"CASE REPORTS AND COMMENTARIES

IN

OBSTETRICS AND GYNAECOLOGY"

SUBMITTED BY

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IN PART FULFILMENT FOR THE DEGREE OF

MASTER OF MEDICINE

IN

OBSTETRICS AND GYNAECOLOGY

OF THE

UNIVERSITY OF NAIROBI

FEBRUARY 2005

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DEDICATION

This book is dedicated to my lovely wife, Rebecca, our two precious sons, Shem and Sammy, and my brother and mentor, Andrew.

ACKNOWLEDGEMENT

It is with solemn and humble submission that I thank God the Almighty for directly or indirectly enablin me to reach this far and to complete this book.

I am sincerely grateful to my sponsor, the Moi Teaching and Referral Hospital, for enabling my pursuit this postgraduate course.

Special thanks and heartfelt appreciation go to my supervisors, Dr. Omondi Ogutu and Dr. Wanyoik Gichuhi, for their invaluable and readily accessible guidance in writing the long commentaries and th short cases in this book. I wish to thank Professor Shadrack B. Ojwang and Dr. Evan Sequeira for the wise counsel during my Elective Term at the Aga Khan Hospital where some short cases in this book we management and the gynaecology long commentary study was carried out.

My gratitude goes to the management and particularly the nurses of Pumwani Maternity Hospital wh unreservedly facilitated my obstetrics long commentary study in the hospital.

While wishing them happiness, I am highly indebted and grateful to the Records Department members staff of the Aga Khan Hospital, Nairobi, who despite their busy work retrieved for me the case records f the gynaecology long commentary without pay.

My sincere gratitude also goes to Alex Wambua who, not only analyzed the data of the two studies, b also facilitated the production of most manuscripts in this book. Tegla Chemalwai and Ruth Korir must acknowledged for their tireless efforts in typing the proposals and most of this book respectively.

I am most grateful to all the Consultants and Senior Registrars for their dedication and commitment seeing that I acquired the necessary knowledge and skills during my training. I acknowledge the close a symbiotic relationship I had with my fellow students from whom I learnt most abundantly.

Equally well appreciated are the nurses, laboratory technicians/technologists, clerks, librarians and all oth people who in one way or another helped me to realize the objectives of my entire postgraduate trainin Most importantly, I thank all the patients without whose cooperation my training would have be impossible.

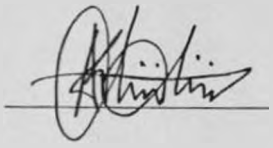
I thank my mother who not only took me to school against all odds but continued to date to pray for me succeed. To my big brother Andrew, thanks for being my mentor and close to replacing my late father.

Lastly but not least, I must thank my wife, Rebecca, and our sons Shem and Sammy for understan tolerating the long duration I was unable to be with them while under training and for encouraging me bad and good times.

DECLARATION

I declare and certify that the short commentaries in this book were managed by me under the clo supervision and guidance of the senior members of staff in the Department of Obstetrics and Gynaecolog of the University of Nairobi.

I further declare that the two long commentaries in this book are my original work and have not be presented for a degree in any other university.

Signed: 

DR. AMON K. CHIRCHIR

FEBRUARY 2005

CERTIFICATION OF SUPERFISION

This is to certify that **Dr. Amon K. Chirchir** researched upon the long commentaries presented in th book under our guidance and supervision and that this book is submitted with our approval.

Signed: _____ Date: 28th January 2005

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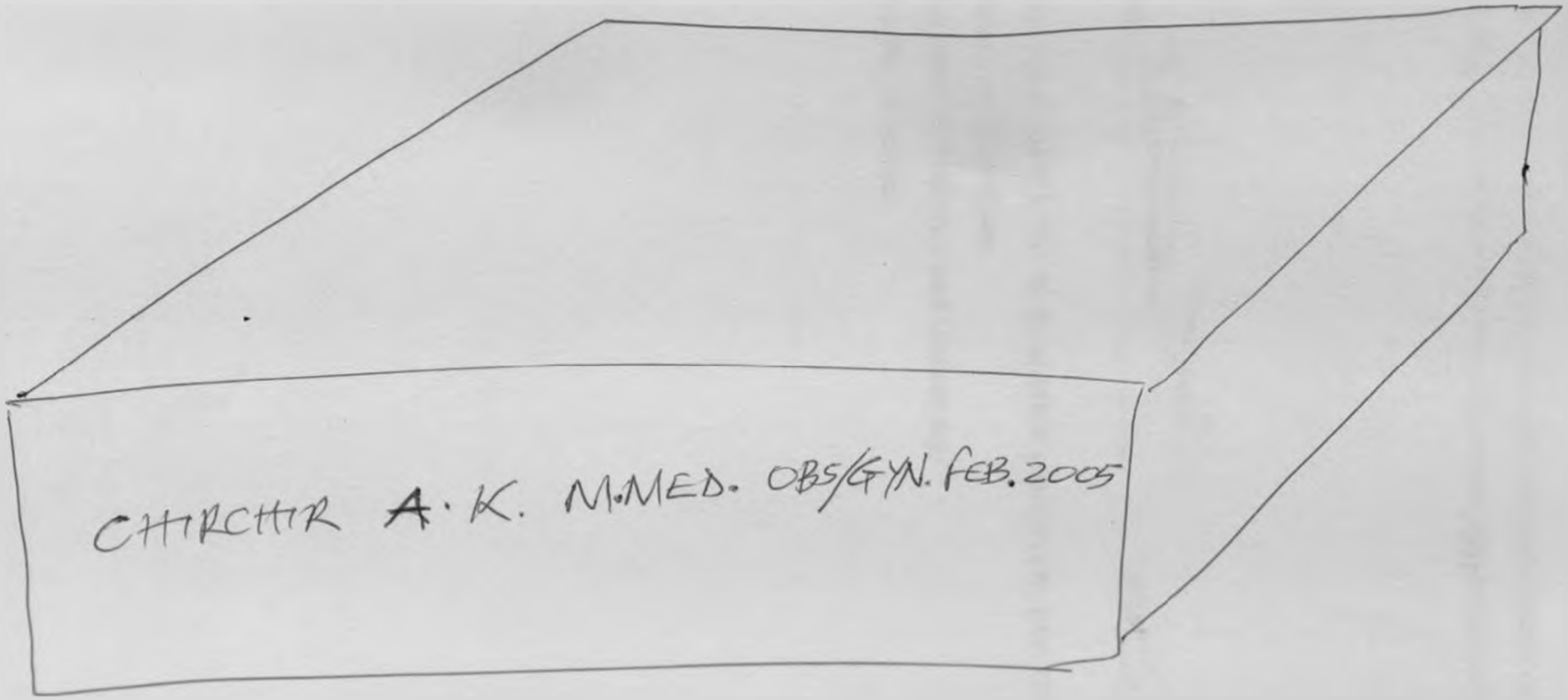
CHIRCHIR A. K. M.MED. OBS/GYN. FEB. 2005

CERTIFICATION

This is to certify that **Dr. Amon K. Chirchir** managed obstetric cases number 7,10, and 13 and gynaecology case number 4 under my supervision and guidance at the Aga Khan Hospital, Nairobi.

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CERTIFICATION

This is to certify that obstetric cases number 4 and 5 and gynaecology cases number 3, 7, 10 and 11 were managed by **Dr. Amon K. Chirchir** under my supervision and guidance at the Kenyatta National Hospital Nairobi.

Signed: _____ Date: 29.01.2005

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CERTIFICATION

This is to certify that obstetric cases number number 2, 3, 6, 14, and 15 and gynaecology cases number 1, 2, 5 and 15 were managed by **Dr. Amon K. Chirchir** under my supervision and guidance at the Kenyatta National Hospital, Nairobi.

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CERTIFICATION

This is to certify that **Dr. Amon K. Chirchir** managed obstetric cases number 2 and 11 under my supervision and guidance at the Kenyatta National Hospital.

Signed: _____ Date: 29/01/05

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CERTIFICATION

This is to certify that **Dr. Amon K. Chirchir** managed obstetric cases number 8, 9and 12 and gynaecology cases number 6, 8, 9, 12, 13 and 14 under my supervision and guidance at the Kenyatta National Hospital.

Signed: Wanjala Date: 29/01/05

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INTRODUCTION

Kenyatta National Hospital (KNH) is currently the only institution in Kenya in which postgraduate degree courses in Obstetrics and Gynaecology are taught by a team of dedicated members of staff from both KNH and the Obstetrics and Gynaecology department of the college of Health Sciences of the University of Nairobi. It is in KNH that most of the short cases were managed between January 2002 and September 2004. The obstetrics long commentary was undertaken in Pumwani Maternity Hospital while some short cases and the Gynaecology long commentary study took place at the Aga Khan Hospital, Nairobi.

KENYATTA NATIONAL HOSPITAL

Established in 1901 as the then Native Civil Hospital, Kenyatta National Hospital (KNH) has over the years grown to be the largest teaching and referral hospital in Kenya with approximately a 2000 bed capacity. It is situated in the heart of Nairobi about three kilometers from the city center. It is built on approximately 304 acres of land. It was called King George's Hospital in the 1950s but acquired its current name in 1964. The hospital serves as a national referral hospital and is also the teaching hospital for both undergraduate and postgraduate students from the University of Nairobi Medical School and the Kenya Medical Training College, both situated within its compound. It serves a large population of Nairobi and its environs as a primary health care institution, and also receives referred patients from neighbouring countries requiring specialized treatment. Under an act of Parliament, the hospital became a state corporation in 1956 managed by a Parastatal Board with the director as the chief executive.

The facility is a complex building that consists of the former old King George's Hospital building and the ultramodern 10-storey tower block that was inaugurated in 1978. The former old hospital building houses the Administration offices the dental department and the faculty of Pharmacy. The 10-storey tower provides the main in-patient facilities while the 1-storey structure has the ICU, Renal, Burns and the Cardiology units and Opthalmology, ENT and Staff clinics in the south of the tower and obstetrics and gynaecology wards in the northern side. Most of the outpatient clinics and the Accident and Emergency (Casualty) unit are located in the east of the tower. To the west are the departments and offices of the College of Health Sciences of the University of Nairobi.

DIVISION OF OBSTERICS AND GYNAECOLOGY

The collaboration of the Department of Obstetrics and Gynaecology of the College of Health Sciences and the department of Reproductive Health of KNH form the Division of Obstetrics and Gynaecology. This division is subdivided into three teams called Firms each headed by a senor consultant. These Firms work separately and collectively to provide training to students and both in-patient and outpatient obstetric and gynaecologic services. The outpatient services are offered at the Accident and Emergency Department, the Antenatal, Postnatal, and Gynaecology outpatient clinics as well as the family planning clinic. The latter clinic also provides day diagnostic laparoscopy and surgical sterilization services. The in-patient services occupy the ground and first floors of the tower block and comprise a labour ward with two operating theatres, three general maternity wards, an emergency gynecology ward, a cold gynaecology ward, a private maternity ward, and Neonatology Unit. The division has been allocated three theatres in the twelve-unit main theatre block.

The Casualty Department

This is one of the busiest units in the division where the Senior House Officer in Obstetrics and Gynaecology sees all patients with obstetric and gynaecologic emergencies. The SHO works with clinical and medical officer-interns and admits those requiring admission to the respective wards and treats and discharges those who may be treated as outpatients. Booked antenatal clinic mothers do not need to pass through the casualty and are usually seen by the Senior House Office in labour ward where they report whenever they have a problem.

Antenatal Care (ANC)

This is provided in the antenatal clinic, which operates every morning from Monday to Friday. Antenatal clients and their partners are booked in on Monday mornings by the Senior Registrars in the unit. Each firm books patients on a weekly rotation and review them on one of the three main clinic days i.e. Firm I on Tuesdays, Firm II on Wednesdays and Firm III on Thursdays. Teenage mothers are seen in a special Monday afternoon clinic specifically for them. Previously the clinic only booked high-risk patients as defined in the World Health Organisation's High Risk Approach to the reduction of maternal mortality. The risk factors included:

- Any pregnancy preceded by a complication that could recur e.g. habitual abortion, neonatal death, caesarean section, vacuum extraction, post-partum haemorrhage etc.
- Primigravidae particularly the adolescents and those above 35 years.
- Medical conditions complicating pregnancy such as hypertension, diabetis mellitus, anaemia and cardiac, renal and thyroid diseases,

- Previous gynaecological problems such as repaired genital fistulae, myomectomy, tubl surgery for infertility or ovulation induction.
- Women with multiple gestation, rhesus negative blood group, breech presentation, pre-eclampsia and grandmultiparous women.

However, this has since been discarded and all pregnancies are treated as a risk and clients are managed as per the Focused Antenatal Care (FAC) programme. In general, for all patients, a record of personal, medical, social and obstetric history is obtained. A physical examination including weight, height, and blood pressure are done at booking and depending on parity and gestational age, a pelvic examination may be done. Antenatal tests done include a urinalysis, haemoglobin measurement, blood group, and serological tests for syphilis. The clients and their partners are offered counseling for HIV testing, which has become a routine test at the clinic. Subsequent management is individualized based on the antenatal profile results and the individual progress of pregnancy.

Antenatal Follow-up:

This is generally carried out once every 4 weeks until 28 weeks, fortnightly till 36 weeks and weekly till delivery. However, since it was shown that number of visits did not reduce risks during delivery, the clinic is in the process of individualizing the number of visits depending on the client and the way pregnancy progresses, with more visits for those with problems and less for those with no problems. This is still being implemented and will gradually take over.

At 36 weeks, all primigravidae have a clinical pelvic assessment unless they already have a condition that precludes vaginal delivery. Clinical and radiological pelvimetry is also performed for those who have had a previous caesarean delivery for a non-recurrent cause and desire a trial for vaginal delivery. Clients who have had repeat caesarean sections and those who have had a caesarean section for a recurrent cause are admitted for elective sections at 38 weeks gestation. If fetal maturity is uncertain, amniocentesis is done and the surfactant bubble test is performed to confirm fetal lung maturity. The clients are admitted whenever delivery is indicated or in labour. All booked clients can report to the labour ward 24 hours a day for any emergency.

THE MATERNITY UNIT AND MANAGEMENT OF LABOUR

The maternity unit consists of the labour ward, three general maternity wards and the Neonatology Unit. The labour ward has an admission office and a records department, 10 rooms for the first stage of labour, 2 delivery suites, a 3 bed acute room, a 5- bed 4th stage room and an operation theatre. All patients are seen

at the admission desk by the Senior House Officer or the Houseman (Intern) and reviewed by the SHO. Those in labour are admitted to labour ward. Antenatal mothers with problems requiring admission are sent to the maternity wards while those who require out-patient treatment alone are treated and discharged to follow-up in the antenatal clinic. Patients in the first stage of labour both from home and the antenatal wards are monitored in the first stage rooms. Each patient is allocated a midwife and is monitored by use of partograph. Once the second stage of labour is reached, the patients are transferred to the delivery suite and prepared for delivery by the midwife. The rooms are equipped with appropriate couches instruments and neonatal resuscitation facilities.

Patients with special needs during labour, delivery and post-partum such as patients with cardiac disease premature labour, severe pre-eclampsia and eclampsia, diabetes, or any medical condition are monitored in the acute room with adult resuscitation and low level support facilities. All emergency and elective obstetric operations are carried out in the adjoining operating theatre. The labour ward team is headed by a senior midwife who acts as the team leader. The others include midwives, two senior house officers housemen and support staff. A senior registrar and a consultant are on call for difficult cases. The neonatal unit is on the firs level and is run by neonatologists from the Department of Paediatrics. The neonatal unit has four nurseries and is in the process of establishing a Neonatal ICU. It also has isolation wards for neonates with infection and those born outside the hospital. The neonatal unit is managed by an SHO in paediatrics and also has a senior registrar and consultant on call. The SHO is available for all deliveries requiring special neonatal care including all babies delivered by caesarean section.

Management of First Stage of Labour:

The partograph is the cardinal instrument used to monitor the progress of labour. Although it has served quite well, the current partograph includes the latent stage of labour that has led to labour being considered prolonged when the prolonged phase was only the latent phase. This has led to interventions that were probably unnecessary. Consequently, trials are currently underway to introduce modified partographs that begins monitoring of labour during the active phase of the first stage of labour rather than the incumbent that includes the latent stage of labour. The partograph is a comprehensive single-paged document on which the progress of labour and foetal and maternal well-being are recorded. The intensity and frequency of uterine contractions, fetal heart rate, maternal pulse and blood pressure are recorded half hourly. An abdominal and vaginal examination to assess descent, cervical dilation in centimeters, presence and degree of moulding, caput succedaneum formation and colour of draining liquor is done every 4 hours. Artificial amniotomy is performed for all clients in active phase of labour at cervical dilatation of 6 cm or more. However, this is not done in patients with unknown HIV sero-status and those who are HIV seropositive

unless there is an obstetric indication or they have attained cervical dilatation of 7 cm or more. For it has been shown that artificaila rupture of the membranes in HIV positive mothers is associated with increased risks of mother to child transmission.

There are two lines on the partograph: the "alert" and the "action" lines. The "action" line is four hours to the right of the "alert" line. Upon admission, for parturients in active phase of labour, cervical dilatation is marked on the "alert" line and the time noted. Cervical dilation of at least 1 cm per hour is expected. Any deviation of cervical dilation curve towards the action line is an indication of some abnormality in the progress of labour and corrective measures are instituted accordingly. Analgesia is provided during labour but is not mandatory if the patient requests not to have any, which is rare. Pethidine and Tramadol are the most used drugs during labour. A 100mg of intramuscular pethidine is given 4 hourly until a cervical dilatation of 6cm subsequent doses are given in aliquots of 25mg intravenous as necessary. Tramadol is also widely used as it has the advantage of causing less neonatal respiratory depression. However, it is causes emesis and is often given with metoclopramide. Augmentation of hypotonic uterine contractions is carried out using an intravenous infusion of oxytocin. Membranes may also be ruptured to augment labour. However, this is done in labour already being augmented to monitor liquor staining. Patients in labour have their blood taken for grouping so that blood may be readily available if required.

Management of second stage of labour

When the second stage of labour is reached, the patient is transferred to the delivery suite. The midwife or doctor conducting the delivery then wears a mask and protective goggles, scrubs and done sterile gown and gloves. The vulva and perineum and swabbed with antiseptic solution such as 2% chlorhexidine. The patient is draped and then asked to bear down once the baby's head has crowned. The baby's head is allowed to gently distend the perineum until it is delivered with the midwife providing perineal support. Once the head is delivered, mucus is cleared from the baby's nose using a soft piece of sterile gauze. A finger is gently passed round the baby's neck to check for the presence of the cord. If found round the neck and loose, it is gently slipped over the head and the rest of the baby delivered. If it is tight, the cord is ligated between two clamps and then the baby delivered. The baby's body is delivered after restitution by gentle downward traction to deliver the anterior shoulder and then the posterior shoulder and the rest of the body. The cord, if not already divided is then divided between two clamps and the baby is wrapped in a towel and put on the mother's abdomen. If suction is required it is done on the resuscitaire which also provides warmth.

Episiotomies

Episiotomies are no longer given routinely during the second stage of labour. The use of episiotomies is now individualized. Episiotomies have been associated with increased risk of mother to child transmission of HIV. When required, a midline but mostly a medio-lateral episiotomy is performed. During a normal vaginal delivery, the distendend perineum is infiltrated with 1% lignocaine solution and using a special mayo episiotomy scissors, an episiotomy is made during the height of a contraction. It is then packed to stem bleeding from cut vessels. The baby should then deliver easily.Episiotomies are also performed during vacuum extractions where the perineum is liable to sustain bad tears.

The episiotomy is repaired using absorbable suture from its vaginal apex by placing a stitch above the apex and continuing till all the vaginal mucosa is closed. The perineal muscles are then approximated using interrupted stitches. The skin edge is then apposed using interrupted subcutaneous stitches with buried knots to prevent perineal irritation and discomfort. The patient is advised to have daily sitz baths till healed.

Management of third stage of labour

The third stage of labour is actively managed in the labour ward. With the delivery of the anterior shoulder, 0.5mg of ergometrine sulphate or 10 IU of oxytocin is given intramuscularly. In unbooked mothers, this is given after delivery of the baby and the presence of a second foetus is ruled out. For patients in whom ergometrine is contraindicated, 10mg of oxytocin is given intramuscularly. In fact oxytocin given intramuscular is becoming the drug of choice in the management of the third stage of labour in the unit as it is associated with fewer side effects compared with ergometrine. The placenta and membranes are delivered by controlled cord traction after the signs of separation have occurred. The membranes and placenta are examined for competeness and the birth canal is inspected for tears. Episiotomies and any perineal rears that need repair are then repaired. The patients's blood pressure, pulse and themperature are then recorded and the patient encouraged to empty her bladder. The mother and baby are then put together in the 4th stage room and observed for one hour until she is taken to the maternity ward to rest.

OPERATIVE VAGINAL DELIVERIES

Vacuum extraction

The vacuum extractor is solely used for all operative vaginal deliveries in the unit. It is mainly used to deliver patients with prolonged second stage of labour with poor maternal effort or in cardiac patients where bearing down is contraindicated or not possible as in sedated patients with eclampsia or pre

eclampsia. It is also used to expedite delivery in cases with foetal distress in the second stage of labour Only low vacuum extractions are usually performed. Mid-cavity vacuum extractions may be performed fo severe fetal distress in the absence of obstructed labour. High vacuums are contraindicated and caesarea sections preferred in their stead.

The patient is informed about what is going to happen and a written consent is obtained for the procedure The patients with cardiac disease are usually prepared antenatally for this mode of delivery. The patient i palced in the lithotomy position or in the case of cardiac patients, in a semilithotomy position. Th perineum cleaned with antiseptioc solution and draped. The perineaum is infiltrated with 1% lignocain and a mediolateral episiotomy is performed if required. Two types of cups are available for use, the meta and the plastic (mouldable) cups. The largest suitable cup is applied to the vertex taking care not to includ maternal tissues. A negative pressure of $0.8kg/cm^2$ is created in a stepwise $0.2kg/cm^2$ every 2 minutes The negative pressure causes the fetal scalp to be drawn into the cup creating a chignon. The plastic cu creates a less noticeable chignon than the metal cup but is prone to slip more. After a pressure o $0.8kg/cm^2$ is reached, traction is applied on the fetal head downwards following the pelvic curve intil th head is crowned, then upwards to deliver the foetal head by extension. After the head is delivered, th pressure is released and the rest of the body is delivered as for a normal vaginal delivery. The neonate i usually then handed over to the neonatologist who is present for the delivery for examination an resuscitaion where necessary.

CAESAREAN SECTION

Nearly all caesarean sections performed in the unit are transverse lower uterine segment caesarean section (LUSCS). Classical caesarean sections are rarely performed except when it is a repeat classical section o when the foetus is in a transverse lie with ruptured membranes and the uterus is moulded on the foetus The transverse incision is the method of choice the world over as it is associated with less blood loss, it i easier to repair, is less likely to rupture during subsequent pregnancies and usually does not promot adherence of bowel or omentum to the incision site.

Procedure for caesarean section

Once a caesarean section is prescribed or elected as the mode of delivery, the patient is counseled an informed about what the procedure entails and why it is being performed. A written consent is obtaine from the patient. For patients under 18 years of age, a written consent is always obtained from th guardians in case any intervention is required during labour. In emergencies or in cases where the patien may not be able to provide her own consent, and in the absence of guardians, the senior consultant in th

unit provides the written consent on the patient's behalf. Blood if not already obtained, is taken to enable crossmatch of units of blood, which are then reserved for the operation. The theatre staff and surgeon are informed and premedication usually atropine 0.6mg is given intramuscular, the abdomen and mons pubis hair is clipped and the patient wheeled into the operating theatre.

Anaesthesia for caesarean section may be spinal (regional) or general anaesthesia with endotracheal intubation and controlled ventilation. For spinal anaesthesia, 0.5% bupivacaine is injected intrathecal under aseptic technique and the patient positioned on the couch tilted about 15° to the left. For general anaesthesia, the patient is put in the same position on the couch and given oxygen by mask.

The abdomen and perineum are cleaned and draped and urethral catheter is inserted to drain urine before and during the procedure. The abdomen is draped and then crash induction of anaesthesia with endotracheal intubation performed for the patient to have the operation under general anaesthesia. The lower midline incision is preferred for abdominal access especially when repeat sections are anticipated. The pfannensteil and Joel Cohen incision are also used. Once the abdomen is entered, the gut is packed away from the uterus using wet warm packs. A doyen retractor is placed over the bladder and the vesico-uterine peritoneum incised transversely. The bladder is deflected downward and retracted away from the operating field. The lower the uterine segment is thus adequately exposed. A small transverse incision is made through the myometrium in the midline 2cm above the deflected bladder. The incision is then extended bilaterally using the forefingers of both hands simultaneously to about 10cmj. or widened using curved round tip scissors, taking care to curve the incision distally to avoid damaging the uterine vessels. The membranes are incised and liquor amni drained by suction. The retractor is removed and the surgeon's hand is slipped into the uterine cavity between the lower segment and the presenting part. The hand then guides the presenting part through the incision.

In cephalic presentation, the head is delivered and the mouth and nose cleared of secretions. The rest of the body is delivered gently as for vaginal delivery. Patients with prolonged obstructed labour may present with the foetal head deeply impacted in the pelvis. The head may be gently disimpacted vaginally by a third operator wearing high-level disinfection gloves while the surgeon maintains flexion to prevent precipitate delivery through the incision once the head is disimpacted. Alternatively, the baby may be easier delivered by breech extraction with gentle disimpaction if the foetal head once the rest of the body is delivered. In breech presentation, gentle total breech extraction is performed.

Once the baby is delivered, the cord is divided between two clamps and handed over to the neonatologist for sesuscitation. 0.5mg ergometrine is given intramuscular or intravenous. 10 iu of oxytocin may also be given. The placenta is delivered manually and examined for completeness. The uterine cavity is cleaned and the edges opposed in one or two layers using absorbable suture – usually No. 2 chromic catgut or No. 1 Vicryl ensuring the edges of the incision are well tied to ensure haemostatis. The vesico-uterine peritoneum may be sutured with 2-0 absorbable suture or left to heal on its own. The abdominal packs are removed and the abdominal organs inspected. The swabs and instruments are counted and once all is accounted for, the abdomen is closed in layers or en mass using No. 1 Vicryl. Subcutaneous fat is approximated with plain catgut to obliterate dead space. The skin is often closed using interrupted vertical mattress or continuous subcuticular stitches. The most popular sutures are nylon 3 – 0, or vycril 3 – 0. The incision site is then cleaned with antiseptic and dressed with dry sterile dressing. Vulvo-vaginal toilet is performed and all uterine clots are expelled. Urine is checked to make sure it is still clear and the catheter is left in situ, to be removed after 12 hours or usually on the first post-operative day. General anaesthesia is reversed and the patient taken to recovery ward. Patient's who have had spinal anaesthesia are joined by the babies and leave the operating theatre faster.

Postoperative Care

The patient's vital sighns are observed continuously in the recorvery ward until completely awake from the anaesthesia then 2 – 4 hourly thereafter. They are then taken back to the labour ward where they join their babies before being transferred to the maternity ward. Patients requiring special care are taken to the acute room in labour ward till well enough to be taken to the maternity wards. Postoperative antibiotics are given intravenously for 24 – 48 hours. Crystalline penicillin G or Ampicillin and Gentamicin are the antibiotics of choice. Metronidazole is added for patients who have been referred with prolonged obstructed labour, prolonged rupture of the membranes, chorioamnionitis, or ruptured uterus. Postoperative analgesia is maintained with pethidine 100mg 6 – 8 hourly regularly for the first 24 hours and then on an as necessary schedule, and 75 mg intramuscular diclofenac sodium. Patients are ambulated within 12 hours of surgery and usually begin to take oral feeds within 12-24 hours. By the third day, patients with uncomplicated caesarean section are on oral medications and many are ready to be discharged. Most of our patients are allowed hom on the 4th day. Post operative haemoglobin measurement is checked on the third day. Removable interrupted sutures are removed once the wound has healed, usually on the 7th day. This can be done as an outpatient procedure. Patients are discharged on oral antibiotics, usually amoxillin and analgesics. They are seen in the post-natal clinic between 4 and 6 weeks post partum.

Baby Friendly Institution

Kenyatta National Hospital is a baby friendly institution meaning that at no time except under very specia circumstances is the baby separated from the mother. Rooming in is practiced and breastfeeding excep where contraindicated like in patients with HIV, is established immediatedly after delivery and encouragec on demand throughout the hospital stay. During the postpartum period, staff provide practical tips on baby care and breastfeeding that is greatly appreciated by new primipara.

Postnatal Follow-up

The postnatal clinic runs every Friday morning. The Senior House Officers see patients who have ha operative deliveries or complications during pregnancy.

The other clients are seen by the midwives or at their nearest health facility. During this time, a history o the puerperium, lactation and baby's health and immunizations is taken. The clients are examined onc again and any problems discussed. Contraception is also discussed during this visit and the suitabl preferred method is provided at the family planning clinic.

The Family Planning and Welfare Clinic

This clinic provides advice and services of most contraceptive methods available, both temporary an permanent methods for both men and women. It also provides counseling on family matters an counseling and treatment of sexually transmitted infections. The contraceptive services provided includ combined oral contraceptive both 2nd generation and the 3rd generation sequential pills, progestagen onl pills, injectable contraceptives (Depot medroxy-progesterone acetate, noresterat® and the one monti injectable - norygynon® norplant® implants, copper based intrauterine devices, and barrier methods Vasectomy and interval bilateral tubal ligation done by minillaparotomy or laparoscopy are performed a day cases. Immediate postpartum tubal ligation is also performed.

GYNAECOLOGY UNIT

This comprises a gynaecology out patient clinic that runs from Monday to Thursday in the afternoons an two gynecology wards. Patients seen at casualty with gynaecologic emergencies are admitted to th emergency gynaecology ward 1D which handles all emergencies 24 hours a day. Cold cases are treate and/or referred to the clinic as well as oncology patients on chemotherapy who report directly to the war then therapy is due.

Gynaecology Outpatient Clinic

This clinic runs from Monday to Thursday afternoon. The Monday clinic is an infertility clinic dealing with infertile couples. Each of the three firms has a clinic day. Common problems seen at the clinic include infertility, uterine fibroids, and abnormal uterine bleeding. Patients from the clinic who require surgical intervention are admitted to the cold gynaecology ward and managed. Oncology patients are also followed up in the clinic once therapy is completed. This includes those whose only intervention was surgical.

Acute Gynacological Admissions – Ward 1D

Admissions to this ward are through the casualty. Patients admitted have been seen by the SHO and need in-patient care, surgery or minor surgical procedures. Most of the patients have complications arising in early pregnancy or the puerperium. Common problems encountered include incomplete abortion and complications arising from abortion such as sepsis and severe anaemia. Evacuation of retained products of conception is the commonest procedure done. This is performed by manual vacuum aspiration using Karman's cannula and a double valve syringe. The procedure is carried out under analgesia and mild sedation. Patients who need cervical dilation are evacuated under general anaesthesia in the operating theatres. Uncomplicated cases are managed as out patients. Other emergencies encountered include ruptured ectopic pregnancies, pelvic abscesses, and torsion of ovarian cysts. Bartholin's abscesses and cysts are also not unusual.

The ward also admits patients with cancer of the cervix who need intervention before specific management such as blood transfusions, rehydration or general work-up before examination under anaesthesia (EUA) and biopsy, or radiotherapy.

Cold Gynaecological Admissions – Ward 1B

Beds in this ward are divided among the 3 operating firms. Each firm admits its patients from their clinics and manages them. Surgery performed includes hysterectomies, repair of obstetric fustulae, oncologic surgery, tuboplasty, colporrhaphy, perineorrhapht and various surgeries to correct development anomalies of the mullerian tract such as vaginal atresia, and uterine septi. Patients are usually admitted when ready for surgery except under special circumstances such as cancer patients who may need in-patient care before surgery. All diagnostic work up is done before admission.

GYNAECOLOGICAL OPERATIONS

All operations, emergency or elective are carried out in the main operating theatres on designated days. Emergency operations are allocated a specific theatre daily. Laparotomy for ectopic pregnancies (ruptured

and non-ruptured), pelvic absecesses, ovarian cyst and other tubo-ovarian masses are done here. Smaller procedures like diagnostic dilatation and curettage of the uterus, removal of misplaced intra-uterine contraceptive devices, marsupilization of bartholin's abscess and suction curettage are also carried out in the same theatre.

Scheduled operations are done on firm basissuch that Firm II operate on their patients on Mondays and Firms III and I on Thursdays. The VVF team also operate on Thursdays and occasionally on Fridays. The operations are generally performed under general anaesthesia as outlined below:

- Intravenous sodium thiopentone and succinylcholine are used for induction of anaesthesia.
- Nitrous oxide, oxygen and halothane provide maintenance of anaesthesia.
- Curare is given intermittently for muscle relaxation.
- Atropine and neostigmine are used for reversal.

Pre-operative preparations

All patients for emergency operations are prepared for theatre on admission. Ruptured ectopic pregnancies are the most common indications for emergency laparotomy. In this case blood is urgently cross-matched and an intravenous drip of N/saline started. The abdomen is cleaned and shaved. Pre-medication is provided by atropine 0.6mg intramuscularly half an hour before theatre. An informed written consent is taken before theatre. For cold (non-emergency) operations, baseline investigations such as the full haemogram, urea and electrolyte levels are done and the date of surgery fixed. The nature and purpose of the operation is explained to the patient and an informed written consent for the operation is obtained. Blood is ordered and reserved for the day of the operation. For most operations gut preparations is done by enema at 6.00 a.m. on the operation day. The operation is cleaned and shaved. Pre-medication is provided by atropine 0.6mg and pethidine 50-100mg intramuscularly half hour before theatre.

Post-operative management

After the operation general anaesthesia is reversed and the patient wheeled to the recovery room where half hour observations of blood pressure, pulse rate, respiratory rate and temperature are monitored until the patient is fully awake and stable. She is then transferred to the ward where observations are done 4 hourly.

Patients who have had uncomplicated laparotomy for hysterectomy, ectopic pregnancy, ovarian cyst etc are usually kept in the ward for 4 days. For the 24 hours the patients are maintained on intravenous fluids. Oral fluids are given when bowel sounds are established. Blood transfusion is given when indicated. Pethidine 50-100mg 6 hourly for 24 to 48 hours is routinely given for analgesia. Prophylactic antibiotics

are given routinely. A check hemoglobin level is determined on the third post-operative day. Before discharge, the patient is informed about the findings at operation and a discharge summary is issued patients are reviewed in the gynecology clinic after six weeks or earlier when there is an indication. The most common acute gynaecological operation is laparatomy due to ruptured ectopic pregnancy while total abdominal hysterectomy is the commonest cold gynaecological operations done in this unit. Total abdominal hysterectomy is described below.

Total abdominal hysterectomy

General anaesthesia, induction and maintenance are done as described above. A vulvo-vaginal toilet is done with antiseptic solution such as hibitane or savlon. Aseptic catheterization is done next and the catheter left in situ to maintain continuous bladder drainage during the operation. Pelvic examination under anesthesia is done and findings noted. The vagina is then painted with methylene blue dye. The abdomen is thoroughly cleaned with hibitane or savlone and painted with iodine and then drapped with sterile towels.

The abdomen is opened in layers either through a Pfannenstiel incision or through a lower midline incision. The intestines are packed away from the intestines with wet gauze packs and a self-retaining retractor applied. The round ligaments are identified and beginning on either side using straight long artery forceps the round ligament is double clamped and divided between the two forceps. The lateral stump is transfixed with No 0 or No. 1 vicryl. This procedure opens the anterior leaf of the broad ligament, which is pushed forwards through this opening with a surgeon's finger and incised with scissors. The same is done for the opposite side. The next step depends on whether the tube and the ovary are to be saved or removed. If they are to be saved, the tube and the ovarian ligament are double clamped en masse and cut using a scalpel. The distal clamp holds the ovarian vessels as they approach the anastomosis with the uterine vessels. This stump is ligated using a transfixed vicryl No. 1 or No 0. The same is done for the opposite side. If the tube and ovary are to be removed with the uterus, the infundibulo pelvic portion of the broad ligament is double clamped with long curved artery forceps with the tips reaching the open window in the broad ligament. The ligament together with ovarian vessels is divided between clamps and ligated using vicryl No. 1 or No. 0. The same is done for the opposite side.

The reflection of the bladder peritoneum onto the uterus is then freed by extending the incision in the anterior leaf of the broad ligament towards the midline. The bladder is thus separated from the lower uterine segment, the cervix and the vagina by careful sharp and blunt dissection of the fascial fibres beneath the bladder wall. Usually the bladder can be displaced into the lower pelvis quite easily, but if it is adherent, it is surgically released and not bluntly forced.

Next the posterior leaf of the broad ligament on either side is cut parallel with the side of the uterus to better demonstrate and skeletonise the uterine vessels between the leaves of the broad ligament for clamping. The uterine vessesl are double clamped and cut using a scalpel and freed from the uterus by extending the incision around the tip of the distal clamp. This enables adequate ligation. Care should be taken to avoid freeing the tissue beyond the tip of the clamp, as this could permit bleeding from the collateral vessels that are not included in the clamp. Before clamping and cutting the uterine vessels, it is always advisable to palpate the lower portion of the pelvic ureters as they course beneath the uterine artery lateral to the internal OS and pass medially through the base of the broad ligaments to the trigone of the bladder. The uterine vessels are ligated with vicryl No. 1. The same is done for the opposite side.

The uterus is retracted forward and upward to demonstrate and stretch the uteroscral ligaments posteriorly. A transverse incision is made through the uterine reflection of the cul-de-sac peritoneum between the attachments of the two uterosacral ligaments. The peritoneum is then incised with the scalpel and reflected, mobilizing it past the cervix to the posterior vaginal fornix. Care is taken not to dissect extensively laterally where the haemorrhoidal vessels are inserted into the rectum. Each uterosacral ligament is double clamped, cut and ligated with a No. 1 vicryl suture. Here particular care is exercised to avoid the pelvic portion of the ureter as it courses along the base of the broad ligament. Next the cardinal ligaments on either side of the uterus are clamped, cut and ligated.

More commonly the uterus is removed by the open technique in which the anterior fornix is opened initially with the scalpel and the vagina is circumcised by a sharp knife or scissors. As the anterior, posteriror and lateral margins of the vagina are opened, straight artery forceps are used to secure vaginal margins. These margins are then closed using a series of figure-of-eight sutures. Particular care is taken when tying the lateral angles to ensure the descending vaginal branches of the uterine vessels are securely ligated.

Suspension of the vaginal vault is done by tying the peritonization suture to the lateral and mid sutures of the vault. Peritonzation is accomplished by means of a continuous No. 1 chromic catgut suture, that first pierces the vaginal walls near the midline and passes throught the posterior leaf of the broad ligament, the free margin of the uterosacral ligament, then through the infundibulo-pelvic ligament, the free margin of round ligament and the anterior bladder peritoneum. The suture is tied at the centre. The same is done for the opposite side with the suture being tied at the midline and lateral angles. If the ovaries have been preserved an alternative suspension may be used in which the tip of the broad ligament is stitched

separately with a purse string of No. 2/0 chromic catgut. The free margin of the pedicle is left high against the pelvic wall and is not anchored to the vaginal vault. This is advised in order to avoid subsequent dyspareunia and avoid stretching of the ovarian vessels with possible thrombosis, ischaemia and cystic changes of the ovary. After this abdominal vicera are well inspected. If haemostasis has been achieved, and instruments and swabs count are normal, the abdomen is closed in anatomical layers.

The post-operative management is as described above.

COUNSELLING CLINICS

There are three such clinics in the hospital, which offer counseling to obstetrics and gynaecology patients. These are the patient support centre, GOPC, teenage clinic and the Nairobi Hospice.

The patient support centre

This is situated in the old hospital buildings wherepatients regularly attend from all the departments of the hospital. Sometimes the counselors are called to the wards to counsel those patients who cannot go there. The counselors consist of psychiatrists, sociologists, psychologists and trained nurses. Mostly, they deal with HIV counseling, puerperal psychosis patients and those patients who are poor and neglected by relatives. They counsel, treat and even assist patients find their way home.

The high risk clinic (HRC)

This clinic is situated on the gound floor next to the maternity wards. It deals with young single mothers who have had an abortion those who have delivered babies and even those who do not want to bear up their children. The counselors are also trained nurses, sociologists and consultant obstetrician/gynaecologists.

They counsel their clients, treat them for any illness they may have with assistance from the obstetric and gynaecology wards, and also provide them with family planning and STD management services. The patients come from other institutions or from the obstetrics and gynaecology wards.

The Nairobi Hospice

Workers here also offer counseling care in addition to management of terminal disease. They also offer narcotic analgesia and encourage homebased care for such patients instead of hospital care. Most of their patients have cancer of the cervix.

The Hospital Chapel

This provides spiritual nourishment to those patients who are in need. It is situated on level 2 of the tower block.

THE MOTHERS' HOSTEL

This accommodates mothers with babies in nursery. When they get sick, they are treated from the wards where they were initially admitted.

COLPOSCOPY AND CANCER OF THE CERVIX

Cancer of the cervix is the commonest cancer occurring in Kenya. It is also the leading cause of cancer deaths in the country. Tragically, cancer of the cervix is preventable. All patients and female staff working in the unit are encouraged to have pap smears regularly in order to prevent this tragedy. The gynaecology clinic offers colposcopy training and services to both medical practitioners and patients respectively. Patients with abnormal pap smears are referred to the clinic and colpscopic directed biopsies performed or ablation of the abnormal zones using laser or cryotherapy using carbon dioxide. Cone biopsies can also be performed at the clinic as a therapy for patients who desire future fertility. Some patients with carcinoma in situ opt to have a hysterectomy. Unfortunately, many patients seen at the clinic present with cancer of the cervix that is too advanced for colposcopy. They are prepared at the clinic to have EUA and biopsy as day cases in preparation for radiotherapy.

LAPAROSCOPY

Diagnostic laparoscopy is carried out in the Family Welfare Clinic usually for patients with infertility to assess suitability for surgical intervention. Recently, laparoscopic surgery was introduced and operations such as tuboplasty, removal of small ovarian cysts and unruptured ectopic pregnancies are being performed. With more users becoming competent, it is hoped that more surgeries can be performed laparoscopically.

OBSTETRIC CASE No 1

PRE-ECLAMPSIA AT 31+ WEEKS, EMERGENCY CAESAREAN SECTION AT 34 WEEKS-LIVE BABY.

Name: S.M	LNMP:	10/9/03
Age: 32 years	EDD:	17/06/04
IP NO: 0956108	GBD:	31+ Weeks
Parity: O+O	DOA:	20/04/04
	DOD:	22/05/04

Presenting complaint

S.M. was referred to Kenyatta National Hospital (KNH) with one week's history of upper and lower limb swelling and elevated blood pressure.

History of Presenting Complaint

She had been booked and followed up uneventfully from 24 weeks gestation at Githurai Medical Centre until a day prior to admission when she was noted to have a BP of 150/95mmHg and leg swelling. She was asked to have a bed rest and to report to the clinic the following day for repeat BP measurement. A repeat BP was 150/110mmHg and she complained of throbbing headache and occasional blurred vision. Foetal movements were unaltered. She was, therefore, referred to KNH. A repeat BP at KNH was 160/110 mmHg and a dipstick urinalysis indicated trace proteinuria. Her antenatal care (ANC) card indicated she had a baseline BP of 110/70 mmHg at 24 weeks.

Past Obstetric and Gynaecologic History

S.M was a para 0+0 gravida 1 at 31+ weeks. Her last normal menstrual period was 10/09/03 and her expected date of confinement was 17/6/04. She attained menarche at 14 years and had regular periods every 28-30 days with menstrual flow duration of 3 – 5 days and associated mild dysmenorrhoea. She had coitarche at 28 years and had used combined oral contraceptive pills for a year before she stopped to conceive.

Past Medical History

She neither had history of elevated BP before nor history of features of hyperthyroidism or renal disease.

Family and Social History

S.M was a married unemployed, trained copy typist who did not smoke tobacco or consume alcohol. She lived in Githurai with her husband who worked as a procurement officer with the Nairobi City Council. She had no history of hypertension, diabetes mellitus or other familial condition in her family.

PHYSICAL EXAMINATION

General Examination

She was in fair general condition with mild facial puffiness and moderate bilateral pitting pedal oedema. She had a BP 160/110 mmHg, Temp 36.6°C, respiratory rate: 18 min, PR: 8.4/min, regular and of good volume.

Abdominal Examination

The abdomen was uniformly distended and had no surgical scar. The fundal height corresponded to 30 weeks; the foetus was in longitudinal lie, cephalic presentation and had a regular foetal heart tone of 136/min. She had no other organomegally, no tenderness and the foetal head floated freely at the brim.

Cardiovascular, respiratory and nervous systems were essentially normal as were the breasts and thyroid glands.

Pelvic examination

This was not done, as there was no indication.

Impression:

Thirty two year-old Primigravida with severe Pre-eclampsia at 31+ weeks gestation.

Management

Sublingual 10mg of nifedipine was given to her in casualty and subsequently got admitted for BP control, investigations and monitoring of foetal and maternal well-being. She received 500mg of methyldopa, 30mg of phenobarbitone and 25mg of hydralazine all 8 hourly. She got 6 hourly BP recordings, daily urinalysis and weighing and recorded daily foetal movements on a foetal kick chart.

Investigations:

These included haemogram, renal function tests, liver function tests, obstetric ultrasound scans, daily urinalysis. Uric acid levels were requested but the reagents for testing it were unavailable.

Renal Function Tests

Date	*21/05/04*	*30/05/04*	*05/05/04*	*09/05/04*	*18/05/04*
K+ (mmo/L)	4.3	4.1	4.2	4.3	4.3
Na+(mmo/L)	130	136	138	135	146
Urea(mmo/L)	2.2	2.4	6.0	6.1	4.6
Creatinine (μmo/L)	61	63	7.3	80	76

Haemogram

Date		*21/05/04*	*30/05/04*	*10/05/04*	*Normal Ranges*
Haemoglobin	(g/dl)	14.2	13.8	12.5	11.5 - 14
WBCC	($x10^9$/l)	8.3	7.9	8.3	4.0 - 11
Platelets	($x10^9$/L)	20.8	18.4	144	>100

Liver Function Tests

Date		*21/05/04*	*09/05/04*	*Normal Ranges*
Total Protein	(g/dl)	75.0	50.0	60 – 80
Albumin	(g/dl)	34.2	23.5	25 – 35
Bilirubin	(μmol/L)	16.2	14.5	upto 17
SGPT	(IU/L)	36.0	37.5	upto 50
GOT	(IU/L)	32.0	34	upto 40

Blood Pressure, Fetal Movement and Urine Analysis Charts

Date	*BP (mmHg)*	*Proteinuria*	*Foetal mvt*	*Treatment*
20/04/04	150/110	2+	OK	Methyldopa 500mg
24/04/04	180/100	2+	OK	+ Hydralazine+adalat
28/04/04	160/100	2+	OK	I.M.hydralizine +dexamethasone
04/05/04	190/110	3+	OK	Above continued
05/05/04	180/100	3+	OK	+Atenolol
10/05/04	190/130	3+	Reduced	Emergency cesarean section

Obstetric Ultra-sonography scans:

23/04/04. Single intra-uterine pregnancy at an average computed gestational age of 27 weeks six days with probable asymmetric intrauterine growth restriction. The foetus had foetal heart rate of 128/min and was estimated to weigh 1058 grammes. Amniotic fluid was adequate and biophysical profile score 7/8.

06/05/04: Average computed gestatinal age of 28 week+4 days. No diastolic flow and umbilical artery resistive index was very high (no figure given). Biophysical profile was 6/8 with reduced amniotic fluid. Because of these findindings and the uncontrolled BP, severe headache and visual disturbances, a decision was made to deliver the baby. However, the patient declined. She was therefore asked to chart a fetal movement chart for fetal monitoring.

Emergency cesarean section

In view of satisfactory foetal kick chart, significant weight gain and patients refusal to undergo delivery earlier decision for delivery was differed. However, on 10/05/04, at 34+ weeks gestation, the patient developed severe hypertension (BP 190/130 mmHg) associated with severe headache, blurred vision but no epigastric pain. She also reported reduced foetal movements. These being some of the 4 signs of impending eclampsia and with poor modified Bishop's score the patient was quickly prepared for emergency lower uterine segment caesarean section. Informed consent was obtained, pubic hair shaved, blood taken for grouping and cross-match and premedication with intramuscular 50mg of pethidine and 0.6mg of atropine administered. Caesarean section was undertaken as described under introduction. By cephallic extraction a life female infant with a birth weight of 1350gms and Agpar score of 6/1, 8/5, 9/10 was delivered. Due to a moderate respiratory distress and the prematurity, the infant was admitted to the newborn unit.

Postoperative care:

The patient's blood pressure, input/output chart and vital signs were closely monitored. By the 10th post operative hour she had BP of 140/90 mmHg and other monitored parameters were satisfactory. Oral sips to graduate to light diet and ambulation were advised. On her 2nd post-operative day, however, she was found to have elevated BP of 160/100mmHg associated with worsening oedema. Though urine output was satisfactory, facial puffiness and headache had recurred. Methyldopa and nifedipine were reinstated and the BP gradually reduced to 140/90 mmHg by the 6th post-operative day.

On the other hand her infant recovered remarkably well from the respiratory distress syndrome. And by the 12th post-operative day both the mother and her baby were ready to be discharged. S.M went home on 20mg nifidepine 12 hourly and advice to be reviewed in a week in the postnatal clinic.

Follow-up

She was seen as scheduled in the postnatal clinic her blood pressure was 135/85 mmHg. She was in good general condition with no periorbital oedema. No abnormality was detected in her breasts, abdominal and vaginal examination. Nifedipine was discontinued and she was allowed home on advice to revisit the postnatal clinic in her 6th postnatal week. On her sixth week postpartum, she had a normal BP of 120/70mmHg. Her breasts, abdomen and pelvic examination revealed no abnormality. The uterus was not palpable abdominally. Nifedipine was discontinued and contraceptive advice given. She opted for copper T. 380 intrauterine contraceptive device and she was referred to the adjoining Family Welfare Clinic for the same.

DISCUSSION

S.M was a 27 year-old primigravida who developed severe pre-eclampsia in the early third trimester of her pregnancy. Despite intake of 4 antihypertensive drugs her worsening elevated blood pressure, deteriorating renal function tests and foetal and maternal well-being and poor bishop score necessitated delivery by emergency caesarean section.

Hypertension is defined as two blood pressure (BP) readings of 140/90 mmHg or more at least 6 hours apart, or an increase in mean arterial blood pressure of at least 20 mmHg. The use of an increase of blood pressure of 30/15 mm Hg over first trimester values is controversial.[1,2]. Hpertensive states in pregnancy can occur at any gestation and are classified into the following groups:[1,2]

1. **Gestational hypertension (formally pregnancy induced hypertension or transient hypertension):** Hypertension for the first time during pregnancy without proteinuria, BP returns to normal in 12 weeks posparutm; hence a diagnosis of exclusion and final diagnosis made only in postpartum period. May have other signs of pre-eclampsia e.g. epigastric discomfort or thrombocytopenia.
2. **Pre-eclampsia** is hypertension associated with proteinuria (of at least 300 mg/24 hours or at least 1+ dipstick) and pathological edema after the 20th gestational week. Edema is not essential for the diagnosis. This is further classified into:

   a) **Mild pre-eclampsia** defined as diastolic BP of 90-110 mm Hg and/or systolic BP of140-.160 mm Hg plus proteinuria of 300 mg – 2 grams/24 hours or 1+ to 2+ on the dipstick.

   b) **Severe pre-eclampsia,** defined as presence of diastolic BP of at least 110 mm Hg and/or systolic BP of at least 160 mm Hg, plus proteinuria of at least 2 grams/24 hours or 2+ and above on dipstick testing, plus any of the following characteristics:

   i) increased serum creatinine: > 110μmol/L unless known to be elevated previously
   ii) cerebral or visual disturbance
   iii) oliguria, ≤500 mls/24 hours
   iv) epigastric pain
   v) elevated liver enzymes
   vi) thrombocytopenia (platelet count less than 100 x 10^9/L)
   vii) retinal haemorrhages, exudates or papilloedema

viii) pulmonary oedema

3. **Eclampsia,** defined as seizure(s) that cannot be attributed to other causes in a pregnant woman with pre-eclampsia.
4. **Superimposed pre-eclampsia (on chronic hypertension),** defined as new onset proteinuria of at least 300mg/24 hours in hypertensive women but before 20 weeks' gestation and/or a sudden increase in proteinuria or BP and/or platelet count ≤100 x 109/L in women with hypertension and proteinuria before 20 weeks' gestation.
5. **Chronic hypertension,** defined as BP ≥140/90 before pregnancy or diagnosed before 20 weeks' gestation, or hypertension first diagnosed after 20 weeks' gestation and persistent after 6 (other authorities: 12) weeks' postpartum.

From this classification, our patient had severe pre-eclampsia due to the BP of more than 160/110 on more than 2 occasions, deranged renal function tests and features of end-organ damage.

Hypertensive disorders in pregnancy occurs in 5-7% of all pregnancies worldwide.[1,2] In Kenya the prevalence of hypertensive disorders in pregnancy has been reported to range from 1.5-9%.[3,4,5] Mati[3] in 1968 reported a prevalence range of 1.5-9% while Bashal[4] (1985) and Kibaru[5] (1992) reported prevalence of 5.7% and 5.4% respectively. It is the 3rd leading cause of maternal mortality in Kenya after haemorrhage and sepsis.[6] The perinatal mortality in eclamptic mothers was reported as 82.5/1000 by Mati[3] in 1968 and 225/1000 by Machoki[7] in 1989 and 162.8/1000 in pre-eclamtic mothers.[7] The maternal mortality rate reported by Mati was 325/100,000 while that reported by Machoki was 4650/100,000! Our patient was lucky not to be in this category. The predisposing factors are nulliparity, black race, maternal age below 20 and above 35 years, low socio-economic status, multiple gestation, polyhydramnios, twins, nonoimmune fetal hydrops, diabetes mellitus, chronic hypertension and underlying renal disease. Our patient's risk factors were the black race, relatively low socio-economic status and nulliparity.

The actiology of pre-eclampsia remains unknown despite decades of intense research.[1,2,8] It, therefore, remains a disease of theories and an enigma.[1,2,8] It is virtually agreed, however, that the placenta is the principle site from where the pathology begins with resultant release of unknown substance(s) into the maternal circulation where it/they cause endothelial dysfunction and subsequent multisystemic vascular end-organ damage. This is substantiated by the fact that placental removal is curative within 48 hours in most women, that pre-eclampsia is more common in women with increased placental tissue - e.g. hydatidiform molar pregnancy and multifetal gestation - and persistence of the disease in women with retained placental tissue such as in women with abdominal pregnancy 3 months postpartum.[1,2,8]

Currently the placental pathology postulated to lead to preeclampsia is impaired placental implantation and/or vascularization attributable to impaired maternal immunological response to the hemialogeneic conceptus. This impaired immunological response lead to inadequate endovascular trophoblast invasion, replacement of the endothelium and destruction of the medial musculo-elastic tissues of the spiral and basal uterine arteries. This leads to inadequate fibrinoid change in the vessel wall and conversion of thin walled muscular spiral arteries into saclike, flaccid and low resistance utero-placental vessels.[1,2,8] The impaired maternal immune response is also thought to cause acute atherosis in the myometrial segments of spiral arteries characterized by fibrinoid necrosis of the arterial wall and resultant various degrees of vascular obliteration and consequent placental infarction.[1,2,8,9]

A relatively recent findings by Sukhatme and Levine et al[9] (2003) is the concomitant impaired implantation and vasculirization of the placenta due to inadequate production of placenta growth factor (PlGF) and/or excessive production of serum fms-like tyrosine kinase-1 (SFLT-1) that inhibit the function of PlGF and vacular endothelial growth factor (VEGF). These findings were also noted by Ecker and Karumanchi[10] (ACOG Feb. 2004) who postulated that low levels of PlGF and high levels of SFLT-1 could be used to positively predict pre-eclampsia in the first trimester. The immunologic response and or inflammatory reaction therein, in the presence of predisposing factors such as maternal vascular disease, genotype etcetera, lead to reduced uteroplacental perfusion and subsequent release of unspecified substance(s) into the maternal circulation that lead to endothelial dysfunction, vasospasm, activation of coagulation system, and impaired secretion of endothelial cell products (endothelins, thromboxane-A2, prostacyclin, prostaglandins, nitric oxide, etcetera).[1,2,8,9] Thus in pre-eclamptic women there is increased levels of vasoconstrictors (endothelin-1, thromboxane-A2, some cytokines) and reduced levels of vasodilators (nitric oxide, kallikrein, prostacyclin). The immunization concept is supported by various studies including the observation by Trupin and colleagues (1996)[11] that multi-parous women impregnated by new consorts are more likely to develop preeclampsia than those who conceive with the same male partner throughout.

Prediction of pre-eclampsia has been attempted with various methods but none has proved effective enough to be widely used. The latest is the use of serum SFLT-1 and PlGF alluded to above.[9,10] Others that have used are angiotensin-II infusion, roll over test, serum levels of calcium, uric acid, fibronectin and markers of oxidative stress. Still others have tried urinary levels of kallikrein and serum coagulation and immunological factors and Doppler velocimetry of the uterine arteries but without reproducible results.[1,2]

Prevention of preeclampsia remains elusive. Earlier findings by Wallenberg et al (1986) that low dose acetyl salicylic acid (Aspirin 60-80 mg daily) prevented development of preeclampsia have been disproved by many multi-centric studies including that by Sibai (1993), the pre-eclampsia guru. Similarly, calcium has not been shown to be useful in preventing the disease despite earlier studies to the contrary. Use of antioxidants such as vitamins A, C and E has been shown to reduce preeclampsia (Chappell et al, 1999). However, this has yet to be demonstrated in double blind, randomized, controlled trials. Our patient had no familial or prior history of pre-eclampsia so she did not benefit from the prophylaxis.

The management of hypertensive diseases in pregnancy is aimed at termination of pregnancy with least possible trauma to the mother and fetus, birth of a fetus who thrives and complete restoration of the health of the mother.[1] The definitive treatment of pre-eclampsia-eclampsia is delivery of the fetus and specifically the placenta.[1,2,8] The timing of delivery is dependent on the fetal maturity, fetal and maternal status and the severity of pre-eclampsia. Upon diagnosis all patients with pre-eclampsia are usually admitted for physical and laboratory evaluation of disease, continuous and close maternal and fetal monitoring, and subsequent delivery. The laboratory tests performed include liver and renal function tests (RFTs), a full haemogram and daily urinalysis and, when needed coagulation profile. Obstetric ultrasonography for estimating gestation and weight, umbilical artery Doppler velocimetry, placental status assessment and biophysical profile score are done. The patient is then classified as having mild or severe disease depending on these findings and the patient managed accordingly. All patients with hypertensive disease who have attained gestation of 37-38 completed weeks are delivered either by induction of labour or by cesarean section where indicated. The management of the other patients will depend on the fetal maturity, severity of pre-eclampsia, maternal and fetal status and the condition of the cervix.[1,2]

Patients with mild pre-eclampsia are managed expectantly either in hospital or by home based care. Hospitalized patients are allowed to be up and about, BP measured 4 hourly, urinalysis and weighing done daily, liver function tests (LFTs) and RFTs and creatinine clearance are done weekly and the patient monitors the baby by a fetal movement chart. Ideal home-based care involves bed rest, daily BP measurement and urinalysis, twice weekly antenatal clinic visits and advice on the danger signals e.g. severe headache, epigastric pain or visual disturbances. Controversy exists on the use of antihypertensive therapy in early mild pre-eclampsia remote from term. In fact a meta-analysis on various antihypertensives (labetalol, nifedipine and isradipine) concluded that antihypertensive-induced decreases of maternal BP affects fetal growth causing growth restricted infants.[14] In mild pre-eclampsia, therefore, antihypertensive therapy is usually withheld unless the BP exceeds 160/100 mm Hg.[1,2] Because pre-eclampsia can rapidly

progress to severe in an unpredictable manner, steroids are administered weekly to accelerate fetal maturity.

Severe pre-eclampsia is managed by either immediate delivery (preferred- for maternal and, to a certain extend, fetal benefit) or by prolongation of pregnancy in a tertiary hospital. Certainly women with severe pre-eclampsia at 34 weeks or more must be delivered immediately. For patients who are under 34 weeks gestation pregnancy prolongation can either be expectant or 48 hours delay to allow action of corticosteroid therapy, the main aim being to improve neonatal outcome without compromising maternal outcome adversely.[2] However, pregnancy prolongation in patients with severe pre-eclampsia at gestation below 34 weeks is controversial with some advocating immediate delivery irrespective of any other factors while others prolong pregnancy in consideration of reducing perinatal morbidity and mortality, particularly in patients with bad obstetric history and prior infertility. Thus patients who are 24-34 weeks who must be delivered but can wait for 48 hours are given corticosteroids then delivered. Expectant management is offered to patients at 24-34 weeks who do not have HELLP syndrome, no deteriorating renal function, no persistent severe headache, no visual disturbances or epigastric pain. The occurrence of these features during expectant management demands immediate delivery.[1,2] Despite having severe pre-eclampsia, which requires immediate delivery, our patient had expectant management for 20 days but immediate delivery by cesarean section was carried out when the features of impending eclampsia occurred. This is because she had declined earlier delivery.

Women with severe pre-eclampsia at 24 weeks or less should be offered induction of labour to terminate the pregnancy.[1,2]

During expectant management daily weighing, urinalysis, fetal kick chart and BP assessment and physical examination are done. Where resources allow and depending on prior findings, RFTs and LFTs are done twice weekly, biophysical profile score and Doppler velocimetry once weekly. Antihypertensives are given to control the BP to the range of 140/90 – 160/100 mm Hg.[1,2,8] This is because reduction of diastolic BP below 90 mm Hg leads to reduced placental perfusion with resultant fetal compromise. The use of sedatives is controversial. Phenobarbitone particularly is disfavoured in certain western centers because of its interferes with fetal heart rate testing and impairs vitamin K-dependent clotting factors.[1,2] In our set up it is still widely used as was the case with our patient.

Eclampsia prophylaxis is indicated in patients with severe pre-eclampsia, particularly those with signs and symptoms of impending eclampsia such as severe headache, visual disturbances, epigastric pain and fulminant hypertension. All women with severe pre-eclampsia in labour should be on anticonvulsant

prophylaxis while awaiting delivery and 24 hours after delivery.[1,2] There is controversy as to whether patients with mild pre-eclampsia should benefit from eclampsia prophylaxis with some authorities advocating for while others disapprove of it.[1,2]

The drug of choice for eclampsia prophylaxis is magnesium sulphate. The alternatives are intravenous diazepam and phenytoin infusion. Both Lucas and colleagues[14] (1995) and the Eclampsia Trial Collaborative Group (1995)[15] found magnesium sulphate more superior to either diazepam or phenytoin in preventing and controlling eclampsia. Though our patient did not develop eclampsia, she should have gotten eclampsia prophylaxis with magnesium sulfate, which is now gaining popularity in our set up. If eclampsia occurs magnesium sulphate (or diazepam or phenytoin) should be given to control the seizures. Further, the complications of severe pre-eclampsia and eclampsia should be looked for and managed accordingly. These include cerebrovascular accidents, blindness from retinal detachment and/or cortical injury, pulmonary edema, abpruptio placenta, rupture of the liver, acute renal failure, tongue injuries and muskuloskeletal fractures and injuries. Delivery must be achieved within 6-12 hours post eclampsia.[1,2]

Postpartum care of pre-eclamptic and eclamptic patients is important because, although 75% of eclampsia occurs before delivery, 50% of the remaining 25% occur in 48 hours postpartum. Besides patients need to be counseled that generally the recurrence risk of pre-eclampsia in nulliparous women who develop pre-eclampsia before 30 weeks is estimated to be 40%, that early onset and/or recurrent pre-eclampsia is associated with development of hypertension in the future and that women who subsequently develop normotensive births have reduced risk of hypertension in future. Pre-eclampsia does not cause hypertension and any hypertension persisting beyond 12 weeks postpartum should be regarded as chronic hypertension. Pre-eclamptic mothers should be warned that any future pregnancy will require attending antenatal clinic as soon as pregnancy is detected and preferably in a center where a comprehensive care can be provided.[1,2,15]

It is important to appreciate that most developing countries including Kenya are hampered by inadequate resources for comprehensive feto-maternal medical care that would be needed in comprehensive care of pre-eclamptic patients. In Kenya for instance many women only realize they had pe-eclampsia after they have developed and recovered from eclampsia!

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OBSTETRIC CASE No.2

SUCCESSFUL VAGINAL BIRTH AFTER PRIMARY CAESAREAN SECTION

Name :	R.W.W.	LMP :	18/11/2003
File No. :	0904557	EDD :	25/08/2004
Age :	27 years	GBD :	38+ weeks
Parity :	1+0 gravida 2	Admitted:	12/8/2004
Delivered:	12/08/04	Discharged:	13/08/2004

Presenting Complaint

R.W. was admitted to the Kenyatta National Hospital (KNH) labour ward at 1 a.m with labour pains for one hour without drainage of liquor.

History of Presenting Complaint

She was well until 1 hour before admission when she developed lower abdominal pains that radiated to the lower back, were intermittent and progressive in intensity and frequency. She reported an associated bloody and mucoid vaginal discharge. There was no history of symptoms of urinary tract infection.

Past Obstetric and Gynaecologic History

RW was para 1+0 gravida 2 with no living child. Her first delivery was in 2002 via an emergency caesarian section (C/S) at Pumwani Maternity Hospital (PMH) at 8 months gestation. The indication was fetal distress in labour that occurred 4 days after pre-term premature rupture of membranes. The duration of labour was 4 hours. Earlier, she had been admitted to PMH and started on antibiotics. No explanation was given for the preterm drainage of liquor. The outcome of the c/s was a live male infant who who eight 2950 grams and was admitted to nursery because of pneumonia. He died 4 days postpartum. Following this she used the combined oral contraceptive pills until August 2003. Her menarche was at 14 years while her menstrual flow occurred for 3-5 days every 28-30 days.

History of Present Pregnancy

Her last normal menstrual period was on 18/11/2003 and her expected date of delivery was 25/08/2004. The gestation by dates was, therefore, 38+ weeks. Her first booking date was at 16+ weeks of gestation at Kenyatta National Hospital (KNH). She was in good general condition. Her weight was 54 kg and her height was155 cm. She had normal vital signs and the uterine fundal height then corresponded with the dates. The antenatal profile was: Hb; 13.2 g/dl, blood group A +ve and negative serology for VDRL and HIV. Her antenatal care visits were 4 weekly until 28 weeks, 2 weekly till 36 weeks and weekly thereafter

The antenatal period was uneventful. At 36 weeks of an erect lateral pelvimetry indicated an inlet of 11.4 cm, a midcavity of 12.8 cm and an outlet of 14 cm with normal sacral curvature. Clinical pelvimetry at 36 weeks' gestation indicated an adequatepelvis. At 37+ weeks an obstetric ultrasound, done to aid in the decision on trial of scar, showed a fetus in cephalic presentation that was estimated to weigh 3000 grams. The placenta was fundo-anterior and had no abnormality. These findings were explained to her and she was advised to undergo "trial of scar" (attempt of vagial birth after c/s). She was to report to labour ward as soon as she thought she was in labour.

Past Medical History

This was not significant

Family and Social History

She was a married housewife who had graduated from a technical college. She neither drank alcohol nor smoked tobacco. She lived in Kayole with her husband. Her partner was a tutor at the Railway Technical Institute, Nairobi. There were no familial illnesses.

PHYSICAL EXAMINATION

She was in good general condition. There was no pallor, edema, jaundice or lymphadenopathy. Her BP was 125/75 mmHg. The pulse rate was 78 beats per minute with good volume. The RR was 81 per minute and the temperature was of 36.7°C.

Abdominal Examination

This was uniformly distended. A sub-umbilical mid-saggital scar was noted. The uterine fundal height was term. A medium sized fetus was in longitudinal lie, cephalic presentation with a descent of 4/5 and regular fetal heart tones of 144/minute. Its estimated weight was 3 kg. Three uterine contractions occurred every 10 minutes each lasting 30-40 seconds. She had no tenderness or any other organomegally.

Pelvic Examination

The external genitalia and the vagina were normal. The cervix was 3 cm dilated, central and 70% effaced. The sacral promontory was not reached, the ischial spines were not prominent and the intertuberous diameter easily accommodated four knuckles.

Diagnosis

A diagnosis of a 27-year-old Para 1+0 gravida 2 at 38+ weeks with an adequate pelvis in labor was made.

Management

Blood was taken for grouping and cross match of 2 pints of blood. A cannula was fixed and 5% dextrose administered. She was shaved; informed consent obtained, and advised to remain nil per os, to lie on her left lateral side and to report any persistent lower abdominal pain to the midwife. A partogram was started for half hourly monitoring of fetal and maternal well-being. At 4 a.m., 3 hours after admission, she complained of some bleeding. She was reviewed. Her uterine contractions remained the same but descent was 3/5. Vaginal examination revealed heavy show from a cervix that was fully effaced and 7 cm dilated. There was no caput succedaneum or moulding. Amniotomy drained scanty clear liquor and no cord was palpated. She was reassured and labor allowed to progress. At 7.00 a.m. she was noted to be in second stage.

Twenty minutes later she had spontaneous vertex delivery of a life male infant who weight 2750 grams an Apgar score of 7, 8 and 10 at 1, 5 and 10 minutes respectively. The placenta was delivered by controlled cord traction 10 minutes later. It was complete and weight 450 grams. Inspection of the birth canal showed no active bleeding and, therefore no exploration of the previous scar was done. Intramuscular ergometrine 0.5 mg was administered and the bleeding from the placental bed reduced to minimal ooze. Her immediate post-partum BP was 130/80 mmHg, PR 80/minute, temperature 36.9 and a RR of 20/minute. She was transferred to the post-natal ward where she was observed closely for 24 hours. Being stable and happy with her newborn she was discharged home on the second post-natal day with advice to visit the post-natal clinic in 6 weeks' time.

Follow up

In her 6th postnatal week she was in good general condition with normal vital signs. Breast, abdominal, pelvic and general examination showed no anomaly. After contraceptive advice, she opted for the combined oral contraceptive pills since she had used them before without any major adverse effects. She was advised to use a progestin only pill (Microlut®)) up to 6 months then she could start the combined pills.

DISCUSSION

The patient presented was a 27-year-old para 1+0 with 1 previous lower uterine transverse c/s scar that was planned for and had a successful vaginal birth after caesarian section (VBAC) to a 2750 gram, life male infant with a good Apgar score.

"Once a caesarian always a caesarian" was a dictum that was coined by Cragin in 1916 presumably in reference to the "classical" vertical c/s that was widely practiced then until Kerr's low transverse c/s was accepted in 1921.[1] This "dictum" seems to have lasted over ½ a century; for by the time Merill and Gibbs reported that they had achieved 83% VBAC in1978, only 2% of American women with previous c/s scars underwent trial of scar.[1] Since then several studies have been carried out to delineate the success (and its determinants) and the safety of VBAC. This is largely because a big proportion of the increase in the c/s rate in both the developing and developed countries has been attributed to repeat cesarean sections.[1,2] For instance, at the Kenyatta National Hospital, Karanja, in 1980, reported a c/s rate of 17.8% of which 51.2% were repeat sections.[2] Twenty-three years later in the same hospital Akula found a c/s rate of up to 92% in patients with a primary c/s scar and an overall c/s rate of about 30%.[3] This was in complete contrast to Walton's study in 1978 which found out that 73.9% of patients on trial of scar had successful vaginal delivery at KNH.[4] Prior c/s was the commonest (53.3%) indication for c/s then. In the developed countries the lowest overall c/s rates (6-7%) have been recorded in Japan and the Netherlands while in the USA and Canada the rate was 25% in 1988.[5] Generally about 60-80% of trial of labour after prior c/s result in vaginal delivery in the USA.[1] This is similar to the success rate of 64% to 84% in sub-Saharan Africa as found by Boulvain's meta-analysis on studies on VBAC.[6] Indeed of all women delivered by c/s in the USA in the year 2000, 37% of them were repeat sections.[1]

The variation in the success rates is due to various factors including the criteria for patient selection. At KNH, 2 or more previous c/s scars contraindicates VBAC. It (VBAC) is allowed only when the following conditions are met:

- Absence of recurrent indications of c/s e.g. a contracted pelvis,
- One previous lower uterine section incision,
- No other obstetric complication,
- Estimated fetal weight of 2500-3500 grams,
- A true conjugate of 10.5 cm or more by x-ray pelvimetry (ELP) and
- Absence of history of wound and/or endometrial (puerperal) sepsis after the first c/s

Our patient did not have any of the aforementioned contraindications.

Although 2 or more deliveries by c/s contraindicates trial of scars in our set up, the American College of Obstetricians and Gynaecologists (ACOG) in 1999 took the position that women with 2 prior low transverse c/s deliveries may be considered for VBAC.[1] It is crucial to find out the indication of the previous c/s, the infant weight and the outcome of the c/s. This patient had preterm premature rupture of membranes with subsequent chorioamnionitis and fetal distress and recovered uneventfully. These factors are non-recurrent. Besides, she met all the criteria mentioned above. The indication of the previous c/s has been used to prognosticate the outcome of trial of scar. Wing and Paul [1] found VBAC success rates of 91, 84 and 77% when the original indication for c/s was breech presentation, fetal distress and dystocia respectively. Further, the VBAC success rate fell to 13% when dystocia had been diagnosed in the second stage of labor. On the other hand prior vaginal delivery either before or after c/s birth, greatly improves the prognosis for a successful VBAC.

The criteria for patient selection and the management of patients on trial of scar are not without controversies. Whereas Walton[4] cited x-ray pelvimetry as the single most investigation in the selection of patients for VBAC, a randomized controlled trial in South Africa[7] found radiological pelvimetry to be of little value while Ogutu (1985) [8] suggested that x-ray pelvimetry should be limited to those with borderline pelvis and should not necessarily be done routinely in all patients. The cut off fetal size is also controversial. There are those who consider estimated fetal weight of more than 3500 grams as a contraindication for VBAC while others take 4000 grams as the cut off weight. Still others base their cut off weight on the previous largest baby delivered vaginally.[1,9]

The worst complication of trial of scar is uterine rupture with the resultant increased maternal and fetal morbidity and mortality. Factors associated with increased risks of uterine rupture include poor patient selection, inadequate facilities and qualified staff for monitoring patients on trial of scar and the use of oxytocis to induce or augment labor among others. Hence the exclusion from VBAC of upper segment uterine scars, which have ten-fold increased risk of uterine rupture than lower segment scars. In the USA the risk of uterine rupture as reported by Miller et al [9] in 1994 was 0.6% and 1.8% for those with 1 and 2 prior c/s deliveries, respectively. The reported rates in KNH for 1 prior c/s delivery are 6.5% and 4.5% by Walton [4] and Akula [3] respectively. Unlike in the USA where cautious use of oxytocin or prostaglandin gel has been recommended by the American College of Obstetricians and Gynaecology[10] oxytocics are contraindicated in our set up largely because of inadequate mandatory monitoring. However, sweeping of membranes and amniotomy are allowed. Once labour is established, a midwife or a doctor by the bedside

does strict partogramming. Maternal and fetal vital signs are observed and signs of impending or actual rupture looked for. These include:

- Signs of fetal distress e.g. late decelerations, variable decelarations and fetal bradycardia,
- Uterine pain that persists between contractions and usually located in the area of prior incision,
- Increased maternal pulse rate (>100) and reduced BP,
- Intrapartum haemorrhage and haematuria and
- Loss of uterine contractions, recession of the presenting part and loss of fetal tones are signs o overt rupture which one should not wait until they occur.

Upon delivery exploration of the integrity of the scar is unnecessary unless there is significant uterine bleeding.[1]

The author's opinion is that in teaching hospitals such as KNH, attempts should be made to reduce the high cesarean rate in patients who undergo trial of vaginal birth after cesarean section. This would include the provision of a cardiotocograph machine which would aid in the monitoring of patients thereby enabling induction and augmentation of labour in patients with primary cesarean section scars. Further the management of these patients should be reviewed based on the current evidence provided by multi-centre studies.

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OBSTETRIC CASE No. 3

RUPTURED UTERUS AND URINARY BLADDER AVULSION - SUBTOTAL HYSTERECTOMY AND BLADDER REPAIR.

Name	: M.K.	LNMP	: 15/10/2003
Age	: 27 years	EDD	: 22/7/2004
File Nº	: 0975033	GBD	: 38+ weeks
Parity	: 3 + 0 gravida 4	DOA	: 13/7/2004
		DOD	: 3/8/2004

Presenting complaint.

M.K was referred from Pumwani Maternity Hospital (PMH) at 5 a.m. on 13/07/04 with a diagnosis o ruptured uterus in a mother with three previous caesarean section scars at 38+ weeks gestation by dates.

History of presenting complaint.

She had been well until 10 pm on 12/7/04 when she developed labour pains. She and her husband could n get means of transport immediately and it was not until 3 a.m. on 13/07/04 that she reported at PMH. Bein a mother with 3 previous caesarean deliveries in established labour by then, she was prepared f emergency caesarean section. Unfortunately this was not possible since there was an ongoing operation i the only operational theatre in PMH.While she was waiting for her turn to be operated, she develope progressive persistent lower abdominal pain associated with very bad feelings. At about 5 a.m., th persistent pain suddenly ceased but the bad feelings persisted and she developed heavy vaginal bleedin associated with dizziness, headache, blurred vision and fainting tendency. She was referred to Kenyat National Hospital (KNH) but again lack of immediate transport led to her being admitted to KNH labo ward at 7:45am; virtually three hours later.

Antenatal History.

She had her antenatal care in a health center in Kisii, her rural home. She had had menses only once a weeks after her last delivery in June 2003.She breastfed exclusively for only one month, did not use an contraceptive method despite being advised and first noted pregnancy symptoms in December. Quickenin was about late march or early April 2004,giving an estimated gestation of 36 to 40 weeks. She had visite the health center only three times from about 26 weeks by extrapolation of the quickening date. Blood an urine had been taken for antennal profile. The ANC card was unavailable but she said the results had bee

all right. Despite being unsure of her dates, an obstetric scan had not been done .The prenatal period had been uneventiful and she had planned to deliver at PMH.

Past Obstetric and Gynaecologic History.

She was a para 3 + 0 gravida 4 at 38 weeks gestation by dates. All her three deliveries had been b emergency cesarean section and all her children were alive and well. The first baby, a life female infant, 3. kg, was delivered in 1996 by emergency cesarean section due to malposition in labour at term. The secon baby delivered in 2000, by emergency cesarean section due to foetal distress with one cesarean section sca was a life female infant with a birth weight of 3 kg. The third baby, a life male infant with a birth weight o 3.4 kg was delivered June 2003 also by emergency cesarean section due to 2 previous cesarean section sca in labour at term. All the caesarean sections were uneventiful. She had not used any contraceptive metho despite being advised to use an intrauterine contraceptive device (IUCD) since she had goitre.

Past Medical History.

M.K.had developed an asymptomatic goitre since 2000 and had no known allergies.

Family and Social History.

She was a married woman who had schooled up to Form 2. She did not smoke tobacco or drink alcohol. He husband and she were small scale grocers in Nairobi and Kisii respectively. The husband also worked wi Keroka bus services limited and lived in Limuru 40, kilometres from PMH in Nairobi.

PHYSICAL EXAMINATION.

General examination

She was sick looking, moderately pale, anxious and moderately dehydrated. She had no jaundic lymphadenopathy or obvious cyanosis but had mild pedal oedema. Her BP was 142/94mmHg, pulse wa satisfactory volume and regular at 92/min. Tempareture was 36.5°C. Two pints of blood ran through cannulae on her forearms.

Thyroid examination

There was diffuse enlargement of the whole gland. It had a smooth surface and moved up with deglutitio Either of the lobes measured about 3X4X5 cm.

Abdominal Examination.

This was distended with an accentuated bulge in the lower abdomen. A subumbilical midline scar wa noted. Palpation revealed tender abdomen with easily palpable foetal parts without contractions Auscultation revealed no foetal heart tones. Deep palpation was not possible because of the generalize tenderness.

Pelvic Examination.

Fresh and altered blood was on the normal external genitalia. The vagina was filled with blood clots and Foleys catheter drained bloody urine.

Cardiovascular Systems.

Though the pulse rate was rising and the volume becoming weaker, the BP and the rest of the system wer not yet decompensated.

Respiratory and Central Nervous Systems.

Besides anxiety and psychological and physical distress due to pain and the life threatening nature of h ailment these were essentially normal. No stigmata of hyperthyroidism were noted in the eyes.

Impression.

An impression of ruptured uterus with foetal demise in a 27 year-old para 3+0 with 3 previous scars wa made.

Management.

Immediate preparations for emergency laparotomy were instituted. Blood transfusion was maintained, bloo taken for urgent grouping and cross-match, atropine given and informed consent obtained. The surgic team including consultant gynaecologist was assembled. Laparotomy revealed haemoperitoneum of abo 1500mls,and an extruded male fresh stillbirth with a birth weight of 3100 grams. An extensive T-shape uterine rupture was found. The transverse tear was annular along the previous scar while the vertical on extended up to the cervix and led to avulsion and vertical tear of the urinary bladder from the fundus to th level of the apex of the trigone. The tear was about 4cm long. The ureteric orifices were patent. Th necessitated subtotal hysterectomy. The bladder tear was repaired in 2 layers with absorbable (Vicryl) si 2/0 and haemostasis achieved. However, on doing vulvovaginal toilet, bloody urine was noted draining p the urethral catheter. This was expected and the catheter was left in situ for 14 days. The reversal of gener anesthesia was fairly smooth.

Post Operative Care.

Half hourly measurements of BP, PR, RR and temperature, chartting of input, output and BP charts an transfusion of 3 pints of blood were instituted. Intravenous fluid infusion, ceftriaxone 1gram 12 hourly metronidazole 500mg eight hourly and lasix 40mg 12 hourly were administered. On the first post- operativ day the patient had normal vital signs, was moderately pale, had stabilized and had normal bowel sounds Systemic examination was satisfactory except that she still had frank haematuria despite flushing th bladder with normal saline overnight. A consultant gynaecologist with expertise in urogenital reconstructiv surgery was consulted. He recommended intravenous and oral fluids to achieve a total of 6-8 litres per day A three-way catheter for bladder irrigation was also ordered for but was unavailable. A total of 6 pints o blood had been transfused. Double dosage of oral haematinics, bromocriptine, minimal ambulation an continuation of intravenous antibiotics were prescribed. Her blood group was A+ve and the haematocrit wa 23%. Appraisal on the outcome of surgery and counseling was provided.

The patient did very well. The breasts remained unengorged, the chest was clear and exposure of the wound on the 3rd postoperative day revealed a well-healed dry wound. Blood was taken for haemogram and plenty of oral fluids advised. However, on the 5th postoperative day she developed fever associated with chills, headache and general malaise. Her breasts were soft and non-tender, the chest was clear, the wound was clean and dry and the abdomen was soft and non-tender. Her scanty lochia was non-foul smelling, the catheter urine remained bloody and the calves were without signs of deep venous thrombosis. A repeat haemogram, urine for culture and sensitivity and blood smear for malaria parasites were ordered. The combined results were as follows:

13/7/04: a) **Haematocrit:** 23%, b) Renal function tests: Normal

16/7/04: **Haemogram:** a) Hb: 7.5 g/dl b) Wbc count : 8.2 x 10^9/L

18/7/04: 1) **Haemogram**: a) Hb: 8.0 g/dl b) Wbc count : 6.2 x 10^9/L c)

: 2) **B.S for malaria parasites :** Negative

: 3) **Urine:** a) Urinalysis: No abnormality,

b) Culture and sensitivity: No growth obtained.

The interrupted stitches were removed on the 8th postoperative day. Her urine cleared. No incontinence occurred. On the 19th postoperative day the vesico-vaginal team who removed the foleys

catheter reviewed her. She had normal voiding thereafter and was subsequently discharged home on the 20th postoperative day through the postnatal clinic.

Follow-up

M.K was reviewed two weeks following discharge in the postnatal clinic. She was in good general condition, not pale and systemic examination revealed no abnormality. She was advised that she still had a chance of developing cervical cancer since only subtotal hysterectomy was done. A request for papanicollaou smear was given to her and was to have the smear taken in six weeks post surgery. Because of massive transfusion, she was advised on testing for HIV, Hepatitis B and C viruses in three months time.

She was also advised to seek surgical evaluation of the goiter and that the IUCD is not contraindicated in patients with asymptomatic goiter or even those with hyperthyroidism unless they have dysfunctional uterine bleeding secondary to the disease.

DISCUSSION

The patient presented was para 3+0 gravida 4 with 3 previous cesarean section scars who went into labour at term and due to delay in performing emergency cesarean section developed uterine rupture and an associated bladder tear and fetal demise. Subtotal hysterectomy and bladder repair were undertaken and she did well postoperatively.

One of the most dreaded obstetric catastrophies is uterine rupture because it is associated with high incidence of maternal and foetal morbidity and mortality rates and reflects poor obstetric care. The incidence of uterine rapture varies from region to region and from institution to institution. The incidence in Pumwani Maternity hospital, Nairobi as found by Wanyonyi in the period 1996 to 2001 was 1:219 deliveries.[1] This compares to the incidence of 1:192 by Webala in his 1979 study at Kenyatta National Hospital.[2]. These local incidences are quite high compared to the incidence of 1 in 1280 deliveries in a Western institution in 1950 and 1 in 18500 in another one in 1994.[3,4]
Uterine rupture is classified into complete (rupture communicates directly with peritoneal cavity) or incomplete (rupture separated from it by the visceral peritoneum). It should be distinguished from dehiscence of uterine scar which is partial separation of the scar with intact overlying peritoneal, unextruded fetus and no or minimal bleeding.[4] Our Patient's uterine rupture was complete.

Causes of uterine rupture are broadly classified into injuries or anomalies sustained before index pregnancy and those occurring during current pregnancy. The former include surgery involving the myometrium (e.g. previous caesarian scar, hysterotomy, myomectomy, metroplasty) coincidental uterine trauma (e.g. abortion, curreting sounds, sharp/blunt trauma) and congenital anomaly such pregnancy in undeveloped uterine horn. The risk of rupture due to previous uterine scar is higher if uterine wound healing was inadequate suche as in metritis and inadequate time (at least 6 months) of healing prior to conception. Causes of rupture during current pregnancy are injudicious stimulation of labour with oxytocics, external and internal version, breech forceps delivery and acquired uterine diseases such as placenta percreta and gestational trophoblastic disease. The patient presented had 3 previous scars in labour when she developed uterine rupture. Besides she conceived 4 months after the 3rd cesarean section. This was because she had erroneously been told that she could not use the IUCD due to her asymptomatic goiter. Further if she could not use the IUCD because of any other reason she could have been offered an alternative such as barrier methods.

In developing countries such as Kenya, however, studies [2,5] have shown that socio-economic factors such as inadequate means of transport, facilities for operative delivery, qualified and motivated personnel and maternal ignorance play a significant role in putting the parturients at risk of uterine rupture. Our patient suffered the tragedy of having multiple risks. She went into labour at night with 3 previous c/s scars. 40

kilometers away from her preferred hospital for delivery to which she arrived 5 hours later only to find the only theatre occupied!

The most common aetiological factor of uterine rupture is separation of previous c/s scar as was the case with our patient.[1,2,4] Prolonged and obstructed labour are other causes attributable to poor obstetric care while (grand)multiparity is a predisposing factor. As mentioned earlier the maternal and prenatal morbidity and mortality are high. For instance, the reported still birth rate ranges from 46% to as high as 70% while maternal mortality ranges from 4.2% to 20%.[1,2,4,6] Our parturient had a stillbirth and her morbidity led to hospitalization for three weeks.

Though not fully reliable a number of symptoms and signs suggests impending or actual uterine rupture. These include persistent and rising lower abdominal pain, maternal tarchycardia, distress, reducing blood pressure and persisted lower abdominal pain or tenderness. Complete uterine rupture presents with sudden cessation of uterine contraction and foetal heart tones, vaginal bleeding and bloody urine when the bladder is involved. All these were noted in our patient. Signs of hypovolaemic shock supervene if urgent intervention is not instituted.

Treament depends on the degree of rupture. Non-bleeding small rents as occurs after some normal vaginal deliveries after successful trial of scar are best managed conservatively. However, complete ruptures require emergent laparotomy preceded by intensive resuscitative measures such as correction of hypovalaemia with colloids and blood transfusion. Operative management depends on the state, age and family size of the patient, the type, and site and extend of the rupture and the surgeon skills. At laparotomy three surgical options are available; repair of the rupture only, repair and bilateral tubal ligation and hysterectomy.

Either subtotal (mostly) or total (rarely) hysterectomy is preferred for complete extensive ruptures. Subtotal hysterectomy is preferred to total because it is faster and safer. Total hysterectomy is reserved for extensive tears involving the cervix and vagina and then a highly skilled surgeon should perform the surgery. Lateral rupture involving the uterine artery with attendant broad ligament haematoma renders the ipsilateral ureter at risk of ligation or other injury. Excessive haemorrhage will, therefore, require ligation of hypogastric artery to clear the operating field of blood.[6] Other pelvic organs must be inspected for possible injury, as was the case in our patient who underwent subtotal hysterectomy and bladder repair.

Possible complications of uterine rupture include haemorrhage with resultant hypovolaemic shock and possible prerenal failure, postoperative sepsis, ureteral injury, thrombophlebitis, amniotic fluid embolism, disseminated intravascular coagulopathy, pituitary failure (Sheehan syndrome) infertility/sterility and death.

Patients and/or relatives with mortalities need counseling. Those whose uteri are repaired and remain fertile should be counseled on contraceptive, and the need for essential selective caesarian section should they conceive. In Kenya most uterine rupture cases would be prevented by improving the socio-economic status of the citizens, education of both staff and expectant mothers, good obstetric care such as partogram usage and early referral of mothers in need of operative delivery.

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OBSTETRIC CASE No. 4

CEPHALO-PELVIC DISPROPROTION IN LABOUR – EMERGENCY CESAREAN SECTION: LIVE BABY.

Name	:	S.I.O.	DOA	:	22/09/2004
Age	:	28 years	DOD	:	25/09/2004
Parity	:	1 + 0 gravida	LMP	:	13/12/2003
File N°	:	0985002	EDD	:	20/09/2004
			GBD	:	40 + weeks

Presenting Complaint

S.I.O was admitted to the Kenyatta National Hospital (KNH) on 22/09/2004 at 8.40 p.m. as a referral from St. Luke's Surgery nursing home with complaints of labour pains for 25 hours.

History of presenting complaints

She had been well until she developed intermittent lower abdominal pains that increased in frequency and intensity on 21/09/2004 at 7 p.m. She had no urinary symptoms or change in bowel habits. She was admitted to St. Luke's Surgery nursing home at 11.20 p.m. and was found to be labor with cervical dilatation of 2 cm. About 9 a.m. on 22/09/2004, a drip (? Syntocinon) was started, but 4 p.m. the cervix remained 7cm dilated and the head was still high above the pelvic brim, hence the referral.

Obstetric and Gynaecologic History

She was para 1 +0 gravida 2. Her last menstrual period (LMP) was on 13/12/2003, the expected date of delivery (EDD) was on 20/09/2004 and therefore the gestation by dates (GBD) was 40 weeks plus 2 days. She had started antenatal care (ANC) at the St. Luke's Surgery nursing home from 24 weeks gestation. Then and throughout the follow-up the fundal height corresponded to the gestation by dates. The antenatal profile done at 28 weeks gestation was: Hb; 13.0 gm/dl, blood group B +ve and serological tests for VDRL and HIV were negative. She received 2 tetanus toxoid vaccines at monthly interval. Her blood pressure remained normal throughout the pregnancy. Urinalysis was done only once at 24 weeks and had no abnormality. The last 4 weeks she developed frequent urination without burning sensation associated with frequent thirst but no increase in appetite was noted. She reported this and was told that it was because of the baby's head pressing on her urinary bladder. The antenatal period was largely uneventful.

Her first delivery was in 2000 at hospital at term by spontaneous vertex delivery to live male infant with a birth weight of 3600 grams. He cried immediately after birth and was alive and well. Pre- intra- and postpartum periods were uneventful. She attained menarche at the age of 15 years and since then had menses for 3 days every 26 to 28 days without any menstrual anomaly. She used the combined oral contraceptive pill six months after the first delivery in 2000 to October 2003 when she stopped to conceive. She had had no Pap smear done.

Past medical history

This was insignificant.

Family and social history

She was a married lady who sold second hand clothes and smoked no tobacco and drank no alcohol. She had attained only secondary education. She lived with her husband in Dagoreti corner, Nairobi. The husband was a counselor and worked with Dagoreti community voluntary counseling and testing (VCT) for HIV. There was no history of chronic illness (such as diabetes mellitus) in the family.

PHYSICAL EXAMINATION

General examination

She was in fair general and nutritional condition, not of short stature, mildly anxious and dehydrated but without pallor, pedal edema, jaundice or lymphadenopathy. The BP was 130/80 mmHg, the pulse was of good volume at 84/minute, the temperature was 36.5 oC and the RR was 22/minute. Urinalysis showed moderate ketonuria.

Abdominal examination

The abdomen was uniformly distended except a small transverse subumbilical depression (?Bandl's ring). The fundal height was term and the fetus was in longitudinal lie, cephalic presentation with regular heart tones of good volume. Three uterine contractions occurred every 10 minutes each lasting 45 seconds. The descent was 4/5 (up). Neither hepatomegally nor splenomegally or any mass were noted. The fetus was estimated to be 3950 grams.

Pelvic examination

She had normal external genitalia and healthy vaginal mucosa. The cervix was 8 centimeters dilated, fully effaced and slightly edematous. There was mild caput succedaneum and no moulding. The sacral promontory could not be reached, the pelvic contours were not suggestive of a contracted pelvis and the ischial spines were not prominent. Meconium staining liquor grade I was present. The knuckles of a clenched fist could easily fit in the intertuberous diameter of the outlet.

Respiratory, cardiovascular, nervous and the musculoskeletal systems were essentially normal.

Impression

A diagnosis of cephalopelvic disproportion (CPD) was made.

Management

The clinical findings and the diagnosis were explained to the patient. She was to undergo an emergency cesarean section. An informed consent was obtained and blood taken for grouping and cross match. She was shaved and premedicated with 0.6 mg of intramuscular atropine.

In theatre vulvo-vaginal toilet and catheterization drained 100 mls of concentrated urine. Repeat vaginal examination found no change of the earlier pelvic examination findings. She was placed in supine position; abdomen cleaned and draped then, under general anesthesia, opened via a subumbilial incision. A lower uterine segment cesarean section was performed and by cephalic extraction delivered a live female infant with a birth weight of 4100 grams. The Apgar score was 8, 10 and 10 at 1, 5 and 10 minutes respectively. The baby was admitted to neonatal unit due to its weight. The placenta and the umbilical cord were delivered by controlled cord traction and were found to be grossly normal. The uterus and the abdomen were then closed as explained under introduction. Repeat vulvo-vaginal examination and toilet revealed clear urine and no active bleeding. Reversal of the general anesthesia was excellent.

Postoperative Care

The immediate postoperative care was as explained in the introduction. Intravenous fluids and antibiotics (crystalline penicillin, gentamicin and metronidazoe) and intramuscular pethidine were started. On the first postoperative day she was ambulant, had passed urine three times, had normal vital signs and was not pale. The breasts were soft and not yet active, the chest was clear, the abdomen was soft and moved with respiration and had normal bowel sounds. The uterine fundal height corresponded to 20 weeks gestation and was well contracted. The lochia loss was normal and the calves were soft and non-tender. In view of

the large for gestationbal age infant and the history suggestive of gestational diabetes, a random blood sugar test was done and was 6.7 mmol/L. A dipstick urinalysis showed glycosuria of 1+. Oral sips preceded gradual introduction of light diet by evening. On the 2nd day, oral medications were started and light diet encouraged. The baby had been found to be without abnormal hypoglycaemia or any other anomaly. Breastfeeding was encouraged. A fasting blood sugar done then was 5.8-mmol/L. She was advised to undergo oral glucose tolerance test (OGGT) in the 6th postpartum week. On the 3rd day, she was ready to go home and was discharged with advice to attend the antenatal clinic in the 6th postpartum week or any time she developed any complications. She was to do the OGTT a day or two prior to attending the clinic.

Follow up

By the 6th week postpartum, she was doing pretty well. Lochia loss had stopped in the 3rd week postpartum. She had not had menses since delivery. She had no pallor or pedal edema. Her Blood pressure, pulse, respiratory rate and temperature were all within normal limits. The OGGT was essentially normal. The breasts were active, soft and without any lumps. Abdominal examination revealed a well-healed incisional scar and no palpable uterine fundus or other mass. While taking the Pap smear the cervix was noted to be grossly normal and no abnormal vaginal or cervical discharge was noted. Contraceptive advice was provided and she opted for the combined oral contraceptive pills, which she got from the Family Welfare Clinic (FWC) adjoining the postnatal clinic. She was to be followed up in the FWC with the Pap smear results.

DISCUSSION

Presented is a 28-year-old para 1 +0 who was referred to KNH with CPD. She underwent an emergency cesarean section and delivered a live female infant with a birth weight of 4100 grams and a good Apgar score. Postoperative recovery was excellent.

Cephalopelvic disproportion (CPD) is defined as the disparity in relation between the fetal head (cephalo) and the pelvis (pelvic).[1,2,3] It may be due to either a big fetus with a normal pelvis or due to an average size fetus with a small pelvis or commonly due to a combination of both factors. This disproportion could be either at the pelvic inlet or in midpelvic plane or at level of the pelvic outlet. However, in order of frequency, CPD due to pelvic outlet disparity is the least while that at the pelvic inlet is the commonest.[1,2] The definition of CPD, however, is highly controversial since it is relates to the fetus and the various levels of the pelvis. In our set up, the diagnosis of CPD is made when the parturient is in active phase of labour with at least three uterine contractions occurring every 10 minutes and each lasting at least 40 seconds and there are signs of obstruction. These signs include unengaged head and associated early rupture of the membranes, absence of descent of the head despite cervical dilatation, a head not well applied to the cervix despite ruptured membranes and occurrence of moderate to severe caput succedaneum and moulding. The diagnosis of CPD in labour is based on the fact that a fetal head is the best pelvimeter so that an apparently contracted pelvis could be adequate for a small fetus while a normal pelvis may be inadequate for a macrosomic fetus.[1,2] It is one of the leading indications of cesarean sections.[1,2,3,4] for instance in his masters thesis, Karanja[4] (1991) found out that CPD was not only the leading indication for cesarean section, but accounted for virtually half (48.8%) of all indications for cesarean sections at Pumwani Maternity Hospital, Nairobi.

Cephalo-pelvic disproportion should be distinguished from contracted pelvis. Whereas CPD commonly occurs in contracted pelvises, it does also occur in normal pelvises when the fetal head is bigger than the pelvic passage. Besides, although clinical pelvimetry can be used to diagnose a contracted pelvis, it is usually confirmed by radiological pelvimetry and unlike CPD is not depended on the size of the fetus and is always recurrent. CPD can be recurrent if there is a contracted pelvis. Although our patient had no clinical signs of a contracted pelvis, the fetus was large for gestational age (LGA-4100 grams), the fetal head remained unengaged despite adequate contractions and vaginal examination revealed caput succedaneum and moulding, hence the diagnosis of CPD.

With regard to the pelvis the normal dimensions of a gynaecoid pelvis are as follows:[1,2,5]

1. Pelvic inlet: a) anterior-posterior (AP) diameter – 11 cm
   b) Transverse diameter - 13.5 cm
2. Pelvic mid-cavity: both transverse and antero-posterior diameters are 12 cm.
3. Pelvic outlet : a) anterior-posterior diameter – 13.5 cm
   b) Transverse diameter - 11.0 cm

Radiological pelvimetry can be done by X-ray, Computerized Tomography (CT pelvimetry) and magnetic resonance imaging (MRI) in that order of efficiency and accuracy.

The widest diameter of the fetal head, the biparietal diameter, in vertex presentation is on average is 9.5 cm.

The pelvis is contracted if on radiological pelvimetry one or all of pelvic dimensions (the inlet, the mid-cavity and the pelvic outlet) are diminished. A contracted pelvis by obstetric definition is the alteration of the size and/or the shape of the pelvis of sufficient degree so as to alter the normal mechanism of labour in an average size baby.[5]The pelvic inlet is contracted if the AP diameter is less than 10 cm and/or if the transverse diameter is less than 12 cm. Clinically, the inlet could be estimated to be contracted if the mother is of short stature (less than150 cm), puts on shoe size 4 and below, the sacral promontory could easily be tipped, the head floats easily above the pelvic brim despite uterine contractions or on performing the Muller-Munro-Kerr test.[5] Other factors associated with inlet CPD include malpresentation and early rupture of the membranes. In women with contracted pelves, face and shoulder presentation are encountered three times more frequently and cord prolapse occurs 4-6 times more frequently.[1,2]

According to Cheng and Huang[6] the pelvic mid cavity is contracted when the sum of the interspinous and posterior sagittal diameters (normal 10.5 and 5 cm respectively) is 13.5 and below. Clinical features of contracted mid pelvis include prominent ischial spines, converging pelvic sidewalls, flat and long sacrum and narrow sacrosciatic notch. The pelvic outlet is usually defined as diminution of the interischial tuberous diameter to 8 cm or less.[1,2,5] Clinically the sub-pubic angle is acute and the knuckles of a clenched fist cannot enter the intertuberous diameter.[5]

There is no definitive fetal size and/or fetal head biparietal diameter that is indicative of CPD.[1,2,3] Although the American college of Obstetricians and Gynaecologists has recommended an estimated fetal weight of 4250-4500 grams as the cutoff point for cesarean section, this is usually for the diabetic mothers where shoulder dystocia is to be pre-empted.[7]

The causes of CPD are either maternal or fetal. The maternal causes include:

1. Malnutrition: rickets in childhood and severe prolonged osteomalacia in adult multipara women, stunted growth due to severe under-nutrition,
2. Disease or injury of the bones: a) pelvic tumours, tubercular arthritis, traumatic and pathologic fractures, b) Spinal deformities: kyphosis, scoliosis, spolilolisthesis, coccygeal deformity, c) lower limbs: congenital dislocation of the hip, hip joint disease,
3. Developmental defects: a) Naegele's and Roberts pelves, sacralization or lumberization of the vertebrae.
4. Endocrine disorders: diabetes mellitus (causing fetal macrosomia), precocious puberty and early fusion of epiphysial plates.

The fetal causes include fetal macrosomia (fetal weight exceeding the 90th percentile for a given gestation but generally birth weight above 4000 grams and certainly above 4500 grams) [1] and cephalic congenital anomalies – hydrocephalus, brain tumour and others.

Our patient could have had undetected gestational diabetes mellitus with subsequent fetal 'macrosomia' with a birthweight of 4100 grams. There were no other maternal or fetal predisposing factors to CPD.

The diagnosis of CPD is made from the history, physical and radiological pelvimetry and more importantly by the "fetal pelvimetry" while in labour for borderline pelvises. Diagnosed contracted pelvis is classified into severe, moderate and mild. Severe contracted pelvis is defined as obstetric conjugate of 7.5 cm and below in the presence of other indicators of CPD. In moderate pelvic contraction the obstetric conjugate is 7.6-9.5 cm, while an obstetric conjugate of 9.6 to 10 cm is indicative of slight or mild disprortion.[5] The presence of unengaged fetal head by 37th completed week in primigravidae, early rupture of the membranes while in labour with a high head, dilated cervix that is not well applied to the head, occurrence of caput succedanium and moulding, poor descent of the head despite strong uterine contractions are all suggestive of CPD.[1,2,5] All these were present in our patient.

The management of CPD depends on the degree of the disproportion, availability of facilities and personnel and fetal and maternal status. The management should begin from the antenatal clinic where patients with CPD are referred to centers specialized with high-risk maternal care. Patient selection for the specified management is then done and delivery planned in advance. Moderate and severe degrees of CPD are managed by

1. Premature induction of labour,
2. Elective cesarean section at term or

3. Trial of labour.

Premature induction of labour is not favored nowadays, should not be done in primigravidae and is performed in only selected multigravidae after confirmed 37 completed weeks.[5] Elective cesarean section is performed for mothers with severely contracted pelvises at 38 completed weeks in our set up. Those with other obstetric complications such as pre-eclampsia, postmaturity, and post cesarean pregnancy also undergo elective cesarean section.

Trial of labour is the conduction of spontaneous labour in a moderate degree of CPD in an institution trained personnel and facilties for fetal and maternal wellbeing monitoring and for performing emergency cesarean section.[2-5] It aims at avoiding unnecessary cesarean section and achieving good maternal and fetal outcome. The patient should tentatively be prepared for, counseled on and consent obtained for emergency cesarean section. Ideally labour should be monitored with a cardiotocogram but otherwise the progress of labor is meticulously monitored using the partogram and if there is inadequate uterine contractions labour is augmented with oxytocin. This requires that cautious amniotomy be performed when the cervix is at least 3 cm dilated and cord prolapse be anticipated if the head is not yet fixed. The presence and the degree of meconium in the liquor should also be looked for once amniotomy has been done.

The length of trial of labor is individualized and depends on the progress of labor, the degree of CPD and the maternal and fetal well-being and. The second stage of labor should not last more than ½ an hour.[1,5] In India the outcome of trial of labour is spontaneous vaginal delivery in 30%, ventouse or forceps delivery in 30% and cesarean section in 40%.[5]

The complication of CPD varies from center to center and depends on the level of obstetric care the patient gets. They include a high cesarean section rate including repeats, obstructed labour and its attendant maternal and fetal morbidity and mortality. These are feta lintracranial haemorrhages, severe caput succedaneum, asphyxia or death, uterine rupture, genital tract injuries including fistulations and lacerations and fetal and/or maternal sepsis and death.[1,2,3,5] Fortunately our patient had none of these complications except the cesarean section which had a good out come.

Prevention of CPD and its complications includes provision of good childhood nutrition and education to the girl child, comprehensive and focused antenatal care that aims at identification of mothers at risk including those with CPD and early referral of them to appropriate centers supported by adequate and accessible social amenities and infrastructure. Our patient was fortunate to have been referred early hence the good outcome.

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OBSTETRIC CASE No. 5

ANTEPARTUM HAEMORRHAGE-PLACENTA PRAEVIA. EMERGENCY CAESAREAN SECTION- LIVE BABY.

Name:	J.W.	LNMP:	10/10/03
Age:	28 Years	EDD:	17/07/04
File No:	0959604	GBD:	29+ Weeks
Parity:	1+0 gravida 2	Admitted:	05/05/04
Deliverd:	26/06/04	Discharged:	03/07/04

Presenting Complaint:

J.W. presented with sudden onset, painless vaginal bleeding for 1 ½ hours.

History of Presenting Complaint:

Our client had been well until she developed the vaginal bleeding at about 5.30 am on 5/5/04 soon after coitus with her husband. The bleeding was without associated abdominal pain, drainage of liquor or external trauma. There was no prior per vaginal discharge or urinary symptoms. The initial blood was frank red, fresh, profuse with clots and tracked down her lower limbs despite use of several pads. She still perceived foetal movements and denied history of any bleeding disorder. She had visited Huruma Nursing Home in Nairobi, thrice from about 20 weeks gestation. Antenatal profile indicted Hb of 12.0g/dl, blood group O +ve and negative HIV and VDRL serology tests. Her pregnancy had been hitherto uneventful.

Past Obstetric and Gynaecologic History:

J.W was a Para1+O gravida 2 with no living child. In 1999 she had a premature spontaneous vertex delivery at about 6½ months gestation to a live female infant who weighed 1.6kg and survived respiratory distress syndrome but died of pneumonia at 1 year of age. The predisposing factor(s) of preterm labour wwere unknown. Her menarche was at 15 years, had regular menstrual periods each occurring every 28 days and with moderate flow in 3 to 4 days without any menstrual disorder. Her coitarche was at 20 years and had had no sexually transmitted infections. She had used depot medroxy-progesterone acetate injections for three years after her 1999 delivery during which her periods were scanty but remained regular.

Past Medical History

This was insignificant.

Family and Social History

She was a married housewife who never drank alcohol or smoked tobacco and had attained only primary education. She lived in Lower Kabete with her husband who worked as a driver with a petroleum transporting parastatal. Her mother had diabetes melitus and hypertension.

PHYSICAL EXAMINATION

General Examination

JW was in fair general condition. She had mild pallor but no jaundice, fever, oedema or lymphadenopathy. Her BP was 110/70 mmHg, PR 95/min; regular and of good volume. Her skirt and lower limbs were blood stained.

Abdominal Examination

The abdomen was uniformly distended and moved with respiration. On palpation, it was soft and non-tender but lower abdominal discomfort was noted. The uterine fundal height corresponded to 30 weeks gestation and the foetus was in longitudinal lie, cephalic presentation with regular foetal heart tones at 144 per minute.

Speculum Examination

She had normal female external genitalia. Blood clots found in the vagina were removed and minimal oozing of altered blood from the grossly normal and closed cervical os was noted. Vaginal mucosa was healthy and had no signs of cervicitis.

Respiratory, Cardiovascular and Central Nervous Systems.

These were essentially normal.

Diagnosis

A diagnosis of antepartum haemorrhage (APH) probably due to placenta praevia at 29+ weeks gestation was made.

Investigations

They included:

1. Urgent blood grouping and cross match of 2 units and a haemogram that showed Hb of 11.0g/dl. WBCC of 6.5x 10^9/L and adequate platelets.
2. An emergent obstetric ultrasound that showed a single intrauterine pregnancy in cephallic presentation at an average computed gestational age of 29 weeks + 5 days. The placenta was on

the left lower uterine segment and covered the entire internal cervical os. This was a major type of placenta praevia.

Management

After securing a wide bore cannula, she was promptly admitted and put on strict bed rest, phenobarbitone 30mg every 8 hours for one week and intra-muscular dexamethasone 12mg 12 hours apart every week until she was 33 weeks (This was an old protocal now considered inappropriate - see Discussionbelow). She received haematinics twice a day. She was counseled on her condition and the need to remain in hospital until she attained a gestation of 38 weeks when scheduled caesarian section would be done. She was to keep all her pads for observation before discarding and to report any bleeding immediately to the nursing staff. Per vaginal spotting of altered blood continued for another 2 days then stopped altogether.

Hospital stay was uneventful for 8 weeks. She never bled again after the first episode. A repeat ultrasound had been requested at 36 weeks but was booked for a week later. At 37 completed weeks, however, she developed spontaneous per vaginal bleeding and was rushed to theatre via labour ward for emergency caesarian section. Informed consent was obtained and premedication with intra-muscular 0.6mg of atropine administered.

In theatre quick aseptic catheterization drained 150mls of clear urine and per vaginal bleeding was noted to be mild. The abdomen was cleaned draped and opened unter general anesthesia through a pfannenstiel incision. Lower uterine segment caesarean section was performed via an elliptical incision on the upper margin of the LUS. A fast but cautious incision was made through the placenta and the amniotic membranes. By cephallic extraction, a life male infant was delivered and the cord quickly clamped to prevent fetal blood loss through the incised and partially separated placenta. The birth weight was 3000 g and the Apgar Score was 8 at 1 minute and 10 at 5 minutes. The placenta (which was located on the left anterolateral site of the lower segment of the uterus, covering the entire internal cervical os) and the umbilical cord were delivered manually. The heavier than usual uterine bleeding was quickly arrested with double layer uterine repair, uterine massage and 40 units of syntocinon infusion. The estimated blood loss was 750mls. She had smooth reversal of general anaesthesia after routine abdominal closure and vulvo-vaginal toilet as described for caesarean section under 'Introduction'.

Post-operative Care

Recovery from surgery was good. She was put on nil per os, intravenous fluids and antibiotics and intra-muscular pethidine. On her first post-operative day oral amoxycillin, mefenamic acid, haematinics and sips

to graduate to light died were commenced. She had mild pallor and had passed urine three times. She had normal vital signs and was, therefore, mobilized and breast-feeding initiated. Wound dressing was removed on her 3rd post-operative day and the wound was clean and dry. Her check Hb was 9.2g/dl. She was discharged home on the 4th post-operative day.

Follow-up

She was reviewed in the postnatal clinic on the 2nd post-operative week. The breasts were active without abnormality. The wound had healed well and the uterus was involuting well and corresponded to 14 weeks. Lochia was serosal and non-foul smelling. Advice on proper breastfeeding and family planning options was given and the patient asked to revisit the clinic on her 6th postnatal week. On the 6th postnatal week the patient was no longer pale and had normal vital signs. The uterus was no longer palpable and lochia loss had stopped. She opted for oral contraceptives and progestin only pill (Microlut®) was prescribed for her. Advice was given to her to report to the Family Welfare Clinic for the combined oral contraceptive pill and pap smear after 6 months.

DISCUSSION

JW was a 28-year-old para 1+O who was admitted with antepartum haemorrhage due to major placenta praevia (type III) at 29+ weeks gestation. By emergency caesarean section at 37 weeks she delivered a live male infant with birth weight of 3000g and a good Apgar score. The mother and her infant did well postoperatively.

Antepartum haemorrhage (APH) is defined, as vaginal bleeding before delivery but after fetal viability has been attained.[1,2] The World Health Organization (WHO) describes fetal viability as known gestation of at least 24 weeks and or a fetus with at least 500 grams of body weight.[3] Fetal viability, however, depends on the facilities that can ensure the 'viability' and, therefore varies from place to place. In the USA for instance viability is taken to be known gestation of 20 weeks while for a long time (and perhaps even now) viability in Kenya was attained from 28 weeks gestation. APH therefore, is not synonymous to third trimester bleeding although APH occurs most frequently during the third trimester.

Causes of APH may be obstetric or non-obstetric. Obstetric causes include 'heavy show', abruptio placenta, placenta praevia, vasa praevia, circumvalate placenta and uterine rupture. Non-obstetric causes include local lesions such as cervicitis, cervical cancer, uterine and cervical polyps, vaginal lacerations, varices, blood dyscracias or other neoplasms. Our patient had APH due to placenta praevia at 29 + weeks.

Placenta praevia is the implantation of the placenta in the lower uterine segment, over or very near the internal cervical os, within the zone of cervical effacement and dilatation.[1,2] Placenta praevia (PP) is classified into four types depending on location relative to the internal cervical os and the degree of associated severity of APH. [1,2,4] These are:

Type I : (also called lateral PP, low lying placenta). The placenta is implanted in the lower uterine segment but does not reach the cervical os.

Type II : (marginal placenta praevia). The placenta extends up to the margin of the internal cervical os.

Type III : (partial placenta praevia). The placenta partially covers the internal cervical os

Type IV : (total or complete placenta praevia). The placenta completely covers the internal cervical os and is centrally placed.

Placenta praevia has also been classified into major (requires delivery by cesarean section; types IIb- IV) and minor (vaginal delivery is possible; type I). Our patient had type III or major type of placenta praevia.

In the literature, the overall incidence of placenta praevia ranges from 0.25% to 1%. [1,2,5,6,7,8] Thirty years ago, Ojwang'[5] in 1974 found an incidence of 0.25% at the Kenyatta National Hospital while Kirima[6] and Mbithi[7] found similar incidence of 0.9% and 1% in 1981 and 1983, respectively in the same hospital. Mbithi used ultrasonography and perhaps that explains the higher prevalence relative to the other incidence rates. In the USA similar incidence rates have been documented. Overall 1 in 200 births (0.5%) is associated with placenta praevia in the USA, though the reported incidence rates range from 0.26% to 0.55%.[1,2,7] Although the aetiology of placenta praevia is unknown, it is postulated that defective vascularization probably resulting from inflammation or atrophic changes could be the cause. Certain factors, however, have been associated with increased risk of placenta praevia. These include previous (lower uterine segment) cesarean section, multiparity, advanced maternal age, anaemia, erythroblastosis, succenturiate lobe or placentae membraneceae, cigarette smoking and increased placental area due to multiple gestation.[1,2,3] Frederiksen and associates[7], in 1999, attributed the increase of previa from 0.3% in 1976 to 0.7% in 1997 to a shift to an older obstetrical population while Babinszki et al[8] reported an incidence of 2.2% for para 5 or greater which was significantly increased compared to women of lower parity. In Sweden, Nielsen and colleagues[9] found a 5 fold increased incidence of placenta previa in women with previous cesarean deliveries. William and associates[10] found the relative risk of placenta previa to be increased 2 fold in women who smoked cigarettes. Our patient had no apparent risk factor though coitus apparently provoked the bleeding.

The cardinal sign of placenta praevia in 90% of patients is painless vaginal bleeding, which occurs rarely at the end of the second trimester but commonly in the third trimester, as was the case in our patient. Initial cramping occurs in 10% of cases. Spotting during the first and second trimesters or just before a torrential bleeding is not uncommon. Bleeding from placenta praevia may be caused by mechanical separation of the placenta from its implantation site, either during the formation of the lower uterine segment (by Braxton Hicks contraction) or during dilatation and effacement in labor, or during intravaginal examination. Placentitis and rupture of poorly supported venous lakes in the decidua basalis are other possible causes of bleeding. Unlike abruptio placenta, the abdomen is usually soft, the uterus is soft, non-tender and the fetal parts are easily palpable with a high presenting part. There is a high prevalence of malpresentation in women with previa due to the low-lying placenta displacing the presenting part. Oblique or transverse lie is found in 16% of patients.[1,2] A deeply engaged presenting part, therefore, highly suggests a minor degree of placenta praevia. Cusco's speculum (and not digital) vaginal examination is used to confirm intra-uterine bleeding and/or rule out non–obstetric causes of APH.

The precise diagnosis and classification of placenta praevia is either by ultrasonography or by digital examination under anaesthesia (EUA) when the patient is prepared for delivery by emergency cesarean section or by amniotomy and induction of labor, i.e. the "double set up". This, however, is reserved for situations where sonography is not possible. Transabdominal ultrasonography is the simplest and safest method of placental location with an accuracy of 95% to 98%.[1,10] Cautious confirmatory transvaginal ultrasonography , which has a higher accuracy in placental location, should be done where doubt exists in terms of the extend and actual placental location.[2,11] Other methods that can be used include, magnetic resonance imaging (MRI), soft tissue placentography, amniography, arteriography, displacement placentography and infrared thermography.

The management of placenta praevia depends on the fetal maturity, the type of praevia, the local infrastructure and the degree of haemorrhage. Initial admission is mandatory for all cases where blood is taken for grouping and cross match and comprehensive evaluation made. Women with preterm fetuses and no active bleeding are managed conservatively. Bed rest, analgesia and tocolytics if there are cramps and or signs of labor, transfusion (if the initial bleeding was heavy) and haematinics are prescribed. If maturity is between 24 and 34 weeks as was the case in our patient, intramuscular corticosteroids such as betamethasone or dexamethasone are given. The current recommended protocal is either intramuscular betamethasone 12mg 24 hours apart (total 48 mg) or intramuscular dexamethasone 6mg 12 hours apart for 2 days (total 48 mg). This regiment is not repeated as repeat administration of corticosteroids were associated with increased maternal sepsis (endometritis) and fetal growth (liver and brain) restriction, adrenal insufficiency, sepsis and placental infarction and neonatal necrotising enterocolitis. Thus our weekly corticosteroid administration, as earlier practised, was inappropriate. The aim of conservative management is to attain 37 completed weeks. Studies in the USA indicate that there is no significant difference in perinatal and maternal morbidity and mortality between carefully selected patients managed at home and those who are hospitalized.[1,12] Patients with access to immediate transportation to hospital in the event of haemorrhage could be allowed home and be re-admitted at about term for delivery. Expectant management is abandoned if there is active labor, uncontrolled bleeding and premature rupture of membranes or fetal death.

Though the delivery method of choice for all placenta praevia is cesarean section, type I and Type II anterior could be delivered vaginaly.[1,2] The two types require EUA and amniotomy in a " double set up" situation and subsequent induction of labor with syntocinon. This has been abandoned in the USA [1]. Emergency or elective cesarean section is performed for types II posterior, III, IV and even type I where

bleeding is excessive. One should be prepared for post partum haemorrhage (PPH) with blood and equipment and staff for hysterectomy. PPH may occur due to poor contraction of the less muscular and more fibrous lower uterine segment and/or placenta increta that is common in patients with previa. In such cases, mattress suturing, oxytocin, prostaglandins or methylergonovine should be used. Hysterectomy is the last resort. Although low uterine incision is commonly used, classical incision may be required to secure sufficient room in case of poor lower uterine development and to avoid incision through the placenta and the potential infant anaemia.[1,2]

Malpresentation in up to 38.3% of cases, premature rupture of membranes (11%), cord prolapse (1.7%), increased perinatal and maternal morbidity and mortality are some of the complications of placenta praevia. Fetal prematurity causes 60% of the perinatal mortality in the USA[1,2] Intrauterine fetal restriction and unexplained fetal anomalies have been reported in 20% and 2.5% respectively.[1,2] None of these complications occurred in our patient.

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OBSTETRIC CASE No. 6

PRIMARY POSPARTUM HAEMORRHAGE—CERVICAL TEAR AND RETAINED PLACENTA: REPAIR AND MANUAL REMOVAL OF PLACENTA.

Name : JNN
Age : 25 years
File No :0975018
Parity : 1+0

DOA : 03/08/2004
DOD : 05/08/2004
LNMP : 25/10/2003
EDD : 31/07/2004
GBD : 40 weeks

Presenting Complaint

J.N.N was referred to Kenyatta National Hospital from a private clinic in Githurai Estate, Nairobi with delayed second stage of labour for 4 hours.

History of Presenting Complaint

She presented to the aforementioned health facility with a 4 hour history of labour pains on 03/08/2004 at 6.30 a.m. Examination then indicated that she was in fair general condition, with uterine fundal height corresponding to term gestation. The foetal lie was longitudinal with cephalic presentaion, descent of 5/5 fetal heart rate of 136/minute and cervical dilatation of 8 cm. Amniotomy was done and clear liquor obtained. Her contractions were strong. At 8.30 a.m, she was noted to be in second stage but three hours later she had not delivered, hence the referral. Her antenatal care had been in the referring clinic from 34 weeks when she had a haemoglobin level of 12.0 gm/dl, normal urinalysis, 0+ve blood group and negative VDRL test. Counseling and testing for HIV had not been done.

Past Obstetrics and Gynaecologic History

JNN was a para 1+0 gravida 2. Her first delivery in 1997 was SVD at term to live male infant with birth weight of 2500 grams. Labour was short; about 3-4 hours, but delivery and puerperium were uneventful. Post delivery she used injectable medroxyprogestrone acetate for only three months then switched to the

combined oral contraceptive pills for up to 8 months prior to conception. She had neverhad a papanicoloau smear done.

Past Medical History

This was not significant.

Family and social History

JNN was a married cook in local hotel who attained only primary education, never smoked but drank alcohol moderately. She lived with her husband who was technician with a petroleum company in Nairobi. There was no history of any familial disease.

PHYSICAL EXAMINATION

General Examination

She was in good general condition with mild dehydration but without pallor, pedal oedema , lymphadenopathy or jaundice.

Abdominal Examination

The fundal height was term. The fetus was in longitudinal lie, cephalic presentation and the head was engaged. Fetal heart tones were present and regular at 136/minute. Uterine contractions were 3 in every 10 minutes each lasting 20-25 seconds.

Pelvic Examination

Her external genitalia were normal. The cervix was soft, 9 cm dilated and less than 0.5 cm long. The pelvis felt adequate. There was no caput succedaneum or moulding. Scanty liquor was clear.

Impression

An impression of delayed second stage due to poor uterine contractions was made

MANAGEMENT

An intravenous cannula was secured and 5 units of syntocinon in 500 mls of 5% dextrose titrated against uterine contractions. Within 10 minutes of syntocinon infusion, she had SVD to a live male infant with a birth weight of 3900 grams and an Apgar score of 9 and 10 at 1 and 5 minutes respectively. This pre-empted a planned vacuum assisted vaginal delivery. She was noted, however, that she was bleeding

profusely. Speculum examination revealed a posterior cervical tear. Besides placental delivery was not achieved 15 minutes postpartum and the patient continued to bleed despite 40 units of syntocinon, bladder catheterization and uterine massage. The patient was quickly appraised of her delivery complications and the need for urgent manual removal of the placenta and repair of the cervical tear under general anaesthesia. Consent was obtained and the patient was wheeled to theatre after drawing blood for grouping and cross match.

Under general anaesthesia (GA), vulvo-vaginal toilet was done then bladder catheterization. Examination under GA confirmed the vertical posterior cervical tear; about 4 cm long and a mild first degree saggital perineal tear. Attempts to remove the placenta by controlled cord traction were futile. This necessitated manual placental removal. With the left hand the fundus of the uterus was held while the right hand was introduced into the vagina and the uterus along the cord. The placenta was found to be fundo-anterior. With the fingers of the right hand apposed and with ulna aspect of the hand the placenta was peeled from the uterine wall and removed. It was noted to be complete. With the aid of an assistant's fundal pressure and exposure with the Sim's speculum, the lips of the cervical tear were held with sponge forceps. Repair and haemostasis were achieved by a continuous stitching with chromic catgut number '0' on atraumatic needle. The stitching began proximal to the apex of the tear. Perineal tear repair preceded smooth GA reversal. She had postpartum haemorrhage (PPH) of an estimated post-partum blood loss of 1100 mls. Postoperative vital signs were: BP, 100/65mmHg, PR 90/mnute, RR 18/min and temperature 36.5oC.

Postpartum Care

Vital signs were observed ½ hourly until she was fully awake. Syntocinon, colloid infusion and start doses of crystalline penicillin, gentamicin and ergometrine were administered. Observation for any vaginal bleeding was made. This did not occur and the patient did not need blood transfusion. She was put on haematinics, amoxycilin and mefenamic acid. Forty-eight hours later she was discharged home with her baby both in good general condition. She was to visit the post-natal clinic in 6 weeks' time. Upon review then she was in good general condition. Breast, abdominal, pelvic and calf examination were without any anomaly. Advice on contraception was given. She opted for injectable depot medroxy-progesterone, which was administered to her in the family welfare clinic after appraisal on its side effects.

DISCUSSION

The patient presented was a 25-year-old para 1 + 0 at term who developed PPH due to cervical tear and retained placenta after vaginal delivery to a 3900- gram live infant. She underwent manual removal of the

placenta and cervical tear repair under general anesthesia and subsequently had uneventful postpartum period.

Post-partum haemorrhage has traditionally, been described as the loss of 500mls or more blood after completion of the 3rd stage of labour. Blood lost during the first 24 hours after delivery is referred to as early or primary post-partum haemorrhage. While that lost between 24 hours and 6 weeks after delivery is referred to as late or secondary post-partum haemorrhage.[1,2] Our patient lost more than 1000mls of blood within the first 24 hours hence the diagnosis primary PPH

Pritchand[3] found that if accurate measurements were taken, blood loss in excess of 500mls is not necessarily an abnormal event in vaginal delivery. However, estimated blood loss is usually under-estimated. He observed that estimated blood loss is commonly only about half the actual loss.[1] The general condition of any parturient after blood loss depends on her initial blood volume, haematocrit and haemoglobin concentration rather than the rigid volumetric blood loss. Some blood loss may be concealed and not measurable. Delayed bleeding after 4 hours of delivery may go unrecognized, hence, the conventional definition of PPH has limitations of under-estimation of blood loss.[4]

Postpartum haemorrhage accounts for 28% of maternal deaths in developing countries and 4.3% of maternal deaths in developed countries. The incidence of primary PPH is 5-8% worldwide.[1,2,3] In the USA, haemorrhage is the 3rd leading cause of maternal mortality and PPH is directly responsible for about one-sixth of maternal deaths. Likewise, in the United Kingdom, half of the maternal deaths from haemorrhage are due to post-partum events.[5] Here in Kenya, haemorrhage is among the leading obstetric causes of maternal deaths. Makokha[6] in 1980 found maternal mortality due to PPH to be 15.2% and was the leading cause of mortality, while Obore,[7] found that haemorrhage was the second leading cause of death, after peuperal sepsis, accounting for 10.3% of all maternal deaths in KNH from 1995-1999. PPH was responsible for half of these deaths.

Causes of primary PPH include uterine atony, retained placental tissue, trauma to the genital tract and coagulation disorders.[1,2,4] Failure of the uterus to contract and retract properly following delivery is the commonest cause of PPH. Factors predisposing to uterine atony include labour either initiated or augmented with oxytocin, prolonged labour, very rapid labour, uterine infections, operative delivery, intra-uterine manipulation, over-distended uterus from large foetus, multiple fetuses or polyhydroamnios.[1,2,4] Other associations include fibroids, full bladder, previous haemorrhage during 3rd stage, vaginal delivery

after caesarian section and use of halogenated anaesthetic agents e.g. halothane. A woman of high parity may be at increased risk of uterine atony. Fuchs et al[8] found a four-fold increase in the incidence of PPH for para 7 or greater compared to the general obstetric population while Babinski et al [9] reported the incidence of PPH to be 0.3% in women of low parity compared to 1.9% in those para 4 or greater

Retained placental tissue and membranes causes 5-10% of cases of PPH. Such retention is associated with abnormal adherence of placental tissue to the uterus (placenta accreta), unrecognized succenturiate placenta, manual removal of placenta and the mismanagement of 3rd stage.[2] In our patient, the placenta did not separate promptly after 2nd stage. A question to which there is still no definite answer concerns the length of time that elapses in the absence of bleeding before the placenta is removed manually. In our set-up, it is usually taken to be 30 minutes after delivery of the infant. A study found the median third stage duration to be 6 minutes and 3.3% were more than 30 minutes.[10] Manual removal of placenta as was done in our patient interferes with normal uterine mechanisms of haemostasis. She also had a cervical laceration, which exacerbated the PPH.

Lacerations usually result from precipitate or uncontrolled delivery or operative delivery of a large infant. It may also occur after any delivery.[2] Persistent bleeding (bright red) and a well-contracted firm uterus suggests bleeding from a laceration, cervical tear or from the episiotomy.[2] To ascertain the role of lacerations as a cause of bleeding, careful inspection of vagina, cervix and uterus is essential.[1] It is uncommon for an episiotomy done to cause severe PPH although blood loss averages 200mls.[1] Lacerations of the vagina and cervixor a rupture of the uterus may cause PPH. Our patient suffered the tragedy of having both retained placenta and cervical tear. The diagnosis of PPH in most circumstances should be obvious except in situations where intra-uterine or intra-vaginal blood accumulation is not recognized or if the uterine rupture is accompanied by intra-peritoneal bleeding.

The treatment of primary PPH starts before delivery of the placenta. It involves massaging of the uterus and oxytoxcic drugs. An intravenous infusion is established and blood is taken for grouping and cross matching. Plasma volume expanders like haemacele or cross-matched blood may be given. The placenta is then delivered by continuous cord traction (CCT) or manually removed under suitable analgesia or anaesthesia.[1,2,4] After delivery of the placenta, the patient is catheterized and the bladder is emptied. All clots should be expelled from the uterine cavity and the uterus should be massaged into a firm contraction. The placenta should be inspected for any missing cotyledons or membranes and if in doubt about its completeness, exploration of the uterus should be done under analgesia or general anaesthesia. Suturing of

lacerations and tears when identified should be done.[1,4] An extra intravenous line and a CVP line should be established depending on the degree of PPH. Strict measurement of hourly urine output is essential. Accurate charting of pulse, blood pressure, fluid balance, central venous pressure, temperature and respiration rate should be maintained.[1]

Management of PPH depends on the cause. Oxytocin infusion, intravenous fluids and the manual removal of the placenta and cervical repair managed to contol PPH in our patient. For patients with uterine atony torrential bleeding intravenous fluids, oxytocin and bimanual compression of the uterus usually stops the bleeding. Intravenous or intramyometrial injections of prostaglandin $F_{2\alpha}$ has been shown to eefectively control PPH. Oleen and Marieno[12] studied the use of 15-methyl $F_{2\alpha}$ prostaglandin (Prostin, Upjohn) in 232 women with PPH. 88% were treated successfully but the remaining 12% in whom drug treatment failed required surgical intervention. They also found the side effects associated with $PGF_{2\alpha}$ affected 20% of the women included, diarrhoea, hypertension, vomiting, fever, and tachcardia. Rectal administration of PGE_2 20mgs suppositories have also been used for uterine atony but not studied on clinical trials.[1] O'Brien et al[13] reported that misoprostol (PGE_1) 1000μg given rectally was effective in women unresponsive to the usual oxytocics.

In patients with retained placenta and PPH due to uterine atony unresponsive to conservative management, it is imperative that the patient and whenever possible, the spouse or family members are consulted and informed about the possibility of hysterectomy. That was the case in our patient. PPH due to uterine atony or unknown cause may require emergency laparotomy. Management options during laparatomy include; pressure occlusion of the aorta to provide valuable time to treat hypotension and identify the source of bleeding and plan the operative procedure.[2] Direct ligation of uterine artery has success rate of 10-15% if unilateral ligation and 75-90% if bilateral ligation is done to control haemorrhage.[14]

In bilateral internal iliac (hypogastric) artery ligation, exposure can be difficult and failure rate is as high as 57%.[2] An alternative to vessel ligation techniques is placement of a B-lynch brace suture to compress the uterus in cases of diffuse bleeding from atony or percreta. A small case serial study showed success and avoidance of hysterectomy using this approach.[15] Hysterectomy is the definitive method of controlling PPH. The procedure is undoubtfully life saving. Laparotomy was not necessary in our patient.

Some of the complications of PPH include hypovolemic shock and defect of homeostatic mechanism.Shock from haemorrhage evolves through several stages. Early in the course of massive

bleeding, there are decreases in mean arterial pressures, stroke volume, cardiac output, central venous pressure and pulmonary capillary wedge pressure.[1] Catecholamines released during haemorrhage causes a generalized increase in venular tone resulting in an autotransfusion from these capacitance reservoir.[2] These changes are accompanied by compensatory increase in heart rate, systemic and pulmonary vascular resistance and myocardial contractility. In addition, there is redistribution of cardiac output and blood volume to selective centrally mediated arteriolar constriction. This results in diminished perfusion to the splanchnic bed, skin and uterus, with relative maintenance of blood flow to the heart, brain and adrenal glands; organs that autoregulate their inflow.[16] As blood volume deficit exceeds 25%, compensatory mechanisms usually are inadequate to maintain cardiac output and blood pressure. At this point, additional small losses result in rapid clinical deterioration.

Controversy exists concerning fluid resuscitation of hypovolemic shock with colloid versus crystalloid solutions. A study found a 4% excessive mortality in non-pregnant patients resuscitated with colloid compared to crystalloid[17] while Cochrane reviewers[18] found a 6% excess mortality in albumin treated non-pregnant patients with shock. Fluid resuscitation preferably should be with crystalloids and blood.[19] Marrison et al[20] recommended transfusion for acute blood loss if haematocrit is less than 24% or haemoglobin less than 8g/dl. Our patient was given crystalloid, plasma expander (haemaccle) and whole blood.

Massive blood loss usually results in depletion of platelets and soluble clotting factors leading to a functional coagulopathy that clinically is indistinguishable from disseminated intravascular coagulopathy.[1] In some cases, frank consumptive coagulopathy may accompany shock and confuses the distinction between dilutional and consumptive coagulopathy. Fortunately, in most situations encountered in obstetrics, treatment of both is the same.[1] Successful therapy involves the replacement of essential factors faster than the body is losing. Because stored whole blood is deficient in factors V, VIII and XI and platelets, fresh whole blood or fresh frozen plasma should be given. Our patient received 3 units of FFP and 10 units of fresh whole blood.

Prevention of primary PPH necessitates:[4]

i) Prophylactic use of oxytocic drugs at the onset of 3rd stage

ii) Active management of third stage of labour

iii) Avoidance of genital trauma

Prophylactic use of oxytocic drugs with the crowning of the head or immediately the body is delivered reduces the risk of PPH by 30-40%.[11] These drugs include, ergometrine, syntometrine and oxytocin

(Syntocinone). Active management of the 3rd stage implies early clamping of the umbilical cord and delivery of the placenta by controlled cord traction (CCT) as soon as the uterus contracts and there are signs of placental separation (bleeding) and descent (lengthening of the cord).[4] The genital tract trauma associated with instrumental delivery can be minimized by ensuring that only people with appropriate competence and training conduct such deliveries.[4]

Patients at risk of PPH should have their blood typed and cross-matched immediately pre-delivery and the blood preserved in the bank for 24 hours after delivery.

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OBSTETRIC CASE No.7

SHOULDER DYSTOCIA, SUCCESSFUL VAGINAL DELIVERY – LIVE BABY

Name	: K.E.W.	DOA	: 12/07/03
Age	: 28 years	DOD	: 15/07/03
Reg. Nº	: 38.13.39	LNMP	: 05/10/02
Parity	: 1 +0 gravida 2	EDD	: 12/0703
		GBD	: 40 weeks

Presenting Complaint

She was admitted to the Aga Khan Hospital labor ward at 10.00 a.m. with 6-hour history of labor pains.

History of presenting complaint

She had been well until about 4 a.m. on 12/07/03 when she developed intermittent lower abdominal pains tha increased in intensity and frequency and radiated to the lower back. She had noted a bloody vaginal discharge 3 hours prior to admission. There was no history of urinary symptoms or preceding vaginal discharge. Her uneventful antenatal care was in Nazareth Hospital in Kiambu district from 30 weeks gestation. Though she reported that her antenatal profile had been done and were without any anomaly. there was no document to confirm the report.

Past obstetric history

K.W was a para 1 +0 gravida 2 at 40 weeks. In 1997, she had a spontaneous vertex delivery to a live male infant with a birth weight of 4000 grams. Her antenatal profile, follow up and delivery at Kiambu District hospital had been uneventful. The infant had cried immediately after delivery and was alive and well. She denied history suggestive of gestational diabetes mellitus. Her menarche was at the age of 14 years and her menstrual flow took 3-5 days every 26 to 28 days without any menstrual disorder. She had uneventfully used the combined oral contraceptive pills from 1998 to 2002. Although she had been asked to do a pap smear, this had not been done.

Past medical history was insignificant.

Family and social history

K.E.W was an employee of the Armed Forces Canteen Organization (Afco). She drank no alcohol, smoked no tobacco and reported no history of familial medical problem. She lived in Nairobi with her husband who did business in (Nairobi) town.

General examination

She was in good general, nutritional and hydrational condition; without pallor, pedal edema, jaundice or lymphadenopathy. Her BP was 130/80 mmHg, PR; 80/minute, RR; 18/minute and the temperature was 36.6 °C.

Abdominal examination

The abdomen was uniformly distended with uterine fundal height corresponding to term pregnancy. The fetus was in longitudinal lie, cephalic presentation and had a regular fetal heart rate of 148/minute. The descent was 4/5 up and the uterine contractions were moderate; 3 in 10 minutes each lasting 20 to 25 minutes. The estimated fetal weight was 4000 grams.

Pelvic examination

She had normal external genitalia and healthy vaginal mucosa. The cervix was central, 80% effaced and 6-7 cm dilated. The membranes were bulging and amniotomy revealed clear liquor and no cord was palpable. The pelvis felt adequate.

Management

She was nursed in the left lateral position and partogramming started. At 2.40 p.m. the uterine contractions were 3 in 10 minutes each 30 seconds. The descent was 3/5 up and the fetal heart tones were satisfactory. The cervix was fully effaced and 8-9 cm dilated. Mild caput succedaneum moulding grade I was noted. In view of the moderate uterine contractions, 5 units of syntocinon in 500 mls of 5% dextrose was started. At 3.00 p.m. she had the urge to push and a vaginal examination by the midwife indicated that she was in 2nd stage of labour. She was transferred to the delivery room; head propped up and encouraged to push with every uterine contraction. At 3.30 p.m. the infant's head was delivered but it remained stuck to the perineum and restitution never occurred. The midwife called for help and the senior house officer on call assisted in the delivery.

The patient was put in abducted lithotomy position and the head propped up further thereby causing hyperflexion of maternal thighs (McRoberts maneuver). Gentle suprapubic pressure by the midwife was unsuccessful. The Rubin's maneuver whereby the posterior shoulder was pushed around towards the fetal chest was also unsuccessful. This necessitated the delivery of the posterior shoulder by using two fingers to flex the elbow joint and deliver the arm. Delivery of the anterior shoulder then followed and a live infant with a birth weight of 4850 grams and an Apgar score of 7, 9 and 10 at 1, 5 and 10 minutes was delivered

Episiotomy was not done because the introitus was adequately lax and the bony pelvic outlet was holding the fetus. The delivery of the baby occurred 3-4 minutes after the head had been delivered. There was no congenital abnormality noted and no fetal injuries occurred. She developed mild postpartum haemorrhage due to uterine atony, which was managed by bladder catheterization, 0.5 mg of intravenous ergometrine, syntocinon infusion and bimanual uterine massage. There were no reproductive tract lacerations.

Post Operative Care

On the 1st postnatal day she was in good general condition with normal vital signs and mild pallor. Breasts were beginning to be active as breastfeeding was encouraged. The uterus was well contracted, the lochia loss was scanty rubral and the calves were soft and non-tender. However, on the 2nd day, she complained of pain in the left lower thigh and calf. Mild tenderness was noted but no swelling or more warmth than the right limb was noted. A Doppler flow velocimetry and coagulation screen showed no features of deep venous thrombosis. Her Hb was 9.1g/dl and the random blood sugar was normal. On the 3rd day the pain in the left lower limb had subsided and she was discharged on haematinics and ibuprofen. Advice to undergo oral glucose tolerance test six weeks postpartum and during the next pregnancy was given.

Follow up

She opted to be followed up in Nazareth Hospital for the postnatal care.

DISCUSSION

K.E.W was para 1+0 who presented in active labour at term. She went on to have a vaginal birth characterized by shoulder dystocia that required active management by use of McRobert's maneuver and posterior shoulder delivery to live infant with a birth weight of 4850 grams without major complications.

Shoulder dystocia is a subjective diagnosis. It is characterized by failure of the shoulders to spontaneously traverse the pelvis after delivery of the fetal head. A more objective definition is based on the acceptable head-body expulsion time of 60 seconds; this being the mean interval between the delivery of the head to expulsion of the body without ancillary obstetric maneuvers such as McRobert's .[1,2] Shoulder dystocia is a devastating obstetric emergency. The incidence of occurrence is varied, but among the general population it ranges between 0.2-2% births.[1,2,3] In the normal mechanism of labour the bisacromial diameter enters the pelvis at an oblique angle with the posterior shoulder ahead of the anterior one, rotating to the antero-posterior position at the pelvic outlet with external rotation of the fetal head. The anterior shoulder can

then slide under the symphysis pubis for delivery. If the fetal shoulders remain in the antero-posterior position during descent or descent simultaneously rather than sequentially into the pelvic inlet the anterior shoulder can become impacted behind the symphysis pubis and/or the posterior shoulder may be obstructed by the sacral promontory.[3]

Risk factors associated with shoulder dystocia are several but the two strongest independent factors are maternal diabetes mellitus and fetal macrosomia. If the estimated foetal weight or birth weight is greater than 4500gm or in some definitions greater than 4000gm then this is considered as fetal macrosomia.[4,5] It should be noted however that not all large for gestatinal age infants develop dystocia and not all infants who develop dystocia are large for gestational age. Rather the shoulder to head and/or the shoulder to chest ratios are the important parameters to consider. Significantly larger shoulder to head and chest to head disproportions in the infant results in shoulder dystocia as compared with equally macrosomic infants with normal proportions and no dystocia.[6] Approximately 50% of cases of shoulder dystocia occur in infants whose birth weight is less than 4000gm.[7] Because of this and as observed by Rogo et al in 1992,[8] shoulder dystocia is, therefore, an unpredictable obstetric emergency whose management should be known by every practicing obstetrician.

Diabetes mellitus results in LGA infants with significantly increased chest to head ratios and shoulder to head ratios. Maternal diabetes increases the likelihood of shoulder dystocia two to six fold over and above the non-diabetic population. Other factors that predispose to shoulder dystocia include operative vaginal delivery with dystocia being more frequent with vacuum than with mid forceps. Another risk factor is previous shoulder dystocia, evidence of which is varied. Some studies estimate the incidence of recurrence to be as low as 1% whereas others as high as 14%.[8] It should be noted however that recurrence may be low because clinicians choose abdominal delivery in subsequent pregnancies following an episode of shoulder dystocia. Other characteristics associated with recurrent shoulder dystocia include greater maternal pre-pregnancy weight, greater maternal weight gain, longer second stage of labour, higher birth weight, postdate infants, male fetal gender, advanced maternal age, fetal shoulder and maternal pelvis disproportion and labour abnormalities.[7,8,9,10] A combination of labour abnormalities and suspected fetal macrosomia are quite good predictors of shoulder dystocia.

Essentials of diagnosis include the 'turtle's sign' that is retraction of the fetal head into the perineum. It should also be suspected in situations where the normal downward pressure on the infant's head fails to accomplish delivery of the body. The goal of management in these cases is to effect delivery before fetal

asphyxia due to cord compression and inability of the chest to expand without causing peripheral neurological injury. In situations where the infant is alive a quick but stepwise approach is recommended. Initial gentle traction on the fetal head to assist the mother's expulsive efforts is made.[11] There are about 5-7 minutes available to the operator to deliver the baby if it was previously well oxygenated.[12] A generous episiotomy or proctoepisiotomy is made, the bladder emptied and the mother encouraged not to bear down during repositioning of the fetal head.

Pressure is applied to the subrapubic area in an attempt to free the impacted anterior shoulder. If this fails McRoberts maneuver may be used.[13] Other maneuvers that can be used are Rubin's maneuver or Woods corkscrew maneuver. Occasionally one has to resort to delivery of the posterior shoulder first then the anterior. Gun Zavanelli O'Leary maneuver necessitates replacement of the fetal head back to the pelvis in the occiput anterior position followed by immediate delivery via the abdominal route, in this situation one should always be prepared for an abdominal delivery of the infant. Gaskin maneuver involves the use of all four maneuvers.[13] Deliberate fracture of the clavicle reduces the bisacromial diameter but this may be difficult to do on a live fetus. Symphysiotomy definitely relieves the obstruction but is assocated with significant maternal morbidity and is generally rarely done.

Complications of shoulder dystocia can be both fetal and maternal. Fetal complications include hypoxia brain injury and sometimes death, fracture of the clavicle or humerus and brachial plexus injury. Maternal complications are both physical and psychological including the agony of a difficult vaginal delivery, risk of haemorrhage from genital tract tears or lacerations and uterine atony due to excessive manipulation and/or use of syntocinon.[11] This happened to our patient. Fortunately the woman discussed here did not have any physical complications following this delivery. Lastly it is important for all women who have had shoulder dystocia to have full documentation in their files. Details of the interval between diagnosis and delivery associated risk factors that she may have the method used to expedite the delivery and the proposed mode of delivery for future pregnancies.

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OBSTETRIC CASE No.8

MALARIA IN PREGNANCY – REMISSION WITH CHEMOTHERAPY

Name	: N.A.A.	DOA	:	29/09/04
Age	: 30 years	DOD	:	02/10/04
Parity	: 2 + 0 gravida 3	LMP	:	29/01/04
File No.	: 0985507	EDD	:	05/11/04
		GBD	:	34 weeks + 6 days

Presenting Complaint

NAA was admitted to one of the Kenyatta National Hospital (KNH) antenatal wards with progressive complaints of global headache, hotness of the body alternating with chills, generalized body weakness, joint pains an nausea and vomiting for 2 days.

History of presenting complaints

She had been well until she developed the above symptoms. She had traveled to the Lake Victoria region three weeks prior to the onset of the symptoms. While there she did not use insecticide treated mosquito nets. She had not taken any prophylactic antimalarials before departing to her rural home in Busia district close to the lake. She denied history of dysuria and urgency and frequency of urination, loin pain or abnormal vaginal discharge. She had vomited twice the previous day and thrice just prior to admission. There was no history of coughing, sore throat neck stiffness or photobia. Fetal movements were unaltered.

Obstetric and Gynanaecologic History

She was para 2 + 0 gravida 3. Her last normal menstrual period was 29/01/04, the expected date of confinement was 05/11/04 and the gestation by dates was 34 weeks plus 6 days. She had had her antenatal care (ANC) at the Makadara city council health center from 26 weeks. The antenatal profile done then was Hb of 11.2 g/dl, blood group B +ve and negative HIV and VDRL serological tests. Hitherto the pregnancy had been uneventful. She had received the first dose of 3 tablets of sulphadoxine-pyrimethamine (SP) comination. She had had two uneventful spontaneous vertex vaginal deliveries to two male infants both at term gestation and in a heath facility. The first was in 2000 and the infant weight3000 grams. The second was exactly one year later in 2001. During the second pregnancy she had symptomatic anaemia and was treated with haematinics. The infant weight was 2.9 kg. Both children were alive and well. She attained her menarche at the age of 15 yearsand since then her menses flowed for 3-4 days every 28 to 30 days without any menstrual disorder. She had used three monthly injections of medroxyprogesterone acetate from 2001

to June 2003 during which she became ammenorrhoeic. No Pap smear test had she doneand neither had she contracted any sexually transmitted disease.

Pat medical history

She was admitted to Busia District Hospital and transfused blood in early childhood due to anemia. She did not know the cause of the anemia though given the location of the district she could have had severe malaria. She had no known allergies.

Family and social history

She was a married housewife who had only attained secondary education, drank no acohol and did not smoke cigarettes. She lived with her children and husband in Kibera, Nairobi. The husband worked with a plastic manufacturing factory in industrial area, Nairobi, and did not consume alcohol or tobacco. There was no history of chronic illness in the family.

PHYSICAL EXAMINATION

General examination

She was sicklooking, febrile and mildly pale but had no dehydration, jaundice, pedal edema, oral thrush or lympadenopathy. Her temperature was 39.5 °C. The PR, BP and RR were 90 minute, 110/70 mmHg and 20/minute respectively.

Abdominal examination

The abdomen was uniformly distended and without any surgical marks. The uterine fundal height corresponded to 34 weeks and the fetus was in longitudinal lie, cephalic presentation with a regular fetal heart rate of 148/minute. There was no splenomegally or hepatomegally; and no tenderness was elicited in the loins or the suprapubic area or anywhere on the abdomen.

Pelvic examination

Her external genitalia were normal. The vaginal mucosa was healthy and the cervix was posterior, 3 cm long, closed and felt grossly normal. There was no abnormal vaginal discharge on the examining finger.

The nervous system

Her pupils were equally reacting to light. The neck was soft and kernig's test was negative. The deep tendon reflexes were normal.

The respiratory and the cardiovascular systems were essentially normal

A quick blood smear for malaria parasite showed moderate Plasmodium faciparum parasitaaemia.

Diagnosis

Malaria in pregnancy with mild anaemia at 34+ weeks gestation in a 30 year-old para 2+0.

Management

Intramuscular injections of 300 mg of paracetamol and 300 mg of β-arthemether were administered prior to her being admitted to the antenatal wards. In the ward she continued to receive daily injections of 100 mg of the arthemether and oral 1 gram of paracetamol 8 hourly. Her vital signs particularly the temperature and fetal heart tones were observed 6 hourly. She was given a fetal kick chart with which to monitor the fetal status. Widal test, haemogram and urine for microscopy, culture and sensitivity were done. Their results were as follows:

1. Haemogram: Hb: 10.1 g/dl, Platelets: 230 x 10^9/L, WBC count: 6.7 x 10^9/L.
2. Widal test : Negative
3. Urine analysis, culture and sensitivity: No abnormality noted.
4. Repeat blood smear test for malaria parasites was negative.

While in the ward the patient received counseling on the modalities of transmission, prevention and treatment of malaria. By the 3rd day of the arthemeter the temperature was back to normal and she had no nausea or vomiting. The fetal kick chart was satisfactory. She was, therefore, discharged on the 4th admission day having completed the dose of arthemeter. She was to take haematinics (Ranferon) twice a day and to be reviewed in the antenatal clinic in a week's time. Besides she was to take 3 tablets of sulphadoxine-pyrimethamine (500 and 25 mg respectively) combination every 2 weeks up to and including the entire puerperium.

Follow up

She opted to be followed up in the Makadara health center since she could not afford antenatal care at KNH.

DISCUSSION

The patient presented was 30 year-old para 2+0 admitted at 34+ weeks gestation with malaria. She had previously traveled to the Lake Victoria Basin of Kenya without taking any chemoprophylaxis for malaria. She was treated with ß-artemether and recovered. She was to take 3 tablets of sulfadoxine-pyrimethamine combination every two weeks after discharge up to and including the puerperium.

Derived from the words mal (for bad) and air, Malaria is caused by protozoa of the plasmodium species that parasitize the red blood cells and the liver after finding their way into the blood circulation through the bite of an infective female Anopheles mosquito. There are four species of plasmodium that can infect man, namely P.falciparum, P.ovale, P.malariae and P.vivax. Of the four, P.falciparum is associated with the most severe forms of malaria and the worst disease outcome.[1,2] P.falciparum is also the predominat species that causes malaria in most parts of Kenya as well as in the rest of Eastern and Southern Africa where it is responsible for 98% of cases. The other species cause the remaining cases although P.vivax is very rare.[2] Our patient had P.falciparum malaria.

The World Health Organization (WHO) estimates that over 300 million acute illness and 1 million deaths per year are caused by malaria.[3] Malaria infection during pregnancy is a major public health problem in tropical and subtropical regions throughout the world. Africa, south of the Sahara bears 90% of this global malaria burden.[4] Each year, more than 30 million African women become pregnant in malaria endemic areas and are at risk of P.falciparum malaria infection during pregnancy, yet less than 5% of these pregnant women have access to effective intervention. Approximately 1.5 million women become pregnant each year in Kenya, the majority in areas of moderate to intense malaria transmission. In 2000, Guyyat[5] reported that it caused severe anaemia in more than 6,000 primigravid women in these areas alone with resultant 4,000 low birth weight infants being born due to malaria during pregnancy each year. The prevalence of malaria varies from region to region. For instance, Rukaria[6] reported a prevalence of 21.2% in Kilifi in the Kenyan coastal region while Nyamogo[7] reported a prevalence of 42% in Kisumu in the lake Victoria region.

Areas where there is a constant repeated infection are said to be hyperholoendemic. This includes the Kenyan coastal and lake regions. The population in these areas have high immunity and epidemics do not occur here (stable malaria).[2] In the regions like Aberdare ranges and Mount Kenya areas, transmission is intermittent as there is poor community immunity and epidemics do occur (unstable malaria).[2] Our patient lived and was brought up in Nairobi (unstable transmission) and had traveled to Nyanza where she

contracted malaria. Pregnant women resident in areas of unstable malaria are at 2-3-fold risk of developing severe disease as a result of malaria infection than are non-pregnant adults living in the same region.[4]

Both humoral and cellular immunity are involved in the development of immunity against malaria. This immunity is maintained by intermittent parasitaemia. Cellular immunity is in the form of phagocytosis by macrophages while humoral factors involve the production of specific antibodies.[8] Individuals living in endemic areas are therefore usually less susceptible to infection except during periods when the immunity is impaired. Pregnancy impairs immunity against malaria so that even in the hyper-endemic regions where tolerance to the parasites has previously been acquired, infection readily occurs. The increased propensity to malaria may be as a result of high cortical levels found in pregnancy as well as the decreased cellular immunity especially seen in the third trimester. The glycoproteins of pregnancy have also been implicated by their inhibition of the transformation of monocytes into macrophages. Additionally, sequestration of the parasites in the placenta shield them from destruction by maternal effector cells.[9] Multiparity appears to conver some protection against this increased susceptibility during pregnancy such that the breakdown in immunity is most marked during the first pregnancy. However, this only holds true for those who have developed immunity.[1,2] Our patient was para 2+0 in the 3rd trimester and was not exposed to persistent malaria challenge necessary for her to mount the semi-immunity found in those living in endemic areas.

Malaria is characterized by fever, joint pains, myalgia, nausea, vomiting, headache, generaliased body weakness and other systemic symptoms depending on the severity. The clinical signs incude pallor, pyrexia and splenomegally. Hepatomegaly and jaundice may occur. Our patient presented with headache, fever, generalized body weakness, joint pains and myaglia without jaundice.

The diagnosis of malaria is usually confirmed through laboratory a blood smear that helps in identifiying the mlaria trophozoites and their quantity. Our patient had moderate parasitaemia of 2.3% of RBCs in the peripheral film. Other features in the peripheral blood picture may include anisocytosis, macrocytosis and polychromasia with or without nucleated red cells. There may also be reticulocytosis.[10] The bone marrow shows megaloblastic changes which may be gross. Malaria pigment is present in the macrophages. Iron stores tend to be increased unless there is concurrent iron deficiency.[10] The mean haemoglobin level in pregnant women with malaria parasites has been found to be lower than in parasite negative women as was the case in our patient whose Hb was 10.1g/dl.[1,6,7] Other investigations that could be done aim at excluding other causes of pyrexia in pregnancy such as urinary tract infection, salmonelosis and meningitis. Our

patient had a negative widal test, white blood cell count and differentials were normal, urine for microscopy was normal and culture did not grow any bacteria.

Both the mother and the fetus are at risk of developing malaria related complications. For the pregnant mothers malaria is associated with increased severity of the infection. This acute severe infection may be complicated by severe anaemia (Hb<5g/dl), cerebral malaria, acute renal failure, hypoglycemia, disseminated intravascular coagulation, acute pulmonary oedema, increased susceptibility to pneumococal infections and postpartum sepsis.[8] The mortality from cerebral malaria in pregnancy is about 50% compared to 20% in non-pregnant adults.[10]Anaemia results from rupture of parasitised erythocytes, opsonization of these cells by reticuloendethelial system, hypertsplenism, folic acid deficiency, hyperferrtinaemia, depression of bone marrow leading to reduced red cell synthesis and probably by production of autoantibodies which result in intravascular haemolysis.[10] Hypoglycaemia may result from release of insulin triggered by stimulation of pancreatic islet cells by products of malaria parasites or macrophages activation. Increased glucose consumption due to fever, consumption by the malaria parasites and foetus also contribute to hypoglycaemia.[11]

Fetal complications due to plasmodium falciparum malaria during pregnancy include increased chances of abortion, prematurity, intra-uterine growth restriction and infant low birth weight, which is the single risk factor for death in the 1st month of life.[12] Malaria has been estimated to cause 8% to 14% of all low birth weight babies and 3% to 8% of all infant deaths in areas of Africa with stable malaria transmission.[3] Impaired foetal growth results from reduced placental blood circulation in the intervillus space that develops from placental parisitization. The foetus is usually protected from acquiring malaria in the uterus by the placental barrier, circulating maternal antibodies and the fact that the foetal haemoglobin (HbF) is more resistant to the parasite. However, congenital malaria may occasionally develop especially in non-immune women.[13] The incidence of congenital malaria in endemic areas is estimated to be <1%.[10] A study in Malawi detected parasites in 35% of cord blood of infants whose mothers were infected with malaria.[14] In Africa, however, clinical disease is rarely seen in neonates.

Management of malaria is both specific and supportive. The treatment is dependent on the severity of the disease the geographical area and the local pattern of drug resistance. Chloroquine is the drug of choice in chloroquine-sensitive areas. The aim of the treatment in malaria is to reduce pyrexia and stop the attack as quickly as possible.[10] Patients with severe malaria are hospitalized and given parenteral quinine tretment. Those with milder forms of malaria are give 4-amnioquinolenes, chloroquine and amodiaquine as drugs of

choice in areas where thereis no resistance to these drugs. The Quinhosa derivatives such as artemesinin or artemether may also be used after the first trimester as was the case with our pattient.[1,6,7,10] WHO recommends that the following drugs should not be used in pregnancy: Halofantrine, Primaquine, Tetracycline and Doxycycline.[15]

Anaemia responds rapidly in most patients following anti-malarial therapy and folic acid, but the haemotocrit does not rise in patients with hyperactive malarious splenomegaly.[10] Blood transfusion is indicated only if the patient is in incipient or established cardiac failure or if the patient is approaching delivery with an Hb<7g/dl. Mothers with severe forms of malaria in labour may need shortening of the second stage of labour by assisted vacuum delivery. Care is taken to avoid postpartum haemorrhage or cardiac failure that may occur in moderately or severely anemic mothers after delivery. Active management of third stage is recommended.[2,10] After successful treatment, malaria chemoprophylaxis is necessary throughout the remaining period of pregnancy including peurperium as happened with our patient. This clears and prevents placental parasitization

The National Malaria Strategy[16] and WHO recommend the use of intermittent presumptive treatment (IPT) and insecticide treated bed-nets (ITNs) in prevention of contracting the disease, particularly in areas of stable malaria transmission. ROLL BACK MALARIA is a global partnership founded in 1998 by the World Health Organization (WHO) the United Nations Development Programme (UNDP), the United Nations Children Fund (UNICEF) and World Bank with the goal of halving the world's malaria burden by 2010. One of the foci of this partnership is to strengthen care management of malaria for all pregnant women and to prevent malaria during pregnancy using cost effective preventive approaches (IPT and ITNs) delivered through antenatal clinics and programmes that provide health services to the community.

The objective of IPT is to provide all pregnant women with at least two preventive treatments of an effective anti-malaria drug; one in the 2nd trimester and another in the 3rd trimester. This approach has been shown to be safe, inexpensive and effective. One study in Malawi evaluating IPT showed a decline in placental infection (32% to 23%) and in the number of low birth weight babies (23% to 10%). It also found that 75% of all pregnant women took advantage of IPT when offered.[15] Insecticide-Treated Nets (ITN's) decrease both the number of malaria cases and malaria death rates in pregnant women and their children. A study in an area of high malaria transmission in Kenya has shown that women protected by ITNs every night during their first four pregnancies produce 25% fewer underweight or premature babies.[15] In addition, ITN use also benefits the infant who sleeps under the net with the mother by decreasing exposure to malaria infection.

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OBSTETRIC CASE No. 9

RHESUS NEGATIVE MOTHER WITH BAD OBSTETRIC HISTORY-SUCCESSFUL INDUCTION OF LABOUR, LIVE BIRTH

Name :	W.M	DOA	: 21/07/04
Age :	26 years	DOD	: 24/07/04
Parity :	2 + 0 gravida 3	LNMP	: 26/10/03
File No:	0901913	EDD	: 02/08/04
		GBD	: 38 weeks

Presenting Complaint

W.M. was admitted following referral from Airport Medical Clinic for delivery due to bad obstetric history.

History of Presenting Complaint

She was known to be Rhesus negative. She had attended antenatal clinic at Airport Medical Clinic and at Kitengela health centre from where she was referred for delivery. She was not in labour, not draining liquor, and had no per viginal bleeding. The fetal movements were unaltered.

Obstetric and Gynaecologic History

She was para 2+0 gravida. Her first delivery was in 1997 when she had a premature birth at 7 months (28 weeks) to a live infant who died 3 days later. Her second delivery was in 1998 when she delivered a fresh stillbirth at 7 months (28 weeks) gestation. During both deliveries she was not given anti-D immunoglobulins and neither was she investigated though she had delivered in a health center.

Her last menstrual period was on 26/10/03 and the expected date of delivery was 4/08/04. Gestation by dates was therefore 38 weeks. She had attended Airport medical clinic for ANC once at 30 weeks and later went to Kitengela health centre where she was seen three times from 32 weeks. At 38 weeks she was referred to Kenyatta National Hospital for delivery. She had no other test for antenatal profile except blood group, which was B Rhesus negative. She had attained menarche at 16 years. Her menses were regular occurring every 28 days and lasting five days. She had no history of use of contraceptives.

The past medical history was insignificant

Family and Social History

She was a married housewife. Her husband was a casual laborer. They lived in Mlolongo informal settlement. She did not smoke cigarettes or take alcohol. There was no family history of chronic illness.

HYSICAL EXAMINAPTION

General examination

She was in good general condition. She was not pale, not jaundiced, and had no edema. Her pulse rate was 62 beats per minute, blood pressure was 100/70 mmHg, her respiratory rate was 22 breaths per minute and her temperature was 36.5^0C.

Abdominal examination

The abdomen was uniformly distended and moved with respiration. The fundal height was term and a single fetus was in longitudinal lie in cephalic presentation. Fetal heart tones were heard and regular at 144 beats per minute.

Pelvic examination

There were normal external genitalia. The cervix was posterior, soft, 3 cm long and its os was closed.

Diagnosis

She was diagnosed as a Rhesus negative mother at 38 weeks with bad obstetric history.

Investigations

Haemogram : Hb: 10.8 g / dl
WBC – 7.1 x 10^9/l
Platelets: 176x10^{12}/l

Renal function tests: Na^+ - 141 mmol/l
K^+ – 3.9 mmol/l
Urea – 2.6mmol/l
Creatinine – 63 umol/l

Indirect Coomb's test: Negative

Blood group : B Rhesus Negative

Management

A decision to induce labour was made, in view of the early 3rd trimester pregnancy losses and her rhesus negative blood blood. She was induced with misoprostol of which 50mcg was inserted into the posterior fornix of the vagina and repeated after 8 hours. She went into labour four hours after the second insertion. She was wheeled to labour ward. On examination she was found to have cervical os dilatation of 4 cm. Artificial rupture of membranes was done and clear liquor obtained. Syntocinon infusion of 5 IU in 500 mls of 5% dextrose was started. She was also started on a partogram. She progressed well and delivered after five hours to a male infant weighing 3000 grams, and scored 9 at 1 and 10 at 5 minutes. The cord blood was taken for Hb level, blood group and direct combs text. Serum bilirubin and reticulocyte count were not done.

The results were:

- Hb - 16g/dl,
- Blood group - O Rhesus positive
- Direct Coomb's test - negative.

The mother was given Anti-D globulin 300 μg on the second day after delivery. The baby remained well and did not develop jaundice. Both baby and mother were discharged 48 hours after delivery for follow up at the postnatal clinic after 6 weeks.

Follow up

At postnatal clinic both the mother and the baby were fine. The mother was counseled on family planning and sent to the family welfare clinic for further counseling and to receive 300 mg of intramuscular medroxyprogesterone acetate DMPA that she had opted for. She was also advised to continue with the well-baby clinic, which she had attended once.

DISCUSION

The patient presented was a 26 year-old para 2 + 0 gravida 3 at 38 weeks with rhesus negative blood group who underwent successful induction of labour. She delivered a live 3000-gram male infant with a good Apgar score and blood group O rhesus positive. She received 300μg of anti-D and thereafter she and her infant had uneventful follow up.

The first suggestion that erythroblastosis foetalis was an alloimmune disorder was made by Levin and Stetson.[1] They suggested that an immunizing property was inherited by a foetus with hydrops foetalis, from the mother, and passed into maternal circulation causing her to develop the agglutinins. Although Landsteiner and Weiner.[2] had discovered the Rhesus factor in 1940 in erythrocytes of the Rhesus monkey, it was not until 1942 that the role of alloimmunisation in the pathogenesis of erythroblastosis foetalis was established. On the cell membranes of red blood cells (RBCs) are specialized groups of glycoproteins that habour antigenic activity. These form the ABO and the Rhesus blood group systems. An individual is blood-typed according to the antigenic glycoproteins present on the cell membranes of one's RBCs. Thus one is said to be rhesus positive when the rhesus antigens are present and negative when they are absent. Our patient's RBCs had no Rhesus antigen and therefore was Rhesus negative.

Although various classification systems have been reported, the most used nomenclature, the Fisher Race, has five major antigens; C,c,D,d,E,e. Three appropriate letters describe a rhesus gene complex . Hence eight gene complexes could exist namely Cde, cde, cDE, cDe cdE, CDE, and CdE.The vast majority of rhesus alloimmunization causing transfusion reactions or serious erythroblastosis foetalis is the result of incompatibility with regard to the D antigen commonly called rhesus antigen. Usually, therefore, rhesus positve denotes presence of D antigen and rhesus negative indicates its absence. The D antigen appears very early in embryonic life. It has been demonstrated on the RBCs of a 38-day-old fetus.[3] The precise function of the rhesus antigen is unknown though they probably have a role in maintaining the cell membrane integrity. Cells, which lack the rhesus antigen manifest with several membrane defects incuding, increased osmotic fragility and abnormal shapes.

Although there is no significant difference in the distribution of the rhesus antigens with regard to gender there exist an important racial difference. The incidence of rhesus negativity ranges from zero in the Mongoloids to the highest incidence rate of 30-35% among the Basque people. Caucasians have an incidence of 15-16%, African blacks 4% Indocaucasians 2% and North American Indians 1% .[4] In Kenya Mulandi found a prevalence of 4.1% in antenatal women at Kenyatta National Hospital (KNH) compared to 3% in the general population as found by Kingo .[6] in Tanzania and noted by Mulandi.

Rhesus isoimmunization occurs when sufficient numbers of foetal rhesus positive erythrocytes gain access to the maternal circulation and the mother has rhesus negative erythrocytes and the capacity to produce antibodies against the D antigen. Fetal RBCs enter maternal circulation during pregnancy and the immediate postpartum period. The amount of fetal haemrrhage necessary to cause isoimmunization varies from patient to patient. As little as 0.1 mls of rhesus positve RBCs have been showed to cause sensitization .[7] However

during normal pregnancies and deliveries, over 50% have fetomaternal haemorrhage or less .[8,9] Risk factors that increase fetomaternal haemorrhage include abortion, caesarean delivery, manual removal of the placenta, multiple gestation, antepartum haemorrhage and intra uterine manipulation. The majority of the fetomaternal haemorrhage occur in patients without risk factors who have uncomplicated vaginal deliveries [8]

The amount of fetal blood in the maternal blood can be calculated using many tests, but the commonest used method is the Keihauer-Betke test. This test is based on the fact that fetal RBCs are less likely to get haemolysed than maternal RBCs because of the difference in the haemoglobin combination between maternal and fetal RBCs. Left untreated with rhesus immunoglobulin, about 16% of rhesus negative mothers will become isoimmunized by their first rhesus incompatible, ABO compatible pregnancy .[9] With no apparent predisposing factor studies [4,8] have detected fetal blood in maternal blood in 6.7% of women during the first trimester, 16% during the second and 29% during the third trimester. This contrasts from findings by Kizza's study at Kenyatta National Hospital, Nairobi that showed fetomaternal haemorrhage in 15.4% of women during the first trimester, 29.5% during the second trimester and 38% during the third trimester. Antepartum sensitization, however, rarely occurs before third trimester. This is because of the cellular feto-maternal blood barrier reduces to to two cells: the syncytiotrophobloast and the capillary endothelium- so called placental aging. To avoid this induction of labour is usually instituted at 40 weeks. However, our patient underwent induction of labour at 38 weeks to pre-empt both the higher incidence of isoimmunization in the late 3rd trimester and the unknown cause(s) of her two previous fetuses in the early 3rd trimester.

The management of women with regard to rhesus isoimmunization begins with blood typing. Any pregnant woman, therefore, should have her blood typed. Husbands of rhesus negative women should have their blood typed too. By direct Coomb's test, antibody screening is performed at 28 weeks gestation in unsensitized rhesus negative mothers. If negative 300 μg of rhesus immunoglobulin (RhIgG) is given. If positive the patient should be managed as Rh-sensitized. Rh-negative mothers should receive intramuscular 300 μg of rhesus immunoglobulin ('anti-D') any time there is a risk of fetomaternal haemorrhage (abortion, antepartum haemorrhage, amniocentecis, etc). Ideally the dosage of RhIgG should be 10μg per milliliter of whole fetal blood. However, this is rarely done due to the cost of the test and the fact that fetomaternal haemorrhage greater than 30 mls (the volume covered by 300 μg) occurs in only 0.4-1% .[4,10] The lifespan of anti-D is 12 weeks. Mothers who are given the drug at 28 weeks should therefore receive another 300 μg at 40 weeks. However, to avoid this they are delivered instead. As happened with our patient, upon delivery, cord blood is taken for haemoglobin and bilirubin levels, direct Coomb's test (DCT), blood typing and, if necessary, cross match. If the baby's RBCs are rhesus positive, the mother is given 300 μg of anti-D. Our patient received the

300μg of anti-D because the baby was Rhesus positive and the negative DCT test and baby's haemoglobin level of 16 g/dl indicated no prior isoimmunuzation.

In those who are with no history of affected fetus, the patients are followed up by antibody titres at booking, at 20 weeks and then every 4 weeks. As long as titres remain less than 1:32, no further intervention is required. But once antibodies reach 1:32 amniocentesis is mandatory. In those who had a prior affected fetus, there is no need to follow antibody titres. Instead amniocentesis should be performed 4-8 weeks earlier than the gestational age in the previous pregnancy when rhesus associated morbidity was first identified. Cordocentsis, amniocentesis and ultra sound with Doppler velocimetry have been used in the follow up and management of these patients. The essence of amniocentesis is that liquor has bilirubin and other blood breakdown products the concentration of which is directly proportional to the haemolysis of fetal blood. The amniotic fluid is analysed by spectrophotometry and by the amount of light (wavelength 450) absorbed by the blood breakdown products (bilirubin) and plotted on semilogarithmic scale versus gestational age.

This forms the Liley's chart, which is divided into three zones: Zone 1, Zone 2 and Zone 3 in severity of fetal affliction .[11] The unaffected or mildly affected fetus falls into zone 1. Amniocentesis is repeated every 2-3 weeks and the fetus delivered near term. The moderately affected fetus falls into zone 2 where amniocentesis is repeated every 1-2 weeks. Delivery is generally before term as soon as pulmonary maturity is achieved. In the severely affected fetus in zone 3, intervention is usually needed to allow the fetus to reach a gestation\al age at which delivery and neonatal risks are fewer than the risks of uterotherapy. Intrauterine transfusion may be necessary to prevent the fetus from dying. This may be done by the old fashion intraperitoneal or the new, prevered intravascular methods.[4] In conjunction with Doppler velocimetry, ultrasonograpgy is used in amniocentesis, cordocentesis, intrauterine blood transfusion,biophysical profile score and as a predictor for significant prehydropic fetal anaemia.[9]

Since adequate and appropriately administered anti-D prophylaxis against isoimmunisation is quite cheap and highly effective relative to management of isoimmunized mother, everyone should endeavor to provide this prophylaxis. Certainly this cannot be overemphasized in the developing countries where advanced technology such as intrauterine blood transfusion and intensive neonatal care are unavailable to most patients. Further it is important to emphasize that an isoimmunized patient requires a multi-disciplinary team of obstetricians, paeditricians, haematologists and sonographers among others.

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OBSTETRIC CASE No. 10

CORD PROLAPSE, EMERGENCY CAESAREAN SECTION: LIVE BABY

Name	: V.W.R.	LMP	:	18/4/03
Age	: 31 Years	EDD	:	25/1/04
File N°	: 38.70.39	GBD	:	40 weeks + 5 days
Parity	: Para 1+ 0 gravida 2	DOA	:	30/1/04
		DOD	:	3/2/04

History of Presenting Complaint

V.W.R. was admitted to the Aga Khan Hospital, Nairobi (AKHN) labor ward with labour pains for 6 hours.

History of Presenting Complaint

She had been well until she developed lower abdominal pains that were intermittent and increased in frequency and intensity and radiated to the lower back. There was an associated show but no drainage of liquor or urinary symptoms. Fetal movements prior to this had been unaltered.

Past Obstetric and Gynaecologic History

She was a para 1+0 gravida 2 at 40 weeks +5 days. Her LNMP was18/4/03 and her EDD was 25/1/04. In 1997 she had spontaneous vertex delivery of a life infant who weight 3500 grams and had an Apgar of 8 and 10 in 1 and 5 minutes, respectively and was alive and well. Pre-, intra- and post- partum periods were uneventful then. She had used the combined contraceptive pills until 2002 when she stopped the in order to conceive. A Pap smear done in 1998 was normal. She attained menarche at 14 years and had normal menstrual flow for 3-4 days every 28 days.

History of the Present Pregnancy

Her uneventful antenatal care was at the AKHN from 27 weeks gestation. Her booking weight was 58.4 kg and the BP; 100/60 mmHg. An obstetric ultrasound then showed a singleton in cephalic presentation at 28 weeks without any anomaly. The antenatal profile was: Hb of 14g/dl, blood group A +ve and VDRL and HIV serological tests were negative. A computed gestational age by a repeat ultrasound at 36 weeks gestation by dates corresponded with the dates and had no anomaly.

Past medical history was insignificant.

Family and Social History.

V.W.R was married and did not smoke any substance nor did she drink alcohol. She lived with her husband. There was no history of a familial medical disorder.

PHYSICAL EXAMINATION

General examination

She was in good general, hydrational and nutritional condition without pallor, pedal edema, lymphadenopathy or jaundice. Her vital signs were: BP; 120/70 mmHg, PR; 70/minute, RR; 18/minute and temperature; 36.7 °C. Her weight was 65kg.

Abdominal Examination

The abdomen was uniformly distended without any scars. Palpation revealed a term fundal height and fetus in longitudinal lie and cephalic presentation. The descent was 4/5 and 2 contractions occurred every 10 minutes each lasting 20-30 seconds. The fetal heart tones were heard at a rate of 144/minute. No other organomegally was noted.

Pelvic Examination

She had normal external genitalia. Digital examination showed a cervix that was 6 cm dilated, central and 80% effaced. The membranes were intact and bulged through the dilated cervix. No cord was palpated. The pelvis was adequate. Upon amniotomy, however, a gush of liquor occurred and further palpation revealed a prolapsed, pulsating umbilical cord at the level of the external cervical os.

Diagnosis

A pulsating umbilical prolapse cord at term in a 31 year-old para 1+0.

Management

The hand was kept in the vagina to prevent any further prolapse. The assisting midwife was asked to alert the theatre staff to prepare for an emergency caesarean section (c/s) and to call in the anaesthetist. The head of the bed was lowered informed consent obtained and blood taken for quick grouping and cross match. Without shaving, the patient was then wheeled to theatre with the hand in the vagina to prevent further prolapse cord spasm upon exposure to the cold exterior.

The theatre staff was ready for the emergency c/s .The surgeon withdrew his hand while a nurse inserted her hand into the vagina. While the surgeon scrubbed general anesthesia was administered. The usual

catheterization was not done. Before the nurse withdrew his hand he confirmed that the cord was stillpulsating and auscultation with the fetosope showed fetal heart tones at 126 beats per minute that were beginning to be irregular. Quick abdominal cleaning and draping preceded abdominal opening via a sub-umbilical incision. Without the usual packing of the paracolic gutters, a lower uterine section c/s and cephalic extraction delivered a live male infant who weight 3550 grams and an Apgar score of 7 at 1 minute and 10 at 5 minutes. Meconeum staining liquor grade I was noted. The placenta the cord and the membranes were delivered by continuous cord traction. They were grossly normal. Uterine repair in two layers achieved haemostasis. After receiving a report of correct account of instruments, gauzes and needles. the abdomen was closed in layers, the skin with 3/0 monocryl. The reversal of the general anesthesia was smooth.

Post Operative Care

She was transferred to the recovery room where vital signs were observed ½ hourly. One hour later she was fully awake and was, therefore, transferred to the postnatal ward. Recovery was good. On the 1st postoperative day, she had passed urine twice, was in good general condition and had normal vital signs. Breasts, abdominal, pelvic and calf examination were all essentially normal. Ambulation, oral sips of warm water to graduate to light diet by midday and oral antibiotics were started. She opened bowels on the 2nd day and was put on normal diet. Her check Hb on the 3rd day was 10 g/dl and breastfeeding had been established. She was ready for discharge home but she opted to stay an extra one-day. The baby and herself were doing well when they were discharged home on the 4th day on oral antibiotics and haematinics. She was to visit the postnatal clinic 1 week later.

Follow up

On the 12th postoperative day she was reviewed in the postnatal clinic. Her general condition was excellent. No pallor, pedal edema or jaundice was noted. Her vital signs were normal. The breast were active, uterine fundal height corresponded to 14 weeks gestation, the incisional wound was well healed and lochia was serosal. Assurance and contraceptive advice were provided. In the 6th postoperative week, she had fully recovered. A pap smear was taken from the grossly normal cervix. Her husband and she had opted for injectable medroxyprogesterone acetate (Depo Provera), which was given to her. She was booked in the Family Planning Clinic in the same hospital where she was to be followed up. Two weeks later she was seen in the FP clinic and the pap smear report indicated she had normal cervical cytology (CIN0/SIL0).

DISCUSSION

V.W.R. was 31-year-old para 1 +0 patient who developed cord prolapse and a subsequent irregular fetal heart tones after amniotomy while in active labour. She underwent emergency c/s with good maternal and fetal outcome.

Umbilical cord anomalies could be divided into developmental and accidental disorders. Developmental disorders include anomalies of length (normal range30-100cm), vascular number (2 arteries + 1 vein) and insertion (usually singly at center of placenta). Accidental abnormalities consists of true knots, loops round parts of the body mainly the neck, torsions, prolapses and strictures.[1] The "accidents" occur as a result of fetal movements. Umbilical cord prolapse (UCP) is defined as descent of the umbilical cord into the lower uterine segment to the level of or beyond the presenting part when the membranes are ruptured. It should be distinguished from cord presentation where the descent is the same but the membranes are intact. There are two types of UCP: occult (descent at the level of the presenting part) and overt (cord lies below the presenting part). Umbilical cord prolapse is one of the most serious obstetric emergencies because of the very high perinatal morbidity and mortality caused by fetal exsanguination by compression of the cord between the foetus and the uterus, cervix, or pelvic inlet.[2]

The incidence of UCP reported in the literature ranges from 0.14 to 0.62 percent and has not changed in years.[3] However, in the USA, perinatal mortality related to UCP, which was as high as 375 per 1000 births in the early 1900s, has fallen to between 36 and 162 per 1000 births within the past few decades.[4] The causes or predisposing factors are those, which lead to inadequate application of the presenting part to the cervix and those due to obstetric interventions. Usta et al in 1999 reported that obstetric interventions contributes to nearly half of cases of UCP.[5] These interventions include amniotmy, scalp electrode application, intrauterine cathether insertion and external cephalic version. Our patient had UCP secondary to artificial rupture of the membranes (amniotomy). It is possible however that occult presentation existed before amniotomy. Factors that lead to inadequate filling of the cervix by the presenting part are prematurity, malpresentation, malposition, and minor degrees of placenta praevia and cephalopelvic disproportion. Others are hydramnios, multiple gestation and preterm premature rupture of the membranes. Malpresentation is one of the leading causes of UCP. It is present in to 41% of UCP.[6] The incidence of overt cord prolapse in cephalic presentation in the USA is 0.5%; in frank breech, 0.5%; in complete breech, 5%; in footling breech, 15%; and in transverse lie 20%.[2] However, the majority of UCPs occur with the vertex presentation because of the low frequency of malpresentation. None of these risk factors were obviously present in our patient.

Preterm deliveries have a higher rate of UCP, probably due to the smaller size of the presenting fetal parts and the increased frequency of malpresentation among premature fetuses. Babies with birth weight less than 1250 grams, for example, had a 19-fold increased risk of UCP in one series[6] while the risk of UCP in a term twin pregnancy is confined to the second born twin, in whom there is an increased probability of malpresentation.[7] Multiparous women have a higher risk of UCP may be due to the increased likelihood of rupture of membranes (ROM) prior to engagement of the presenting part, since engagement in multiparas often occurs after labour has begun and later than in nulliparas.[5] On the other hand the risk of cord prolapse during expectant management of patients with premature rupture of membranes (PROM) is small, but should be considered due to the potential for an adverse outcome. As an example, a review of nine studies that included 731 patients with PROM reported a 1.9 percent incidence of cord prolapse.[5] Polyhydramnios is often associated with an unstable lie. Rupture of membranes may be followed by a forceful gush of fluid that carries the cord ahead of the unengaged foetus and through the cervical os. Mean cervical dilation and station at the time of UCP are 5.8cm and 1.6, respectively, although the ranges of values are braod.[8] Although there was no evidence of this in our patient, there was a gush of liquor on amniotomy at 6 cm cervical dilatation. There was no evidence of other risk factors.

The diagnosis of UCP is by inspection of an already prolapsed cord beyond the vaginal introitus, digital palpation and having a high index of suspicion based on the fetal presentation and cardiotogographic findings when available. Moderate to severe variable fetal heart decelarations and/ bradycardia that are relieved by lying on the side are highly suggestive of occult cord prolapse/presentation and calls for continuous cardiotogographic monitoring, pelvic examination or urgent Doppler ultrasonography.[2,3] The management of UCP depends on whether there is fetal viability, associated obstetric complications, degree of cervical dilatation and station of the fetal head among other factors. However, the mode of delivery of choice when the fetus is alive is emergency caesarean section unless the cervix is fully dilated and the presenting part is at or below the ischial spines so that the baby is delivered by assisted vacuum delivery.[1,3] When the baby is alive and preparation for emergency cesarean section are underway, measures to prevent or to minimize complications of UCP should be instituted. These include postural treatment in the tiring and inelegant knee chest position or high Trendelenberg position or exaggerated Sim's position with placement of pillows below he hips. The cord should be kept in vagina and possibly protected by displacing the presenting part away keeping it between two gloved fingers. In our case the foot of the bed was raised thereby putting the parturient in Trendelberg position and the hand was retained in the vagina to prevent further prolapse and to prevent the fetal head from compressing the prolapsed cord.

A protruding overtly prolapsed cord must be placed into the vagina to minimize vasospasm and desiccation but where this is not possible warm packs are gently wrapped around it. Other methods which have been used to displace the fetal head from compressing the cord include rapid filling of the urinary bladder with 500 to 700 mls fo normal saline and concomitant intravenous administration of tocolytics such salbutamol or ritodrine and funic (cord) reduction by lifting the fetal head from the vagina and digitally elevating the cord above the widest part of the vertex so as to place in the nuchal area. The patient should be put on oxygen. These measure are important especially if delay in delivery is inevitable as when the patient is to be transferred to another center for delivery. [3,9]

The complications due to UCP are maternal and fetal. The perinatal morbidity and mortality rates are high and depend on the degree and duration of cord compression and the resuscitation measures instituted. Complete cord compression in the development of profound metabolic acidosis in 10 to 20 minutes. The perinatal mortality due to overt UCP in the USA is about 20%, but rates of 30-50% have been documented.[1,2,3,10] When delivery is achieved within 30 minutes of diagnosis, however, the mortality is reduced to 10%.[1,10] Maternal complications are those related to general anaesthesia, haemorrhage and sepsis after cesarean section or operative vaginal delivery. Our patient received correct resuscitative measures and luckily delivery was achieved in about 20 minutes hence the good maternal and fetal outcome.

Knowledge of preventive measures particularly in developing countries, Kenya included, is crucial. These include anticipation of UCP in patients at risk. Amniotomy after ascertaining there is no cord presentation should be performed with controlled release of liquor by slight pressure of the presenting part onto the pelvic brim. After the amniotomy palpating the infeior pole of the presenting part does careful search of the cord. The fetal heart rate should be checked after any amniotomy including spontaneous ones. Where facilities allow, cord tracing by Doppler ultrasonography, should be done incase of variable deceleration of fetal heart tones before amniotomy.[1]

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OBSTETRIC CASE NUMBER 11

GRADE IV CARDIAC DISEASE IN PREGNANCY AT 14+ WEEKS, PRETERM LABOUR AT 35 WEEKS: GOOD OUTCOME.

Name	: V.M.M	DOA	:	25/11/2003
Age	: 32 years	DOD	:	14/05/2004
Parity	: 1+1	LMP	:	13/08/2003
File No	: 0859736	EDD	:	20/05/2004
		GBD	:	14 weeks + 5 days

Presenting complaint

VMM was a known cardiac patient who was admitted through the Kenyatta National Hospital (KNH) labor ward with complaints of worsening shortness of breath, coughing, bilateral leg swelling, palpitation and chest pain for 3 months.

History of presenting complaint

She had been well until 5 years ago (in 1998) when she developed progressive dyspnoea on exertion. Initially she could get shortness of breath and palpitations only when she had fetched water from the river about 200 meters away from their house or if she walked uphill too fast. This continued for 4 years. In January 2003, she developed chest pain, persistent palpitations and paroxysmal nocturnal dyspnoea. By February 2003 she had orthopnea and coughed a lot at night. She sought medical assistance at Makindu health center where she treated for pneumonia severally without improvement. She was finally referred to KNH in August 2003. She was seen in the cardiology clinic where clinical evaluation, a chest X-ray, an electrocardiogram and an echocardiogram indicated that she was in congestive cardiac failure secondary to moderate mitral stenosis and mild mitral regurgitation. There was no atrial fibrillation. No clinical or laboratory signs of infective endocarditis were noted.

She was started on 0.125 mg of digoxin and 40mg of frusemide daily. She was to receive monthly intramuscular 2.4 mega units of benzathine penicillin in the nearest health facility and to avoid exertion beyond her comfort. Any sore throat was to be treated with amoxycillin or any other appropriate antibiotic after seeing a doctor. She had dramatic improvement thereafter. However, 6 weeks after the improvement of her symptoms, the symptoms began to get worse despite intake of the medications that had hitherto relieved her symptoms. She came to KNH cardiology clinic and the dosages of frusemide and digoxin were

doubled. In addition she was to take a banana every day. She was asked to attend the clinic in a month's time. When she turned up on 25/11/2003, she was pregnant in congestive cardiac failure and was subsequently admitted through labour ward.

Obstetric and Gynaecologic History

VMM was para 1 + 1 with 1 living child. She had a spontaneous abortion in 1994 at about 8 to 10 weeks and uterine evacuation was done at Masaba Hospital. Post abortion period was uneventful. Her second pregnancy reached full term. She had spontaneous vertex delivery in 1996 in a local health facility to a live female infant who cried immediately and weight 3000 grams. Pre-, intra- and postpartum periods were uneventful. She denied history of any symptoms suggestive of heart disease in pregnancy. She had never used any contraceptive method and neither had she done any Pap smear cytology test. Her menarche and coitarche were at 14 years and 15 years respectively. She had regular menses for 3-5 days every 26-28 days without any menstrual disorder.

Past Medical History

She had been admitted to Mtito Andei Sub-district Hospital in 1987 and 1992 for management of malaria. She gave a positive history of recurrent sore throat in childhood that was not serious enough to warrant any medical attention. She denied history of any feature of rheumatic fever or skin infection save for the usual childhood bruises. She had no known allergy to any substance.

Family and social history

She was a single lady who lived with her mother in Mtito Andei, Taita Taveta District. She had completed secondary education and initially worked in various Nairobi estates as house help. Later she did her own business in Mtito Andei. She did not consume alcohol or smoke tobacco. There was no history of familial heart disease or any other medical disorder.

PHYSICAL EXAMINATION

General examination

She was sick looking on a propped up bed at about 45°. Moderate bilateral pitting pedal and mild sacral edema was easily noticeable. She had no pallor, cyanosis, facial puffiness, finger clubbing, lymphadenopathy, Osler's nodes, Chenewey's lesions or splinter haemorrhages. Her pulse was regular, of good volume and rate of 100 beats per minute. The blood pressure was 92/47 mmHg. Her respiratory rate was 26/minute and the temperature was 36.7 °C.

Cardiovascular system

She had a regular pulse of good volume, non-collapsing and without radio-femoral delay. The jugular venous pressure was elevated but there was no Corrigan's sign. The praecordium was hyperactive and the apex beat was in the 6th intercostals space just lateral to the mid-clavicular line. The heart rate was 100 /minute. The 1st and 2nd heart sounds were heard. An apical grade 3/6 pansystolic murmur was heard associated with minimal radiation to the back. A mid-diastolic grade1/6 murmur was also heard in left lower sternal border.

Respiratory system

The upper lung zones were clear bilaterally except the posterior lower fields that had fine crepitations bilaterally. There was no other anomaly.

Abdominal examination

The abdomen was uniformly distended in the lower aspect and moved with respiration. The fundal height corresponded to 14 weeks. There was tender smooth-surfaced hepatomegally of up to 4 cm below the right costal margin. No splenomegally was noted.

Musculoskeletal system

She had no skeletal deformity (such as kyphoscoliosis) that could predispose to or precipitate a cardiac disease.

The nervous system was essentially normal except that she was slightly anxious.

Impression

Cardiac disease grade IV (CCF secondary to rheumatic heart disease) in pregnancy at 14 weeks gestation. Sub-acute bacterial endocarditis was to be ruled out.

MANAGEMENT

She was put on bed rest on a cardiac bed propped up at an angle of about 30°. Oxygen was placed by the bedside in case the need for it arose. Intravenous frusemide at a dose of 80 mg 12 hourly and daily oral 5mg of enalapril, 50 mg of atenolol and 0.25 mg of digoxin were administered. In addition 50 mg of aldactone twice a day was administered. Haematinics were deferred subject to exclusion of endocarditis. In

view of the bed rest and possible deep venous thrombosis (DVT) and the mitral stenosis that could predispose to atrial fibrillation, clot formation and subsequent embolization, prophylactic oral 5 mg of warfarin was given to her daily. Investigations done to aid in the management included those on the table below and the next enlisted ones.

I.

DATES	26/11/03	15/12/03	21/01/04	11/02/04	19/03/04	30/03/03	19/04/04
TESTS	**Antenatal period**						Postpart.
Haemogram							
Hb (g/dl)	11.5	11.7	11.6	11.4	12.0	11.1	11.4
Wbcc (x 109)	6.5	6.3	7.6	10.1	7.4	8.0	6.07
Neuts (%)	65.9	69.0	70.0	68	72.0	71.0	66.0
Lymphs (%)	32.0	35.0	29.0	31	26.0	27.0	32.0
Platlets (x 10^9)	265	240	265	300	310	290	330
ESR (mm/hr)	44						
RFTs							
BUN (mmol/L)	4.8	2.1	3.6	2.1	3.4	Not	4.3
Cr (μmol/L)	93	44	60	84	56	Done	80
K+ (mmol/L)	3.5	3.7	3.8	3.4	N/D		4.0
Na+ (mmol/L)	134	130	138	135	N/D		140
Urine	**9/12/03**			**20/2/04**	**17/3/04**		
Analysis	NAD	NAD	NAD	NAD	NAD	NAD	Not
Culture	No gro	No gro	No gro	S.epid.	N/D	No gro	Done
Sensitivity	N/A	N/A	N/A	Nitrfn	N/D	N/A	
LFTs	----	NAD	----	----	----	----	----

KEY: 1. NAD: No abnormality detected

2. N/A: Not applicable

3. S. epid.: Staphyloccocus epidermidis

4. N/D: Not done

5. No gro.: No growth obtained.

II. **Echocardiography**: 1. Upon admission: Moderate to severe mitral stenosis, mild mitral and tricuspid regurgitation, mild pulmonary hypertension and no vegetations.

2. 17/01/04: As in the first echocardiogram.

3. 24/02/04: As in the first echocardiogram.

III. **Chest X-rays** : 1.Cardiomegally, double cardiac shadow (suggestive of mital stenosis), pulmonary plethora with reticular interstitial infiltrates

2. Marginal cardiomegally, increased bronchovascular markings, sharp costophrenic angles. Conclusion: ? pneumonitis.

IV. **Coagulation profile**: 15/12/03: Prothrombin time test: 29.9 seconds
Prothrombin time test control: 12.3 seconds
Prothrombin time index: 41%
INR (International normalized ratio): 2.92: WARFARIN REDUCED.
4/02/04: INR: 3.3 : WARFARIN DISCONTINUED.
Haemogram: Normal in both occassions.

V. **Electrocardiogram**: Upon admission: Sinus rhythm,pulse rate 96/minute, no atrial fibrillation, mild P mitrale and the rest normal.

VI. **Blood culture**: Done upon admission: No growth obtained.

VI: **antenatal profile**: 1. Haemoglobin: 11.5%

2. HIV ELISA: Negative

3. VDRL: Negative

4. Blod group: O +ve

VII. **Obstetric ultrasound scan**: Single viable intrauterine fetus in variable lie at an average ultrasound age of 14 weeks + 3 days. Placenta: fundo-posterior, adequate liquor.

While waiting for the above results she received intravenous 2 mega units of crystalline penicillin and 80 mg of gentamicin as treatment for possible endocarditis. Salt and spicy food was restricted. She improved remarkably so that 1 week later when a cardiologist reviewed her, the pulse rate was 84/minute, the BP was 110/70 mmHg and had no gallop rhythm, the chest was clear and the findings on the murmurs were the same. She was no longer in congestive cardiac failure. Haematinics (Ranferon) were started and frusemide (Lasix) was converted to oral 40 mg twice a day while she continued with the rest of the other drugs

prescribed earlier. Two comprehensive counseling sessions regarding her ailment and the management were held. The necessity of continuous monitoring in the hospital was emphasized. Clinically she remained out of CCF for the rest of the stay in the ward. The doctors and the nurses reviewed her every day. The uterine fundal height persistently corresponded to the gestation by the dates and ultrasound extrapolation.

As shown in the table above serial haemogram, urine for urinalysis culture and sensitivity, echocardiograms and renal function tests were generally satisfactory throughout the pregnancy. However on 20/02/04, she complained of lower abdominal pains associated with terminal dysuria and though urine analysis was normal the urine culture grew 10^5 colonies per ml of Staphyloccocus aureas sensitive to nitrofurantoin, which she received. A repeat urine analysis and culture a week later was without any abnormality. The symptoms had resolved. At 32 and 34 weeks gestation she complained of headache and general weakness and on both occasions tests for malaria were negative.

On 17/04/04, at 35 + weeks, she went into preterm labour while in the antenatal ward. She was quickly rushed to labor ward where she was monitored in the acute room. Re-evaluation at 10.30 a.m. indicated that she was in good general condition with a BP of 110/70 mmHg, a PR of 84/minute, a RR of 20/minute and the temperature was 36.6 °C. Abdominal examination showed a uterine fundal height corresponding to 34 weeks and a fetus in longitudinal lie, cephalic presentation and with a descent of 4/5. Regular fetal heart tones were heard. Three uterine contractions occurred every 10 minutes each lasting 25 to 35 seconds. Aseptic vaginal examination revealed normal external genitalia, healthy vaginal mucosa and a cervix that was 0.5 cm long, central and 4 cm dilated. The membranes were intact and flat. The presenting part was well applied to the cervix and the pelvis felt adequate. Amniotomy was not done. As she was being examined, the vacuum extraction set, the resuscitation and the emergency tray equipment and drugs were being assembled and tested for use when the need arose.

She was nursed in the left lateral and propped up position and ½ hourly strict partogramming performed by a midwife who stayed in the room throughout the labor period. The anesthesiologist in the intensive care unit (ICU), the senior registrar and the cardiologist on call were all informed of the patient's condition and labor. Two intravenous access lines were fixed (in case one came out during labour) and intravenous crystalline penicillin and gentamicin, 100mg of intramuscular tramadol and oxygen by mask were administered intrapartum. She progressed very fast without any signs of decompensation. At 1.30 p.m. she had spontaneous rupture of the membranes. Examination then indicated that the descent was 2/5, the cervix was fully effaced and 8 cm dilated. There was no palpable cord, caput succedaneum or moulding. At 2 p.m. she reported the urge to bear down. Repeat pelvic examination indicated that she was in 2nd stage of labour.

Bearing down was discouraged. Eighty milligrams of intravenous lasix was administered then she was transferred to the delivery room where she was put in semi-Fowler's and semi-lithotomy position, vulvovaginal toilet and draping done. A medio-lateral episiotomy was made and with one pull of vacuum extraction, delivered a live female infant with a birth weight of 2200 grams and an Apgar score of 7, 9 and 10 at 1, 5 and 10 minutes respectively. The placenta was delivered by controlled cord traction. There was no active bleeding. Five units of oxytocin were given by the intramuscular and the episiotomy repaired under local anaesthesia after inspection of the entire genital tract revealed no lacerations and no active uterine bleeding. The uterus was massaged after emptying the bladder aseptically. The uterus was then well contracted.

Postpartum Care

She remained in the labour ward acute room under close monitoring of the vital signs, any vaginal bleeding and any signs of cardiac decompensation Intravenous antibiotics and lasix and oxygen when needed were administered while propped on bed. On the first and second postnatal days, she was quite well and was transferred to the postnatal ward where she had been before onset of labour. However, on the third day postpartum, she developed marked palpitations, chest pain and persistent dyspnoea even on ordinary activity. The dose of lasix was increased and the patient stabilized. As shown in the table above, a haemogram and renal function tests done on the 2nd postnatal day (19/14/04) was essentially normal. The Hb was 11.4 g/dl. By the 4th postnatal day she had class II cardiac disease symptoms. Oral lasix was introduced and the intravenous one discontinued. She took coamoxi-clavulanic acid (Augmentin) antibiotic for a week and continued with oral lasix, enalapril, atenolol, digoxin and haematinics up to the time she was discharged 2 weeks postpartum. Advice on contraception, breastfeeding, prevention of recurrent rheumatic fever and the need to adhere to drug dosages as prescribed was provided. She opted for interval bilateral tubal ligation (BTL). In the meantime she was to use a condom or progesterone-only pill if ever she had coitus.

Follow up

She was seen in the cardiac and the postnatal clinics 2 weeks later. She was in good general condition with class II cardiac disease symptoms. The uterus was well involuted and breastfeeding was established. The dosages of lasix, enalapril and atenolol were halved and she continued with the rest of the drugs. She was to have BTL performed by a private practioner at 8 weeks postpartum.

DISCUSSION

Our patient was para 1 + 1 gravida 3 at 14 weeks gestation when she was admitted in congestive cardiac failure. She was managed in the ward until gestational age of 35 weeks when she had uneventful assisted vaginal delivery with a good outcome.

Cardiac disease in pregnancy is one of the biggest obstetric challenges because of the associated increased maternal and fetal morbidity and mortality that occurs in 0.4-4% of pregnancies worldwide.[1] However, the incidence varies from region to region. In the USA, cardiac disease complicates about 1% of pregnancies and is the third leading cause of death in 24-44 year old women.[2] In the United Kingdom, from 1994 through 1996 cardiac diseases accounted for 38% of maternal deaths.[3] At Kenyatta National Hospital, it was reported to be 0.5% in 1969[4] and 0.6% in 1982.[5] In developed countries, the overall incidence is less than 1%.[6,7,8] In both Kenya and Tanzania, majority of the patients were found to be in the age group of 20 – 24 years.[5, 9]

Aetiological causes of cardiac disease vary from region to region. In the developing countries such as Kenya, rheumatic heart disease with valvular lesion(s) is the most common cause of cardiac diseases in pregnancy. Ngotho in his 1982 tudy at KNH [5], found rheumatic heart disease to be responsible for 86.4% of cardiac disease in pregnancy. In the developed countries, the incidence of rheumatic heart disease has continued to decline and that of congenital heart disease has become relatively more important.[6, 7] In the west congenial heart lesions now constitute at least half of all cases of heart diseases encountered during pregnancy, while rheumatic heart disease has almost disappeared.[8] Other types of heart diseases that may complicate pregnancy include corpulmonale, constrictive pericarditis, various forms of heart block, cardiomyopathy and isolated myocarditis. Others include hypertension, coronary, thyroid, syphyllitic and kyphoscoliotic heart diseases.[1, 6,7]

The dominant lesion in rheumatic heart disease has been reported to be mitral stenosis.[1, 7] In a study in KNH, Bhatt[9] in 1978 found that majority of the study subjects had combined mitral senosis and regurgitation, while in Tanzania Spense and Makena[10] in 1972 found mitral regurgitation to be the commonest lesion. Our patient had predominantly mitral stenosis with mild mitral, tricuspid and aortic regurgitation secondary to rheumatic heart disease.

Pregnancy related cardiovascular physiological changes that tend to worsen or unmask cardiac disease include increase in cardiac output by almost 30-50% and the total blood volume by 50% above non-pregnant level by 32 weeks.[1, 7] During labour, cardiac output increases by a further 34% in 1st stage and even further in 2nd stage due to increase in stroke volume and heart rate. There is also a steady increase in

blood pressure.[7] Clinical indicators of heart disease include; symptoms of progressive dyspnoea or orthopnoea, nocturnal cough, haemoptysis, syncope and chest pain related to exertion or emotions. Sign are cyanosis, finger clubbing, persistent neck vein distention, systolic murmur grade 3/6 or greate diastolic murmur, sustained arrhythmia, persistent split second heart sound and pulmonary hypertension. Physiological changes of normal pregnancy that may make diagnosis of heart disease difficult ar functional systolic murmur, oedema in lower extremities and accentuated respiratory effort suggestin dyspnoea.[1, 7]

The New York Heart Association (NYHA) grading system first published in 1928 and was revised for th eighth time in 1979, is widely used to grade the severity of heart disease. It is based on past and presen disability and is uninfluenced by physical signs, thus: [1,7]

Grade I: Uncompromised: Patients with cardiac disease but no limitation of physical activity.

Grade II: Slightly compromised: Patients with cardiac disease and slight limitation to ordinar physical activity but no symptoms at rest.

Grade III: Markedly compromised: Patients with cardiac disease and marked limitation of physic activity. They are comfortable at rest but have dyspnoea on mild physicial activity.

Grade IV: Severe compromised: Patients with cardiac disease and inability to perform any activi without discomfort. They have dsypnoea at rest.

Patients with pure mitral stenosis and those who have had previous congestive cardi failure or cardiac surgery are classified as Grade IV.

The American College of Obstetricians and Gynaecologists has further classified the various heart diseas into 3 Groups according to the risks of mortality during pregnancy. [11]. These are:

Group 1: Minimal Risk – Mortality <1%:

These include atrial septal defect, ventricular septal defect, patent ductus arteriosus, corrected tetralogy of Fallot, bioprosthetic valve, pulmonary or tricuspid disease and mild mitral stenosis (NYHA disease I and II).

Group 2: Moderate Risk Mortality 5-15%. This is further divided into 2A and 2B.

2A: - Symptomatic mitral stenosis (NYHA) Classes III and IV), aortic stenosis, aortic coarctation and artificial valve. without valvular involvement, uncorrected tetralogy of Fallot, previous myocardial infarction, Marfan's syndrome with normal aorta.

2B - Mitral stenosis with artrial fibrillation

Group 3: High risk – Mortality 25 – 50%

These include pulmonary hypertension, aortic coactation with valvular involvement, Marfan's syndrome with aortic involvement.

The patient presented had features of congestive heart failure evidenced by a gallop rhythm of the heart sounds, hepatomegally, cardiomegally, pulmonary edema and bilateral pitting pedal oedema secondary to symptomatic mitral stenosis. She was, therefore, NYHA grade IV and had moderate (5-15%) risk of maternal mortality.

The diagnosis of heart disease in pregnancy is safely aided by electrocardiography, echocardiography and chest radiography. However, cardiovascular changes in normal pregnancy cause a challenge in interpretation. Due to the elevation of the diaghragm with advancing pregnancy, there is a 15° left axis deviation seen in the ECG, with mild ST changes in the inferior lead. Pregnancy does not alter voltage findings.[7] The heart silhouette is normaly larger in pregnancy in the chest x-ray. However, gross cardiomegally can be suggestive. A lead apron to shield foetal radiation exposure must be used.[7] Some normal pregnancy induced changes in echocardiography include tricuspid regurgitation, increased left atrial size increase in left ventricular outflow cross sectional area.[7]

A combined team of a cardiologist and obstetrician best manages patients with cardiac disease in pregnancy. Where pre-existing cardiac disease is known, management should commence before the woman conceives. Assessment of the disease and the likely maternal and foetal risk should be discussed with her. Patients with high risks should be advised to terminate pregnancy in 1st trimester if possible. During the antenatal period, time should be taken to advice her on ways of avoiding aggravation of her

cardiac condition. Factors aggravating pre-existing disease include anaemia, infections (especially in the urinary tract), hypertension, anxiety, arrhythmia, thromboembolic disease and rigorous physical activity.[1,6,7] Patients should be advised to avoid added salt intake and contact with any persons with respiratory infection and if possible, to get vaccination against pneumococcal and influenza infections.[7] Those who smoke cigarettes should stop because of its effect on the heart and its propensity to cause respiratory infection.[7] Patients with grade I and II cardiac disease are seen in the antenatal clinic weekly and assessed by both the obstetrician and cardiologist during every visit. They are to be admitted at 35 weeks to await spontaneous onset of labour. Those with grade III and IV disease, as was our patient, are hospitalized for the duration of the pregnancy and have to be routinely assessed by the cardiologist

Vaginal delivery is the preferred method of delivery. Caesarian section is limited to obstetric indications.[1,6,7] During labour, the patient should be propped up into the most comfortable position. The pulse and respiratory rates should be taken after every 15 minutes during second stage.[1,7] The temperature should be recorded two hourly and urine output recorded hourly as a decrease may be an early sign of cardiac failure.[1,7] Morphine should be given to allay anxiety and pain though epidural analgesia is recommended for most situations (helps reduce cardiac output by reducing pre-load). The major danger is maternal hypotension especially in intra-cardiac shunt or aortic stenosis because ventricular output is dependent on adequate pre-load.[7] Oxygen is also given to ensure optimal saturation of blood and also to prevent decompensation.[7] Intravenous fluids should be carefully monitored to avoid fluid overload and pulmonary oedema. American College of Obstetricians and Gynaecologist recommend pulmonary artery catheterization for continuous haemodynamic monitoring.[11] This is not feasible in our set-up. However,intravenous fluid is restricted. Second stage of labour should be shortened by elective assisted delivery to minimize dramatic increase in blood pressure. The patient should not be encouraged to bear down forcibly and the episiotomy is made once the head is on the perineum.[4] In our set up, elective vacuum extraction is carried out as was done for our patient.

The most dangerous time for the development congestive cardiac failure or pulmonary oedema is immediately after delivery.[1,7] Oxytocin is preferred to ergometrine in prevention of postpartum haemorrhage as ergometrine causes sudden peripheral vasospasm and hypertension associated with sudden intravascular overload.[1,7] A bolus of intravenous frusemide immediately after delivery may offset the haemodynamic changes of sudden mobilization of extravascular fluid and release of blood from the lower extremities as the gravid uterus is emptied and venacaval compression released. Our patient was given both oxytocin and frusemide. Postpartum period is critical and patients should be monitored closely for

congestive heart failure, infective endocarditis and thromboembolic disease. Early rising and early ambulation are encouraged. If there is no evidence of cardiac embarrassment during labour, delivery and early peurperium, breastfeeding is usually not contraindicated, as was the case with our patient.

Eighty five percent of the 0.5% maternal mortality rate in the USA, occur in patients with grade III and IV disease.[1,5] Other complications of cardiac disease in pregnancy include; premature labour and delivery, low weight babies and high incidence of congenial heart diseases.[1,7] Post-partum counseling on contraception is important and surgical sterilization is the preferred method of choice.[1,7] Where the husband consents, vasectomy offers a permanent but less risky mode of contraception. If tubal ligation is done, mini-laparotomy under local anaesthesia six weeks postpartum is preferred. Other methods than can be used include barrier methods (condom) or progesterone only contraception. Intrauterine devices are not frequently used due to high frequency of infection and predisposition to bacterial endocarditis.[1,6,7] Estrogen based contraception should be avoided in cases of mitral valve diseases and those with mechanical valves where risks of thrombosis and embolism are high. Our patient was to undergo interval BTL under local anesthesia.

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OBSTETRIC CASE NUMBER 12

TWIN GESTATION IN A PRIMIGAVIDA IN LABOUR - EMERGENCY CESASERIAN SECTION, LIVE BABIES.

Name : J.N.K	DOA	:	16/09/2004
Age : 21 years	DOD	:	19/09/2004
Parity : 0+0 Gravida one	LMP	:	13/12/2003
File No : 0983627	EDD	:	20/09/2004
	GBD	:	39 weeks + 3 days.

Presenting Complaint

JNK presented to the Kenyatta National Hospital with labor pains for 8 hours.

History of presenting complaint

She had been well until she developed intermittent painful uterine contractions that progressively increased in intensity and frequency. There was no associated drainage of liquor, change in bowel or urinary habits but show had been noted. Fetal movements had been unaltered prior to the onset of the contractions.

Antenatal care

Her antenatal care had been at the Riruta Health Center from 20 weeks gestation. An obstetric ultrasound done then showed that she had a twin gestation. Both twins were without any anomaly and appeared binovular. Her antenatal profile was Hb 11.3g/dl, blood group; O +ve and HIV and VDRL tests were negative. Her prenatal period was uneventful save for the pressure symptoms she experienced during her late 3rd trimester. Her blood pressure remained within normal limits and her weight gain was 11.3 kg. In view of the twin gestation she was to undergo elective cesarean section at 38 completed weeks and was to report to a hospital with a functional operative theatre as soon as she developed labor pains. She had initially opted to undergo the cesarean section at Pumwani Maternity Hospital.

Past obstetric history

She was a primigravida whose menarche was at 15 years. Her menses flowed for 3-4 days evry 28 to 30 days without any menstrual disorder. She had uneventfully used the combined oral contraceptive pills for 2 years before she stopped to conceive. She had not done any Pap smear test and denied any history of sexually transmitted infections.

Past medical history was not significant.

Family and social history

She was a married housewife who had only completed primary education, took no alcohol, smoked no tobacco and had no history of familial disease or multiple gestations. She lived with her husband in Riruta in Nairobi. Her husband was a security guard with one of the security firms in Nairobi.

PHYSICAL EXAMINATION

General examination

JNK was in good general condition. She had no pallor, pedal edema, jaundice or lymphadenopathy. Her height was 155 cm. Her BP was 120/75 mmHg, PR; 80/minute, temperature 36.7°C and RR was 18/minute.

Abdominal examination

The abdomen was uniformly distended and the uterine fundal height was term. The first twin was in longitudinal lie, breech presentation and had regular fetal heart tones at 144 /minute. It was difficult to delineate the presentation of the 2nd twin whose fetal heart tones were regular at 136/minute. The descent of the breech of the first twin was 3/5 and the uterine contractions were 3 in 10 minutes each lasting 25 seconds.

Pelvic examination

She had normal external genitalia and healthy vaginal mucosa. The cervix was 5cm dilated, central and 80% effaced. The membranes were mildly bulging and the presenting breech was frank. The pelvis felt adequate and there was show on the examining finger. No umbilical cord was palpable.

Diagnosis

Twin gestation at term in a primigravida in active labor with the first twin in frank breech presentation

Management

She was admitted to the labor ward and preparation for emergency cesarean section (c/s) instituted. Her pubic hair was shaved, informed consent obtained, blood taken for grouping and cross match, theatre team alerted and she was not to eat or drink anything before the operation. Intramuscular atropine was administered before she was wheeled to theatre. While being catheterized, she had spontaneous rupture of the membranes with subsequent prolapsed umbilical cord. Quick abdominal toilet and opening via a sub-umbilical midline incision and lower uterine segment cesarean section as described under introduction, led to the delivery of the twins. The first twin was a live female infant delivered by breech extraction with a

birth weight of 2800 grams and an Apgar score of 6, 9 and 10 at 1, 5 and 10 minutes respectively. The second twin was a live male delivered by cephalic extraction with a birth of 2300 grams and an Apgar score of 7, 9 and 10 at 1, 5 and 10 minutes respectively. They had distinct placentas, cords, chorions and amnions. The senior house officer in the Pediatrics Department reviewed both twins. They had no anomaly and were to join the mother as soon as she was able to take care of them. The rest of the c/s was as per the description under introduction. The estimated blood loss was 500 mililiters.

Postoperative care

Her recovery from both the general anesthesia and the surgery was good. She received intravenous crystalin penicillin and gentamicin and intramuscular pethidine for 24 hours. Ambulation and oral sips were started 8 hours after surgery followed by light diet 6 hours later. On the 2nd post-operative day she and her infants were doing well. She was mildly pale with normal vital signs. She was commenced on oral antibiotics, analgesics haematinics and breast-feeding advice given. The breast-feeding advice included sitting on a firm sit with back support while breastfeeding, taking plenty of warm fluids such porridge and tea, a balanced diet based on the available and acceptable foodstuffs locally, proper breast feeding techniques and breast care. The lach-on technique whereby the entire nipple and most of the areola (and not just the areola) are placed in the baby's mouth was demonstrated. On the 3rd day she was in good general condition. Lactation had been established, the uterine size corresponded to 18 weeks' gestation, the wound was clean and dry, lochia was scanty rubra and the calves were soft and non-tender. A check Hb done then was 9.8 g/dl. She was discharged home with the oral medicines and advice to attend the postnatal clinic in 6 weeks or earlier if there was a problem. She was also advised on contraception and asked to discuss the same with her spouse.

Follow-up

Her general condition in the 6th postnatal week was excellent. She was no longer pale. The breasts were highly active without anomaly, the uterus was completely involuted and pelvic examination revealed no anomaly. She had not had menses and denied any sexual activity since her delivery. Further advice on contraception was provided and she opted for the intra-uterine contraceptive device (IUCD). A pap smear was done and she was referred to the Family Welfare Clinic for insertion of the IUCD and subsequent follow-up.

DISCUSSION

The patient presented was a 21 year- old primigravida who presented in active labour at term (39+ weeks) with twin gestation diagnosed clinically and by ultrasound. She was delivered by caesarian section to two live infants who, together with their mother, did well in the puerperium.

Multiple or multifetal pregnancy is the occurrence of two or more embryos/fetuses in one pregnancy. Twin-gestation, therefore is the presence of two fetuses in one pregnancy. Twins occur in 1 of 100 pregnancies of white women, 1 of 80 pregnancies of black women and in only 1 of 155 pregnancies in Asian women.[1] Monozygotic twining occurs in 1 in 250 births and is independent of race, heredity, age and parity. Dizygotic twinning is affected by these factors and by use of fertility drugs. In general the frequency of dizygotic twins is low in Asians, intermediate in whites and high in blacks. The Yorubas of Western Nigeria have a frequency of 45 twins per 1000 births and about 90% are dizygotic twins.[2] Oyieke[3] found a high twinning rate at Kenyatta National Hospital (KNH) of one in 59 births (1.7%). In a later study Mutungi[4] found an incidence of 2.2% of all deliveries at Kenyatta National Hospital and Pumwani Maternity Hospital.

Worldwide, the incidence of twin and higher order multiple gestations has increased significantly over the last 15 years primarily due to the availability and increased use of ovulation inducing drugs and assisted reproductive technology (ART). In the USA, multiple gestations now comprise 3% of all pregnancies and twins comprise 25 – 30% of deliveries resulting from ART.[5] Monozygotic twins result from a single fertilized ovum and comprise slightly more than 30% of all twins. Dizygotic twinning is the result of fertilization of two separate ova. The two ova are released from separate follicles or rarely from the same follicle at approximately the same time. They comprise nearly 70% of all twins. The timing of monozygotic division has important implications. Division within the first 72 hours after conception results in a diamniotic, dichorionic monozygotic twin pregnancy. This occurs in 30% of twins with a mortality rate of 9%.[1] Division 4-8 days after fertilization results in a diamniotic, monochorionic twin pregnancy. It is the most frequent of the monozygotic twining,about 68%, with mortality as high as 35% due to complications of vascular anastomoses within the placenta.[1] Division during days 8-13 results in monoamniotic, monochronomic twins. These monozygotic twins occur least often (2%) but with a high mortality rate of up to 50%. Division at two weeks of fertilization after the amniotic sac and embryonic disk have been formed results in conjoined twins.[1] Our patient had dizygotic twins in view of the different sexes and, less importantly, different amniotic sacs and separate placentas. Monozygotic twins without chromosomal anomalies are always of the same sex and usually (but not always) share the same physical

characteristics; same genetic features and could be mirror images of one another. However, because monozygotic twins result from 'teratogenic' division of one ovum, the incidence of chromosomal and, therefore, phenotypic inequalities in monozygotic twins is higher. They are therefore less identical than the dizygotic twins and their fingerprints differ[5]. Chromosomal anomalies can result in heterokaryotypic monozygotes for instance; where one twin has Downs Syndrome and the other twin normal.

The cause of monozygotic twinning is unclear. However, conventional ovulation induction methods[6] and use of other ART[7] methods have resulted in an increase in monozygotic twinning. In addition, evidence suggests that delayed transport through the tube increases the risk twinning. Progestational; agents and the combined contraceptives were shown by Bressers and associates (1987)[8] to increase twinning in pregnancies conceived in close proximity with contraceptive use. Dizygotic (fraternal) twins maybe of the same or different gender. About 75% of dizygotic twins are of the same sex with both twins being males 45% and both twins female 30%.[3] Many factors influence dizygotic twinning. Race is a factor with most dizygotic twinning occurring in blacks, than whites and least common in Asians. Dizygotic multiple pregnancy tends to be recurrent and women who have borne dizygotic twins have a 10-fold increased chance of subsequent multiple pregnancy. Age also increases the rate of twinning with advancing age of over 35 years having an increased risk. Parity doesn't influence the incidence of dizygotic twinning.[5] Women of increased height and weights have a higher incidence twinning. Dizygotic twinning is also more common among women who become pregnant soon after cessation of long-term oral contraception. The use of ART and ovulation induction methods increases multiple pregnancies with higher order pregnancies being more common. Besides the black race and the use of combined oral contraceptives, our patient had no other risk to twinning.

Early diagnosis of multiple gestations in the ante partum period is important for specialized care. Undiagnosed multiple gestation presents special problems of management and ultimately contributes significantly to higher perinatal and maternal morbidity. Oyieke[3] reported that in 38% of twins seen at Kenyatta National Hospital, the diagnosis was made either in labour or after delivery of the first baby. Multiple gestation should be suspected whenever the uterus seems larger than dates, hydramnios or unexplained maternal anaemia develops, auscultation of more than one fetal heart is suspected or when pregnancy has occurred following ovulation, induction or in-vitro fertilization. Ultrasound diagnosis is a simple, safe and effective tool in diagnosis of multiple gestations. Cetrulo[9] (1980) argued that universal ultrasound screening for all pregnant women during the second trimester would result in early diagnosis of multiple gestation with almost 100% accuracy. This has been validated by the results of the Routine

Antenatal Diagnostic Imaging with Ultrasound (RADIUS) study from which the separate gestational sacs can be identified ultrasonographically as early as six weeks from first day of last menstrual period. Its important however to visualize separate fetuses with independent cardiac activities. Ultrasound can help determine the zygotism. If fetal gender can be identified on ultrasound, twins of opposite sex are almost always dizygotic. Separate placentas and a thick separating membrane greater than 2mm between the twins also probably points towards dizygosity. In the absence of these findings, monozygosity is likely.[1,5] In our patient diagnosis of twin gestation was made by ultrasonograpy by the 20th week of gestation.

Maternal serum alfa-fetoprotein (MSAFP) screening at 14-20 weeks has been used to screen for multiple gestation. The median MSAFP level will be 2.5 times that of the medial level for singleton pregnancies at a similar gestation.[1,5] Its three times and four times higher in triplets and quadruplets respectively.[1,5] The hematocrit and haemoglobin values and red cell count are considerably reduced in direct relationship to the increased blood volume. Maternal hypochromic normocytic anaemia occurs so frequently in multifetal pregnancy that it has been suggested that all patients with the process be suspected of having a multiple gestation.[5] Glucose tolerance tests show that gestational diabetes mellitus and gestational hypoglycaemia are much higher in multiple gestation compared to singleton of the same gestational age.[10] After 32 weeks, the combined weight gain of both twins is approximately equivalent to that gained by a singleton for the remaining portion of the pregnancy.[10] The median weights of twins at birth is just over 2270 grams in the USA.[5] Our patient delivered low birth weight babies weighing 2300 and 2800 grams at 39+ weeks.

Antepartum care for twin pregnancy includes an early diagnosis of twin pregnancy, which is associated with improved perinatal outcome.[11] Ultrasound diagnosis may be used as early as the 4th week of pregnancy. The mother should make frequent antenatal visits at least every fortnightly from 20-36weeks.[1] She will need diet supplementation with additional calories at least 300 calories per day above the normal pregnancy requirements, protein intake of up to 80 grams per day and folate at least 1 milligram per day. She will require extra bed rest and the value of bed rest in hospital is controversial.[12,13,14] Preterm labour should be anticipated and recognized early and aggressively treated if it occurs. Prophylactic administration of tocolytics to these women with twin pregnancy has been tried with varying degrees of success. Since an increased incidence of maternal cardiovascular complications has been reported in women with multiple gestation treated with β-agonist, it seems prudent to restrict the use of these agents to women who are confirmed to be in latent labour.[15] Results of studies using prophylactic cervical cerclage in these women with multiple gestation has been disappointing.[16] Its now recommended that cervical cerclage placement should be restricted to women with documented cervical incompetence. Cervical assessments

score based on the length of cervical canal minus the dilatation of the internal os in centimeters,[17] ultrasonographic assessment of cervical length and fetal fibronectin have been validated as useful adjuvant tests in the prediction of pre-term labour in multiple gestation.[17]

Fetal growth should be monitored by serial ultrasound examinations to detect discordant fetal growth and intrauterine growth restriction. The serial ultrasound should include monitoring fetal well-being by use of non stress tests. Blake and others[18] confirmed that antepartum nonstress testings is a highly reliable and predictive test in assessment of multiple gestations. It is recommended that ultra sound follow-up be done four weekly during the third trimester and more frequently if intrauterine growth restrictions is encountered.[5] Pregnancy induced hypertension and pre-eclampsia, should aggressively be looked for and managed effectively to reduce both maternal and fetal morbidity. Other less common complications such as polyhydraminios, twin to win transfusion should be looked for and managed effectively. The parents should also be advised to make plans for two babies at home. Our patient did not have any of the complications associated with multiple gestation such as polyhydramnios, pregnancy induced hypertension and antepartum hemorrhage among others.

Intrapartum management of twin gestation depends on several factors, which affect the delivery outcomes. All combinations of intrapartum twin presentations can be classified into three groups. Twin A vertex, Twin B vertex – 40%, Twin A vertex, Twin B non-vertex – 40%, Twin A non-vertex, Twin B vertex or non vertex – 20%. The patient should be admitted to hospital at the first sign of labour. An ultrasound evaluation should be performed to ascertain the presentation of each fetus and is estimated fetal weight. Routine continuous fetal monitoring is recommended with facilities ready for immediate caesarian section. Generally if no other obstetric indication for caesarian section, vertex-vertex presentations should be allowed vaginal delivery.[19] In category with twin A vertex and twin B non vertex with each twin weighing over 2000 grammes, vaginal delivery can be accomplished for both. This is generally accomplished by external version of twin B immediately after delivery of twin A. Total breech extraction is an alternative to delivery of breech B.[19] If twin B weighs less than 2000 grammes and external version is unsuccessful, then caesarian section is warranted. This is in contrast of the twin B who weighs more than 2000 grammes who should undergo vaginal breach delivery.[19] Twin A non vertex with twin B having any presentation should be delivered primarily by caesarian section.[19] Locked twins may occur if twin A is breech and twin B is vertex. Our patient had first twin in breech presentation and hence the decision to deliver the mother primarily by caesarian section.

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OBSTETRIC CASE NUMBER 13

DELAYED SECOND STAGE OF LABOUR, VACUUM EXTRACTION – LIVE BABY.

Name	: C.A.M	LNMP	:	22/10/02
Age	: 32 years	EDD	:	29/07/03
File N°	: 38.40.39.	GBD	:	39 weeks + 3 days
Parity	: 0 + 0 gravida	DOA	:	25/07/2003
		DOD	:	27/07/2003

Presenting complaint

C.A.M was admitted to the Aga Khan Hospital labor ward via the antenatal clinic with a 36-hour history of reduced fetal movements at 39+ weeks gestation for induction of labor.

History of presenting complaint

She had been generally well until 36 hours prior to admission when she noticed that the fetal movements had reduced relative to the previous days. There were no associated features such as urinary symptoms, abnormal vaginal discharge, headache or blood vision or those suggestive of diabetis mellitus.

Past obstetric and gynaecologic history

She was a primigravida. She had had primary infertility for 5 years due to azoospermia found on semen analysis of her husband who declined artificial insemination by donor despite adequate counseling by the private physician attending to them. This led to their divorce 2 years prior to conception by another partner.Her menarche was at 15 years and the menstrual flow took 4 days and occurred every 26 to 30 days without any menstrual disorder. Her private physicians had done hormonal profile, pelvic ultrasound and a pap smear and all were reportedly normal.

History of the index pregnancy

She was a para 0 +0 with an LMP of 22/10/2002, an EDD of 29/7/2003 and, therefore, her gestation by dates on admission was 39 + weeks. She had her antenatal care at the Aga Khan Hospital, Nairobi, from 1[illegible] + gestation by dates. Her weight then was 62.2 kg and was 164 cm tall. The antenatal profile done then was: Hb; 11.9 g/dl, blood group B +ve and VDRL and HIV serological tests were negative. The uterine fundal height and the average ultrasonographic age corresponded with the dates then and on the subsequent follow up. She was admitted twice during her antenatal period. The first was at 18 weeks when she was treated for acute severe pharyngitis and anxiety with Augmentin® and Lexotanil® respectively. She was discharged 2 days later in good general condition. Her second admission was at 30 weeks for reactive

depression. Counseling and oral paroxetine for 1 week resolved her problem. She attributed the psychological problems to lack of psychosocial and financial support by her partner. She was discharged after 3 days. The obstetric care both as an out – and in-patient was uneventful save for mild placental calcification that was noted on ultrasound at 36 weeks.

Past medical history

She had been treated three times for reactive depression with serotonin specific inhibitors prior to conception. This was soon after she divorced from her husband. Her new partner had, not only failed to marry her, but he also provided no psychosocial and financial support as he had promised he would if she conceived. She was not allergic to any substance and had no other significant medical history.

Family and social history

She was a divorced, college graduate who worked with the National Bank of Kenya in Nairobi as a teller. She did not smoke any substance nor drink alcohol. There was no history of any psychiatric illness or any chronic disease in the family. Her new partner worked with in the same institution and was married with two children. She lived in Eastland's alone.

General examination

CAM was in good general, nutritional and hydrational status. She had no pallor, pedal edema, lymphadenopathy or jaundice. Her vital signs were temperature of 36.5oC, BP of 120 80 mmHg. RR of 18/minute and a PR of 80 per minute that was regular with a good volume.

Abdominal examination

The abomen was uniformly distended and no surgical or therapeutic marks were noted. Palpation revealed a term uterine fundal and no other organomegally. The fetus was in longitudinal lie, cephalic presentation and had regular fetal heart tones at 148/minute. The head was 5/5 above the pelvic brim and no tenderness or contractions were noted.

Pelvic examination

Her external genitalia were normal and vaginal mucosa felt healthy. The cervix was soft, central, 50% effaced and 2 cm dilated; giving a modified Bishop score of 5.The intact membranes were swept. The pelvis felt adequate

Management

A non stress test (NST) was done and 45 minutes later was found to be reactive. Since the fetal head was 5/5 above the pelvic brim, amniotomy could not be done because of the potential umbilical cord prolapse Istead enema was given and observation of contractions was done. Four hours later she had had good results of the enema and was having 1 contraction in 10 minutes lasting about 15-20 seconds.She was observed over night and she continued having the above contractions. Eight hours later abdominal examination indicated that the fetal head was 4/5 above the pelvic brim and on pelvic examination the cervix was 3 to 4 cm dilated and 60 to 70 % effaced. Amniotomy was done and scanty clear liquor was drained. There was no palpable cord on the pole of the presenting part. Syntocinon infusion was commenced at 6.30 a.m. Labour progressed well and after 7 hours since induction of labour the patient reached second stage. She was transferred to the delivery room. She was propped up and urged to exert valsalva manouvre whenever there was a contraction. Five units of syntocinon in 5% dextrose was running at 60 drops per minute and she had 3 contractions in 10 minutes each lasting 35 to 40 seconds. Descent was 2/5 (up). Pelvic examination showed full cervical dilatation. The fetal head had no moulding or caput succedaneum and was in occiput anterior position. Sixty-five minutes of second stage, however, she had not delivered. Cardiotocographic tracing showed mild late decelarations and fetal heart rate of 120 to 12 beats per minute. A decision to perform assisted vacuum delivery was made. She was put in lithotomy position. Vulvovaginal toilet, draping, catheterization, infiltration of 1% of lignocaine on the mediolateral aspect of the vulva and episiotomy performed on the same side were done in that order.

The vacuum extraction set was prepared. The center of a 50 mm cup was placed on the vertex 3 cm anterior to the posterior fontanelle. Palpation of the edge of the cup confirmed that there was no vaginal tissue being held by the cup. Vacuum pressure was then exerted up to 0.6kg/cm^2. When uterine contraction occurred the mother was asked to bear down maximally and a sustained traction in line with the pelvic curve led to the delivery of a live male infant with a birth weight of 3600 grams and an Apgar score of 8 and 10 at 1 and minutes respectively. A complete 500-gram placenta was delivered 5 minutes later by controlled cord traction. Both the cord and the placenta appeared normal grossly and the liquor remained clear. The episiotomy had not been extended and no lacerations were noted. Intramuscular syntometrine was given and the episiotomy repaired. The immediate postpartum vital signs were normal. A doctor from the Paediatric department reviewed the baby. It had no chignon or any other abnormally. He was to join the mother as soon as possible. The mother received prophylactic antibiotics and analgesics. Both the parturient and the infant did well postnatally. By the second postpartum day the mother's breasts were active without anomally, the uterus was involuting well and corresponded to 18 weeks gestation. The lochi

was rubra and scanty and the calves were soft and non-tender. Her 2nd postnatal day Hb was 10.6 g/dl. She was allowed home with advice to return to the postnatal clinic in 6 weeks.

Six weeks later she was in good general condition. Her breasts were fully active without any anomally, the uterus was fully involuted and the episiotomy well healed. She had not had her menses. Advice on contraception was given and she opted for the combined contraceptive pills. She was given a prescription of a progestin-only pill for six months after which she could start the combined contraceptive pills. A pap smear was to be done then (six months postpartum) in the Family Welfare Clinic.

DISCUSSION

The patient presented was a 32-year-old para 0+0 gravida 1 at 39+ weeks geastation who had delayed second stage due to poor maternal effort that necessitated vacuum extraction of a live baby with good outcome.

Though Simpson was the first person to suggest the use of a vacuum extractor in the 1840s, it was not until a century later, in 1954, that Malmstrom described, designed and produced the vacuum extractor. (Simpson was the man who designed the outlet delivery forceps – the Simpson forceps). Until then operative vaginal delivery could only be achieved by the use of the obstetric forceps. The vacuum extractor is an apparatus for delivery of a vertex- presenting foetus effected by traction applied to the foetal head through a suction cup. It has replaced the forceps in many countries in Northern Europe,where it is called the ventouse, while in Africa, vacuum assisted deliveries are the commonest type of operative vaginal delivery. [1,2,3]

The extractor of Malmstrom has the following components: A specially designed suction cup (which differed from the previous sucker by having the greatest diameter of the cup not at its point of application to the scalp but at a level 8mm from this); a hose connecting the suction cup to a pump with intervening trap bottle and manometer, and a chain inside a hose pipe that connects the suction cup to a crossbar for traction. The design of the cup permits the scalp to be anchored in the periphery and oedematous centrally formed chignon is held within its rim. Three cup sizes are available; 40mm, 50mm, and 60mm.[1]

The use of the ventouse has been made easier by two modifications. Binds modification of the suction cup permits more efficient traction and also eliminates the need to thread the chain through a hose.[4] Secondly the use of silastic cups as opposed to metal cups reportedly simplifies the procedure.[5] a 50mm silastic cup was used in this patient. One advantage of the silastic cups is that the vacuum can be applied rapidly as

opposed to the metal cup, which requires a stepwise application of vacuum over a period of time to enable the formation of a chignon. Just like for forceps delivery, the pre-requisites for vacuum extraction are: vertex presentation, fetal head engagement (station of 0 or less) and absence of cephalopelvic disproportion. The indications for vacuum extraction in the 1st stage of labour include lack of advance and delay at the end of 1st stage in the absence of cephalopelvic disproportion. In the second stage of labour, indications include prolonged second stage of labour. The American College of Obstetricians and Gynaecologists (1991) defines prolonged second stage of labour as second stage for more than 3 hours with regional analgesia and more than 2 hours without regional analgesia primigravida and more than 2 hours with regional analgesia and more than 1 hour without analgesia in a parous woman.[6]

Other indications include patients with conditions in which bearing down in the second stage of labour is not desirable such as cardiac disease, bronchial asthma, hypertension, and anaemia. The vacuum extractor is used to shorten the second stage of labour. Foetal indications for vacuum extraction include cord prolapse with cephalic presentation in late first or the second stage of labour, and foetal distress in late first stage or in the second stage of labour.[7] the vacuum extractor is used to expedite delivery and prevent further foetal compromise. The indication for assisted vacuum delivery in this patient was poor maternal effort and impending foetal distress. The vacuum extractor is contraindicated in any other presentation other than vertex presentations. It is contraindicated in premature fetuses (32 weeks or less) and in cephalo-pelvic disproportion. The membranes must be ruptured. Failure rate with the silastic cup was 0.9% among 1062 women in another series[8] The commonest reason for failure of vacuum extraction is poor or wrong technique especially when applying traction to the foetal head. Other reasons include faulty equipment and inadequate patient assessment (CPD, malposition of the foetal head). If delivery is not accomplished with four pulls synchronized with uterine contractions, caesarean section must be performed.[1, 3] In our case delivery was achieved with the first pull. Episiotomies (as performed in our parturient) if needed are given before the vacuum is applied and after anaesthetizing the vulva with local anesthetic agent.

Complications of vacuum extraction can be fetal or maternal. Maternal complications are usually injuries to the birth canal when maternal tissues are sucked into the cup and pulled with the foetus. Cervical and vaginal lacerations may cause postpartum haemorrhage and genital tract sepis. Iatrogenic vesico-vaginal fistulae have also been caused in this manner. Foetal complications are few in carefully selected patients and include cephalhaematoma and occasionally intracerebral haemorrhage and tentorial tears.[2] Rarely, subgaleal haemorrhage may occur if an intracerebral vein is ruptured. Intracranial haemorrhage is related to

the duration and strength of pulling of the vacuum extractor.[7] There were no foetal or maternal complications experienced during this delivery. The foetus scored well and there was no obvious cause for the foetal distress/reduced fetal movements. The placenta and the cord grossly appeared normal and the liquor remained clear throughout the labour period. The baby had no obvious anomaly even after paediatrian's evaluation. This is a case, which needed fetal scalp blood blood gases, and pH. Analysis. Both mother and baby were discharged on the third postnatal day in good general condition.

Compared to the forceps, the vacuum extractor was found by Johanson and Menon to significantly cause less maternal trauma (3rd and 4th degree lacerations, urinary and rectal incontinence, etc) but increased infant cephalhaematomas, jaundice and retinal haemorrhages.[9] Several other studies have showed that the vacuum extractor has fewer complications relative to the forceps.

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OBSTETRIC CASE NUMBER 14

BREECH PRESENTATION IN 2ND STAGE OF LABOR –ASSISTED BREECH DELIVERY- LIVE BABY

Name	: C.A.N.	DOA	:	18/09/2004
Age	: 32 years	DOD	:	19/09/2004
Parity	: 2 +1 gravida 4	LNMP	:	27/12/03
File Nº	: 0983651	EDD	:	3/10/2004
		MBD	:	38 weeks

Presenting complaint

CAN presented to the Kenyatta National Hospital (KNH) labor ward at 10 a.m with complaints of labor pains for 8 hours and drainage of liquor for 1 hour.

History of presenting complaint

She had been well until she developed intermittent lower abdominal pains that increased in intensity and frequency with subsequent passage of bloodstained, mucoid vaginal discharge. She had neither prior vaginal discharge nor any symptoms of urinary tract infection or change of bowel habits. Drainage of clear liquor occurred 1 hour prior to admission and though the fetal movements had subsided, they were still present.

Obstetric and Gynaecologic History

She was para 2+1 gravida 4. Her last normal menstrual period was on 27/12/2003, the expected date of confinement was 3/10/2004 and the fetal maturity by dates, therefore, was 38+ weeks. She had attended Eregi Mission Hospital for her antenatal care 4 times from 28 weeks gestation. The antenatal profile was; Hb 12.3 gm/dl, blood group O +ve and negative HIV rapid test. VDRL had not been done. She had been told that she would need to be delivered by elective cesarean section because she had breech presentation at term. Besides curdish vaginal discharge and associated one day's vaginal spotting that was successfully treated with vaginal pessaries, the antenatal period had been uneventful. She had, therefore, come to Nairobi where her husband stayed and worked so that she could undergo the operation.

She first delivered in 1997 to a live male infant whose birth weight was 3.7 kg and cried immediately. Her 2nd delivery was in 2001 to a live female infant with a birth weight of 3.2 kg. Both deliveries were by

spontaneous vertex delivery in hospital at term and the infants cried immediately after birth. Labor took about 8 to 10 hours and the puerperal periods were uneventful. In August 2003, she had a spontaneous miscarriage at 3 months gestation during which she bled a lot necessitating blood transfusion with 2 pints after uterine evacuation had been carried out. No contraceptive advice was given. Her menarche was at 14 years and had menses every 26 to 28 days each lasting 3 days. She had associated intra-menstrual dysmenorrhoea that was relieved by non-steroidal anti-inflamatory drugs. She had used the combined contraceptive pills from 1997 to early 2001 and in 2002 without any major adverse events. She had never had a pap smear done. She had had no history of post-coital or intermenstrual vaginal bleeding.

Past medical history

Besides the aforementioned blood transfusion this was insignificant.

Family and social history

She was a married housewife who had completed secondary school, drank no alcohol, smoked no tobacco and lived in Eregi village in Western Province, Kenya. Her husband was a hotel attendant and lived in Kibera slums of Nairobi. There was no history of familial disease.

PHYSICAL EXAMINATION

General examination

She was in good general condition with mild pallor but without pedal edema, jaundice or lymphadenopathy. Her BP was 130/80 mmHg; PR: 84/minute, RR: 20/minute and temperature of 36.5 ^{o}C.

Abdominal examination

The abdomen was uniformly distended. The uterine fundal height corresponded to 36 weeks gestation. The fetus was in longitudinal lie, breech presentation with regular fetal heart tones at 132/minute. Three uterine contractions occurred every 10 minutes each lasting 30 to 40 seconds. The descent of the presenting part was 3/5^{ths}. There was no other organomegaly.

Pelvic examination

She had normal external genitalia and healthy vaginal mucosa. The cervix was 9 cm dilated, the membranes had ruptured and complete breech presentation was noted. No umbilical cord was palpated.

Systemic examination was essentially normal.

Impression

An impression of breech presentation in advanced active phase of labor at term in a 32 year- old para 2+1 was made.

Management

In view of the high descent and the hospital policy to deliver all breech-presenting fetuses by cesarean section, the patient was prepared for emergency cesarean section. Informed consent was obtained, blood taken for grouping and cross match and the patient wheeled to theatre. However, while on the operating table, she rapidly progressed to 2nd stage. The breech and the lower trunk were delivered spontaneously. However, when the anterior scapula became visible, the arms were noted to be extended necessitating assisted breech delivery using Lovset's manouver. This entailed holdinding the fetus with both hands by the femoro-pelvic grip keeping the thumbs parralel to the vertebral column. The baby was then lifted to cause lateral flexion. The trunk was rotated through 180° keeping the back anterior and maintaining downward traction. The trunk was then rotated counterclockwise to the first rotation keeping the trunk anterior thereby delivering the shoulder that was initially anterior. Once the shoulders were delivered; the head was delivered by modified Mauriceau-Smellie-Veit procedure. This involved placing the trunk of the baby on the supinated left forearmwith the lower limbs hanging on either side of the forearm. The middle and the index fingers were placed over the malar bones on either side to maintain flexion of the head. The ring and little fingers of the pronated right hand were placed on the baby's right shoulder, the index finger was placed on the left shoulder and the middle finger was placed on the suboccipital region. With an assistant keeping giving suprapubic pressure mainly on the sinciput (to maintain flexion), traction was then applied downwards and backwards till the nape of the neck was visible.the fetus was then carried in upward and forward direction towards the mothers abdomen releasing the face, brow and lastly the tunk was depressed to release the occiput and the vertex. A mildly depressed female infant with a birth weight of 3300 grams and an Apgar score of 6, 8 and 10 at 1, 5 and 10 respectively was delivered. There were no anomalies (such as congenital hydrocephalus) noted. The placenta and cord were delivered and found to be normal grossly. Inspection of the entire genital track revealed no cervical, vaginal or perineal lacerations. Five hundred micrograms of intramuscular ergometrine was given and the uterus was noted to be well contracted with resultant minimal lochia loss. She was observed for one day and rooming in encouraged.

On the 1st postpartum day, she was in good general condition with mild pallor but without symptoms of anaemia. The breasts were beginning to be active; the uterus was well contracted at about 20 weeks gestation and the lochia loss and calves were normal. The baby was already breastfeeding. She was discharged on haematinics with advice to attend the postnatal clinic 6 weeks postpartum.

Follow-up

She opted to attend the Eregi Mission Hospital near her rural home.

DISCUSSIION

The patient presented was a 32 year-old para 2 + 1 gravida 4 at term in advanced active face of labour with breech presentation. She delivered by assisted breech delivery to a live male infant with a birth weight of 3300 grams and a good Apgar score and without any maternal complications.

Breech presentation is defined as the presentation to the pelvic brim of the podalic pole or the lower extremities of a fetus in longitudinal lie.[1,2] It is the commonest type of malpresentation with an overall incidence of 3-4% of all pregnancies.[1,2,3] In 1979, Njuki[4] found an incidence of 3.5% of all deliveries at KNH while the Nairobi birth survey found an incidence of 2.7%.[5] There are three types of breech described as per the fetal attitude.

- **Footling (incomplete) breech** occurs when one (single footling breech) or both (double footling breech) knees and/or legs are extended below the level of the buttocks.
- **In flexed (complete) breech**, both hips and knees are flexed. This is common in multipare.
- **Frank breech** occurs when the hips are flexed and both knees are extended.

Complete, footling and frank breech occurs in 10%, 25% and 65% of birth weights above 2500 grams respectively.[3] Our patient being a multipara had complete breech. The causes of breech presentation are not known for certain. However, factors that predispose to breech presentation include:

- Prematurity. This is the commonest cause of breech. The small size of the fetus and the relatively larger volume of amniotic fluid enable the fetus to undergo spontaneous version by kicking movements until 36th week when the position becomes stabilized. Thus the incidence of breech drops from35% at 28 weeks to 2-4% at term.[1,2,3]
- Favorable adaptation factors: hydrocephalus, placenta praevia, contracted pelvis and cornu-fundal placental attachment,
- Spontaneous version inhibiting factors: Twins, oligohydramnios, breech with extended legs,bicornuate uterus, short cord and intrauterine fetal death.
- Excess fetal mobility: Multiparae with lax abdomen, polyhydramnios.

Besides being multipara, our patient had no other obvious predisposing factor to breech presentation.

The diagnosis of breech presentation is made by abdominal and pelvic examination and imaging techniques (particularly ultrasonography). This should be made antenatally in order to prepare for the correct management. The Leopold maneuvers are used. During the first manouvre, a hard, round, readily ballotable fetal head is found to occupy the uterine fundus. The second manouvre indicates the back to be on one side and the small parts to be on the other one. If engagement has not occurred, the third manouvre identifies the breech above the pelvic brim. If egagement has occurred the 4th manouvre shows the firm breech to be above the pubic symphysis. As opposed to cephalic presentation the fetal heart tones are best heard above the umbilicus unless engagement has occurred when the fetal heart tones are heard below the umbilicus.[2,3] These findings may vary slightly with the type of breech. For instance, in frank breech the head is not easily ballotable as in complete breech due to limitation by the extended legs.

Vaginal examination before labor indicates soft and irregular parts felt through the fornices in complete breech while a hard feel of the sacrum often mistaken for the head is noted in frank breech.[1,2,3] During labor palpation of ischial tuberosities, anal opening, sacrum and the feet by he side of the buttocks indicates complete breech while palpation of the anal opening, ischial tuberosities and the sacrum without the feet indicates frank breech. The foot is identified by the prominence of the eel and the lesser mobility of the big toe. In incomplete breech one or both of the legs or knees are felt or seen below the buttocks. Clinical diagnosis may not be conclusive especially in obese women.

Imaging is not only important in diagnosing breech presentation but also in planning the mode of delivery in centers where vaginal breech delivery is practised. At KNH all but dead and grossly abnormal breech presenting fetuses and mothers in advanced labor are delivered by cesarean section. Ultrasonography is useful in confirming the diagnoisis, excluding fetal congenital anomalies such as hydrocephalus and uterine abnormalities such lower uterine segment myomas or bicornuate uterus. Further, it is used to determine the placental location, gestational age and number and the fetal weight, attitude and the biparietal diameter. These and clinical and radiological pelvimetry are important factors used in deciding the mode of delivery. X-ray imaging is not only used in pelvimetry but also for diagnosis of any bony congenital malformation and fetal attitude and size. In centers where it is readily available, computed tomography reduces the degree of irradiation and gives better pelvimetric assessment.[4] Magnetic resonance imaging (MRI) is non-irradiating and gives better pelvimetric assessment but was found to be of no added clinical value in reducing the cesarean rates in breech presentation.[5] In our patient, sonography was used to confirm the breech presentation and to exclude fetal anomalies.

The gestation at diagnosis determines the type of management of breech presentation. Expectant management is applied to await spontaneous version if the diagnosis is made before 36 weeks. In the absence of contraindications, external cephalic version is performed between 36 and 40 weeks. The preferred gestation is 37 weeks.[1,2] Version is a manipulative procedure designed to change the lie (of a longitudinally lying fetus) or to bring the comparatively favourable pole to the lower pole of the uterus and over or into the pelvic brim.[1,2] When the cephalic pole of the fetus is brought down to the lower pole of the uterus, it called cephalic version and when the podalic pole is brought down it is called the podalic version. The contrindications to external cephalic version (ECV) include engagement of the presenting part, premature rapture of the membranes, maternal rhesus negativity, oligohydramnios, placenta praevia, previous uterine surgery and systemic diseases such as diabetes mellitus, cardiac disease in pregnancy and thyroid diseases. Experience and adequate training, multiparity (lax abdomen), tocolysis, adequate amniotic fluid and complete breech are among the factors associated with successful ECV.

External cephalic version should be done in a setting with facilities for emergency cesarean section and preferably under tocolysis with β-adrenergic recepter agonists such as salbutamol, terbutaline or ridotrine. Where available a non-stress test should be done before the procedure. With an empty bladder the patient lies on her back with the shoulders slightly raised and the thighs slightly flexed. Using the forward somersault technique the breech is mobilized using both hands to one iliac fossa to which the back of the fetus lies. Pressure is then exerted to the head and the breech in the opposite direction to keep the trunk well flexed which facilitates version. Once transverse lie is attained the hands are exchanged one after the other to prevent crossing of the hands. Intermittent pressure is exerted till the head is brought down to the lower uterine pole. Fetal auscultation or non-stress test is repeated after the procedure and the patient observed for at least 30 minutes to rule possible cord accidents or abruptio placenta. Other risks of ECV are uterine rupture, fetal distress and/or demise, amniotic fluid embolism and preterm labor.[1,2]

The reported success rate of ECV ranges from 35-85% with a mean of 60%.[1,2,6] It has been found to reduce the cesarean rate in breech presentation from 77-88% to 30-37%.[6,7] When cephalic version is not attained then breech delivery is planned.

Breech delivery is either by cesarean section or vaginal. In our set up, however, planned vaginal breech delivery is not performed based on studies[2,10] that showed increased perinatal morbidity and mortality and relatively lower intelligence among individuals who were delivered by breech delivery. Besides our unit does not have the facilities (continuous fetal monitor, bedside ultrasound machine, etc) required for planned

breech delivery. Vaginal breech delivery is either spontaneous or assisted or total extraction. In spontaneous breech delivery no traction or manipulation of the infant is done. Assisted or partial breech delivery is when the infant is allowed to deliver spontaneously up to the umbilicus, and the maneuvers are initiated to assist in the delivery of the remainder of the body, arms and head. In total breech extraction the entire body of the fetus is extracted. This method is commonly reserved for the non-cephalic second twin.

For planned vaginal breech delivery to be carried out strict screening criteria should be used to select the patients. Factors that favor vaginal delivery include flexed fetal head, fetal weight between 2000 and 3500 grams and absence of maternal or fetal indications of cesarean section, documented lethal fetal congenital anomalies, previable fetus (gestational age below 25 weeks and/or weight <700g) and adequate maternal pelvis by radiological pelvimetry. The pelvic inlet should have an anterior-posterior diameter of at least 10.5 cm and a transverse diameter of at least 11.5 cm while the mid pelvis anterior-posterior diameter should be at least 11.5 cm and the transverse diameter 10.0 cm. Elective cesarean section is done in case of contracted or borderline pelvis, the estimated fetal weight is more than 3500g, premature fetus (25-34 weeks), elderly primigravida, prolonged rupture of membranes and deflexed head (increased risk of cervical spine injury). Others are a mother with poor obstetric history or infertility and footling breech (increased incidence of cord prolapse and coiling round the fetal limbs). Other criteria have been developed including the Zatuchni-Andros scoring system shown below.

	Add 0 point	**Add 1 point**	**Add 2 points**
Parity	0	1	2
Gestational age (weeks)	39	38	37
Est. fetal weight lb (Kg)	8 (3.6)	7-8 (3.6-3.2)	<7 (3.2)
Previous breech	0	1	2
Dilatation	2	3	4
Station	-3	-2	-1

A total score of 0-4 requires cesarean section. Our patient presented in advanced labour and, therefore this scoring system was not utilized.

Vaginal breech delivery (particularly planned) should ideally be carried out by a skilled obstetrician assisted by a paediatrician and an anaesthesiologist and in a facility equipped with tools for emergency caesarean section (c/s) and neonatal resuscitation. Upon admission vaginal examination should be done to

exclude cord presentation or prolapse in which case caesarean section is performed if imminent delivery is not feasible. Amniotomy is avoided as long as possible to allow the forewaters to act as a dilating wedge on the cervix. It is done only after excluding cord presentation. The patient should be in bed to reduce chances of spontaneous rupture of the membranes (SROM). The patient should be on nil by mouth just in case there is need for c/s.[1,2] Controversy exists on the use of oxytocin. In our setup oxytocin is contraindicated. In the USA some centers use it cautiously only for hypotonic uterine dysfunction. In assisted breech delivery the breech is allowed to be delivered spontaneously up to the umbilicus after which assistance is used to deliver the rest of the body. In frank breech, however, the extended legs may impede the delivery and then breech decomposition is performed by the Pinnard's manouver (1889). It involves use of two fingers to push the medial aspect of the nearest knee laterally away from the trunk. Spontaneous flexion of the knee then makes the foot accessible for grasping and downward pull. This method is applicable to total breech extraction.

Once the fetal umbilicus is past the perineum, the infant is held at the hips and gentle, outward and downward traction of the infant is made until the scapula and the axilla are visible. The Lovset's manouver is then used to deliver the shoulders. The infant is rotated backwards through 90° while applying downward traction, and the anterior shoulder is delivered by pressing on the inner side of the elbow. The infant is then rotated through 180° in the reverse direction and the posterior shoulder is swept out of the vagina. Upon delivery of the arms the infant is rotated back by 90° so that the chin is posterior. If the Lovset's manouver is not successful the posterior shoulder is delivered first by grasping the feet and elevating them in an oblique direction on the ventral aspect of the infant with one while the other hand pulls the posterior arm at the elbow joint.[1,2] The head is then delivered by Piper's forceps or by one of the Mauriceau-Smellie-Veit, Prague or Bracht maneuvers. These maneuvers are equally useful in breech delivery in cesarean section; for breech presenting infants are delivered by breech extraction in cesarean section.

The modified Mauriceau (1721)-Smellie (1876)-Veit (1907) manouver involves application of the index and the middle fingers of one hand over the maxilla (previously the jaw), to flex the head, while the body rests on the palm and forearm of the same hand. Two fingers of the other hand are then hooked over the fetal neck and gentle traction applied while the assistant applies suprapubic pressure to maintain head flexion. Once the nape of infant is visible, the body is then elevated towards the pubic symphysis to deliver the face, the brow and lastly the occiput. If the fetal occiput fails to rotate interiorly, then the modified Prague manouvaer is used to deliver the head with occiput-posterior position. Two fingers of one hand grasp the shoulders of the back-downfetus from below while the other hand draws the feet up over pubic

symphysis. The Bracht manouver is similar to the Mauriceau one but gravity and maternal efforts rather than traction are used. If the above maneuvers fail then the Piper's forceps is used. In our patient both the Loveset's and the Mauriceau-Smelie-Veit maneuvers were used.

Breech presentation is associated with increased perinatal morbidity and mortality. Maternal morbidity and mortality is increased by the high operative deliveries (vaginal and cesarean section). Perinatal mortality is increased 2 to 4 fold regardless of the mode of delivery. This is due to birth asphyxia due to difficult delivery of the aftercoming head and cord prolapse, which occurs in 15% and 5% of footling and complete breech respectively, compared to 0.5% in cephalic presentation.[2,3,10] In KNH Njuki[4] found the perinatal mortality in breech presentation in 1979 to be 2.5 times greater than that of cephalic presentation. Vaginal breech delivery is associated rupture of the sternocleidomastoid muscle, brachial palsy, fracture of the long bones and injuries of fetal adrenal glands, liver, anus, genitalia, hip joint, sciatic nerve and spine. Our unit, therefore, may be justified in the policy of delivering all but a few exceptions of breech presenting infants by cesarean section by the increased perinatal morbidity and mortality. However, this policy might need review as better facilities and research findings become available.

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OBSTETRIC CASE NUMBER 15

FOETAL DISRESS IN LABOUR – EMERGENCY CESAREAN SECTION, LIVE BABY

Name	: M.N.K	LNMP	: 03/12/2003
Age	: 19 years	EDD	: 10/09/2004
Parity	: 0 + 0 gravida 1	GBD	: 41 weeks +1 day
File No	: 0985091	DOA	: 25/09/2004
		DOD	: 01/10/2004

Presenting complaint

M.N.K. presented to the Kenyatta National Hospital (KNH) labour ward at 10.30 p.m. with complaints of intermittent lower abdominal pains for 12 hours.

History of presenting complaint

She had been well until she developed non-progressive intermittent lower abdominal pains that did not radiate to the lower back and were not associated with altered urination or bowel habits. She had not had any drainage of liquor neither had she noted any bloody mucoid vaginal discharge. The fetal movements were present and unaltered. She had no fever or loin pains.

Obstetric and Gynaecologic History

The patient was para 0 +0 gravida 1. Her last normal menstrual period was on 3/12/2003 and, therefore, the expected date of confinement was 10/09/2004. The gestation by dates was 42 weeks + 2 days. The pregnancy had been uneventful. She had had antenatal care at Gachii health center from 34 weeks. Her antenatal profile then was Hb of 12.1 g/dl, blood group A + ve and negative serological tests for HIV and VDRL. Then and thereafter the fundal height corresponded to the dates. Her coitarche was at 14 years and menarche was at the age of 16 years. Her menstrual flow occurred every 28 days and took 3 days. She had no history of abnormal menstruation, use of contraceptives or genital tract infections.

Past medical history was not significant.

Family and social history

She was married and earned her living by selling second hand clothes. She lived in Gachii with her husband who worked with her. Both of them did not drink alcohol neither did they smoke tobacco. There was no history of chronic illness in the family.

PHYSICAL EXAMINATION

General examination

MNK was in good general and nutritional status. She had no pallor, pedal edema, oral thrush, jaundice or lymphadenopathy. Her BP was 122/79 mmHg, temperature; 35.9 °C; PR; 74/minute and RR was 18/minute. Her height was 160 cm. No body weights had been measured.

Abdominal examination

This was uniformly distended and moved with respiration. There were no surgical or therapeutic scars. The uterine fundal height was term and the fetus was in longitudinal lie and cephalic presentation. The descent was 5/5 and the fetal heart tones were regular at 144/minute. No contractions, tenderness or other organomegally were observed, particularly in the suprapubic and the lumber regions.

Pelvic examination

Her external genitalia were normal. The vaginal walls were healthy. The cervix was posterior, 2 cm long, of medium consistency and closed. No show or any abnormal vaginal discharge was noted on the examining finger. The pelvis felt adequate and the fetal head could be felt on bimanual examination.

The respiratory, cardiovascular and the nervous systems were essentially normal.

Impression

Post-term in a teenage primigravida with possible intrauterine growth restriction (IUGR) and probable latent/false labour or urinary tract infection.

Management.

She was admitted to the antenatal wards to await labour. If she had false labour, induction of labour was to be performed after an urgent biophysical score had been done in view of the possible IUGR. However, 6 hours after admission she developed spontaneous rupture of the membranes (SROM) and an immediate vaginal examination indicated that the cervix was 2 cm dilated, of medium consistency and 2 cm long. No cord was palpated. She was draining fresh meconium stained liquor grade II. The uterine contractions were mild (1-2 occurred every 10 minutes each lasting 15-25 seconds). False labour was, therefore, ruled out. She was transferred to labour ward where an abdominal examination revealed an irregular pattern of the fetal heart tones at 100 to 128 beats/minute. A repeat vaginal examination confirmed the earlier findings

only that the meconium staining of liquor was now grade III. An emergency cesarean section (c/s) was to be carried out due to fetal distress.

She received explanation on the diagnosis and gave informed consent for the c/s. Pubic hair was shaved, premedication with intramuscular 0.6 mg of atropine was administered and the patient wheeled to theatre whose staff had already been informed to prepare for the operation.

In Theatre

In semi-lithotomy position, vulvovaginal toilet was done and 100 mls of clear urine drained by aseptic catheterization. Vaginal findings remained the same. The abdomen was cleaned, draped and opened via a Pfannenstiel incision. A lower uterine segment c/s was performed and by cephalic extraction, a live female infant with a birth weight of 2450 grams and an Apgar score of 6, 7 and 9 at 1, 5 and 10 minutes respectively, was delivered. The umbilical cord was tightly round the fetal neck twice while the placenta had moderate infarcts. Grade III liquor was noted. Fallopian tubes, ovaries and other abdominal viscera were found to be normal. The uterus and abdomen were repaired as per the description of c/s under introduction after accounting for the surgical equipment used. Vulvovaginal examination showed clear urine and no active uterine bleeding. Reversal of the general anesthesia was smooth. The paediatric team from the newborn unit received the baby and provided resuscitation. This included placing the baby on a tilted, warm resuscitare so that the head faced downwards; suction to clear the airway of mucus and meconium and provision of oxygen by mask. Spontaneous respiration was good so intubation was not indicated. The baby was then admitted to the unit because of the small for gestation (SGA). There were no overt signs of postmaturity syndrome or meconium aspiration syndrome.

Postoperative Care

Vital signs were observed ½ hourly until she was fully awake when the signs were then recorded every 4 hours. She received intramuscular pethidine for pain and intravenous crystalline penicillin and gentamicin for 24 and 48 hours respectively. On the first postoperative day, she was in good general condition without pallor, fever or dehydration. Breasts, respiratory, abdominal and pelvic and calf examinations were satisfactory. She was started on ambulation and oral sips to graduate to light diet by evening. On the second day she was fully ambulant and on light diet. She received her baby on the 2nd day and started to breastfeed. By the 3rd day antibiotics were discontinued and only took mefenamic analgesics. She was discharged with advice to attend the pediatric outpatient clinic in a week's time and the postnatal clinic in 6 weeks' time.

Follow up

By the end of 6 weeks postpartum, she was in good general condition without any complaint. Breastfeeding was well established and the baby looked healthy. She had no pallor; the breasts were normal and the uterus was completely involuted. There was no lochia loss or abnormal vaginal discharge. Advice on contraception and screening for cervical cancer was given and she accepted to have both levonorgestrel (Jadelle) insertion and Pap smear test, which were to be done in the Family Welfare Clinic.

DISCUSSION

The patient presented was a 19-year-old primigravida admitted in latent labour with post-maturity. Due to fetal distress, she underwent an emergency cesarean section and delivered a live SGA infant with mild asphyxia. She and her baby did well in the postoperative period.

Fetal distress is defined as a physiological state in which there is fetal metabolic acidosis secondary to hypoxia and is characterized by abnormal fetal heart rate and rhythm, poor Apgar scores, temporary or permanent injury or even death.[1, 2] Meconium passage is not always indicative of fetal distress but when combined with fetal heart rate abnormalities is a significant indicator.[1, 2,3] It is either acute or chronic.

The causes of fetal compromise are maternal, pathologies of the placenta and cord, fetal or a combination of these. Maternal causes of acute fetal compromise include decreased uterine blood flow (acute hypotension, shock, and cardiac failure), decreased blood oxygenation (hypoxia, hypercapnia) and uterine hypertonia (injudicious use of oxytocin, tetanic contractions). Placenta and cord problems include abruptio placenta, placenta praevia, umbilical cord compression (knots, prolapse or entanglement) placental "aging" and ruptured vasa praevia. Intrauterine fetal infection due to amnionitis could lead to fetal distress.[1, 2,3] The fetal distress in our patient was due to multiple factors including cord compression postmaturity and placental insufficiency due to infarction and calcification.

Chronic fetal compromise results from long standing fetal deprivation from the above and other causes that retard fetal growth and development. Fetal causes of chronic distress include multiple gestation, postmaturity, congenital anomalies and infections and erythroblastosis foetalis. Pre-eclampsia, chronic hypertension and diabetes mellitus are common causes of vascular abnormality that lead to decreased placenta perfusion and chronic fetal distress (FD).[1,2,3]

The diagnosis of fetal distress is based on presence of an SGA fetus as per ultrasonography and or the fundal height versus the dates; mother's perception of reduced fetal movements, poor biophysical score of less than 4 and more importantly abnormal fetal heart rate and rhythm. Abnormal heart rate characteristics include tarchycardia greater than 160 beats per minute (bpm), persistent bradycardia of less than 110 beats per minute, variable decelarations of greater than 50 beats per minute, reduced baseline variability of less than 5 beats per minute and persistent late decelerations. The use of continuous fetal cardiotogography to

diagnose FD is controversial.[5,6] For instance, Ayres-de-Campos[7], using inter-observer interpretation of fetal heart rate patterns, found out that experts agreed on only 62% of normal patterns, 42% of suspicious patterns and only 25% of pathological patterns. However, Dellinger and associates (2000),[6] using a combination of zero variability plus late or moderate-severe variable decelerations or baseline rate less than 110 bpm for 5 minutes to define FD, were able to predict normal outcomes for fetuses as well as discriminating true FD. Perhaps the true diagnosis of FD is made when the pH of fetal scalp blood, determined within 30 minutes of sampling, is less than 7.2.[1,2,4] other methods of detecting fetal distress include fetal response to stimulation (scalp stimulation or vibroacoustic stimulation) and fetal pulse oximetry. The latter and fetal scalp blood sampling require an open cervix and ruptured membranes.[1] In our set up continuous fetal cardiotocogram machines are unavailable and the diagnosis of fetal distress, as made in our patient, is based on BPP score, unsatisfactory fetal kick chart, abnormal fetal heart rate patterns on auscultation with the Pinnard's fetal sthetoscope and the presence of meconium stained liquor.

Abnormal cardiotocographic (CTG) findings suggestive of fetal distress include:

I. Abnormalities of fetal heart rate:

1. Bradycardia: A 10-minute baseline fetal heart rate less than 110 bpm (earlier: 120 bpm).[2,4]
    a) Mild: 100-119 bpm
    b) Moderate: 80-100 bpm
    c) Severe: less than 80 bpm in 3 minutes
2. Tarchycardia: Fetal heart rate more than 160 bpm
    a) Mild: 160-180 bpm
    b) Severe: more than 180 bpm

II. Abnormal fetal heart rate rhythm due to uterine contractions:

1. Late deceleration: A smooth, gradual, symmetrical decrease in FHR beginning at or after the peak of uterine contraction and returning to baseline only after the contraction has ended. Signifies diminished utero-placental perfusion.
2. Recurrent variable decelerations: Defined as recurrent decelerations of at least 15 bpm for at least 15 seconds, lasting no more than 30 seconds and with variable onset with successive contractions. Significant variable decelerations as described by the American College of Obstetricians and Gynecologists are those that decrease to less than 70 bpm and last more than 60 seconds.[7] They signify cord compression
3. Others: prolonged deceleration, saltatory and sinusoidal baseline heart rates and cardiac arrythmias.
4. Early decelarations indicate fetal head compression and are not indicative of FD.

Both fetal bradycardia and late decelerations are thought to be due to brain stem reduction of the sympathetic activity (leaving the vagal parasympathetic system to cause bradycardia) by chemicals prevailing in metabolic acidosis and by direct depression of the myocardium by acidaemia.[1, 2,8]

Meconium passage as an indicator of fetal distress is equally controversial because it could be as a result of a normal physiological process in term and particularly postterm infants or pathological.[2,4] Miller (1975)[1] found that the perinatal mortality when meconium was present without other observed signs of fetal distress was approximately 4.5%, but as high as 18.4% when other signs of FD such as fetal heart rate abnormality were present. Fongoh (1984) [9] at KNH found that meconium stained amnionic fluid in labour was associated with lower 1 and 5 minute Apgar scores and higher early perinatal morbidity and mortality. He also found a perinatal mortality rate of 80 per 1000 live births for cases of meconium stained amnionic fluid in labour. Just as in the aetiology of fetal bradycardia, meconium passage has been attributed to unopposed and increased vagal activity leading to increased gut peristalsis and subsequent meconium passage.[1, 2,3] Our patient had posmaturity but the presence of other causes of fetal distress indicates that the use of meconeum stained liquor to diagnose FD in this patient was justified.

Due to the controversies of diagnosis of fetal distress, its true incidence is unknown.[1, 2] However, the Nairobi birth survey conducted by Mati and colleaques in 1983 found a prevalence of FD to be 5.2% at the Kenyatta National Hospital.[10] In the western countries, the reported incidence ranges from 4.5% to 9.3%.[4]

Management of fetal distress if overt is delivery. However, the timing and mode of delivery depends on the cause, the gestation at the time of diagnosis, the type of FD and, if in labour, the stage and progress of labour. Chronic FD with resultant severe IUGR before 34 completed weeks requires lung maturation with steroids and subsequent delivery by cesarean section (c/s). Intrapartum FD characterized by non-reassuring patterns requires urgent vaginal examination to exclude cord compression and determine the stage of labour. In the absence of cord compression the parturient is nursed in the left lateral position and the legs elevated if there is maternal hypotension. Oxygen at a rate of up to 10 L/minute (6-10 L/min) is administered by mask or nasal catheter. Infusion of 10% dextrose may alleviate FD. If delivery is imminent and signs of FD persist, then assisted delivery by vacuum or forceps extraction is performed as long as there are no contraindications.

However, if delivery is not imminent then tocolysis is performed by use of beta-mimetics such as intravenous 0.25 mg of terbutaline or nitroglycerin or even magnesium sulfate. An emergency cesarean

section is then performed. Tocolysis is particularly useful in cases compromised utero-placental perfusion due to hypertonic uterus and placental insufficiency. Cook and Spinnato (1994) found significantly better pH values in fetuses whose mothers received terbutaline tocolysis than those whose mothers did not.[11] Fetal resuscitation should be anticipated and appropriate equipment and qualified pediatric personnel should be available for the resuscitation including intubations and Intensive Care Unit management.

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OBSTETRIC LONG COMMENTARY

ACCEPTABILITY OF HIV TESTING AND NEVIRAPINE ADMINISTRATION TO HIV SEROPOSITIVE MOTHERS IN EARLY LABOUR

LIST OF ABBREVIATIONS

ACU	-	AIDS Control Unit
AIDS	-	Acquired Immune Deficiency Syndrome
ANC	-	Antenatal Clinic
ARM	-	Artificial Rapture of Membranes
ARVs	-	Antiretrovirals
AZT	-	Azidovudine
CS	-	Caesarian Section
CT	-	Counselling and Testing
DNA	-	Deoxyvibonucleid Acid
HIV	-	Human Immuno-deficiency Syndrome
MCH	-	Maternal-Child Health
MTCT	-	Mother to Child (HIV) Transmission
NACC	-	National AIDS Control Council
NASCOP	-	National AIDS and STD Control Programme
NVP	-	Nevirapine
PCR	-	Polymerase Chain Reaction
PLWHAs	-	People Living with HIV/AIDS
PMH	-	Pumwani Maternity Hospital
PMTCT	-	Prevention of Mother to Child (HIV) Transmission
RNA	-	Rebonucleid Acid
UNAIDS	-	Joint United Nations Programme on HIV/AIDS
VCT	-	Voluntary Counselling & Testing (for HIV)
WHO	-	World Health Organization

INTRODUCTION

The number of people infected with Human Immunodeficiency Virus (HIV) continues to rise globally. The Joint United Nations Programme on HIV/AIDS (UNAIDS) estimates indicate that by the end of the year 2004 an unprecedented 4.9 million people contracted HIV.[1] Of these 640,000 were children; most of whom (90%) acquired the virus from their HIV infected mothers. Majority (4.4 million) of these new infections occurred in developing countries and particularly in Sub-Saharan Africa (3.1 million).[1]

In Kenya, estimates show that 120,000 children are living with HIV/AIDS.[2] They are among the estimated 1 million AIDS orphans in the country.[2] The risk of mother to child transmission (MTCT) among HIV infected mothers ranges from 20% to 42%.[3] This transmission (20 – 42%) occurs during pregnancy through the placenta (5-10%) during labour and delivery (10-20%) and through breast-feeding (10-15%).[3]

Prevention of mother to child transmission (PMTCT) is feasible as exemplified by reduction of the MTCT rate in developed countries from a range of 15% to 25% in mid nineties to as low as 2% to 5% in the year 2000.[3,4,5] This reduction stems from elaborate and effective PMTCT programmes instituted and implemented in mid and late nineties by the well-endowed developed countries.

The National AIDS and STD Control Programme (NASCOP) in collaboration with other institutions involved in the fight against the HIV/AIDS pandemic has developed guidelines on PMTCT that generally include primary prevention of HIV/AIDS in adults (to reduce the HIV prevalence), core intervention for prevention of MTCT and care and support of HIV seropositive mothers. The core interventions of PMTCT include, among others, short-course anti-retroviral (ARV) prophylaxis for mother and infant after counselling and testing (C.T) for HIV.[3] Unfortunately, a considerable number of pregnant women are not tested for HIV and even in places where voluntary counselling and testing (VCT) services are available some pregnant women still turn up in labour untested.

The NASCOP and the U.S. based Centre for Disease Control (CDC) PMTCT guidelines provide for counselling and rapid testing of expectant women in early labour.[3,4] However, whereas studies have been done in Kenya and elsewhere [6,48,51,52], on acceptance of CT in the antenatal clinic (ANC) acceptability of counselling and rapid testing for HIV in early labour had not been studied, in our set-up.

This study, therefore, aimed at determining the acceptability of counselling and rapid HIV testing in early labour and provision of short course nevirapine prophylaxis to seropositive clients. This was an interventional/operational, cross-sectional study that hoped to provide data that would be used to formulate appropriate evidence-based guidelines on PMTCT. It was carried out at Pumwani Maternity Hospital in Nairobi.

ABSTRACT

Background: Concerted efforts are now being made to provide counseling and HIV testing to all pregnant women at all points of contact with a health worker. These points include counseling and testing in early labour acceptability of which was hitherto unknown. Elucidating the determinants and the level of this acceptability would provide evidence-based guidelines aimed at enhancing PMCT.

Objective: To determine the acceptability of HIV testing and nevirapine administration to seropositive mothers in early labour.

Study site: The study was carried out in the labour ward of Pumwani Maternity Hospital, Nairobi, Kenya from January to April, 2004.

Study design: This was a cross-sectional descriptive study.

Methodology: A structured and pre-tested questionnaire was administered to all eligible clients in early labour. Pretest counseling was provided after which rapid HIV testing using Unigold® and Determine® test – kits was done to consenting pregnant women. HIV seropositive women were further asked whether or not they would accept nevirapine for PMCT. Results and responses were analyzed using the SPSS computer program version 11.

Results: Two hundred women were interviewed. 48% were aged between 20 – 24 years, 48% were nulliparus and 87% earned less than a dollar per day. The acceptability rate of counseling and testing in early labour was 76.5%. Women who had not been offered antenatal counseling and testing (CT), those who would have liked to have antenatal CT and those who knew that MTCT occurred during labour and delivery were significantly more likely to accept CT in early labour (P values 0.022, 0.013 and 0.004 respectively). Those who accepted CT and those who declined were similar in terms of socio-demographic factors. The HIV seroprevalence rate was 18.3%. Acceptability of nevirapine administration was 93% while 94.1% of those tested said they would disclose their HIV status to their spouses/partners and refer them for VCT.

Conclusion: Intrapartum counseling and HIV testing and nevirapine administration was acceptable to the study population and HIV seropositive women in early labour respectively. The study population had a significantly higher HIV sero-prevalence than that of the general population from the same setting. There is need for enhancing pre- natal and early post-partum PMTCT services in the study area and improving the services by including intrapartum counseling and HIV testing in the PMTCT program.

Key words: Acceptability, counseling and testing, Immunodeficiency virus, nevirapine, mother to child transmission.

LITERATURE REVIEW

HIV/AIDS has become a pandemic with worldwide devastating health and socio-economic problems. The Joint United Nations Programme on AIDS (UNAIDS) and World Health Organization (WHO) estimates indicate that by the end of the year 2002, 42 million people were living with HIV/AIDS.[1] Of this population were 3.2 million children who innocently acquired HIV through mother to child transmission (MTCT).[1,2] By the end of the same year (2002) an estimated 21.8 million people had died of the scourge since it was first clinically defined in 1981.[7]

The overwhelming majority of people living with HIV/AIDS (PLWHAs), 95% of the global total, live in the developing world. Sub-Saharan Africa is the most adversely affected with an estimated 29.4 million PLWHAs (or 70% of the global total).[1]

Kenya has a population of 30 million people, 2.2 million of whom are PLWHAs. Among the PLWHAs are 120,000 children under 5 years of age. About 1.5 million Kenyans have died of the syndrome with a resultant 1 million AIDS orphans.[2] This has led to enormous adverse health and socio-economic impact in the country. For instance, about 50 to 70% of medical-ward beds are occupied by patients with AIDS related illnesses [2,8] while the national life expectancy dropped from 58 years in 1995 to 46 years by the end of 2001.[2] Consequently, the Kenya Government in November 1999 declared HIV/AIDS a national disaster and established the National AIDS Control Council (NACC) to "provide a policy and strategic framework for mobilizing and coordinating resources for prevention of HIV transmission, and provision of care and support to the infected and affected people in Kenya". NACC consequently formulated the 2000-2005 National HIV/AIDS Strategic Plan.[9]

NACC is a multi-sectoral overall body incorporating the Kenya AIDS NGO consortium, other AIDS stakeholders and AIDS Control Units (ACUs) in each ministry. The ACU in the Ministry of Health is the National AIDS and STD Control Programme (NASCOP), which provide surveillance of the spread and prevalence of the epidemic among other things.

Using antenatal sentinel surveillance points spread throughout the country, NASCOP has progressively estimated Kenya's adult HIV prevalence rate, which was 5.1% in 1990, 13.5% in 2000 and 10.2% in 2002.[2,10] The 2002 prevalence rate was controversial given the very drastic reduction from 13.5% in 2000 to 10.2% in 2002 (within 2 years). Within the country regional prevalence, vary considerably ranging from

2% in Garissa, North Eastern province to 34% in Suba District, Nyanza province. Nairobi's prevalence stood at 16.8% in 2000 but NASCOP currently estimate 14% prevalence rate in the city.[10]

The HIV Virus

The Human Immunodeficiency Virus (HIV) was isolated in 1985, four years after the AIDS clinical definition had been made in 1981.[7] There are two types of HIV; HIV-1 and HIV-2. HIV-1 is more widespread and virulent than HIV-2, which is predominantly found in West, Africa.[11] Both HIV-1 and HIV-2 are members of the lentiviridae sub-family of retroviruses. HIV-1 is further sub-classified into serotypes A, B, C, D, etc. The predominant serotype in East Africa is A. Both HIV-1 and 2 are about 100 nm in diameter and bear an RNA genome. They cannot be extra-cellular for more than two hours before they die and are, therefore, obligate intracellular viruses. That it has many sub-types and the fact that it undergoes rapid mutations has made vaccine development rather difficult.

Its transmission occurs when HIV bearing human body fluids such as semen or blood from an infected person enters the bloodstream of uninfected individual. Specifically, its cytopathic effect begins with entry into CD4 antigen bearing cells such as T-helper lymphocytes, macrophages and placental trophoblasts cells, among others. Other antigens such as CD26 and CCR3 have been postulated as receptors for HIV leading to infection of the gut and bone marrow cell progenitors and placental trophoblast respectively.[12]

Once inside the cell an enzyme, reverse transcriptase, converts RNA into DNA, which is then incorporated into the victim's DNA and transcribed into virions. The transmembrane release of these virions causes cytopathic injury and/or death; hence the reason for reduction of CD4 T-helper lymphocytes. These lymphocytes, through lymphokines and other chemical substances, play a central role in coordinating both cellular and humoral immunity.[13]

There are several ways of HIV transmission including intimate sexual contact (heterosexual and homosexual), mother to child transmission (MTCT), transfusion with contaminated blood and being pricked or cut with contaminated instruments, as is the case with drug abusers. In Kenya the main modes of transmission are heterosexual (90%), MTCT (approximately 9%) and via blood transfusion (1%). Other forms of transmission exist but make a negligible proportion.[2]

Mother to Child HIV Transmission (MTCT)

This is a tragic HIV/AIDS phenomenon in that innocent children acquire the deadly virus from their infected mothers. Without any intervention an infected pregnant woman has 14% to 50% cumulative chance of transmitting the virus to her baby depending on her health and socio-economic status. MTCT

occurs antenatally (5 to 8% chance), during labour and delivery (10 to 20%) and through breastfeeding (10 to 15%).[3] Given a HIV prevalence rate of 13% and MTCT rate of 40% it is estimated that about 50,000 HIV infected children are born in Kenya each year.[3]

A number of factors determine the rate of MTCT in each of the stages. Generally, however, these include viral, maternal, foetal/infant and breastfeeding factors. Viral factors include the viral subtype, serotype/variant or type and the viral load.[14,15] Menu E. et al[15] showed in a study in Cameroon that certain subtype A variants could not pass through placental trophoblastic cells. High viral loads such as occurs in AIDS state and infection/seroconversion during pregnancy or breastfeeding increases the MTCT rate. An HIV infected mother with low CD4 lymphocytes count has a high chance of MTCT. Mayoux et al[16] in their French cohort study showed the risk of MTCT rose from 15% for the women whose CD4 lymphocyte count was above 600/mm^3 to 43% for those with counts below 200/mm^3. Studies with similar results have been done.

Other maternal factors that increase the MTCT rate are nutritional deficiencies (e.g. of Vitamin A), anaemia, presence of sexually transmitted diseases (especially ulcerating types), chorioamnionitis, frequent, unprotected sex, smoking and alcohol intake.[17]

Obstetric factors that increase the HIV vertical transmission are vaginal delivery (versus scheduled/elective caesarian section), rapture of membranes for more than four hours, intrapartum haemorrhage and invasive procedures. These procedures include amnioscopy, amniocentesis, episiotomies, instrumental deliveries, foetal scalp blood sampling, and external cephalic version.[18] Mandelbrot et al[18] showed an increased risk of HIV transmission during amniocentesis and amnioscopy (33% vs. 18.5%, P< 0.003), sexually transmitted diseases (26.4 vs. 17,3%, P< 0.003), preterm delivery (25.5% vs. 17.9%, P< 0.003), premature rapture of membranes (23,8% vs. 17.1%, P< 0.009), intrapartum haemorrhage (34,4% vs. 18.1%, P< 0.003) and bloody amniotic fluid (60% vs. 17.2%, P< 0.00001). The International Perinatal HIV Group[19] found out that among women diagnosed with AIDS the probability of transmission increased from 8% to 31% when the duration of ruptured membranes was 2 hours and 24 hours respectively (P< 0.01). The first twin has been noted to have a much higher risk of MTCT compared to the second twin probably due to longer exposure to cervical and vaginal excretions.[20]

Longer duration of breastfeeding, mixed feeding (breast milk and other feeds), presence of mastitis and cracked nipples and presence of infant oral thrush are some of the breastfeeding factors that increase the MTCT. De Cock et al[21] showed that the risk of HIV-1 transmission through breastfeeding between birth

and 18 to 24 months was 10 to 20%. In another study, Gray and McIntyre[22] estimated that worldwide 30% to 50% of perinatal HIV-1 transmission may be attributed to breastfeeding. In our country Nduati et al[23] in their randomized clinical trial estimated that 44% of all infant HIV infections were attributed to breastfeeding.

Timing of HIV Transmission

Most studies indicate that most HIV vertical transmission occurs in the peripartum period. Rouzioux et al[24] using viral kinetics (and other factors) among children born to HIV infected mothers estimated that 33% of MTCT occurred in utero, less than two months before delivery, while 65% occurred at birth. Mirochnik et al[25] estimated that 75% of MTCT occurred in the peripartum period.

Transmission through breastfeeding seems to be higher in the peripartum period than later, although the longer the breastfeeding period, the higher the transmission. Becquart et al[26] in a study in Bangui, Central African Republic, found 19% infection rate through breastfeeding in 6 months among 43 initially PCR-tested HIV negative neonates. A South African study by Coustoudis et al,[27] however, showed that those infants who were exclusively breastfed for at least three months had similar infection rates to infants who received formula feeding.Though perinatal transmission is the highest, MTCT has been showed to occur as early as eight weeks gestation although the transmission rate is low. This was noted by Brossard et al[28] who used thymuses of 100 spontaneously aborted or electively terminated fetuses. They found 2% transmission rate in early pregnancy.

Since most (75%) of MTCT occur during the peripartum period, majority of the intervention measures for PMTCT are geared towards this period. This is so particularly in developing countries where inadequate resources may not allow long-term intervention measures.

Prevention of Mother to Child Transmission (PMTCT)

Strategies for prevention of MTCT of HIV revolve around reducing and avoiding the factors that increase the MTCT rate. Broadly, these interventions include reduction of HIV prevalence among adults (aged 15-49 years), instituting specific interventions for PMTCT and care and support for HIV infected mothers.

Primary prevention of HIV/AIDS among adults remains the main means of combating the epidemic since there is no cure yet for it. These include, among others, promotion of behaviour change through appropriate information, education and communication aimed at promoting abstinence, faithfulness to one

sexual partner/spouse and correct and consistent condom use for those who cannot abstain, are single and sexually active. The objective is to reduce the overall HIV prevalence rate among reproductive adults and, therefore, mitigate PMTCT.

The 1998 Kenya Demographic and Health Survey (KDHS) indicated that most (79.9%) adult Kenyans were aware of HIV/AIDS and a considerable proportion of them knew that abstinence, being faithful to one sexual partner and correct and consistent condom use were key ways of avoiding contracting HIV. It also showed that majority (86%) of men and women (85%) knew that the virus could be transmitted to the foetus and the baby from an infected mother. Furthermore, only 14.4% women and 16.9% men had undergone HIV test while 63% of untested women and 65.5% of men desired to be tested.[29]

Despite 86% of women being aware of MTCT and 63% of them wishing to be tested, only a few of them actually get tested especially for purposes of MTCT. Studies on acceptability of antenatal counselling and testing of pregnant Kenyan women showed high acceptability rates of up to 99.4%.[6] The challenge, therefore, is to translate this desire into reality through widespread voluntary counselling and testing (VCT) in the general population and pregnant women in particular. Couple counselling is even more important given studies showing discordant couple rates of 18% in Uganda and 40% in Kisumu, Kenya.[30,31] Such couples are in the greatest chance of contracting HIV (and conceiving) because of unprotected, repeated sex.

The principle intervention measures for PMTCT consists of provision of comprehensive mother and child health (MCH) services, counselling and testing of pregnant women with concurrent provision of antiretrovirals to HIV infected mothers and their infants and prenatal care. Also included is provision of counselling on alternative infant feeding and practices and contraceptive services to all women generally and HIV infected ones particularly.

Intrapartum Care

This consists of active management of labour and specifically use of a partogram to reduce the risks of prolonged labour and the associated increase of MTCT rate. Avoiding unnecessary artificial rupture of membranes (ARM), episiotomy, foetal scalp blood sampling and digital vaginal examination mitigate MTCT.[18,19,20] Unless indicated ARM should not be done before cervical dilation of 7 cm. The National Guidelines for Prevention of Mother to Child HIV/AIDS Transmission[3] recommends that the vagina and

cervix should be cleansed with 0.25% chlorhexidine (Hibitane) at contact with the client and with every digital vaginal examination.

Once the baby's head is delivered, the mouth and nostrils should be wiped with a soft gauze or towel. Upon complete delivery, the cord should be clamped immediately while avoiding milking of cord blood towards the baby. Suction of the newborn with a nasogastric tube should be avoided unless the baby inhaled meconium-stained liquor. The baby should then be washed with warm chlorhexide solution or just wiped dry with a soft clean towel to remove maternal body fluids and thereby reduce MTCT. These measures should be offered to all women in labour whose HIV serostatus is unknown.

Mode of Delivery

Several studies on modes of delivery versus HIV transmission have showed that elective caesarian (CS) reduces prenatal MTCT by 40 to 80%. The International Perinatal HIV Group[32] using meta-analysis of 15 prospective cohort studies on scheduled/elective CS showed perinatal HIV transmission reduction by 55 to 80% whether or not patients received antiretrovirals (ARVs). In an international randomized trial of mode of delivery, transmission was 1.8% in women randomized to elective CS delivery though most had taken AZT.[33] Kabare[34] showed that there is no significant difference in wound sepsis rate between HIV infected and HIV non-infected women who underwent caesarian section. Scheduled CS should, therefore, be offered to all HIV infected pregnant women. However, due to shortage of resources and the high HIV prevalence rate, elective CS in developing countries is relatively difficult to accomplish. Scheduled CS, when combined with ARVs, has a higher MTCT reduction rate than when done without prophylaxis.

Antiretrovirals (ARVs) for PMTCT

Since Connor et al[35] of the Paediatric AIDS Clinical Trial (PACT-076) showed significant reduction of PMTCT using zidovudine (AZT), many other studies have been done on ARVs for PMTCT. Whereas Connor et all showed MTCT reduction from 22.6% (placebo) to 7.6% (AZT) a 66% reduction rate, the cost of this regimen (Kshs.15, 000 to 20, 000/- per client) is not affordable in most developing countries, Kenya included.[36]

In a short course antenatal/intrapartum AZT prophylaxis trial on non-breastfeeding women in Thailand, administration of 300 mg twice daily for four weeks antenatally and 300 mg every three hours during labour was shown to reduce perinatal MTCT by approximately 50% compared to placebo.[37] The cost per client would be approximately Kshs.3, 360.[36] A randomized double-blind placebo controlled (the PETRA)

study in South Africa, Uganda and Tanzania on HIV infected breastfeeding women showed that lamivudine (3TC)/AZT combination given orally from 36 weeks gestation, orally intrapartum and for one week postpartum reduced MTCT by about 50% at the age of six weeks.[38] This regimen would cost Kshs.3, 500/- per client.[36] Another arm of the PETRA study whereby 3TC/AZT combination to the mother at onset of labour and postpartum in both mother and infant showed reduced MTCT at six weeks from 15% in the placebo group to 9% with the two part 3TC/AZT regimen; a 40% reduction.

Perhaps the most cost-effective is the one based on the HIVNET 012 study done in Uganda. In this study 200 mg oral dose of nevirapine (NVP) given to the mother at onset of labour combined with a single 2 mg/kg oral dose given to her infant at 48-72 hours of age reduced MTCT by 47% at the age of six weeks compared to AZT given orally at onset of labour and to the infant for one week.[39] The cost of NVP in this protocol is estimated to be only Kshs.60 to 70.[36] Another study in Cote d'Ivore where AZT, started at 36 weeks and given orally intrapartum and for one week to the infant was combined with 200 mg NVP oral dose at onset of labour, showed MTCT reduced to 7.1% at 4 weeks. This was lower than PMTCT due to AZT alone.[40]

A closely related study to this proposed research is the one whose preliminary results were presented by Taha E.[41] to the 14th International AIDS Conference in Barcelona, Spain, in July 2002. In this study, mothers in early labour were counseled and tested for HIV. HIV infected mothers received a single oral dose NVP (200mg tablet) while their infants were randomized to receive either a standard oral single dose of NVP (2mg/kg weight) or a single oral dose of NVP plus AZT (4mg/kg weight) orally twice daily for a week. Preliminary results indicated no additional benefit from the combined neonatal NVP and AZT relative to NVP alone given to infants of HIV infected mothers who received intrapartum NVP. However in both cases there was a significant reduction of vertical HIV transmission.

Antiretrovirals for PMTCT in Developing Countries

Most antiretrovirals remain relatively expensive drugs that are out of reach to most developing countries, Kenya included. Affordability, availability and adherence to antiretroviral treatment protocols are essential for, not only effective PMTCT, but also prevention of development of drug-resistant HIV variants. The WHO and UNAIDS, in collaboration with other institutions and individuals, have, therefore, recommended cost-effective ARV regimens for PMTCT in developing countries.[3,21] These guidelines are short course regimens that do not require extensive laboratory tests, close monitoring and intravenous equipment. The

cost of HIVENT-012 regimen recommended in Kenya, for instance, is only Kshs.70/- compared to Kshs.15, 000 to Ksh 20,000/- for the PACT 076 AZT regime used in the U.S.A.

Nevirapine (NVP Viramune)

This is a non-nucleoside reverse-transcriptase inhibitor that has been shown to be a cost-effective ARV for PMTCT.[39] Its efficiency is due to its rapid absorption (IC_{50} in < 1 hour), wide distribution throughout the body including brain, high transplacental transfer, activity against cell free virions and long plasma half life ($t_{1/2} \geq 72$ hrs).[42] Besides, mothers who took 200 mg of NVP had high levels of the drug in their breast milk for the whole of the first week postpartum.[25]

Nevirapine has a few side effects most of which are dependent on the dose and/or duration of use. These side effects include skin rash of various degrees, hepatotoxicity and a form of hyper-sensitivity syndrome that includes fever, myalgia, anthralgia, hepatitis and eosinophilia (The syndrome may or may not precede the rash which usually occurs in the first two to four weeks of treatment in about 17% of patients.[43] Serious (grade 3 to 4) rash requiring treatment discontinuation occurs in about 6 to 8% of patients.[43,44]

Toxicity of single dose nevirapine prophylaxis appears to be minimal. Two phase I safety and pharmacokinetic studies in the USA[25] and Uganda[42] and three large randomized, comparative phase III clinical trials in Uganda[39], in the USA, Europe, Brazil and the Bahamas[46] altogether showed no significant clinical or laboratory toxicity with single dose nevirapine. Hence HIV negative mothers and their babies who will receive nevirapine in the basis of discordant results will be expected to have minimal side effects. It should be noted, however, that babies on long-term treatment with NVP will develop mitochondrial abnormalities.[39]

Resistance to Nevirapine

This may pose a very big problem to the cheap, easily administered single dose NVP. Although a study by Eshleman S. H. et al[47] showed 20% resistance at 6 weeks, the mutant HIV-1 variants could not be detected 12 to 18 months after treatment implying NVP could still be used later since the resistant HIV mutants appeared to die within two years.

RATIONALE

Kenya has a high (10.5 to 13.5% - NASCOP 2001[10]; 9% in women aged 15-49 – KDHS, 2003[49]) adult HIV prevalence rate which when combined with a relatively high birth rate leads to high MTCT. Prevention of MTCT is, therefore, a big challenge that should be tackled by all acceptable means possible. Counselling and rapid testing for HIV in labour and administration of ARV prophylaxis to HIV infected parturients is recognized as one way of reducing PMTCT. However, whereas studies have been done in acceptability of voluntary counselling and testing (VCT) for HIV in the antenatal period,[6] no study has been carried out in our set up on acceptability of counselling and testing in early labour. Besides a considerable number of women in Kenya do not attend antenatal clinic and, therefore, do not benefit from the antenatal VCT and ARVs for PMTCT. Still a substantial proportion of those who attend do not undergo HIV testing because of various reasons including inability to pay for the test.

Since the administration of NVP will be directly observed as opposed to NVP given to HIV sero-positive clients after antenatal VCT, the problems of reduced adherence compared to uptake as noted by Sinkala[46] in Lusaka, Zambia will be reduced.

The study is also rationalized by virtue of its operational nature. Since NVP will be offered to HIV infected mothers and their infants the study will help to reduce PMTCT among study subjects. Other aspects of HIV/AIDS including advice for partner referral for VCT will also be provided to both HIV sero-positive and sero-negative mothers.

Counselling and testing in early labour and intrapartum, use of NVP is hoped to be a "safety net" intervention to mitigate MTCT, especially in areas with high HIV prevalence rates.[3,4,11]

OBJECTIVES

1.1 Broad Objective

To determine the acceptability of HIV testing and nevirapine administration to HIV sero-positive mothers in early labour.

1.2 Specific Objectives:

1. To determine the acceptability of HIV testing among women with unknown HIV serostatus in early labour
2. To determine the socio-demographic characteristics of women in early labour at Pumwani Maternity Hospital (PMH).
3. To determine the knowledge of the study population on perinatal HIV transmission.
4. To determine the HIV prevalence and its correlates among women in early labour at PMH.
5. To determine the acceptability of nevirapine for PMTCT among HIV seropositive women in early labour.
6. To determine the correlates of test acceptance and nevirapine uptake

METHODOLOGY

The Study Design

This was an interventional, cross-sectional descriptive study.

The Study Site

The study was done at Pumwani Maternity Hospital (PMH), which is located in the eastern side of Nairobi city about 2 km from the city centre. It is the largest maternity hospital in East and Central Africa with an average patient/client turnover of 30, 000 per year. This makes it a suitable study site for a survey study where results are needed within a relatively short time. It is managed by the Nairobi City Council public health department through a team of Obstetricians and Gynecologists, Paediatricians, Medical Officers, Anaesthetists, nurses of various cadres and other paramedical and subordinate members of staff.

Study Population

The study population consisted of pregnant women at term presenting in Pumwani Maternity Hospital (PMH) in early labour. Being a public referral hospital to all the Nairobi City Council MCH health centres, PMH provides maternal services to about 70 to 80% of the city's 2.3 million people. Its clients / patients are relatively of low socio-economic status compared to clients who attend privately owned hospitals within the city.

Sample Size and Sampling Method

It being a cross-sectional descriptive study, the sample size was calculated using the formula:

Sample size, $n = \frac{Z^2 pq}{d^2}$

Where n = the desired sample size

z = value which is the normal standard deviation usually set at 1.96 which corresponds to 95% confidence interval.

p = prevalence taken to be the probable acceptance rate for HIV screening in this study population. A previous study by Kamau (7) showed acceptance rate of 88.3%. Hence p will be taken to be 0.883.

q = 1.0 - p = 0.117

d = degree of accuracy with which p was determined, set at 0.05.

Thus sample size = $\frac{1.96 \times 1.96 \times 0.883 \times 0.117}{0.05 \times 0.05}$

= 159 study subjects

This sample size was increased to 200 study subjects who were recruited using simple systematic sampling method whereby every third client was selected.

Personnel and Participant Recruitment

The study team comprised of the principal investigator and two assistants. The assistants were nurses with experience in counseling and testing (CT) for HIV. The principal investigator and the assistants underwent training in testing for HIV and in the administration of the questionnaire. The Kenyatta National Hospital VCT training centre provided the training and supervision for CT.

Pregnant mothers in early labour were recruited after satisfying the inclusion criteria provided below. Pre-tested questionnaires were used to obtain data from clients who consented to the study (Appendix I & II).

Inclusion Criteria

Women were eligible for the study for the study if they:

- Consented to the study
- Were in early labour (with cervical dilatation of not more than 4 cm) and with mild contractions.
- Were at term.
- Had unknown HIV serostatus.

Exclusion Criteria

Women were excluded from the study if they:

- they had other complications such as antepartum haemorrhage, mental disturbance, eclampsia, etc.
- Those who are under 18 years and/or those who are not "mature minors" aged at least 15 years and / or those who are at least 15 years and less than 18 years but not accompanied by a guardian aged at least 18 years.

Determination of whether a client met the above criteria was made after the client had been clerked and a pelvic examination done by a midwife or a doctor to ascertain the stage of labor. A mother who met the criteria was then led to the counseling and testing room in labour ward. The room had a bed suitable for mothers in labour and all the equipment for conducting counselling for HIV testing. The standard counseling for HIV testing and particularly the meaning and/or implication of negative and positive results were provided.

Thus, every third mother in early labour was randomly recruited by being clerked, undergoing pelvic examination, being counseled, tested and administered nevirapine, as long as consent had been given.

Procedure for HIV Testing using Determine® and Unigold® Test Kits

Since study subjects were in labour, rapid HIV testing kits were used in order to get results quickly and decide on PMTCT using nevirapine. The test kits were Determine for screening and Unigold for confirming the results of the screening test. In case the results from the screening test and the confirmation

test were different, the blood sample was to be sent to Kenya Medical Research Institute (KEMRI) for a Tie Breaker test. However, none of the participants had indeterminate results. Determine's screening test has a sensitivity of 98.5% to 100% and a specificity of 97.6%, while Unigold's sensitivity and specificity are 95% and 97.9% respectively.

Using a clean microfilter tube a 50 µl drop of blood, obtained by a lancet finger prick, was placed onto prepared Determine and Unigold test strips; both at the same time. One drop and two drops of respective buffer solutions were then added to the Determine and Unigold strips respectively. The investigator together with the client read the results fifteen minutes thereafter.To ensure quality assurance every 5th sample was sent to KEMRI through the Kenyatta National Hospital VCT Centre for quality control testing. The specific steps and associated illustrations are provided in Appendix III.

Administration of Nevirapine

Once the results were provided the participants were then counseled accordingly. HIV – infected mothers in early labour were soon thereafter offered a single oral dose of a 200mg nevirapine tablet after ensuring absence of contraindication to the drug (e.g. jaundice, liver disease). They were also asked to give consent to administration of single oral dose of nevirapine to their neonates at a dose of 2mg/kg/body weight within 48 hours postpartum.

Single dose nevirapine was already being offered to HIV – infected pregnant women tested antenatally at PMH. The safety measures established by the hospital with regard to nevirapine administration, therefore, applied. Besides, involved mothers were advised to report to the study site in the event of occurrence of any adverse effect of the drug both to themselves and/or their neonates.

Evaluation of the drug's efficacy, though desired was not possible due to financial constraints. Its efficiency was, therefore, not assessed, but was assumed to be equivalent to the efficacy found in the HIV-NET 012 study.

ETHICAL CONSIDERATIONS

HIV infection is still stigmatized and since there is no cure as yet for the scourge, participants' right to confidentiality was highly upheld. HIV testing involved only study subjects who met the inclusion criteria, signed consent to be tested and had undergone pretest counseling (including being briefed on the meaning of positive and negative results). The actual physical appearance and the interpretation of the results were provided after which post-test counselling was given.

HIV seropositive study subjects were counseled on and offered the use of nevirapine for PMTCT. They were administered the drug only after signing consent to do so. The drug was provided free of charge to both mother and infant. All study participants and non-participants were entitled to the usual optimum obstetric and other health care provided at the study site. Potential study subjects were informed that participation would be absolutely voluntary. A copy of the information and consent forms appears as Appendix I.

Patient Support Mechanisms:

It being an interventional study, participants benefitted from a number of ways including:

a) Treatment of STIs, opportunistic infections and minor ailments.

b) Referral to other health institutions for treatment and/or follow-up. HIV-infected women, in particular, were referred to various patient support centers such as the one at Kenyatta National Hospital. They were counseled on safe sex, breastfeeding options, family planning, nutritional requirements, and the use of anti-retrovirals, etcetera.

c) All participants were encouraged to disclose their HIV status to their spouses and to ask them (spouses) to also undergo counseling and testing for HIV. Mothers and their neonates (and their spouses) also benefited from weekly group counseling on HIV/AIDS/STI and reviews when necessary as in the occurrence of a serious adverse effect of nevirapine. This took place up to three weeks after completion of the study.

DATA MANAGEMENT

Data obtained using the questionnaire (Appendix II) by the investigator and his two assistant investigating nurses trained in CT, was crosschecked by the investigator before being entered into a microcomputer using SPSS data entry programme.

Univariate analysis was performed for each of the dependent variable for acceptability to HIV testing. Examples of these variables are age, level of education, residence and marital status. All dependent variables that were significantly associated with acceptability to HIV testing in early labour were subjected to multi-variate logistic regression analysis. A p-value of <0.05 was considered significant.

RESULTS

From January through April 2004 two hundred women who presented to Pumwani Maternity Hospital (P.M.H) in early labour and were eligible for the study were recruited. Their socio-demographic characteristics are shown in Table 1 below.

Table 1 Socio-demographic Characteristics of the Study Population

Background Characteristics N = 200	N	%
Grouped Age (yrs)		
15 – 19	33	16.5
20 – 24	95	47.5
25 – 29	50	25.0
30 – 34	13	6.5
35 – 39	9	4.5
Mean Age = 24 years	24	100
Parity		
0	96	48
1	58	29
2	18	9
3	24	12
4	2	1
>5	2	1
Mean Parity = 1.0	1.0	100
Marital Status		
Single	23	11.5
Married	173	86.5
Divorced or widowed	4	2.0
Level of Education		
None	1	0.5
Primary	113	56.5
Secondary	71	35.5
College/university	15	7.5
Occupation		
None/Housewife	128	64.0
Self employed	51	25.5
Salaried/formal	16	8.0
Student	2	1.0
Casual	3	1.5
Average Monthly Income (Ksh.)		
<1000	11	5.5
1000-1999	11	5.5
2000-3999	24	12
4000-5999	14	7
>=6000	15	7.5
None	125	62.5

Most (89%) of the clients were less than 30 years of age with the majority (47.5%) being within age group 20 – 24 years. Majority (48%) of the clients were nulliparous, had had some or complete primary education (56.5) and were housewives and or had no occupation (64 %). Most (86.5%) earned less that a dollar per day (were below the poverty line). Most (70%) of the spouses of married clients earned less than Ksh. 6000 per month and were unemployed (16.8%), self employed (43.4%) or worked as casual workers (20.3%). These results are illustrated in Table 2.

Table 2: Occupation and Income of Clients' Spouses.

Spouses Occupation	**N**	**%**
Unemployed	29	14.5
Self-employed	75	37.5
Salaried/formal	33	16.5
Student	1	0.5
Casual worker	35	17.5
Not applicable	27	13.5
Spouse's Average Monthly Income (Ksh.)		
<1000	4	2.0
1000 – 1999	12	6.0
2000 – 3999	30	15.0
4000 – 5999	41	20.5
> 6000	57	28.5
None	56	28.0

Clients were asked whether they had attended any antenatal clinic during the index pregnancy and if so how many times. They were also asked whether they had been tested for HIV and were only included in the study if they had unknown HIV serostatus. The results of their responses are shown in Table 3.

Table 3: Antenatal Clinic Attendance, Pre-test HIV serostatus and Reasons for Not Being Tested During Prenatal Period.

Characteristic	N	%
Attended ANC	200	100
No of visits		
• 1	13	6.5
• 2	19	9.5
• 3	41	20.5
• 4	36	18.0
• >=5	91	45.5
HIV serostatus		
• Unknown	171	85.5
• Negative > 1 year ago	29	14.5
Reason for Not Being Tested in Antenatal Period		
• Afraid of knowledge of HIV status	26	13
• Not Offered VCT	122	61
• Didn't know where to go for VCT	10	5
• Assumed had been tested	23	11.5
• Needed time to decide	14	7
• Others	5	2.5

All women attended at least one ante-natal clinic; most (84%) at least three times; yet a considerable proportion (61%) of them said they were not tested in the prenatal period because they were not offered counseling and testing. Other reasons for not being tested in the antenatal clinic included: fear of knowledge of positive serostatus (13%), lack of knowledge of where to be tested (5%), "assumed had been tested" (11.5%) and "needed time to decide" (7%).

Knowledge of Vertical HIV Transmission and Its Prevention.

For purposes of determining predictor variables of acceptability of HIV testing in early labour knowledge of mode of HIV transmission in general and the timing and/or mode of mother to child transmission in particular, were assessed. Each client was asked to name as many as possible the means of transmission of HIV, prevention of MTCT and the timing of MTCT. The client was not asked whether this method and/or timing was correct ("YES") or not ("NO") but a "NO" was marked against a response when the client did not name the particular response. The results to these questions are given in figures 1,2 and 3 respectively.

In order to know whether knowledge of the mode of HIV transmission had any influence on the acceptability of HIV testing and nevirapine administration in early labour participants were asked to name as many as possible the modes of HIV transmission in the general population. The modes of transmission

enlisted in the questionnaire were sexual intercourse, mother to child transmission (MTCT), not using condoms/unprotected sex and transfusion with contaminated blood.

In addition sharing razors/ sharps, other no-specified modes and lack of knowledge of any transmission were also short-listed as options. This was unlike past studies where participants were asked to indicate whether a mode of HIV transmission, each of which was read out to the participant, led to the actual HIV transmission. The results to these questions are shown in figure 1 below.

Figure 1: Knowledge of Mode of HIV Transmission.

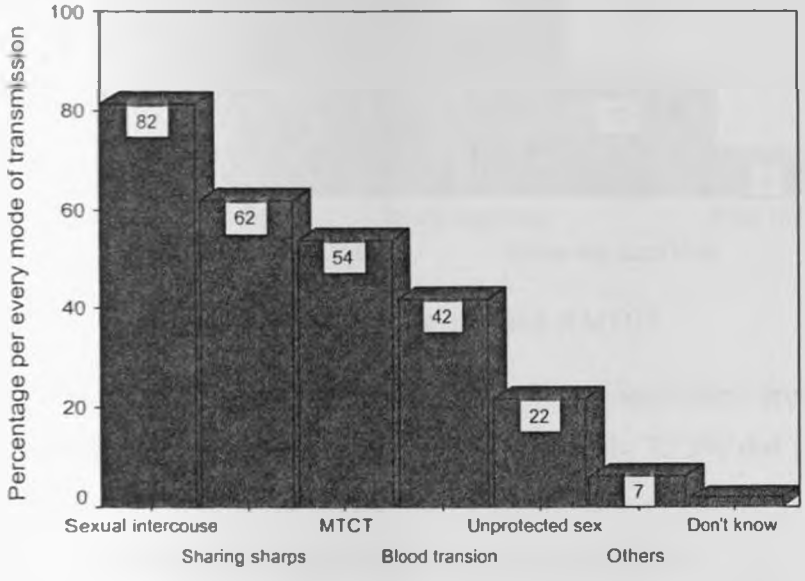
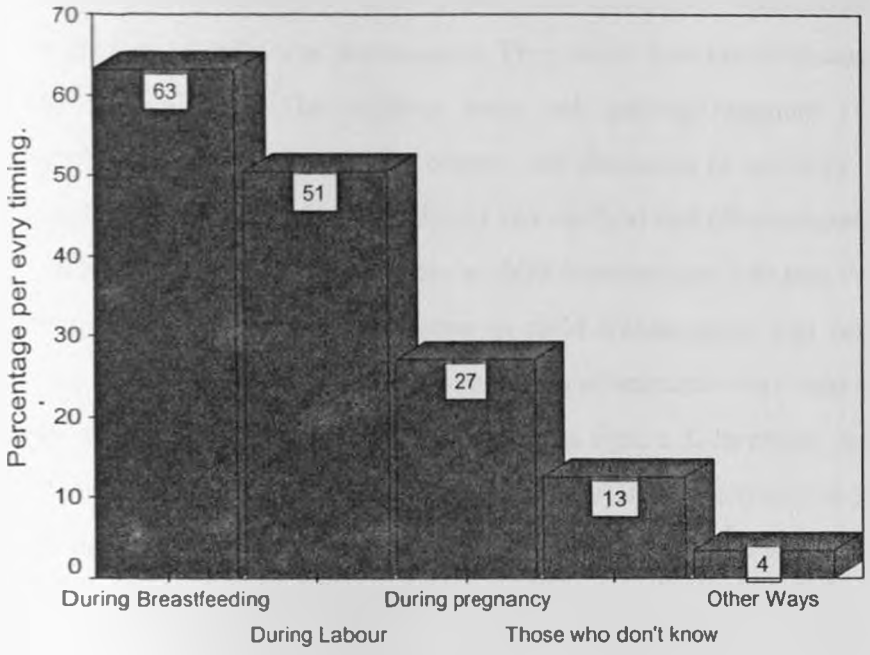


Figure 1 illustrates that virtually every one (99.5) of the study population knew at least one mode of HIV transmission. Most (77.5%) knew that HIV is sexually transmitted but only 44 (22%) clients specified unprotected sex/not using condom as the means of HIV transmission. Vertical transmission was named by 104 (54.0%) clients as a mode of HIV transmission.

Participants were asked to name as many as possible the time and/or activity during which mother to child transmission could occur. The options enlisted in the questionnaire were during pregnancy, during labour and delivery, during breastfeeding, others and lack of knowledge of any of the mode and/or activity that lead to mother to child transmission. The responses to this question are shown in figure 2 below.

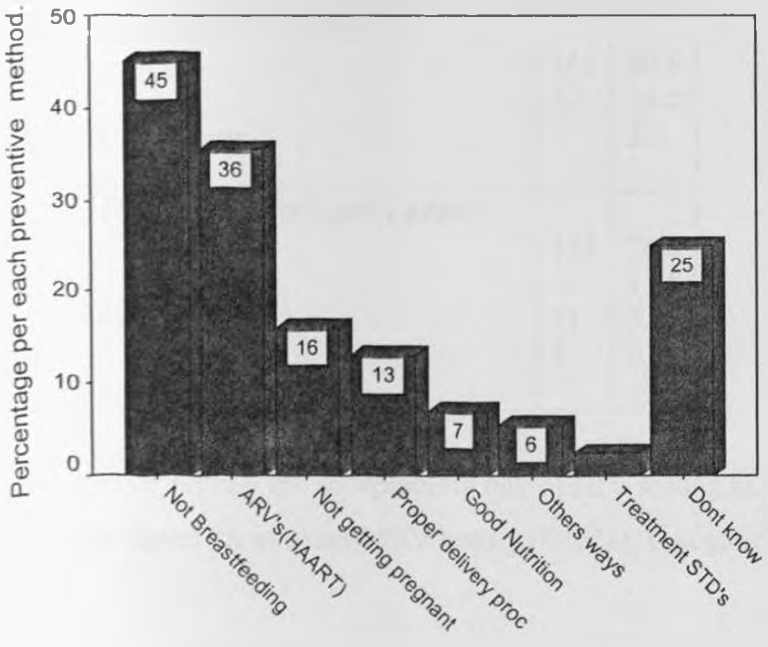
Figure 2: Knowledge of Timing of MTCT



Knowledge of timing of MTCT

As shown in figure 2, majority (63%) of the clients mentioned breastfeeding followed by during labour and delivery (51%) and during pregnancy (27%). Only 12.5% did not know the timing of mother to child transmission.

Figure 3: Knowledge of mode of Prevention of MTCT



Knowledge of mode of PMTCT

To determine whether knowledge of methods of prevention of mother to child transmission would influence the acceptability of HIV testing and nevirapine administration, participants were first asked whether mother to child was preventable. They were then asked to name methods of prevention of mother to child transmission. The options were not getting pregnant if one is HIV-seropositive, taking antiretrovirals, proper delivery procedures and treatment of sexually transmitted diseases. Other options were good nutrition, lack of knowledge of any method and other unspecified ways.

Most (76%) of clients said that mother to child transmission was preventable. Sixteen percent did not know or were not sure while 8% said mother to child transmission was not preventable. After being told that mother to child tansmission was preventable, participants were asked to name as many as possible the methods of prevention of MTCT. As shown in figure 3, however, only a modest 45% and 35% of the women mentioned not breastfeeding and taking ARVs respectively as the methods of preventing mother to child transmission. A quarter (25%) of the women did not know of any method of prevention of mother to child transmission.

ACCEPTABILITY

The desire for antenatal HIV testing was assessed prior to counseling and subsequent testing once consent had been granted to do so.

Table 4: Acceptability of HIV Testing in Early Labour and Desire for Antenatal HIV Testing

Characteristics N=200	N	%
Desired Antenatal HIV Testing		
Yes	161	80.5
No	32	16.0
Not Sure/Don't know	7	3.5
Accepted HIV Testing in Early Labour		
Yes	153	76.5
No	35	17.5
Yes but later	11	5.5
Unsure	1	0.5

As indicated in Table 4 the acceptability rate of HIV testing in early labour was 76.5%, which was slightly less than the desire for antenatal HIV testing (80.5%). Categorical ("NO") refusal rate was 17.5%

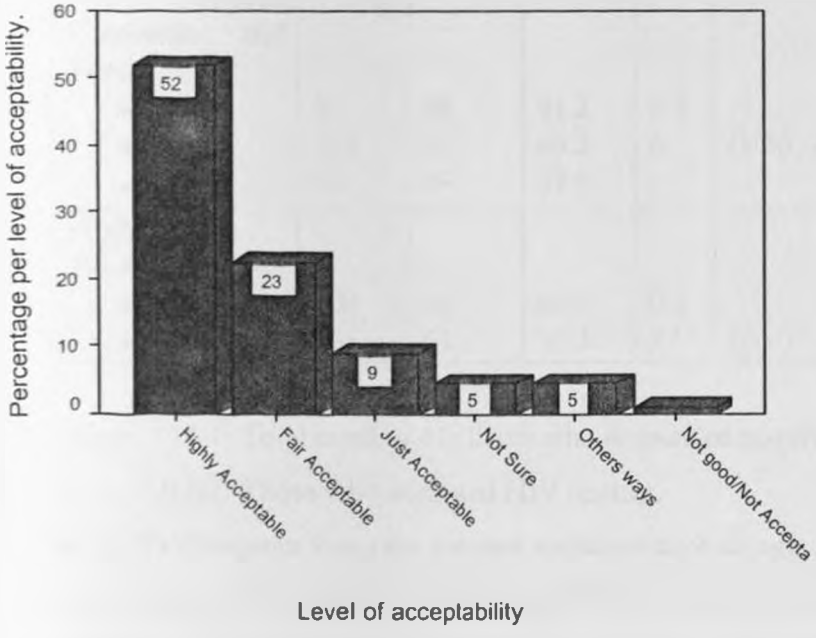
Forty- seven (23.5%) of the study subjects who were not ready to be tested were asked to give reasons for their decisions. Their responses are tabulated in Table 5 as shown.

Table 5: Reason for Declining HIV Testing in Early Labour.

Reasons: N = 47	N	%
• Afraid of knowing am infected with HIV.	16	34.0
• Am in Labour	5	10.6
• Have to inform my spouse first	5	10.6
• Was not prepared for the test	9	19.1
• Need more time to decide	7	14.9
• It is unnecessary	5	10.6
• Others (specified)	4	8.5

This denotes that majority (34%) of the clients were afraid of knowing that they had been infected with HIV. Another 34% were unprepared for the test (needed time to decide, had to inform spouse first and/were unprepared) while being in labour, the test being unnecessary and having to inform the spouse first each received 10.6% of the responses.

Figure 4: The Level Of Acceptability Among Clients.



Acceptability of HIV testing was further tested by asking all the women interviewed (tested and untested) to indicate their level of acceptance to the testing in early labour. As indicated in the bar graph below, (Figure 4), most (83.5%) said that the testing was acceptable, [just acceptable (9%) fairly acceptable (22.5%) and, the majority, highly acceptable (52%]. Sixty-two (31%) women spontaneously said they would prefer antenatal testing. Only one (0.5%) said the service was not good.

Table 6: Predictor Variables Associated with Acceptability.

	Acceptability					
Variable: N=200	N1	N2	%	OR	95% CI	P value
Knowledge that MTCT occurred during Labour and delivery.						
• Yes	101	86	85.1	0.3		
• No	99	67	67.7	65	(0.183,0.729)	0.004
Would have liked to be tested during antenatal period.						
• Yes	161	132	82.0	4.5		
• No	32	16	50.0	5	(1.90, 10.95)	0.013
• Not sure	7	5	71.4			
Not offered Counseling and Testing						
• Yes	99	86	91.2	3.3		
• No	101	67	66.3	6,	(1.56, 7.31)	0.022
• N/A	42	34	89.0			
Highly Acceptable						
• Yes	104	92	88.5	0.2		
• No	96	61	63.5	27	(0.107,0.472	< 0.001

Key: 1) N1: Total number of clients who responded positively to the specific variable.

2) N2: Those who accepted HIV testing.

Using the SPSS Computer Program various variables such as age, level of education, number of antenatal visits, parity, income, knowledge of MTCT and PMCT and reasons for not being tested in early labor were cross-tabulated with acceptance (dependent variable) to see whether there was statistical difference

between those who accepted to be tested and those who declined with regard to the specified predictor variable. There was no significant difference between accepting and declining clients regarding all other variables except on knowledge that MTCT occurred during labour and delivery (P=0.004), having not been offered CT in the antenatal clinic (P= 0.022), "highly acceptable" level of acceptability (P<.001) and having liked to be tested for HIV antenatally (P=.013). Table 6 illustrates these findings.

The results in Table 6 above indicate that clients who knew that MTCT occurred during labour and delivery were significantly more likely (85.1%) to accept testing than those without the knowledge (67.7%) OR .365 (95% CT .183- .729) P= .004. It also shows that women who wished to be tested during the antenatal period and clients who were not offered counseling and testing had significantly higher acceptability rates than their counter parts (P Value = .013 and .022 respectively). As would be expected study subjects (tested and untested) who indicated that counseling and HIV testing in early labour was highly acceptable, had significantly higher acceptability rate than those who said otherwise; OR .227 (95% CT, .107-.474) P value< .001.

To determine which predictor variable had the most significant association with acceptability of CT in early labour, the above four predictor variables were subjected to SPSS multivariate logistic regression analysis with acceptability as the dependent variable.

Table 7: Multivariate Logistics Regression Analysis

	Characteristics	Wald	P -Value	OR	95% CI
1	During labour and delivery	9.301	.002	.365	(0.183, .729)
2	Would have liked to be tested Antenatally	7.437	.006	____*	____*
3	CT is highly acceptable	15.541	< .0001	.227	(.107, .472)

- OR and 95% CI not possible to calculate because not a 2 X 2 data entry table.

Only the knowledge that MTCT occurred during labour and delivery, having liked to undergo antenatal HIV testing and the CT being highly acceptable remained significantly associated with acceptability of CT in early labour.

(P Values: .002, .006 and < .0001 respectively). The odds ratios and other parameters are indicated in Table 7 above.

Table 8: Socio-demographic Factors, Willingness to Disclose Own Serostatus and HIV Seroprevalence.

	Background Characteristics	Seronegative N 1	Seropositive N 2	%	OR	OR at 95% CI	P Value
1	Age in years						
	• 15 – 19	25	4	13.8			
	• 20 – 24	57	14	19.7			
	• 25 – 29	32	7	17.0			
	• 30 – 34	5	1	16.7			
	• 35 - 39	6	2	25.0	**	**	.946
2	Parity						
	• Nulliparous	61	15	19.7			
	• Higher Parity	64	13	16.9	.826	(.363,1.878)	.625
3	Marital Status						
	• Unmarried	14	7	33.3			
	• Married	111	21	15.9	2.095	(1.019,4.308)	.069
4	Level of Education						
	• =<Complete Primary	69	14	16.9			
	• =<Complete Secondary	46	12	20.7			
	• =Complete College/University	9	3	25.0	**	**	.769
5	Occupation						
	• None/Housewife	94	17	18.1			
	• Some Employment	59	11	18.6	.960	(.390, 2.420)	.898
6	Income per month (Ksh.)						
	• Less than 4,000	103	26	20.16			
	• More than 4,000	22	2	8.33	2.78	(.57,18.26)	.277
7.	Not Willing to Disclose Status	3/116	4	16.7	.129	(.027, .622)	.003

Key: N1 – Count of Seronegative women

N2 – Count of Seropositive women

**_Odds Raito not possible to calculate because not a 2x2 data entry table.

HIV seroprevalence

Out of 153 clients who underwent counseling and testing 28 of them were HIV seropostive, giving a sero-prevalence rate of 18.3%. There was no significant difference between seropostive and seronegative women with regard to all socio-demographic factors (Table 8). However, though not statistically significant there seemed to be an increased risk of HIV seropositivity with higher level of education (25%; college/university Versus 16.9% Primary) P= .769 and monthly income higher than Ksh. 4,000 (20.2% verses 8.3%) P= .277. Although unmarried women appeared to have higher (33.3%) seroprevalence rate than married ones 15.9%, this difference was not statistically significant; OR 2.095 (95% CT 1.019,4.308) P= .069.

Acceptability of Nevirapine

All the 28 seropositive women underwent post-test counseling after which a 200mg of nevirapine tablet was offered to each of them. Twenty-six (92.9%) of the 28 accepted to take the drug and actually swallowed it in the presence of the respective counselor. One participant declined to take the nevirapine tablet because "it was too late to take the drug for useful PMCT" but said she would administer the medicine to her infant. Another client cited uncertainty of safety of research drugs as her reason for declining uptake of nevirapine.

The only significant difference between HIV seronegative and seropositive clients was that HIV seropositive women were less likely to disclose their serostatus than their counterparts. (16.7% versus 2.5%) OR .129(.027- 0.622); P=0.003.

DISCUSSION

The main finding of this study was that the acceptability rate of counseling and HIV testing in early labour at Pumwani Maternity Hospital was high (76.5%). Of the 153 women who were counseled and tested 28 (18.3%) were seropositive 26 (93%) of whom accepted and actually took nevirapine for PMCT for themselves and their infants.

Majority (47.5%) of the women interviewed were aged between 20 and 24 years and were of low socio-economic status as indicated by majority (64%) being housewives and/or unemployed and most (86.5) earned less than a dollar per day. This population consisted of women in early labour most of whom attended Nairobi City Council public health centers for antenatal care. They were, therefore, similar to the study populations studied by Kiarie in 1996[6] and Malonza et al in 1999[48] in the East and West of Nairobi, respectively. Both investigators studied women attending NCC health centers on acceptability of pre-natal HIV testing using HIV ELISA and rapid HIV testing respectively.

The two investigators (Kiarie and Malonza) and this study found no significant difference between accepting and declining and between seropositive and seronegative women with regard to socio-demographic factors. In this study, however, acceptance was higher in clients who knew that MTCT occurred during labour and delivery (P=0.004), those who had not been offered antenatal CT (P=0.022) and those who would have liked to undergo prenatal testing (P= 0.013). As was the case with Kiarie's study most of our clients were aged between 20 and 24 years (47.5 vs. 51.4%), were married (86.5 vs. 79.4%) and of low parity (nulliparous women: 48% versus 43.2%).

The acceptability of prenatal (as opposed to intra-partum) HIV testing has widely been studied in Kenya and stood at 50 to 70% in early and mid nineties.[3,6] Recent studies[7,48] indicate that it (acceptability) has risen up to between 88% and 97.7%. Although the acceptability of HIV testing in early labour as found by this study was high and similar to acceptability of prenatal testing, it was slightly lower relative to prenatal testing. Moreover it is virtually the same as the acceptability rate of 76% among 4,303 pregnant and non-pregnant women eligible for CT during the 2003 Kenya Demographic and Health Survey (2003 KDHS).[49] This survey indicated that women in Nairobi had the highest categorical refusal rate of 22% that compares well to the refusal rate of 17.5% found in our study. Unlike the participants in our study, however, the 2003 KDHS study subjects were not in early labour, had a second chance of counseling and testing and the results of the tests were anonymous.

Factors that may have led to acceptability being similar but slightly lower than prenatal HIV testing probably include:

1. Lack of adequate knowledge on MTCT and its prevention modalities,
2. Inadequate time for participant to prepare for and accept testing,
3. Prior decision not to be tested and
4. Being in labour

Knowledge on MTCT and PMTCT was generally low relative to findings in other studies[7,29] For instance, while the 1998 KDHS found that 85% of women interviewed knew that MTCT is one of the routes of HIV transmission, our study revealed that only a modest 54% of study subjects spontaneously named the route of transmission (Figure 2). This level of knowledge could be explained in part by the way the questionnaire was administered. In this study, women were asked to name as many as possible the modes of HIV transmission (or otherwise) that they knew as opposed to asking them to indicate whether this mode or timing of HIV transmission was correct ("Yes") or not ("No"). Prompted or provoked knowledge and guessing were, therefore, likely to have been omitted; possibly leading to reduced knowledge.

The relatively low level of knowledge on PMTCT could also be explained by inadequate information, education and communication (IEC) by antenatal clinics visited by our clients as demonstrated by 80.5% of them having not been offered prenatal CT while 15% of them presumed they had been tested when their blood was taken for antenatal profile tests. In addition, our clients were of low socio-economic and education level compared to that of the general population surveyed by the 1998 KDHS team and the KNH clients studied by Kamau[7] whereby only 29.2% of the subjects were unemployed compared to 64% in this study.

That the level of knowledge my have contributed to relatively low acceptability rate is underscored by the fact that acceptance was significantly higher in women who knew that MTCT occurred during labour and delivery compared with those without the knowledge (P=0.004). Since this was not reflected in other aspects of knowledge it is possible that women accepted testing knowing that provision of nevirapine to them and their infants would be provided should they turn to be seropositive.

Nearly a half (44.6) of the 47 participants who were not tested were unprepared for the test (needed to tell spouse first (14.9%) were psychologically unprepared (19.1%) and needed time to decide (14.9%). Whereas prenatal HIV testing provides for repeated counseling, this was not possible in this study and this

probably led to the slightly low acceptance. Although being in labour was thought would be the main reason for declining testing, it was mentioned by only 5 (10.6%) of clients who declined to be tested.

Given that most (68%) of the reasons advanced for declining testing (afraid of knowing am seropositive and being unprepared) are issues that would be dealt with by prolonged counseling, intraparitum CT should be complimented by early postpartum (ePP) counseling and testing of parturients preferably with their spouses/partners. This will cater for parturients who were unprepared and wished to be counseled with their spouses/partners. It also indicates the kind of influence the partner has on decision making among the women in this study. Being unprepaired would probably be reduced if more PMTCT information, education and communication was provided by all health workers at all levels of obstetric care.

The effect of introduction of intrapartum CT of HIV on number of clientele seeking delivery services at the study site, though desired, could not be studied in this research. This would be a significant finding since studies [6,48] have indicated that CT of HIV testing may reduce the number of clients seeking antenatal services.

HIV Seroprevalence:

The HIV seroprevalence in our study population was 18.3%. Though higher, it is within the range of (10-20%) of HIV seroprevalence in Nairobi's pregnant women (50). Jericho, the nearest NASCOP sentinel surveillance center currently has HIV seroprevalence of 15% while Baba Dogo's is 18% [50]. Our seroprevalence, however, was lower than the prevalence of 21% found in 773 women [eligible (tested in early labour) and not eligible (tested postnatally) to this study] who concurrently underwent CT in the same study site. It is however, much higher than the most recent national seroprevalence of the general population and the national seroprevalence of pregnant women which currently stands at 7% and 9.4% respectively.[49]

This may be explained by the low socio-economic status and level of knowledge on HIV/AIDS that was noted in the study population. The high seroprevalence calls for enhancing PMTCT programmes in the NCC health centers.

There was no significant difference between seropositive and seronegative clients with regard to all other factors except on willingness to disclose one's HIV serostatus to the spouse/partner. The study showed that HIV seropositive women were significantly less willing to disclose their serostatus than the seronegative ones; OR .129 (95% CI .027 - .622) P=0.003. This is consistent with Muliro's study[51] done in Kitale

District Hospital (P=0.05). Age, level of education, parity, marital status, occupation and monthly income were not significantly associated with seropositivity. However, though not statistically significant, single women had a higher seroprevalence rate (33.3%) than married ones (15.9%). This is comparable to Kiarie's study[6] where there was no significant association of marital status and serostatus.

Of the 28 seropositive women the uptake and adherence of nevirapine offered to them and their infants was 93%. This figure validates findings of Kiarie et al[52] that showed that overall, more women were compliant with the HIVNET – 012 regimen than with the Thai-CDC regimen (87% versus 41%), P<. 001. Only two (7%) seropositive women declined to take nevirapine. One said it was too late and that in any case she would still breast-feed. Another participant said she did not trust drugs given in research studies. She was given a prescription of nevirapine syrup and referred to the hospital pharmacy or any other pharmacy or PMTCT providing institution of her choice. She did not turn up at the hospital pharmacy. Both clients declined to take nevirapine despite extended counseling.

CONCLUSIONS

1. The acceptability of rapid on-site counseling and HIV testing in early labour was high (76.5% and similar to acceptability of prenatal HIV testing found in other studies.[34,48]
2. Majority of the women studied were aged less than 25 years, married and had low soci economic status.
3. The knowledge of the study population on perinatal HIV transmission was low but similar that found in other studies in Kenya.
4. The HIV seroprevalence in women in early labour at Pumwani Maternity Hospital was high than the current national and Nairobi province HIV seroprevalence rates among women reproductive age.
5. The acceptability of nevirapine to HIV sero-positive women in early labour was quite high.
6. Acceptability was significantly higher in women who knew that MTCT occurred in peripartu period but there was no significant difference with regard to socio-demographic factors betwe women who accepted and those who declined HIV testing.
7. Both acceptability of HIV testing in early labour and uptake of nevirapine were significant higher among women with knowledge of peripartum HIV transmission and those who h previously not been offered counseling and testing.

RECOMMENDATIONS

Based on the study findings, I would recommend that;

1. Intrapartum counseling and HIV testing be included as part of a continuous and wider PMT programme that includes prenatal and postnatal care.
2. There's need to enhance VCT and PMTCT programmes in the N.C.C health centers and Pumwani Maternity Hospital.
3. Institutional or field efficacy of the HIV NET- 012 as used in this study be studied farther since t was limited by financial constraints.
4. The effect of introducing intrapartum CT for HIV on the number of women seeking delive services at Pumwani Maternity Hospital be studied, and
5. Compulsory HIV screening on all pregnant women be considered as part of PMTCT programme

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GYNAECOLOGY CASE NUMBER 1

AMBIGUOUS EXTERNAL GENITALIA-CONGENITAL ADRENAL HYPERPLASIA

NAME : F.K LMP: Primary Amenorrhoea

AGE : 18 YEARS DOA: 28/6/2004

Parity : 0+0 DOD: 5/08/2004

File N^{o} :0966604

Presenting Complaints:

F.K presented with monthly low abdominal pain for 1 year and associated lack of menses since s attained puberty.

History of Presenting Complaints.

She had been well till about 1 year ago when she developed colicky, lower abdominal pains that occurred intervals of 28 to 30 days. The severity of the pain ranged from moderate to severe, crampy, suprapul pain that occasionally forced her to prostrate and stopped her from going to school. Pubarche was at years. She had never had menarche. She had noted that her facial hair had receded, grown masculine bo built and her voice was tenor as from 16 years. Although she had been brought up as a girl, she beca aware of her abnormal genitalia at 15 years. She had been approached by boys but was not interested in s at all, and, therefore, declined history of sexually transmitted infection, coitus or other sexual activity.

Past Obstetric and Gynaecologic History was as above

Past Medical History:

She underwent clitoridectomy by a surgeon at KNH at about 3 months old and NO medication w prescribed for her. There was no history of features of Addisonian crisis.

Family and Social History.

F.K. is a first born among five orphans. Both Parents died when the patient and her siblings were under years. She and her siblings have been staying with their grandmother in their rural home in Kisii.B parents were reported to be taller and lighter in colour than she is and so were her siblings. Her 16 and year-old sisters attained menarche at 14 and 13 years respectively. Both her 13-year-old brother and year old sister had no symtoms similar to hers. None of her clinical features have been noted in extended family, which has no history of chronic illness.

PHYSICAL EXAMINATION

General examination

She was in a good general condition with a height of 143 cm, weight of 43 kg and dark-skined. She had BP of 100/60 mmHg, temperature 36.8^{0} C and PR of 92 Bpm.She had no acne but her voice was tenor.H general stature was masculine as characterized by broad shoulders, receded hairline, masculine muscles a presence of fine hair on the upper lip that appeared like a poorly developed moustache. She also had a ma pattern of coarse pubic hair (male escutcheon).

Pelvic and Rectal Examination

She had no clitoris. Her labia majora were larger than usual and appeared like scrotal skin. The lab minora were not visible. The external urethral meatus was more posterior (lower) than usual. Only t vestibule was visible as there was no vaginal opening. Small nodular masses were palpable about ovari fossa (? gonads) on rectal examination but no uterus was palpable. There was bogginess (? haematocolpo anteriorly, but mild tenderness was observed on bimanual examination.

Respiratory, abdominal, cardiovascular and the nervous systems were essentially normal.

Impression

An impression of ambiguous external genitalia possibly due to congenital adrenal hyperplasia was made.

MANAGEMENT

F.K was given a brief appraisal of the suspected pathology and due to her financial constraints, w admitted for investigations and multi-disciplinary consultation and management.

Investigations

1.Haemogram : hemoglobin: 15.9g/dl

: WBCC: 5.15 x 10^{9}/l

: Platelets: -229x10^{9} /l

2.Renal Function test: Sodium –146mmol/l

: Potassium-4.6mmol/l

: Urea- 4.7 mmol/l

:Creatinine- 91μmol

3. Abdominal-pelvic ultrasound:

: Normal bladder

: Uterus, antevertted, normal sized, normal echogenicity and without anomally, both "ovaries" were visible and appeared normal and there was marked fluid in the P.O.D. Visible hyperplastic adrenal glands not usually visible; normal viscera.

4.Serum: a). cortisol____7.30 a.m: 130.6nmol/l (116-1060)

____7.30p.m: 40.22nmol/l(47-458)

b). Oestradiol- 23 pg/ml (Normal for male upto 60 pg/ml)

c). Testosterone_40.22 (Normal range for 8.09 –36.7nmol)

d). Progesterone —6.0 (Normal male –upto 2.9ngml)

e). 17 hydroxyprogesterone- 94.0nmol/L Normal ranges:

Follicular phase -(0.4-2.1 nmol)

Luteal phase------(1.0-8.7 nmol/L)

e). Dehydroepiadrosterone-12.8μmol/L (3.93-10.72)

6.Bar body-11% (Normal female range 9-36%)

7.Diagnostic laparoscopy and punch biopsy: -

-Rudimentary and flappy uterus with grossly normal healthy fallopian tubes.

-Bilateral gonads that appeared like ovaries.

-Histology of punch biopsy of gonads, though inadequate, was highly suggestive of ovarian tissue.

8.Urinalysis - Normal findings.

Diagnosis

Based on the aforementioned history, physical examination and laboratory findings the conclusive diagnosis of congenital adrenal hyperplasia with resultant ambiguous external genitalia was made.

TREATMENT.

This was multidisciplinary involving, gynaecological, endocrinological and urological consultants, counselors the guardian and the patient and all other health workers. The objective was to maintain and reinforce the gender identity and role and establish feasible and acceptable gender orientation. Upon endocrinologist review, the patient was started on prednisolone tablets: 5mg to be taken in the morning and 2.5mg in the evening, 12 hours apart. Mefenamic acid capsules were to be taken whenever there were crampy lower abdominal pains. The doctors and the counselors from the Hospital provided counseling to the patient alone and together with the guardian. The options available for her treatment were discussed at length and F.W. opted not to undergo vaginoplasty. She was advised to consider other options (discussed under 'Discussion') as an outpatient on follow-up.

DISCUSSION

The Patient presented was an 18 year old with typical features of congenital adrenal hyperplasia with resultant ambiguous external genitalia, primary amenorrhoea and virilism.

The human gender or sex is determined by the genetic sex (46XY: male, XX: female), gonadol sex (ovary: female, testis: male) and the phenotypic appearance of the external genitalia (vulva: female, penis and scrotum with testis: male). At birth, gender assignment is primarily based on the appearance of the external genitalia. When an individual's external genitalia is not clearly formed (is ambiguous) and/or the genetic, gonadol or genital sex are not tallying then the individual is said to be a hermaphrodite or having intersexuality.[1] A true hermaphrodite posseses both ovarian and testicular tissue. A male pseudohermaphrodite has testes but external and (occasionally) internal genitalia take on female phenotypic aspects. A female pseudohermaphrodite has ovaries but genital development display masculine characteristics.[1,2] The classification of gonado-genital abnormal development and some of their causes is as follows:

I. Disorders of Fetal Endocrinology.

1.*Female pseudohermaphroditism (partial virilization) congenital adrenal hyperplasia (CAH)*

- P45021a –hydroxylase deficiency (CAH-type I & II)
- P45011B-hydroxylase deficiency (CAH type III)
- 3B- hydroxysteroid dehydrogenase (CAH type IV)
- Drug intake (Progestagens, danazol, androgens)
- Maternal disease (adrenal tumor, arrhenoblastoma placental aromatase deficiency).

2.*Male Pseudohermphroditism (inadequate vilization)*

- Antimullerian hormone defect
- Impaired androgenization.
- *Androgen insensitivity syndrome*
- 5-reductase deficiency
- Testosterone biosynthesis defects
- 3B-hydroxysteroid dehydrogenase (CAH type IV)
- P450-Side chain cleavage enzyme (desmolase) deficiciency (CAHtype VI)
- P450 17α –hydroxylase deficiency (CAH type V)
- 17β-hydroxysteroid dehydrogenase deficiency

II Disorders of Gonadol Development

- *Male pseudohermaphroditism:*
  - Primary gonadal defect
  - Y chromosome defect
- True hermaphroditism
- Gonadal dysgenesis
- Tunners' syndrome
- Mosaicism
- Structural abnormally
- Normal karotype.

Genetic sex is determined at fertilization while phenotypic sex is dependent on absence or presence of unimpaired hypothalamo-pituitury-testis-end-organ, paracrine and endocrine systems. Complete development of the female reproductive organs, therefore, depends on the genotype (46,XX) and the absence of the testicular (male) paracrine and endocrine substances.[1,2,3,4] The promordia of the female reproductive organs consists of the indifferent gonad (formed by the sixth week), the paramesonephric (Mullerian) ducts, the urogenital sinus, the sinus tubercle, the phallus, the urogenital folds and labioscrotal swelling.

In the absense of the sex-determining region of the Y-chromosomes (SRY) that codes for testis differentiating factor (TDF), the indifferent gonads develop into ovaries. Consequently there will be no Mullerian inhibiting factor (MIF) leading to the development of the paramesonephric ducts into fallopian tubes and (by fusion of the tubes) the uterus. Similarly, absence of androgens (testosterone) leads to development of the urogenital sinus into the lower two thirds of vagina and the sinus tubercle forms the hymen. The phallus forms the clitoris; the urogenital folds develop into the labia minora and the labiosrotal swelling form the labia majora.

Congenital adrenal hyperplasia (CAH) arises due to deficiency of any of the six enzymes involved in the synthesis of cortisol. Deficient cortisol production lead to excessive adrenocorticotrophic hormone (ACTH) production leading to suprarenal (adrenal) gland hyperplasia. There are six major types of CAH all transmitted as autosomal recessive disorders as shown above. Both males and females can be affected but males are rarely diagnosed at birth unless they have ambiguous genitalia, are salt losers and manifest with adrenal crises or are screened because they have an affected sibling. Defects of type I-III are confined to the adrenal gland and produce virilization of the female i.e. congenital virilizing adrenal hyperplasia (CVAH).

They cause female pseudohermaphroditism. On the other hand defects of the type III-IV have in common blocks in synthesis of cortisol and sex steroid synthesis in both the adrenal and the gonads. The latter three types produce chiefly incomplete masculinization in the male and little or no virilization in the female. They cause male pseudohermaphroditism.[1,2,3,4]

Our patient had clinical features of congenital virilizing adrenal hyperplasia both at birth (hence the erroneous clitoridectomy) and at the time she presented to us. She had no history of adrenal crises and her renal function tests were normal. Although karyotyping was not done the 11% of bar bodies on mucosal cells, ovarian biopsy indicating ovaries and the laparoscopic findings of normal oviducts and rudimentary uterus all pointed to a female genotype. Further, the markedly elevated 17-hydroxy-progesterone and androgens and the lower limit level of cortisol suggested 21-hydroxylase deficiency. Our patient, therefore, had type I CAH and the attendant female pseudoherphrodism due to 21-hydroxylase-enzyme deficiency.

The degrees of deficiency of 21 hydroxylase is variable depending on the type and extend of mutation of the gene coding the enzyme. This gene is located on the short arm of chromosome 6 close to the HLA supergenes. The highly variable mutations result in phenotypic spectrum that range from late onset of virilization or simple congenital virilization (type I) to variable degrees of salt losing CVAH (type II). The commonest (75%) type of mutation is point mutation or micro gene conversion. The remainder (25%) have gene deletion or macrogene conversion.[2] Overall, the prevalence of 21 hydroxylase (type II) deficiency is 1:14,000 live births in Caucasians.[3] However, others studies have postulated that the "nonclassic", non-virilizing 21-hydroxylase deficiency is the most common autosomal recessive disorder, affecting one in 100 persons of all ethnic groups, but having an incidence two to three times higher in Hispanics and Ashkenazi Jews.[2,3] Attempts to get prevalence in our set up and in blacks were futile.

In type I CAH (without salt loss), 21-hydroxylase deficiency leads to impaired cortisol synthesis, increased ACTH, increased androgen precursers (e.g. DHEA & 17-hydroxy-progesterone) and increased androgen secretion. Left untreated, the high androgen levels lead to rapid growth and bone maturation and physical signs of excess androgen secretion. These features were found in our patient whose height was 143 cm and had masculine features all due to failure of early treatment.

The degree of musculization of the external genitalia depends on the onset of the secretion of excessive androgens. Beyond 12 weeks after fertilization, excess androgens result in only clitoromegally while onset before 12 weeks produces varying degrees of labioscrotal fusion.[1]The onset of hyperandrogenmaemia in our patient was earlier than 12 weeks because she had no vagina and urogenital sinus was not visible.

Other causes of female pseudohermaphrodism include 11-hydroxylase deficiency (type III of CAH), musculinizing maternal adrenal tumours (e.g benign adrenal adenoma) and ovarian tumours especially arrhenoblastomas.[4] Exogenous androgen and excessive progestogens used for treating threatened abortion have resulted in female pseudohermaphrodism.

To achieve comprehensive and optimal results, treatment of CAH should involve gynecologists (primary physician), endocrinologist, urology surgeon, pediatrician, psychologist, sexicologist, the parents/guardians and all other health workers including a genetic specialist when available. Broadly the treatment is medical, surgical and psychological. Gender assignment soon after birth requires proper diagnosis based on comprehensive history, physical examination and appropriate laboratory and imaging tests. Corrective treatment should then be instituted once the diagnosis has been made to avoid progression of the disease and its sequelae and to enhance development of gender identity, role and orientation.

The treatment is divided into acute and chronic phases depending on the type of CAH. Adrenal crisis due to deficiency of both glucocorticoids and mineralocorticoids (aldosterone) requires quick assessment of eloctrocyte and glucose levels and correction of any type of hypokalaemia, hypoglycaemia and replacement of fluids in the presence of shock with saline. The chronic phase entails the supply of deficient hormone. Cortisol, or its equivalent synthetic glucocorticoid, is the primary treatment of CAH.The Drug of choice is hydrocortisone (approximately 10 mg/day). Predrisone at a dose of 3.5-5 mg/m^2 is the alternative.

For type II and III (salt losing) CAH, fludrocortisone is used to replace the deficient aldosterone.[1,2] Corrective surgery is dependent on the age at diagnosis, the gender identity and role, and the type/degree of ambiguous genitalia. Plastic surgery for ambiguous externatal genitalia should be performed before one year of age. Clitoroplasty rather than clitoridectomy is the preffered corrective surgery on hypertrophied clitoris. Unfortunately our patient missed both treatment options. Clitoridectomy was done at 3 months and she did not get glucocorticoid replacement until she was 18 years. Clitoroplasty is performed as per Rajfer technique where a mid portion of clitoral corpora is excised through posterior incision and glands anastomosed to the proximal stump of corpora.[5]

Vaginoplasty is best performed at puberty when mature compliance is possible and the pelvic structures are bigger. The type of corrective surgery depends on the position of the vagino-sinus. The position of vagino-sinus and the respective corrective surgery are summarized on the table below.

Position of vagino-sinus	Corrective surgery
1.When vagina is present.	With urethral catheter in situ, identity vagina; incise the urogenital sinus to within 2-3 cm from anus, free vagina posteriorly and laterally then attach to the edges of the skin. Do clitoroplasty
2. When the vaginal orifice is obscured.	Two stages (a). First clitoroplasty and incision of urogenital sinus. (b). Second vaginal orifice identification mobilization and exteriorization.
3.When the vagino-sinus communication is deep.	Disconnect the urethra from the vagina and reposition the vagina in the perineum. Serial manual dilatations.
4.When the vagino-sinus communication is blind without urethral communication.	Laporatory, hysterotomy, uterine sound in blind vagina, reposition vagina in perineum as above. Serial manual dilatations.

Our patient has obscured vagino-sinus communication and, therefore, could be having urethrovagina communication or a blind vagina, which then requires urethroscopy and type 3 or 4 reconstructive surgery.

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GYNAECOLOGY CASE NUMBER 2

CERVICAL CARCINOMA IN SITU - EXTENDED HYSTERECTOMY

Name: G. M. M.	LNMP: 21/05/04
Age: 35 years	DOA: 31/05/04
File No:0920956	DOD: 07/06/04
Parity: 4+0	

Presenting Complaint

G.M.M. was admitted through the Gynaecological Outpatient Clinic (GOPC) with a 7-month history of post-coital per vaginal bleeding, dyspareunia and offensive vaginal discharge.

History of Presenting Complaint

G.M.M was well until 7 months prior to admission when she developed progressive-dyspareunia and associated post-coital vaginal bleeding. She also developed offensive vaginal discharge that was brownish in colour and would ensue predominantly two to three days after coitus. Her menstrual cycle remained unaltered. She had no urinary symptoms nor lower abdominal pain or distension. She, therefore, sought medical advice in our GOPC.

Past Obstetric and Gynaecologic History

The patient was para 4+O. All her deliveries, including the last in 1993, were by spontaneous vertex delivery and were uneventful. Her menarche occurred at 15 years. Her last menstrual period was on 21/05/04 and her menses occurred every 28 days and flowed for 3 to 5 days. Her coitarche was at 18 years and had had 7 different sexual partners in her lifetime. She had used combined oral contraceptive pills in between pregnancies uneventfully and denied history of any sexually transmitted infection. A Papanicollaou smear had been done. It was haemorrhagic and unsatisfactory. A repeat was requested but in view of suspicious history colposcopy was offered instead.

Past Medical History

This was not significant

Family and Social History

G.M.M was a married businesswoman who lived with her family in one of the Nairobi suburbs. She did not consume alcohol neither did she smoke tobacco. She had no history of familial disease. Her husband did business with her. She believed they had been faithful to each other in their 12-year marriage.

PHYSICAL EXAMINATION

General examination

She was in good general condition and nutritional status, without pallor, jaundice lympahdenopathy or oedema. Her BP was 120/75mmHg, pulse rate 80/min, and temperature: 36.5°C.

Abdominal Examination

The abdomen was slightly obese moved with respiration; was soft and had no palpable masses.

Pelvic Examination

On Cusco's speculum examination she had normal female external genitalia and her vaginal mucosa was healthy. There were hyperaemic patchy areas at 12, 3-5 and 6-8 o'clock without any obvious malignant lesion. Digital examination showed a normal sized, mobile antevertted uterus and free adnexae and pouch of Douglass. There was no abnormality on rectal examination.

Respiratory, Cardiovascular, Mucosculoskeletal and Central Nervous Systems were normal.

Colposcopic Examination and Biopsy

Cervical staining with acetic acid showed dense acetowhite patches at 12, 3 – 5 and 6 – 8 o'clock. Schiller's test was positive. Histology of colposcopically directed biopsy revealed mucosal cells that had lost polarity, with frequent mitotic figures and hyperchromasia in all the mucosal layers. Although microinvasion could not be ruled out it was not observed.

Impression.

An impression of cervical carcinoma in situ/severe dyspalsia/high grade squamous intraepithelial lesion (HSIL) with possible micro-invasion.

Management

The patient was appraised on the results and the possible treatment options. These options included loop electroexcision procedure (LEEP), cone biopsy and extended total abdominal hysterectomy. She opted for the later and investigations in preparation for the surgery were as follows:

1. Renal Function Tests:

Sodium	137mmol/L
Potassium	3.8mmol/L

Urea	6.1mmol/L
Creatinine	72mmol/L

2. Haemogram:

Hb	13.2g/dl
WBCC	$3.6x10^9$/L
Platelets	$230x10^9$/L

3. Intravenous urogram (I.V.U)- Normal outline of kidneys, ureters and bladder.
4. HIV ELISA – Negative

Extended Hysterectomy

Upon admission informed consent was obtained from the patient in the presence of her husband. One pint of blood was drawn from her and blood taken for grouping and cross-match of an extra pint. A low fiber diet was instituted. Enema and 1gm of metronidazale were given at 6 p.m on the preceding day and enema repeated at 5.30 a.m. Shaving of pubic hair occurred 1 hour before surgery and pre-medication with intra-muscular 0.6mg of atropine and 50mg of pethidine half an hour later.

In theatre general anaesthesia was administered. In semilithotomy position, aseptic catheterization was done then examination under anaesthesia. This revealed a normal sized anteverted uterus that was freely mobile, free adnexae and pouch of Douglass (POD). Rectal examination showed no palpable masses or nodes and the rectal mucosa was not adherent to the rectovaginal septum. The patient was put in supine position, cleaned, draped and then opened through a subumbilical incision. The findings were grossly normal ovaries, tubes, uterus and abdominal viscera. Extended hysterectomy type II was then done. This included double clamping, incision between clamps and ligation of the stumps of the round ligaments, the fallopian tubes and the ovarian ligaments bilaterally. The pelvic peritoneum was then opened to expose the parametrium, the pelvic vasculature and the pelvic fascia including the sacral and the cardinal ligaments. No extension of the tumor outside the cervix or parametrial tumor involvement was noted. The pelvic lymph nodes were dissected and found to harbor no obvious tumor involvement. The uterine artery was ligated where it crossed the ureter and the uterosacral and cardinal ligaments were divided midway towards their attachment to the sacrum and the pelvic sidewall. The upper 3rd of the vagina was resected and vault closed and peritonized. Peritoneal lavage with normal saline was done and the abdomen closed after accounting for the equipment used. The indwelling Folleys catheter that drained clear urine was to be

removed in 12 hours. The reversal of the general anaesthesia was uneventful. The estimated blood loss was 500 mls.

Postoperative Care

The patient's vital signs were observed ½ hourly until she was fully awake then 4 hourly. She was started on intravenous antibiotics (crystalline penicillin, gentamicin and metronidazole) and fluids and pethidine for pain. On the first postoperative day she was in good general condition with normal vital signs, mild pallor and no major complaint. The chest was clear. The abdomen was soft, the wound dressing was clean and the bowel sounds were present and normal. The urine draining from the urethral catheter was clear and adequate. There were no signs of deep venous thrombosis. She was therefore, started on oral medications and analgesia, mobilsed and oral sips to graduate to light diet by evening started. The catheter was removed. By the 3rd postoperative day the wound was clean and she had passed stools. She was discharged on the 4th day with instruction to have the stitches removed by the 7th day. She was to be reviewed in 2 weeks time.

Follow up

GMM was well 2 weeks later when she was seen in the gynaecology outpatient clinic. The wound had healed welland vaginal examination revealed no abnormal discharge. The histology report indicated that she had moderately differentiated stage IA_2 microinvasive squamous cell carcinoma of moderate malignancy. No lymph node involvement was noted. The importance of this report was communicated to her and she was asked to strongly adhere to subsequent follow up with vaginal vault Pap smears every 3 months until she was negative for 4-6 times.

DISCUSSION

The patient presented was a 35-year-old para 4 + 0 who underwent successful extended hysterectomy for preoperative diagnosis of severe dysplasia but was found to have microinvasive squamous cervical cancer stage $1A_2$ on histopathological examination. Her postoperative recovery was uneventful and by the time of writing this commentary she was doing well.

Globally, cancer of the uterine cervix is the second most common cancer in women after breast cancer.[1,2,3] In 2000, it was estimated to be afflicting 1.4 million women. The estimation indicated that 470,000 new cases and 230,000 deaths occurred annually worldwide.[1] Eighty percent of these cases occurred in developing counties. The "tragic" situation of cervical cancer in Africa has remained unchanged for the two decades. The disease prevalence remains high,[2] still affects young women,[3] treatment facilities remain less optimal and a new burden in form of HIV has been added.[4] The national incidence of cervical cancer in Kenya is unknown as there is no population based cancer registry. However, it is estimated that the incidence is between 37 and 47 per 100,000 women per year.[2] Based on unpublished data of reported cases obtained from national cancer registry, between 2000 and 2003, cervical cancer was the second commonest (8.1%) cancer in both sexes after cancer of the breast (13.8%). It was also second (14.8%) to breast cancer (23.6%) in women. At KNH, between 1991 and 2000, 4836 cancer cases were reported/recorded. Of these, cervical cancer was the commonest (21.2%) reported/recorded cancer in both sexes ahead of skin (9.5%), breast (9.2%), esophagus (8.9%), lymphoma (6.6%), postnasal space (4.3%), stomach (3.6%), lips and oral cavity (3.1%), eye (3.0%) and prostate (2.7%). This was in big contrast to data obtained from Nairobi Hospital where out of 616 cancer cases in both sexes, cervical cancer was the 12th commonest cancer (2.1%) while breast cancer was the commonest accounting for14.5%.

Previous studies on invasive cervical cancer (ICC) in Kenya, have shown that the mean age at presentation was 42-45 with peak age being 40-49 years[3,5] However, Gichangi's study on the impact of HIV infection on invasive cervical cancer in Kenyan women indicated that at presentation, HIV seropositive patients with ICC were 10 years younger than HIV seropositive ones (40 vs 50 years, $p<0.001$) suggesting that the overall age at presentation may have gone down with the advent of HIV/AIDS.[4]

Cervical neoplastic disorders range from cervical intraepithelial neoplasia (CIN, dysplasia, squamous or glandular cell intraepithelial lesion) I, II, and III to microinvasive, macroinvasive and metastatic cancer.

With regard to aetiology, cervical cancer has been regarded by some as a sexually transmitted disease because of the recognition of the causal role of the sexually transmitted aetiologic agent, the human papilloma virus, and the association of the disease with sexual behaviour.[3,4,6] The risk factors for cervical cancer include early coitarche, having multiple sexual partners either directly (self) or indirectly (through regular sexual partner), history of sexually transmitted disease (STDs), immunosuppression, HIV and Herpes simplex II virus infection, multiparity, long term oral contraceptive use, low socio-economic status and tobacco smoking/chewing/sniffing. Our patient's risk factors were multiparity, 7 multiple sexual partners, relatively early coitarche and use of oral contraceptives for about 5 years.

The human papilloma virus (HPV) as an oncogenic factor of the cervix exists in 70 subtypes, half of which infect the anogenital region.[3,7] Based on their malignant potential, HPV is classified into low-risk HPV types (6,11,42,43,44), which are associated with condylomata and low grade lesions, intermediate-risk HPV types (33, 35, 51, 52) commonly found in CIN II and III and high-risk HPV types (16,18,31 39, 45, 56, 58, 59 and 68) that are associated with both CIN II,III and invasive cancer.[3,4,7] HPV is epithelotropic and becomes either asymptomatic or actively infectious or undergoes neoplastic transformation by incorporating its DNA into the human DNA. This transformation lead to upregulation of oncogenes E_6 and E_7 which then disable the tumour suppressor genes p53 and Rb.[7] The human immunodeficiency virus (HIV) on its own or, more commonly, in conjunction with HPV has been shown to cause the development and/or rapid progression of CIN to frank cancer and mestastasis of the cervical cancer into unusual places.[3,4,7] It is because of this that since 1993 invasive cervical cancer has been included as an AIDS defining cancer alongside Kaposi sarcoma and non-Hodgkin's lymphoma (types:high grade immunoblastic, small non cleaved and primary non-Hodgkin's lymphoma of the CNS)

With regard to sexual behaviour, many studies including one carried out by Were[8] (2001) at the Moi Teaching an Referral Hospital, Eldoret, Kenya, have out found that a woman with one sexual partner could get HPV (and other STDs) through a regular partner with multiple sexual partners.[3,4,7] Our patient was HIV sero-negative and the HPV status could not be established due to lack of resources.

Cervical cancer is preceded by premalignant lesions that are classified into CIN I (or mild dysplasia or low grade squamous intraepithelial lesion-LGSIL), CIN II (moderate dysplasia or part of high grade squamous intraepithelial lesion- HGSIL) and CIN III (severe dysplasia or HGSIL or carcinoma in situ- CIS).[7] The CIN classification depends on the proportion of the epithelium affected by the dysplasia. In CIN I the lower third of the epithelium has disordered growth (dysplasia). In CIN II and III the dysplasia has affected the

lower 2/3 and the entire epithelial thickness respectively. In America and indeed the world over including Kenya, the Bethesda Reporting System[8] has been advocated. It consists of adequacy of smear, general categorization, descriptive diagnosis and epithelial abnormalities. It involves the term squamous intraepithelial lesion to encompass all the grades of CIN. It is further divided into low grade (CIN I) and high grade (CIN I and III). Part of the Bethesda Reporting System is shown below.

BETHESDA CLASSIFICATION

1. Within normal limits
2. Infections (organisms should be specified)
3. Reactive and reparative changes
4. Squamous cell abnormalities:
    a) Atypical squamous cells
        1) of undetermined significance (ASC-US)
        2) cannot excude HSIL (ASC-H)
    b) Low-grade squamous intraepithelial lesion (LSIL): include HPV/mild dysplasia/CIN I.
    c) High grade squamous intraepithelial lesion (HSIL): include moderate and severe dysplasia, CIN II and CIN III/CIS
    d) Squamous cell carcinoma
5. Glandular cell abnormalities
    a) Atypical (AGC): endocervical cells, endometrial cells or glandular cells not otherwise specified (NOS)
    b) Atypical favour neoplastic: endocervical cells or glandular cells NOS.
    c) Endocervicl adenocarcinoma in situ (AIS)
    d) Adenocarcinoma: 1) endocervical 2) endometrial 3) extrauterine 4) not otherwise specified (NOS)
6. Other malignant neoplasms (specify).

Based in this classification our patient was classified as having HSIL on the Pap smear cytology. However, the histological report of the excised cervix showed microinvasive cervical cancer.

Since its introduction in 1940s by Papanicolaou[10], the Papanicolaou cytological test has been and remains the gold standard method of cervical cancer screening. Besides the Pap smear, new cervical cytological techniques that aim at increasing the reliability of the screening have been developed. These include the Thin Prep and the Auto pap 300 QC. The Thin Prep method is a fluid based method whereby cervical cells

are filtered of blood, mucus and inflammatory cells before they are read. The Auto pap 300 QC is an automated (machine) screening device. The use of pap smear screening test in developed countries has shown that cervical cancer is preventable. Prevention of cervical cancer is either primary or secondary. Primary prevention entail reducing the exposure to the risks of cervical cancer alluded to above while secondary prevention involve the use of screening tests such as the pap cytology test.

Inadequate screening programs and other factors in developing countries including Kenya has led to most women presenting in advanced stages of cervical cancer.[3,4,5] Because of the inability of the developing countries to afford population based pap smear screening programs, studies are ongoing in Kenya that aim at introducing relatively cheaper use of direct visual inspection (DVI) and/or visual inspection after application with acetic acid (VIA). The sensitivity and specificity of these methods need to be tested before they are incorporated into programs. Colposcopy and the use of a gynoscope can be used for both screening and diagnosis. The patient presented had not had a pap smear done until she developed symptoms of cervical cancer. She underwent colposcpic evaluation and biopsy which showed HSIL.

About 70-75% of cervical carcinomas are squamous cell; the remainder are adenocarcinomas (20-25%), adenosquamous carcinomas (3-5%) and undifferentiated ones.[7]

The symptoms of cervical cancer include abnormal vaginal bleeding, foul-smelling disharge, postcoital bleeding and pelvic pain and stool or urinary incontinence in advanced cases. Signs include cachechia and anaemia in advanced cases, grossly normal or barrel shaped or a typical cauliflower, fungating easily friable lesion on the cervix. The cervical lesion can be exophytic, ulcerative or endophytic. Other features depend on the disease stage as per the Federation of Gynaecology and Obstetrics (FIGO) of 1995 outlined below.

Stage 0: Carcinoma in situ, CIN III, pre-invasive carcinoma

Stage I: Carcinoma confined to the cervix

IA: Invasive cervical cancer diagnosed by microscopy only

a) IA_1: stromal invasion no deeper than 3mm, no wider than 7mm in horizontal spread

b) IA_2: stromal invasion > 3mm but <5mm and no wider than 7mm horizontally

IB: Clinically visible lesion confined to the cervix or microscopic disease > stage IA

a) IB_1: lesion not greater than 4 cm

b) IB_2: lesion greater than 4 cm

Stage II: Tumour extends beyond the uterus but not pelvic side wall or lower third of vagina.

IIA: Vaginal involvement without parametrial involvement

IIB: Parametrial involement

Stage III: Tumour extends to pelvic sidewall and/or cause hydronephrosis and/or extends to lower 1/3 vagina

IIIA: Involvement of the loer 1/3 of vagina without pelvic side wall or hydronephrosis

IIIB: Extension to pelvic siewall and/or hydronephrosis

Stage IV: Extension beyond the true pelvis or into mucosa of rectum or bladder

IVA: Extension into adjacent organs

IVB: Distant metastases

Our patient had microinvasive cervical cancer stage IA_2

Management of premalignant lesions of the cervix depends on the degree of neoplasia (ASC-US or HSIL the patient's ability for follow up, presence of high risk factors (e.g. HIV seropositive or high risk HPV an desire for fertility. Ideally if the pap cytology is ASC-US then the patient should have repeat cytolog every 6 months for 1 year. If normal, routine pap smear cytology is done. If abnormal then colposcopy i done. Patienst with HSIL need colposcopic evaluation. If colposcopy is satisfactory and indicates ASC-U expectant management or cryotherapy is done. But if there is ectocervical HSIL cryotherapy or lase ablation or loop electroexcision procedure (LEEP) is done. On the other hand if there is endocervical lesio or ectocervical lesion with positive endovervical curretings (EEC) or colposcopy is unsatisfactory or i there is discrepancy betweewn colposcopy and the high grade cytology or if microinvasion is suspecte then either cold knife cone biopsy and EEC or deep LEEP and EEC are performed. If the findings of th biopsy are HSIL then 3 monthly pap smears and 6 monthly EECs are done for 1 year then routine follo up. If the biopsy indicates microinvasion then individualise management as per desire for fertility. In a levels if there is invasive cancer then definitive therapy is provided. Because of the limited resources i Kenya, this protocol is not feasible and the protocol has been modified to suit our set up. Our patient ha completed her desired family size, had clinical presentation suggestive of cervical cancer, Pap smea cytology of HSIL and colposcopy was suspicious of microinvasion. Hence the management by extende TAH.

Although the overall 5-year survival rates for surgery and for radiation in patients with operable early stag (IIA2 to IIA) cervical cancer is approximately equal, surgery has the advantage that ovaries can b transposed, proper staging of the disease is better intraoperatively with pelvic lymphadenectomy an vaginal stenosis caused by radiation is avoided. Radical hysterectomy and pelvic lymphadenectomy as firs described by Wertheims (and modified by Meigs and Okayabashi) was less extensive as is performe

today. Wertheim's hysterectomy involved the removal of only enlarged and palpable lymph nodes and only the medial half of the cardinal ligaments.[11] Five types/classes of extended/radical hysterectomy have been described for various stages of cervical cancer.[7,11] Type I hysterectomy, indicated for patients with stage IA1 involves extrafascial hysterectomy with removal of all cervical tissue without dissecting into the cervix itself. Type II hysterectomy (IA_2) involves the ligation of the uterine artery where it crosses over the ureter, division of uterosacral and cardinal ligaments midway towards their attachment to the sacrum and the pelvic sidewall and the removal of the upper third of the vagina. In type III (modified Wertheim's/radical hysterectomy meant for stage IIA cervical cancer, the uterine artery is ligated at its origin from superior vesical or internal iliac artery,the uterosacral and cardinal ligaments are resected at their attachment thorough pelvic lymphadenectomy and the upper ½ of the vagina is removed. In our set up limited resources limit the use of LEEP or cone biopsy for staging into IA_1-IIA first before deciding the type of hysterectomy to perform. Hence most patients undergo type III hysterectomy irrespective of the stage. Our patient underwent type II hysterectomy for HSIL but retrospectively this was a right decision since histology indicated that she had stage IA_2 cancer.

Patients with stage IIB and above are best treated by primary radiotherapy. Unfortunately this facility is only available at KNH and a high cost private hospital in Nairobi in Kenya. Primary radiotherapy with concomitant cisplatinum-based chemotherapy has been shown to be superior to radiotherapy alone.[7,8] Radiotherapy is also used as adjuvant therapy after surgery and palliative therapy to control bleeding.Other treatment modalities of cervical cancer include primary chemotherapy for stage IVB disease, pelvic exentaration for isolated pelvic recurrence and importantly, palliative care. Primary chemotherapy with cisplatin, ifosmide,paclitaxel and vinorelbine, usually as combination of 2 drugs such as cisplatin and paclitaxel or cisplatin and ifosfamide has shown 31-36% response rates.[7]

Supportive and palliative care should be part and parcel of the treatment of cervical cancer patients. Depression, pain and anxiety should be treated. Pain has been controlled with morphine, transdermal fentanyl patch and continuous morphine instillation through a peridural catheter placed and connected to a subcutaneous pump. Thus depending on the stage of the disease, cervical cancer requires a multidisciplinary team of gynoncologists, radiotherapists, psychologists, urology surgeons and motivated nurses.

Attempts are underway to produce a vaccine against HPV.

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GYNAECOLOGY CASE NUMBER 3

OVARIAN SEROUS CYSTADENOCARCINOMA - TOTAL ABDOMINAL HYSTERECTOMY BILATERAL SALPINGO-OOPHERECTOMY AND CHEMOTHERAPY

Name	: J.W.G	DOA : 10/05/04	DOD : 29/07/04
Age	: 44 years	DOA : 18/08.04	DOD : 08/09/04
Parity	: 2 + 0		
File No	: 0959291		
LMP	: 03/04/04		

Presenting Complaint

JWG was admitted to the acute gynaecological ward (1D) with progressive abdominal pains and swelling for 2 ½ months.

History of presenting complaint

She had been well till she developed intermittent generalized abdominal upsets characterized by flatulence hyperacidity and discomfort several weeks prior to the onset of the above symptoms. The abdomina swelling was insidious and was associated with a dull abdominal pain and feeling of abdominal fullness She gradually developed low appetite and subsequent weakness and constipation on and off. Her friend told her that she had lost weight, which she confirmed when she put on some of her skirts. She was treate severally for various ailments by the clinician in the nearest private clinic without any improvement. He menstrual flow remained unaltered and she had had no inter-menstrual or post-coital vaginal bleeding There was no history of localized weakness, cough or any other swelling elsewhere.

Obstetric and Gynaecologic History

She was a para 2 + 0 with two living children. She underwent an emergency cesarean section in 1994 du to fetal distress and subsequently had a successful trial of scar in 1996. Her menarche was at the age of 1 years. Her menstrual flow took 4 days and occurred every 28 days regularly. Her last menstrual period wa on 03/04/04. She had used the combined oral contraceptive pills since 1996 up to early 2004 without an adverse effects. No Pap smear cytological test had been done on her.

Past medical history

This was non-contributory

Family and social history

She was a single mother who lived in Maringo estate in Nairobi with her 2 children. She worked with Homegrown company which packaged beans for export. She neither drank alcohol nor smoked cigarettes. Her mother and elder sister and brother were all diabetic. In addition, her mother was hypertensive. None of maternal relatives had had a similar disease as she, particularly breast, colonic or ovarian cancer. There was no history of other chronic illness in the family.

PHYSICAL EXAMINATION

General examination

She was sick looking and mildly wasted and mildly pale. She had no jaundice, no pedal edema, no oral thrush and no lymphadenopathy. She had a BP of 110/70 mmHg, a PR of 78/minute, a RR of 20/minute and a temperature of 36.3°C.

Abdominal examination

The abdomen was distended more on the right side of the lower abdomen. There was a subumbilical midline scar (made for the cesarean section) and no other surgical or therapeutic marks. The skin bore striae and palpation revealed multiple masses in the lower abdomen that were not quite mobile and bu tender. She had ascites as demonstrated by a positive shifting dullness and fluid thrill. The liver was no enlarged, neither was the spleen. Inquinal lymph node examination revealed no abnormality.

Pelvic examination

JWG had normal external genitalia. Speculum vaginal examination showed healthy vaginal mucosa and cervix and no bleeding or lesion was observed. Digital examination indicated that she had a huge right adnexal mass that was fixed, tender and associated with other multiple masses in the pouch of Douglas (POD - which was also full) and the left adnexae. Deep palpation was associated with tenderness. Recta examination showed no abnormality.

Respiaratory system

The chest was clear with vesicular breath sounds and no abnormality.

Cardiovascular, musculoskeletal and the nervous systems were essentially normal

Abdimino-pelvic ultrasound done at admission showed an abdominopelvic mass that was multiseptated heterogeneous and measured 13 x 14 cm and marked ascites was also noted. The uterus was reported to be

normal sized and the endometrial thickness could not be measured properly. The pancreas, the liver, the pancreas, the biliary tree, the spleen and the kidneys were reported to be normal.

Impression

An impression of probable ovarian malignancy was made. Abdominal lymphoma, tuberculous peritoniti and other abdominal tumours were to be excluded.

Management

She was admitted to the cold gynecological ward (1B) for preparation for explorative laparotomy.

Counseling on the possible diagnosis and the planned explorative laparotomy was provided and the patien prepared for the surgery by doing the following investigations.

Investigations:

Abdominal computed tomography (CT) scan (13/05/04): Showed a huge lobulated pelvic mass wit variegated density measuring 25 x 23 x 19 cm with necrotic areas. The liver had fatty infiltration otherwis normal and so were the pancreas, spleen and the kidneys and the retroperitoneal lymph nodes. The uteru was adhered to the mass posteriorly and the tumour also involved the left adnaxae.

1. **Haemogram (25/05/04)**: Hb – 12.1 g/dl, Wbc count – 6.6 x 10^9/L, platelets – 431 x 10^9/L
2. **Renal function tests (25/05/04)**: K+ - 4.6 mmol/L, Na+ - 137 mmol/L, Urea – 2.4 mmol/L Creatinine – 87 μmol/L.
3. Preoperative **blood grouping and cross matching** (26/05/04): Blood group A +ve; 2 pint available.
4. **Chest X-ray**: Normal

The above results were explained to the patient. On the eve of the operation the patient was given furthe counseling regarding the surgery and informed consent obtained. She was given 1 gram of metronidazol and 15 mg of bisacodyl at 8 p.m and enema at 12 midnight. Pubic hair was shaved 1 hour before theatr and intramuscular atropine was administered ½ an hour before the patient was wheeled to theatre.

Explorative Laparotomy, TAH and Bilateral Salpingoopherectomy.

In theatre, and under general anaesthesia, aseptic catheterization in semilithotomy position precede examination under anaesthesia, which confirmed the earlier findings. Abdominal cleaning with savlo solution, draping and opening followed. The findings were huge bilateral ovarian tumours that wer haemorrhagic and adherent to the omentum, the rectum, the uterus and the fallopian tubes and the smal gut. The left measured 16 x 8 cm and the right, 24 x 20 cm. There was moderate ascites of straw-coloure

fluid. A single metastatic tumour was noted on the left lobe of the liver. Based on the gross features of the tumour, the tentative diagnosis was stage IIIc ovarian cancer.

Total abdominal hysterectomy (TAH), bilateral salpingoopherectomy (BSO) and omentectomy were done while avoiding gut injury when separating the tissues adhered to each other. The estimated blood loss was 1000 and 2 pints of blood were transfused. Despite the extensive surgery, the reversal of anaesthesia was uneventful. The Folley's catheter that was left in situ drained clear urine.

Postoperative Care

She was transferred to the recovery room where vital signs were meticulously observed ½ hourly for 2 hours when she was fully awake. The vital signs were within normal limits. She was observed for another 2 hours and then transferred to the ward. Intravenous antibiotics (metronidazole, crystalline penicillin and gentamicin) and fluids and intramuscular pethidine were administered soon after the surgery and in the ward for 48 hours. On the first postoperative day the patient was started on oral fluids graduated to light diet by evening, ambulated, catheter removed and explanation on the findings of surgery given. On the second day she was ambulant, raised no complaint and systemic examination indicated no abnormality. The wound was exposed on the 3rd day and was found to be clean and dry. Thereafter the patient did well and only awaited the histology results. Meanwhile preparation for chemotherapy was instituted. This included taking the weight and the height in order to calculate the body surface area and checking that the renal and liver function tests were normal. She and her relatives were informed of the possible need for long-term hospital stay and financial needs of chemotherapy.

The histology report indicated that the patient had ovarian serous cystadenocarcinoma of borderline malignancy.

Chemotherapy and supportive care.

Her body weight was 63 kg and her height was 155 cm giving a body surface area of 1.65 m^2

The chemotherapy protocol consisted of the following:

1. Cisplatin given once as an intravenous infusion at a dose of 50 mg/m^2 (50-75mg/m^2)
2. Doxorubicin (Adriamycin) given once intravenously at a dose of 50 mg/m^2 (50-100/m^2)
3. Cyclophosphamide given intravenously daily for 5 days at a dose of 500mg daily.

Prior to administration of the chemotherapy 1000 mls of intravenous fluids were given and another 1500mls thereafter. The above regimen was repeated every 3 weeks unless one of the haemogram, LFTs or RFTs tests was deranged. She got her first course on 18/06/04 consisting of 80 mg of cisplatin and 75 mg of Adriamycin (instead of 82.2mg - for ease of administration) as stat doses on day one and 500mg of

cyclophosphamide from day one to the 5th day. One week later she was noted to be quite pale. A full haemogram on 25/06/04 showed a pancytopenic picture with a white cell count of 1.02 x 10^9/L and neutropenia of 0.602 x 10^9/L. This necessitated the administration of Granulocyte Colony Stimulating Factor (or Neupogen) and antibiotics to ward off any infection in view of neutropenia.

On 28/06/04 she developed fever of 39.5oC necessitating a full septic screen, which was as follows:

1. Haemogram: Hb- 5.86 g/dl, Wbc count 0.737 x 109/L (neutrophils 0.367 x 109/L),
2. Urine for microscopy, culture and sensitivity – No growth obtained,
3. Blood smear for malaria parasites was negative.
4. Blood microscopy, culture and sensitivity no growth or organisms seen.

In view of the neutrophilia she was put in the side room and minimal visits made. Three pints of blood were transfused to build up the Hb for the next chemotherapy. A repeat haemogram a week later showed a neutrophilia of 18.4 x 109/L suggesting that the Neupogen had worked since she did not have any fever or other constitutional symptoms. The laboratory results are shown in the table below.

Pre- and Post Chemotherapy Laboratory Tests.

Date	18/06/04	28/06/04	20/07/04	10/08/04	30/08/04	22/09/04	30/09/04
Tests	1st chem	Post Rx	2nd chem	Post Rx	3rd chem	Post Rx	Post Rx
Haemogram: a) Hb g/dl	12.8	5.86	12.6	8.3	10.0	7.4	10.0
b)Wbcc x 10^9/L	6.3	0.737	4.92	3.8	5.1	2.46	5.2
c) plts x10^9/L	415	90	207	112	159	Adequate	118
RFTs a) K+ mmol/L	3.9	ND	4.2	ND	3.6	ND	ND
b) Na+mmol/L	136	ND	138	ND	143	ND	ND
c) Ure mmol/L	4.3	ND	6.2	ND	6.3	ND	ND
d) Cre µmol/L	69.0	ND	91	ND	79	ND	ND
LFTs	Normal	ND	Normal	ND	Normal	ND	ND

Key: 1. ND: Not done

2. chem.: chemotherapy

3. Post Rx: post treatment

During the second course of chemotherapy the patient received 300μg of Neupogen on the 4th and 6th day and although there was a mild degree of bone marrow depression, the patient did not develop any signs o sepsis such as fever after the 1st course. However, during the 3rd course she received only 1 dose o Neupogen and subsequently developed severe signs of meningitis and went into coma 22 days after the chemotherapy had been given. As shown on the table above the full haemogram done on 22/09/04, showed a pancytopenic picture. She was treated with ceftriaxone (Rocephine) and she recovered.

By the time of writing this commentary the patient was stable and was awaiting review of the dosages o the chemotherapy by the gynaecology oncologist and the general oncologist in view of the repeated post chemotherapy pancytopenia.

DISCUSSION

JWG was a 44 year-old para 2 + 0 who had features of ovarian malignancy, underwent TAH plus BSO and omentectomy and was found to have ovarian serous cystadenocarcinoma of borderline malignancy on histology. She had received 3 courses of cisplatin, adriamycin and cyclophosphamide with subsequent development of bone marrow suppression that required review of the chemotherapy.

Worldwide, ovarian cancer accounts for 10-15% of all female genital malignancies.[1] It is the third commonest after cervical cancer and choriocarcinoma but has a poorer prognosis than the other two.[2] The prevalence of ovarian cancer varies from region to region. It is higher in the United States of America (USA) and Scandinavian countries than in Oriental countries such as Japan. In the USA, a woman's risk of having ovarian cancer sometime in her lifetime is nearly 1.5% and that of dying from the disease is 1%.[2,3] It is the 5th leading cause of cancer related mortality among American women accounting for 5% of all such deaths , and more cancer related deaths than all other primary pelvic tumours. Only malignancies of the lung, breast, colon and lymphoreticular system claim more lives.[1,2] At the Kenyatta National Hospital (KNH), the incidence of ovarian cancer in women with genital malignancies was found to be 8% in 1979 by Njuki[4] and 9.4% by Karanja[5] in 2002. Unlike Njuki's study where ovarian cancer was the 3rd commonest genital cancer after cervical and choriocarcinoma, Karanja's study found ovarian cancer to be the 2nd commonest genital tract cancer in women at KNH. This is probably because choriocarcinoma is sometimes diagnosed on the basis clinical and biochemical diagnosis alone. From data obtained from the National Cancer Registry at the Kenya Medical Research Institute (KEMRI) courtesy of Dr. Abinya, a renowned oncologist based at KNH, ovarian cancer was the 5th (2.9%) commonest cancer in all women after those of breast (23.6%), cervix (14.8%), uterus/(?endometrium) (4.3%) and oesophagus (3.8%).

The cause of ovarian cancer is unknown, but a number of risk factors have been identified. These include familial tendency, nullipairity and/or infertility, early menarche and late menopause, certain types of diet, environmental factors, talc, asbestos, ovulation induction, prolonged estrogen exposure and endometriosis among others.[1,2,3,6]. Repeated ovulation is thought to lead to repeated disruption of the germinal epithelium, inflammation and repair that lead to metaplastic and subsequent carcinogenic changes.[1,6]. Hence anything that reduces ovulation decreases the risk of developing ovarian cancer. These include lactation, oral contraceptive pills, pregnancy and multiparity.

Pregnancy reduces risk of ovarian cancer by 30-60% while the combined oral contraceptive pills also reduces the risk by 30-60% and 50% if used for at least 5 years. This reduced risk persists for 10 years after cessation of the pill use.[1,6,7] Talc in talcum powder and corn powders applied to the perineum has been associated with increased risk of developing ovarian cancer. That tubal ligation and hysterectomy reduces the risk of developing ovarian cancer suggests that there could be carcinogenic factors from the uterus itself or from the environment via the vagina and the cervix. The perineal talcum powder is thought to reach the ovaries in this manner. Other environmental factors such as diets consisting of saturated animal fats, lactose and cholesterol have higher risk while fresh and yellow vegetables are protective. Cigarette smoking is positively associated with the disease.[1,2,7,8] These environmental/community-based factors probably explains increased incidence of ovarian cancer among Japanese women who migrate to the USA.[1]

Women with two first-degree relatives with ovarian cancer have a 50% likelihood of developing ovarian cancer up to age 70. Hereditary ovarian cancer occurs in 3 forms, either as breast and ovarian cancer syndrome (BOC), or the less common, Lynch II syndrome also known as hereditary nonpolyposis colorectal cancer syndrome (HNPCC syndrome) or site-specific ovarian cancer. Patients with Turner's syndrome (45, X0) have increased risk of dysgerminoma and gonadoblastoma.[1]

Although ovarian cancer is predominantly a disease of the postmenopausal woman and the prepubescent girl, it occurs in all ages.[1] However, unlike in the Western countries where the median age is 63 years, Karanja[5] found a median age of 46 years. Our patient was 44 years old and had no history of exposure to any risk of ovarian cancer. On the contrary she had used the oral contraceptive pills for more than 7 years, which was supposed to reduce the risk of developing ovarian cancer by 50%.

Of all ovarian neoplasms, 20% are malignant. Among the malignant tumors, 80% are primary (i.e. arise directly from the ovaries) and 20% are secondary.[1,2] The primary tumors fall into three major histopathologic categories. These are epithelial, germ cell and sex cord and stromal tumours.[1,2,3] The common primary site from which secondaries arise are gastrointestinal (pylorus, colon and rarely small intestine), gall bladder, breast and endometrium. The secondary tumours are either typical or atypical. In typical the histologic picture is the same as the primary cancer while in the atypical, as the Krugenberg tumour, the histological tumour is different from the primary site and sometimes it is difficult to locate the primary site.[1,2,3]

In the USA epithelial tumours form 90% of the primary tumours.[1,2,3] In our set up (KNH), Karanja[5] (2002) found epithelial tumours in 71% of cases and germ cell and sex cord and stromal tumours in 8.4% and 10.7% respectively. Epithelial cell tumours are bilateral in 50% of cases and include serous, mucinous,

endometrioid, clear cell, transitional and undifferentiated. Serous tumours are the commonest accounting for 35-55% of epithelial tumours. As was found in our patient who had epithelial, serous cystadenocarcinoma, they are bilateral in up to 60% of cases and are associated with extra-ovarian spread at the time of diagnosis.

Mucinous tumours are the 2nd commonest epithelial tumours, accounting for 10-20% of the cases. They are bilateral in less than 10% of the cases. They are associated with pseudomyxoma peritonei which results from progressive accumulation of mucus in the abdominal cavity following slow leakage from the neoplasm. It most usually occurs in association with cystadenocarcinoma of the ovary and appendix as well as mucocele of the appendix.[1,2]. Endometriod ovarian carcinoma exhibit adenomatoid pattern and resembles endometrial adenocarcinoma. It is bilateral in 30-50% of cases and arises from a focus of endometriosis in less than 10% of cases. Thirty percent of patients with endometrioid carcinoma have concomitant endometrial carcinoma that is a primary tumour of the uterus rather than it being a secondary of the ovarian tumour.[1,2,3] About 5% of ovarian tumours are mesonephroid, also called clear cell carcinoma. They are biologically active and could be associated with hypercalcaemia and/or hyperpyrexia. Transitional cell carcinomas resemble the transitional cell carcinoma of the urinary bladder. Patients typically present with advanced disease stage and exhibit a poorer prognosis than those of other histologic types. Undifferentiated carcinoma of the ovary occurs in less than 10% of epithelial neoplasms.[1,2,3]

Malignant germ cell tumours arise from the embryonic germ cells. They have varying degrees of malignant potentiality. They occur predominantly in children and young adults.[1-3] Some of these tumour produce biologic markers which can be used to monitor the response to therapy. They include dysgerminoma, endodermal sinus tumour, non-gestational ovarian choriocarcinoma and immature teratoma.[1 2] sex cord and stromal cell neoplasms include granulosa cell tumour, thecomas, androblastomas (Sertoli-Leydig cell tumours) and gynandroblstoma. Granulosa cell tumours are associated with hyperestrogenism and may cause precocious puberty in young girls and adenomatous hyperplasia and vaginal bleeding in postmenopausal women.[1,2,3]

The diagnosis of ovarian cancer is usually late since most ovarian malignancies develop as an insidious disease with few nonspecific symptoms and/or signs until the disease is widely disseminated in the abdominal cavity. As was the case with our patient, the diagnosis is made on the basis of clinical, laboratory, imaging, surgical and histological evaluation.

The outcome of patients with ovarian cancer depends on the stage of the disease at the time of diagnosis and availability of specialized personnel, financial resources and facilities. Staging is based on clinical, surgical, histologic and pathologic findings including cytologic findings off effusions or peritoneal

washings. Where present, therefore, pleural effusion should be tapped. The current staging of ovarian cancer approved by the International Federation of Obstetrics and Gynecology (FIGO) is shown below.

Stage I. Growth limited to the ovaries

Ia- one ovary involved

Ib- both ovaries involved

Ic- Ia or Ib and ovarian surface tumor, ruptured capsule, malignant ascites,or peritoneal cytology positve for malignant cells.

Stage II. Extension of the neoplasm from the ovary to the pelvis

IIa- extension to the uterus or the fallopian tubes

IIb- entension to other pelvic tissues

IIc- IIa or IIib and ovarian surface tumour, ruptured capsule, malignant ascites, or peritoneal cytology positive for malignant cells

Stage III. Disease extension to the abdominal cavity.

IIIa – abdominal peritoneal surfaces with microscopic metastases

IIIb – tumour mestastases less than 2 cm in size

IIIc – tumour metastases > 2 cm in size, or metastatic disease in the pelvic, para-aortic, or inquinal lymph nodes.

Stage IV. Distant metastatic disease.

Malignant pleural effusion,

Pulmonary parenchymal metastasis,

Liver or splenic parenchymal metastasis (not surface implants),

Metastasis to the suptraclvicular lymph nodes or the skin.

Our patient had stage IIIc disease on the basis of extensive disease involving the entire abdominal cavity including the surface of the liver, malignant ascites and no liver, pulmonary, splenic, parenchymal involvement.

The treatment of ovarian cancer involves surgery and then either chemotherapy or radiotherapy depending on the cell type and grade. The cornerstone of ovarian cancer therapy is surgery regardless of the cell type or stage of the disease.[1,2,3] A midline incision is always preferred. At laparotomy the sequence of the

operation include sampling of ascitic fluid or peritoneal washings for cytology, complete abdominal exploration, intact removal of tumour by bilateral salpingoopherectomy, hysterectomy, infracolic omentectomy biopsies of peritoneal implants, pelvic and para-aortic lymph node biopsies and cytoreductive surgery (debulking) to remove all visible and operable disease. This was done to our patient.

The rationale of aggressive debulking is based on three main considerations:

1. The removal of the tumour mass often relieves gastrointestinal symptoms and improves the patient's nutritional status,
2. Reduced tumour mass improves the efficiency of either chemotherapy or radiotherapy since large bulky tumours are often poorly vascularized and oxygenated and hence more resistant to therapy. Besides the more resistant cells in the resting phase found in large tumours are removed.
3. Debulking reduces the immunosurpressive capacity of the tumour.

Chemotherapy should be given as soon as possible once debulking has been done. Currently the most effective regimen for epithelial tumours uses the combination of paclitaxel (Taxol) and carboplatin. This combination is regarded the best followed by the combination of paclitaxel and cisplatin and others such as cisplatin and cyclophosphamide in that order.[1,2,3,8,9] Other drugs which can be used with platinum are topotecan and gemcitabine. The efficacy of therapy is monitored by clinical or imaging findings of regression of the tumour and/or correlating the pre-treatment CA-125 serum levels with intra-or post-treatment levels.[1,2,3] Dysgerminomas are the most radio-sensitive neoplasms identified.[1,2,3] Germ cell tumours, however, can be treated by chemotherapy with cisplatin and other combination regimens such as vincristine-actinomycin-cyclophosphamide. These cytotoxic drugs have side effects such as bone marrow suppression as occurred in our patient.

Second look operation (laparotomy or laparoscopy) should be done once cure is thought to have been achieved. Second-line and other forms of therapy include intraperitoneal chemotherapy, secondary cytoreduction and chemotherapy. Hormonal therapy and immunotherapy are being studied.[1,2,3,8] Surportive therapy is crucial in the management of ovarian cancers.

Unfortunately our patient received the first course of chemotherapy late and could not afford paclitaxel that was not available in the hospital. A combination of cisplatin, Adriamycin and cyclophosphamide was used instead. Although the tumour had not recurred as of the time of writing this commentary, the patient had developed very severe myelosuppression with consequent meningitis.

Prevention of ovarian cancer has been elusive. However, women at risk such as those with first-degree relatives (mother, sisters) with history of breast ovarian cancer syndrome and the Lynch II syndrome and those with history of ovarian cancer in first-degree relatives should undergo early CA-125 levels and pre-emptive bilateral oopherectomy as soon as they have completed their families or if the CA-125 begins to rise. Other methods of ovarian cancer prophylaxis that have been tried without proven success include regular pelvic examination, ultrasonography and where available at reasonable costs, CT scan done at regular intervals individualized as per the previous findings.

Worldwide the prognosis is poor with an overall 5-year survival rate of 40% in the USA. In the same country (USA) the 5-year survival rate for stage I disease is 76-93%, 60-74% for stage II, 23-41% for stage III and only 11% for stage IV disease.[1,2,3] Although the prognosis of ovarian cancer has not been studied in Kenya as much as in the USA, our patient was informed of the implication and possible prognosis. There is need to enhance cancer therapy in our set up.

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GYNAECOLOGY CASE NUMBER 4

GESTATIONAL TROPHOBLASTIC DISEASE-SUB-TOTAL HYSTERECTOMY AND CHEMOTHERAPY

Name:	P.N.K	D.O.A.:	15/11/2002	D.O.D:	7/12/2002
Age:	45 years	D.O.A.:	17/12/2002	D.O.D.:	22/12/2002
File No:	20:80:21 AKH	D.O.A.:	5/01/2003	D.O.D.:	11/01/2003
Parity:	3+1	D.O.A.:	20/01/2003	D.O.D.:	31/01/2003
L.M.P:	17/06/2002	D.O.A.:	10/02/2003	D.O.D.:	16/02/2003
		D.O.A.:	3/03/2003	D.O.D.:	9/03/2003
		D.O.A.:	30/03/2003	D.O.D.:	6/04/2003

Chief complaint

P.N.K presented to the Aga Khan Hospital, Nairobi (AKHN) casualty with complaints of intermittent vaginal bleeding for two months.

History of presenting complaints

She was well until 2 months prior to her admission on 15/11/2002 when she developed the aforementioned complaint. This followed termination of a three months gestation and concurrent bilateral tubal ligation (BTL) in a private clinic for an undesired pregnancy. She was put on antibiotics. The bleeding was mainly spotting with episodes of heavy bleeding often presenting with clots. There was an associated mild abdominal pain. She also continued to experience symptoms of pregnancy. These included, nausea, vomiting and breast tenderness but she did not experience any abdominal distension or foetal movements. She also complained of generalized body weakness and backache. She did not give any history of headache, blurring of vision, dysponoea passage of vesicles or palpitations. She did not experience any changes in her urination or bowel habits. She denied history of a bleeding disorder.

Obstetric and gynaecologic history

Her parity was 3+1 with 3 living male children. Her first delivery was in 1987 via spontaneous vertex delivery (SVD). This was after she underwent laparoscopic ovarian drilling and subsequent ovulation induction in 1986. Her 2nd delivery in 1989 was through a cesarean section for breech presentation. Her third delivery was in 1992 through caesarean section for premature rupture of membranes for 3 days without labour at 38 + weeks with a primary scar. Her last normal menstrual period (LNMP) was on

17/06/2002. Before this, her menstrual flow took 5 days every 28 days. It was moderate in amount and was not associated with dysmenorrhoea. She had only used copper T IUCD between 1992 and 1997. Two of her previous Pap smears done in 1992 and 1997 had been normal.

Past medical history

P.N.K. had been admitted twice before to the Aga Khan hospital, Nairobi. The first was in 1997 for laparoscopic cholecystectomy due to gallstones and the second was in March 2002 for subtotal thyroidectomy in the right lobe of euthyroid goitre. There was no history of chronic illness or allergy to any substance. The two operations were uneventful and no drug allergies were known.

Family and social history

She was a married banker who worked with Barclays bank, Nairobi, did not smoke nor consume alcohol. Her husband worked with the Ministry of Health. There was no history of familial disorder.

PHYSICAL EXAMINATION

General examination

Her general condition was fair and was mildly pale and without fever, jaundice, oedema or lymphadenopathy. Her vital signs were: BP 130/80mmHg, pulse rate; 86/min, temperature; 36.6°C. she weighed 90kg.

Abdominal examination

The abdomen was slightly distended at the supra-pubic region. It moved with respiration and had multiple healed surgical scars consisting of a sub-umbilical mid-saggital scar for a previous caesarean section, a peri-umbilical BTL scar and two small right and left hypochondriac scars for laparoscopic cholecystectomy. Her abdomen was soft. A pelvic mass corresponding to 16 weeks gestation that was globular, mobile horizontally and non-tender was noted. No other masses or organomegally was found.

Pelvic examination

PNK had normal external genitalia. Speculum examination showed healthy vaginal mucosa and a brownish discharge at the posterior fornix. The cervix was dilated with a reddish mass attached to the posterior lip. Digital examination showed the cervix was 4cm dilated and soft a mass was felt adherent to the posterior

lip. The uterus was enlarged, mobile and non-tender and corresponded to 16 weeks gestation. The adnexae and the pouch of Douglas (POD) had no abnormality.

Respiratory, cardiovascular and central nervous systems were essentially normal

Impression

An impression of gestational trophoblastic disease with differentials of incomplete septic abortion, prolapsed uterine mass and a cervical polyp was made.

Investigations

1. **Ultrasound:** This showed a markedly enlarged uterus with widespread mixed echo lesion throughout the uterus more on the lower pole, which was also enlarged. The upper pole of the uterus showed a large hypoechoic area measuring 4.19cm x 1.52cm which could either be a haemotoma or a collapsed gestational sac. Right ovary appeared normal, left ovary could not be visualized. There was free fluid in the pouch of Douglas. Infective uterine mass or molar pregnancy was suspected.
2. **Haemogram:** Hb-9.8g/dl
   WBC-8.9X10^9/l
   Platelets- 367x10^9/L
3. **ß-hCG: 435,149 mIU/ml**
4. Blood grouping and cross matching of 2 units. Her blood group was A +ve.
5. Urea, electrolytes and creatinine were within normal limits.

Management

Based on the ultrasound findings and the serum ß-hCG levels, a working diagnosis of molar pregnancy was made. Preparation for examination under anaesthesia and suction curettage entailed obtaining informed consent after appraisal of the working diagnosis, intended procedure and the possible risks. She was starved overnight. The following morning, she was given an enema at 6 a.m. She was cleaned and shaved; and premedication with 0.6mg of intramuscular atropine 0.6mg administeredprior to being taken to theatre.

In theatre

Under general anaesthesia, she was put in lithotomy position and vulvo-vaginal toilet and draping with sterile clothes done. By aseptic urethral cathetirization, 100ml of clear urine was drained. An infusion of 20

units of syntocinon in 500ml of 5% dextrose was started. On Auvard speculum examination the vaginal mucosa was found to be normal and the cervix was dilated with a red mass protruded from the uterus. When digital examination was attempted, she developed torrential bleeding that did not respond to intravenous ergometrine and prostaglandin F2 alpha. Blood transfusion was commenced and an extra venous access obtained for syntocinon and colloids infusion. The consultant on call was immediately called and an emergency laparotomy was performed.

Abdominal toilet, draping and openning via the sub-umbilical midline incision, after removal of the old caesarean section scar, revealed an enlarged uterus corresponding to 16 weeks gestation and a mass of about 6 x 6 cm arising from the cervix and extending upwards into the lower uterine segment and parametria on both sides. The abdominal viscera, ovaries and tubes had no obvious abnormality. The heavy bleeding necessitated sub-total hysterectomy. Despite this, patient continued to bleed from the suture sites. Urgent coagulation profile, an extra 6 units of whole blood and 3 units of fresh frozen plasma were prepared for fear of disseminated intravascular coagulopathy (DIC).

A Portovac drain was left in situ and the abdomen closed in layers after a report of correct account of all the surgical equipment had been made. Vaginal examination showed moderate and a vaginal pack was left in situ. Reversal from anaesthesia was successful despite the haemorrhagic shock characterized by post-operative blood pressure of 80/40-mmHg and pulse rate of 120/minute. Intra-operatively, she received 3 units of whole blood. A PTI of 81.3% and INR of 1.26 were suggestive of DIC and, being in shock, she was admitted to ICU where a central venous pressure line was inserted into the right subclavian vein. She was put on oxygen by mask at rate of 6L/minute. Intravenous 500mg of tranexamic, 4mg of ondansetron (Zofran), 1.2 grams of amoxycillin-clavulanic acid combination (Augmentin) and 500mg of metronidazole were all given 8 hourly as well as 25mg of intramuscular pethidine as required. Extra 6 units of whole blood, 3 units of fresh frozen plasma were administered. In view of the massive blood transfusion,a full course of intramuscular beta artemisinin (Paluther – 80mg 12 hourly on the first day then 80mg daily for 4 days) to prevent infection with malaria.

Postoperative Care

She had dramatic recovery. On the first postoperative day she was in fair general condition with a BP of 130/80mmHg and a pulse rate of 80/min. The urine output was satisfactory and was, therefore, transferred

back to the gynaecology ward where preparation for chemotherapy for gestational trophoblastic disease was made. This included:

1. **Plain abdominal x-ray**: There was no pathological calcification or radio opaque calculus visible. Bowel gas distribution was normal.
2. **Pelvic Ultrasound**: There was a large mixed echo mass in the pelvis measuring 9.43 x 6.49 x 7.53cm. The ovaries could not be visualized. There was an encysted collection of fluid pestero-lateral to the mass measuring 4.7 x 4.6 cm. A CT scan of the pelvis was suggested.
3. **CT scan whole abdomen**: The spleen was not enlarged. The pancrease was normal in shape and size.Liver parenchyma was homogeneous, there was no biliary obstruction, and no gallstones were seen. There was no evidence of para-aortic abdominal lymphadenopathy. Both kidneys excreted the contrast medium and showed a normal pelvi-calyceal system. No obstructive uropathy was present. Small bowel distribution was normal. The bladder was normal. The uterine remnant was fairly large. No inguinal lymphadenopathy was seen. The rectum did not appear to be involved.
4. **Chest x-ray**: Cardiac size and configuration were within normal limits. Both lung fields were clear. Positiong of CVP was satisfactory. No mestatasis was seen.
5. **Liver ultrasound:** Liver parenchyma was of normal homogeneous echogenicity and no dilated biliary ducts were seen. The gall bladder was normal and no biliary calculi were evident. CBD was 4mm. Pancrease was normal in size and echo-pattern. Spleen appeared normal.

As part of the preparation for the chemotherapy the patient was scored as per the World Health Organisation (WHO) prognostic scoring system as follows:

Characteristic Feature	Score
• Blood group (A x O)	1
• Site of metastases	0
• Number of metastases	0
• Largest tumour size (6cm)	2
• Age of 45 years	1
• Antecedent pregnancy (abortion)	1
• Interval of antecedent pregnancy (2months)	0
• Serum ßhCG (435,149mIU/ml)	4
Total	**9**

It was because of her weak state that she was started on low doses of chemotherapy every fortnight and after evaluating the βCG levels, haemogram and renal and liver function tests. The doses were as follows:

Intravenous cyclophosphomide 250mg once daily for 5 days

Intravenous methotrexate 15mg once daily for 5 days.

Intravenous dactinomycin 0.5mg once daily for 5 days.

The serum ßhCG levels became negative (ref.<5mlIU/ml) after 6 courses and 3 additional courses were given thereafter. The results of the aforementioned tests are summarized in the table below.

DATES TESTS	1st course 19/11/02	2nd course 3/12/02	3rd course 18/12/02	4th course 6/01/03	5th course 21/01/03	6th course 11/203	7thcourse 04/03/03	8th course 31/03/03
ßhCG	57,948	317	45	10.7	7.7	2.44	<2.0	<2.0
LFTs								
T. bil(μmol/L)	11	7	4	3	2	4	4	3
D. bil(μmol/L)	5	3	1	-	-	-	-	-
A. phos (iu/L)	106	158	397	196	214	129	126	139
Gam GT(iu/L)	15	40	181	64	71	52	58	89
SGOT (iu/L)	18	16	57	12	25	25	22	24
SGPT (iu/L)	10	7	86	16	20	26	22	31
Haemogram								
WBC-x 10^9/L	16.5	9.2	6.1	6.5	8.0	6.6	7.8	10.2
Plts x 10^9/L	113	962	406	683	297	386	517	704
Hb (g dl)	7.0	9.0	10.6	10.6	11.8	11.3	11.1	13.0
RFTs								
Na^+ (mmol/L)	144	138	135	143	136	137	136	138
K^+ (mmol/L)	3.0	3.2	4.0	3.8	4.3	4.7	3.3	4.5
Cl^- (mmol/L)	104	103	104	109	100	100	103	102
Cr. (μmol/L)	60	53	63	52	-	71	88	-
Urea (mmol/L)	4.1	1.4	3.3	2.0	2.4	2.7	4.7	3.2

In view of the massive transfusion serum ferritin levels done on 5/12/2002 was noted to be high at 350 nanograms/ml (normal range: 13-150ng/ml). Since she was stable and LFTs were normal, this was considered harmless.

Follow-up

Her subsequent monthly follow up was in AKHN gynaecology outpatient clinic. In each visit, a pelvic examination was performed and no abnormality was found. The subsequent ßhCG levels were:

May - <2mIU/ml

June - <2mIU/ml

July – 0.25mIU/ml

August-- <2mIU/ml

October- <2mIU/ml

June 2004 - <2mIU/ml

She had a Pap smear done on 15/07/03, which showed atrophic features and was classified as SILO/CIN0.

DISCUSSION

PNK was a 45 years-old para 3+1 who developed choriocarcinoma following an abortion of a3 month old gestation. She was underwent subtotal hysterectomy after she developed uncontrolled uterine bleeding when suction curretage was attempted. Subsequently she was treated for high risk choriocarcinoma by a combination of methotrexate, actinomycin-D and cyclophosphamide with remission.

Choriocarcinoma is one of the neoplasms classified under gestational trophoblastic diseases. These are disease characterized by expression of human chorionic gonadotropin in the absence of fetal heart tones and fetal structures. They are highly curable by chemotherapy. Gestational trophoblastic disease is a general term for a spectrum of proliferative abnormalities of the trophoblast. These include hydatidiform mole, invasive mole (chorioadenoma destruens) and choriocarcinoma.[1,2] In addition to being the first and only disseminated solid tumours that have proved to be highly curable by chemotherapy they elaborate a unique and characteristic tumour marker, human chorionic gonadotrophin (Hcg).[1,2] Choriocarcinoma may develop following a pregnancy (gestational choriocarcinoma) or in the absence of a pregnancy (non-gestational choriocarcinoma).[1,2] Non-gestational choriocarcinoma are rare and are often associated with some some teratoma or seminoma and other germ cell ovarian tumours.[1,3]

Choriocarcinoma does not contain chorionic villous structures but is composed of sheets of both anaplastic cytotrophoblast and syncytiotrophoblast. Placental site trophoblastic tumour (PSTT) is an uncommon variant of choriocarcinoma composed entirely of mononuclear intermediate trophoblast and does not contain chorionic villi. Choriocarcinoma has a propensity for early vascular invasion with widespread dissemination. The most common metastatic sites are the lung, vagina, brain and liver in that order.

Metastasis to the lungs has a good prognosis when compared with other areas of metastasis.[1,2,4] In this case, there were no metastatic lesions.

Choriocarcinoma occurs in about 4% of patients after the evacuation of a molar pregnancy and infrequently following other pregnancies.[1,4] Available data show that 50% of cases of choriocarcinoma follow evacuation of a hydatidiform mole, 25% follow an abortion, 20% follow term pregnancy and 5% follow extrauterine pregnancy.[5] After molar evacuation persistent gestational trophoblastic tumour (GTT) may exhibit the histological features of either hydatiform mole or choriocarcinoma. After a non-molar pregnancy however, persistent GTT have the histologic pattern of choriocarcinoma. The ultimate cause of gestational trophoblastic diseases may be genetic. Choriocarcinoma is said to be more likely to arise as a consequence of first pregnancy by a particular man and that if it occurs in a multiparous women the chances are that the pregnancy might be of a different paternity.[6] our patient developed choriocarcarcinoma following an abortion of a 3 month gestation.

The incidence of choriocarcinoma in the U.K and the U.S.A is of the order of 1:50,000 to 1:70, 000 pregnancies and is ten times more common in South East Asia.[5] The incidence at KNH was reported as 1:1, 118 deliveries.[6] The antecedent pregnancy was hydatidiform mole in 57% of cases, normal pregnancy in about 26% and an abortion, as in the case of our patient, in about 17% of cases. The risk of choriocarcinoma after a hydatidiform mole is about 2 to 4% while that of a partial mole is 4 to 8%.

The diagnosis of choriocarcinoma requires a high index of suspicion. It is mainly clinical. History of persistent or irregular uterine haemorrhage following an abortion, a molar pregnancy or a normal delivery, should always raise the suspicion of choriocarcinoma. The bleeding is usually profuse, but sometimes there may be only blood stained. Later there may be offensive discharge, pyrexia or cachexia[7]. When amenorrhea occurs it is due to a very high level of HCG secreted by the metastatic growth outside the uterus.

The disease may also present by way of its metastasis. Dyspnea and haemoptysis are noticed with lung metastasis. Metastases often develop early with common sites being lungs (over 75%). Neurological symptoms like hemiplegia, epilepsy, headache and visual disturbances may occur with brain metastases. Vaginal metastasis appears as a bluish red vascular tumour that bleeds easily on touch. The uterus may be enlarged and granulosa lutein cysts may be palpable. Ovarian theca lutein cysts are identified in over 1/3 of the cases.[1,7] Elevated BhCG together with any of the above symptoms makes the diagnosis of choriocarcinoma more likely.

The predisposing factors associated with development of choriocarcinoma include pregnancies towards the beginning or end of childbearing periods, history of previous molar pregnancy, multiparity, low socio-economic status and folic acid deficiency.[8] Recognition of the possibility of lesion is most important factor in diagnosis. Women with molar pregnancy are at risk of developing choriocarcinoma and need to be followed up.

All patients with suspected choriocarcinoma should undergo a careful pretreatment evaluation, which includes: -

1. Complete history and physical examination
2. Measurement of the serum BhCG value
3. Hepatic, thyroid and renal function fests.
4. Determination of baseline peripheral white blood cell and platelet counts

Metastatic work-up should include: a chest x-ray or computed tomography, ultrasonography of the abdomen and pelvis, CT scan of the head and in some cases selective angiography of abdominal and pelvic organs. Head CT scans help in the early diagnosis of asymptomatic cerebral lesions.[1,4] An abdominal CT scan, a chest X-ray, an abdominal and liver ultrasound and other baseline investigation were undertaken in our patient.

Choriocarcinoma is staged according to the international Federation of Gynaecologist and Obstetricians (FIGO) staging system. The staging is as follows: -

Stage 1: Persistency elevated BhCG levels and tumours confined to the uterus.

Stage II: Tumour outside the uterus but limited to he genital structures (adnexa, vagina, broad ligament).

Stage III: Pulmonary metastases with or without uterine vaginal or pelvic involvement.

Stage IV: All other sites of metastases

At each stage patient are further classified in to A, B, C, depending on the risk factors. 'A' is when there is no risk factor, 'B' only one risk factors and 'C' two risk factors. The risk factors are: -

1. Human chorionic gonadotrophin levels more than 100, 000 mLu/Ml
2. Duration of diseases of more than 6 months from termination of antecedent pregnancy.

Other factors taken into consideration include prior chemotherapy and placental site tumours.

Our patient did not have metastasis, the human chorionic ginadotropin levels were below 100, 000 mLu/mL and the duration of diagnosis from last delivery that was an abortion was less than 6 months. She was staged at 1A.

Apart from staging, selection of chemotherapy and the prediction of drug resistance depends on a prognostic scoring system. The prognostic scoring system in use currently is the WHO prognostic scoring system.

The scoring system is as follows: -

PROGNOSTIC FACTOR	**SCORE**			
	0	1	2	4
Age (years)	≤ 39	> 39		
Antecedent pregnancy	H. Mole	Abortion	Term pregnacy	
Interval between end of antedecent pregnancy and start of chemotherapy (months)	<4	4 – 6	7 – 12	>12
HCG (I, U/L)	$<10^3$	10^3- 10^4	$10^4 – 10^5$	$>10^5$
ABO groups (female x male)		O x A, A x O A x unknown	B x A or O AB xA or O	
Largest tumour including uterine tumour (cm)		3 –5	5	
Sites of Metastases		Spleen Kidney	G.I.Tract, liver	Brain
No. of metastases identified		1-3	4-8	>8
Prior chemotherapy			Single drug	>1 drug

The total score for a patient obtained by adding the individual scores for each prognostic factor. When the prognostic score is 8 or greater the patient is considered high risk and requires intensive combination chemotherapy to achieve remission. A score of less than 4 is considered low risk and a score of 5 – 7

middle risk. Generally patients with stage I disease have a low-risk score and patients with stage IV have a high-risk score[1,4]. our patient was classified as low risk.

Choriocarcinoma is highly curable even with widespread metastases. Chemotherapy is the main stay of treatment of choriocasrinoma. Low risk patients are treated with single agent drug therapy. Methotrexate with or without folinic acid rescue is used [1,4,7] Actinomycin D, 5-Flourouracil or Bleomycin may also be used[7]. in our set up methotrexate is mainly used. Medium and high risk disease are usually treated with combination chemotherapy,. Several combination regimens have been advocated but currently the most commonly used are the M.A.C and EMACO regimens. The M.A.C. Protocol is a combination of methotrexate given in doses of 0.4mg/kg/d intravenously for 5 days, Actinomycin D given in doses of 10 – 12ug/kg/d intravenously for 5 days and cyclophosphamide in a dose of 3 –5 mg/kg/d intravenously for 5 days. The cycle is repeated after 14 days or as toxicity allows. The EMACO regimen encompasses Etoposide, Methotrexate, Actinomycin-D and Leucovorin calcium alternating with vincristine and cyclophosphamide[1,4,7]. The EMACO regimen results in response rates of about 90% and survival rates of 80 to 1000 percent[3,8]. in our set up the MAC regimens is commonly used. In the event of MAC therapy failure EMACO regimen is used. Our patient had good remission with Methotrexate monotherapy regimen. Patients with brain metastases should be treated with whole head irradiation with 3000 rads[1,2].

In the follow-up, all patients with stages I, II, and III should have weekly BhCG measurements until normal for 3 weeks and then monthly hCG until levels are normal for 12 consecutive months. Patients are encouraged to use effective contraception during the entire perid of follow-up. Patients with stage IV disease are followed by weekly HCG values until normal for 3 weeks and then monthly HCG values until normal for 24 months[1,4].

Hysterectomy may be required in choriocarcinoma to control uterine hemorrhage or sepsis. In patients with extensive uterine tumour hysterectomy may substantially reduce the trophoblastic tumour burden and therapy limit the need for multiple courses of chemotherapy. Hysterectomy is also indicated for placental site tumour because it is quite resistant to chemotherapy. Hysterectomy is preceded and followed by chemotherapy to prevent the risk of dissemination and development of distal metastasis.

Patients with choriocarcinoma and other gestational trophoblastic diseases who are successfully treated with chemotherapy can except normal reproduction in the future. The frequency of congenital anomalies is not increased, although chemotherapeutic agents are known to have teratogenic and mutagenic potential.

However, women who have had trophoblastic diseases are at an increased risk for developing trophoblastic disease in a subsequent pregnancy [1,3,4].

Women with low risk choriocarcinoma who are treated aggressively with single or multi agent chemotherapy have remission rate of almost 100%. In high risk choriocarcinoma remission rates have been reported to vary from about 45 to 65 percent. Among the prognostic factors are the site of metastases, and number of metastases[3,5].

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GYNAECOLOGY CASE NUMBER 5

LONG-TERM REVERSIBLE CONTRACEPTION: INSERTION OF JADELLE® AFTER REMOVAL OF NORPLANT®

Name	: S.W.W	LMP	: 25/09/2004
Age	: 23 years	Last delivery	: October 1999
Parity	: 1 + 1	Date of visit	: 13/10/2004
Card No	: 1344/99		

Presenting Complaint

SWW presented to the Kenyatta National Hospital (KNH) Family Welfare Clinic (FWC) with the need for another long-term contraceptive method such as the levonorgestrel (Norplant) implants that she had used for 5 years and was due for expiry on the day of the visit.

History of presenting complaint

She had had an unwanted pregnancy at the age of 18 years. After having a spontaneous vertex delivery to live female infant in October 1999, she was advised on contraception and she opted for Norplant, which was inserted on 12/10/1999. She was to have it removed in 5 years time, the time which the implant ceases being active as a contraceptive. Hence her visit to the FWC.

Obstetric and Gynaecologic History

She was para 1 + 1. She had a spontaneous abortion of a 3-month pregnancy in 1998 and underwent uterine evacuation at KNH without any major complication. She was not transfused blood. Her recovery after then was good. Her five-year-old daughter born with a birth weigh of 3.5 kg via spontaneous vertex delivery was alive and well. She attained menarche at the age of 14 years and her coitarche was at 15 years. Before the use of Norplant her menstrual flow took 3-4 days and occurred every 30 days without any menstrual disorder. For three months after the use of the Norplant, she developed menometrorrhagia. This was managed with the combined oral contraceptive pills given to her for two consecutive months. Thereafter she had regular scanty menses but the flow became longer (5-7 days). She and her husband were counseled on the side effects of the implants and they were satisfied with the contraceptive method. She had visited the FWC twice thereafter and on both occasions had been advised on doing a Pap smear cytology test. Unfortunately she had not done so citing financial constrains.

Past medical history was not significant

Family and social history

SWW was a married housewife who lived in Matasia in Ngong, 25 km from Nairobi city center. Her mother was diabetic. She and her husband did not drink alcohol neither did they smoke tobacco. The husband worked as clerk with the Nairobi Law Courts.

PHYSICAL EXAMINATION

General examination

She was in good general and nutritional condition. She had no pallor, jaundice, edema or lymphadenopathy. She had a BP was 120/70 mmHg, a RR of 18/minute, PR of 70/minute and a temperature of 36.5°C.

Breasts examination

These were Tanner stage V. There was no tenderness, no lumps and no galactorrhoea.

Abdominal examination

The abdomen was flat and moved with respiration. There was no organomegally, no tenderness and masses.

Pelvic examination

She had normal external genitalia. The vaginal mucosa felt normal. The uterus was anteverted and of normal size and shape. The cul de sac and the adnexae were free. There was no abnormal vaginal discharge on the examining finger.

The respiratory, cardiovascular, musculoskeletal and the nervous systems were essentially normal.

Impression

A twenty-three year-old para 1 + 1 with a desire for a long-term contraceptive method.

MANAGEMENT

She was counseled on the new formulation of levonorgestrel with a trade name of Jadelle® and contraceptive use equivalent to that of Norplant. She accepted to use Jadelle for the next 5 years, which was inserted after removal of the 6 capsules of the Norplant.

Removal of Norplant and insertion of Jadelle

The equipment for removal of Norplant and the insertion of Jadelle was prepared. This included local anaesthetic, scalpel, two (Mosquito and Crile) forceps, sterile cloths, gloves, gauzes and tray, iodine (Betadine) solution and 21-gauge needle and 5 cc syringe among others. The client was placed in supine position and being right handed, the left upper limb was rested on a side table also in supine position. The medial aspect of the left arm was cleaned and painted with iodine solution before it was draped with sterile cloths. The base of the fan of the previous Norplant insertion was then infiltrated with 4 mls of 0.5% lignocaine and then under the sticks of Norplant. A 4 mm transverse incision was made, the capsules pushed towards the incision one by one. Using the Mosquito forceps, the tip of each capsule was grasped, gently pulled out through the incision. Where necessary, a scalpel was used to release tissue capsule around the capsule. The Crile forceps was then used to pull the capsule.

To insert the two capsules of Jadelle, a trocar was pushed subdermally in the opposite direction from that of the Norplant capsules up to the mark close to the hub. A capsule was then inserted into the trocar and using the plunger, the capsule was pushed until resistance was felt. The trocar was then withdrawn - while steadying the plunger – until the mark at the tip of the trocar was visible. Similarly, without completely withdrawing the trocar the second capsule was inserted. The incision was then applied with iodine solution, gently apposed with the Crile forceps and dressed with sterile gauze. She was advised to keep the wound site dry for 4 days and to be seen in the FWC in a week's time when a Pap smear would be taken.

Follow up

She was seen as scheduled. The wound had healed well. A Pap smear was taken but the report of the cytology was unavailable by the time this commentary was being written.

DISCUSSION

The patient presented was a 23 year-old para 1+1 who had used Norplant implants for 5 years uneventfully and needed another reversible long-term contraceptive method, for which a new formulation of levonorgestrel, Jadelle, was inserted.

Norplant is a sub dermal implant composed for six match stick size capsules (34mm long and 2.4mm wide) made of silastic capsules each containing progestin levonorgestrel 36 mg in crystalline form. The capsules are inserted under the skin of woman's upper arm in a fan shaped pattern using a simple trochar. The hormone is released at a steady state starting at 85 ug per day and decreasing to 50 ug at 9 months, 35 ug at

18 months and 30 ug thereafter[1,2]. Upon insertion of the capsules, the release of levonogestrel reaches a protective level after 24 hours.[2] The implants are protective for 60 months.

The mode of action of Norplant is similar to other progestin only contraceptives. It causes thickening of cervical mucus, anovulation in up to 50% of users, accelerated ovum growth and, when ovulation occurs, degeneration of the corpus luteum.[3] The method had been approved by 27 countries by 1992. It was introduced in Kenya in 1986, with regulatory approval obtained in August 8th 1989.[1]

The five-rod Norplant has, however, been phased out and a new two-rod device with a brand name of Jadelle® or Norplant-2 has been introduced. Jadelle is a second-generation progestin only contraceptive implant. It consists of two rods containing 75mg of leveonogestrel. Unlike Norplant where levonorgestrel crystals are freely encased in silastic capsules, the levonorgestrel crystals in Jadelle are embedded in copolymer and encapsulated in a rod measuring 2.5 mm wide and 43 mm long. The major advantages of Jadelle are that insertion and removal are easier and take less time because there are only two rods versus six capsules for Norplant. Norplant and Jadelle are bioequivalent and are used for 5 years.[4]

Norplant failures rates are rare in the first year of use at 0.2% and from the second to fifth year, pregnancy rates are 0.5%, 1.2%, 1.6% and 0.4% respectively. The pregnancy cumulative rates after 5 years is 3.7%[5] This efficacy was exemplified by the fact that our patient had used Norplant for 5 years without conceiving.

Continuation rates of Norplant users after 1 year are 85%-95% and 33-78% complete 5 years.[1,6] The reasons for discontinuation are mainly the desire to conceive and the occurrence of side effects. Major potential health sequelae have not been identified in association with use of levonorgestrel subdermal implants (Norplant or Jadelle), but side effects are fairly common. Some degree of menstrual irregularity such as increased flow or spotting has been reported in up to 60% of Norplant users in the 1st year. However, the occurrence of such side effects is time dependent with the rate declining by about 50% after one year. Headache is sited as the reason for discontinuation in upto 20% of women. Weight change, mastalgia, nervousness, nausea, acne, dermatitis, hair loss or growth and change in appetite are commonly reported. It is contradicted in pregnancy and in unexplained vaginal bleeding. It is relatively contraindicated in patients with a history of stroke or ischaemic heart disease, and when on drugs that affect liver enzymes.[2,8]

Insertion is usually done within seven days of menses after abortion or 6 weeks post partum.[9]

Despite the effectiveness, safety, and patients satisfaction with this excellent contraceptive, it has become the target of personal injury lawyers (in the west). This is based on the silicon content producing "illness". The patient presented here had no complaints at all after the 60 months follow up and had received her periods as usual though it was slightly scantier and longer by 2 days. The menorrhagia she had in the first 3 months following Norplant insertion was expected and she and her husband were counseled to expect this. It (menorrhagia) was managed with combined oral contraceptive pills.

Insertion site complications include infection, bleeding, expulsion of the capsules and pain.[2] The cost is also slightly higher at the time of insertion.

Upon removal. return to fertility is always assured in not a distant future.

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GYNAECOLOGY CASE NUMBER 6

SECONDARY INFERTILITY DUE TO TUBAL BLOCKAGE – LAPAROSCOPIC TUBOPLASTY

Name	: M.W	DOA	: 11/08/04
Age	: 36 years	DOD	: 14/08/04
Parity	: 1 + 0	LD	: 1994
File No	: 0970818	LMP	: 04/08/04

Presenting Complaint

M.W was admitted through the Kenyatta National Hospital (KNH) infertility clinic with a diagnosis of secondary infertility for 10 years due to bilateral tubal blockage.

History of presenting complaint

She had been physically well but emotionally distressed for the last 8 years since she realized that she could not conceive despite satisfactory unhindered coitus with her husband with whom she got the first child. For 6 years she hoped that she would eventually conceive. However, this did not happen and she, therefore, sought medical assistance with a local clinician who then referred her to KNH. Her menstrual flow and frequency was not different from what she had before and after she conceived. The menstrual flow took 3-4 days every 26-30 days regularly and without any menstrual disorder such as dysmenorrhoea. She reported positive history of increased secretion of clear mucous in the middle of her cycle, some thing which has always been present even before the first pregnancy. She denied any history of headache or blurred vision, features of thyroid disease, diabetes, connective tissue disease and any other systemic disease. Further, there was no history of chronic lower abdominal pain, abnormal vaginal discharge, dyspareunia or ever contracting any sexually transmitted disease both herself and her husband. Her husband had never had any genital ulcer disease, abnormal urethral discharge, swelling of the scrotum or mumps. They denied any history of multiple sexual partners. She had used the combined oral contraceptive pills for 1 year after the delivery to her only child.

Obstetric and Gynaecologic History

She was para 1 + 0 with one living child born via spontaneous vertex delivery without any adverse events. Her last menstrual history was on the 04/08/04. She attained menarche at 16 years. She had never done a Pap smear cytology test.

Past medical history was not significant

Family and social history

She was a married business lady who lived with her husband. Her husband and she bought and sold second hand clothes. Neither of them drank alcohol or consumed tobacco. There was no history of chronic disease in the family.

Investigations

1. Hysterosalpingograph (HSG): Bilateral ampulo-fimbrial hydrosalpinx. No spill noted bilaterally. Uterine and cervical anatomy essentially normal.
2. Semen analysis : Normal
3. Pap smear cytology : Normal
4. HIV ELISA test : Negative
5. Haemogram : White cell count - 6.5 x 109/L
   Hb – 13.9 g/dl
6. Renal function tests : Urea – 3.2 mmol/L,
   Na+ - 146 mmol/L,
   K+ - 4.5 mmol/L
   Creatinine - 77μmmol/L
7. Pelvic ultrasound, hormonal profile and endometrial biopsy had not been done.

Diagnosis

Thirty-six year-old para 1 +0 with secondary infertility due to bilateral fallopian tubal blockage.

Management

The patient had been informed of the diagnosis and the mode of management in the infertility clinic. This explanation was provided further in the ward. She was to undergo laparoscopic (and if need be open) tuboplasty under general anaesthesia. The success rate, risks and the procedure of the operation were explained to her after which informed consent was obtained. She had been investigated as shown above. She received 1 gram of metronidazole orally at 8 p.m. She was starved from 12 midnight on the night preceding the day of surgery and pubic hair shaved an hour before the surgery. She also received 0.6 mg of atropine half an hour before she was wheeled to theatre.

Laparoscopic Tuboplasty Under General Anaesthesia

The patient was put in the supine position, general anaesthesia given and then in lithotomy position, abdominal and vulvo-vaginal toilet, draping and aseptic catheterization was done. The anterior cervical lip was grasped with single tooth volselum forceps and uterine elevator inserted. A stab incision was made just inferior to the umbilicus and using a Veress needle, pneumoperitoneum was created with carbon dioxide. The stab incision was then extended to 1 cm and a 10 mm port was then inserted through the same incision and the camera and scope followed. This revealed:

1. Bilateral hydrosalpinx both from the isthmal level with fimbrial closure by marked adhesion,
2. Filmy adhesions arising from the posterior uterine wall and covering the ovaries, the pouch of Douglass (POD) and the adjacent gut.
3. Both ovaries appeared to be polycystic albeit minimally,
4. The appendix and the other abdominal viscera were essentially normal.
5. Normal sized uterus without anomaly such leiomyomata.

Two extra ports were inserted in either of the iliac fosae. Using a dissecting scissor, the filmy adhesions were released. A Babcok forceps was used to grasp either of the tubes and bilateral salpingostomy and fimbrial intosuception performed. A clear fluid from the hydrosalpinx was taken for microscopy, culture and sensitivity. A dye test with methylene blue showed free dye spill from the left tube and minimal oozing of the dye from the right tube. The ovaries were then drilled with diathermy. Peritoneal lavage with normal saline preceded infiltration with heparin and hydrocortisone. The two ports on the iliac fossae were then removed under vision. Pneumoperitoneum was then deflated and the camera and its port withdrawn also under vision. The abdomen was closed and endometrial biopsy curretage performed. The reversal of the general anaesthesia was uneventful.

Postoperative Care

Her vital signs were observed ½ hourly till she was fully awake then 4 hourly. She received intravenous antibiotics consisting of metronidazole, crystalline penicillin and gentamicin for 48 hours. Analgesia was achieved by use of pethidine and later mefenamic acid. On the first postoperative day she was well, ambulant and had already started oral sips and light diet was to be introduced gradually. By the second postoperative day, after 48 hours of antibiotics, she was discharged home on oral amoxi0clav and metronidazole antibiotics with advise to avoid conception in the next 3 months to prevent ectopic pregnancy. She was to be reviewed in the infertility clinic in 2 weeks time.

Follow up

In 2 weeks time she had healed well and had no complaints. The endometrial curretings showed proliferative phase of the endometrial cycle while the fluid from the hydrosalpinx had no organisms and grew nothing. She was to repeat an HSG in two months and thereafter to attempt conception.

DISCUSSION

Presented here is a para 1 + 0 patient who had secondary infertility due to a tubal factor for which laparascopy was done. The peritubular and ovarian adhesions were released and salpingostomy was done on bilaterally with a good dye spill from the left tube and minimal dye spill from the right one.

Infertility is defined as one year of unprotected intercourse without pregnancy. This condition may be further classified as primary infertility in which no previous pregnancies have occurred and secondary infertility in which a prior pregnancy, although not necessarily a live birth, has occurred.[1]Our patient had secondary infertility.

Fecundability is the probability of achieving pregnancy within a single menstrual cycle, and fecundity is the probability of achieving a live birth within a single cycle. The fecundability of a normal couple has been estimated at 20% to 25%.[1,2] On the basis of this estimate, about 90% of couples should conceive after 12 months of unprotected sex.[2]Childlessness may be a tragedy to the married woman and can be a cause of marital upset as well as of personal unhappiness and ill health. This can affect either the male or the female partner.[3]The diversity and multiplicity of possible cause of infertility needs meticulous investigations to

reveal them. Female infertility contributes most of the cases than male. However, 10% of males are absolutely sterile.[3,4,5, 6]

In the USA it is estimated that 1 in 6 couples will experience difficulty in conceiving during 1 year and that 1-2% of couples are involuntarily sterile. An apparent increase in the prevalence of infertility is suggested by analysis of trends in medical visits, which reveals an exponential increase in the number of visits for infertility in the last decade. The reasons for the increase in attention given to infertility are multiple. Couples in some cases have voluntarily delayed child bearing in favour of establishing careers and may experience age-related decline in fertility, in some cases the choice of prior contraception may have contributed to infertility, as with the use of some intrauterine contraceptive devices (IUCDS); having an increased number of sexual partners leads to a greater potential for exposure to sexually transmitted diseases, which may contribute to infertility; and couples are less willing to simply accept childlessness and are increasingly aware of the available services and options for resolving infertility.[1,3,5,7]

Both partners in a relationship contribute to potential fertility and both may be sub-fertile. A primary diagnosis of a male factor is made in a bout 30% of infertile couples and the man may be contributory in another 20-30%. The woman is responsible for the remaining 40-50% of cases.[5,7]

Various factors have been associated with female infertility. They can have failure to produce ova frequently or to produce ova capable of being fertilized. Pelvic adhesions interfere with the passage of the ovum from the ovary to the tubes. [3,4,6]The patient presented had peritubal adhesions, hydrosalpinx and terminal tubal blockage all being dactors that impeded ova transfer and fertilization.Partial or complete tubal blockage, contributes up to 20% of all cases of infertility. The main causes of tubal blockage are previous salpingitis commonly from gonococcus, Chlamydia species or tuberculous infections. Also spasm of the utero- tubal junctions, congenital hypoplasia of the tubes also interferes with ovum transfer.[3,6] There was no history of genital track infections in our patient though it is estimated that virtually one half of patients with tubal damage or pelvic adhesions have no history of antecedent disease.[7]

Investigations for infertility should be commenced after 12 to 18 months of regular coitus without conception. The initial clinical assessment, while focusing on the history and physical status of the female partners, should also include the historical factors of importance that pertain to the male partner and to the couple. The initial test should be the least invasive and progress to invasive procedures. The first test should be that of the male factor which is semenalysis. Once the male factor has been ruled out, the female

can then be further evaluated. Investigations for tubal patency should be carried out 2-5 days following the conclusion of menstruation. This is to avoid disturbing a possible fertilized ovum. Vaginal sonosalpingography, utilising air and saline as the contrast has been used and is a reliable simple and well-tolerated method to assess tubal patency in an out patient setting. This procedure can be performed without prophylactic antibiotics using a regular paediatric Foleys catheter instead of an expensive hysterosalpingography catheter. Though not practiced in this setup, it has a positive predictive value of 94.9% and a negative predictive value of 71.4%.

Hysterosalpingogram (HSG) is the method commonly used in our set up. Radiographic liquid dye is instilled into the uterine cavity using either a paediatric Foley catheter, to occlude the cervical canal or a suction catheter. After 3-5 mls of dye is insufflated, an image is obtained and additional dye is added to fill the uterine tubes. The procedure is witnessed under an image intensifier or key films are obtained. This will demonstrates the uterine contour, the patency of the tubes and the ability of the dye to freely spill into the pelvis. Abnormal findings include congenital malformation of the uterus sub-mucous leiomyomas, intrauterine synechiae (Asherman's syndrome), intrauterine polyps, salpingitis, isthmic nodosa and proximal or distal tube occlusions.[5] HSG done for this patient showed bilateral hydrosalpinx and fimbrial adhesions which were confirmed at laparascopy. As used in our patient dye laparascopy is complementary to HSG. Unlike HSG laparascopy allows careful assessment of the external architecture of the tubes and in particular visualization of the fimbria. Identified abnormalities include tubal obstruction, pelvic adhesions, and endometriosis.

Therapies that directly address correcting tubal factor infertility are entirely surgical and include correction of periadnexal disease, correction of proximal, distal or combined tubal disease and correction of iatrogenic tubal abnormalities (e.g. tubal sterilization). Surgical treatment of periadnexal disease causing tubal factor infertility have proved effective whether by laparatomy or laparascopy. However, laparascopy has been proved to be superior in preventing recurrence of adhesions.[8,9]

Proximal tubal occlusion may be corrected by selective salpingography performed under fluoroscopy where contrast is injected directly into the tubal lumen in an attempt to overcome obstruction resulting from mucous plugging. If selective salpingogram fails to create tubal patency, proximal tubal cannulation can be performed using a guide wire under direct visualization or using radiological guidance. Proximal tubal cannulation has a reported success rate of 85% in establishing tubal patency. Re-occlusion occurs in 30% of cases. The risk for tubal perforation with cannulation is reported to be 3% to 11% but tubal damage is

usually mild and heals spontaneously.[10] Tubal anastomosis for proximal tubal occlusions is no longer practiced or encouraged. Instead microsurgical tubocornual anastomosis has become the preferred surgical approach with post surgical ongoing pregnancy rates averaging 47.4% in some series.[11,12] Distal tubal occlusion is treated by surgical correction. This can be by fimbriostomy or neosalpingostomy. This can either be done laparascopically or by laparatomy. Regardless the method used the efficiency of neosalpingostomy or fimbrioplasty as treatment for distal tubal occlusion rests largely on the extend of tubal and peritubal disease.[7,11]

The tubal mucosa is the most important factor in tubal disease process and hence success of any correction. The mucosa is graded into good, intermediate and poor quality. Poor qualities are those with loss of rugae and vascularity and presence of adhesions. This can be assessed before surgery by use of falloscope.[7] Severely damaged tubes may not benefit from surgical procedures. The patient presented had neosalpingostomy done. This will hopefully enable her to conceive.

In situations where tubal damage is too extensive in vitro fertilization may be advised. In this situation controlled ovarian stimulation is one and several follicles are harvested during each cycle. These are then mixed with spousal sperm and the resultant embryo reimplanted into the uterus.

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GYNAECOLOGY CASE NUMBER 7

POSTPARTUM BILATERAL TUBAL LIGATION

Name	: E.W.N	DOA	: 15/08/04
Age	: 38 years	DOD	: 19/08/04
Parity	: 6 + 1 with 7 children	LMP	: 15/11/03
File No	: 0975960	EDD	: 22/08/04
		GBD	: 39 weeks

Presenting Complaint

W.N had just delivered her 7th child and being single wanted no more children

History of presenting complaint

She had been well until she went into labour on 14/08/04. On 15/08/04 she was admitted to the Kenyatta National Hospital (KNH) labour ward in advanced active labour at 39 weeks gestation by dates. She had spontaneous vertex delivery to a live male infant with a birth weight of 3000 grams and an Apgar score of 7, 8 and 10 at 1, 5 and 10 minutes respectively. The 3rd and 4th stages of labour were uneventful and so was the antenatal period. She had had her antenatal care in a local clinic from about 32 weeks but no antenatal profile had been done. After her delivery she had discussed her predicament with the nurse who conducted the delivery and the doctor in the postnatal ward and had opted for bilateral tubal ligation as permanent solution to her problem. She declined HIV testing despite counseling by a trained counselor. Her blood group was B-Rhesus positive.

Obstetric andGynaecologic History

EWN was now para 6 + 1 with 7 living children all delivered vaginally. Her first delivery was in 1985 when she was 19 years old. She had had a spontaneous abortion in 1999 at about 10 weeks and no evacuation was done. She had twin delivery in the year 2000 and had sworn never to conceive again. Consequently, she used the combined oral contraceptive pill but was non-compliant, hence the 6th pregnancy. After the 3rd pregnancy she had used medroxyprogesterone acetate injections but stopped 9 months later because of the heavy uterine bleeding that she developed. She had not tried the intrauterine contraceptive device (IUCD) because a friend of hers told her that she had developed heavy vaginal bleeding upon the IUCD insertion. She did not want another experience of bleeding. She had not visited any family planning clinic though for advice on other contraceptive methods. No Pap smear testing had

been done on her. She denied any history of contracting a sexually transmitted disease despite being single and having had 3 sexual partners the last 10 years each at different periods of time. She had menarche at 15 years and thereafter had menses for 3-4 days every 30 days.

Past medical history was insignificant

Family and social history

She was a single mother who lived with her children and parents. She did various business activities to make ends meet. Neither did she drink alcohol nor smoke cigarettes. There was no history of chronic illness in the family.

PHSICAL EXAMINATION

General examination

She was in good general and fair nutritional condition with mild pallor but without edema, jaundice or lymphadenopathy. Her BP was 130/80 mmHg and the PR was 80/minute,the RR was 18/minute and the temperature was 36.9°C.

Breasts examination

These were Tanner class V. The nipples were evertted, no cracks were noted and they were just beginning to be active. Suckling had been commenced and was noted to be appropriate.

Abdominal examination

It was uniformly distended and moved with respiration. The uterine fundal height corresponded to a 22-week gestation and was well contracted and mildly tender. There was no other organomegally.

Perineal inspection

She had appropriate rubral lochia for the first postnatal day that was not foul smelling. The vulva had no lacerations.

The calf muscles, respiratory, cardiovascular and the nervous systems were essentially normal.

Impression

Desired family size in a single 38 year-old para 6+1 with 7 living children on her first postnatal day.

Management

She had been asked to starve from mid night for possible BTL. Informed consent was obtained and the patient wheeled to theatre after she had passed urine and premedication with intramuscular 0.6 mg of atropine had been administered.

Bilateral tubal ligation under general anaesthesia.

The urinary bladder was noted to full as she had stayed for too long in the waing bay. Aseptic catheterization was done in semilithotomy position then in supine position the abdomen was cleaned and draped with sterile linen. A 3-cm semi circular, transverse subumbilical incision was made and a minilaparotomy performed. Using hand held retractors and by moving the uterus side by side, the right fallopian tube was held with a Babcock clamp, identified by the fimbriae, exteriorized and tubal occlusion achieved by use of the modified Pomeroy's method using Vicryl number 1/0 suture. Having achieved haemostasis the tube was returned into the abdomen. The same procedure was carried out for the left tube and then the abdomen was closed in layers using the same suture material. The skin was stitched with subcuticular stitches and dressed with iodine-laced gauze. Reversal of the general anaesthesia was smooth.

Postoperative Care

Vital signs were observed ½ hourly till she was fully awake before she was transferred to the ward to continue with 4 hourly observations. She was to start light diet after 4 hours and oral amoxycillin and mefenamic acid were prescribed for her. Twelve hours later she was well enough to be discharged home with advice to visit the postnatal clinic after 1 week

Follow up

One week postpartum the wound had healed well and breastfeeding was well established. The lochia loss was sero-sanguinous and no abnormality was noted in the systemic examination. She was to be followed up in the Family Welfare Clinic where Pap smear was to be done. Further counseling on HIV testing was to be done there and in the KNH voluntary counseling and HIV testing center.

DISCUSSION

The patient presented was a single 38 year-old para 6+1 with a desired family size on her first postnatal day. She underwent postnatal BTL and had uneventful postoperative and early postnatal period.

Bilateral tubal ligation (BTL) is the mechanical blocking of both fallopian tubes to prevent the sperm from reaching the egg. It is a safe and simple surgical procedure, which can usually be done with just local anaesthesia and light sedation. It is also called voluntary female surgical contraception or female sterilization, tying the tubes and "the operation".[1] Bilateral tubal ligation can be done during the immediate post partum period (within 48 hours of delivery as happened with our patient), caesarean section or at 4 or more weeks after delivery (interval sterilization).[2]

Voluntary surgical contraception has become the most widely used method of family planning in the world for both developed and developing countries. In 1990, more than 170 million couples of childbearing age in developing countries used voluntary surgical contraception.[3] In the United States approximately 700,000 women a year currently choose voluntary sterilization.[4] However in most African nations use of tubal sterilization is low.[3] In Kenya it is the third commonly used modern method of contraceptive after injectable contraceptive and oral contraceptive pills. However data from the 1998 and 2003 Kenya Demographic and Health Surveys[5,6] indicate that female sterilization among married women in Kenya fell from 6.2% in 1998 to 4.3% in 2003.

There are several advantages of female sterilization. These include the procedure is permanent, the failure (pregnancy) rate is low at 0.1% to 0.5%, the patient has nothing to buy or remember, no significant long-term side effects, the partner does need to co-operate, lovemaking need not be interrupted and does not affect breastfeeding. Female sterilization is ideal for couples who are certain they wish to have no further children and who need a reliable contraceptive method. It is also indicated in women with medical disorders in whom subsequent pregnancy may have an adverse effect on the woman's health. The medical conditions include cardiac diseases and diabetes mellitus.[3,6]

The disadvantage of female sterilization include: the procedure is permanent, techniques to reverse the sterilization are difficult and expensive, sterilization procedures are technically difficult, the operative procedure requires a surgeon, an operating room, trained assistants, medications and surgical equipment and it does not protect against sexually transmitted disease.[1,3] The surgical approach is mainly abdominal.

The approach may be through a minilaparotomy incision or laparoscopy. The patient presented had mini-laparotomy. The mini laparotomy procedure can be done under sedation and local anaesthesia or under general anaesthesia. In our patient it was done under general anaesthesia.

There are various techniques of tubal occlusion. The most popular methods with the minilaparotomy procedure include:-

1 Irving procedure - the medial cut end of the oviduct is buried in the myometrium and the distal end is buried in the mesosalpinx.
2 Pomeroy procedure - a loop of oviduct is ligated and the knuckle of the tube above the ligature is exercised
3 Parkland procedure - a mid segment of tube is separated from the mesosolpinx at an a vascular site, and the separated tubal segment is ligated proximally and distally and then exercised.
4 Madlener procudure - a knuckle of oviduct is crushed and then ligated without resection.
5 Kroener procedure - the tube is legated across the ampulla and the distal portion of the ampulla including all of the fimbriae is resected.

At KNH, Pomeroy's method is the most commonly used as happened with our patient.

Laparoscopic techniques of tubal occlusion include:-

1 Pomeroy's method.
2 Fulguration – electrocoagulation with or without excision or division.
3 Clips such as spring loaded clips or tantalum clips
4 Bands such as Falope ring

As mentioned earlier the timing of female sterilization can be post partum (immediately after delivery) or at 4 or more weeks after delivery (interval sterilization) or at caesarean section or laparotomy. Post partum BTL should be done within 48hours after delivery. The decision for the procedure should as much as possible, be made before the onset of labour. At this time, the fundus is near the umbilicus, permitting a small incision to be made and easy access to the tubes. Also unnecessary long hospital stay is prevented and an early ambulation achieved. On some situations a delay of 12-24 hours may be necessary for a more accurate assessment of the baby's chances for survival. After 48 hours. A lower and larger incision is required and the tubes are not easily accessible.[2]

Before the procedure, each patient should be given information and instructions in a language they can understand. These information must include the steps of the operations, instructions for care of the wound, what pain and discomfort might occur, common anticipated complications and what to do in each case, use of medication prescribed. when to return to work and resume sexual relations and timing of follow-up visit. To prepare for the operation, the client is told to bathe and wear clean loose clothing and if possible bring someone with her to accompany her home after the operation. Consent should be obtained in writing.
During the procedure, proper infection-prevention procedures are required. The operation site is properly cleaned. In the event of local infection, the procedure should be differed until this has been treated. Scrubbing and facemasks are mandatory for operators and assistants.

Complications of BTL may be divided into immediate and late complications. Immediate complications include injury to internal organs e.g. bladder, intestines, blood vessels, uterine perforation with uterine elevator, wound infection, hemorrhage, thermal burns and pain. Late complications include wound sepsis, failure of the method, ectopic pregnancy and regret.[2,3,4,6,7] Failure rates of BTL are lower than temporary contraceptive methods. Most studies of the common occlusion techniques, the Pomeroy and Parkland techniques, silastic ring, Filshie and spring clips, electrocoagulation and the Irving technique – report failure rates o less than 1% usually 0.1-0.8%.[2]
The reasons for failures are

1 Surgical errors accounting for 30 – 50% of cases
2 Patient already pregnant
3 Faulty clips or the fallopian tube undergoes faulty re-anastomosis.
4 Equipment failure such as defective electric current for the electrocautery.[4]

Although BTL is considered permanent some women do request for reversal following a divorce, remarriage, a child's death or if they desire more children.[2,3] The success rate of reversal depends on the method used for tubal occlusion. Occlusion techniques that damage the smallest segment of the oviduct have the highest success rate. The Filshie and spring clips have a success rate of about 88%. The Pomeroy's method has a success rate of approximately 50% and the electrocoagulation method approximately 43%.[2,3] the success rates are generally achieved through the use of microsurgical technique which requires special training.

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GYNAECOLOGY CASE NUMBER 8

UTERINE LEIOMYOMATA MANAGED BY TOTAL ABDOMINAL HYSTERECTOMY

Name	: A.N	DOA	: 29/09/04
Age	: 44 years	DOD	: 04/10/04
File No.	: 0975361	LMP	: 16/09/04
Parity	: 4 + 1		

Presenting Complaint

A.N. was admitted to the cold gynaecology ward (1B) via the gynaecology outpatient clinic (GOPC) with a year's history of progressive heavy and painful menstruation associated with low back pain due to uterine fibroids.

History of Presenting Complaint

She had been well until a year ago when she noticed that she was using pads more than usual when having menses. Besides the duration of flow increased from a maximum of 4 days to 10 days by the time she was being seen. Unlike in the past she noticed that the menstrual flow was with blood clots. She had had intermenstrual bleeding though this was not as heavy as the menstruation. The bleeding had made her weak and occasionally would get dizziness. She was no longer able to till in the farm as she was used to. Menstruation was much more painful than before and had to use painkillers such as ibuprofen to relieve the pain. The low back pain was dragging in nature and was not associated with menstruation but was aggravated by standing suddenly and walking for a long distance. She had also noticed a firm growth in her lower abdomen. Three months prior to admission, she was started on treatment with a progestagen norethisterone (Ubisterone®) a 5 mg tablet three times a day for 21 days then monthly from day 16 to day 25) and Ranferon (haematinic combination) capsules. The bleeding had subsided but was still heavy. Though her urination had slightly increased in frequency, she had no dysuria or urgency of urination. She only had some discomfort on defaecation.

Obstetric and Gynacologic History

She was para 4+ 1. Her last delivery was in 1992. All her deliveries were at term, in hospital and vaginal and her 4 children (3 sons + 1daughter) were alive and well. She had had spontaneous abortion at about 3 months in 1986 and underwent uterine evacuation in Kisii District Hospital. She attained menarche at about 15 years and thereafter had menses for 3-5 days every 28-30 days without any menstrual disorder other than mild lower abdominal discomfort. She used the combined oral contraceptive pills between pregnancies

and up to the age of 40 years when she was advised to use other methods because of her age. She opted to use natural methods and the condom. She and her husband had no desire for more children. She had never had a Pap smear test done neither had she had any sexually transmitted infection.

Past Medical History

A.N had been admitted to Kisii District Hospital and treated for malaria four times. She had not undergone any blood transfusion or operation and had no known allergies.

Family and Social History

She was a married lady who lived with her husband and some of her children in Nyakoiba village in Kisii district. She had attained only incomplete primary education. She and her husband were farmers and neither of them drank alcohol nor smoked tobacco. There was no history of chronic illness or any familial disorder in the family.

PHYSICAL EXAMINATION

General examination

She was in good general condition and fair nutritional status with mild pallor but without fever, jaundice, edema or lymphadenopathy. She had a BP of 130/80 mmHg, a PR of 70/minute, a RR of 18/minute and a temperature of 36.7°C.

Breasts examination

They were flappy, soft and without tenderness, lumps or galactorrhoea.

Abdominal examination

The abdomen was full and moved with respiration. No surgical or therapeutic marks were visible. It was soft and generally non-tender except on a firm iiregular mass that corresponded to a 16 weeks gestation arising from the pelvis. The mass was nodular, mobile from side to side, centrally placed in the suprapubic region and one's fingers could not go over the suprapubic aspect. There was no ascites or any other organomegally or mass.

Pelvic examination

She had undergone partial clitoridectomy otherwise the rest of the external genitalia were normal. Cusco's speculum examination revealed a healthy vaginal mucosa and a parous cervix that looked grossly normal. No bleeding or abnormal discharge was noted. Digital examination indicated that the uterus was enlarged with a firm mass in the posterior aspect that felt like a myoma. The cervix moved up freely on elevation of

the mass with the left hand. The uterine size corresponded to 16 weeks gestation. Bimanual examination of the adnexae and the pouch of Douglass (POD) revealed no abnormality.

The nervous, cardiovascular, musculoskeletal and the respiratory systems were essentially normal.

Investigations

1. Pelvic ultrasound: Enlarged uterus with multiptle fibroids. The largest is intramural in the posterior wall and measured 7.3 x 5.1 cm. The adnexae and the POD were without any abnormality.
2. Haemogram : Hb : 10.4 g/dl
   Wbcc : 4.8×10^9/L
   Platelets : 599×10^9/L
3. Renal function tests: Potassium : 3.8 mmol/L
   Sodium : 142 mmol/L
   Urea : 4.2 mmol/L
   Creatinine : 53 μmmol/L
4. Pap smear : Satisfactory; SIL0/CIN0
5. Blood group B +ve: 2 units available.

Diagnosis

Symptomatic uterine fibroids in a 44-year-old para 4+1 with 4 living children and a desired family size.

Management

She was prepared for total abdominal hysterectomy. Part of the preparation had been done as an outpatient in the GOPC including the aforementioned investigations. Counseling on the operation, the procedure, implication and the possible risks were discussed with her before informed consent was obtained from her. Blood was drawn for grouping and cross match and two units were available for her. On the eve of the operation, she was given 15 mg of bisacodyl (Dulcolax) and 1 gram of metronidazole tablets respectively. She was starved from midnight. At 5.30 a.m. she was given enema with good results. Pubic hair was then shaved and ½ hour prior to being wheeled to theatre she received intramuscular 0.6 mg of atropine and 50 mg of pethidine.

Total abdominal hysterectomy and left unilateral salpingo-oopherectomy

In theatre the patient was put under general anaesthesia. She was then placed in semi-lithotomy position and aseptic catheterization done. Pelvic examination under anaesthesia confirmed the earlier findings only

that the ovaries felt bigger than usual for her age. Using methylene blue the vagina was painted and the patient placed in supine position. The abdomen was cleaned, draped with sterile linen and opened via a subumbilical midline incicision. The abdomen was without adhesions. The uterus was found to have one large posterior wall myoma of about 7 x 7 cm and multiple small ones both anteriorly and posteriorly. There were no subserosal myomas. The overies were slightly enlarged and appeared granulated. About 100mls of straw-coloured fluid was found in the POD

The gut was packed away with wet abdominal packs. A self-retaining retractor was inserted and the uterus lifted out of the pelvic cavity and anchored with a myomectomy screw. The round ligaments were identified bilaterally, double-clamped and divided between clamps. Stay sutures were left in the distal stumps. The anterior leaves of both broad ligaments were opened bilaterally and the right pedicle and the proximal part of the right tube were double clamped, divided between clamps and ligatured. The distal aspect of the left tube and the infundibular ligament (excluding the ovarian ligament) were double clamped, divided between clamps and ligatured. The posterior leave of the braod ligament and the uterosacral ligaments were dissected away. Having opened the vesico-uterine fold of peritoneum, the bladder was dissected away by blunt dissection using a mounted gauze swab. The uterine vessels were identified bilaterally, skeletonized, double clamped and divided between clamps and ligatured proximally. Similarly the cardinal ligaments were double clamped and divided between clamps. Two Littlewood's were used to elevate the anterior cervico-vaginal junction and then the vaginal vault was circumcised while clamping any bleeders from the vault edges.

The vaginal vault was closed with interrupted sutures then it was reperitonised with anchoring to cardinal ligaments and indirectly to round ligaments. Haemostasis was achieved. The abdomen was cleaned with warm saline before it was closed in layers (the skin with subcuticular stitches) and the wound dressed. The catheter, which was noted to be draining clear urine, was to be left for 24 hours. Reversal of the general anaesthesia was smooth. The patient was extubated and taken to the recovery room. The estimated blood loss was 250 mls.

Postoperative Care

Half hourly observation of vital signs took place till she was fully awake when she was taken to the ward where 4-hourly observation continued. Treatment included intravenous fluids, 80 mg of gentamicin 8 hourly, 2 MU of crystalline penicillin 6 hourly and 100 mg of intramuscular pethidine 6 hourly for 24 hours. Twelve hours later she was in good general condition with normal bowel sounds and no

abnormality. She was started on oral sips to graduate to light diet. Explanation on the unilateral oopherectomy was given to her and she understood. After 48 hours she continued with only mefenamic acid for pain and haematinics. She did not need blood transfusion. On the 3rd day the wound was exposed and was found to be clean and dry. She did well postoperatively and by the 4th day was well enough to be discharged home.

Follow up

Four weeks after the operation she was well. The wound had healed well. The histology report, which showed multiple uterine leiomyomata with normal cervix and left ovary, was discussed with her. She was advised to have coitus after three months and to be seen in the Kisii District Hospital in the event of any problem.

DISCUSSION

This was a 44-year-old patient with multiple symptomatic uterine leiomyomata for which she underwent a total abdominal hysterectomy and intraoperative unilateral salpingo-oopherectomy. Postoperative period was uneventful.

Among the most common problems encountered by the gyneocologist are uterine leiomyomata commonly known as fibroids. They are the most common tumours of the female genital tract.[1,2] They are benign tumours of the myometrium composed of smooth muscle cells and fibrous connective tissue and whose precise aetiology is unknown.[1,2] Fibroids as they are more commonly known occur in 20 – 25% of women in the reproductive age, are 3 – 9 times more common and occur at an earlier age (30s) in women of African descent compared to Caucasian women (40s).[3] In a study conducted by Wanjala[4], 67% of patients with leiomyomata were between the ages of 26 – 40 years. Leiomyomata are rare below 20 years of age. An incidence of 50% has been found in post-mortem examinations.[1] The incidence of leiomyomata is the same in pre- and postmenopausal women although the average size is smaller in the latter. Leiomyomata depend on oestrogen for growth which is maximal during the reproductive years.[1,2] Our patient had the risk of being of the black race and reproductive age.

Myomas may be single but most are multiple. They are classified by anatomic location as intramural, submucous, and subserosal.[2] They may also become pedunculated and occasionally parasitic. Leiomyomata undergo degenerative changes, which may be related to symtomatology.[1] The commonest form of degeneration is hyaline degeneration. Others include cystic degeneration, calcification, infection,

carneous degeneration (common in pregnancy), fatty degeneration and, the most serious but rare, sarcomatous degeneration whose incidence is about 0.7%.[1,2] Our patient had only intramural leimyomata without obvious degenerative changes.

Despite the postmortem findings of 50% incidence rate in all women, relatively few leimyomata are associated with symptoms. Most uterine leiomyomat (60 – 80%) are asymptomatic and are incidental findings on routine examination.[1] Less than 50% of patients with leiomyomata have symptoms which depend on size, number, location of the tumours and any degenerative changes that have occurred. The symptoms include abnormal menstrual bleeding usually menorrhagia and intermenstrual bleeding, which may cause anaemia as was noted in our patient. Others are pressure symptoms on nearby viscera (urinary sumptoms, constipation and intestinal obstruction), pelvic pain, pelvic congestion or low back pain, abdominal swelling, spontaneous abortions and other pregnancy related problems and infertily.[1,2] Wanjala[4] in his series reported the main complaints to be abdominal pain (58%), menstrual disorders (55%) and abdominal swelling in 38%. Menorrhagia has been reported in up to 65% patients. Leiomyomata are thought to be related to infertility though no mechanism has been identified. Factors associated include anovulation, interference with sperm transport, tubal blockage by leiomyomata, endometrial changes, vascular congestion, menorrhagia, dyspareunia and age. Myomectomy has been shown to restore fertility in these patients.[5,6]

The diagnosis of leiomyomata can be made clinically though ultrasonography is often used to confirm the diagnosis and to exclude other attendant anomalies such as ovarian tumours and pelvic kidney(s). Submucous leiomyomata can be diagnosed by hysterosalpingography or directly visualized hysteroscopically.[1,2] This patient had leiomyomata diagnosed clinically and confirmed by ultrasonography. A Pap smear was done to rule out concurrent cervical disease, which if present requires extended hysterecomy. Though it was not done in our patient an intravenous urograph (IVU) is usually performed for markedly enlarged myomas to delineate the course of the urinary tract, particularly the ureters whose anatomical location could be altered b the myomatous uterus rendering them vulnerable to injury.

The management of uterine leiomyomata includes the use of three main modalities: medical, radiological and surgical.[1] Surgical therapy includes hysterectomy (abdominal, vaginal, laparoscopically assisted or total laparascopic) and myomectomy. Hysterectomy is the definitive mode of management for uterine leiomyomata and is the most common mode of management for symptomatic leiomyomata. In fact 33% of all hysterectomies are due to leiomyomata making them the commonest reason for hysterectomies.

Hysterectomy is curative. This patient had a total abdominal hysterectomy with unilateral oopherectomy and had all her presenting symptoms resolved.

Medical management of leiomyomat is temporary and not definitive and is reserved for patients who do not desire surgery, cannot withstand surgery or just prior to surgery. They provide symptom relief while others shrink the tumours. Drugs used include progestins and danazol, non-steriodal anti-inflammatory drugs, the combined oral contraceptive pill, mifepristone and other antiprogestins, gestrinone (antioestrogen/antiprogestin) and gonadotropin releasing hormone analogues.[1] Myomectomy is reserved for patients with symptomatic leiomyomata who wish to preserve fertility. With myomectomy the recurrence rate of the leiomyomata is 15 – 40%.[2]

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GYNAECOLOGY CASE 13

VAGINAL SEPTUM WITH HAEMATOCOLPOS AND HAEMATOMETRA

Name:	P.G	Parity:	Para 0 + 0
Age:	16 years	D.O.A:	13-09-04
IP No:	0970802	D.O.D:	21-09-04

Presenting Complaints

P.G presented with complaints of failure to achieve menarche and lower abdominal pains for 2 years.

History of presenting complaints

She was a 16-year old para 0 + 0 referred from Embu Provincial Hospital where she had presented with complaints of failure to achieve menarche and 2 years history of lower abdominal pains. The pain was cyclical coming every 21 days and lasting 7 days. Non-steroidal analgesics relieved this pain. There was no associated per vaginal discharge. No diarrhoea or vomiting. She had not achieved menarche yet. She had no urinary symptoms.

Past medical history

She had been admitted to Embu Provincial Hospital for the same problem before being referred to KNH after examination under general anaesthesia. She had no history of any chronic illness, and no known drug or food allergies.

Family and social history

She was a class 8 pupil in Embu, where she lived with her parents. She was a 4th born in a family of 4 siblings. Her other 2 sisters had no similar illness.

Systemic enquiry elicited no significant findings.

PHYSICAL EXAMINATION

General examination

She was in good general condition. She was not pale, had no oedema, lymphadenopathy or oral thrush. She had well-developed female secondary sexual characteristics. Her breast development was Tuner Class II with normal pubic hair distribution (feminine) and habitus. Her blood pressure was 110/70 mmHg; the pulse rate was 74 beats per minute and the temperature was 36.5°C

Abdominal examination

The abdomen was not grossly distended and moved with respiration. There were no surgical or therapeutic marks seen. She had a pelvic mass corresponding to 14 weeks gestation, associated with slight suprapubic tenderness.

Pelvic examination

External genitalia were found to be normal with normal female pubic hair distribution. The labia majora, minora and the clitoris were all normal. The vaginal cavity was found to be sealed by a thick ?mucous tissue and the cavity could not be accessed digitally.

Per rectal examination

Upper vagina was found to be distended with what was thought to be possible accumulated menstrual blood. Uterus was found to be bulky, soft and boggy.

The nervous, respiratory, cardiovascular and musculoskeletal sysyems were essentially normal.

Impression

An impression of vaginal septum with haematocolpos and haematometra was made.

The following investigations were done to confirm diagnosis:

1. **Pelvic Ultrasound Scan**: Showed that the uterus and cervical cavities were filled with echogenic material dilating the cavities indicating haematometra and haematocolposthe vaginal stripe appeared short and thickened. Both ovaries were normal in size and echopattern. No adnexal mass was seen.

2.	**Urea and electrolytes**:	Urea	6.5 mmol /L (1.7 - 8.3)
		Na^+	136 mmol/L (135 - 145)
		K^+	3.7 mmol/L (3.5 - 5.0)
		Creatine	5.2 umol/L (40 - 110)
3.	**Haemogram**	Hb	13g/dl

Management.

The diagnosis was communicated to her as well as her parents. The mode of management was also discussed with her and the parents both of who were in agreement. A written consent was obtained from her father and surgery was scheduled for Thursday (under the elective list).

The evening before theatre, she was shaved and enema given at 6pm and repeated again at 6am. She was starved from midnight. Atropine 0.6mg intramuscularly and Pethidine 75 mg were given half-hour before theatre.

Reconstructive surgery and postoperative care.

In theatre, general anaesthesia was induced. She was put in dorsal lithotomy position and aseptic catheterisation done. Examination under anaesthesia (EUA) confirmed earlier findings. Blunt dissection of the thick septum was done. This was extended up to the cervical region. Dark - chocolate coloured blood was drained. An inflated Folley's catheter was left in the uterine cavity, which was to be removed after 72 hours. A vaginal pack with souffratoule was also left in the vaginal canal and was to be removed after 48 hours. The urinary catheter was also left in situ for 72 hours.

General anaesthesia was reversed successfully. Her vital signs were monitored 1/4 hourly till she was fully awake and there after 4 hourly. She received Pethidine 100mg intramuscularly 8 hourly for analgesia for the first 24 hours, then changed to Diclofenac 50 mg per oral 8 hourly. She also received parenteral antibiotics i.e. Augmentin and Metronidazole as well as Betadine vaginal douches twice daily. She did well and was discharged home on the 5th post-operative day. She was to be reviewed in GOPC after 6 weeks.

Follow up.

She was seen at the gynaecology out patient clinic after six weeks. She was found to be in good general condition. Her vital signs were within normal and she did not raise any complaints. The operation site had healed well and she had experienced her first normal menstrual period with no difficulties. She was discharged from the clinic.

DISCUSSION

The patient presented at 16 years of age with a vaginal septum and haematocolpos as well as haematometra. She underwent treatment where the septum was dissected off and the haematometra and haematocolpos drained. She started experiencing normal menstrual flows. A review 6 weeks later confirmed the patency of the hymen was still present.

The vaginal lumen is separated from the urogenital sinus by the hymeneal membrane. The hymen is the junction of the sinovaginal bulbs with the urogenital sinus.[1] The hymen usually ruptures before birth due to degeneration of the central epithelial cells. However, a thin fold of mucous membrane persists around the vaginal introitus.[2]

Hymeneal anomalies are derived from incomplete degeneration of the central portion of the hymen. These anatomic variants include imperforate, microperforate, septate, and cribriform hymen.[3]

An impeforate hymen is one of the most common obstructive lesions of the female genital tract. At birth, infants may have a bulging introitus due to mucous plug from the vaginal secretions stimulated by maternal estradiol. If the diagnosis is not made in the newborn period and the hymen remains imperforate the mucus will be reabsorbed and the child usually remains asymptomatic until menarche. At that time, the adolescent girl may present with a history of cyclic abdominal or pelvic pain and hematocolpos, which may give the hymeneal membrane a bluish discoloration. Marked distension of the vagina may also result in back pain, pain with defecation or difficulties with urination.[4]

Repair of the hymen can be performed at any age; however the repair is facilitated if the tissues have undergone oestrogen stimulation. Therefore, surgery is ideal in the newborn, postpubertal, or premenarchal time periods. Surgical repair consists of an incision in the membrane close to the hymeneal ring followed by evacuation of the obstructed material. Extra hymeneal tissue is excised to create a normal sized orifice and the vaginal mucosa is sutured to the hymeneal ring to prevent adhesion and recurrence of the obstruction.

Review is done after 4-6weeks to reduce chances of introducing infection while doing a pelvic assessment.[5] The great distensibility of the vagina protects the adolescent patient with imperforate hymen from abnormal retrograde menstruation. Subsequent development of pelvic endometriosis is unlikely as long as the diagnosis is made early.[2]

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CASE NUMBER 10

ACUTE RUPTURED TUBAL PREGNANCY – PARTIAL SALPINGECTOMY

Name	:	M.E.O.	DOA	:	20/09/2004
Age	:	30 years	DOD	:	23/09/2004
Parity	:	4 + 1	LMP	:	15/6/2004
File No	:	0983825	Ammenorrhoea	:	14 weeks

Presenting complaint

MEO presented to the Kenyatta National Hospital (KNH) casualty at 10.05 a.m with a sudden onset lower abdominal pain for 6 hours.

History of presenting complaint

She had been well until about 6 hours prior to admission when she suddenly developed lower abdominal pains that were initially more on the left iliac fossa. The pain then spread to involve the lower abdomen and later the entire abdomen. There was no history of prior abnormal vaginal discharge, symptoms of urinary track infection or change in the bowel habit. Her last menstrual period (LMP) was on 15/06/04. She was on 3 monthly injections of medroxyprogesterone acetate (Depo provera), which had made her to have menses for 3 days as usual but prolonged the inter-menstrual interval from 30 days to 60 to 90 days. Therefore, her ammenorrhoea of 14 weeks could be less for real. Her last injection of Depo provera was on 16/08/04 and she had received another one about 16/05/04. There was a positive history of shoulder tip pain.

Obstetric and Gynaecologic History

She was para 4 + 1 with ammenorrhoea for 14 weeks. She had had uneventful spontaneous vertex deliveries to 4 male infants all in hospital at term. Their birth weights ranged from 3 to 4 kg. The first 2 deliveries occurred in 1994 and 1996 respectively. In 1997 she had a right tubal ectopic pregnancy and underwent laparatomy and right partial salpingectomy at KNH. Surgery was uneventful but she had to be transfused 2 pints of whole blood. The next 2 deliveries took place in 2000 and 2001. She attained menarche at 15 years and her normal menstrual cycle was of a flow for 3 days every 28 to 30 days without any menstrual disorder. She had always used Depo provera injections between pregnancies since 1994. When on the Depo Provera the inermenstrual period is prolonged to 60 to 90 days. She denied history of sexually transmitted disease, abnormal vaginal discharge or chronic lower abdominal pain. She had never done any Pap smear cytology test.

Past medical history

Besides the blood transfusion mentioned above, this was insignificant.

Family and social history

Our client was married housewife who schooled up to Form 3, drank no alcohol and did not smoke any tobacco. She lived in Karen, Nairobi; with her husband who worked as an accountant with a legal in Nairobi. He did not smoke cigarettes neither did he drink alcohol. There was no history of chronic illness.

PHYSICAL EXAMINATION

General Examination

She was sick looking, moderately pale, anxious and in pain. Her pulse was of low volume at 96 beats per minute. The blood pressure was 105/65 mmHg, RR was 22/minute and temperature was 36.2 °C. There was no jaundice, fever, pedal edema or lympadenopathy.

Abdominal examination

It was mildly distended and moved less with respiration. She had a sub-umbilical midline scar (previous ectopic pregnancy), guarding and rebound tenderness. There was no organomegally.

Pelvic examination

She had normal external genitalia, healthy vaginal mucosa, smooth and closed cervix and a bulky anteverted uterus. Marked bilateral adnexal tenderness and full cul de sac were noted. No abnormal discharge was noted on the examining finger.

Systemic review and examination bore no contributory findings.

Paracentesis was positive for no-clotting blood.

Diagnosis

Ruptured Ectopic Pregnancy with Impending Hypovolaemic Shock.

Management

The patient was appraised on the diagnosis and the management. Blood was taken for urgent grouping and cross match and haematocrit. Consent was obtained while resuscitating her. Resuscitation included fixation of a wide bore cannula and infusion of crystalloids (0.9% normal saline) and colloids (Haemacel/ dextran

70%). Premedication with intramuscular atropine 0.6 mg was administered and the patient wheeled to theatre for emergency laparotomy. She requested surgical sterilizationand signed consent for it.

In Theatre

She was positioned in semi-lithotomy, vulvovaginal toilet done and catheterization drained 100 mls of concentrated urine. She was then put in supine position, abdomen cleaned and, under general anaesthesia, opened via the previous subumbilical laparatomy scar. There was haemoperitoneum of 1500 mls, dense adhesions covering both fallopian tubes and ovaries. Previous partial salpingectomy was noted on right tube. There was a ruptured left fimbrio-ampulary tubal ectopic pregnancy with dense blood clots around it. The uterus was slightly bulky without any anomaly. The rest of the abdominal viscera were essentially normal. Partial salpingectomy was done thereby rendering her surgically sterile. Peritoneal lavage was done and abdomen closed after accounting for all the instruments used. Blood transfusion was started as soon as the bleeding ectopic pregnancy had been clamped. A second cannula was fixed for concurrent fluid infusion. Reversal of the general anesthesia was smooth. The immediate postoperative urine output was 50 mls of clear concentrated urine within 45 minutes while the vital signs were: BP; 110/ 70 mmHg, PR; 90/minute. RR 20/minute and temperature of 36.6 o C.

Postoperative Care

Her recovery was good. She got 3 units of blood in total and her pre- and 3rd day postoperative haematocrit were 21.2% and 29.7 respectively. Intravenous antibiotics and fluids were discontinued on the 1st postoperative day when bowel sounds were found to be present and normal and oral medications started. Oral sips preceded gradual introduction of light diet and by the 2nd postoperative day was on normal diet and ambulant. She was discharged on the 3rd postoperative day on haematinics with appraisal on the surgery and advice to attend the Gynaecology outpatient clinic (GOPC) in 6 weeks' time. HIV testing was advised in view of the blood transfusion.

Follow up

She was seen in the GOPC as advised. Her general condition was good. She had no pallor. The abdominal scar had healed well and her normal LMP took place 1 week prior to the review date. She was advised to have a Pap smear done and was to be reviewed with the result 1 month later.

DISCUSSION

M.E.O was a 30 year old para 4+1 who had a left ruptured fimbrio-ampulary ectopic pregnancy. She had a previous right tubal pregnancy and partial salpingectomy done. Laparotomy and partial salpingectomy was done. This was followed by an uneventful recovery.

Blastocyst implantation in a site other than the endometrial lining of the uterine cavity is termed ectopic pregnancy [1,2]. The true incidence of ectopic pregnancy is difficult to determine accurately due to the difference in population groups studied with different risk factors. The incidence varies with race and socio-economic factors. In the United States, it is reported to vary between 0.25% and 1.4% of all pregnancies with an average of 1%. The incidence for non-white women was higher in every age category than for whites with an overall 1.4 times increased risk for non-white women compared to white women [1,2]. In Jamaica, the evidence is reported at 1 in 28 deliveries [3] while at the Kenyatta National Hospital, Webala reported an incidence of 1 ectopic pregnancy for every 15 full term pregnancies [4]. Mwathe found that 4-5 patients are admitted with ectopic pregnancy every week in the same hospital [5]. The incidence is increasing worldwide, for example, in the USA between 1970 and 1989, the rate has increased five-fold [6]. This has been attributed to an increase in the incidence of sexually transmitted disease and pelvic inflammatory disease, the efficacy of modern therapy for pelvic inflammatory disease which in the past would have resulted in complete tubal occlusion and sterility, the widespread use of intrauterine contraceptive devices, an improved method of diagnosis and reporting and an increase in tuboplasties and assisted conception [1,3,7,8]. After an ectopic pregnancy, there is a 7-13 fold increase in the risk of a subsequent ectopic pregnancy. The chance that the subsequent pregnancy will be tubal is 10-25% 9. Our patient had a previous ectopic gestation although she was lucky to have had 3 normal pregnancies after the first ectopic pregnancy.

Despite the significant rise in the incidence, the mortality rate due to ectopic pregnancy has decreased due to early diagnosis and inervention [7,8]. However, it remains one of the main causes of maternal mortality. In the USA, 15% of all maternal deaths are attributed to complications of ectopic pregnancies[9] while here in Kenya, Makhokha found that ectopic pregnancy caused 5.1% of all maternal deaths at the Kenyatta National Hospital between 1978 and 1987 [10].

Ectopic pregnancy is most commonly found in the oviduct in 95% of the cases, and over 75% are diagnosed before the 12th weeks of gestation [1,2]. The ampulla is the commonest site with approximately 55% of tubal pregnancies, isthmus-25%, infindibulum and fimbriae-17% and interstitial segment 2-4%. Our patient had an ampulo-fimbrial pregnancy. Other sites include the ovary, abdomen, broad ligament, the rudimentary horn of the bicornuate uterus, the cervix, vagina and the myometrium all accounting for the

remaining 5%. At Kenyatta National Hospital, Webala found that 61-64% of the ectopic pregnancies occurred in the distal 2/3 of the tube [4]. Ectopic pregnancy occurs more on the right side than on the left side probably due to local influence of appendicitis [4].

Other rare forms of ectopic pregnancies include heterotropic pregnancy, which occurs when there is a co-existing intrauterine pregnancy and ectopic pregnancy. This is common in women undergoing in vitro fertilization [9]. Multiple ectopic pregnancies have also been reported though they occur less than heterotropic gestation. Pregnancy after sub-total hysterectomy may occur because the patient has a cervical canal that may provide intra-peritoneal access. It could also occur during the peri-operative period after hysterectomy secondary to a vaginal mucosal defect that allows sperm into the abdominal cavity [9].

The primary causes of ectopic pregnancy include conditions that prevent or impede passage of fertilized ovum through the uterine tube. These include; chronic salpingitis, adherent folds of tubal lumen due to salpingitis, isthmica nodosa, congenital abnormalities of the tube, abnormal tubal anatomy due to di-ethylstilboestrol (DES) exposure in utero, previous tubal or pelvic organ microsurgery, tubal ligation, conservative management of unruptured tubal pregnancy, extrinsic adhesions, pelvic tumours, endometriosis, excessive tubal length or tortuosity, tubal spasm or inadequate peristalsis [1,2,3,7]. Webala found evidence of chronic salpingitis in 69% of the cases at KNH [4]. Ovarian factors predisposing to ectopic pregnancy include fertilization of an unextruded ovum, ovum transmigration, post mid-cycle ovulation and fertilization and treatment with ovulation induction drugs such as clomiphene citrate [1,3]. Zygote abnormalities such as chromosomal anomalies and neural tube defects are also risk factors for ectopic pregnancy [1,3]. Exogenous progesterone hormone administration such as progesterone-only pill, failure of post-coital contraceptive pill and the use of IUCD's containing progesterone have been shown to increase the risk of ectopic pregnancy. This was the case in our patient. Progesterone is thought to act by decreasing ciliation and cell height in the tubes [2,3]. Other risk factors include the maternal age (with the highest rate occurring in women aged 35-44 years), tubal abortion and subsequent implantation, any form of intra peritoneal bleeding and assisted conception techniques such as in vitro fertilization and embryo transfer [1,3,7,8,9]. Current cigarette smoking has been associated with more than two fold risk of tubal pregnancy [9].

In the pathophysiology of tubal pregnancy, the fertilized ovum promptly burrows into the epithelium of the tube with limited resistance for the trophoblasts and at the same time, maternal blood vessles are opened [7]. The foetus or embryo is often stunted. The uterus undergoes some element of early pregnancy changes. These changes include enlarged epithelial cells, with hypertrophic and hyperchromatic, lobular and irregularly shaped nuclei. The cytoplasm is vacuolated, roomy with occasional mitosis. These changes in

the endometrium ('Aria Stella' reaction) are not specific for ectopic pregnancy and may occur in normal pregnancy [2]

Fifty percent of all ectopic pregnancies may abort, get absorbed, mummify or become chronic [7]. Rupture is usually spontaneous. Isthmic pregnancies tend to rupture at 6 to 8 weeks gestation, due to the small diameter of this portion of the tube. Ampullary pregnancies rupture later, generally 8-12 weeks. intestitial pregnancies are the last to rupture, usually at 12-16 weeks, as myometrium allows more room to grow than the tubal wall [1].

No specific symptoms or signs are pathognomic for ectopic pregnancy. The patient may or may not have symptoms pointing to pregnancy and she may not have a period of amenorrhoea [1,3,7]. Thus, a high index of suspicion is very important for the diagnosis of ectopic pregnancy. Lower abdominal pain or pelvic pain as was seen in our patient is the commonest symptom and occurs in 99% of the cases. It is often present even before rupture. The pain may be caused by distention of the tube and separation of the layers of the muscle by the blood but more severe pain is due to presence of blood in the peritoneal cavity. The pain may be generalized, unilateral, sub-diaphragmatic or sharp shoulder pain from irritation of the diaphragm [1,3]. Abnormal uterine bleeding occurs in 75% of the women irrespective of the site and may be mistaken for late onset of menses. The bleeding is scanty and results from sloughing of the deciduas. This was not there in our patient. The classical picture of ectopic pregnancy is a triad of abdominal pain, amenorrhoea and irregular vaginal bleeding [1,3]. A rough working rule is that, if a patient who is a few weeks pregnant complains of a little pain and heavy vaginal bleeding, the pregnancy is probably intra-uterine, whereas if she has much pain and little bleeding, it is more likely to be ectopic [1]. Other symptoms my include syncope in 37% of cases [1].

The commonest physical sign is abdominal tenderness often with rebound. Cervical excitation may also be present. About 50% of the patients will have an adnexal mass and in the majority, the uterus is normal size. Blood pressure and pulse correlate with the amount of haemoperitoneum. Most patients are afebrile unless they have a concomitant infection. This aids in the differntial diagnosis from acute PID in which fever is present [1]. Our patient had hypotension and tachycardia, she had abdominal tenderness, cervical excitation was positive and an adnexal mass was appreciated. The major gynaecological conditions that mimic ectopic pregnancy are ruptured or twisted ovarian cyst, acute pelvic inflammatory disease, and tubo-ovarian abscess. Others are appendicitis, uterine abortion, urinary tract infection, degenerating fibroids and normal intra-uterine pregnancy [1,2,3].

Investigation that may aid in the diagnosis of ectopic pregnancy include [1,2,3,7,9].

- Biochemical tests which include pregnancy test to detect ß-hCG. Most urine pregnancy tests have a sensitivity equivalent to serum ß-hCG of 500-750 mIU/ml and are positive on 50-80% of cases. A negative test, therefore, does not rule out ectopic pregnancy.
- Serum progesterone levels may also be used to rule out ectopic pregnancy with those with levels greater than 25ng/ml seen in less than 2% of ectopic pregnancies and in less than 4% of abnormal pregnancies. A progesterone level less than 15ng/ml is seen in 80% of ectopic and 90% of abnormal intrauterine pregnancies and 11% of normal pregnancies.
- Others are alpha-feto-protein and serum amylase, which may be elevated. These were not done in our patient.
- Ultrasonography:- transvaginal sonography can detect an intra-uterine gestational sac by 33 days gestation and 6 week gestation by trans-abdominal sonography. Vaginal sonography in diagnosing tubal ectopic has a sensitivity of 96% and specificity of 99%. A combination of pregnancy test and sonography gives a positive predictive value in 95% and the negative predictive value in 100% of cases.
- Laparoscopy is now the gold standard in diagnosis of early unruptured ectopic pregnancy. It is advantageous in that it can also be used as definitive management of early ectopic pregnancy.

Other tests are:

- Haemogram: which may show leucocytosis
- Culdocentesis and paracentesis: useful in cases of intra-peritoneal bleeding. Culdocentesis reveals non-clotting blood in 95% of cases. In our patient, paracentesis was positive for non-clotting blood.
- Culdoscopy and posterior colpotomy have also been used but are associated with other risks.
- Dilation and curettage shows curettings without chorionic villi. 'Arias-stella' reaction may be demonstrated.

The definitive management of tubal pregnancy is either surgical or medical. Shock should be urgently treated in case of ruptured ectopic as was the case in our patient. Intravenous fluid infusion should be commenced immediately and blood drawn for grouping and cross matching with request for sufficient pints for transfusion. In life-threatening situations, immediate control of further bleeding via laparotomy is called for. Auto-transfusion of blood from the peritoneal cavity can also be done.

Surgical treatment may be conservative or radical. Conservative surgical treatment my be either salpingostomy, segment resection and anastomosis or fimbria expression [11]. Conservative management should be attempted for all suitable cases normally, unruptured isthmic or ampullary tubal pregnancy in a patient desiring future fertility. The tubes can be accessed by laparoscopy or laparotomy. Laparoscopic constructive surgery is now the gold standard for the management of ectopic pregnancy in a haemodynamically stable patient. It should be used for unruptured tubal pregnancy less than 5cm in diameter and not in the cornua [11]. Uncontrolled bleeding calls for immediate laparotomy. Radical treatment includes salpingectomy as in the case of our patient or slapingo-oopeherectomy. The rate of repeat ectopic pregnancy is similar for both radical and conservative surgery but the rate of intrauterine pregnancy is higher following conservative treatment.

Non-surgical treatment involves the use of systemic drugs such as methotrexate and actinomucin D or local administration of drugs such as methotrexate, potassium chloride, hyperosmolar glucose, mifepristone, and prostaglandin F2α (PGF2α) or PGE2α [12]. Systemic methotrexate has been the most used drugs with a single dose of 50mg/m2 given intra-muscularly.

Selection criteria for methotrexate treatment are:

- Haemodynamical stability of the patient.
- No evidence of tubal rupture of significant intra-abdominal haemorrhage
- Tube less than 3-4cm in diameter.
- No contra-indication to methotrexate.
- Patient's availability
- No contra-indication to methotrexate.
- Patient's availability

With this criteria one expanded clinical trial showed that 94.2% had complete resolution with treatment and 5.8% required surgical anagement of the ectopic pregnancy while 3.3% required a 2nd course of methotrexate [13]. Methotrexate 10mg injected directly into the ectopic gestational sac under trans-vaginal ultrasound guidance has also been successful in 83% of cases [14].

Anti-D immunoglobin should be given to rhesus negative mothers [2]. Post-operatively, serum ß-HCG should be measured weekly following conservative surgery. Conception rate following ectopic pregnancy is about 60% with only half of them resulting in live births. In vitro fertilization offers hope for some patients who have been rendered infertile after ectopic pregnancy [8]. Our patient had her desired family size the partial salpingectomy completed the bilateral tubal ligation that began with the previous ectopic pregnancy.

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GYNAECOLOGY CASE NUMBER 11

PELVIC INFLAMMATORY DISEASE: ACUTE ABDOMEN – EMERGENCY LAPARATOMY: PYOSALPINX

Name	:	J.W.K	DOA	:	18/08/2004
Age	:	32 Years	DOD	:	21/08/2004
Parity	:	1 + 4	LMP	:	06/08/2004
File No	:	0188076	Ammenorrhoea	:	None

Presenting complaint

JWK presented to the Kenyattta National Hospital casualty unit with 3 days' history of sub-acute onset lower abdominal pain that was colicky with a baseline burning pain.

History of presenting complaint

She had been well until the onset of the abdominal pains that were associated with a mild greenish-yellow vaginal discharge. The pains were progressive and were not associated with symptoms of urinary track infections or change of bowel habits. She had associated hotness of the body and chills but no headache or vomiting.

Obstetric and gynaecologic history

She was a para 1 + 4. her last normal menstrual period was 12 days prior to admission, on 6/08/04.Her first pregnancy ended in a term normal vaginal delivery of a term male infant who cried immediately, weight 3.4 kg and was alive and well. Her 2nd pregnancy was a tubal ectopic pregnancy that led to an emergency laparotomy at KNH and subsequent blood transfusion. Intraoperative findings were ruptured left ampullary pregnancy, matted gut and right fimbrial adhesions to the ovary. These were released and recovery was excellent. The next 3 pregnancies ended in 2 abortions in 1994 and another one in 2002 all at 2 months gestation. No obvious cause had been found despite uterine evacuation in Kiambu District Hospital every time she had an abortion. She attained menarche at 14 years and since then had menstrual flow for 3 days every 28 to 30 days. She had no postcoital or intermenstrual bleeding or any other menstrual disorder. She had had no Pap smear done and denied history of multiple sexual partners with both her husband and/or herself. Because she had only one child and was keen on another one, she had not used any modern contraceptive method.

Past Medical History

This was insignificant

Family and Social History

She was a married business lady who only completed primary education, smoked no tobacco and drank no alcohol. She ran a vegetable stall in Mathare North where she lived with her husband who worked in a dry-cleaning shop. There was no history of chronic illness.

PHYSICAL EXAMINATION

General examination

She was sickloocking, febrile with no pallor, jaundice or lymphadenopathy. Her blood pressure was 120/80 mmHg, Pulse was 84/minute; RR 18m/minute and her temperature was 37.8 °C. The skin had "goose pimples".

Abdominal examination

The abdomen was flat and moved minimally with respiration. There was a subumbilical midline scar (for the previous laparotomy and salpingectomy). Superficial palpation indicated suprapubic tenderness while deep palpation showed guarding and rebound tenderness. No organomegally or masses were felt.

Pelvic examination

She had normal external genitalia, but scanty purulent vaginal discharge was noted on the introitus. The vaginal mucosa was healthy. The cervix was 3 cm long, with a smooth surface and a closed os. The uterus was normal sized but retroverted. Positive bilateral cervical excitation test was noted and the pouch of Douglass was boggy. The right adnexae was quite tender and it was not possible to delineate whether there was a mass or not. There was sero-purulent, foul-smelling discharge on the examining finger.

The respiratory, cardiovascular, muculo-skeletal and the nervous systems were essentially normal.

Impression

An impression of acute pelvic inflammatory disease with possible pelvic abscess was made.

Management

She was admitted and started on intravenous crystalline penicillin, metronidazole and gentamicin. Blood was drawn for a full haemogram and urea and electrolytes. A blood smear for malaria parasites, urine for microscopy, culture and sensitivity and pregnancy tests and a pelvic ultrasonograph were also done. The results were as follows:

Investigations:

1. **Haemogram:** Haemoglobin 10.9 gm/dl
   White cell count 11.6 x 10^9/L: neutrophils; 80%, lymphocytes 17%.
   Platelets 230 x 10^9/L
2. **U/E/Cs**: Potassium: 3.8 mmol/L, Sodium 140 mmol/L, Creatinine: 66μmol/L
3. **Pregnancy test**: Negative
4. **Pelvic ultrasound**: Retroflexed, normal sized, non-gravid uterus with normal endometrial stripe; mixed echo mass (tubo-ovarian mass) in the right adnexal region with associated free fluid in the pouch of Douglass (POD) and the right adnexa. The left adnexal region was free. Features suggestive of PID.
5. **Counselingg for HIV** testing was offered but she opted to go for couple counseling and testing later.
6. **Urine analysis**, culture and sensitivity were with no abnormality.
1. **Endocervical swab** for microscopy, culture and sensitivity though desired could not be done, as there were no swab sticks.

On the second day of antibiotics the suprapubic tenderness persisted and on the basis of the above findings the patient was scheduled for emergency explorative laparatomy. Informed consent was obtained, patient shaved and premedication with intramuscular 0.6 mg of atropine and 50 mg of pethidine administered. Blood was also taken for grouping and cross match.

In Theatre

Under general anesthesia, vulvovaginal toilet preceded drainage of 50 mls of clear urine by catheterization. Examination under anesthesia revealed normal sized retroverted uterus and a right tubo-ovarian mass that felt cystic and measured about 6 x 7 cm. Abdominal toilet and opening via the previous subumbilical scar revealed normal sized uterus with fundal adhesions to the omentum. The right tube was distended and adherent to the POD. The right ovary was covered by dense adhesions. Only a small stump of the left tube was present indicating previous salpingectomy. Purulent fluid found in the POD was drained and taken for microscopy, culture and sensitivity. Further, 30mls of pus from the right pyosalpinx was drained by a

longitudinal incision and marsupialization of the superior aspect of the dilated tube. Adhesions were then released gently, peritoneal lavage done and the irrigation with rifocin and heparin undertaken. The abdomen was then closed after confirming correct account of all instruments. Reversal of the general anesthesia was smooth and postoperative urine per catheter was clear. Vital signs were normal.

Postoperative Care

The patient did well thereafter on continuation of the above antibiotics: crystalline penicillin, metronidazole and gentamicun. Her temperature settled to normal within 24 hours. The bowel sounds gradually improved to normal by the 2nd postoperative day. By the third day the patient was ready to go home. Microscopy of the pelvic speciments was pyogenic but no growth was obtained. She was discharged home on the 4th postoperative day on advice to be seen in the gynaecology outpatient clinic (GOPC) in 2 weeks' time. The nylon sutures were to be removed on the 7th day post surgery in the nearest health facility. Her husband was advised to purchase oral doxicyline and metronidazole for 10 days. They were to abstain from sex during treatment and if need be use the condom. She was advised to complete her family size as soon as possible three months after surgery and to have a pelvic scan to rule out possible ectopic pregnancy if she conceived. Further she was appraised on possible subfertility due to the PID.

Follow up

Two weeks later she turned up in the GOPC in good general condition. The abdomen was soft and vaginal examination revealed no abnormal discharge. She was referred to the KNH Voluntary Counseling and Testing (VCT) center for HIV testing and was to come back to the GOPC for Pap smear testing after paying for the test (she did not have the money that day).

DISCUSSION

The patient presented was a 32-year-old married lady who was para 1 + 4. She presented with a 3 day history of progressive increase in lower abdominal pain and per vaginal discharge which begun one week of her menses. At admission she had features of pelvic abscess. She underwent emergency laparotomy and salpingostomy of a pyosalpinx was done with good postoperative outcome. She was discharged after being counseled on preventive methods against sexually transmitted infections including the human immunodeficiency virus.

The term pelvic inflammatory disease (PID) denotes the clinical syndrome resulting from infection of the female upper genital tract, including the uterus, fallopian tubes, ovaries and peritoneal surface, not associated with surgery or pregnancy. It is thought to occur after the ascending spread of organisms from the cervix or vagina to the upper tract.[1] PID therefore comprises a spectrum of inflammatory disorders of the upper genital tract including any combination of endometritis, salpingitis, tubo-ovarian abscess or pelvic peritonitis.[2] These features were found in our patient.

Because PID cannot be diagnosed reliably from clinical symptoms and signs and is often asymptomatic or sub-clinicalits exact incidence is unknown

Factors associated with PID mirror those for sexually transmitted infections. Risk factors and risk markers for PID include[2] young age, multiple sexual partners, chlamydial and gonococcal cervicitis and vaginitis, vaginal douching, tobacco smoking and intra-uterine contraceptive device (IUCD). Besides history of prior tubal ectopic pregnancy that suggest prior PID, our patient had no obvious risk factor since she was married, denied history of multiple sexual partners and did not smoke cigarettes or drink alcohol. Barrier contraceptive use protects against PID. Oral contraceptives use modifies the manifestation of PID towards a less symptomatic disease. Oral contraceptive use may in fact protect against manifested PID.[2] Our patient had not used any contraceptives

Typically, PID is a polymicrobial infection with sexually transmitted organisms (65%) particularly C. trachomatis (45%) and N. gonorrhoeae (37%) implicated in most cases.[3] It has been estimated that 10-30% of women with gonococcal or chlamydial cervicitis develop PID if left untreated. Other organisms isolated are those associated with bacterial vaginosis. These include anaerobes like bacteroides, peptococcus, peptostreptococcus, clostridium welchii and veilonella. Other enteric and vaginal flora include escherichia

coli, group B streptococci, enterococci, gardnarella vaginatis, mobiluncus spp., genital mycoplasms and other gram-negative bacilli. Other less commonly associated microorganisms include actinomyces spp., which are typically linked to IUCD usage. PID may also be associated with mycobacterium tuberculosis, due to dissemination of the microorganisms via the blood stream rather than via ascending spread from the lower genital tract. In some areas of the world, Kenya included, organism such as schistosoma spp may cause granulomatous salpingitis. In our patient, heavy growth or proteus mirabillis and escherichia coli were grown on culture from the endocervical swab. Due to lack of pus swab kits, we were unable to do microscopy, culture and sensitivity of the pyosalpinx in our patient.

The most important natural barrier to the ascending spread of microorganisms into the uterus is cervical mucus. Several studies have suggested that the risk for developing PID may increase during menses or immediately after menses as in the case of our patient.[2] This may be due to the breach of the cervical mucus plug. Hormonal factors may also play a role in the pathogenesis of PID by affecting the structural and functional barriers preventing infection of the upper female genital tract. Since age is an important risk factor for PID, specific age related, hormonal or other host immune response-related factors in the cervix or cervical mucus may also manifest PID. As the organisms ascend, endometritis is an early manifestation of PID and most but not all women with PID have plasma cell endometritis. Next, salpingitis develops, which can lead to Pyosalpinx or tubo-ovarian abscess formation. Peri-hepatitis is associated with PID in 10-20% of cases. Our patient did not have right upper quardrant abdominal tenderness. In the case of gonoccocal PID, as it ascends from the endocervix, it causes oedema and induces an intense polymorphonuclear leucocyte response. Gonococci readily attach to the microvilli of non-ciliated mucosal epithelial cells, which they enter, resulting in cell damage and sloughing of ciliate cells hence the yellowish purulent discharged noted from the cervix. This was seen in our patient. Chlamydial organisms also attache to non-ciliated epithelial cells.

No concrete data is available on the pathogenesis of non-gonococcal, non-chlamydia PID as cited by Molander and Paavonen.[2] However, in bacterial vaginosis, this is characterized by a complex change vaginal ecology. The concentration of hydrogen peroxide producing lactobacilli is decreased. This leads to a massive increase in the concentration of microbial by-products, which are thought to destroy cervical host defense barriers, leading to ascent of microorganisms and their by-products to the upper genital tract.[1]

Clinical manifestation of PID ranges from asymptomatic or subclinical endometritis to symptomatic salpingitis, pyosalpinx, tubo-ovarian abscess, pelvic peritonitis and sometimes peri-hepatitis. Bilateral

lower abdominal pain is most common presenting symptom. Peri-hepatitis causes right upper quadrant abdominal pain mimicking acute cholecystitis. Other common symptoms are abnormal vaginal discharge, metrorrhagia, post coital bleeding, dysuria, fever and nausea. Our patient had symptoms of lower abdominal pain and abnormal vaginal discharge. She did not have urinary symptoms. Clinical signs PID, as seen in our patient, include cervical motion tenderness, uterine tenderness and bilateral adnexal tenderness.[2]

Investigations for PID include laboratory studies for detection of suspected organisms. The endocervical gram stained smear if positive for gram-negative, intracellular diploccoci is suggestive of N. gonorrhoeae. Cell culture has long been the 'gold standard' for confirmatory diagnosis of C. trachomatis. However, recently developed polymerase chain reaction tests have largely replaced cell culture and antigen tests in the diagnosis of C. trachomatis infection. Laparoscopy is now considered the 'gold standard' for confirming a diagnosis of PID. This is because it allows direct inspection of the fallopian tubes and surrounding pelvic anatomy. It also enables microbiologic sampling from the upper genital tract. Laparoscopy can also be used to characterized and grade the severity of PID. In addition, operative procedures can be performed during laparoscopy such as liberation of adhesions, peritoneal lavation, drainage and lavage of abscesses, which shortens the hospital stay and may improve outcome.[4] Operative laparoscopy also facilitates management of other conditions that cause differential diagnostic problems for instance, endometriomas, ruptured ovarian cysts adnexal torsions and appendicitis. Thus laparoscopy also augments the management of non-PID cases, which are difficult to discriminate from acute PID by clinical examination alone. However, in our set up, the routine use of laparoscopy to confirm a diagnosis of PID is limited by cost and availability. Hence instead of diagnostic and operative laparoscopy, our patient underwent an emergency laparotomy.

Trans-vaginal sonogrphy (TVS) and magnetic reasonance imaging (MRT) are other techniques introduced to augment clinical diagnosis of PID. Earlier studies have shown that TVS performs well in diagnosis of PID when the criteria include thickened fluid-filled tubes. TVS is superior to trans-abdominal sonography in diagnosis of pelvic abnormalities. Specific TVS findings include; wall thickness >5mm, cog-wheel signs, incomplete septa and the presence of cul-de sac fluid.[5] In the case of our patient she had a trans-abdominal pelvic ultrasound which showed slightly bulky uterus with some fluid collection in the pouch of Douglas.Other laboratory tests, which are not specific to PID but are often abnormal or elevated in an acute infection of inflammation and which may help in the diagnosis and monitoring of PID include erythrocyte sedimentation rate (ESR), quantitative C-reactive protein (CRP) and the total white blood cell count

(WBC). Other tests also include, pregnancy test, and urine for microscopy, culture and sensitivity. Our patient had a haemogram plus ESR, pregnancy diagnostic test and urine for microscopy culture and sensitivity done on her. These were not deranged. Another test often omitted is the HIV test. Reports have shown that HIV positivity rate in acute PID are more likely to have pelvic abscess, require prolonged hospitalization, respond more slowly to antimicrobial therapy and more often require change of antibiotics.[2] This test was not done in our patient although it was requested and the patient opted to have it done later.

It is on the basis of the above clinical features and investigations that the Centres for Disease Control and Prevention (CDC) have developed a criteria for diagnosis of pelvic inflammatory disease. These are,[6]

Minimum Criteria

- Lower abdominal tenderness
- Adnexal tenderness
- Cervical motion tenderness

Additional Criteria

- Oral temperature >38.3°C (101°F)
- Abnormal cervical or vaginal discharge
- Elevated erythrocyte sedimentation rate
- Elevated C-reactive protein
- Laboratory documentation of cervical infection with N.gonorrhoeae or C.trachomatis

Definitive Criteria

- Histopathologic evidence of endometritis on endometrial biopsy
- Tubo-ovarian abscess on sonography or other radiologic tests
- Laparoscopic abnormalities consistent with PID

Our patient satisfied the minimum criteria. She also had features suggestive of PID on sonography.

Patients with P.I.D. can either be managed as outpatients or as inpatients. Indications for admission according to CDC include[6];

- Surgical emergencies cannot be excluded (e.g. appendicitis)
- Pregnancy (PID may be seen in early pregnancy but is rare after 6 weeks gestation).
- Poor clinical response to oral antimicrobials

- Unable to follow or tolerate an outpatient oral regime
- Severe illness (toxic), nausea, vomiting, high fever >39°C.
- Tubo-ovarian abscess
- Immunodeficiency (e.g. AIDS, immuno-suppressive drugs, etc)

Our patient was admitted because she had features of acute abdomen and pyosalpinx on sonography

Due to the complex microbiology of PID, broad-spectrum antimicrobial coverage is recommended. The antimicrobial regimen should include agents known to be active against C.trachomatis, N. gonorrhoea and the broad spectrum of aerobic and anaerobic bacteria commonly detected in the upper genital tract of women with PID. In our set up, a combination treatment of doxycycline plus metronidazole is usually used for both in-patient and outpatient. It is well tolerated, easy to administer, rarely causes enterocolitis and is not very costly. However, one important disadvantage is that it does not provide adequate coverage against gonococci, which commonly a problem in our population. In this case, a single dose therapy for gonorrhoea is given for instance norfloxacin 800mg or ciprofloxacin 500mg. Recent data, although limited, show somewhat lower microbiological and clinical cure rates for doxycycline and metronidazole than other antibiotic combination regimens.[7]

The CDC has come up with recommended regimens for the treatment of PID in both out-patients and in-patients.[6] For the outpatient, it is recommended to give orfloxacin 400mg twice daily for 14 days plus metronidazole 500mg twice daily for 14 days. Alternatively, one can give a single dose of ceftriaxone250mg intramuscularly plus doxycycline 100mg twice daily for 14 days. Regardless of the regimen used, close outpatient follow-up should include re-evaluation within 72 hours. Those patients who fail to respond to outpatient therapy should be re-evaluated to confirm diagnosis and should be started on parenteral therapy. In the case of in-patients,intravenous ofloxacin 400mg is given 12 hourly plus metronidazole 500mg 8 hourly are given until the acute symptoms have subsided and continued for an additional 72 hours prior to changing to oral medication. An alternative regimen is intravenous ampicillin/salbactum (Amoxicillin/clavullanic acid in our set-up) 1.2g 8 hourly and doxycycline 100mg intravenously or orally 12 hourly. A third alternative is intravenous ciprofloxacin 200mg 12 hourly plus doxycycline 100mg 12 hourly plus metronidazole 500mg 8 hourly. Most clinicians advocate a conservative medical approach rather than surgical treatment of patients with tubo-ovarian abscess. However, treatment requires the use of clindamycin because of its superior ability to penetrate abscess cavity.[8] Failed medical therapy occurs often in patients who have an abscess larger than 8cm in diameter or

bilateral abscesses.[11] Our patient was put on intravenous metronidazole, gentamicin and crystalline penicillin and, though no recommended by the CDC, worked quite well. She was weaned of parenteral therapy after 48 hours and continued with oral antibiotics.

Surgical management of PID ranges from laparoscopy as mentioned above to laparatomy that is sometimes used to manage cases of ruptured tubo-ovarian abscess and severe peritonitis as was performed on our patient. Other surgical procedures include posterior colpotomy and drainage of the pus and preferably; irrigation with sterile solutions every 4 hours. This may negate the need for laparatomy. If colpotomy is to be done, the pocket of pus must be mid-line or nearly so, it should be adherent to the cul-de-sac and must dissect the reco-vaginal septum and it should be flactuant.[10] In our unit, colpotomy is not routinely done.

The patients should be counseled to abstain from coitus while ill and during treatment, as was done in our patient. Since most infected men are asymptomatic, male sex partners should be treated empirically with regimens that are effective against N. gonorrhoeae and C.trachmiatis.[2] Patients should be counseled on preventive methods especially use of barrier methods. In the case of our patient, her partner declined to accompany her for the counseling session. She was however given a full dose of the necessary antibiotics to give to him.

Complications of PID include subfertility due to tubal damage and pelvic adhesions, severe generalized peritonitis, septicaemia, pelvic abscess and increased rates of ectopic pregnancy occurrence. After a single episode of PID the risk for tubal factor infertility is approximately 8—11%. It is 20-30% for two episodes and 40-50% for three episodes.[11] Women with a history of PID have an approximately six-fold increased risk of tubal ectopic pregnancy compared to women with no history of PID. Chronic pelvic pain occurs in about 18% of those with previous PID infection. They are also approximately 10 times more likely to be admitted for pelvic pain and hysterectomy rates are eight times higher than in other women. Women with PID are also more likely to suffer long-term dyspareunia later in life. Our patient was counseled on the risk of sequelae associated with PID and advised to delay pregnancy by 3 months but to try conception as soon as possible therafter.

Strategies for prevention of PID can either be at primary, secondary or tertiary levels.[2] primary prevention involves counseling of lifestyle and health education. These include asking questions about risk-taking sexual behaviour, encouraging screening test for those at risk, ensuring that male sex partners are evaluated and treated and counseling about safe sex practices. Effective school health education programmes should be implemented especially among adolescents. Secondary prevention involves screening and treatment of pathogens, which include C. trachomatis. A programme for screening of C. trachomatis was undertaken in

Sweden. This lead to a dramatic decrease in the rates of hospitalization for PID.[12] Five to ten years later, this was followed by a significant fall in the rate of ectopic pregnancies especially in the young age groups. Tertiary prevention includes treatment that prevents upper genital tract infection from leading to tubal dysfunction or obstruction. Early diagnosis and therapy can reduce the need for surgical intervention.

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GYNAECOLOGY CASE NUMBER 12

UTERINE PROLAPSE, CYSTOCOELE AND RECTOCOELE – TOTAL VAGINAL HYSTERECTOMY AND ANTERIOR AND POSTERIOR COLPORRHAPHY

Name	: J.W.M	LNMP	: 28/01/04
Age	: 49 years	D0A	: 16/02/04
File No	: 0939304	DOD	: 25/02/04
Parity	: 7+1	Re-admitted	: 21/06/04
		Discharged	: 05/07/04

Chief complaint

JWM presented with a one-year history of feeling of fullness in the vagina.

History of Presenting Complaint

JWM was admitted via the gynaecology out-patient clinic (GOPC) with a diagnosis of relaxation of pelvic support structures and resultant cystocoele, rectocoele and uterine prolapse. She was to undergo a scheduled total vaginal hysterectomy (TVH) and anterior and posterior colpo perioneorrphy. She had been referred from Lamu District Hospital via the Coast General Hospital for the aforementioned surgery. This was because her eldest son lived in Nairobi and would take care of her after the operation. JWM had been well 1 year prior to admission when she experienced a feeling of vaginal fullness each time she coughed or opened bowels. This progressed to a feeling of something protruding in the vagina even on minimal stress like suddenly standing up and eventually the feeling of fullness persisted even without straining. She denied history of dysuria, post-micturation dribbling of urine, urgency of micturation or leakage of urine on straining but occasionally had feeling of incomplete bladder emptying and had to attempt to empty severally.

She was a known case of bronchial asthma for 23 years on ventolin tablets. She frequently coughed a lot and strained to breathe when with asthmatic attacks. There was no history of chronic constipation but conceded to carrying heavy loads to the local market. There was no associated vaginal bleeding or discharge neither did she have inter-menstrual or post-coital vaginal bleeding.

Obstetric and Gynaecologic history

J.W.M. was para 7 + 1 having had spontaneous vaginal delivery to all her seven living children and one spontaneous abortion. Her last delivery in 1988 was difficult in that she had delayed second stage for 2 to 3 hours before vacuum extraction was done only to find the infant had been in face to pubes position and weight 3.8kg. The other deliveries were uneventful and the birth weights ranged from 3 to 3.9kilogrammes. She had had three episiotomies. Besides bilateral tubal ligation in the early post-partum period after last delivery, she had used no other contraceptive method. She attained menarche at 16 years and had menstrual flow duration of 5 days with intermenstrual interval of 28 days regularly. The menstrual pattern had not changed.

Past Medical History

J.W.M. had had bronchial asthma for 23 years, had no specific allergies and controlled her asthmatic attacks with ventolin tablets. Besides episiotomies she had had no other pelvic surgery neither had she been admitted for other conditions other than for obstetric care and asthma.

Family and Social History

She was a married lady who sold foodstuffs in the local market. She did not consume alcohol or smoke tobacco and denied any familial disorder exept that her elder sister was asthmatic too. Her husband did miscellaneous businesses in Lamu.

PHYSICAL EXAMINATION

General Examination

J.W.M was a middle-aged woman in good general condition. She had no pallor, jaundice, pedal oedema or lymphadenopathy. Her blood pressure was 120/70 mmHg, pulse rate; 80 beats per minute, regular and of good volume; temperature, 36.5° C and respiratory rate of 18 breathes per minute.

Breast and Abdominal Examination

Her breasts appeared normal for her age and parity. There were no palpable masses. The abdomen moved uniformly with respiration, was flat and a subumbilical transverse BTL scar was visible. It was soft, non-tender and had no palpable masses.

Pelvic Examination

In semi-lithomy and a slight propped up position inspection revealed normal female external genitalia. Speculum examination showed a protrusion of anterior vaginal wall extended up to the level of hymenal ring on valsava manouvre and a positive cough impulse was noted. The cervix was in the mid position, looked parous, grossly normal and protruded up to the level of hymenal ring on straining. There was also a positive cough impulse on a slight protrusion of the middle posterior vaginal wall that did not extend up to the level of hymenale carunculae on straining. Digital examination confirmed the two protrusions and the associated positive cough impulses. The uterus was mobile and normal sized. Minimal pelvic muscle tone was appreciated when the patient was asked to tighten her perineal muscles. There was no abnormal vaginal discharge on examining finger. The sacral promontory could not be reached.

Based on the Pelvic Organ Prolapse Quantification (POP-Q) system of classification (see under "Discussion"), the patient had POP-Q stage II uterine prolapse and cystocoele and POP-Q stage I rectocoele. These stages correspond to second degree uterine prolapse and cystocoele and first degree rectocoele.

Respiratory Examination

There was no wheezing and the chest was symmetrical and without rhonchi, rales or any other sounds other than the vesicular breath sounds.

Musculo-skeleful, nervous and cardiovascular systems were essentially normal.

Impression:

The impression of relaxation of pelvic support structures with attendant second-degree uterine prolapse and cystocoele and first-degree rectocoele was made.

Investigations

1. Pap smear: Reactive inflammatory cells but no malignant cells noted.
2. Pelvic ultrasound: Normal sized anteverted uterus without fibroids and free adnexae and POD: No hydronephrosis was observed.
3. Renal Function Tests.
    - Sodium 134 mmol/L
    - Potassium 3.6 mmol/L
    - Urea 5.4 mmol/L

- Creatinine 72 mmol/L
- Haemogram: WBC: 70 x 10^9/L, Hb 14.2g/L, Platelets 294x10^9/L.

MANAGEMENT

Preparation for total vaginal hysterectomy was part of the aforementioned management and consisted of comprehensive counseling on what the surgery entailed, the entire procedure and the expected outcome. Informed consent was then obtained after which one unit of auto-donated blood was taken. A sample was taken for crossmatch of an extra unit. She was given 1 gram of metronidazole, starved overnight and enema given at 5.30 p.m. on the day of surgery. Abdominal and pubic hair was shaved that morning, premedication with atropine 0.6 mg and pethidine 50mg was given by the intramuscular route.

Operation

Aseptic spinal anaesthesia using 3mls of 2% bupivacaine was given in sitted and dorsiflexed position through the inter-vertebral space L3 - L4. The patient was then propped up at an angle of about 15° to prevent anaesthesia of proximal nerve roots. Once the anaesthesia had taken effect, she was put in dorso-lithotomy position, vulvo-vaginal toiled done and painted with iodine. On cathetirization with indwelling Folley's catheter, 50mls of clear urine was obtained. Examination under anaesthesia confirmed the 2nd degree uterine prolapse and cystocoele and 1st degree rectocoele. The adnexae and cul de sac were free.

An ovum speculum was placed in situ, cervix held with double-toothed volsellum forceps and traction applied downwards and posteriorly. A local vasoconstictor concoction ("Jungle juice") made of a mixture of adrenaline, lignocaine and 0.9% saline was infiltrated to create a line of cleavage and to prevent excessive bleeding. An elliptical incision was made on the anterior vaginal wall at the vesicouterine arch of the vaginal mucosa. By blunt dissection and using the Foley's catheter as a guide, the bladder was freed from the uterus. Serially, the vesico-uterine peritoneum was identified and divided laterally on both sides, the cervix was pulled anteriorly, the posterior fornix identified and the incision at the vesico-uterine level extended posteriorly avoiding rectal injury. Dissection was made until the cul de sac was identifiedthen the incision was extended laterally up to the base of the utero-sacral ligaments. The cardinal ligaments, the utero-sacral ligaments and the uterine vessels were progressively clamped and divided between clamps, ligated and transfixed. The tubes, the round and ovarian ligaments were also double clamped and ligated. The infundibulopelvic ligaments were then were then ligated between clamps and transfixed. The uterus and its appendages were then delivered. The pelvic peritoneum was then closed using purse string suture and the vginal vault was closed by approximation of the cardinal ligaments. The redundant vaginal mucosa

Post Operative Care

This consisted half hourly observations in the left lateral recovery position till she was fully awake then as per routine. Treatment comprised 3000mls of intravenous fluids of normal saline alternating with 5% dextrose in 24 hours, crystalline penicillin G 2 MU 6 hourly gentamicin, 80mg 8 hourly and flagyl 500mg 8 hourly for 48 hours followed by oral amoxycillin, and metronidazole. She received pethidine 100mg 6 hourly for 48 hours and later mefenamic acid. Her fluid input and output was satisfactory and the vaginal pack and foley's catheter were removed 24 hours and 48 hoursl respectively. Although recovery was uneventful she complained of feeling of fullness in the posterior aspect of the vagina. Pelvic examinations indicated the rectocoele had not been corrected. She was, therefore, discharged on the 4th post-operative day for review after 6 weeks in the GOPC.

When she turned up in the GOPC the rectocoele was noted to be persistent and was scheduled for posterior colpoperineaorrhaphy she was, therefore, admitted 3 months after the initial surgery. Her repeat renal function tests and haemogram were normal. Preparation for surgery and the anaesthesia was as before. Posterior colpoperineorrhphy was undertaken. This involved placing a pair of Allis tissue forceps on each side of the lower side of labium minus and 3rd forceps is place on the posterior vaginal wall in the midline well above the rectocoele bulge. A horizontal incision was made on the mucocutaneous junction joining the 2 Allis tissue forceps. The vaginal epithelium was then separated off the underlying rectovaginal fascia up to the 3rd forceps. A vertical incision was made from the apex to the middle of the horizontal incision (inverted 'T' shaped incision). Digital rectal examination identified the defect. Using size 2/0 polyglitin (Vicryl) on a round body, interrupted sutures were used to repair the recto-vaginal fascia particularly the defect and the lavetor ani muscles. Redundant vaginal epithelium was excised and the edges approximated using the same suture but on a cutting body. The anaestesia was reversed and the patient received the similar post-operative care as in the initial surgery. However, she developed moderate headache and nape pain on the second postoperative day. Her vital signs were normal and the repeat haemogram and a blood smear for malaria parasites had no abnormality. Analgesia was achieved with tramadol capsules and by the 4th post-operative day she was well enough to be discharged home to be seen in the GOPC in another 6 weeks.

Follow up

Compliant as she was the patient visited the GOPC in six weeks without any complaint. Pelvic examination revealed a well-healed vaginal vault and no recurrent cystocoele or rectocoele. The histology report, which indicated she had normal uterus and cervix save for features of chronic cervicitis, was discusses with her.

indicated she had normal uterus and cervix save for features of chronic cervicitis, was discusses with her. She went home as a happy patient on advice to resume coitus after 3 months, use the Kevigel's isometric pelvic exercises and to be reviewed after six months in the GOPC.

DISCUSSION

The patient presented was a 49-year-old para 7+1 who presented with second-degree uterine prolapse and cystocele and first-degree-rectocoele. She underwent total vaginal hysterectomy with anterior and posterior colpoperineorrhaphy with good postoperative outcome.

Genital prolapse is a downward or forward displacement of one of the pelvic organs from its normal location. It usually follow the relaxation of fascial and ligaments supporting the pelvic organs. Traditionally prolapse has referred to the displacement of bladder, uterus and rectum.[1,2] Utero-vaginal prolapse is a common gynaecological problem and is responsible for about 20% of women on the waiting list for major gynaecology surgery in Britain.[3] In a study in Oxford, utero-vaginal prolapse was the primary reason for 6.5% of all hysterectomies.[4] Genital prolapse is relatively uncommon in East Africa as compared with Europe and United States, though it was reported among the Pokot of western Kenya.[5] Mwalali reported an incidence of 0.1% at Kenyatta National Hospital.[6] Majority of the patients with genital prolapse are post-menopausal and of high parity.[1,2,3,4,7] Among the Pokot it was reported to occur in young and low parity women.[5]

All forms of female genital prolapse are described with reference to the vagina.[1] Classification of prolapse has usually been graded as scale of 0-3 with 0 referring to no prolapse and increasing in severity so that grade 3 is total prolapse. The following are variety of terms, which are used to describe female genital prolapse:

- A cystocele is a downward displacement of bladder.
- A cystourethrocoele is a cystocele that includes the urethra as part of the prolapsed organ complex.
- A uterine prolapse is descent of the uterus and cervix down the vaginal canal towards the vaginal introitus.
- A rectocele is a prolapse of rectum into the posterior vaginal lumen
- An anterocele is a herniation of the small intestines into vaginal lumen usually after hysterectomy.

The use of the most dependent position of the pelvic organ to rate the "degree" of prolapse into 1st, 2nd and 3rd degrees was noted to be subjective to some extend and hence the Pelvic Organ Prolapse Quantification system was formulated and first published in 1996.[8] It has more precise description of anatomy and site

specific measurements of the of the vaginal and perineal anatomy with the reference point being the plane at the level of the hymen, which is defined as zero.

Pelvic organ prolapse is generally a disease of the elderly.[8] The etiology is considered in terms of weakening of the pelvic floor and increased downward pressure.[3,8] Histochemical studies on biopsies of pelvic floor muscles have demonstrated evidence of denervation in these women.[9] Predisposing factors include congenital or developmental weakness of the supports, injury sustained during child birth (more in multiparous women) to endopelvic fascia, pelvic floor ligaments, laceration of pelvic muscles and those of perineal body,[1] iatrogenic injury as during hysterectomy, atrophy of supporting tissues at climacteric, and causes of increased intra-abdominal pressure such as chronic cough, obesity, ascites, pelvic tumours, heavy lifting, chronic constipation and sacral nerve disorders, caudal anaethesia, presacral tumour and fracture of the pelvis. [1,5,10,11].

Iatrogenic factors that may contribute to genital organ prolapse include failure to adequately correct all pelvic support defects at the time of surgery, ventrosuspension of the vagina that increases the exposure of the cul de sac to increase in intra-abdominal pressure, failure to detect and correct occult enterocele and, excessive shortening of the vagina.[10,11] Female genital prolapse occurs most commonly in multiparous women. However, the prolapse of the uterus can occur in nulliparous women especially when the cervix is congenitally long.[1,11] Our patient had the risks of developing uterine prolapse in that she was a grand multiparous lady with multiple episiotomy scars, had had a 3.8 kg baby in face to pubis delivered by vacuum extraction, had bronchial asthma for 23 years with resultant chronic cough and was perimenopausal.

Symptoms and signs associated with utero-vaginal prolapse include sensation of swelling or fullness in the vagina, bearing down sensation, a dragging discomfort in the lower abdomen and pelvis, backache, voiding difficulties, difficulty in emptying the rectum and discomfort during coitus. Completeness of voiding and defecation may require manual reduction of prolapse by the patient. The commonest symptom is a sensation of something coming down the vagina.[7] Mwalali at KNH found that majority of patients (97%) presented with a feeling of something coming down the vagina while 12% presented with urinary symptoms.[6] This patient presented with complaints of a mass coming down the vagina.

Complications of utero-vaginal prolapse include keratinization of the vagina, decubital ulceration of the prolapse, urinary tract infection resulting from incomplete emptying of the bladder, leucorrhoea, abnormal uterine bleeding, urinary tract infections and haemorrhoids resulting from straining to overcome constipation.[1,10] Downward movement of the uterus causes the lower ends of the ureters to be constricted

and this may lead to obstruction with resultant hydroureter and hydronephrosis.[10,11] The differential diagnosis includes cervical elongation, cervical tumour and endomentrial tumour such as endometrial and/or cervical polyps.

The patient presented did not have any of the above complications.

Management of uterine prolapse includes preventive, conservative and surgical measures. Asymptomatic pelvic prolapse does not require management. Preventive measures include prenatal and postnatal exercise, avoidance of traumatic delivery and oestrogen therapy for menopausal women.[1,9,11] For mild degrees of prolapse with mild or no symptoms, expectant management can be done. This will include taking measures to prevent or correct problems associated with prolapse such as obesity, constipation, and chronic cough. Postmenopausal women can be advised on estrogen replacement therapy. Patients should be taught the technique of perineal muscle exercises and be encouraged to do them regularly.[11] Vaginal pessaries may be used as a palliative therapy if surgical treatment is contraindicated or as a temporary measure in mild to moderate prolapse.[1,10,11] A vaginal pessary can also be used to promote healing of decubital ulcer prior to surgery. Infrequent removal and cleaning of the vaginal pessary can result in vaginitis and if forgotten in situ, may lead to erosion and fistula formation into the bladder.[11]

Surgical management is indicated in advanced and symptomatic pelvic organ prolapse 10, 11. Selection of surgical approach for uterine prolapse depends on the patient's age, her desire for future fertility or preservation of coital function, degree of prolapse and presence of associated conditions such as cystocele, stress incontinence or rectocele1. The type of operation done includes vaginal hysterectomy, anterior colporrhappy, posteriror colpoperineorrhapy, transvaginal enterocele repair and vaginal vault suspension. A combination of the above procedures can be done depending on individual patients. Abdominal hysterectomy can also be done [1,2,3]. For women of reproductive age who have completed their family size and postmenopausal women, vaginal hysterectomy is routinely done. The repair of cystocele and rectocele is done in same sitting. Complications of vaginal hysterectomy include, injury to bladder, infection of the vault, vault prolapse and haemorrhage. Prophylactic antibiotics are indicated to prevent sepsis. The patient presented did not develop any of these complications after vaginal hysterectomy and posterior and anterior coporrhaphy although posterior colporrhaphy was initially omitted erroneously. Such an error would not have occurred had the patient been examined in the standing or propped up position. Further, a rectal examination would have probably revealed a defect on the recto-vaginal septum.

When the preservation of fertility is important Manchester-Fothergill operation that includes anterior and posterior colporrhaphy with amputation of the cervix is performed.[1,11] Other methods include sacral cervicopexy-hysteropexy or shrodkar sling cervicopexy.[2]

Complications of surgery include haemorrhage, infection and injury to the contigious organs, blood vessels and nerves. Possible long-term complications include postoperative urinary incontinence, dyspareunia and recurrent pelvic organ prolapse.[1,3] The Manchester procedure is associated with cervical incompetence due to shortened cervix, or infertility due to loss of cervical mucus.[3]

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GYNAECOLOGIC CASE N°.13

VESICO-UTERINE FISTULA AND LIGATED LEFT URETER – REPAIR OF FISTULA AND URETERIC RE-IMPLANTATION

Name	:	K.M.M	DOA	:	13/07/2004
Age	:	40 years	DOD	:	04/08/2004
Parity	:	10 + 0	Ammenorrhoeic since last delivery on 17/12/2003		
File No	:	09666032			

Presenting Complaint

K.M.M admitted to the Kenyatta National Hospital (KNH) cold gynaecology ward (1B) with uncontrolled continuous leakage of urine since she delivered by emergency cesarean section on 07/12/2003. The leakage was noticed on the first postoperative day.

History of presenting complaints

She had been well until she went into labour at home for two days from 5/12/2003. She had intended to deliver at home as she had done on many occasions but when labour became protracted for more than 24 hours, she was taken to Mwingi District Hospital in Ukambani where she underwent an emergency cesarean section for obstructed labour. It was not clear whether concurrent bilateral tubal ligation (BTL) had been done. The outcome was a live male infant with a birth weight of 4.5kg and unknown Apgar score. No documentation of surgery was available. She noted urine leakage on the first postoperative day despite having an indwelling catheter, which was left in situ for 2 weeks. The leakage never stopped. She was referred to KNH in April 2004 but due to financial constraints she came to KNH in June 2004. She was examined in the vesico- vaginal fistula (VVF) clinic where a dye test indicated the dye oozing from the uterine external cervical os. This was suggestive of a high (type III) vesical fistula.

Obstetric and Gynaecologic History

KMM was para 10 +0 with 9 living children. All but the last deliveries were vaginal. Only 4 of these deliveries had been in hospital and only after protracted labour or in the presence of other complication. Her first delivery was in 1981. She had a stillbirth in 1997 after delivery at home. She had never used any contraceptive method neither had she done any Pap smear. Her menarche was 15 years and had menses for

3-4 days every 30 days without any menstrual disorder. She had not had menses for 7 months (since the cesarean section) unlike in the other deliveries when she could get menses 2-4 months postpartum.

Past medical history

This was of insignificant contribution.

Family and social history

She was a married lady who had not gone to school, drank no alcohol and smoked no tobacco. She lived in a rural home in Mwingi District with her peasant husband and children. Their home was 40 km from Mwingi District Hospital.

PHYSICAL EXAMINATION

General Examination

She was in fair general and nutritional status without pallor, pedal edema, jaundice or lymphadenopathy. Her BP was 130/80 mmHg, the PR; 80/minute, RR 20/minute and the temperature; 36.8 °C. The smell of urine was noted.

Abdominal examination

The abdomen was flat and moved with respiration. A sub umbilical midline scar (for the cesarean section) was noted. Palpation revealed no tenderness, organomegally or any masses.

Pelvic examination

She had excoriation of the perineum, grossly normal external genitalia and urine soaked clothing and a vaginal pad. Digital examination revealed anteverted normal sized uterus and free adnexae and the cul de sac. Sim's speculum examination showed healthy vaginal mucosa and urine leaking from the otherwise grossly normal external cervical os. This leakage was confirmed by the dye test performed by instilling methyline blue dye into the urinary bladder via a urethral Folley's catheter. Rectal examination showed intact external and internal anal sphincters and no rectal fistula or masses were noted. In view of this and the fact that the urine leakage occurred immediately after the cesarean section, the fistula was considered to be high (type III). The differentials were vesico-uterine, vesico-cervical, uretero-uterine or uretero-cervical.

The nervous, respiratory, cardiovascular and musculoskeletal systems were essentiqally normal.

Impression

High (type III) VVF. The differentials were vesico-uterine, vesico-cervical, uretero-uterine or uretero-cervical fistulae.

Management

The patient received a comprehensive explanation of the diagnosis of her disease and the necessary corrective surgery required through the abdominal route given the nature of her fistula. She was admitted with haemogram and renal functiona tests results, which were as follows:

1. Haemogram: Hb : 12.2 g/dl
   WBCC : 6.4 x 10^9/L, neutrophils; 67%; lymphoctes: 29%
   Platelets : 215 x 10^9/L
2. Renal function tests : Potassium: 5.0 mmol/L
   Sodium : 132 mmol/L
   Creatinine: 50 μmol/L

3. Intravenous urogram (IVU), though highly recommended in her case, was not done, because of financial constrains.

Preparation for the fistula repair included light diet two days prior to the operation day. Informed consent was obtained and the patient starved after supper on the eve of surgery. Ten milligrams of bisacodyl (Dulcolax) and enema at 6p.m. and at 5.30 a.m. were administered. Prior to being wheeled to theatre, she was shaved and premedication with 50 mg of pethidine and 0.6 mg of atropine given.

The Operation

Under general anesthesia, vulvo-vaginal toilet and catheterization with an 18 French Folley's catheter preceded examination under anaesthesia, which confirmed the earlier vaginal examination findings. Abdominal cleaning, draping and opening via a subumbilical incision were done. There was a small uterus that was adherent to the posterior vesical wall. BTL had been done. The right ureter was identified with normal peristaltic movement and caliber. The left ureter had marked hydronephrosis and no peristalsis was noted. The distal end was at the level of the isthmus of the uterus. There was no ureteric fistula. The bladder was separated from the uterus and the vesico-uterine fistula was identified. The uterine side of the fistula was refreshed by gentle dissection and then it was repaired in 2 layers with size 0 vicryl on round body (atraumatic) needle. The bladder was opened in the North- South direction. The right ureteric stoma was easily identified but the left could not be visualized despite intravenous frusemide injection and rigorous propping. The left ureter was dissected and resected at the distal end. It was then re-implanted into

the urinary bladder trough a small transverse incision on the bladder wall and fixed onto the bladder wall by external size 2/0 vicryl around the entry point. A ureteric catheter was inserted and minimal urine drained via the catheter suggesting possible left kidney atrophy due to chronic hydronephrosis. The bladder was then closed in 1 layer while avoiding its mucosa. To avoid chemical peritonitis, thorough peritoneal lavage was undertaken. After receiving a report of the correct account of all the surgical equipment, the abdoment was closed in four layers. Fifty milliliters of bloody urine was noted in the Folley's catheter. Reversal of the general anesthesia was uneventful.

Post Operative Care

She was transferred to the recovery room where vital signs were observed half hourly for 2 hours and then 4 hourly. Infusion of 4 liters/24 hours consisting of 5% dextrose alternated with 0.9% saline infusion was started in the recovery room and continued in the ward. Intravenous crystalin penicillin and gentamicin and intamuscular pethidine for infection and pain control respectively were administered. The ureteric and the urethral catheters were to be removed on the 12^{th} and 14^{th} days respectively.

Her recovery was satisfactory. On the first postoperative day the vital signs were normal, the abdomen was soft with normal bowel sounds. The urine in the Folley's urethral catheter was much clearer than the immediate postoperative period while that in the ureteric catheter was clear but only 30 mls had collected. Ambulation, 5 liters of oral fluids and oral amoxycillin and mefenamic acid were started. By the 3^{rd} day both catheters drained clear urine. The wound was exposed. A small portion through which the ureteric catheter passed was wet and iodine solution (Betadine) was to be applied twice daily. Apus swab taken from the portion grew E. coli sensitive to ciprofloxacin that the patient received and the wound healed well thereafter. No urine leakage occurred throughout the postoperative period even after removal of the two catheters on the 12^{th} and 14^{th} days as ordered. A methyline blue dye test done on the 19^{th} postoperative day was negative (No leakage was noted). She was discharged on the 20^{th} postoperative day through the VVF clinic

Follow up

She was reviewed in the VVF clinic 2 and 6 weeks later. In both occasions the patient was in good general codition and no urinary leakage was noted. Contraceptive advice was not offered because BTL had been done. She was to do an IVU in 6 weeks when she hoped to have gotten some money for the test then she would be reviewed with it.

DISCUSSION

Our patient was a 40 year-old- para 10 + 0 who had a vesico-uterinel fistula after an emergency cesarean section for obstructed labour. She underwent successful fistula repair and left ureteric re-implantation.

A communication between two epithelial surfaces is called a fistula. Examples include enterocutaneous fistulae (communication between the gut epithelium and the skin epithelium) and vesico-vaginal fistulae (VVF). VVFs are a subtype of female urogenital fistulae. VVF is an abnormal fistulous tract extending between the bladder and the vagina that allows the continuous involuntary discharge of urine into the vaginal vault. In addition to the medical sequelae from these fistulas, they often have a profound effect on the patient's emotional, mental, social and psychosomatic well being that results in a syndrome that has been dubbed "Fistularia" by one of our VVF specialists.[1,2]

The true incidence of VVF and other urogenital fistulae is unknown.[1,2] This is because most of the cases are not reported due to the social stigmization of the disorder and the occurrence of most of these disorders in remote parts of developing countries such as among the Pokots in northern Kenya, where Mabeya (2003) found an incidence of 1 VVF per 1000 deliveries.[1,2,3] However, Waaldjik et al (1994) and other authorities estimated a minimum worldwide incidence of 1-2 per 1000 deliveries and a prevalence of 1-2 million cases.[1,3] Besides the localized study by Mabeya[3] in West Pokot district in Kenya, the incident of VVF in Kenya has not been established. Margolis cites an incidence of 3-4 cases per 1000 deliveries in West Africa[2], while Gunaranthe at Kenyatta National Hospital found that 87.8% of VVF were obstetrics and the highest incidence occurred in the 20 – 24 age group. The majority of these cases complicated a first or second confinement.[4] Smith and Williams estimate that the prevalence of VVF is 500,000 cases of worldwide.[5]

Causes of urogenital fistulae are varied and the proportion of each cause varies from place to place depending on the quality of obstetric and gynaecologic care. In developing countries, the predominant cause (85-97%) of VVF is prolonged obstructed labour.[1,2] In the USA, in 1984, 85% of VVFs followed surgery, 10% radiotherapy and only 5% obstetric causes, mainly from operative vaginal delivery. About the same time (1983) Tahzib[3] found that 83% of VVF resulted from obstructed labour and only 1% were from surgical injury. In the same year at KNH, Orwenyo,[6] found out that obsteric causes accounted for 92% of the VVFs then. A year before, 1982, Guranathine and Mati[4] had found that 40-80% of VVFs occurred in the primigravida of whom 70% had obstructed labour with cephalo-pelvic disprortion.

VVF occurring as a result of birth trauma especially in Africa occurs in young girls who have conceived at a young age often before full pelvic growth has been achieved. Chronic malnutrition and rickets further limit pelvic dimensions increasing the risks of mal presentation and cephalopelvic disproportion. This is further compounded by poor health seeking behaviour during pregnancy and poor access to medical facilities during childbirth and or lack of skilled attendance at birth.[1,3,6] Obstructed labour in these settings may be protracted for days. While operating the patient presented, Dr. Khisa, a specialist in VVF repair, indicated that he had observed an increase of VVFs that occur secondary to operative delivery particularly after cesarean section following obstructed labour as happened to our patient.

Prolonged obstructed labour causes VVF mostly by marked pressure ischaemic necrosis and less so by uterine and concomitant bladder injury. In obstructed labour, the bladder is displaced upwards and the base and the urethra are compressed between the presenting part and the posterior part of the pubis. This causes ischaemic necrosis, oedema of tissue and sloughing and scarring. When infection supervenes the sloughed off fistula widens even further. The effect of prolonged impaction of the foetal presenting part in the pelvis is one of widespread tissue oedema, hypoxia, necrosis, and sloughing resulting from prolonged pressure on the soft tissues of the vagina, bladder base, and urethra. The sloughing off occurs between the 3rd and 10th days resulting in urinary incontinence.[1,3,7] Typically the resulting fistula is large and involves the bladder, urethra, bladder trigone, and the anterior cervix. Complex neuropathic bladder dysfunction and urethral sphincteric incompetency often result, even if the fistula can be repaired successfully. Other sequelae of VFF include bladder praolapse, stone formation, loss of pelvic floor muscles and/or labiae minora, pressure ulcers over prominent bone areas such as over the sacrum, urine induced dermatitis and cachecia.

Other factors that increase the likelihood of obstetrical VVFs include operative vaginal deliveries using forceps or vacuum extraction, destructive vaginal deliveries, outlet obstruction due to female circumcision, infections such as genital tuberculosis and lymphogranulom venereum, the practice of symphysiotomy, and Gishiri and Angurya 'cuts' incisions (anterior vaginal walls incisions).[3] Other causes of urogenital fistulae including VVF, ureteral-genital fistulae include gynaecologic surgery especially total abdominal hysterectomies, pelvic malignancies like advanced cervical cancer, surgery for pelvic malignancy, vaginal surgery and radiation therapy. In our patient the bladder may have been incised accidentally and, unnoticed, sutured to the anterior uterine wall. Alternatively, a suture may have been passed through the posterior vesical wall while repairing the uterus. This may have been exacerbated by sepsis as commonly occurs in

patients with obstructed labour. That it was a cut is supported by the fact that she developed incontinence soon after the operation.

Classically, patients with VVF present with uncontrolled leakage of urine from the vagina. The onset of leakage is importantin estimating the cause of VVF. Iatrogenic lacerations associated with obstetric fistulas as may have happened with our patient, typically presents in the first 24 hours of delivery. Fistulas attributed to obstructed labour present within the range of 3-30 days but commonly from 7th day postpartum. Radiation induced fistulae present 30 to 3 months later.[1,3,8] The leakage is usually continuous but may be dribbling with small fistulae. The patient is initially examined to confirm presence of urine in the vagina. Once confirmed, the source of the urine (ureter, bladder, and urethra) is confirmed. The differential diagnosis for the discharge of urine into the vagina includes single or multiple vesicovaginal, urethrovaginal or ureterovaginal fistulas and fistula formation between the urinary tract and the cervix, uterus, vagina, vaginal cuff, or (rarely) ureteral fistula to a fallopian tube.[9] In this patient, methylene blue was instilled into the bladder using a urethral catheter. The vagina was then exposed and examined for areas of leakage of the dyed urine. The point was clearly documented in a diagram. If non-dyed urine leaks then the fistula is presumed to involve the ureter. Unexplained amenorrhoea, as noted in our patient, is commonly seen in patients with VVF. Haematuria during menses is suggestive of utero-vesical fistula that would have occurred in our patient had she not been ammerrhoeic.

Urogenital fistulas have been classified variously for purposes of description, rating success of repair and prognostigating the outcome of the repair. Ten years ago the Nigerian VVF specialist, Kees Waaldjik,[1] came up with the following classification that remains widely in use to date including in our set up:

1. Type I : Fistulas not involving the closing mechanisms.
2. Type II : Fistulas involving the closing mechanism:

A : Without total involvement of the urethra

a. Without circumferential defect

b. With a circumferential defect

B : With a total urethral involvement

a. Without a circumferential defect

b. With a circumferential defect

3. Type III : Miscellaneous e.g. utero-vesical as was the case with our patient, utero-vaginal and other exceptional fistulae.

Each of the above fistulae is further classified as per the widest diameter of the fistula as follows:

1. Small : <2 cm diameter
2. Medium : 2-3 cm diameter
3. Large : 4-5 cm diameter
4. Extensive : > 6 cm diameter

Our patient, therefore, had small type III type of urogenital fistula.

Laboratory investigation needed include a urine culture with sensitivity testing, haemogram, renal function tests and radiological examinations including intravenous urography, hysterosalpingography and cystography where ureteral fistulae or other urogenital fistulae are suspected. Unfortunately IVU was not done in our patient.

Treatment of VVF can be conservative or surgical. Conservative treatment is usually carried out immediately the fistula is diagnosed by continuous per-urethral catheter placement and maintanance of free drainage of urine for 4-6 weeks. This way up to 40-60% of fistulas less than 4 cm have been shown to heal.[8] Prolonged passive bladder drainage encourages natural healing and small fistulae can resolve. The rate of success is however usually unpredicatable for individual patients. Drainage is usually continued for 2 – 6 weeks.

Surgical treatment of obstetric VVF is indicated for those, which do not heal, by conservative management. It can be difficult and successful repair depends on correct identification of the fistula, proper pre-operative preparation, correct surgical technique, and good post-operative nursing care. Skill is acquired through experience and practice. Patients with fistulae should, therefore, be operated on by gynaecologists who have the interest and have acquired the necessary skills in fistula repair and work in well-staffed and equipped centers.[10] This patient was referred to Kenyatta Hospital for specialized care but arrived 7 months after she developed the fistula. Controversy surrounds timing of fistula repair. However, most authors agree that fistulae due to pressure and tissue necrosis are best-repaired 8 – 12 weeks after the insult to allow full resolution of inflammation and oedema and revascularisation.[9] Types I and II VVFs are best repaired by the vaginal route while type III commonly requires the abdominal route as happened with our patient.

The vaginal route is also preferred to the abdominal route for repair though this depends on expertise and the size of fistula.[8] In our unit, most VVF's are repaired vaginally under regional anaesthesia in lithotomy position (mostly) and knee-chest position occasionally. The vaginal route has the advantage of minimal blood loss, low postoperative morbidity, shorter operative time and shorter postoperative time.Spinal anaesthesia has the advantage of reduced cost, easy accessibility and the fact that the surgeon can anaesthetize the patient. Nearly all patients are referred so that repair is often many months after the fistula was acquired. In this patient, repair was 7 months after the fistula developed.

It is the objective of any VVF repair to make the patient continent and to preserve and restore sexual and urination function. The size, location and amount of fibrosis are re-evaluated before a definitive surgery is carried out. A circumferential incision is made at the fistula edge with bilateral transverse incision in small fistulas. The anterior vaginal wall is widely dissected sharply and bluntly from the bladder. The bladder is mobilized sufficiently to allow tension-free suturing and closure of the fistula. Closure is made from the lateral margins to the middle. The bladder mucosa is avoided from suture bites and the first layer is inverted with interrupted stitches. Next, the first dye test is made and if watertight then the vaginal, (or uterine in the case of our patient) mucosa is closed with interrupted averting interrupted mattress stitches. A Folley's catheter is left in situ and held above the urethra usually on the anterior abdominal wall to avoid traction of the catheter on the repaired VVF site. This is left for 14 days postoperatively when the patient is re-examined for urine leakage.[11]

Continuous bladder drainage is vital for a successful bladder repair. To achieve this a large caliber urethral catheter is inserted to minimize potential catheter blockage by blood mucus and calcaneous deposits and the patient drinks at least 6 liters of water daily to maintain bladder washout. If the catheter blocks, it must either be flushed out or changed or changed. Any delay will result in tension of sutures and the take of repair may fail. Despite the age, our patient was quite compliant with the water intake. The use of antibiotics is controversial. The VVF specialists at KNH do not recommend antibiotics postoperatively unless there is evidence of potential or real infection. Other surgeons administer oral antibiotics prophylaxis until the catheter is removed. Antibiotics were administered in to our patient because the abdominal route of repair and the surgery is more prone to sepsis. The patient is adised against coitus for 6 months following the operation and removal of the catheter. Further she is strongly advised to undergo an elective cesarean section in the event she becomes pregnant. Our patient had undergone BTL during the cesarean section. Though not required in premenopausal women, oestrogen replacement in postmenopausal women has been shown to enhance fascularisation and mucosal healing.[8]

The most serious complication of VVF repair is breakdown of repair. Haemorrhage, sepsis, ureteral obstruction, urinary incontinence and complications of anaesthesia may also occur. Vaginal repair also carries the risk of vaginal stenosis and dyspareunia or sexual dysfunction. Although she had not resumed sexual activity so dysfunction could not be determined, this was unlikely to occur because the repair route was abdominal. Progression or persistence of urge and stress incontinence may occur.[8] The success of VVF repair depends on the determination and motivation of both the patient and the entire health service providing team in the unit. It (success) has improved tremendously at KNH, thanks to Drs. Khisa, Qureshi, Tekle and Rasen. As happened to our patient the success rate of VVF repair with optimal care is 80-90% at the first attempt. Further attempts are usually less successful as more scar tissue is involved in each repair. At KNH This patient experienced wound sepsis but was treated successfully. Although unpublished data indicates that the success rate at KNH is well over 80%, Orwenyo[6] reported cure rate in the period 1979-1983 as 60%. He found the highest success rate (80.6%) in juxta-cervical fistulae while Guranatine and Mati[4] (1982) found the highest rate (85.7%) in mid vaginal.

Non-repair related complications of VVF include very high perinatal mortality (80%, Guranatine and Mati) and still birth rates (70%, Orwenyo), peroneal nerve injury leading to foot drop, psycho-social disturbances including separation and divorce and 'fistularrhoea' in the wards – a pessimistic belief in the patient (expressed to other patients) that no therapy is amenable by surgery as was the VVF repair in the past.

Prevention of urogenital fistulae in our country requires a strong political will that is supported by tailor-made socio-economic policy frame work that will not only increase and equip maternity health facilities, but will also make them affordable, accessible and readily available countrywide. Further improvement of the general socio-economic status of the citizenry, economic empowerment of the women and education of the girl child is crucial in prevention of VVF. To discourage marriage before the age of maturity, stringent laws should not only enacted, but should also be enforced diligently. Perhaps our patient is an embodiment of what needs to be done in our country to achieve reproductive health. She was uneducated peasant with 10 children most delivered at home, was 40 kilometers away from the nearest health facility where she ultimately went only to get her left ureter tied and a uretero-vesical fistula.

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GYNAECOLOGY CASE NUMBER 14

BARTHOLINS ABSCESS – MARSUPIALIZATION

Name	: M.O.W	DOA	: 29/07/04
Age	: 29 years	DOD	: 30/07/04
Parity	: 2 + 1 Last delivery 2002	LMP	: 2 ½ years ago on DMPA
File No	: 0977930		

History of presenting complaint

MOW presented to the Kenyatta National Hospital with a painful swelling on the right side of her external genitalia for 2 days.

History of presenting complaint

She had been well prior to the onset of the above symptom, which was of rapid and progressive onset. The pain was throbbing and persistent in nature and could not allow her to sleep the previous night. It felt like a boil and walking was now difficult. There was no history of trauma, abnormal vaginal discharge or lower abdominal pain. Her husband had not complained of any unusual urethral discharge.

Obstetric and Gynaecologic history

She was a para 2 + 1 with 2 living children both delivered by spontaneous vertex delivery. Her last delivery was in 2002 after which she used depot medroxyprogesterone acetate (DMPA). Since then she had not had her menses. She underwent an emergency laparotomy and left salpingectomy in 1999 due to a ruptured tubal pregnancy. She received 2 units of whole blood thereafter and recovered well. She attained menarche at the age of 15 years. For two to three years after menarche her menstruation was irregular and heavy but thereafter she had regular menses every 21 days each lasting for 5 days. She had never done a Pap smear test and denied any history of either she or her husband ever contracting any sexually transmitted disease.

Past medical history

She was on treatment for pulmonary tuberculosis (PTB) since February 2004. She had been admitted to Mbagathi District Hospital with chest symptoms and sputum tests indicated that she had the disease. She and her husband had undergone HIV testing after counseling. Both of them were HIV seropositive. She had not suffered from any other opportunistic disease and was not any other medication. Although CD4 count

test had been recommended, she had been able to raise the Ksh. 1500 for the test. She was not on antiretrovirals. The coughing she had had stopped.

Family and social history

She was a married housewife whose husband lived with her and worked as a carpenter. Neither she nor her husband consumed tobacco or alcohol. She was the only wife to her husband whom she suspected had been unfaithful to her. There was no history of other chronic illness in the family. Their children were alive and well and neither of them was HIV seropositive.

PHYSICAL EXAMINATION

General examination

She was sick looking, febrile and mildly pale. She had a temperature of 37.5°C, BP of 110/70 mmHg, PR of 84/minute and RR of 20/minute. She had generalized healed skin lesions and discrete non-tender lymphadenoparhy. No oral thrush or edema was noted.

Respiratory system

She had no signs of respiratory distress. Though a few transmitted breath sound were auscultated, there were no dullness, no reduced air entry or bronchial breathing.

Abdominal examination

The abdomen was scaphoid and moved with respiration. A subumbilical midline scar (laparotomy for ectopic pregnancy) was visible. No tenderness, masses or organomegally were elicited on palpation.

Pelvic examination

There was a cystic and tender swelling on the postero-medial aspect of the right labia majora and minora that measured about 4 cm in diameter. It was warm to touch and quite tender. There was no vaginal discharge. The left labiae were normal. Further vaginal examination was limited by the tenderness.

The cardiovascular and nervous systems were essentially normal.

Diagnosis

A diagnosis of right Bartholin's abscess with secondary amenorrhoea in a patient with positive HIV serostatus was made.

Management

The patient was prepared for an emergency marsupialization under general anesthesia. The dianosis and the mode of management were explained to her and informed consent obtained. She received intamuscular 0.6 mg of atropine and 100 mg of pethidine for both premidication and pain. Stat doses of intravenous 2 MU of crystalline penicillin, 80 mg of gentamicin and 500 mg of metronidazole were administered before she was wheeled to theatre.

Marsupialization

In theatre and under general anaesthesia, she was put in lithotomy position, vulvovaginal toilet was done and then she was draped with sterile towels. Aseptic catheterization was done and pelvic examination confirmed the earlier findings. The uterus, adnaxae and the cul de sac were essentially normal. With a needle and a syringe, 5 mls of pus was aspirated and taken for microscopy, culture and sensitivity. A 3-cm vertical incision was made on the postero-medial (vestibular) aspect of the right labia minora just outside the hymenal ring. This drained 15 mls of pus. Any loculi of pus were broken with exploration with a finger and mounted gauze laced with hydrogen peroxide. The edges of the incision were held with atraumatic toothed tissue (Allis') forceps on either side, everted and stitched to the vestibular mucosa on the inside and the labia minora mucocutaneous skin on the outside using interrupted Viryl number 2/0. The abscess cavity was then cleaned with normal saline and an iodine laced gauze left in situ. The gauze was to be removed after 24 hours. Reversal of the general anaesthesia was smooth.

Postoperative Care

Vital signs were observed ½ hourly until she was fully awake and then 4 hourly. She continued with the above intravenous antibiotics for 24 hours when the gauze was removed and she was discharged on mefenamic acid for 3 days and oral metronidazole and coamoxiclav (Augmentin) for one week. She was advised on daily sitz baths at least three times in a day and whenever she passed stool. She was to be reviewed in the gynecology out patient clinic in a week's time.

Follow up

She was reviewed in the GOPC as scheduled and was noted to be in good general condition. The labial incision was healing well. The microscopy, culture and sensitivity results were unavailable but since the patient was doing well on coamoxiclav she was given another prescription for 3 days and referred to the comprehensive care center for possible CD4 count testing and antiretroviral therapy. Condom use by the husband was emphasized.

DISCUSSION

This is a presentation of a 29 year old lady who was para 2 + 1 and presented with acute Batholins abscess for which marsupulization was done with good outcome.

In 1677 Caspan Bartholin, the Danish anatomist accurately described the location of the paired vestibular glands which were named after him. Batholin's glands are normally two rounded, pea-sized glands deep in the perineum. They are located at the entrance of the vagina at 5 and 7 o'clock. Each gland has got a duct which measures about 2 cm and opens into the vestibule outside the hymen at the junction of the anterior 2/3 and posterior 1/3, in the groove between the hymen and the labium minus.[1,2,3] The main function of the Batholin's gland is to keep the vestibular surface of the vulva moist by its continuous secretion.[1]

A normal Batholin's gland cannot be palpated. Approximately 2% of adult women develop enlargements of one or both glands, of which there are 3 common causes. The most common cause is cystic dilatation of the gland. Symptomatic enlargement of the Batholins gland may be secondary to adenitis or abscess formation. Mechanical obstruction of the duct usually precedes overt infection.[2]

The main causative agent for acute Bartholinitis is gonococcus and to a lesser extend, escherichia coli, staphylococcus, streptococcus, or chlamydia trachomatis or mixed types.[3,4,5]

Acute symptoms resulting from infection are pain, tenderness and dyspareunia. The surrounding tissues become edematous and inflamed and a fluctuant mass is usually palpable.[4]

Bartholin's abscess constitutes 1-7% of acute gynecological admissions at Kenyatta National Hospital.[7] The condition appears most commonly during the reproductive years. Eighty three percent of the patients are between 20 and 50 years of age.[1] The case presented here was 27 years.

Many methods have been used for treatment of Batholin's gland with varying results. The aim of treatment is principally to preserve the gland and prevent recurrence. Simple incision and drainage may provide temporary relief. However, the opening tends to become obstructed and recurrent cystic dilation and infection may result. The recurrence rate after simple incision and drainage is 68-75%.[4] The classic and widely practiced method of treatment of a Batholins abscess is to develop a fistulous tract by marsupulizing the gland. Another mode of treatment is by insertion of a Ward catheter.[1,4,7]

In marspulization, a wedge shaped vertical incision is made on the vaginal wall at the center of the cyst. The cyst is drained and then everted and sutured using delayed absorbable sutures. Sitz baths from the third postoperative day is recommended. The patient M.W. was treated using this procedure. Definitive treatment is excision of the cyst, but cannot be done during infection as it would lead to spread infection and hemorrhage. Recurrence following marsupulazation is 10-15%. [4]

In rare instances, Batholins glands may be a site of adenocarcinoma.

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GYNAECOLOGY CASE NUMBER 15

SEPTIC INCOMPLETE ABORTION AND ANAEMIA – MANUAL VACUUM ASPIRATION (MVA)

Name	: D.K.W.	DOA	: 18/09/2004
Parity	: 0 + 1	DOD	: 23/ 09/2004
Age	: 22 years	LMP	: 22/06/2004
File N^{o}	: 0983818	Ammenorrhoea:	13 + weeks

Presenting complaint

D.K.W presented to the Kenyatta National Hospital (KNH) casualty department with 3 weeks' history of vaginal spotting and a day's history of heavy vaginal bleeding.

History of presenting complaint

She had been well until 3 weeks prior to admission when she developed bloody vaginal spotting that necessitated use of sanitary towels twice a day. The spotting gradually increased so that by the 5th day, she had to use 3 towels per 24 hours. Intermittent mild crampy pains on the 6th day forced her to seek medical advice from the nearby private clinic. She was put on unknown medications that were meant to stop both the pains and the spotting. However, none of the two stopped. Instead they increased. On the 14th day since the onset of the symptoms she sought another clinic's help. She was told that the bleeding was due to previous use of contraceptive pills and was given an injection to stop the bleeding. All along she never thought that she was pregnant and no pregnancy test had been done. The night prior to admission she developed severe intermittent cramps associated with heavy vaginal bleeding with clots. She gradually became week and dizzy and by the time of admission she had fainted twice. She also developed hotness of body and chills. She did not have a bleeding disorder.

Past Obstetric and Gynaecologic History

She was para 0 +1 having had a spontaneous abortion in May 2003 at about 10 weeks gestation. No uterine evacuation was done then although a private clinician saw her. Subsequently, she had vaginal spotting for 2 weeks with no foul smelling discharge. Prior to this she had used the combined oral contraceptive pills for 2 years without any major side effects. Her menarche was at 17 years and the menstrual flow took 3 days each 26 to 30 days. There was no menstrual disorder. She became sexually active soon after completing

secondary education 4 years back (2000) and since then had had 2 sexual partners. She hoped to ge married to the current boyfriend hence she never bothered to use the pills or the condom any more. Sh denied any history of contracting a sexually transmitted disease and believed her boyfriend was equall save and faithful to her. She had never heard of a Pap smear test.

Past Medical History

This was non-contributory.

Family and social history

She lived with her parents in Ngong' town 20 kilometers south of Nairobi. She neither took alcohol no smoked tobacco. She sold second-hand cloths in Ngong' town. She was the 5th and last-born in a family o 5. Her eldest sister had been married at the age of 20 years due to lack of tuition fees. There was no histor of chronic illness in the family.

PHYSICAL EXAMINATION

General examination

She was sick looking, severely pale, dehydrated, febrile and unable to walk or stand without support. He BP was 100/60 mmHg . The pulse was of low volume at 96 beats/minute. The temperature was 38.9 °C and the RR was 20/minute.

Abdominal examination

It was flat and moved less with respiration. There were no surgical marks. There was marked suprapubi tenderness with attendant guarding but no rebound tenderness. The uterus was palpaple, quite tender an the fundal height corresponded to 12 weeks gestation. There was no hepatomegally or any othe organomegally.

Pelvic examination

The otherwise normal external genitalia were covered with old blood that was foul smelling. The vagin felt warm to touch. The cervix was 4 cm dilated and products of conception could be felt protrudin through it. The uterus was about 12weeks gestation and tender. There was bilateral adnexal tenderness. Th POD was boggy though not full as in the typical pelvic abscess. No signs of lacerations that could sugges induced abortion. There was no active bleeding.

Respiratory, cardiovascular, musculoskeletal and the nervous ststems were essentially normal.

Impression

An impression of septic incomplete abortion at 13+ weeks with moderate anaemia was made.

Management

She was admitted, blood taken for grouping and cross match, full haemogram and renal function tests.the results were as follows:

1. Haemogram: Hb; 7.2 g/dl, Wbc count; 14.7 x 10^9/L, neutrophils- 85%, platelets- 143 x 10^9/L
2. U/E/Cs : Potassium – 3.6 mmol/L, Sodium- 137 mmol/L, creatinine- 96 μmol/L, urea 3.5 mmo/L
3. Blood group A Rhesus positive; 2 units available.

A wide bore cannula was fixed and intravenous fluids and crystalin penicillin, metronidazole and gentamicin were administered. She was informed of the clinical findings and the need for uterine evacuation by manual vacuum aspiration (MVA). Once blood was available another intravenous access was obtained and oxytocin infusion started. Informed consent for the MVA was obtained and she was taken to the procedure room after passing urine. She was placed in lithotomy position, vulvo-vaginal toilet done and then draping with sterile cloths done. Examination with Cuscos speculum revealed protruding products of conception through a 4 cm dilated cervix. No lacerations were noted. The anterior lip of the cervix at 12o'clock was held with a double tooth tenaculum. The necrotic and malodorous placental tissue was removed with an ovum forceps and taken for microscopy, culture, sensitivity and histopathology. The cervix was cleaned with a swab soaked in diluted savlon's solution. Manual vacuum aspiration was then done with size 12 Karman's cannula.

The cannula was inserted into the uterine cavity and the vacuum syringe attached to it via the valve. The cannula was gently pushed into the uterine cavity until it reached the fundus then it was slightly withdrawn. The pinch valve on the syringe was released thereby transferring the vacuum through the cannula into the uterus. By moving the entire set back and forth and while rotating the cannula through 360°. The rotation was repeated in anti clockwise manner. The procedure was considered complete when red foam with no tissue was seen in the cannula; when a gritty sensation felt as the cannula was passed over the endometrial surface and when there was resistance while moving the cannula. A total of 110 mls of foul-smelling products of conception were evacuation. To enhance uterine contraction, intravenous 0.5 mg of ergometrine was given. Minimal bimanual massaging was limited by the tenderness but helped to stop mild uterine bleeding. Sucking and plunging the syringe and dismantling the set one by one then cleaned the instruments. The parts were then placed in hypochlorite solution. Blood transfusion was then started and

the patient wheeled back to the ward to continue with intravenous antibiotics and other post-abortal care. This included observation of vital signs half hourly and observation for any vaginal bleeding. Mefenamic acid and haematinics were added to the treatment. Further, intravenous co-amoxiclav (Augmentin) was added and crystalline penicillin discontinued. She stabilized after blood transfusion of 2 units and intravenous fluids. Counseling on contraception and protection from sexually transmitted diseases including HIV/AIDS was provided. She opted for the familiar combined oral contraceptive pills and preferred to undergo counseling for HIV testing together with her boyfriend. She received intravenous antibiotics for three days and on the fourth day was discharged on oral equivalent of the antibiotics with advice to attend the gynaecology out patient clinic (GOPC) in 2 weeks' time or if she developed any complications. She never turned up at the GOPC.

DISCUSSION

The patient presented was a single 22-year-old para 0+1 who had septic incomplete abortion at 13+ weeks gestation with attendant moderate anemia. She underwent manual vacuum aspiration, postabortal counseling and intravenous antibiotic therapy with good outcome.

Abortion is defined as termination of pregnancy by any means before fetal viability has been attained. The gestational age at which viability is tenable depends on the availability and quality of neonatal care, and, therefore, varies from place to place. Though the World Health Organization has defined abortion as termination of pregnancy of a fetus less than 500 grams and /or at 22 weeks gestation, in the USA, the cut off gestation for abortion is 20 weeks.[1,2] In our set up the WHO definition is used. Abortion can be spontaneous or induced. Induced abortion is when there is deliberate interference with pregnancy leading to abortion. This could be legal or illegal depending on the laws of the country in question. In Kenya induced abortion is illegal unless it is done because the life of the mother is in danger.[3] Induced abortion is either safe or unsafe. It is unsafe if it is done either by persons lucking the necessary skills or in an environment lacking the minimum medical standards or both in such a manner that the life of the mother is endangered.[2,4]

Worldwide number of abortions per year is about 50 million, 20 million of which is unsafe and most take place in developing countries where the risk of death is close to 1 in 280 abortions.[5] In Kenya the true incidence of abortion is unknown due to the secrecy involved in illegal abortions all over the country. However, abortion remains one of the commonest gynaecological problems. For instance of all acute gynaecological admissions at the Kenyatta National Hospital (KNH), abortions have over the years consistently accounted for over 40% - (44%, Khehar, 1969) [6], (60%, Aggarwal, 1982),[7] (51%, Fomulu, 1989) [8] and (41.8%, Wanyoro, 2001). [9] These studies indicated that induced incomplete (illegal) abortions accounted for 30-60% of the abortions and most occurred in single young, primigravid girls with unwanted pregnancies. In 2002, abortions were estimated to cause 30% of the 600 maternal deaths per 100,000 per year in Kenya.[4] Our patient probably had illegal, induced, septic incomplete abortion since she denied knowledge of any pregnancy, stayed without seeking any serious medical advice for 3 weeks, was young and single and without any meaningful employment.

Spontaneous abortion is the most common complication of pregnancy occurring in about 15% in clinically evident pregnancies and in about 60% in chemically evident pregnancies. Eighty percent of spontaneous abortions occur prior to 12 weeks.[10] This is based on studies on the failure rates of in vitro fertilization programs. Many factors have been attributed to spontaneous abortion. Chromosomal abnormalities are responsible for 50% of all first trimester abortions. These abnormalities include monosomies, trisomies and

polyploidy which cause abnormal development of the zygote, embryo or fetus.[10] Maternal causes of abortion include infections such as protozoa (malaria,toxoplasmosis), bacteria (listeriosis, brucelosis, tuberculosis, syphyllis, ureaplasma, mycoplasma) and viruses (HIV, cytomegalovirus, herpes simplex typeII). Maternal systemic illnesses such as diabetes mellitus, hyperthyroidism, hypothyroidism, cardiovascular-renal disease and connective tissue disorders. Increasing parity, increasing maternal and paternal and shorter pregnancy intervals are also associated causal factors. [2,4,10] Other causes include immunological factors (systemic lupus erythematosus, anticardiolipin and antiphospholipid antibodies) and alloimmune disorders (blood group incompatilities, antipaternal), corpus luteum insufficiency and uterine defects. Uterine defects may be congenital (abnormal Mullerian formation or fusion, congenital incompetence) or acquired (cervical incompetence, uterine fibroids, uterine synechiae).[2,10]

Environmental factors such as irradiation and intoxication with some gases e.g. anaesthetic gases and passive smoking are causative. Others are ingestion of alcohol and/or tobacco and certain drugs such as cytotoxics. Intrauterine contraceptive devices are associated with 54% abortion rate after contraceptive failure compared with 25% abortion rate if the device is removed promptly when pregnancy is diagnosed. Trauma in form of direct inury to the gravid uterus by penetrating abdominal injuries such as gun short injuries or indirect trauma such as burns, electric shock or surgery (excision of corpus luteum of pregnancy or appendicectomy).[2,10] Although our patient seems to have procured the abortion, other factors such as chromosomal anomalies and HIV testing ought to have been excluded.

The pathogenesis of spontaneous abortion begins with haemorrhage into the deciduas basalis followed by necrosis and inflammation in the region of implantation. The pregnancy becomes partially or entirely detached and is in effect an intrauterine foreign body.[10] Uterine contractions and dilatation of the cervix often results in expulsion of products of conception. The foetus and placenta are likely to be expelled in abortion occurring before the 10 weeks, but separately thereafter. Once spontaneous abortion has set in the symptoms will depend on its progression or the stage/type of abortion. Both spontaneous and safe and unsafe induced abortion (depending on the mode of induction) can progress from threatened abortion to inevitable, incomplete to complete or blighted ovum or missed abortion. Although any of these types of abortions can be infected or septic, incomplete abortion is the commonest to be septic as was the case with our patient. The symptoms and signs of abortion depends on the type or stage of abortion. The common symptom is uterine bleeding during the phase of pregnancy when the fetus is not viable.

In threatened abortion there is usually scanty bleeding but the cervix remains closed. It occurs in 20-25% of all pregnancies.[2,10] Fifty percent of threatened abortions will abort whatever the intervention.[2,10] Inevitable

abortion occurs when there is bleeding with rhythmic lower abdominal pain,low back pain and dilated, effaced cervix and/or rupture of the membranes. Incomplete abortion, defined as partial passage of products of conception, is the leading cause of morbidity and mortality in abortions.[7,8,9] Complete abortion is the passage of the entire conceptus while a missed abortion is the retention of pregnancy despite fetal death. Blighted ovum or unembryonic gestation is a failed development of the embryo so that only a gestational sac with or without a yolk sac, is present. Symptoms of incomplete abortion include lower abdominal pain, backache, vaginal bleeding and expulsion of products of conception. Bleeding is the main symptom and sometimes could be severe to cause hypovolaemic shock. The lower abdominal pain results from uterine contractions.[2,10] Physical examination may reveal pallor and shock depending on the degree of haemorrhage. Fever, jaundice and lower abdominal tenderness mauy be a feature of overwhelming infection. The uterine size may be smaller than the expected gestational age. Vaginal examination may reveal a dilated cervical os.[2,10] Our patient presented with vaginal bleeding, lower abdominal pain, hypovolaemic shock and signs and symptoms of sepsis. Vaginal examination showed foul-smelling, septic products of conception prodruding via the uterine cervix. She had septic incomplete abortion with moderate anaemia.

At KNH, the incidence of septic abortion has over the years ranged from 15-30% and was commonest in the young teenage girl who had illegal and unsafe abortion.[6,7,8,9]

Besides the clinical features, other aids to diagnosis of abortion include ultrasonography.[2,10] Pelvic ultrasound especially by vaginal route is highly accurate in diagnosis of impending spontaneous abortion and prognosis in threatened abortion. If there is no foetal heart activity, the pregnancy is not viable and the clinician can evacuate the uterus. Laboratory findings of the falling or low plasma levels of β-hCG are predictive of complete abortion. A falling blood or urine osestrogen level may signify impending abortion. Pregnanediol and serum progesterone drops precipitously in abortion. In view of the obvious clinical features shown in our patient, the diagnosis was purely based on the signs and symptoms.

The management of abortion depends on the type of abortion and the related complications. In all cases, it is important to assess the blood loss and the general condition of the patient. If significant bleeding has occurred, blood studies will indicate anaemia and if infection is present, the white cell count will be elevated (12,000-2000μl) as well as the sedimentation rate.[2,10] If the patient has lost a lot of blood and is in shock, resuscitation by volume expansion is best done by blood transfusion. Our patient was critically ill with hypovolaemic shock and needed blood transfusion and resuscitation with crystalloids and colloids. Broad-spectrum (co-amoxiclav) antibiotics were instituted to counter the potentially fatal post-abortal sepsis.

The management of threatened abortion is by bed and pelvic rest and sedation is recommended though no sufficient evidence has tested the efficacy of this treatment. Coitus and douches are contraindicated.[2,10] Management of incomplete and inevitable abortion involves prompt evacuation of the uterus to avoid further haemorrhage and infections. Uterine evacuation can be done either by sharp curettage or by suction curettage. In our unit, manual vacuum aspiration is used to achieve this as was done in our patient. This procedure is safe, easy to perform, cheap and does not require prolonged hospital stay. Before the procedure, good counseling of the patient is required. A sepsis rate of 5.4% after manual vacuum aspiration procedure was noted in KNH.[8] For patients with septic abortion, 24 hours of parenteral antibiotics are recommended before evacuation as happened with our patient.[2,4,10] However, before beginning treatment, intrauterine and blood cultures should be obtained. Missed abortion, especially if the pregnancy has been retained for more than 4-5 weeks after the foetal death, requires coagulation profile evaluation to rule out disseminated intravascular coagulopathy (DIC).[2,4,10]

Although this is not routinely being done in our set up, products of conception curetted from the uterus should always be taken for histopathology examination. Regardless of completeness of abortion, the clinician should know the woman's Rhesus factor. Spontaneous abortion is a potentially sensitizing event for Rhesus negative women at risk. At 12 weeks or earlier, 50 μg of anti-D should be given to Rhesus negative mothers with abortion. Abortion after 12 weeks warrants a dose of 300μg.[4,10] Our patient was blood group A+ ve and was, therefore, not given anti-D.

Included among complications of abortion are haemorrhage leading to shock, sepsis and its sequalae and anaemia as noted in our patient, gestational trophoblastic diseases, uterine perforation and injury to the bowel and or bladder.[2,10] The psychological effects include the profound grief and guilt which may follow both induced and spontaneous abortion. This often receives little attention. Patients should always be counseled after evacuation.[2,10,11] Because ovulation may also occur as early as two weeks after abortion, it is important that effective contraceptive advice be given as happened with our patient who opted for the combined oral contraceptive pill, though the use of these could not be confirmed as she did not turn up for follow up.

Prevention of abortion (especially unsafely induced abortion) and its complications includes prevention of unwanted pregnancies through sex education, effective contraception, improving measures for treating women who medically need abortion and by providing treatment promptly.

The author's opinion is that we should stop being hypocritical and act to stop maternal morbidity and mortality from abortion. If abortion is indeed illegal and immoral, then we should see the action of the police (and the society aiding) in the prosecution of those who break the law by procuring abortion; and the religious organizations should have excommunicated millions of their members who are known to their leadership to have procured abortion. On the other hand abortion can be legalized in public health facilities only, where for it to be performed, stringent criteria to be ascertained by a committee consisting of members from the Medical Association of Kenya, the Law Society of Kenya, the Attorney General's Office and that of the faith/religion of the applicant, must be met. Thus abortion will be legal in so far as the criteria is met and illegal if it is done without meeting the criteria as stipulated by law. This will require new legislation on abortion.

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GYNAECOLOGY LONG COMMENTARY

THE PREVALENCE OF ENDOMETRIOSIS AMONG WOMEN UNDERGOINGLAPAROSCOPIC SURGERY AT THE AGA KHAN HOSPITAL, NAIROBI, (AUGUST, 2002 TO AUGUST, 2003)

1.0 ABSTRACT

Background: Operative and diagnostic laparoscopy has led to increased recognition of endometriosis, the prevalence of which remained hitherto unknown in Kenya. Evaluating the prevalence and treatment of the disease would provide evidence-based guidelines for future management of and research on the disease.

Objective: To determine the prevalence of endometriosis in women undergoing laparoscopic surgery

Study Site: The study was carried out at the Aga Khan Hospital, Nairobi, Kenya.

Study design: This was a hospital-based descriptive and cross-sectional study.

Methodology: 150 case records of eligible women were retrieved sequentially and retrospectively. A structured and pre-tested questionnaire was then used to extract data on socio-demographic, reproductive and menstrual characteristics of the women. The preoperative and laparoscopic diagnosis and the type of treatment and its outcome were also recorded. The data was analyzed using Version 11 SPSS and/or Epi Info computer programs.

Results: The prevalence of endometriosis on laparoscopy was 22.7% in the study population, 12.5% in Africans, 27.3% in Asians and 60% in Caucasians. The disease was significantly higher in Caucasians and Asians than in Africans (P=0.039) and in women with nulliparity (P=0.003), secondary dysmenorrhoea (P<0.001), chronic pelvic pain (P<0.001), dyspareunia (P<0.001) and primary infertility (P=0.009). Most (86.7%) patients had mild endometriosis (rAFS) and received hormonal therapy (76.5%) consisting mainly of either progestagens (40%) or goserelin plus "add back therapy" (32%). Symptoms were significantly relieved in 68% of patients and side effects encountered were not unexpected.

Conclusion: The prevalence of endometriosis in the study population in general and in Africans in particular was similar to prevalence found in other studies done worldwide and, therefore, the disease should be highly considered in women with symptoms suggestive of the disease in our set up.

Key Words: Endometriosis, prevalence, laparoscopy, race, Africans, infertility, therapy, Aga Khan Hospital, Nairobi.

2.0 INTRODUCTION

With the advent of both diagnostic and operative laparascopy, there is an apparent increase in incidence of endometriosis among all races including blacks.[1,2] While the true prevalence of endometriosis is unknown all over the world, it is estimated to be 10% among women in reproductive age in the USA.[1,2] There is need, therefore, to know the true prevalence of the disease in Kenya and to clearly understand its diagnosis and treatment. This is so particularly because of the increase in improved global transport and communication that has widened the racial distribution of (gynaecological) patients.

Although Kenya's population is largely of the black race, there is a significant population of Asian origin, among whom the prevalence of endometriosis was found to be higher than among Caucasians in one study.[2] Besides patients treated for chronic pelvic inflammatory diseases (PID) and/or infertility could actually be having endometriosis instead. This may be so given the fact that the prevalence of endometriosis in most of black Africa and particularly in Kenya is unknown and most patients do not undergo diagnostic laparascopy.[3]

This study, therefore, aimed at determining the prevalence of endometriosis among patients who underwent diagnostic laparoscopy at the Aga Khan Hospital, Nairobi. It was a descriptive, retrospective and cross – sectional study that also determined the various modes of treatment of endometriosis and their outcome and the racial distribution among other things.

Using a pre-tested questionnaire data was obtained from 150 case files of patients who underwent diagnostic laparoscopy. The case files were retrieved from the Medical Records Department of the hospital having satisfied the hospital's research and ethics committee requirements.

Univariate and multivariate analysis using a micro-computer SPSS data entry programme were then carried out.

3.0 LITERATURE REVIEW

Endometriosis is a reproductive female disease characterized by abnormal growths of tissue that histologically resemble endometrium, outside the uterus. It is typically characterized by dysmenorrhoea, progressive pelvic pain, dyspareunia and infertility among other symptoms. It is usually found in the pelvis but other rare sites have been described.[3] The endometrium is the inner lining of the uterine cavity, which consists of the mucosa made up simple columnar ciliated epithelium and an underlying stroma.

3.1 History

Presumably endometriosis has been a disease of women since time immemorial. This is because it is found only in other primates other than man such as the baboon, which is more primitive than man.[4] However, Sharoen[5] first described the disease in his "Disputatio Inaugaralis Medica de Ulcerribus Uteri" in 1860. Rokitansky also descrived the disease in detail in 1860, but perhaps the person who studied the disease in greatest detail in the 20th Century was Sampson.[6] He (Sampson) is reputed for his theory of retrograde menstruation as an aetiological process of pelvic endometriosis.[7] The theory remains the main presumed process of development of endometriosis.[4,6,7]

Other landmark events in the management of endomtriosis include the discovery that the oral contraceptive pill could be used to treat endometriosis and Jacobeus[8] of Sweden who introduced a Nitze cystoscope into the peritoneal cavity and coined the term laparoscopy. Both diagnostic and operative laparoscopic surgery has markedly improved the management of endometriosis.The development and use of gonadotropin releasing hormone (GnRH) analogues in the 1980s has also led to improved medical treatment of the disease.

3.2 Epidemiology

The true prevalence of endometriosis is unknown the world over.[1,2] This is because the disease manifests itself in a wide range of symptoms that range from asymptomatic state to crippling severe dysmenorrhoea. Chronic pelvic pain and dyspareunia. Besides the actual diagnosis of the disease requires surgery (either laparoscopy or laparatomy).[9] Its prevalence in women of reproductive age, however, has been estimated to range from 4% to 18%.[1,2,10,11] The average prevalence of the disease is estimated to be 10% in the United States of America (USA).[11]

The prevalence varies with certain factors such as age, race and personal reproductive characteristics including age at menarche type of menstrual cycle and family history of the pathology.With regard to age the disease is more common in women in reproductive ages of 15 to 49 years and hardly seen before menarche.[11] In USA women are usually, 25 to 29 years of age at the time of diagnosis, which is frequently delayed in those who present with infertility rather than pain.[9,11]

Caucasians have about twice the prevalence rate of endometriosis as blacks in the USA.[12] However, a 1995 study by Sange et al[2] indicated that women of Asian origin had a higher risk of developing endometriosis than Caucasians. Chatman's[13] laparoscopic studies on 190 black women, most of whom had been treated for pelvic inflammatory disease (PID), found 21% of these women had endometriosis. In a study by Osefo et al[14] the incidence of endometriosis among the Igbos of Nigeria was 4.3%. This corresponds to findings in the USA that blacks have half the prevalence of endometriosis as the Caucasians.[11,12]

In Kenya, no study has been done on the prevalence of endometriosis. However, D'Hooge et al[4] has done a number of studies on endometriosis in the baboon at the Institute of Primate Research, Nairobi, Kenya.

Women with infertility have higher risks of having endometriosis. About 25% to 35% of infertile women undergoing laparoscopy in the USA have endometriosis compared to 4- 7.4% prevalence rate among asymptomatic women undergoing laparoscopic sterilization.[1,2,9,10,11] This is because endometriosis causes infertility. A Norwegian study[10] on women undergoing laparscopic tubal sterilization found a prevalence rate of 18% compared to US studies that found prevalence rate between 3.7% and 7.4%.[1,9,13]

Dysmenorrhoea and/ chronic pelvic pain have been associated with increased risk of endometriosis. Eskenazi et al[11] found endometriosis in 24% of women with pelvic pain and up to 53% of adolescents with pelvic pain severe enough to warrant surgical intervention had the disease (9). Chatman's study[13] on 190 black women most of whom had chronic pelvic pain found 21% of the women had endometriosis. In Italy, Ajossa et al[15] found that the prevalence of the disease was higher in patients with infertility (30.5%), chronic pelvic pain (45%) and benign ovarian cyst (43%) than in patients with uterine myomas (8.5%).

3.3 PATHOGENESIS

The aetiology of endometriosis is unknown but a number of theories have been postulated. The widely accepted ones are:

- Retrograde menstruation,

- Coelemic metaplasia and transformation into endometriosis,
- Differentiation of mullerian remnants into endometriotic tissue,
- Abnormal immune response to endometrial tissue (a relatively recent theory currently under intense research) and
- Lymphatic spread from the uterine endometrium to ectopic sites.

Endometriosis, however, is probably multifactorial in origin.

3.4 Retrogarade menstruation

The most widely embraced theory of regrograde menstruation was first postulated by Sampson in 1927.[6,7] Several studies have since been done and a considerable number support the theory. In Kenya, D'Hooghe et al,[4] by injecting menstrual endometrium into the pelvic peritoneum of baboons (Papio cyanocephalus and Papio anubis) demonstrated 60% development of histologically proven and 70% laparoscopically diagnosed endometriosis. That the prevalence of endometriosis is higher among women with menstrual outflow obstruction, and, therefore, increased retrograde menstruation, lends credence to the theory.[16]

Women with long duration of uninterrupted menstrual cycles (such as nulliparous women), those with menorrhagia, and / or menstrual cycle less than 27 days and those who use tampons had high risks of dev eloping endometriosis according to some studies.[16,17,18] Further, endometriosis usually occurs after, (not before) menarche. In all the aforementioned menstrual cycle situations the chances of retrograde menstruations is increased. It is thought that the fact that sites and structures closely opposed to the fallopian tube ostia i.e the ovaries, the recto – uterine pouch and the utero-sacral ligaments are the ones frequently found with endometriosis supports the theory of retrograde menstruation.[9]

3.5 Impaired Immunity

That endometriosis is more frequent among women with autoimmune disease,[20] has familial tendency[1] and is more common in whites than blacks,[12] led to the association of impaired immunity and causation of endometriosis. Oosterlynck et al[20] in Belgium found decreased lymphocyte-mediated cytotoxicity to autologous endometrium and to natural killer – cell – sensitive targets in women with endometrioisis. Further, Tabibzadeh et al[21] noted increased peritoneal leucocyte production of interleukin –1 (IL-1), interleukin – 6 (IL – 6) and tumor necrosis factor (TNF) in patients with mild endometriosis. These cytokines are produced by the endometrial lymphoid cells and are thought to act as autocrine or paracrine endometrial growth factors.[22] Peritoneal fluid obtained from women with endometrosis increases endometrial stromal cell proliferation.[21]

Studies indicating that 7 – 9% of endometriosis patients' first-degree female relatives (sisters etc) diagnosed with the disease compared to the control rate of 1 – 2% suggest a genetic influence in t aetiology of the disease.[23] In addition, it has been shown that expression of HLA – B7 inhibit the cytoto activity of natural killer – like T – lymhocytes.[24]

3.5.1 Other Theories

The theory of coelomic epithelium undergoing metaplastic transformation into endometriosis is defend by the following arguments:

1. Endometriosis occurs in adolescent girls in the absence of mullerial anomalies, and it can discovered a few years after menarche before many menstrual cycles are experienced.
2. Endometriosis has been reported in a pre-pubertal girl.[25]
3. Endometriosis in unusual sites such as thumb, thigh or knee can be explained by the fact t mesenchymal limb buds develop adjacent to coelomic epithelium during early embryogonesis.
4. Endometriosis has been encountered in women who never menstruated.
5. Although usually associated with high dose estrogen treatment. Endometriosis does occur in men

Other theories without wide acceptance have been postulated.

3.5.2 Risk Factors Associated with Endometriosis.

Risk factors that have been associated with endometriosis include

- Genetic factors
- Socio-economic characteristics
- Menstrual and reproductive history
- Environmental factors.

As mentioned earlier Semino et al[24] in 1995, reported association of expression of HLA – B7 w endometriosis. This genetic influence is backed by findings that 7 – 9% of first-degree relatives of patie with endometriosis had the disease compared to 1 – 2% of the controls.[23]

The associations of endometriosis with personal reproductive menstrual, biometric and socio-economic characteristics have been controversial. Pan et al[26] in Peking, China, found that higher level of education increased the risk of developing endometriosis. In the same study parity, gravidity, contraception and weight were not associated with significant risk of endometriosis. While cadre of occupation was associated with endometriosis as found by Zhou et al,[19] Motarass et al[18] found no significant relationship between social class, reproductive history, contraception and family history. While Darrow et al[27] found no difference between cases of endometriosis and controls with regard to biometric parameters, McCann et al[28] found endometriosis was inversely proportional to both waist-to-hip and thigh-to-hip ratios. Though still controversial smoking and exercise have been shown to be protective to development of endometriosis.[29] Several other personal characteristics have been studies including a study that found 83% of red haired women with the disease compared to 42% of no-red-haired ones.[30]

3.6 PATHOLOGY

Endometriotic tissue occur as macroscopic and microscopic lesions.

3.6.1 Macroscopic Features

Physical and histologic appearances of endometriosis are markedly varied and depend on the stage and extend of the disease among others. Early stages of endometriotic lesions appear as red, petechial lesions on the peritoneum.Since endometriosis tissue responds to cyclic ovarian steroid hormones, further growth and menstrual detritus accumulates within the lesions giving them dark brown, dark blue or black appearance. Inflammatory process "against foreign endometriotic tissue" may lead to fibroids and development of "powder burn" implants and massive adhesions.

Progressive cyclic accumulation of menstrual like detritus usually around the ovaries may lead to cystic growths called "endometriomas" or chocolate cysts that may be several centimeters in diameter notable on ultrasonography. Non-classical features of endometriosis are clear vesicles, white or yellow spots or nodules, circular folds of peritoneum ("pockets" or "pseudopouch") and visually normal peritoneum whereby lesions are so small they can only be detected microscopically.

Although endometriosis is predominantly found within the pelvis and adjacent structures, it has been described in many parts of the body including the brain where it caused catamenial seizures.[9,25] The commonest site of the disease is the ovary where about 50% of the lesions are found. This is followed by the pouch of Douglas (POD), utero – sacral ligaments, the posterior surface of the uterus, the broad

ligament and the remaining pelvic peritoneum in that order. This site distribution of lesions is in keeping with the retrograde menstruation theory in that the most proximal structure to the tubal ostia has the highest frequency of lesions.[9,11] Implants may occur, over the bowel, bladder and ureters, and may be deep in the tissues especially the cervix, posterior vaginal fornix, the rectovaginal septum and umbilicus. Lesions may also be found in abdominal incisional and episiotomy scars.

3.6.2 Microscopic Features

Microscopy is used in two ways to diagnose endometriosis. First, to identify microscopic implants and, secondly, to confirm endometriosis by histopathological examination of tissues thought to be endometriotic. Small early lesions are composed of tissues that histologically resemble the endometrium; i.e. have stroma and glands. However, with advanced disease, cyst formation and fibrosis develop leading to distortion of the stromal and glandular aspects of the lesion. Typical chocolate cysts have a monolayer cellular epithelium surrounded by fibrotic membranes. Within them are hemosiderin–laden macrophages and altered blood ("chocolate"). When found in peritoneal fluid aspirate the haemosiderim laden macrophages are also suggestive of the pathology.

Immunocytochemistry has demonstrated oestrogen and progesterone receptors in endometriotic stromal and glandular cells.[9,25] Studies on these receptors and the cyclic appearance of the lesions relative to the patients endometrium obtained by pain during sexual intercourse (dyspareunia) especially on deep penetration occurs as a result of compression of implants in the POD, uter-sacral ligaments or rectovaginal septum. It may also be due to adhesions and associated traction during intercourse.

3.6.3 Endometriosis and Infertility

Endometriosis diagnosed by laparoscopy is reported in a higher proportion of infertile women (38.5%) than fertile ones (5.2%).[9] Moreover, fecundity rates in women with endometriosis tend to be lower than the normal fecundity rate.[25] The aetiology of infertility among women with endometriosis is multi-factorial. Moderate and severe endometriosis is associated with pelvic adhesions that distort the pelvic anatomy, prevent the normal tubo-ovarian and fimbrial ovum collection and encase the ovary. Occasionally implants can destroy ovarian and/ or tubal tissue.

Mechanical effect of adhesions does not explain infertility noted in patients with minimal to mild disease. Although studies on peritoneal fluid have yielded inconsistent results, Syrop and Hulme[35] reported

increased peritoneal fluid volume from the usual 5mls to 20mls among affected patients with minimal and mild disease. He and others demonstrated elevated levels of inflammatory cell products such as prostaglandius, IL –1 and IL – 6, TNF, proteolytlic enzymes, compliment factors and other cytokines. These biochemical products have been postulated to reduce fertility by interfering with folliculogenesis, ovulation, tubal motility, nidation and luteal phase adequacy.[20,35] Phogocytosis of sperms by increased peritoneal macrophages has been thought to cause infertility. However, other studies showed no difference in sperm count between women with and those without minimal and mild endometriosis.[35,36]

Certainly dyspareunia due to endometriosis can reduce fertility but is probably not a major factor of infertility in this case.

3.6.4 Extra – pelvic Endometriosis

Endometriosis can occur in virtually every organ of the body.[37] Clinical presentation of extra-pelvic endometriosis, therefore, depends on the site, size and depth of the disease. It has been documented in surgical scars of 0.1% of women who have undergone caesarean section.[38] On the laparotomy scar, it may similate harmatoma, incisional hernia or metastatic tumour. Okunlola[3] reported monthly umbilical bleeding ("menstruation") from an isolated umbilical endometrioma. In Uganda, Banyima[39] noted that thoracic endometriosis had been described severally and reported a case of thoracic endometriosis. It may present with pleural effusion, chest pain, haemoptysis and recurrent catamenial pneumothoraces. The disease has been operated as an inquimal hernia and has been reported to cause catamenial seizures due to brain implants.

3.6.5 Complications

These, too, are varied, site and size dependent. Due to the infiltrative nature of the lesions endometriosis has been reported to penetrate the gut, urinary tract (bladder and ureter) causing haematochezia and haematuria respectively. Massive adhesions have been documented to cause intestinal and uretic obstruction causing silent impaired kidney function. Increased incidence of clear cell ovarian carcinoma has been noted in patients with endometriosis.[40]

Other complications are site dependent while complications due to treatment will be discussed later under treatment.

3.6.6 Physical Examination Findings

These include tender lower abdomen and localized dark blue nodules if external. Pelvic examination reveals tender nodules in the cul-de-sac and along the uterosacral ligaments. The uterus is often in fixed

retroverted position (due to adhesion) while the ovaries may be enlarged (due to endometriomas) or not felt at all owing to distorted pelvic anatomy. Generalized pelvic tenderness with positive cervical excitation test mimics findings on patients with pelvic inflammatory disease.

3.7 DIAGNOSIS AND CLASSIFICATION

3.7.1 Diagnosis

As mentioned earlier, the diagnosis is based on the patient's comprehensive history, physical examination and surgery (laparoscopy or laparotomy). In addition magnetic resonance imaging (MRI), ultrasonography (particularly transvaginal) and other ancillary diagnostic procedures unique to the clinical presentation may be used to diagnose the disease.

3.7.2 The CA – 125 Assay

This, CA – 125, is a cell surface antigen found on the derivatives of the coelomic epithelium that includes the endometrium. Besides being used as a tumour marker of epithelial ovarian (and other) tumours, serum CA – 125 levels can be used to estimate the degree of the disease and the response to treatment.[25] Though earlier thought it could be used for screening endometriosis, its low sensitivity limits this purpose. Moreover, its levels are elevated in other conditions such as acute pelvic inflammatory disease, leiomyomata and menstruation.

3.7.3 Differential Diagnosis

Because of the varied clinical presentation, endometriosis similates virtually all pelvic diseases but more so chronic pelvic inflammatory disease, adenomyosis (endometriosis interna), ovarian tumours (chocolate cysts), degenerated uterine meiomyoma, etcetera.

3.7.4 Classification of Endometriosis

Being a progressive disease with various stages, endometriosis requires classification. It is for purposes of describing the degree of the disease, for comparing the results of different treatments and for international communications regarding the disease, that various classification systems have been formulated. However, the revised American Fertility Society classification system of 1985 is the mainly used and internationally recognized classification system.[41]

As shown in "Appendix IV", a weighted point system, based on the number, size, depth and location of endometrial implants, plagues, endometriomas and/or adhesions. Certain affected anatomical structures such as the ovary and the fallopian fimbriae are assigned the highest points because of the clinical sequelae (especially on fertility) that the involvement of these structures would pose.

To ensure comprehensive evaluation of the endometriosis, inspection of the pelvis in a clockwise or anti-clockwise manner is advised. Alternatively a pre-designed pelvic map can be used but, again, systematically. The presence of endometriosis in sites not included in the classification system such as the skin, or vagina should be documented under "additional endometriosis"' while other pathology such as leiomyomata should be recorded under "other pathology"'

The weighted points are added up and classification done based on the sum of points as follows: -

1. Stage I (minimal endometriosis: 1 – 5)
2. Stag II (mild endometriosis: 6 – 15)
3. Stage III (moderate endometriosis: 16 – 40)
4. Stage IV (Severe endometriosis:> - 40)

3.8 TREATMENT

Since endometriosis similates other diseases, ideally laparoscopy or laparotomy should be done to confirm and stage the disease before commencing any treatment. However, empirical treatment may be given to patients with typical symptoms and signs of endometriosis who cannot afford surgery. Therapeutic options are, nevertheless, dictated by the patients desire for future fertility, symptoms, disease stage and site and to some extend, her age. These options are either medical, surgical or a combination of both medical and surgical therapies.

They include:

1. Observation with or without analgesia,
2. Hormonal therapy,
3. Surgical treatment,
4. Treatment of complications,
5. Specific therapy to extra-pelvic endometriosis and
6. Supportive care (counseling etc)

Although guidelines on therapy exist, patient management should be holistic and individualized.

3.8.1 Observation

Expectant management may be offered to asymptomatic patients, those with mild discomfort and infertile women with minimal or mild endometriosis. Although endometriosis is a progressive disease there is no evidence that treating an asymptomatic patient will prevent or ameliorate the onset of symptoms later. Moreover, despite studies showing reduced fertility and fecundity among patients with minimal and mild disease, cumulative pregnancy rates after five years of 90% have been reported in women not treated for mild or minimal endometriosis.[42]

3.8.2 Analgesic therapy

Studies[35,36] showing increased prostanoids and cytokines in peritoneal fluid of patients with pain due to endometriosis, justify the use of non-steroidal anti-inflammatory drugs and prostaglandin synthetase inhibiting agents as sole therapy for endometriosis with no abnormalities on pelvic examination and no desire for immediate fertility.

3.8.3 Hormonal Therapy

Since endometriotic tissue hypertrophy and bleed following oestrogen and progesterone stimulation, hormonal therapy aims at interrupting this cyclic process that lead to pain, adhesions and infertility. Agents available include oral contraceptive pills, progestagens, danazol, gestrinone, mifepristone and gonadoctropin releasing hormone analogues (GnRHas). All these agents (save mifepristone) are comparable in terms efficacy but have different side effect profiles.[9,25] They are mainly suppressive rather than curative and are, therefore, mainly used to treat pain (dyspareunia, dysmenorrhoea, etcetera) due to endometriosis.

3.8.3.1 Oral Contraceptive Pills (OCPs)

Monophasic contraceptive pills are generally preferred and are given continuously for 6 to 12 months. This leads to amenorrhoea and decidualization of endometrial glands. Consequently, break-through bleeding may occur and this is ameliorated by adding oestrogen to the therapy such as conjugated oestrogen 1.25mg or oestradiol 2mg daily for 1 week. Other side effects are bloating, nausea, headache, irritability, breast tenderness, chloasma and other skin changes, reduced vaginal lubrication in some patients and other side effects attributed to OCPs.

3.8.3.2 Progestational Agents

These also cause decidualization and subsequent atrophy of endometrial tissue. Commonly used progestagens are oral medroxyprogesterone acetate and megestrol acetate. Medroxyprogesterone acetate in an oral dose of 30mg daily has been shown to be as effective as danazol (below) in treating endometriosis.[42] For this reason, and because it is more cost effective and has fewer side effects, medroxyprogesterone acetate is often the first choice for medical treatment of endometriosis. Unlike the oral formula, the usefulness of depot medroxyprogesterone acetate in infertile patients is limited by the varying length of time it takes for ovulation to occur. Megestrol acetate given in a dose of 40mg daily yields similar results.[41] Side effects of progestagens include, depression, weight gain, fluid retention, and break through bleeding which is usually cleared by short term (7days) administration of oestrogen. They are effective in relieving symptoms but not for treating infertility attributed to endometriosis.[43]

3.8.3.3 Danazol

This is a weak androgen that is an isoxazole derivative of the synthetic steroid 17 – ethynyltestosterone whose multiple actions produce a high adnrogen, low oestrogen environment that does not support the growth of endometriosis.[25] In addition the amenorrhoea it causes reduce new seeding from peritoneum into the peritoneal cavity. It is administered at a dose range of 400 to 800 mg daily for 6 months. Its cost and side effect profile have reduced its use despite its efficacy in reducing pain in, up to 90% of patients.[25] The most common side effects are weight gain, acne, deepening of the voice, oily skin, hirsutism, fatigue, atrophic vaginitis, hot flushes, muscle cramp, menstrual disturbances and emotional lability. Further more, a number of patients develop permanent deepening of their voices. Besides the drug is quite expensive.

3.8.3.4 Gestrinone

This is a 19-nortestorerone derivative that decreases the secretion of follicle stimulating hormone (FSH) and leutenizing hormone (LH) thereby similating danazol both in action and side effects profile. Several studies including a Brazilian study by Halbe et al[44] have demonstrated similar efficacy between danazol and gestrinone. Halme found danazol superior to gestrimone with regard to acne and irregular bleeding. Gestrinone has the advantage of being taken only twice a week, being cheap and causes less leg cramps than danazol.[44]

3.8.3.5 Gonadotropin Releasing Hormone Agonists/Analogues (GnRHAs)

These are synthetic peptides that are structurally similar to the octapeptide gonadotropin releasing hormone (GnRH) produced by the arcuate nucleus of the hypothalamus. Unlike GnRH, whose half-life is 2-4

minutes, these analogues have a long half-life, and once administered are not bound on GnRH-receptors in pulses but persistently, thereby causing receptor down-regulation; reduced FSH and LH and ultimately profound hypo-oestrogonic state. This so called "medical oopherectomy" (or "castraction" in the case of a male has been used to treat endometriosis, prostatic and breast carcinomas and to desensitize the pituitary before induction of ovulation by gonadotrophins for in vitro fertilization (IVF).[25] They are also used for shrinking myomas and reducing menorrhagia due to these fibroids and hence building up the haemoglobin level before myomectomy is done. Examples include goserelin (Zoladex®), buserelin, nafarelin, leuprolide, histreline, tryptorelin and deslorelin. The mode of administration varies such that leuprolide acetate is give intramuscularly 3.75mg once a month, nafarelin 200mg intranassly twice daily and goserelin 3.6mg sucutaneously once a month. Their long term use is limited to six moths due to the profound hypo-oestrogenic state that cause pseudo-menopausal side effects one of which is loss of total body bone mineral content (TBBMC) by 6 – 25.4%.[25,45,46] For instance, Fukashima M.,[45] using serial computerized tomography (CT) scans of third lumbar vertebrae of women randomized into buserelin and danazol groups, foud 10-25.4% decrease in BMC in the buserelin group by end of 6 months' treatment. This decrease in BMC was not fully reversed by end of 6 months since the discontinuation of buserelin and found bone mass loss of 3.3% 12 months after stopping leuprolide. Similar findings were found by Dawood et al[46] who used leuprolide. The adverse events due to hypogonadotrophic-hypogonadism that stems from GnRH agonists have been reduced by "add back" therapy whereby a combination of oestrogens and progestins are added to the GnRH agonists.[47,48] Kilholma P et al,[47] using goserelin and a combination of 1mg of norethisterone ecetate and 2 mg of 17-beta estradiol, showed that hormone replacement therapy (HRT) did not reduce the efficacy of goserelin but diminished the post-menopausal symptoms during treatment. An English study whereby endometriotic patients were randomized into those who used goserelin 3.6mg subcutaneously alone and those who were given goserelin 3.6mg with "add back" therapy of 25micrograms of transdermal 17 beta oestradiol daily and 5mg medroxyprogesterone acetate orally daily found no difference in efficacy of treatment of endometriosis but the hypo-oestrogenic effects were markedly reduced, though not abolished in the "add back" therapy group.[48] Other combinations of add back therapy have been studied and are in use.

3.8.4 Surgical Treatment

This is either conservative or definitive ("radical") surgery. Definitive surgery involves hysterectomy, bilateral sulpingo-oopherectomy and excision of all removable endometriotic tissue. Conservative surgery on the other hand preserves the reproductive organs (ovary/ovaries and uterus) and function of the patient. Surgery is generally indicated for moderate and severe disease for treating both pain and infertility due to

endometriosis. However, Marcoux S. et al,[49] using a randomized controlled trial, found that laparoscopic resection or ablation of minimal or mild endometriosis enhances fecundity in infertile women.

Operative laparoscopy is increasingly being preferred to paparatomy because both diagnostic and operative are done in the same sitting and is generally cost-effective in that there is less morbidity less, hospital stay, and is aesthetically acceptable. Besides other laparoscopy assisted procedures can be done including:

1. Chromo-pertubation
2. Hysteroscopic assisted tubal catheterization and
3. Carbon-dioxide laser ablation.

However, Crosignani[50] et al reviewed 63 references and concluded that intestinal vesical. Peri-ureteral, rectoperitoneal and vaginal lesions and large endometriomas associated with extensive adhesions still benefit from classical surgery at laparotomy.

Conservative surgery for infertility leads to pregnancy rates of 75% for mild diseases 50 – 60% for moderate disease and 30 – 40% for severe disease.[9,25] Definitive surgery is indicated for patients without desire for future child bearing and with severe disease or symptoms. Hormone replacement therapy with oestrogen-progestin combination at usual doses can be started immediately post-operatively with an essentially negligible risk of inciting growth of residual endometriosis.[25] A combination of medical and surgical therapy is indicated where conservative surgery is inadequate, symptoms persist and or recurrence occurs. Specific surgery to localized emdometriosis such as excision of surgical scar endometriomas are performed as per the site and specialist surgeons may be involved.

3.8.5. Recurrence of endometriosis

as stated earlier hormonal therapy is suppressive and, therefore, unless definitive surgery is performed endometriosis recur at a rate of 5 – 20% per year depending on the initial stage prior to therapy. The recurrence rates 5 years after women were treated with various GnRH agonists were 37% for minimal disease and 74% for severe disease.[53] The recurrence rates after treatment with GnRH agonists are similar to those after danazol, and both are greater than that obtained with surgical excision.[25]

3.8.6. Assisted reproduction for infertility due to endometriosis

The use of assisted reproductive technology (ART) though rapidly rising, is very expensive and unavailable in most developing countries including Kenya. This involves ovulation induction, ovum retrieval, in vitro

fertilization (IVF), gamete intro-fallopian transfer (GIFT), zygote intra-fallopian transfer (ZIFT), and tubal embryo transfer (TET). These procedures are indicated in infertile women who are older, or those who have failed to conceive after medical and/or surgical treatment. The reported fecundity and pregnancy rates are variable with some studies suggesting similar rates with those of infertile women due to other causes.[25,55] A Cambridge study in the United Kingdom showed higher rates of pregnancy were achieved after IVF that followed long term down regulation in women with extensive endometriosis.[52]

3.8.7. Prognosis and Supportive care

The course, treatment outcome and symptomatology of endometriosis are impossible to predict partly because the disease state does not correlate with its sequelae. Moreover, the relatively high recurrence rate of the disease, the expensive treatment of infertility with unpredictable success rates and the adverse events of hormonal treatment require adequate and appropriate counseling of patients, their spouses and relatives. In the developed countries endometriosis clubs have been established in which patients with the disease share their experiences, their disappointments and boost each other's morale. Group counseling is also offered through such clubs, which have been found to be useful.

4. RATIONALE

The prevalence of endometriosis in Kenya is not known. There has been a perception that endometriosis was uncommon in black women. Chatman[13] reported that 21% of black women who were previously thought to have pelvic inflammatory disease actually had endometriosis on laparoscopy. Osefo et al[14] in Nigeria found 4.3% of the Igbo (black) women with endometriosis.

The study, therefore, was justified by the need to determine and document the prevalence and other factors of endometriosis in our set up. On the other hand, it would be quite appropriate to just know the magnitude in our set up and particularly its impact on fertility.

This study will not only create a new area of research in our country, it will also create awareness of endometriosis and its management. This is so particularly because improved transport and information technology demands that we acquire a holistic knowledge of our respective areas of specialization. For our clients today and in future will hail from a global village.

5.0 AIMS AND OBJECTIVES

5.1. Broad Objective

To determine the prevalence of endometriosis among women undergoing laparoscopic surgery.

5.2 Specific Objectives

1. To determine the prevalence of endometriosis among the study subjects,
2. To determine the socio-demographic characteristics of women diagnosed with endometriosis,
3. To determine the racial distribution of endometriosis among study subjects,
4. To determine the various stages of endometriosis in study subjects,
5. To determine the various modes of treatment of endometriosis at the study site.
6. To determine the complications of endometriosis in women with the disease.

6. METHODOLOGY

6.1. The Study Design

The study was a hospital – based, descriptive and cross- sectional study.

6.2. Study Site and Study Population

The study was carried out at the Aga Khan Hospital located in Kenya's Capital City, Nairobi. The hospital is classified as a tertiary, referral health institution. It is situated about 3 kilometers north – west of Nairobi's central business district. It has a bed capacity of 254 beds spread in wards administratively divided into the departments of Obsterics and Gynaecology, Surgery, Internal Medicine and Paediatrics. Other important units are the Outpatient, Pharmacy, Radiology, Records, Accident & Emergency and the Operative theatre departments among others.

The hospital through the Department of Obstetrics and Gynaecology is recognized by both the Royal College of Obstetrics and Gynaecology and the University of Nairobi as a training center for post–graduate students. It runs outpatient infertility, gynaecology, antenatal, family planning and gynaecologic oncology clinics. Out of an average of 600 non-vaginal gynaecologic surgeries done by the department annually, about 300 of them are performed by laparoscopy. Most of these are diagnostic–cum operative laparoscopies.

The hospital receives referred patients from East and Central Africa. However, the majority of the patients are Nairobi city residents including those from foreign missions, multinational co-operate institutions and

the United Nations bodies based in Nairobi. They are, therefore, of multiple races in origin. However, the majority of the clients and/or patients are blacks and Asians from Nairobi's estimated population of 3 million people. For instance in 2002, a total of 11,986 blacks and Asians were admitted to the hospital. Of these, 9,679 (80.75%) were blacks and 2,307 (19.25%) were Asians.

The patients are divided into general patients managed by a team of appointed doctors of various cadres and private ones under the care of private doctors. On average a total of 15,000 patients (private and general) are admitted annually. In keeping with the hospital policy each patient (private or general) admitted to the hospital must be clerked by at least a senior house officer and copies of investigation results, discharge summaries and other relevant documents are kept in the patients' case files.

The hospital has a total of five busy theatres that operate 24 hours a day. In each theatre, details of the operations, their indications and the patients' bio-data and registration numbers are recorded in a register. Since 1996, two of these theatres have been used to carry out both operative and diagnostic laparoscopy using modern laparoscopic and hysteroscopic equipment. The files are kept in the Records department in a system that enables easy file retrieval.

6.3. Sample Size and Sampling Method

It being a descriptive, retrospective cross-sectional study, the sample size was calculated using the formula:

$$\text{Sample Size } n = \frac{Z^2pq}{d^2}$$

Where

n	=	The desired sample size
Z	=	Value which is the normal standard deviation usually set at 1.96 which corresponds to 95% confidence interval
p	=	Prevalence taken to be the probable prevalence of condition (endometriosis) in the study population. Being a multiracial population the general prevalence of endometriosis of 10% (11) will be used. Hence "P" will be taken to be 0.1

q = 1 – P =0.9

d = The degree of accuracy with which p was determined; set at 0.05.

Thus Sample Size = $\frac{1.96 \times 1.96 \times 0.1 \times 0.9}{0.005 \times 0.005}$

= **139 study subjects**

The sample was increased to 150 study subjects whose files were retrieved starting sequentially from the file of the latest patient to undergo laparoscopic surgery by 31st August 2003. The increment took care of the possibility of non-retrieval of the case files.

6.4.Participant Recruitment

Using the theatre registry books 150 files of women who satisfied the following inclusion/exclusion criteria were retrieved sequently and retrospectively beginning with the last patient to undergo laparoscopic surgery to the 150th one.

6.4.1. Inclusion Criteria

1. Women aged at least 15 years who underwent laparoscopic surgery
2. Study subjects managed by doctors who consent to the study
3. Study subjects who were clerked prior to laparoscopic surgery.

6.4.2.Exclusion Criteria

1. Women aged less than 15 years at the time of laparoscopy
2. Women aged more than 50 years at the time of laparoscopic surgery
3. Women who underwent laparoscopic surgery done by not-gynaecologist (s)
4. Study subjects not clerked prior to laparoscopy
5. Study subjects managed by doctors who do not consent to the study.

7.0. ETHICAL CONSIDERATION

Patients confidentiality was highly upheld by omitting their names and only using their registration numbers. No specific doctor or team of doctors and/or health workers was mentioned by name. Instead doctors were regarded as private and/or departmental doctors managing general patients.

The study commenced after approval by the Kenyatta National Hospital and the Aga Khan Hospital (Nairobi) research and ethics committees (Appendix VII).

Permission was sought from private doctors in the event there was need to get details from files kept in their clinics for their patients who were operated at the hospital. A detailed explanation of the nature of the study was provided to such doctors and consent obtained from them. The consent form appears as Appendix V

8.0. STUDY LIMITATIONS

1. The study was conducted in a private hospital whose hospital fees limit the number of the average Kenyan patient from undergoing diagnostic laparoscopy at the hospital. This might have created a bias on the true prevalence of the disease.
2. Being a retrospective study some might have been omitted as opposed to a prospective study whose data is recorded as they become available.
3. Due to professional etiquette the study did not take into consideration the level of experience of the surgeons particularly with regard to classification of endometriosis and operative laparoscopy – factors that are experience dependent and would influence the quality of both diagnostic and operative laparoscopy for endometriosis.

9.0. DATA MANAGEMENT

Data obtained using the questionnaire (Appendix VI) by the investigator was entered into a microcomputer using the SPSS-11 data entry programme.

Univariate analysis was performed for each of the dependent variables on endometriosis. All dependent variables that were significantly associated with endometriosis were subjected to multi–variate logistic regression analysis. A P – value of <0.05 was considered significant.

RESULTS

Case records of 150 study subjects were retrieved from the Records Department beginning with the file of the last patient to undergo laparoscopic surgery. Their Socio-demographic characteristics are indicated in Table 1. below: -

Table 1: Socio-demographic Data

Characteristic		
Age	N.	%
<20	2	0.3
20 – 25	12	8.0
25 – 30	32	21.3
30 – 35	35	23.3
35 – 40	32	21.3
40 – 45	23	15.3
45 – 50	14	9.3
Mean Age = 37 years		
Marital Status		
Single	23	15.3
Married	126	84.0
Widowed	1	0.7
Occupation		
Student	5	3.3
Housewife	42	28.0
Employed	87	58.0
Self-employed	16	10.7
Race		
African	80	53.3
Asian	55	36.6
Caucasian	15	10.0

The mean age among the study subjects was 37 years with the majority (66%) being aged between 25 and 40 years. While 23(15.3%) of the study subjects were single, most (84%) were married. Eighty-seven of the women (58%) were employed while about 1/3 (28%) were housewives. There were 80 Africans (53.3%) 55 Asians (36.6%) and 15 Caucasians (10%) in the studypopulation.

Table 2: Parity and Number of Abortions

Characteristic	N.	%
Parity		
0	66	44
1	30	20
2	29	19.3
3	20	13.3
4	4	2.7
>5	1	0.7
Number of Abortions		
0	124	82.7
1	12	8.0
2	8	5.3
3	3	2.0
4	1	0.7
>5	2	1.3

The reproductive characteristics of the study population (Table 2) was such that the higher the parity the lower the frequency of patients. Majority (44%) were nulliparous and parity ranged from 0 to 5; the average being 2.13. Most of the patients (82.3%) had had no abortion while 4% had had habitual miscarriages.

Table 3: Menstrual Characteristics of the Study Population:

Characteristics	N	%
Menarche		
ND	120	80%
<13	1	0.7
13 – 15	25	16.7
15 - 17	4	2.7
Duration of flow (days)		
<3	4	2.7
3– 5	80	57.7
5- 7	33	22.0
>7	3	2.0
Not Documented	30	20.0
Cycle length		
<21	1	0.7
21 – 25	6	4.0
25 – 30	101	67.3
30 – 35	5	3.3
>35	6	4.0
Not Documented	31	20.7
Menorrhagia		
Yes	33	22.0
Not Documented	26	17.3

Although menarche of most (80%) study subjects was not documented, majority of the remaining 20% had their menarche within the normal range (Table 3). Two percent had menstrual flow beyond 7 days while

majority (67%) had menstrual flow within 3 – 5 days. Only 33 (26.6%) of 124 patients had menorrhagia, while two thirds had cycle length of 25 – 30 days and 6 (4%) had oligomenorrhoea

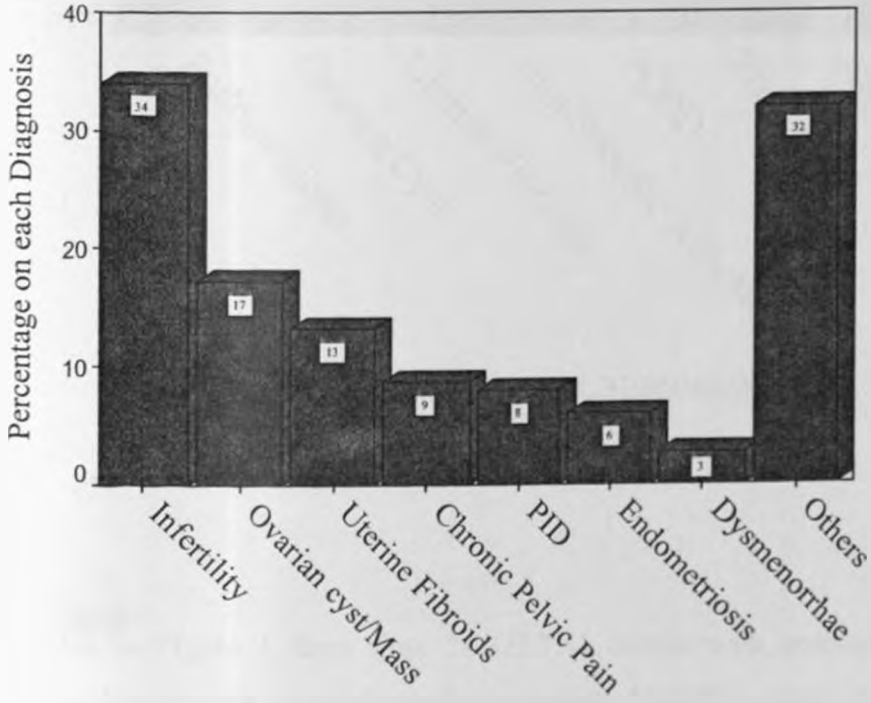
Table 4: Symptoms of Endometriosis:

Symptoms	N	%
Dysmenorrhoea	46	30.7
Chronic Pelvic Pain	25	16.7
Dyspareunia	8	5.3
Pelvic Congestion	10	6.7
Low Back Pain	6	4.0

Table 4 shows the cardinal symptoms of endometriosis as found in this study. Dysmenorrhoea, chronic pelvic pain, dyspareunia, pelvic congestion and low back pain were present in 30.7, 26.7, 6.7 and 4 per cent of the study population respectively.

Diagnosis before laparoscopy was documented for purposes of correlating with diagnosis on laparoscopy.

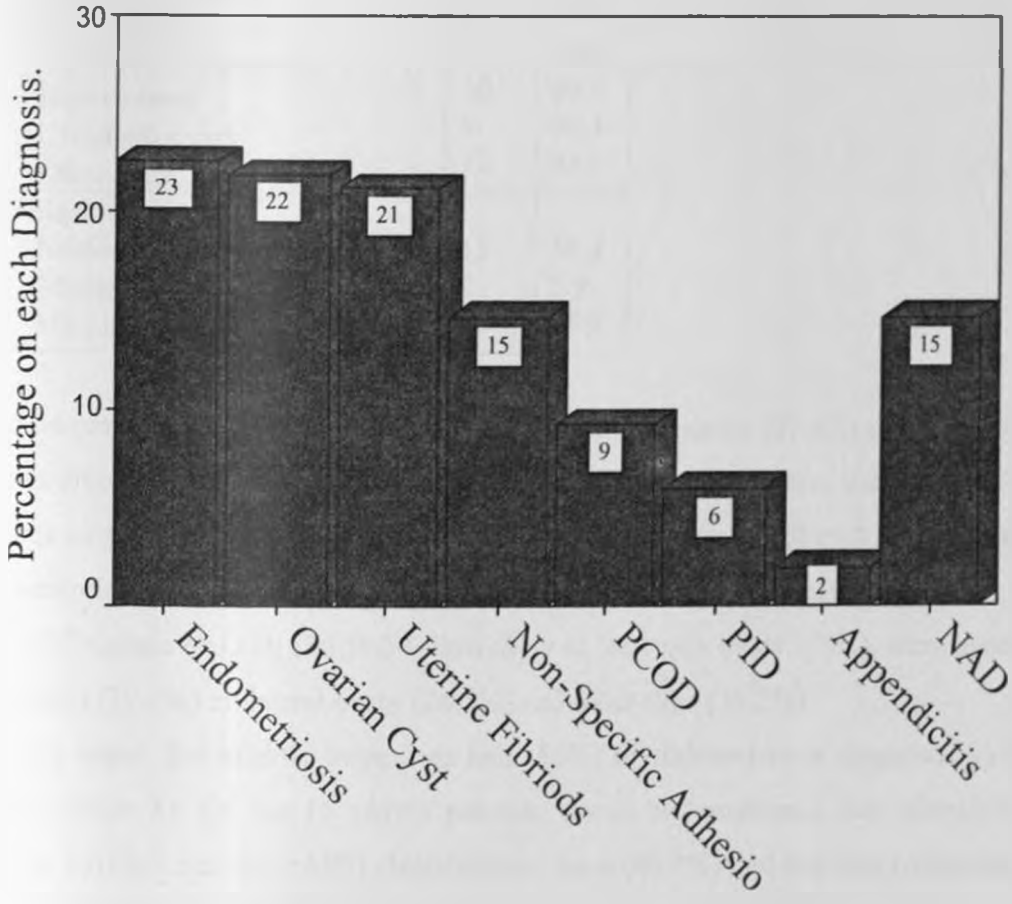
Figure 1:Diagnosis/Impression Prior to Laparoscopy



Diagnosis Before Laparoscopy.

The graph shows one or more indication(s) for which each patient underwent laparoscopy. The leading indication was infertility (34%) followed by ovarian mass/cyst (17.3%) uterine fibroids (13.3%) and chronic pelvic pain (8.7%) in that order. Other conditions (32%) formed a considerable fraction of indications and included desired family size (for BTL).

Figure 2: Diagnosis On Laparoscopy of the Study Population.



As indicated in Figure 2, there were 34 (22.7%) women with endometriosis on laparoscopy. Other diagnoses at laparoscopy included ovarian cyst/mass, 33(22%), uterine fibroids, 32(21.3%), non-specific adhesions 22 (14.7%) and polycystic ovary disease (PCOD); 14 (9.3%). Twenty-one (14%) had no abnormality detected at laparoscopy.

Table 5: Description, Site and Classification of Endometriosis on Laparoscopy.

Description N=34	N	%
Endometriotic lesions	23	67.6
• Pseudo-pouch	16	47.0
• Chocolate cyst/endometrioma	11	32.4
• Dark brown/blue lesion	7	20.6
• Others	5	14.7
Site		
• Pouch of Douglass	26	76.5
• One or both ovaries	19	56.0
• Utero-sacral ligaments	11	32.4
• Both ovaries	10	29.4
• Unilateral ovary	9	26.5
• Other sites	12	35.3
RAFS class		
• Minimal (I, 1-15)	13	38.2
• Moderate (II, 16-40)	2	5.9
• Not classified	19	55.9

Of the 34 patients with endometriosis on laparoscopy, majority (67.6%) of the lesions were described as endometriotic lesions (Table 5). Other signs of endometriosis were described as pseudopouch (47%) chocolate cyst/endometrioma (32.4%), dark blue or brown lesions (20.6%) and various other signs (14.7%). Endometriosis has preponderance to certain sites in the pelvis. The study found the commonest site as the pouch of Dauglass (P.O.D) (76.5%) followed by at least one ovary (56%), utero-sacral ligaments (32.4%) both ovaries (29.4%) unilateral ovary (26.5%) and other sites (35.2%).

The study found that slightly more than half (56%) of endometriosis diagnosed by laparoscopy was not classified(Table 5). Of the 15 (44%) patients whose endometriosis was classified as per the revised American Fertility Society (rAFS) classification, most (86.7%) had minimal endometriosis. The remaining (14.3%) had moderate endometriosis.

Table 6: Surgical Procedures on Laparoscopy

Procedure N=34	N	%
• Adhesiolysis	12	35.3
• Excision/ Cauterization of lesion(s)	11	32.4
• Ovarian cystectomy	3	8.8
• BTL	2	5.9
• Others	A	11.8
• None	11	32.4

On laparoscopy(Table 6), the commonest surgical procedure on endometriotic patients was adhesiolysis 12 (35.3%) followed by excision and/or cauterization of endometriotic lesions 11 (32.4%), ovarian cystectomy 3(8.8%) and others 4 (11.8%). Eleven (32.4%) patients underwent only diagnostic laparoscopy. Two out of 19 women who underwent laparoscopic BTL had endometriosis giving a prevalence rate of 10.5% in asymptomatic women.

Table 7: Cytology, Histology and Race in Patients with Endometriosis

Characteristics	Africans		Asians		Caucasians		Total	
	N	%	N	%	N	%	N	%
Cytology								
• Endometriosis	1	1.3	1	1.8	2	13.3	4	27
• No endometriosis	2	2.5	1	1.8	1	6.7	4	2.7
• Other Pathology			1	1.8	1	6.7	2	1.3
• Not done	77	96.2	52	94.6	11	73.3	140	93.3
Histology								
Endometriosis	3	3.8	3	5.5	1	6.7	7	4.7
Equivocal pathology	1	1.3			2	13.3	3	2.0
Other pathology	31	38.8	20	36.4	4	26.7	55	36.7
No pathology	4	5.0	8	14.5			12	8.0
Not done	41	51.3	24	43.6	8	53.3	73	48.7

Cytology was undertaken on fluids taken from P.O.D of 10 out of 34 women with endometriosis. Four of these had features of endometriosis (Table 7). On the other hand, 7 out of 13 histopathalogical examinations of biopsies of women diagnosed with endometriosis on laparoscopy indicated that they actually had the disease. Three specimens had equivocal pathologies while other pathology was found in another 3 specimens. Thus the prevalence of endometriosis based on laparoscopy and histopathology would be 4.7%. When cytology, histology and laparoscopy were combined, the prevalence of endometriosis came down to 2.7%. However, this would lead to under-estimation since cytology and histology were not done in 70.6% and 61.8% respectively of patients with endometriosis on laparoscopy.

Following the diagnosis 25 (73.5%) of women with endometriosis were put on hormonal therapy. The rest were asymptomatic and were put on no treatment or observation only. Majority 10(40%) of the patients put on hormonal therapy were on progestagens; followed by GnRH analogues plus add back therapy (32%),

GnRH analogues only (12%), danazol (8%) and combined oral contraceptive pills (8%). The GnRH analogue used in all patients was goserelin (Zoladex®). Four patients were lost to follow up and/or the outcome of their treatment was not documented. These findings are illustrated on table 9.

Table 8: Outcome of Hormonal Therapy Used:

Characteristics	**Progestagens N-10**		**Danazol N=2**		**Goserelin N=3**		**Goserelin + Add back therapy N=8**	
Lost to follow up	1		1		0		2	
	N	%	N	%	N	%	N	%
Menstrual Disturbance	4	44.4	1	100	3	100	A	66.7%
Vasomotor symptoms	0	0	0	0	2	66.7	1	16.7
Amenorrhoea	2	22.2	1	100	3	100	6	100%
Breakthrough bleeding	3	33.3	0	0	0	0	0	0
Conceived (after therapy)	1/5	20%	0	0	1	0	2/4	50

The symptoms of most (73.9) patients were significantly relieved while symptoms of 26.1% of patients subsided but persisted. Specifically all the 11 patients on goserelin (only and plus add back therapy) had their symptoms significantly relieved as opposed to 5 out of 9 (55.6) patients on progestagens. In the same vein, 2 out of 4 (50%) women who had primary infertility conceived after goserelin plus add back therapy compared to one out of three (33.3%) who were on progestogens. These patients were too few to determine whether the difference was statistically significant Besides the two patients were on ovulation induction for concurrent polycystic ovarian syndrome. As shown in Table 9 all the complications of treatment were hormonal in nature. There was no surgical complication noted/documented. Four (44.4%) of patients on progestagens had menstrual disturbances that warranted medical attention compared to two thirds of the six patients (66.7%) on goserelin plus add back therapy. Three patients on progestagens developed breakthrough bleeding and two became amenorrhoeic. As expected, amenorrhoea occurred in

all women on goserelin.Two out of three (66.7%) of patients on goserelin only developed vasomotor symptoms compared to one out of six (16.7%) on goserelin plus add back therapy. Of the patients on donazol only one became amenorrhoeic.

Using the SPSS computer program version 11, various presumed predictor variables were cross- tabulated with endometriosis as the dependent variable to see the degree of association. Factors found to be significantly associated with the disease included race (P<0.001), parity (P=0.034), dysmenorrhoea (P<0.001), chronic pelvic pain (P<0.001) and dyspareunia (P<0.001).

Table 9: Factors that were significantly Associated with Endometriosis:

Characteristics	Without endometriosis N=116		With Endometriosis N=34		OR	95% CI	P Value
	N	%	N	%			
Race							
• Caucasians	6	40	9	60			
• Asians	40	72.7	15	27.3	4.00	1.06 – 15.62	0.039
• Africans	70	87.5	10	12.5			
Parity							
• Zero	43	65.2	23	34.8			
• >One	73	86.9	11	13.1	3.55	1.47 – 8.67	0.003
Symptoms							
• Dysmenorrhoea	24	20.9	21	61.8	6.13	2.684– 13.977	<0.001
• Chronic Pelvic Pain	10	8.6	15	44.1	8.368	3.278-21.367	<0.001
• Dyspareunia	1	0.9	8	23.5	35.38	4.16 – 787.68	<0.001
• Primary infertility	13	13.8	11	37.9	3.81	1.33 – 10.97	0.009
Endometriosis	27	19.1	7	77.8	14.778	2.906-75.160	<.0001

As shown in Table 9 Caucasians had the highest (60%) prevalence of endometriosis followed by Asians (27.3%) and lastly Africans (12.5%). They were significantly more likely to have endometriosis than Asians (P=0.039) and Africans P<0.001). Although Asians had more than two fold prevalence of endometriosis than Africans, this difference was not statistically significant (P=0.052) on the other hand, nulliparity was significantly associated with the disease since 23 (34.8%) of nulliparous women had endometriosis compared to 13.1% in women with at least on viable birth; OR 3.55(95% CT 1.47 – 8.67) P=0.003. Age, marital status and occupation were not found to be significantly associated with endometriosis.

There was no significant difference between patients with endometriosis and those without regarding number of abortions and all menstrual features except dysmenorrhoea. The disease was strongly associated with its symptoms which included dysmenorrhoea (61.8%) versus 20.9%); P<0.001, chronic pelvic pain (44.1% versus 8.6%); P<0.001 and dyspareunia (23.5% versus 0.9%) P<0.001.

Generally, infertility was more common with women with endometriosis (47.1%) than those without the pathology (32.2%). However, this difference was not significant (P=0.068). But when primary infertility was taken alone women with endometriosis were found to be significantly less fertile than those without the disease; OR 3.81 (95% CT 1.33 – 10.97) P=0.009. These findings are illustrated in Table 11.

Table 11: Endometriosis and Infertility

Type of infertility	Women without endometriosis N=116		Women with endometriosis N= 34		OR	95% CI	P Value
	N	%	N	%			
Any Infertility	35	30.2	16	47.1	2.057	0.942 – 4.494	0.068
Primary Infertility	13	13.8	11	37.9	3.810	1.333 – 10.973	0.009
Secondary Infertility	22	19.0	5	14.7	1.022	0.291 – 3.38	0.809

DISCUSSION

The main objective of this study was to determine the prevalence of endometriosis among women undergoing laparoscopic surgery at the Aga Khan Hospital, Nairobi. We also sought to determine the socio-economic and reproductive characteristics of the study population. In addition, it was our objective to asses the racial distribution of endometriosis, its impact on subjects' reproductive health and treatment modalities of the disease.

The study population consisted of 150 women of reproductive age who underwent laparoscopic surgery at the Aga Khan Hospital, Nairobi (AKHN) between the first of August, 2002 and 30th August 2003. There were 80 (53.3%) Africans, 55 (36.6%) Asians and 15 (10%) Caucasians. Their mean age was 37 years and ranged from 18 to 50 years. Most women were married (84%) and had had no abortion (82.3%) while majority were employed (58%) and nulliparous (44%). These socio-demographic features are in keeping with those found in Rono's study[54] carried out in the same hospital on laparoscopic gynaecologic surgery. That the majority of women were married (84%) yet were nulliparous(44%) and had the mean age of 37 implies that a considerable proportion of them were sub-fertile. It being a retrospective study, it was not possible to determine the income and level of education of the study population.

Of great significance was the finding by this study that the prevalence of endometriosis in the study population was 22.7%. This is because the expected prevalence of about 10% was twice lower than the prevalence that was found. Further the prevalence of the disease was 12.5% in 80 Africans, 27.3% in 55 Asians and a staggering 60% in 15 Caucasians.

Specimens for histological examination were taken from only 13 out of 34 women diagnosed with endometriosis. Subsequently endometriosis was diagnosed based on both histology and laparoscopy in 7 specimens: three each from Africans and Asians and one from a Caucasian. Assuming specimens from all women had been taken, the histological-based prevalence of endometriosis would be 4.7% in the general population, 3.8% in Africans, 5.5% in Asians and 6.7% in Caucasians. However, since more than half of the women did not have their specimen taken for histological examination, then the aforementioned prevalence rates would be an under-estimation. But if the obvious error were disregarded, the 3.8% prevalence in Africans would be similar to the 4.3% prevalence rate found in the Igbo women of Nigeria by Osefo et al.[14] Perhaps the surgeons did not take specimens for histology because laparoscopy has been deemed by many as the "gold standard" tool for diagnosing endometriosis.[9,11,13,15] Since most of the reported prevalence of the disease was based on laparoscopic diagnosis, and for purposes of discussion, it will be assumed that laparoscopic diagnosis was accurate.

The 22.7% prevalence rate of endometriosis diagnosed by laparoscopy in our study population was relatively higher than the estimated prevalence range of 4 to 18% in women of reproductive age in the general population in the U.S.A.[1,2,10,11] However, it is within the reported prevalence range of 4% in the general population and up to 60% in women with chronic pelvic pain in the same country.[2,11,15] The relatively high prevalence in our study population may be attributed to the fact that more than half (62.3%) of women underwent laparoscopy for conditions that are usually associated with endometriosis (Figure 1). These conditions included infertility (34%), chronic pelvic pain (8.7%), PID (8%), probable endometriosis (6%) and dysmenorrhoea of unknown cause (2.7%).

When cross-tabulated with endometriosis, these (and other) factors were found to be significantly associated with the disease. Endometriosis was significantly more likely to be found in women with primary infertility (P=0.009), chronic pelvic pain (P<0.001), dysmenorrhoea (P<0.001) and dyspareunia (P<0.001). These findings are similar to those found in other studies worldwide.[9,11,13,15] For instance, in Italy, Ajossa et al,[15] found that the prevalence of endometriosis was significantly higher in patients with infertility (30.5%), chronic pelvic pain (45%) and benign ovarian cyst (43%) than in patients with uterine myomas (8.5%). Similarly our study found out that the disease was significantly higher in women with primary infertility (45.8%), chronic pelvic pain (60%) and dysmenorrhoea (17.6%). Unlike Ajossa's findings, however, the prevalence of the disease in women with the above symptoms was relatively higher in our study (infertility: 45.8%V30.5% and chronic pelvic pain: 60%V.45%). These differences may be real or apparent as laparoscopic diagnosis could have led to over-diagnosis of the disease.

Other factors that were significantly associated with endometriosis included nulliparity (P=0.003) and race (P=0.039). Caucasians were more than twice (60%) more likely to have endometriosis than Asians (27.3%) who were in turn more likely to have the disease than blacks (12.5%); P=0.039. This racial distribution of the disease is comparable to those noted by Sanaz et al., Houston et al and Osefo.[9,12,14] However, whereas Houston et al found that Caucasians had twice as much prevalence of endometriosis as blacks, our study indicates that Caucasians had about four fold prevalence of the pathology as the blacks. Besides, unlike the 1995 study by Sangi et al,[2] which showed that Asians had higher prevalence of the disease as the Caucasians, this was the opposite in this study.

As stated earlier the mean parity in patients with endometriosis (1.56) was significantly lower than that of women without the disorder (P=0.002; standard error of the difference = 0.233 and 95% CI – 1.194 to – 0.275). This may be due to the fact that a significant proportion (47.1%) of the 34 women with the disease

(compared to 30.2% in those without the disease) had infertility of some sort and, therefore, of low parity. This revelation and the fact that women with primary infertility were significantly more likely to have endometriosis (P=0.009) suggests that the disease had a causative role of infertility in the individuals involved as has been noted in many studies.[9,11,12,15] It denotes the impact of the disease on the study population's fertility and justifies laparoscopy in subjects with subfertility of unknown cause.

Two out of nineteen asymptomatic women who underwent bilateral tubal ligation (BTL) had endometriosis on laparoscopy. This gives a prevalence of 10.5% in asymptomatic women, which is higher than the prevalence rates of 5.7 to 7.4% found by studies in the U.S.A.[1,9,13] But it is lower than the 18% prevalence rate found in Norwegian women undergoing BTL.[10]

Although endometriosis has been found by some studies[16,17,18] to be higher in women with menorrhagia, those with menstrual cycle les than 21 days and/or early menarche, this was not the case in this study. This implies that the theory of retrograde menstruation as postulated by Sampson[6,7] did not apply to our study population and since 90% of women have retrograde menstruation, this theory alone may have to be relegated to gynaecological archives.

Cognizant that clinical diagnosis of endometriosis could be as good as 78-87% of laparoscopic diagnosis as found in one study[55] we correlated the diagnosis of the disease before and during laparoscopy. Seven out of nine (77.8%) of women who had prior clinical diagnosis of endometriosis actually had the disease on laparoscopy. Thus the positive predictive value of clinical diagnosis was not only good in this study, but was also quite comparable to that found by Ling.[55] This finding suggests that clinical judgments when used with experience and high index of suspicion could be adequate for one to start empirical treatment with cheaper first line drugs (combined pill or progestagens). It is possible though that a surgeon who had clinically made a diagnosis of endometriosis would more likely "see" the same on scoping the patient.

It is known that the colour, depth, site and extend of endometriotic implants are useful in deciding the mode of therapy,[9,25] Our study found modest use of these parameters in that the implants were in most cases (67.6%) described just as endometriotic lesions. In addition only 44% of endometriosis diagnosed on laparoscopy was staged as per the revised American Fertility Society (rAFS) classification system. Of the 15 women whose disease was classified most (86.7%) had minimal disease. Only two (13.3%) women had moderate disease. There was no severe disease.

Though the rAFS classification should not be the only parameter for deciding mode of therapy, it is the most internationally recognized system that is used to stage the disease and compare mode of treatment and its outcome. Further, although the rAFS classification does not always correlate with the symptomatology and impact of the disease on fertility and fecundity, as has been criticized by some,[56] the classification plays a significant role in research. Attempts should, therefore, be made to, not only stage the disease, but also to describe its lesions with regard to their colour, site, depth and extend.

As stated earlier, treatment mainly entailed conservative surgery and hormonal therapy (73.5%) Adhesiolysis (35.3%) and excision and/or cauterization of endometriotic implants (32.4%) formed the bulk of the conservative surgery. None of the patients underwent definitive surgery or the more advanced laser ablation surgery widely practiced in some centers in the West. The only GnRHa used in the study site; goserelin (Zoladex®), was given to 44% of 25 women put on hormonal therapy while ten (40%) received progestagens. Attempts to establish the basis for deciding which of the two drugs to prescribe were futile in that there was no correlation between the symptoms (dysmenorrhoea, chronic pelvic pain, dyspareunia) and the classification on the choice of drug. One could only infer that since apparently most patients had minimal disease and since the two drugs are similar in terms of efficacy but different in side effect profile, the drug choice was individualized, on the basis of desire for fertility, affordability and contraindications of either drug. Goserelin seemed to be more effective than progestagens in that symptoms were significantly, relieved in all the 11 women put on this drug compared to five out of nine (56.6%) in patients on progestagens. In addition two out of four (50%) women who had primary infertility conceived after goserelin plus "add back therapy" compared to one out of three (33.3%) patients who were on progestagens. This difference is apparent because, first, the patients involved were too few to determine whether the difference was statistically significant. Secondly, the study was not a randomized, controlled and prospective study that would ideally be used to determine any significant difference in efficacy between any two drugs. Thirdly, the period of use of the two drugs varied considerably between patients and/or the drugs.

It is worth mentioning that the side effect profile of the drugs were as expected with the majority being disturbances of menstruation. Three (33.3%) patients on progestogens developed breakthrough bleeding while all patients on goserelin became amenorrhoeic. Goserelin with add back therapy as expected had better side effect profile in that only one out of six (16.7%) of the patients on this regimen had vasomotor symptoms compared to two out of three (66.7%) on goserelin alone. These findings on "add back therapy" are similar to results of other studies[47,48] and underscore the importance of the use of "add back therapy" (with estrogen or progestagens) instead of GnRH analogues alone.

CONCLUSIONS

1. The prevalence of endometriosis in the study population was similar to the prevalence found by other studies done worldwide in women with similar characteristics.
2. Majority of the women studied were married and aged above 30 years.
3. Africans had a higher prevalence of endometriosis than earlier stated; but as found in other studies, Caucasians and Asians had higher prevalence rates than Africans.[15]
4. Most women with endometriosis had rAFS stage I (mild form) of the disease.
5. The treatment of endometriosis, though diverse, was mainly hormonal consisting of progestagens and/or goserelin plus "add back therapy".
6. A significant proportion of women with sub-fertility in our society could be having endometriosis.
7. Endometriosis is a significant gynaecological disorder that has hitherto been underestimated in our region.

RECOMMENDATIONS

1. A multi-centre prospective study be undertaken to determine the prevalence of endometriosis in Kenya.
2. Endometriosis be considered and investigated more thoroughly than previously in women with symptoms suggestive of the disease.
3. Guidelines to be established on the diagnosis, classification and treatment of endometriosis in the study site particularly for purposes of future research.
4. Histology and cytology be carried out in all patients diagnosed with endometriosis on laparoscopy/laparotomy.

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APPENDIX I

THE CONSENT FORM

2.1. Information on the study

You are being asked to take part in a medical study entitled, "To Determine the Acceptability of HIV Testing in Early Labor". This study aims at finding out whether rapid testing of HIV among pregnant women who have not been tested can routinely be offered the option of being tested in early labour so as to reduce the MTCT of HIV using a medicine called nevirapine.

Your participation in the study will be purely voluntary and should you decline to participate you will still be entitled to the usual optimum health care provided in this health facility. If you accept to participate in the study, you will be asked a number of questions about yourself and with your consent, counseled and tested for HIV. The test is rapid (results ready in 15 minutes), accurate and involves pricking a finger to get a drop of blood, which is placed, on some slides. Appearance of 2 lines on the slide indicates a positive test and a negative test is denoted by a single line.

A positive test means you are infected with HIV and that you have about 20 to 40% chance of passing the virus to your unborn baby if no intervention is done. It does not mean you are ill or have AIDS. It just means you have the virus and would live for as long as 15 years or as short a time as 1 year before you become ill. A negative test means that upto 3 months ago you had not been infected with HIV and, therefore, you will need another test in three months to confirm this particular test.

Your answers about yourself and the result of the test will be treated with utmost confidentiality and will be used only for purposes of achieving the objectives of the study. If your test turns out to be positive, you will be given the option of orally taking a tablet of a medicine called nevirapine in order to reduce HIV transmission to your baby by about 47%. Research on human use of this medicine has so far not showed any major side effects in both mothers (who have taken it) and their infants.

More counseling will be given to you later as per the outcome of the test. You have the right to ask any questions regarding this study and to decline to take part in the study even in the middle of participation.

2.2 Authority/Consent

Having read the explanation, been explained about the type, objectives and method of the study, and having had the opportunity to ask questions regarding the study (all of which have been answered to my satisfaction), I freely and voluntarily give consent to: -

a) Participate in filling the questionnaire and being counseled for HIV/AIDS testing only.

Signed by participant_____ Date _____

Left thumb print _____ Date _____

b) Participate in, filling the questionnaire and being tested for HIV only.

Signed by participant_____ Date _____

Left thumb print _____ Date _____

c) Participate in filling the questionnaire, being tested for HIV and to be administered nevirapine.

Signed by participant_____ Date _____

Left thumb print _____ Date _____

I _____, the undersigned, have fully explained to the above participant the relevant details of the study and have witnessed her give consent as signed above.

Signed _____ Date _____

APPENDIX II- THE OBSTETRIC LONG COMMENTARY QUESTIONNAIRE

Code No. _____

1. Age in years
   1. 15-19
   2. 20-24
   3. 25-29
   4. 30-34
   5. 35-39
   6. 40-44
   7. 45-49

2. Parity
   1. 0
   2. 1
   3. 2
   4. 3
   5. 4
   6. >5

3. Marital status
   1. Single
   2. Married
   3. Separated
   4. Divorced
   5. Widowed

4. Highest level of education
   1. None
   2. Incomplete primary
   3. Complete primary
   4. Incomplete secondary
   5. Complete secondary
   6. College/University

5. Occupation
   1. None/housewife
   2. Self employed
   3. Salaried/formal
   4. Student

6. Average monthly income in Kshs. 12 months.
   1. ≤ 1000
   2. 1000-2000
   3. 2000-4000
   4. 4000-6000
   5. > 6000
   6. None/N/A

7. Residence
   _____
   _____
   _____

8. Did you attend antenatal clinic during this pregnancy?
   1. Yes
   2. No

9. If "Yes" how many times?
   1. Once
   2. Twice
   3. Thrice
   4. Four times
   5. More than 4 times

10. If "No" reason?
    1. Lack of money
    2. Unware of need/did not need
    3. Discouraging circumstances in health facility
    4. Other (specify)

11. Ever been tested for HIV?
    1. Yes
    2. No
    3. Not sure/don't know

18. How is HIV/AIDS transmitted?
    1. Sexual intercourse
    2. MTCT
    3. Not using condom/unprotected sex
    4. Transfusion with contaminated blood
    5. Sharing razors/sharps
    6. Others (specify)

7. Don't know

12. If yes, when?
    1. During current pregnancy
    2. Not more than three months before current pregnancy
    3. At least once a year ago
    4. Not applicable

13. What was the result?
    1. Positive
    2. Negative
    3. cant remember/not told
    4. Not applicable

14. What is the occupation of your spouse?
    1. Unemployed
    2. Self-employed
    3. Salaried/formal
    4. Student
    5. Casual worker
    6. Not applicable

15. What is his average monthly income Kshs.?
    1. ≤ 1000
    2. 1000 - 2000
    3. 2000 - 4000
    4. 4000 - 6000
    5. ≥ 6000
    6. Don't know
    7. Not applicable

16. Does he have another wife/wives/sexual partner(s)
    1. Yes
    2. No
    3. No Dont know/unsure
    4. Not applicable

17. If "Yes" how many?
    1. 1
    2. 2
    3. ≥3
    4. Not applicable

19. When can a mother with HIV transmit the virus to the baby?
    1. During pregnancy
    2. During labour and delivery
    3. During breastfeeding
    4. Others (specify)
    5. Dont know

GIVE ANSWERS AFTER PARTICIPATION'S REASON (S)

20. Can M.T.C.T. be prevented?
    1. Yes
    2. No
    3. Unsure/Don't know

21. How can M.T.C.T. be prevented?
    1. Not getting pregnant if one is HIV sero-positive
    2. Taking A.R.V./HAART
    3. Proper delivery procedures
    4. Treatment of STDs
    5. Good nutrition
    6. Not breastfeeding
    7. Others (specify)
    8. Don't know

22. Ever hear of voluntary counseling and testing for HIV?
    1. Yes
    2. No
    3. Not sure/Don't know

23. Would you have liked to be tested for HIV through V.C.T. in the antenatal period?
    1. Yes
    2. No
    3. Not sure/Don't know

24. If not tested for HIV antenatally, what reasons?
    1. Afraid of knowing am infected with HIV
    2. Didn't know where to go for VCT
    3. Lacked money for the test
    4. Not offered V.C.T.
    5. Wont be unnecessary
    6. Needed time to decide
    7. Others (Specify).

PROVIDE PRE-TEST COUNSELLING

25. Having been counselled for HIV testing, are you willing to be tested?
    1. Yes (Go to No. 27/28)
    2. No
    3. Yes but later
    4. Unsure

26. If "No" what reasons made you decline?
    1. Afraid of knowing am infected with HIV
    2. Am in labour
    3. Have to inform my spouse/partner first
    4. Was not prepared for the test
    5. Need more time to decide
    6. It is unnecessary
    7. Others (Specify)

CONDUCT TESTING & POST-COUNSELLING

27. (For sero-positive mothers only)
    Now that you know you have been infected with HIV, are you willing to take Nevirapine for M.T.C.T.?
    1. Yes
    2. No
    3. Unsure
    4. Not applicable

28. If "No" what reasons?
    1. Unsure of safety
    2. Dont think it is useful
    3. It is too late
    4. Others (Specify)
    5. Not applicable

29. (For both sero-positive and sero-negative mothers) Are you willing to disclose your status to your spouse/partner and also refer him to HIV testing?
    1. Yes
    2. No
    3. Yes, but better you tell him yourself
    4. Unsure.

30. What are your feelings/views about testing women in early labour for HIV (if they had not been tested antenatally) in order to reduce MTCT?
    1. Highly acceptable
    2. Fairly acceptable/alright
    3. Just acceptable
    4. Would prefer antenatal testing
    5. Not good/not acceptable
    6. Not sure
    7. Others (specify).

KISWAHILI VERSION OF QUESTIONAIRE

1	Una Umri gani (Miaka)? a) 15 - 19 b) 20 - 24 c) 25 - 29 d) 30 - 34 e) 35 - 39 f) 40 - 44 g) 45 - 49	8	Ulitembelea kliniki ya waja wazito tangu ushike mimba? a) Ndio b) La
2	Umezaa watoto wangapi? a) 0 b) 1 c) 2 d) 3 e) 4 f) >5	9	Kama "ndio", mara ngapi? a) Moja b) Mbili c) Tatu d) Nne e) Zaidi ya nne
3	Je umeolewa? a) Sijaolewa b) Nimeolowa c) Tumewachana d) Tumevunja ndoa e) Ni mjane	10	Kama "La", sababu zako a) Ukosefu wa pesa b) Sikuhitaji/sikujali uzuri wake c) Visuhizi zahanatini d) Nyingine
4	Kiwango cha juu cha mosomo a) Sijasoma b) Schemu ya shule ya msingi c) Shule ya msingi d) Schemu ya shule ya upili e) Tamatisha shule ya upili f) Chuo cha elimu/chuo kikuu	11	Umewahi kuchunguzwa kama una viruzi vya ukimwi? a) Ndio b) La c) Sina hakika/sijui
5	Kazi yako a) Hamna/ya nyumbani b) Nimejihajiri d) Nimeajiriwa e) Mwanafunzi	12	Kama "ndio", lini a) Katika hii mimba b) Si zahidi ya miezi tatu kabla ya hii mimba c) Si chini ya mwaka mmoja
6	Mapato yako kwa mwezi kwa miezi 12 iliyopita (Ksh) a) <1000 b) 1000 - 2000 c) 2000 - 4000 d) 4000 – 6000 e) >6000	13	Majibu yalikuwaje a) Nilipatikana na virusi vya HIV b) Sikupatikana na virusi vya ukimwi c) Sikumbuki/sikuambiwa
7	Mahali unakaa _____ _____ _____ _____	14	Mume wako anafanya kazi gani? a) Hajeajiriwa b) amejiajiri c) Ameajiriwa d) Mwanafunzi e) Kibarua

15 Mapato yake kwa mwezi katika miezi 12 iliyopita ni ngapi (Kshs)
a) <1000
b) 1000 – 2000
c) 2000 – 4000
d) >6000

16 Ako na mke (wake) mwingine?
a) Ndio
b) La
c) Sijui/sina hakika

17 Kama ndio wangapi?
a) 1
b) 2
c) >3

18 Virusi vya ukimwi vinasambazwaje?
a) Kufanya mapenzi
b) Kutoka kwa mama hadi mtoto
c) Kutotumia mpira ya kondomu/mapenzi ya kiholela
d) Kuongezewa damu iliyo na virusi vya ukimwi
e) Kutumia vyombo makali pamoja
f) nyingine

19 Wakati gani ambapo mama mzazi anaweza kuambukiza mtoto virusi vya ukimwi?
a) Akiwa mja mzito
b) Anapozaa
c) Anaponyonyesha mtoto
d) Nyingine (fafanua)

20 **PEANA MAJIBU BAADA YA MHUSIKA KUJIBU**
Je, maambukizo kutoka kwa mama hadi mtoto, yanaweza kuzuiliwa?
a) Ndio
b) La
c) Sina hakika/sijui

21 Je, maambukizo hayo yanaweza kuzuiliwa vipi?
a) Kutopata mimba iwapo mzazi ana virusi vya HIV.
b) Kutumia dawa za kupunguza virusi vya
c) Uganga unaofaa
d) Kutibu magonjwa ya zinaa
e) Kutumia vyakula vinavyofaa
f) Sijui
g) Nyingine (fafanua)

22 Umewahi kusikia kuhusu huduma za ushauri na uchunguzi (wa virusi) kwa hiari; yaani VCT?
a) Ndio
b) La
c) Sina hakika/sijui

23 Ulipokuwa na mimba hii ungalipenda kuchunguzwa virusi vya ukimwi?
a) Ndio
b) La
c) Sijui/sina hakika

24 Kama "La" sababu zako ni nini?
a) Naogopa kujua nina virusi vya ukimwi
b) Sikujua mahali pa kwenda kuchunguzwa
c) Ukosefu wa pesa
d) Sikushauriwa
e) Kupimwa hakungefaidi chochote
f) Nilihitaji nafasi ya kuamua
g) Nyingine (fafanua)

25 **PEANA MASHAURI KABLA YA UCHUNGIZI**
Je, uko tayari kupimwa ili ujue hali yako ya ukimwi?
a) Ndio
b) La
c) Ndio lakini baadaye
d) Sina uhakika

26 Kama sivyo ni sababu gani zimekufanya usiwe tayari?
a) naogopa kujua nina virusi vya ukimwi
b) nina uchungu (mwingi)
c) inabidi nimweleze mpenzi wangu kwanza
d) nahitaji wakati zaidi wa kuamua
e) kuchunguzwa hakuhitajiki
f) nyingine (fafanua)

27 **TEKELEZA UCHUNGUZI NA USHAURI BAADA YA UCHUNGUZI**
(Kwa walio na virusi vya ukimwi pekee):
Kama vile uchunguzi unaonyesha una virus vya ukimwi, uko tayari kutumia au kumeza

	HIV mwilini a) Ndio b) La c) Sina hakika/sijui		tembe ya "nevirapine"
28	Kama "La", sababu zako ni? a) Sina hakika kuhusu dhara au usalama wake b) Sidhani inafaidi chochote c) Wakati wake umepita d) Nyingine (dhibitisha)	30	Je, maoni yako kuhusu kuchunguzwa kwa waja wazito walio na uchungu wa kuzaa (Labour pains) na ambapo hawajawahi kuchunguzwa ni nini? a) Napendelea sana b) Ni sawa c) Ni sawa tu d) Napenda kuchunguzwa mbele ya kupata uchungu wa kuzaa e) Sina hakika f) Mengine (thibitisha)
29	**(Kwa walio na wasio na virusi vya ukimwi)** uko tayari kumweleza mume au mpenzi wako hali yako ya ukimwi na pia umshauri aende kupimwa kwa hiari baada ya kushauriwa? a) Ndio b) La c) Ndio lakini afadhali umweleze wewe mwenyewe d) Sina hakika		

Steps to Perform the Finger-Stick Procedure

1. Wash hands with soap and water.

2. Put on latex gloves.

3 Remove test covers and open pouches of test.

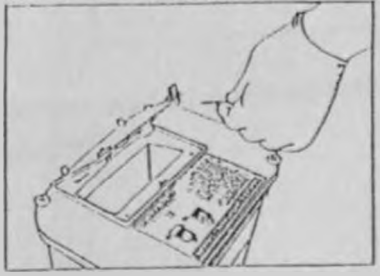
4. Label test in front of client with his or her unique identification number.

5. Holding the palm up, choose the least callused fingertip of client's middle three fingers.

6. Clean client's fleshy area of fingertip with alcohol and cotton pad.

7 Tell client that you are going to prick his or her finger, and it may be uncomfortable Hold finger lower than elbow Prick clean finger with lancet (finger-prick device) Use a swift motion when pricking client's finger A slower motion is very uncomfortable for the client.

8. Place lancet in puncture-resistant container. Never reuse the lancet!

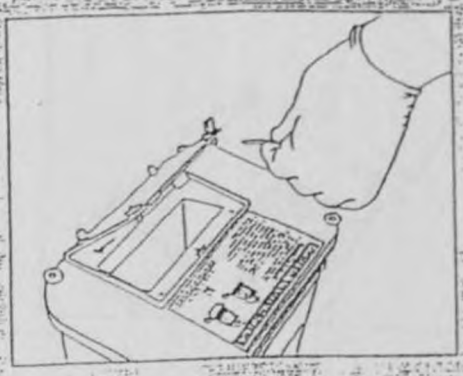


Steps for Conducting the Uni-Gold HIV Rapid Test

1. Take clean micro-filter tube and place gently on finger. Keep your thumb on the tube and gently tap so tube can fill up with blood.

2. When filled, keep thumb on tip of tube. Move thumb away place 50 µl or 1 healthy drop of blood in the circle area of test.

3. Put tube in puncture-resistant container. Do not reuse tube.



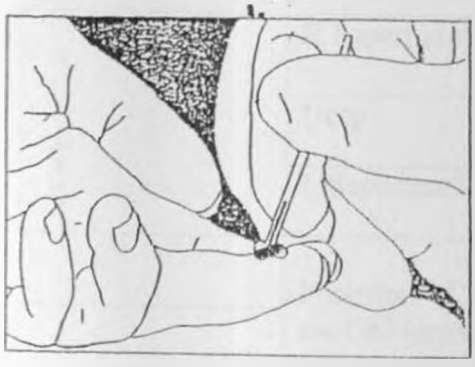
4. Add two drops of buffer solution* in the circle area.

5. This test develops in 15 minutes.

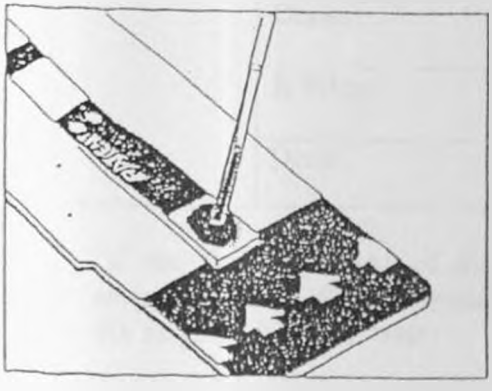
Note: Use only the buffer solution made for the specific HIV Rapid Test you are processing.

Steps for Conducting the Determine HIV Rapid Test

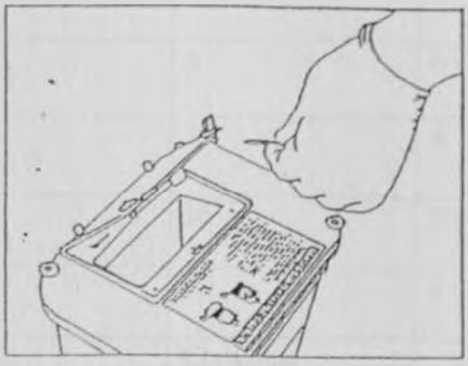
1. Take clean micro-filter tube and place gently on finger. Keep your thumb on the tube and gently tap so tube can fill up with blood.



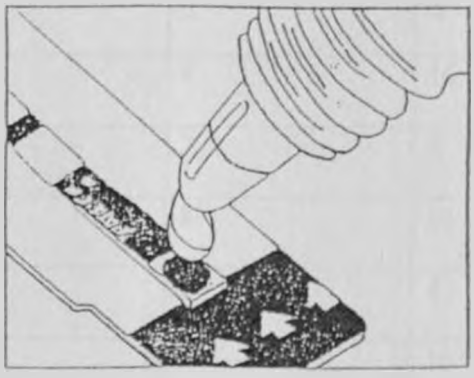
2. When filled, keep thumb on tip of tube and place tube on the gauze. Move your thumb away to place 50 µl or 1 healthy drop of blood on gauze part of strip.



3. Put tube in puncture-resistant



4. Add one drop of buffer solution over the blood on the strip to activate test.



5. This test develops in 15 minutes.

APPENDIX IV

The Revised American Fertility Society Classification System of Endometriosis

Peritoneum	Endometriosis	< 1 cm	1 – 3 cm	> 3 cm
	Superficial	1	2	4
	Deep	2	4	6
Ovary	R Superficial	1	2	4
	Deep	4	16	20
	L Superficial	1	2	4
	Posterior Cul-de-sac Obliteration	Partial	Complete	
		4	40	
	Adhesions	<1/3 Enclosure	1/3-2/3 Enclosure	>2/3 Enclosure
Ovary	R Filmy	1	2	4
	Dense	4	8	16
	R Filmy	1	2	4
	Dense	4[1]	8[1]	16
Tube	R Filmy	1	2	4
	Dense	4[1]	8[1]	16
	R Filmy	1	2	4
	Dense	4[1]	8[1]	16

[1]If the fimbriated end of the fallopian tube is completely enclosed change the point assignment to 16.Stage 1 (minimal): 1 – 5; stage II (mild): 6 – 15; stage III (moderate): 16 – 40; stage IV (severe): >40

APPENDIX V

INFORMED CONSENT ON THE LONG COMMENTARY STUDY

PREVALENCE OF ENDOMETRIOSIS AMONG WOMEN UNDERGOING LAPAROSCOPIC SURGERY

The above study is being carried out at the Aga Khan Hospital, Nairobi, by reviewing patients' case records. All the details on the patients' history, physical examination and management will be treated with utmost confidentiality and only for achieving the objectives of the study. The specific data needed will be derived from the questionnaire attached hereunder.

This study has not been done in Kenya before and therefore, its findings will be useful in reviewing the management of endometriosis and will form a basis of reference for future research on the disease.

Your consent for retrieval and perusal of the patient' case records is, therefore, humbly requested.

Thank you.

Consent granted:

Name.....

Signed.....

Date.....

Witnessed by:

Name.....

Signed.....

Date.....

APPENDIX VI – GYNAECOLOGY DATA COLLECTION QUESTIONNAIRE
QUESTIONNAIRE ON ENDOMETRIOSIS

REG. NO_____

1. Age in Years
   01] 15-20
   02] 20-25
   03] 25-30
   04] 30-35
   05] 35-40
   06] 40-45
   07] 45-50

2. Marital Status
   01] Single
   02] Married
   03] Separated
   04] Wodowed

3. Occupation
   01] Student
   02] Housewife
   03] Employed
   04] Self-employed

4. Race
   01] African
   02] Asian
   03] Caucasian
   04] Other (s), specify_____

5. Parity
   01] 0
   02] 1
   03] 2
   04] 3
   05] 4
   06] >5

6. Number of Abortions
   01] 0
   02] 1
   03] 2
   04] 3
   05] 4
   06] >5

7. Menstrual history
   01] Menarche_____
   02] Duration of flow_____
   03] Cycle of length_____
   04] Menorrhagia
   05] 1° Dysmenorrhoea-absent/present
   06] 2° Dysmenorrhoea-absent/present
   07] Other(s), specify_____
   08] Not Documented

8. Symptoms of endometriosis
   01] Dysmenorrhoea
   02] Chronic pelvic pain
   03] Dyspareunia
   04] Pelvic congestion
   05] Low back pain
   06] Other(s) specify
   07] None

9. Findings on physical examination
   01] Lower abdominal tenderness
   02] Pelvic mass
   03] Adnexal mass
   04] Adnexal tenderness
   05] Retroverted uterus
   06] Nodules in the P.O.D/uterosacral ligaments
   07] Other(s) specify_____
   08] NAD

10. Ultrasonogrphic findings
   01] Ovarian Cyst/mass
   02] Adnexal mass/cyst
   03] Uterine fibroids
   04] Significant fluid in P.O.D.
   05] Enlarged uterus.
   06] Other(s) specify_____
   07] NAD
   08] Not done

11. Hystero-salpingraphy findings prior to laparoscopy
01] Not done
02] Unilateral tubal occlusion
03] Bilateral tubal occlusion
04] Uterine cavity anomally
05] Other (s), specify_____

12. Diagnosis before laparoscopy
01] Endometriosis
02] P.I.D
03] Ovarian cyst/mass
04] Chronic pelvic pain? Cause
05] Infertility
06] Dysmenorrhoea? Cause
07] Other(s), specify_____
08] Unknown

13. Treatment prior to laparascopy
01] None
02] For endometriosis
03] For P.I.D
04] For pelvic pain/dysmenorrhoea
05] For infertility
06] Other(s), specify
07] Not documented

14. Type of infertility
01] None
02] Primary
03] Secondary

15. Duration of infertility in years
01] 0-1
02] 1-2
03] 2-3
04] 3-4
05] 4-6
06] 6-10
07]>-10

16. Presumed known cause of infertility
01] Unknown
02] Endometriosis
03] Hormonal imbalance
04] Tubal factor
05] Other structural anomaly
06] Other(s), specify

17. Treatment for infertility
01] None
02] For endometriosis
03] For hormonal inbalance
04] Tuboplasty
05] Surgery for structural anomaly
06] Other(s), specify_____

18. If treated for P.I.D, how many times?
01] Once
02] Twice
03] Thrice
04] Four times
05] >Five times

19. Laparoscopic findings
01] Endometriosis
02] Ovarian mass/cyst
03] Features of P.I.D
04] Non-specific pelvic adhesions
05] Appendicitis
06] Other(s) specify_____

20. Signs of endometriosis
01] Red petechial lesion(s)
02] Dark brown lesion
03] Dark blue lesion
04] "Powder burn" implants
05] Chocolate cyst/endometrioma(ta)
06] Pseudopouch
07] Other(s) specify_____
08] Not indicated

21. Site of endometriosis
01] Entire pelvic peritoneum
02] Unilateral ovary
03] Both ovaries
04] Pouch of Douglass
05] Uterosacral ligaments
06] Broad ligament(s)
07] Other(s), specify.

22. Tubal patency at dye lap
01] Not tested
02] Both patent
03] Unilateral occlusion
04] Bilateral occlusion
05] Unilateral delayed spill
06] Bilateral delayed spill
07] Other(s), specify_____

23. Revise American Fertility Society Classification of endometriosis
01] Minimal (I, 1-5)
02] Mild (II, 6-15)
03] Moderate (III, 16-40)
04] Severe (IV, >40)
05] Not classified
06] Other(s), specify_____
07

24. Surgical procedure at initial laparoscopy
01] None
02] Adhesiolysis
03] Salipongostomy
04] Excision of endometriotic tissue
05] Fimbriolysis
06] Tuboplasty
07] Other(s), specify_____

25 Cytological findings on P.O.D fluid aspirate
01] Endometriosis
02] Equivocal findings
03] No endometriosis
04] Features suggestive of endometriosis
05] Other(s) Specify_____
06] Not done

26. Histological findings on biopsy tissue
01] Endometriosis
02] Equivocal pathology
03] Other pathology
04] No pathology
05] Not done

27. Treatment given
01] Observation/none
02] Hormonal only
03] Conservative surgery only
04] Conservative surgery and hormonal
05] Definitive surgery only
06] Definitive surgery and hormonal
07] Assisted reproduction
08] Other(s), specify_____

28. Type of hormonal treatment given
01] Combined contraceptive pill
02] Progestational agents
03] Danazol
04] Gestrinone
05] GnRH agonist only
06] GnRH agonist and add back therapy

29. Treatment outcome
01] Symptoms relieved significantly
02] Symptoms subsided (but persisted)
03] Symptoms not relieved at all
04] Conceived
05] Developed complications
06] Did not conceive
07] Other(s), specify
08] Unknown/not documented

30. Complications of treatment
01] None
02] Gut injury
03] Urinal Tract Injury
04] Hormonal side effects warranting medical attention.
05] Other(s), specify_____

31. Hormonal side effects
01] None/not documented
02] Menstrual disturbance
03] Vasomotor symptoms
04] Mental disturbance (specify)
05] Masculinization
06] Breakthrough bleeding
07] Other Hypo-estrogenic side effects (specify)_____
08] Metabolic disorder (specify)
09] Other(s), specify_____

APENDIX VII- RESEARCH APPROVALS

KENYATTA NATIONAL HOSPITAL
Hospital Rd along, Ngong Rd
P O Box 20723, Nairobi

Tel 726300-9
Fax 725272
Telegrams "MEDSUP", Nairobi.
Email KNHplan@Ken.Healthnet.org

Ref: KNH-ERC/01/1940

Date: 25 August 2003

Dr. Chirchir Amon K
Dept. of Obs/Gynae
Faculty of Medicine
University of Nairobi

Dear Dr. Chirchir,

RESEARCH PROTOCOL "TO DETERMINE THE ACCEPTABILITY OF HIV TESTING AND NEVIRAPINE ADMINISTRATION TO SEROPOSITIVE MOTHERS IN EARLY LABOUR" (P14/2/2003)

This is to inform you that the Kenyatta National Hospital Ethics and Research Committee has reviewed and **approved** the revised version of your above cited research protocol for the period 26 August 2003 – 25 August 2004. You will be required to request for a renewal of the approval if you intend to continue with the study beyond the deadline given.

On behalf of the Committee, I wish you fruitful research and look forward to receiving a summary of the research findings upon completion of the study.

This information will form part of database that will be consulted in future when processing related research study so as to minimize chances of study duplication.

Yours sincerely,

PROF. A. N. GUANTAI
SECRETARY, KNH-ERC

Cc Prof. K. M. Bhatt, Chairperson, KNH-ERC
The Deputy Director (C/S), KNH
The Dean, Faculty of Medicine, UON
The Chairman, Dept. of Obs/Gynae, UON
CMRO
Supervisors: Dr. Omondi Oguttu Dept. of Obs/Gynae, KNH
Dr. Wanyoike Gichuhi, Dept. of Obs/Gynae, UON

PUMWANI MATERNITY HOSPITAL

Tel: 02/6763291 - 4
Fax: 02/6762965

P.O. Box 42849
Code: 00100- GPO
Nairobi

REF: PMH/DMOH/79/130
DATE: 21ST JANUARY 2004

DR. AMON CHIRCHIR
UNIVERSITY OF NAIROBI,
DEPARTMENT OF Obs & GYNAECOLOGY

RE: RESEARCH UPDATE MEETING

The Research and Ethics Committee at Pumwani Maternity Hospital would like to invite you to a meeting to be held on Thursday 29th January 2004 at Pumwani Maternity Hospital conference hall at 9.30am.

The meetings purpose is to

- Meet all the students undertaking researches at Pumwani Maternity Hospital
- Introduce the members of the Research and Ethics Committee and internal supervisors assigned to the students.

Kindly avail yourself for this meeting to enable us to facilitate your research undertaking.

Yours faithfully

DR. DAVID KIRAGU
MEDICAL SUPERINTENDENT

KENYATTA NATIONAL HOSPITAL
Hospital Rd. along, Ngong Rd.
P.O. Box 20723, Nairobi

Tel 726300-9
Fax 725272
Telegrams "MEDSUP", Nairobi

Ref: KNH-ERC/01/2097

Date: 15 January 2004

Dr. Chirchir Amon K
Dept of Obs/Gynae
Faculty of Medicine
University of Nairobi

Dear Dr Chirchir,

RESEACH PROPOSAL "TO DETERMINE THE PREVALENCE OF ENDOMETRIOSIS AMONG WOMEN UNDERGOING LAPARASCOPIC SURGERY AT THE AGA KHAN HOSPITAL, NAIROBI (P117/10/2003)

This is to inform you that the Kenyatta National Hospital Ethics and Research Committee has reviewed and **approved** the revised version of your above cited research proposal for the period 15 January 2004 – 14 January 2005. You will be required to request for a renewal of the approval if you intend to continue with the study beyond the deadline given.

On behalf of the Committee, I wish you fruitful research and look forward to receiving a summary of the research findings upon completion of the study.

This information will form part of database that will be consulted in future when processing related research study so as to minimize chances of study duplication.

Yours sincerely,

PROF. A. N. GUANTAI
SECRETARY, KNH-ERC

Cc Prof. K Bhatt, Chairperson, KNH-ERC
The Deputy Director (C/S), KNH
The Dean, Faculty of Medicine, UON
The Chairman, Dept of Obs/Gynae, UON
Supervisors Dr. O. Ogutu, Dept. of Obs/Gynae, UON
Dr. Wanyoike Gichuhi, Dept of Obs/Gynae, UON
Dr. Evans Sequeira, Dept. of Obs/Gynae, Aga Khan Hospital, Nairobi

The Aga Khan Hospital, Nairobi
An Institution of the Aga Khan Health Service, Kenya

P O Box 30270 00100 GPO Nairobi, Kenya
Telephone 3740000 / 37425311 / 3
Fax 3741749

January 27, 2004

Dr. Anon Chirchir
P O Box 19676
Nairobi

Dear Dr. Chirchir

Re: To determine the prevalence of Endometriosis among women undergoing Laparoscopic surgery at the Aga Khan Hospital, Nairobi

I am in receipt of your request to carry out a research in this hospital.

Please note that your request has been approved and permission granted to carry out your study, under the supervision of Dr. E. Sequeira – the Clinical Director, Obs & Gynae Department. The following are the terms and conditions of the study: -

1. This hospital will in no way be responsible for funding of this project.
2. No material belonging to the hospital e.g. files, diskettes, etc may be taken out of the hospital premises.
3. On completion of the study, a copy of the report will be presented to the Hospital or the result of the study may be given in a lecture form to the medical fraternity in the hospital.
4. No part of the study may be published without written permission from The Aga Khan Health Service, Kenya.

Yours sincerely,

Mr. M. M. Qureshi
Medical Director

Copy to: Chairman - Education Committee
Dr. S. Malik - Chairman, Ethics Group
Dr. E. Sequeira - Clinical Director, Dept. of Obs & Gynae