CASE REPORTS AND COMMENTARIES IN

OBSTETRICS AND GYNAECOLOGY

PRESENTED IN PART FUFILLMENT FOR

THE EXAMINATION OF

MASTER OF MEDICINE IN

OBSTETRICS AND GYNAECOLOGY

OF THE OF UNIVERSITY OF NAIROBI BY

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NOVEMBER 2005

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DEDICATION

This book is dedicated to my entire family and to all those whose lives are closely intertwined with mine for their love, support and encouragement.

ACKNOWLEDGEMENT

I wish to express my gratitude to the Department of Obstetrics and Gynaecology of the University of Nairobi for accepting me into the department for this training. My sincere gratitude to the Government of Kenya through the Ministry of Health for sponsoring me through this programme.

I would like to thank all the consultants, lecturers, senior registrars and fellow registrars in the department of obstetrics and gynaecology for their dedication and commitment to see that I achieved the necessary knowledge and skills during my training at the University of Nairobi.

I also express my sincere gratitude to my supervisors, Dr Njoroge Waithaka and Dr. Magoha for their efforts to see that my proposals and long commentaries were properly written by offering expert advice and guidance and also making meaningful critique for the long commentaries and the short cases. I also thank Dr Oyieke J.B for his invaluable assistance, guidance and advice during my training.

I thank the records department of Kenyatta National Hospital especially Mrs. Mbela for their assistance during data collection for the obstetric long commentary.

I thank the Ministry of Education especially Mr. B.O. Adewa (Research authorisation office) for allowing me to conduct the gynaecology long commentary in secondary schools in Laikipia district. Many thanks to head teachers of schools that participated in the study particularly Mr. Kamanja of Rumuruti for facilitating easy entry even in other schools apart from his school. I would not forget the students who participated in the study.

I thank Mr. Francis Njiiri for data analysis for the long commentaries. I would not forget to appreciate the work of Anne and Sharifa in typing this work.
It was not possible to mention all to who I am grateful but whoever and wherever you are thank you very much for your assistance and cooperation. God bless you all.

DECLARATION

This is to certify that the case reports and commentaries presented in this book are my original work and were managed by me under supervision of senior members of the Department of Obstetrics and Gynaecology, Kenyatta National Hospital, Nairobi.

Signature.....

Date.....16/12/2005.....

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CERTIFICATE OF SUPERVISION

This is to certify that the long commentaries in this book by Dr David Gicheru Kariuki were researched upon under my guidance and supervision and that this book is submitted with my approval

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   Date.....16/12/2005.....

CERTIFICATION

This is to certify that Dr David Gicheru Kariuki managed Obstetric case Nos. 2, 5, 10, 11, 15 and Gynaecology case Nos. 1, 2, 6, 11 and 13 under my supervision at Kenyatta National Hospital

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Date 10/02/06.....

CERTIFICATION

This is to certify that Dr David Gicheru Kariuki managed Obstetric case Nos. 1, 6, 12 and Gynaecology case Nos. 7, 8 under my supervision at Kenyatta National Hospital

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Date.....13th February 2006.

CERTIFICATION

This is to certify that Dr David Gicheru Kariuki managed Obstetric case Nos. 4, 7, 9, 13, 14 and Gynaecology case Nos. 3, 5, 9, 10 and 14 under my supervision at Kenyatta National Hospital

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Date.....16/12/05.....

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This is to certify that Dr David Gicheru Kariuki managed Obstetric case Nos. 3, 8 and Gynaecology case Nos. 4, 12, 15 under my supervision at Kenyatta National Hospital

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Signed.....

Date.....31 January 2006.....

INTRODUCTION

The obstetric and gynaecology short cases presented were managed at the Kenyatta National Hospital. The hospital is situated about 3 kms from the Nairobi city centre along Ngong Road. In addition to being a national referral hospital, it also serves as a teaching hospital for the college of health sciences of the university of Nairobi offering facilities for undergraduate and postgraduate medical courses. Nursing and paramedical courses are also offered within this hospital.

OBSTETRIC AND GYNAECOLOGY UNIT

The obstetric and gynaecology outpatient services are offered at the antenatal and gynaecology clinics, the casualty department and the family welfare clinic (Now based at clinic 18). The inpatient services are provided for in the labour ward, acute gynaecology ward (1 D) and the lying in wards (Antenatal and Postnatal wards-GFA, GFB and 1A) and the elective gynaecology ward (1B).

In addition to the hospital laboratory services, the Department of Obstetric and Gynaecology offers other laboratory services including the following for the hospital; semen analysis, hormonal radio-immunoassay, cytology, chromosome analysis, surfactant test, spectrophotometry and glucose tolerance tests, and post-coital test. Ultrasound foetal monitoring and radiological examinations are provided for in the radiology department of KNH and also at the University of Nairobi's Department of Radiology.

CASUALTY DEPARTMENT

This offers services 24 hours a day and all obstetric and gynaecology emergencies are screened here. Most of the patients are treated and discharged while the more serious cases are admitted either to labour ward or acute gynaecological ward. Senior house officers offer obstetric and gynaecology coverage 24 hours a day.

ANTENATAL CARE

This is mainly a high-risk antenatal clinic although hospital staff members are also booked in this clinic. Booking is done every Monday morning. The criteria for booking include – primigravida, grandmultipara, medical conditions complicating pregnancy, previous operative deliveries, bad obstetric history and those who have had difficult gynaecological operations such as repair of urinary fistula or myomectomy- among other high risk factors.
For those who are booked, a detailed history of the patient's past obstetric and gynaecological history, medical and social history is taken. The patients are then sent to the laboratory for antenatal profiles which includes; blood group and rhesus factor, serologic test for syphilis (VDRL), urine analysis for protein and sugar, full haemogram especially the haemoglobin level, and voluntary counseling and testing for HIV. For first pregnancies or previous pregnancies with more than three year intervals, two tetanus

second trimester. Proteinuria, glycosuria, blood pressure and weight gain are checked during every clinic visit.

The teenage mothers have had their own antenatal clinic on Monday afternoons. This was started when adolescent pregnancies were found to be an important public health concern in the country. The important consideration is that teenage pregnancy is associated with more complications such as higher incidence of hypertensive disease, low birth weight, anaemia and sexually transmitted infections.

During the antenatal period, any antenatal morbidity is managed either as outpatient or inpatient. At 36 weeks gestation, clinical pelvimetry is done on all primigravida. Erect lateral pelvimetry is done on those with one previous caesarean scar to assess those destined to deliver vaginally. Amniocentesis is done in few selected cases at 38 weeks in those mothers who are planned for elective delivery and gestation is not ascertained. In addition, during the antenatal period appropriate the staff provides health education on pregnancy and its related problems in all clinical sessions. Emphasis is laid on nutrition, regular clinic attendance, recognition of risk factors (such as excess weight gain, severe headache, vaginal bleeding, reduced foetal movements), preparation for labour and delivery, post partum care, prevention of mother to child transmission of HIV (PMTCT), breastfeeding and birth spacing/contraception.

VOLUNTARY COUNSELLING AND TESTING FOR HIV

This is offered to all pregnant mothers; those found negative are encouraged to continue protection against infection and about exposure prophylaxis. Those who are positive are informed about the various methods available for preventing mother to child transmission of HIV. Currently they are given zidovudine (300mg twice daily) from 34 weeks of gestation up to the time of labour when this is given every three hours until delivery and nevirapine 200mg stat in labour while the infant gets nevirapine 2mg/kg. For those discovered positive after 36 weeks or test positive in labour are given nevirapine 200mg stat while the infant gets nevirapine 2mg/kg (within 72 hours of delivery) and zidovudine 2mg.kg 12 hourly for one week. They are also given the option of caesarean section as method for PMTCT and formula milk for those who opt not to breastfeed. Highly active antiretroviral therapy (HAART) is also offered to those found with low CD4 counts (below 200) mainly with combination of zidovudine, lamivudine and nevirapine.

HOSPITAL ADMISSIONS

These fall into three categories namely:

- Booked patients from our antenatal clinic
- Referrals from other hospitals or health centers
- Those without prior antenatal care.

The last two categories constitute the majority of admissions previously but this trend has changed after antenatal booking at Kenyatta National Hospital was allowed to all mothers who can afford without necessarily being high risk. Booked patients report directly to

labour ward admission area when they are in labour or when they develop any problems and the clinics are closed. Unbooked patients are seen first in the casualty before being sent to labour ward admission area. The patients are then seen by a house officer (intern) in conjunction with a senior house officer (registrar).
Other members of be called if need arises. Those in labour are admitted to the labour ward while those not in labour are admitted to the lying in wards if so required or discharged home. Patients who are very ill are admitted to the acute room in labour ward and managed accordingly.

MANAGEMENT OF LABOUR

The first stage of labour

During the first stage, both the doctor and primary mid-wife keep an accurate record of the progress of labour. The partogram is utilized and consists of the following: -

1. Patient identification, parity and time of admission
2. Date, time and mode of rupture of membranes, and the colour of the liquor.
3. Date and time of onset of labour
4. Half hourly foetal heart rate monitoring
5. Progress on descent of the foetal head into the pelvis
6. Progress on cervical dilatation, recorded 4 hourly
7. Uterine contractions each 10 minutes, their frequency and duration
8. Use of oxytocin, its concentration and rate of infusion
9. Other drugs used, dosage and time of administration
10. Maternal vital signs every half hour

Active management of labour is done aiming at delivery within 12 hours of onset of active labour. Vaginal examinations are preformed every 4 hours or earlier where indicated; aseptic technique is employed and after the bladder has been emptied. Amniotomy is routinely done at or after 4cm of cervical dilatation. Analgesia is usually given early in the active phase of labour. Usually intramuscular pethidine is used but tramadol can also be used. With the partogram, foetal distress, impending uterine rupture and maternal distress are noted early and intervention instituted. In HIV patients' amniotomy is delayed since it has been shown that more than 4 hours of ruptured membranes increases the chances of mother to child transmission of HIV. The patient is nursed in the left lateral position and is reviewed at regular intervals by the registrar, during which appropriate interventions are effected if the progress is poor.

Induction of labour is routinely performed in the morning, usually by amniotomy and oxytocin drip. Where indicated, prostaglandin vaginal pessaries may be inserted the night before to ripen the cervix. Extra-amniotic prostaglandin induction is used for cases with intrauterine foetal death.

Second stage of labour

Second stage is reached when the cervix is fully dilated and the patient may experience an urge to bear down. When this is confirmed, she is transferred to the second stage delivery room. In the delivery room, she is placed in lithotomy position, the vulva and perineum cleaned with antiseptic solution (chlorhexidine) and draping done. The mid-wife or doctor encourages the patient to bear down during a uterine contraction. The foetal heart is monitored every five minutes. If the perineum is tight, 10mls of 2% lignocaine hydrochloride is infiltrated locally and a mediolateral episiotomy is made during crowning of the foetal head. A sanitary pad is used to support the perineum as the head is delivered. This prevents trauma to the perineum and foetal head in the preterm babies. When the head is delivered, the baby's mouth, nostrils and eyes are wiped with sterile gauze. A finger is passed to feel the neck and ensure the umbilical cord is not around it. If the cord is felt and tight, it is clamped at two points 3cm apart and cut in between. If loose, it is slipped over the head. Restitution is then allowed to occur by supporting the head between the palms of the hands. Gentle downward traction is applied to deliver the anterior shoulder, and then upward traction applied to deliver the posterior shoulder. The rest of the body quickly follows with further bearing down by the mother. Where the cord is still intact, is clamped and cut. The baby is handed over to the receiving mid-wife or paediatrician who resuscitate the baby where necessary, check the Apgar score and weigh the baby. Ergometrine 0.5mg intramuscularly is given by an assistant at delivery of the anterior shoulder unless it is contraindicated as in cardiac disease, hypertension or before completed delivery in multiple delivery. Intravenous ergometrine or an infusion of oxytocin is given where postpartum haemorrhage is anticipated.

Third stage of labour

Spontaneous separation of the placenta is usually awaited. Separation is indicated by a sudden gush of blood from the introitus, lengthening of the cord and contraction of the uterus with rising of the fundus into the abdomen as the placenta passes down into the lower segment. The placenta and membranes are then gently delivered by controlled cord traction. The placenta, umbilical cord and the membranes are inspected for normalcy and completeness. Blood clots are expelled from the vaginal cavity. The cervix, vaginal walls and the perineum are inspected and explored for lacerations or tears that are repaired if present, and any bleeding checked for. The episiotomy is then repaired using chromic catgut No.1 in three layers. The placenta is weighed and total blood loss recorded. The patient is encouraged to empty the bladder immediately, post delivery blood pressure, pulse rate, uterine contraction and lochia loss are observed and recorded.

Fourth stage of labour

All mothers after delivery are observed in the fourth stage room until stable and ensuring that they have no bleeding. The vital signs are recorded and observations continued half hourly for about 2 hours. The patient keeps her bladder empty and a vulva pad monitored for any excessive blood soaking. 'Rooming in' is encouraged and early initiation of

breastfeeding within 30 minutes is advocated as long as there is no contraindication. The patient is adviced on perineal hygiene and sitz baths until the episiotomy heals. They are also given advice on neonatal and infant care and proper breast-feeding as well as how to recognize infection. If no complications are noted, she is transferred to the lying-in wards to continue resting and observations. If no problems arise, and both mother and baby are well, immunization has been started, they are discharged home after 24 hours to be followed up in the post-natal clinic after six weeks.

SPECULUM EXAMINATION

Speculum examination is performed on patients complaining of bleeding or premature rupture of membranes where immediate delivery is not indicated. The patient is placed in the lithotomy position, the vulva is cleaned with antiseptic solution such as savlon or hibitane then draped with sterile towels. A Cuscos speculum is gently introduced in the vagina and the blades slowly opened. Using a light source lesions of the vaginal wall and cervix are identified. Bleeding or drainage of liquor through the cervical opening is noted.

The Sim's speculum is also used. It is either single or double ended. It is used mainly to view the vagina or cervix for any lesions, for examination under anaesthesia, genital fistulas repair or McDonalds stitch insertion. The patient lies in Sims position i.e. in the left lateral position, with the left lower limb extended at both the hip and knee while the right lower limb is flexed at the hip and knee joints and pushed interiorly ever the left limb. The surgeon, already gloved, retracts the right buttock to expose the vaginal introitus with the left hand, while with the right hand and under good vision, he introduces the already lubricated speculum into the vagina along the posterior vaginal wall in the transverse diameter. The speculum is then gently pushed towards the vaginal vault, while at the same time inspecting the vaginal wall and cervix. The patient could also be positioned in the lithotomy position, as the condition may deem necessary e.g. during examination under anaesthesia, repair of cervical tear, and cervical cerclage. The Auvard speculum, a weighted speculum is also used during procedures such as during insertion of McDonalds stitch.

OPERATIVE PROCEDURES

1. EPISIOTOMY

This is done when necessary in the second stage of labour. A midline or medio-lateral episiotomy is performed at crowning of the foetal head at the perineum in all cases where the perineum is very tight and for preterm deliveries and for other operative deliveries such as vacuum extraction. A medio-lateral episiotomy is the more commonly used procedure in our unit because it has less risk of extension to the anal sphincter and rectum. The perineum is infiltrated with local anaesthetic (1% lignocaine hydrochloride) at the site where episiotomy is to be given. During the procedure, the index and middle fingers of one hand are introduced into the introitus, between the perineal wall and the baby's head to spread out the perineum and to protect the baby's head from injury by the

scissor blades. An adequate episiotomy is then done using blunt tipped Mayo's scissors to cut through the bulbospongiosus and superficial perineal muscles.
The episiotomy is then repaired immediately after delivery of the placenta. The patient still in lithotomy position, the vagina and perineum are inspected for any lacerations that may require repair. The vulva and perineum are then cleaned with antiseptic solution and a piece of gauze placed in the vagina beyond the apex of the episiotomy to keep off uterine bleeding. The apex of the episiotomy is identified by retracting the vaginal walls with the fingers of one hand. Using the other hand repair is done in layers starting at the apex of the incision. The vaginal mucosa is repaired using continuous chromic catgut 2/0 suture. The perineal muscles are repaired using interrupted or continuous suture with chromic catgut 2/0. The skin edge is then apposed using interrupted or continuous catgut 2/0 burying the knots and starting from the lateral edge. Some obstetricians use vicryl 2/0 or catgut subcuticular sutures for the skin.
The gauze in the vagina is then removed and the uterus squeezed to effect contraction and to expel any clots. The perineum is finally cleansed with antiseptic solution and a sterile sanitary pad applied over the vulva and perineum and the patient placed in supine position, then lateral position and covered. She is then adviced on perineal hygiene and frequent sitz baths until healing occurs.

2. VACUUM EXTRACTION

The vacuum extractor (ventouse) is used to accomplish delivery in prolonged second stage due to poor maternal efforts or where bearing down is contraindicated as in patients with cardiac disease or hypertensive disease or where delivery expedition is desired as in foetal distress occurring in the second stage of labour.
The patient is placed in the lithotomy position. The vulva and perineum are cleaned with antiseptic solution and draped. Aseptic catheterization of the urinary bladder is done and repeat vaginal examination performed to rule out any contraindication to vacuum delivery such as cephalopelvic disproportion and foetal malpresentation. The foetal head should be in the pelvis with only one fifth palpable above the pelvic brim. The largest suitable ventouse cap is placed on the foetal scalp close to the occiput taking care not to include maternal soft tissue by running a finger around the cap. Under local anaesthesia, a medio-lateral epsisiotomy is performed and the vacuum negative pressure is increased stepwise at intervals of $0.2kg/cm^2$ every 2 minutes to a maximum of $0.8kg/cm^2$. At each increase in pressure, a check is repeated for any maternal tissue around the perimeter of the cap. During this process an artificial caput or "chignon" is created. When a caput is already present, the negative pressure may be achieved faster. Traction is then applied with each contraction, in a downward direction until the head descends and then upwards to allow delivery by extension. On delivery of the foetal head, the ventouse cap is released immediately and the second and third stages of labour conducted as usual.

3. CAESAREAN SECTION

The lower segment caesarean section is the commonest major obstetric operation performed either as elective or emergency operation. The classical caesarean section is rarely done except for cases of transverse lie with ruptured membranes and live baby.

PRE-OPERATIVE MANAGEMENT

For those undergoing elective caesarean section, haemoglobin estimation and renal function are checked together with cross matching for two units of blood reserved for the operation. Informed consent for general anaesthesia is taken. The abdominal wall, vulva and perineum hair is shaven. Clipping of the hair instead of clean shaving is the recommended method nowadays as its more comfortable to the patient, wounds are less septic and transmission of diseases like Hepatitis and HIV is minimized. For those undergoing elective operation, starving for 6 hours prior to the procedure is adviced. Premedication is given in the form of atropine 0.6mg intramuscularly half hour before going to theatre. In cardiac patients hyoscine 0.4mg is used instead of atropine.

Surgical procedure

In theatre, the patient is placed in supine position and an intravenous infusion is started through a large bore needle (this is started while in the lying-in ward for elective cases). In the semi-lithotomy position, the vulva and perineum are cleaned with 1% savlon solution. Aseptic catheterization is then done and all urine drained and the catheter retained to provide continuous bladder drainage during the operation. The patient is then repositioned to the supine position. The anterior abdominal wall is cleaned with antiseptic solution and iodine/spirit solution (Betadine). Sterile draping is then done exposing only an area bounded by the mons pubis below to about 4cm above the umbilicus and 2cm on each side of the midline if subumbilical incision is to be used. If pfannenstiel incision is to be used the upper border need not be placed above the umbilicus.
100% oxygenation is given to the patient for 5 minutes then general anesthesia is induced using intravenous thiopentone sodium 250-500mg depending on the patient's weight. A short neuromuscular blocking agent suxamethonium 100mg is used to provide muscle relaxation. Anaesthesia is maintained with nitrous oxide and oxygen in the ratio of 1:1 before the baby is delivered then a ratio of 2:1 is continued. A total of 6-8 litres per minute is used depending on the circuit used. Throughout the operation, halothane 0.5% or trilene 0.35% is used to maintain unawareness. When the effect of suxamethonium has worn off, pancuronium or D-tubocurare, long acting muscle relaxant is used.

Abdominal incisions

The abdominal incisions commonly used in obstetric operations in our unit are the sub-umbilical midline and the Pfannenstiel incisions with the former being more popular than the latter though the trend is changing.

The midline incision

A midline skin incision is made with a sterile scalpel extending from 2cm below the umbilicus to the pubic hairline. A second knife is then used to open the fat in the same way up to the rectus sheath, the bleeding points being picked up along with Dunhill forceps and tied with No 1 catgut suture or coagulated with diathermy. The rectus sheath is incised at the midline with a scalpel making a small opening through which a pair of curved scissors is introduced and used to undermine the rectus sheath to separate the

muscle from their attachment to it and the incision extended in a vertical direction superiorly or inferiorly. The rectus sheath is then deflected to one side of the midline to expose the parietal peritoneum, which is lifted up with two long artery forceps placed about 1cm apart and palpated to exclude gut or omentum being interposed between the two artery forceps. A small incision is made in the peritoneum between the 2 artery forceps using a pair of curved scissors. Under direct vision and with guidance of the index and the middle fingers of the other hand, the incision is extended upwards and downwards the whole length of the skin incision, care being taken not to injure the bladder and bowels.

Pfannenstiel incision.

A transverse incision is made along the skin approximately 2cm above the pubic symphysis using a sterile scalpel. The scalpel is changed and the fat opened up transversely up to the rectus sheath, which is also opened up transversely and peeled off the rectus muscles in all directions by blunt dissection. The rectus muscles are separated vertically along the linea alba to expose the parietal peritoneum that is lifted up with 2 long artery forceps and after excluding the presence of gut, it is opened in the vertical lane as explained earlier.

Joel Cohen incision

This incision is made 3cm below a line joining the anterior superior iliac spines. Using a scalpel an incision is made on the skin leaving the subcutaneous tissue. A small incision is made on the subcutaneous tissue at the middle deep up to include the rectus sheath. The incision on the rectus is then extended on either side using a pair of partially opened dissecting scissors under the layer of subcutaneous tissue that was left intact. Using some force, the rectus muscles are separated apart and the underlying peritoneum opened transversely using both index and forefinger of either hand.

THE LOWER UTERINE SEGMENT CAESAREAN SECTION

Under general anaesthesia in supine position the abdomen is opened via any of the above-described incisions. The uterus is then identified. Tagged moist abdominal packs may then be placed on either side of the uterus to prevent blood and liquor from running into the general peritoneal cavity and protect the gut. A Doyen's retractor is placed to deflect the bladder away as well as expose the uterovesical fold of peritoneum. The peritoneal fold is then picked up with a non-toothed forceps and opened at the middle and undermined on either direction. The incision is then extended on either side transversely and the peritoneum stripped off the lower uterine segment with a mounted swab. The Doyens retractor is shifted to include the lower part of the peritoneal fold in retracting the bladder from the lower uterine segment.

A small incision of about 2cm is made in the lower segment about 2cm below the uterine attachment of the uterovesical fold. Once the membranes are reached, the incision is extended on either side using curved scissors directed by two fingers of the left hand or stretched transversely using the right thumb to steady and left index finger to separate the muscle fibres towards the right. The opening is in an upward directed semi-lunar incision to avoid the uterine arteries at the angles and more to the right as the uterus is

dextrorotated. The incision is enlarged enough to allow delivery of the head and trunk. The membranes are then ruptured if present. If the placenta is encountered in the line of incision, it is either deflected or incised but in the latter case severe feto-maternal haemorrhage may occur and therefore the cord has to be clamped quickly.

The retractor is then removed. If the presentation is vertex, a hand is then slipped into the uterus between the foetal head and symphysis pubis and the head lifted gently with fingers and palm through the incision while a modest transabdominal pressure is applied. After delivery of the head, the nostrils and mouth are sucked. The shoulders are then delivered using gentle traction and still with some fundal pressure. The trunk delivery follows readily. Intravenous ergometrine 0.5mg is given as the baby's shoulders are delivered. The cord is clamped and divided then handed over to an assistant for resuscitation.
The placenta is delivered manually unless it separates spontaneously. The cut edges of the uterus are held with green armytage uterine clamps to control any bleeding that might be occurring as the inside of the uterus is wiped off clots and membranes. The placenta is also inspected for completeness. The uterus is lifted out of the abdominal incision and covered with a wet abdominal pack. If the cervix was not dilated in labour, it is dilated at this juncture with a mounted swab to allow postpartum lochia drainage. The uterus is then closed in 2 layers with No. 2 chromic catgut as a continuous stitch for both layers, the second layer burying the first and extending beyond the lateral edges of the first. The visceral peritoneum is then closed with No. 1 chromic catgut continuous stitch.

The abdomen is mopped and the abdominal packs removed. The pelvic viscera are then inspected for any abnormalities. Instruments and swabs are counted and if they tally with the initial count, then the abdominal wall is closed in 3 layers. The peritoneum is closed with continuous No. 1 chromic catgut suture, the rectus with No. 2 chromic catgut (currently vicryl No.1 is also available for use) and the skin with interrupted nylon or silk suture or subcutaneous vicryl No. 2/0. The wound is then cleaned with antiseptic and dressed with iodine solution. A light strapping is used to hold the gauze dressing in place. The catheter is then removed and the uterus is massaged and clots evacuated from the vagina. A clean vulval pad is placed to monitor any bleeding and to keep the patient clean. General anaesthesia is reversed with 1.2mg of atropine and 2.5mg of neostigmine intravenously. Extubation is then done and oropharyngeal suctioning for any secretions carried out. Blood loss is estimated and recorded and the patient then transferred to the recovery room, then later to the labour ward when fully awake from anaesthesia.

POST-CAESAREAN SECTION CARE

The patient is observed half hourly noting the blood pressure, temperature, pulse rate and respiratory rate on a chart, until she is fully awake, then four hourly. Intramuscular pethidine 50mg 6 hourly is given for 48 hours to relieve pain. Intravenous 5% dextrose and normal saline are given alternately as 500mls four hourly until bowel sounds are established. Prophylactic antibiotics are given to those at high risk of getting sepsis. On the third postoperative day haemoglobin level is checked and urine culture done. The stitches are removed after seven days of the operation, after which the patient is

discharged home with a case summary and having been explained about the nature and findings of the operation. The mother is seen in the postnatal clinic after six weeks and the child is seen in the child welfare clinic in 2 weeks.

CARE OF THE NEWBORN

All the newborn babies who are normal join their mothers after delivery unless the mother is moribund. The babies with problems or where complications are anticipated together with babies delivered by operative vaginal delivery or by caesarean section are all reviewed by the paediatric registrar. Those having problems or who may develop some problems are transferred to the newborn unit in a warm incubator. The premature babies are managed in nursery until their weight is about 2000g when they are discharged. All babies are immunized with BCG before discharge.. The normal mothers who have babies in nursery are lodged in a mother's hostel.

POSTNATAL FOLLOW UP

The clinic is held every Friday morning. Only those who had a complicated or operative delivery are seen. Patients with normal deliveries are followed up in the health facilities nearest to their homes.
The blood pressure and weights are taken. Urinalysis is done. A history of the puerperium lactation and immunization of the baby is taken. The patient is then examined and any problems managed. Family planning advice is given and the patient referred to the family planning clinic for the various methods available.

THE GYNAECOLOGY UNIT

This consists of the outpatient wing at clinic 18 and two gynaecological wards 1B and 1D on the first floor of the hospital's tower block.
Ward 1D is the acute gynaecological ward whereas ward 1B caters for non-emergency cases.
The unit is ran by the three firms in the department.

Gynaecology outpatient services

These are mostly conducted in the clinics, which are held on three days every week by the three firms; Firm 1 on Tuesday, Firm II on Thursday and Firm III on Wednesday. Consultants, senior registrars and senior house officers run the clinics. Medical students are usually also in attendance. There is also an oncology clinic, which is on Friday mornings for follow-up of oncology patients. A colposcopy clinic is held every Friday morning. A fertility clinic is held every Monday afternoon. The majority of patients attending the gynaecology clinic are referred from casualty and emergency gynaecology ward after emergency consultation and treatment. Other patients are referred from other specialist clinics of Kenyatta National Hospital, other hospitals in and around Nairobi as well as from district and provincial hospitals.

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Infertility cases constitute two thirds of the gynaecology consultations, followed by uterine fibroids, abnormal uterine bleeding and adnexal masses. In the clinic, history is taken, thorough physical examination is conducted and most of the investigations are done from the clinic to reduce hospital stay upon admission. These investigations include haemogram, urea and electrolytes, semen analysis, Pap smear, hysterosalpingogram, pregnancy tests, pelvic scans among others.

FAMILY PLANNING CLINIC

It is situated at the family welfare center (clinic 66). Hormonal contraceptives, intrauterine devices, barrier and surgical methods of contraception are offered. Also situated in this clinic is a theatre for laparoscopy and tubal ligation procedures. Patients requiring interval sterilization are counseled and referred to this clinic for the procedure by mini-laparotomy or laparoscopy.

RADIOTHERAPY AND ONCOLOGY SERVICES

Department of radiotherapy offers services to all disciplines of medicine and also serves the entire country and neighbouring countries. Patients sent for radiotherapy from our unit are mainly those with advanced and inoperable carcinoma of the cervix and other radiosensitive tumours. For carcinoma of the cervix, external beam radiation using Cobalt-60 is offered. Once a full course of this has been given, patients are given intracavitary radiation using Caesium-137. They are followed up in the radiotherapy clinic. Occasionally a specialist oncologist is consulted to review treatment of gynaecology oncology patients in the wards.

COLD GYNAECOLOGY IN-PATIENT SERVICES (WARD 1B)

This is the non-emergency ward to which patients are usually admitted from the clinic or are transferred from the acute gynaecology ward for further management. The ward has 36 beds divided among the three firms. Commonly, the patients admitted here have uterine fibroids, genito-urinary fistulae, gynaecological malignancies and infertility among others.

ACUTE GYNAECOLOGY IN-PATIENT SERVICES (WARD 1D)

This is the emergency gynaecology ward having 32 beds. On average, about 15 patients are admitted daily, and more than two thirds of these are cases of abortion, admitted mainly through the casualty department. All patients are reviewed by the houseman and reviewed by the registrar who undertakes the management in consultation with senior members of the department. Apart from the incomplete abortion, other common cases admitted are pelvic inflammatory disease, ectopic pregnancies and pelvic abscesses.

Uncomplicated cases of incomplete abortion have uterine evacuation done in the procedure room using Karman's cannula and discharged home on the same day if stable. Patients who have undergone emergency laparotomies for pelvic abscess, ectopic

pregnancies have a minimum stay of four days postoperatively. All patients with incomplete abortion and have had uterine evacuation are counseled about contraception before discharge.
Patients with suspected carcinoma of the cervix are admitted to this ward for work-up and emergency care such as blood transfusion, antibiotics and analgesic therapy. Routine clerking is done and laboratory investigations done. Thereafter examination under anaesthesia for staging and biopsy is done. When the histology report becomes available they are either transferred to ward 1B or radiotherapy clinic for definitive management.

GYNAECOLOGICAL OPERATIONS

A theatre is reserved in main theatre for emergency gynaecological operations daily. Laparotomies for ectopic pregnancies (ruptured and non-ruptured), pelvic abscess, ovarian cyst and other tubo-ovarian masses are done here. Smaller procedures like diagnostic dilatation and curettage of the uterus, removal of misplaced intrauterine contraceptive devices, marsupialisation and suction curettage are also done here.

Elective operations are done on a firm basis, Firm II on Mondays, Firm I and III on Thursdays. The operations are done from 8.00 a.m. to 5.00 p.m. The operations are performed under general anaesthesia in which intravenous sodium thiopentone and succinyl choline are used for induction of anaesthesia, nitrous oxide, oxygen and halothane for maintenance of anaesthesia, curare is given intermittently to provide muscle relaxation and atropine plus neostigmine are used for reversal from anaesthesia.

PRE-OPERATIVE MANAGEMENT

Patients for emergency laparotomy are prepared for theatre immediately in ward 1D. The abdomen is cleaned and shaved, stomach contents aspirated if the patient has fed just before admission. Premedication is provided by atropine 0.6mg intramuscularly half hour before theatre. Blood is urgently cross-matched and an intravenous drip started.
For non-emergency operations, basic and special investigations are done and the date of surgery is fixed. The nature and purpose of the operation is explained to the patient after which she gives an informed consent. Blood is ordered and reserved for the day of operation. The patient starves from midnight prior to the morning of operation. The skin over the area of operation is cleaned and shaved. A soap enema is given on the morning of operation. Premedication is given as atropine 0.6mg and pethidine 100mg intramuscularly half hour before being wheeled to theatre.

POST-OPERATIVE MANAGEMENT

After the general anaesthesia is reversed and the patient wheeled to the recovery room, half hourly observations of blood pressure, pulse rate, respiratory rate and temperature are taken until she is fully awake. She is then transferred to the ward where observations are taken four hourly.
Most laparotomy patients stay in the ward for seven days. Those who are stable are allowed home on the fourth day. For the first 24 hours the patients are maintained on

intravenous fluids. Oral fluids are given when bowel sounds are established. Blood transfusion is given where indicated. Prophylactic antibiotics are given routinely in our set up but may not be necessary if prophylactic antibiotics are given at the beginning of the operation. If the operation was for a septic condition like abscesses, then combination of broad-spectrum antibiotics covering aerobic and anaerobic organisms are given. A check haemoglobin level is determined on the third postoperative day. Stitches are removed on the 7th postoperative day if non-absorbable stitches were used.
Before discharge, the patient is informed about the findings at operation and a discharge summary is issued. Patients are reviewed in the gynaecology clinic after six weeks or earlier when there is an indication. Total abdominal hysterectomy is the commonest cold gynaecological operation.

COMMON GYNAECOLOGICAL OPERATIONS

1. TOTAL ABDOMINAL HYSTERECTOMY

General anaesthesia is induced as described above. Vulvo-vaginal toilet is performed with hibitane solution and the bladder aseptically catheterised. The catheter is left in situ to provide continuous bladder drainage during the operation. Pelvic examination under anaesthesia is done and the findings noted. The vagina is painted with methylene blue. The abdomen is then cleaned with hibitane solution and painted with iodine followed by sterile draping.
The abdomen is then opened in layers as described previously for caesarean section. The most commonly used abdominal incision for this procedure in our unit is the sub-umbilical midline although other incisions such as pfannenstiel and para-median may be used at times. The abdominal and pelvic organs are inspected i.e. the liver, spleen, stomach, omentum, intestines, uterus, fallopian tubes and ovaries, paying particular attention to the pelvic organs notably the uterus, tubes and ovaries which are examined for any pathology, mobility and presence of adhesions. Warm moist packs are then used to pack the intestines away from the operating field.
The uterus is held with a myomectomy screw or a strong stitch (usually No. 2chromic catgut) and if its mobility allows, it is delivered through the abdominal incision. A self-retaining retractor is then placed, opened and fixed to provide better exposure during the operation.
The round ligament of one side is held with two Kocher's (Oschner) clamps near the uterine cornu, divided between the clamps, and the distal portion transfixed with No. 2 chromic catgut. From this incision site, the anterior leaf of the broad ligament is opened and cut in an elliptical fashion towards the utero-vesical peritoneal reflection. Two fingers (the index and middle) of one hand are used to push the posterior leaf of the broad ligament forward and with the other hand an incision is made between the tips of the fingers with a pair of scissors to make a window in the broad ligament. If the tubes and ovaries are to be conserved, curved Kocher's clamps are laced across the tube and ovarian ligament as close to the uterus as possible. A scalpel divides these structures between the clamps and the distal pedicle is secured with a transfixion suture. If the ovary and tube are to be removed, the infundibulopelvic ligaments are doubly clamped with long artery forceps with the tips reaching the open window in the broad ligament, divided

and the distal portion transfixed with No. 2 chromic catgut. The same procedure is repeated on the other side.

Using a swab on a holder or swab around the finger. Blunt dissection is done gently to separate the bladder from the lower uterine segment, cervix uteri and the upper part of the vagina. The ureters are reflected inferiorly by wide mobilization and displacement of the bladder base from the cervix, aided by traction on the uterine corpus.
The uterine vessels are skeletonised and exposed by trimming off the loose areolar tissue over the lateral aspect of the uterus and are then divide between two strong Kocher's clamps and transfixed distally with No. 2 chromic catgut or vicryl. Extending the incision around the tip of the distal clamp enables adequate ligation. Care should be taken to avoid freeing the tissue beyond the tip of the clamp, as this could permit bleeding from 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 cross beneath the uterine artery, lateral to the internal os, and pass medially through the base of the broad ligament to the trigone of the bladder.

The uterus is retracted forwards and upward to demonstrate and stretch the uterosacral ligaments posteriorly. A transverse incision is made through the uterine reflection of the cul-de-sac peritoneum between the attachments of the two utero-sacral ligaments. The cardinal ligaments are then divided between two clamps and transfixed. The peritoneal flap over the cervix posteriorly is dissected away to isolate the uterosacral ligaments, which are clamped, cut and transfixed adjacent to the cervix. The lowest part of the cervix is palpated and the upper end of the vagina just beneath the cervix is held with two littlewood clamps. A stab incision is made with a scalpel between the two clamps opening into the vaginal canal. The incision is then extended circumferentially with a pair of curved scissors and the entire cervix is removed with the uterine corpus. The specimen removed is sent for histology. The vaginal vault is held with strong long artery forceps and starting from the corners, closure is done with No. 2 chromic catgut ensuring that good haemostasis is achieved. The lateral stitches are left long to be incorporated in the peritonisation stitch.

Using No. 1 chromic catgut peritonisation over the vault is done to cover the raw areas by stitching together the reflected anterior and posterior peritoneum, and incorporating the round, infundibulopelvic and uterosacral ligaments. The suture is tied at the midline from both sides.
The abdominal viscera are inspected, packs are removed, instruments and swab counts are confirmed correct, haemostasis is satisfactory, the abdomen closed in layers as described above. General anaesthesia is reversed and postoperative care instituted as described above.

2. MANUAL VACUUM ASPIRATION

This procedure in our unit is indicated mainly for incomplete abortions, missed abortions or termination of early pregnancy on medical grounds. It is indicated for pregnancies of 14 weeks or less. The same procedure is done when endometrial biopsy is needed. It is

performed in the procedure room in ward 1D. The procedure is explained to the patient, she is counseled about what to expect during the procedure and consent obtained. Normally no anaesthesia is given but for anxious patients para cervical block using local anaesthesia is used. Intravenous pethidine can also be given at a dose of 100mg for an average size adult of average build. Usually it can be calculated at 1mg/kg body weight. Resuscitation drugs are kept standby in case resuscitation becomes necessary.

The patient is placed in the lithotomy position, vulvovaginal toilet done and perineum covered with sterile towels. A vaginal examination is done and dilation of the cervix if present noted. The size of the uterus is also noted. Uterine size of up to 14 weeks is preferred for this procedure. The products of conception if palpable or visible are removed manually. A Cusco's speculum is gently introduced into the vagina and the anterior lip of the cervix held with a tenaculum. Depending on the size of cervical opening, a Karman's canula of appropriate size to fit the cervical opening is selected. If the cervix is closed or tight it can be dilated using plastic dilators that are nowadays available. If this fails, the procedure is done in main theatre under general anaesthesia or sedation. The canula is introduced till the fundus is reached. A vacuum is then created within the Karman's syringe that is then connected to the to the canula and the valves released. The products of conception are aspirated into the syringe as it is gently rotated and moved in and out. Aspiration is stopped when no more uterine contents are coming out and a grating sound is heard in the uterus and the canula has bubbles in it with the cervical os gripping on it. The amount, colour and smell of aspirates are noted, if the procedure was meant for biopsy or if the products of conception appear like grapes, they are sent for histology. The canula is withdrawn from the uterine cavity and the cervix and vagina are cleaned. A sanitary pad is placed on the vulva and the patient taken back to the ward for observations.

COUNSELLING CLINICS

There are three such clinics in the hospital, which offer counseling to obstetrics and gynaecology patients. These are the patient support center for HIV pretesting and post-testing counseling, the under 25 years single mother clinic and the Nairobi hospice.

THE PATIENT SUPPORT CENTRE

This is situated in the old hospital building where patients 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

This clinic is situated on the ground floor next to the maternity wards. It deals with young single mothers who have had abortions, those who have delivered unplanned and unwanted babies. The counselors are also trained nurses, sociologists and consultant obstetricians/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 STI 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 home-based care for such patients instead of hospital care. Patients are referred there from the wards or clinics whenever there is an indication for terminal care e.g. advanced cancer of the cervix.. For patients who are hospitalized and very sick to walk out of the ward or to go home, the hospice workers come to the ward and administer the treatment from there.

THE HOSPITAL CHAPEL

This provides spiritual nourishment to those patients who are in need. It is situated on level 2 of the main tower block. The spiritual leaders also see patients in the ward when need arises.

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.

OBSTETRIC CASE 1

DEEP VENOUS THROMBOSIS: LIVE INFANT –SVD

NAME:	A. A.	L.M.P:	19.01.02
AGE:	21 Years	E.D.D:	26.10.02
I.P NO:	0827733	PARITY:	0 + 0 Gravida 1
D.O.A:	6.8.02		
D.O.D:	2.11.02		

PRESENTING COMPLAINT

AA presented to the hospital casualty with complaints of pain and swelling of the left lower limb for six days.

HISTORY OF PRESENTING ILLNESS

She was well until six days prior to admission when she developed pain and swelling of the left lower limb. This started spontaneously, had no preceding trauma and no associated difficulty in breathing. The pain started in the inguinal area. Elevating the limb eased the pain. She had no fever and had never had such an episode before. She went to Pumwani hospital when the illness started and was adviced to seek treatment at the Kenyatta national hospital.

ANTENATAL CARE

She started antenatal clinic from four months gestation in a City Council clinic at Umoja, having made two visits. The antenatal period was uneventful until the above complaints started. Her blood group was A positive, VDRL and HIV tests were negative, and her haemoglobin level was 9.8g/dl. Her last menstrual period was on 19.01.02 and her expected date of delivery on 26.10.02, and she was therefore at a gestation of 28 weeks.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

AA attained menarche at the age at 13 years, her periods were regular occurring every 28 days and lasting for three days. She had no dysmenorrrhoea. Her parity was 0 +0. She had never used any form of contraception previously.

PAST MEDICAL HISTORY

She had no prior history of admissions, any chronic illnesses or allergies.

FAMILY AND SOCIAL HISTORY

AA was a married housewife and lived with her husband in Kayole. She was the eighth born in her family all of whom were alive and well. She had no family history of chronic illnesses. She did not use alcohol or smoke cigarettes.

PHYSICAL EXAMINATION

She was in a good general condition and well nourished. She was not pale, had no jaundice or oedema. Her blood pressure was 110/70mmHg, pulse rate 78 beats/minute and respiratory rate 19-breaths/ minute. She was afebrile with a temperature of 36.70C. Her central nervous, respiratory and cardiovascular systems were normal.

ABDOMINAL EXAMINATION

Her abdomen was uniformly distended with no surgical or therapeutic scars. She had a fundal height of 28 weeks corresponding with her gestation by dates. The foetus was in longitudinal lie with a cephalic presentation. The foetal heart rate was 144 beats/minute and regular.

LOCAL EXAMINATION OF THE LOWER LIMBS

She had a swollen left thigh, measuring 51.5 cm in diameter at a level of 30cm below the anterior superior iliac spine. The corresponding level of the right thigh measured 46cm. The left calf was similarly swollen, measuring 38cm in diameter 10cm below the tibial tuberosity. The corresponding right calf dimension was 33cm at the same level. The thigh was warm, and there was tenderness in the left medial inguinal region. She had no visible injury on the left limb.

IMPRESSION

Deep vein thrombosis of the left femoral vein at 28 weeks gestation.

INVESTIGATIONS

1. Haemogram haemoglobin 10.6g/dl
   white cell count 4.3x109/l
   platelets count 303x109/l

2. Activated partial thromboplastin time index (APTT) after one day of fractionated heparin was 37 seconds for the test and 33 seconds for the control.

3. Venous Doppler done revealed left common femoral vein occlusion with turbulent flow. Compressibility of the vessel was not easily demonstrated. The left common femoral vein was also dilated.

4. Obstetric scan revealed a single intrauterine pregnancy, in cephalic presentation,

with normal cardiac activity at 28 weeks gestation which corresponded with AA's dates. The placenta was fundoposterior.

DIAGNOSIS

Deep vein thrombosis of the left femoral vein at 28 weeks gestation.

MANAGEMENT

She was admitted and started on fractionated heparin infusion 7500 units every six hours until the acute symptoms subsided after which she was weaned to use subcutaneous heparin 7500 units six hourly and later to oral warfarin 7.5mg once daily. Treatment monitoring was done using APTT for heparin and prothrombin time index (PTI).The results of the tests during monitoring are shown below

Date	8/8/02	15/8/02	28/8/02	2/9/02
A.P.T.T.- Test	33	47.7	50	51
-Control	37	34	33	33.8
I.N.R.	1.07			2.1

On the 4th of September 2004, she was discharged home on warfarin treatment, as delivery was still not anticipated immediately. She was to be followed up in the antenatal clinic and be readmitted at 36 weeks gestation. She visited the clinic twice and during both visits had no limb pain or any swelling. She was adviced to continue with the warfarin tablets. She was readmitted on 14/10/04 and converted back to heparin use subcutaneous at a dose of 5000 units three times daily. She was then at 38 weeks gestation. Warfarin tablets were stopped. The APTT was maintained at 1.5 during this time and at 40 weeks gestation she went into spontaneous labour. Heparin injection was stopped and withheld until 12 hours after delivery. Two units of freshly donated blood were cross matched and kept available for her. Protamine sulphate was ordered and kept available in the labour ward for use in case she had excessive bleeding intra- or post-partum. The haematologist was alerted to be on stand-by. Labour was monitored by use of a partogram. At initial examination, she was found to have cephalic presentation, in longitudinal lie and the head was three fifths above the pelvic brim. On pelvic examination she was at 4cm dilation and the cervix was well effaced. Artificial rupture of membranes was done and labour allowed to progress. She progressed well in labour having good contractions and descent of the foetus. She delivered a live male infant who weighed 2950g and scored 7 in one and 8 in five minutes. She lost about 200mls of blood during delivery and did not develop either immediate or late post partum haemorrhage. She had no major trauma to the genital area during labour and delivery. She remained stable during the postpartum period; 12 hours after delivery she was started on heparin 5000 units subcutaneously together with warfarin 5mg once a day. After three days her PTI and INR (International normalized ratio) was tested and found to be acceptable at 2.32 (Test 34 seconds and control 15 seconds, index 44%). She was then allowed to go home on warfarin 5mg once a day for 6 months and to be reviewed in the postnatal and haematology clinics.

FOLLOW UP

She was seen in the haematology clinic after one week and was well. She had no pain in the lower limb and the swelling had resolved. At six weeks postpartum both she and the baby were well. She was adviced to go through the family planning clinic for contraception advice where she chose to use an intrauterine device. She was also adviced to start antenatal care soonest during her next pregnancy in order to receive prophylaxis against developing deep vein thrombosis. She continued with warfarin 5mg daily and did not have any bleeding episodes while on it. Her PTI ratio was done monthly and remained within the acceptable range of 2-2.5. She was reviewed in the haematology clinic monthly until six months after which she was discharged from the clinic.

DISCUSSION

The case presented is of a 21 year old primigravida with deep venous thrombosis (DVT) in pregnancy and managed with heparin and warfarin and delivered vaginally. She was then discharged in stable condition on oral warfarin.

DVT is a vascular occlusive disorder caused by pathological thrombus formation in the blood vessels. A thrombus is a solid mass consisting of platelets, polymorphonuclear cells and fibrin strands with trapped red blood cells.

Out patient presented at the beginning of the third trimester, which is the commonest time of presenting during pregnancy. Antepartum DVT occurs more commonly in the second and third trimester than the first trimester. Antepartum venous thrombosis is more common than the postpartum type[1,2,3]

The risk of venous thromboembolism is five times higher among pregnant women as among non-pregnant of similar age [1]. The incidence of thromboembolism in pregnancy varies from 1 in 1000 to 1 in 2000 according to various studies. At the Kenyatta hospital the incidence of DVT was reported to be 0.16% of all pregnancy admissions [4]. Out of the 80 cases analysed by Waweru, 61% were associated with pregnancy and 76% of these occurred in the left lower limb. Our patient had DVT in the left lower limb. This is thought to be due to compression of the left iliac vein by the right iliac and ovarian arteries, which cross the vein on the left but not on the right side.

This patient had deep vein thrombosis. There are three forms of thromboembolic disease namely superficial thrombophlebitis, deep venous thrombosis and pulmonary embolism [1,2,3]. Thrombophlebitis indicates thrombosis in a vein preceded by inflammation of vessel wall due to trauma, infection or hypertension. Plebothrombosis means coagulation or thrombosis occurring in veins without antecedent inflammation as can occur with an increase in clotting factors and platelets or decrease in fibrinolytic activity. When a solid thrombus is dislodged, embolism occurs. A thrombus formed in the deep veins of the leg can dislodge to the lungs causing pulmonary embolism. Superficial thrombophlebitis is

the most common thrombosis associated with pregnancy usually in varicose veins in the calf. DVT may be a sequel of superficial venous thrombosis.

The patient presented had pregnancy as the main risk factor for having developed DVT. Various factors increase the risk for thrombosis including; pregnancy, age (above 35 years), surgery of the pelvis, immobilization, cancer, fractures, prior pelvic radiation therapy, obesity, varicose veins, leg oedema, diabetes mellitus, hypertensive disorders, heart disease, prolonged labour, operative delivery, postpartum endometritis, combined oral contraceptive use, previous episode of thromboembolism. Blood group A has been related to increased risk of DVT [2,7]. Our patient is blood group A and this may also have contributed. More recently deficiencies of antithrombin III, protein C, protein S, and plasminogen have been found to be predisposing factors as well as presence of factor V-Leiden, a mutation in coagulation factor V (Arg 506-Gln) that results in resistance to activated protein C [1,3,6]. Hyperhomocysteinuria is also associated with the occurrence of venous thrombosis [8].These tests were not done in the patient presented because due to their high costs they are not routinely done in the unit.

Our patient had the proximal type of DVT.DVT can be divided into proximal (iliofemoral) and distal (calf) DVT. Proximal DVT involves popliteal, femoral and iliac veins and forms 80% of cases. Distal DVT involves the calf veins and forms 20% of cases. Proximal vein thrombosis is associated with higher incidences of pulmonary embolism, which occurs in 50% of patients with documented DVT. Proximal extension of calf DVT occurs in 30% of cases [1-3]. The patient presented had proximal DVT but did not get pulmonary embolism. Almost 90% of DVT affects the left lower limb among pregnant women compared with 55% among non-pregnant women [3]. At the KNH, Waweru [4] found DVT was three times common on the left than the right side. This difference may reflect compression of the left iliac vein by the right iliac and ovarian arteries, which cross the vein on the left but not the right side.

The patient presented was admitted with leg swelling, pain and tenderness. She did not develop any signs of pulmonary complications. DVT in pregnancy can present or be associated with low abdominal pain due to periovarian collateral circulation or thrombosis. When coupled with the mild pyrexia and leucocytosis of thromboembolism, this pain can be mistaken for other intra-abdominal disorders such as urinary tract infection or appendicitis [3]. Most patients with DVT are asymptomatic. Classical features include swelling, pain, tenderness, local cyanosis, fever and positive Homan's sign. However three quarters of patients who present with suspected signs and symptoms of DVT actually have non-thrombotic causes of leg pains such as leg trauma, cellulites, obstructive lymphadenopathy, superficial vein thrombosis, postphlebitis syndrome and ruptured Baker's cyst. Clinical evidence of DVT of the legs precedes pulmonary embolisation in only about half of cases. More importantly, nearly 40% of asymptomatic patients with DVT were found to have concomitant pulmonary embolism. Shortness of breadth, chest discomfort, air hunger, tachypnoea or obvious apprehension are signs and symptoms that should alert the obstetrician to a strong likelihood of pulmonary embolism. The most reliable symptom is breathlessness [1,2,3,5].

Since clinical signs are unreliable, confirmatory investigations need to be done. In our patient, Doppler ultrasonography and compression studies confirmed the diagnosis of DVT of the left common femoral vein. The presently available techniques for the objective diagnosis of DVT include contrast venography, non-invasive methods and biochemical assays. Venous ultrasonographic imaging is the most widely used. Proximal veins are compressed under gentle pressure with the ultrasound transducer and the inability to compress the vein indicates presence of DVT. Impedance plethysmography, real time B mode ultrasonography, magnetic resonance imaging (MRI) and computed tomographic (CT) scanning are other tests that can be done. Venography remains the standard for confirmation but has the risk of inducing thromboembolism itself. Radioactive iodine laden fibrinogen scanning and D-dimer assays are also used for diagnosis of DVT. Various D-dimers are formed when cross-linked fibrin contained within a thrombus is proteolysed by plasmin. Assays available include ELISA, latex agglutination and whole blood agglutination tests [1,2,3].

Our patient had heparin infusion during the acute phase of treatment. In addition she was started on bed rest, elevation of the left limb and given analgesics to relieve the pain. Management of DVT can be divided into the acute and chronic (remission) phases of treatment. Management options include anticoagulation, caval filters, fibrinolytic therapy and surgical thrombectomy. The last three therapeutic approaches have been less extensively assessed and are rarely used. Anticoagulation therapy is the treatment of choice for most patients with established venous thromboembolism. The main objective in treatment is to stop further growth of the existing thrombus using anticoagulants, control pain and swelling with analgesics. Ancillary measures are also taken to improve venous return and reduce discomfort to the patient including bed rest in the trendelenburg position, local application of heat and elevation of the limb to reduce oedema. Physiotherapy and mobilization is undertaken after pain subsides.

Heparin is the anticoagulant of choice in pregnancy. It is used in the acute phase and throughout the first and third trimesters. It may be given by several regimes:

- Following a 5,000 i.u bolus (80u/kg), heparin is given by continuous intravenous infusion to a total of 30,000-40,000 i.u per day (15-25u/hour). This is the regime our patient was started on.
- Intermittent intravenous injections at 5000iu every 4 hours or 7500iu every 6 hours.
- Subcutaneous heparin at a dose of 10000iu every 8 hours or 20000iu every 12 hours. The APTT should be maintained at 1.5 above the control value as determined at six hours after the last injection.
- Use of one of the low molecular weight heparins.

Our patient did not develop any bleeding tendencies while on heparin treatment. She received intravenous heparin during the acute phase and was continued on subcutaneous heparin once the acute symptoms subsided.
The action of heparin is immediate through acceleration of the activity of antithrombin III which in turn decreases the activity of factors IX, X and XI. Heparin has a half-life of 60-90 minutes. Its main disadvantages are its cost and can only be given parenterally. It's

never given intramuscularly as it can cause bleeding in the muscles; it's also not given when the platelet count is less than 50000/ml. Heparin does not cross the placenta and is not secreted in breast milk due to its high molecular weight. Its side effects include bleeding, allergic anaphylactic reactions, thrombocytopaenia, transient alopecia, osteoporosis and spontaneous fracture of ribs and vertebrae, fat necrosis at injection sites and the rare but dangerous adrenal haemorrhage and necrosis. It has also been associated with increased foetal loss. Calcium diphosphate and anabolic agents appear to be beneficial. Heparin's anticoagulant adequacy is monitored using the activated partial thromboplastin time (APTT) or the kaolin cephalin coagulation time (KCCT), which should be between 1.5 –2.5 times the control value.

The antidote used in case of heparin overdose is protamine sulphate in a dose of 1mg of protamine sulphate to 100iu of heparin. Our patient did not require use of protamine sulphate during the period she was on heparin. If more than 30 minutes have elapsed after heparin administration, half of this dosage is required. Protamine sulphate itself possesses anticoagulant activity. Because of this, it is considered unsafe to exceed the dose of 100mg over a short period. Intravenous injection of protamine may cause sudden fall in blood pressure, bradycardia, transitory flushing and a feeling of warmth. Protamine is non-antigenic [1,2,3,5].

More recently, low molecular weight heparin (clexane) was released for clinical use. The conventional heparin has a molecular weight of 16000 Dalton while clexane's is 4000 Dalton. Clexane has been shown to be safe, effective for treatment and prophylaxis of proximal vein thrombosis in non-pregnant patients. Its safety in pregnancy has not been well assessed. Its main advantage is the low risk of complication, fewer injections per day, does not require much laboratory monitoring and does not require an antidote. The disadvantage is that it is more expensive [1,2,9]. The low molecular weight heparin was not used for our patient due to its prohibitve cost. She was discharged on warfarin and was reconverted to heparin when she reached term gestation. Its therapeutic levels were monitored using the prothrombin time index.

Warfarin can also be used for anticoagulation in pregnancy. It can be used after the acute phase but should be stopped at 36 weeks and heparin restarted. The action of warfarin starts within three days and it acts through inhibition of vitamin K dependent clotting factors synthesis in the liver. It is given in doses of 5-15mg once daily orally depending on achieving therapeutic levels, which is measured using the prothrombin time that should be 1.5-2.5 times the control value, and the prothrombin time index (PTI) should be 50%. Vitamin K is the antidote in cases of warfarin overdose. It is given in a dose of 5-10mg intravenously but the process of antidote is slow taking up to 72 hours to reach maximum effect. Being teratogenic, warfarin is contraindicated in the first trimester. It causes nasal hypoplasia, alopecia, opthalmological abnormalities, retarded development, urticaria and severe dermatitis apart from haemorrhage. After 36 weeks, warfarin crosses the placenta and may cause bleeding tendency in the foetus especially intracranial haemorrhage. It can also cause abruptio placenta.

If intravenous heparin is introduced at the same time as warfarin, then heparin can be safely discontinued after five days [3].The patient presented was on heparin in the acute phase, warfarin in the chronic phase and was reconverted to heparin after admission after 36 weeks.

During labour, heparin does not cross the placenta and its effects on blood loss at delivery will depend on various factors including the dose, route and last time of administration, the magnitude of incisions and lacerations, the intensity of postpartum myometrial contraction and retraction, and the presence of other coagulation defects. In our patient, heparin was stopped during labour and restarted 12 hours after delivery as she had no major genital trauma at delivery and she was discharged on warfarin, which she used for six months while on, follow up in the haematology clinic. In general, heparin therapy should be stopped during labour and delivery. If the uterus is contracted and there is minimal trauma as a result of the delivery, it can be restarted after several hours. Otherwise a delay of 1-2 days may be prudent. Anticoagulation should be continued throughout the puerperium preferably using the oral anticoagulant warfarin. This should continue up to six weeks and up to three months if disease was extensive [10]. Warfarin has no significant transfer across breastmilk thus is safe to use during lactation. Patients with underlying congenital or acquired thrombophilia will require antenatal prophylaxis, the timing of which depends on the patient's history and thrombophilic disorder [1,2,5].

Several measures can be taken to prevent thromboembolic disease. General measures include; early mobilization in the post-operative and puerperal period, physiotherapy [11] and discontinuation of oral contraceptives and cigarette smoking if a patient is to be admitted for major surgery. Anticoagulants are provided to the high risk group patients such as those to undergo major gynaecologic surgery, those with artificial heart valves, and previous thromboembolic disease. In women with multiple thrombotic events during previous pregnancies antenatal prophylaxis should start at least 4-6 weeks in advance of the gestation at which the previous episode occured. Deep venous thrombosis deterrent stockings may be useful in pregnancy as they prevent distension of veins and hence prevent endothelial damage [1,2,3,5].

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OBSTETRIC SHORT CASE 2

BREECH PRESENTATION AT TERM:ELECTIVE CAESAREAN SECTION-LIVE BABY.

NAME: S.N.

AGE : 24 Years

I.P NO. 0986521

D.O.A. 28/09/04

D.O.D 02/10/04

L.M.P. 27/12/03

E.D.D. 03/10/04

PARITY 0+0

HISTORY OF PRESENTING ILLNESS

She was admitted from the antenatal clinic for elective caesarean section. She had been referred from a clinic in Kayole where they had noted she had breech presentation at 36 weeks gestation. In our clinic this was confirmed on further follow up and she was then counseled and admitted for elective caesarean section.

ANTENATAL HISTORY

Her last menstrual period was on 27/12/03 and her expected date of delivery on 3/10/04 giving a maturity of 39 weeks 3 days at the time of admission. She had attended antenatal clinic from 20 weeks gestation at Kayole and later at Kenyatta National hospital from 36 weeks. Her antenatal profile done was: -

Haemoglobin 10.6g/dl
VDRL -Negative
HIV -Negative
Blood group –B Positive
Urine –normal

The antenatal period was uneventful.

OBSTETRIC AND GYNAECOLOGY HISTORY

This was her first pregnancy. She had attained menarche at 14 years. Her menstrual cycles were regular, occurring every 21 days with flow duration of 3 days in normal amounts. She did not have dysmenorrhoea. She had no prior use of any contraceptives.

PAST MEDICAL HISTORY

This was not significant.

FAMILY AND SOCIAL HISTORY
She was a married housewife who lived in Kayole with her husband. She was educated up to form 4.The husband was a college student. There was no family history of chronic illnesses

SYSTEMIC ENQUIRY
She had no complaints in any other systems.

PHYSICAL EXAMINATION
She was in good general condition, was not pale, not jaundiced, had no lymphadenopathy or pedal oedema.

VITAL SIGNS
Her blood pressure was 110/60 mmHg
Respiratory rate 18/min
Temperature 36.9 C
Pulse rate 82/min

CARDIOPULMONARY AND CENTRAL NERVOUS SYSTEMS
These were essentially normal on examination

ABDOMINAL EXAMINATION
The abdomen was uniformly distended and moved with respiration. There were no surgical scars and the abdomen had striae gravidarum. The fundal height was term, lie was longitudinal and presentation was breech. The foetal heart was 138/min and regular. She had no contractions and no organomegaly was palpable.

VAGINAL EXAMINATION
The external genitalia was grossly normal. The cervix was closed, posterior, thick and about 2cm long. There was normal vaginal discharge.

DIAGNOSIS
Breech presentation at term in a primigravida was made.

MANAGEMENT
She was admitted to the antenatal ward for work up as planned for elective caesarean section. The diagnosis and the planned mode of delivery were explained to her. She gave informed consent for the operation.

INVESTIGATIONS
1. Haemogram: Hb 10.6g/dl
WBC 6.4 x 103/dl
Platelets 264 x 103/mm3

2. Urea and electrolytes

Na+ 137mmol/l
K+ 4.2 mmol/l
Urea 1.6mmol/l
Creatinine 43umol/l

3. Obstetric ultrasonography

Was done a day prior to admission to confirm the diagnosis, revealed a single intrauterine pregnancy in breech presentation .The foetal heart rate was138beats/min. The computed gestation by BPD, AC and FL was 37weeks.The placenta was fundoposterior, the foetal spine. Body were normal, the liquor was adequate.

4. Blood group and cross match.

Blood group was B positive and two units of cross-matched blood were requested.

A theatre list was made and consent obtained. On the eve of operation, the patient was starved from midnight and an intravenous drip with 5% dextrose set up on the morning of the operation. The abdomen was shaved and she was premedicated with atropine 0.6mg intramuscularly. She was taken to labour ward theatre with two units of cross-matched blood.

INTRA OPERATIVE

In theatre, the patient was placed on the operation table and re-examined to confirm the diagnosis. The foetus was in breech presentation with a regular heart rate. Spinal anaesthesia was then given. She was then placed in semi-lithotomy position and vulvo vaginal toilet done. The perineum was draped and aseptic catheterization done. The catheter was left in situ to ensure continuous urine drainage during the operation. She was then repositioned to supine and the abdomen cleaned surgically and draped, then opened via a pfannenstiel incision. The paracolic gutters were packed with wet gauze rolls. Routine low uterine segment caesarean section was then done and a live female infant weighing 3600g with an Apgar score of 8/1 ,9/5,10/10 was delivered by breech extraction. The foetus was in complete breech position and had its cord round the neck. The placenta was delivered whole by controlled cord traction and caesarean section completed as explained earlier in the introduction. The packs were removed and the abdomen closed in layers after all swabs and instruments counts were confirmed correct. Vulvo vaginal toilet together with gentle uterine massage to expel clots was done. The catheter was removed and the urine noted to be clear postoperatively. Estimated blood loss was 500 millilitres.

POSTOPERATIVELY

The patient's vital signs were observed half hourly and patient kept in bed until the effects of spinal anaesthsia were over, and then continued 4 hourly. She was started on oral sips after six hours. She was started on pethidine 100mg 8 hourly for 48 hours, crystalline penicillin 2MU 6hourly and gentamicin 80mg 8 hourly for five days.
On the first postoperative day she was found to be in fair general condition, the breasts were soft non-tender and had started lactating. The abdomen was not distended, uterus well contracted equivalent to 20 weeks gestation, and the bowel sounds were heard and

normal. She was started on light diet and eventually on full diet. On the third day she was started on Ranferon 10mls daily for two weeks. On the fourth day, she was stable with no major complaints, the wound was clean and healing well. She was discharged home and booked to be followed up in the postnatal clinic in six weeks time.

FOLLOW UP.

She came to the clinic and both she and the baby were well. Her wound had healed well and her uterus also was well involuted being non palpable. She was discharged through the family planning clinic. She was adviced to be followed and deliver in a hospital(with facility for c/section) during her next pregnancy due to her having delivered by caesarean section this time. She was assured that this was a non recurring indication and being her first caesarean there was likelihood of having vaginal delivery in subsequent pregnancies.

DISCUSSION

A case is presented of a 24 years old primigravida with breech presentation at term and delivered by elective caesarean section.

Breech presentation occurs when the foetal pelvis or lower extremities (podalic pole) present at the maternal pelvic inlet. It is common remote from term, most often however sometime before the onset of labour the foetus turns spontaneously to a cephalic presentation so that breech presentation persists in only 3-4% of singleton deliveries [1].
There are three types of breech presentations namely

a) **Frank breech**. Constitutes about 65% of all breech presentation. Both legs are extended at knees and the hips are flexed
b) **Footling breech** (incomplete breech) occurs in about 25% of cases. One or both hips are not flexed and one or both feet or knees are lowermost in the birth canal.
c) **Complete breech** occurs in 10% of cases and both thighs are flexed on the abdomen (hip flexion) and both knees are also flexed.

This patient had frank breech presentation. She was noted to have breech presentation when at 36 weeks gestation.The incidence of breech presentation globally ranges from 3-5% at term [1,2]. At the KNH, Njuki found incidence of 3.5% [3]. The incidence varies with gestational age being 3-5% at term, 8-10% at 34 weeks, 25% at 30 weeks and 30-35% at less than 28 weeks gestation.

There was no obvious cause found as a predisposing factor for breech presentation in the patient presented.The actual cause of breech presentation is unknown in over 50% of the cases. Other than gestational age, other factors that appear to predispose to breech presentation include previous breech delivery, multiple gestation, congenital malformations especially hydrocephalous and anencephaly (deformed head cannot fit into the pelvis), congenital hip dislocation, spina bifida, meningomyelocoele, congenital skeletal deformities and chromosomal abnormalities. Maternal factors that predispose to breech presentation include polyhydramnios, oligohydramnios, multiparity, placenta praevia, maternal uterine anomalies such as bicornuate uterus, uterine septa, and fibroids especially in the lower segment, pelvic tumours and contracted pelvis. Finau[4] provided sonographic evidence of a much higher prevalence of placental implantation in the cornual fundal region for breech presentation (73%) more than for vertex presentation (5%).

In our patient, diagnosis of breech presentation was by clinical presentation and confirmation done with an obstetric scan. Diagnosis of breech presentation is mainly clinical with confirmation done by ultrasound. Clinically, the foetal head is felt as a hard, firm and smooth swelling at the fundal region, while the soft breech is found next to the pubis and mostly is not engaged. The foetal heart tones are heard above the umbilicus. If in labour, vaginal examination will reveal the type of presentation. In frank breech, the sacrum with soft tissues is palpable rather than the hard head. With ruptured membranes, the leg or foot in footling breech will be palpable.

The most useful investigation is sonography, which documents the foetal size, attitude, estimates foetal weight, foetal abnormalities, placental localization and estimates liquor volume [2,5,6]. In our patient, a scan done confirmed breech presentation at term and with no notable anomalies. The significance of persistent breech presentation as in the case presented is the increased frequency of complications which include [1,2]:

- Low birth weight from preterm delivery, growth restriction or both.
- Perinatal morbidity and mortality from difficult delivery
- Cord prolapse especially if membranes rupture spontaneously and there is associated footling breech
- Foetal abnormalities
- Placenta praevia with antepartum haemorrhage
- Uterine anomalies and pelvic tumours
- Operative intervention especially caesarean section
- Multiple pregnancy

Our patient did not have any of these complications except that she had an elective delivery by caesarean section. External cephalic version (ECV) was not done as the procedure is not routinely done in our unit due to lack of continuous monitoring facilities during the procedure. The introduction of ECV will help reduce the number of operative deliveries for breech presentation at KNH.
The management of patients with breech presentation is fraught with controversy both in the antenatal period and during labour, with decisions having to be made between vaginal or abdominal delivery. Antenatal follow up is valuable with some patients benefiting from external cephalic version only if strict criteria are used in selection [7, 8].
External cephalic version (ECV) is considered at 37 weeks and above since if done earlier the foetus might revert back to the breech position due to excessive room and liquor in the uterus[9]. ECV should be done only at a hospital with facilities for emergency caesarean section. Tocolytics may or may not be used. ECV should be done preferably near term so that in case of preterm labour resulting, a mature baby is delivered. ECV is done by external rotation of the foetus from breech to cephalic presentation under monitoring either by sonography (preferred) or external foetal monitors. It has a success rate of about 65% and is considered safe. ECV is contraindicated in presence of rhesus incompatibility as it increases the risk of feto-maternal haemorrhage and sensitization. Other contraindications include labour, ruptured membranes, pre-eclampsia, oligohydramnios, placenta praevia or abruptio, multiple gestation and previous uterine scars. Various complications accompany the procedure including abruptio placenta, cord entanglement resulting in foetal distress or demise, rupture of membranes and rarely rupture of the uterus [1,7,8]. ECV is done only when one anticipates successful vaginal delivery.

If vaginal breech delivery is anticipated, x-ray will provide information about the type of breech, presence or absence of flexed foetal head and measurements of the pelvis [10]. Radiation exposure may be reduced considerably by using computerized tomography pelvimetry [11]. The role of x-ray pelvimetry in deciding mode of delivery for breech presentation is controversial. Most authours have found no correlation between radiographic measurements and outcomes of labour. In our patient, x-ray was not done as

she had been scheduled for delivery by caesarean section and x-ray pelvimetry is usually done in selected cases of patients with prior cesarean section scars for radiographic assessment of pelvic cavity adequacy.

In general, the prognosis of the foetus in breech vaginal delivery presentation is considerably worse than when in cephalic presentation [12]. Major contributing factors are; preterm deliveries with low birth weight, increased risk of congenital anomalies (6.3% vs 2.4%), birth trauma (brain, spinal cord, liver, adrenal glands, and spleen), delayed delivery of the aftercoming head leading to hypoxia/acidaemia, forced delivery, cord compression, traction or both.
Caesarean delivery has become a feature of most schools of thought for managing breech presentations. Caesarean section is commonly used in the following circumstances to deliver all but the extremely immature whose potential for survival is negligible;

- Large foetus with a weight of over 3.5kg
- Any degree of contraction of unfavorable shape of the pelvis
- A hyper-extended head. Ultrasound is used to confirm the attitude of the head antepartum.
- Footling breech presentation
- Ruptured membranes especially for more than twelve hours
- Uterine dysfunctions
- Severe foetal growth retardation/restriction
- Request for sterilization
- Previous history of perinatal death or children suffering from birth trauma.

Vaginal delivery of breech presentation needs strict guidelines to avoid morbidity and mortality. Criteria vary from place to place. The prognostic Zatuchni and Andros index is used in most cases to assess the possibility of vaginal breech delivery. It takes into account the following scoring systems and points:

	Add 0 points	Add 1 point	Add 2 points
Parity	0	1	2
Gestational age in weeks	39+	38	Less than 37
Est. foetal weight (kg)	Over 3.6	3.2-3.6	Less than 3.2
Previous breech delivery	0	1	2
Dilatation	2	3	4
Station	-3	-2	-1

If the score is 0-4, caesarean section is recommended [3].

This patient scored 1; parity 0[score 0], gestational age of 38 weeks [score 1], estimated foetal weight of 3.6kg [score 0], station at –3 [score 0] and was delivered by caesarean section. A paediatrician was available to assess the baby at delivery.

It is essential that the delivery team should include an obstetrician skilled in the art of vaginal breech delivery, an associate to assist with the delivery, a paediatrician trained to resuscitate the infant including tracheal intubation and an anaesthesiologist who can ensure adequate analgesia/anaesthesia when needed.

In our unit (KNH), breech delivery is preferably by caesarean section but vaginal delivery is allowed in cases of very advanced labour (2^{nd} stage of labour), documented lethal foetal anomalies, and pre-viable foetus. Presentation of the mother in advanced labour with no foetal or maternal distress, even if caesarean section were originally planned, would necessitate a carefully performed controlled vaginal delivery, which is safer in such cases than a hastily executed caesarean section.

Complications of vaginal breech delivery that increase neonatal morbidity and mortality are due to birth trauma and asphyxia, which are related to cord compression and the difficulty of delivering of the aftercoming head. Hyper extension of the neck is associated with a high incidence of spinal cord transection if delivered vaginally. Other complications associated with breech presentation include; cord prolapse, cord compression, tear of tentorium cerebellae, intracranial haemorrhage, hip joint dislocations, cervical nerve plexus injuries, fractures of foetal humerus/femur/clavicle, maternal cervical tears including vesicovaginal fistula [2,5,12].

Presence of other obstetric risk factors such as primigravidity, contracted pelvis or borderline pelvis, large foetus, footling breech, extended foetal head and congenital malformations such as hydrocephalous and conjoint twins make both elective and emergency caesarean section necessary. Koo [13] found that even with strict selection of patients, vaginal delivery of breech presentation was associated with increased neonatal morbidity compared to elective caesarean section. Njuki [3] at the KNH found a perinatal mortality of breech deliveries as 5.6 per 1000 live babies. Babies with birth weight less than 1500g had mortality rate of 100% irrespective of the mode of delivery. Mati [14] et al noted that mortality associated with vaginal breech delivery was 9.2 times that associated with vertex delivery, 3.5 times that associated with caesarean section and 3.8 times that associated with operative vaginal delivery.

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OBSTETRIC CASE 3

UNSENSITISED RHESUS NEGATIVE MOTHER- VAGINAL DELIVERY, RETAINED PLACENTA-MANUAL REMOVAL

NAME: N.G.K.

AGE: 34 Years

I.P NO: 0992577

D.O.A: 16/11/04

D.O.D: 19/11/04

L.M.P: 16/2/04

E.D.D: 23/11/04

PARITY 0 + 2

PRESENTING COMPLAINT

NGK presented to labour ward with complaints of low abdominal pain that was intermittent for one day.

HISTORY OF PRESENTING COMPLAINT

The pain was increasing in intensity, radiated to the back and accompanied by vaginal discharge that was mucoid. She had no vaginal bleeding.

ANTENATAL CARE

She attended antenatal clinic at KNH from 27 weeks gestation. Her blood group was B rhesus negative. Her indirect coomb's test done at 28 weeks gestation was negative. Other profile done showed a haemoglobin level of 14.0 g/dl, her VDRL and HIV tests were negative. Her partner's blood group was A rhesus positive. Her last menstrual period was on 16/2/04 and her expected date of delivery on 23/11/04, she was thus at 39 weeks gestation.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She attained menarche at 13 years. Her periods were regular and occurred every 21-24 days and lasting 4 days. She did not experience dysmenorrhoea. Her parity was 0+ 2. All pregnancies were of the same paternity. In 1998 she had an induced abortion at about six weeks of gestation. In 2004 January she had a spontaneous abortion at about 10 weeks gestation and evacuation was done at the KNH. Anti-D was not given post evacuation. She had not used any contraceptives previously.

PAST MEDICAL HISTORY

She had had laparotomy in 1975 for intestinal obstruction and in 1980 for appendicitis. She had no other chronic illnesses, no known allergies and was currently not on any medication. She had never had any blood transfusions

FAMILY AND SOCIAL HISTORY

She was a single lady who lived with her sister in Umoja. She was the fifth born in a family of seven siblings. Her father had diabetes mellitus; she neither smoked cigarettes nor drank alcohol. She worked as a computer operator with the Kenya medical research institute.

PHYSICAL EXAMINATION

She was in a good general condition, not pale, had no jaundice and no lymphadenopathy. Her vital signs were within normal. Her blood pressure was 110/60mmHg, respiratory rate 22 breaths/minute, and pulse 79 beats/minute. Her respiratory and cardiovascular systems were normal.

ABDOMINAL EXAMINATION

Her abdomen was uniformly distended and had a right paramedian scar. She had a fundal height that was at term. The foetus was in longitudinal lie with a cephalic presentation. The presenting part was four fifths up. She had mild contractions. The foetal heart tone was heard and regular at 140 beats/minute.

PELVIC EXAMINATION

She had normal external genitalia. The cervix was soft, central and 3 cm dilated. The membranes had ruptured and she was draining clear liquor.

INVESTIGATIONS

1.Indirect Coomb's test at 28 weeks was negative.

2. Obstetric scan done at 33 weeks gestation showed a single intrauterine pregnancy at 31 weeks and four days with a biophysical profile of 8/8. The placenta was fundal and posterior. The amount of liquor was adequate.

DIAGNOSIS

Rhesus negative mother in labour at term.

MANAGEMENT

Artificial rupture of membranes was done and she was started on an oxytocin drip. Labour progressed well, was monitored by means of a partograph and she delivered a live female infant who weighed 3100g and had an Apgar score of 10 in one and 10 in five minutes. The duration of labour was about 8 hours. At the time of delivery umbilical cord blood was taken to confirm the baby's blood group and rhesus factor, direct coomb's test and the haemoglobin level. The baby was taken to the newborn unit for observation. She developed a retained placenta during third stage. The placenta had not separated ten minutes after delivery. The bladder was emptied by catheterization and controlled cord traction was attempted to try and separate the placenta. As this was not successful, she was also started on oxytocin 20 units in 500mls normal saline and a blood sample for grouping and cross matching taken as precaution in case blood for transfusion was necessary.Half an hour after delivery the placenta had still not been delivered and a diagnosis of retained placenta was made. She was informed of this development and the need for manual removal of the placenta under anaesthesia, for which she consented after being counseled. The oxytocin drip was continued and one unit of blood cross matched. In theatre, anaesthesia was induced and she was placed in lithotomy position. After sterile draping of the perineum and lower abdomen, one hand was inserted into the vagina along the cord and into the uterus. The other hand was used to support the uterus and provide counter traction during removal of the placenta. The hand in the uterus was moved to locate the edge of the placenta and a cleavage identified between the placenta and the uterine wall. From this cleavage point, the placenta was detached from its implantation using the edge of the hand until the whole placenta was detached from the uterine wall. The placenta was then held in the hand and the hand then slowly withdrawn from the uterus while the other hand on the abdomen was used to provide counter traction by pushing the uterine fundus upwards above the pubic symphysis. The inside of the uterine cavity was explored to ensure that all placental tissue had been removed. The uterine surface of the placenta was examined and found to be complete. The uterus was massaged and the oxytocin drip continued at 60 drops per minute. No cervical, vaginal or perineal tears were noted and anaethesia was reversed. Post operatively, vital signs were monitored every half hour in the first two hours, then every four hourly. They remained normal. She was monitored for vaginal bleeding which was not excessive. She was started on metronidazole 400mg and amoxicillin 500mg 8 hourly for five days. The baby's result indicated the blood group as B positive and the direct coombs test was negative. The haemoglobin level was 19g/dl. The baby was discharged from the newborn unit.

NGK was then given 300ug of anti-D immunoglobulin 24 hours after delivery. Both mother and baby were well and were discharged home and were to be reviewed in the postnatal clinic in six weeks time.

POSTNATAL FOLLOW-UP

She came for the postnatal clinic and had no complaints. The uterus was not palpable. The baby was well and growing well. She was discharged to the family planning clinic and also adviced to have an indirect coombs test done.

DISCUSSION

The patient presented is a rhesus negative Para 0+2 who was admitted in labour and had a spontaneous vertex delivery and was given anti-D because her baby was rhesus positive. There was no evidence of sensitization in the mother antenatally and the baby had no features of haemolytic disease of the newborn (HDN). She also developed a retaned placenta and manual removal had to be done under aanaesthesia.

HDN is a condition in which the life span of the foetal or newborn infants red blood cells is shortened by the action of maternal antibody against antigens present in the placenta and enter the foetal circulation. Red cell destruction begins in the uterus and may lead to severe anaemia and even hydrops foetalis or death in utero. Individuals who lack specific red cell antigens can produce an antibody when exposed to that antigen. The vast majority of humans have at least one such factor inherited from their fathers and lacking in the mothers.

The mother presented was D-rhesus negative.In the red blood cells there are about 250 recognised antigenic factors of which the most common are ABO, Rhesus, Kell, Lutheran, Duffy, Kidd, P and MNS, Diego, Lewis [1,2,3]. The Rhesus factor was discovered by Landsteiner and Weiner in 1940.They are antigens confined to the red cell membrane and are lipoproteins coded for by 3 pairs of allelic genes on the short arm of chromosome 1. They are denoted D/d, C/c, and E/e. Inheritance follows Mendelian principles independent of other blood group antigens. D is the most powerful and important of these antigens and those with D antigens are said to be rhesus positive. 45% of rhesus positive people are homozygous for D while 55% are heterozygous for D [1-4].

There are considerable variations in the distribution of the rhesus blood blood group. The Basque population has the highest incidence of rhesus negativity at 30-35%. Among Caucasians, 15-16 % are negative, American blacks 7-8%, African blacks about 4%. In the mongoloid community the incidence of rhesus negative is nil. Mulandi found a prevalence of 4.1% among ANC mothers at KNH [2,3,6].

Our patient was not isoimmunised as anindirect coombs test at 28 weeks gestation was negative.Rhesus isoimmunisation occurs when an individual lacking the antigens is exposed to massive and/or prolonged contact with rhesus positive blood and develops antibodies against the rhesus antigens [3,4]. This may occur when a woman is pregnant with a rhesus positive foetus. It is now extremely rare for isoimmunisation by a rhesus D positive foetus [3,4]. In normal circumstances foetal blood cells enter the maternal circulation but in small numbers, which are destroyed by the maternal immune system before provoking an antibody reaction especially when the ABO blood group of the mother and foetus are incompatible. In these cases the mother would be sensitized if enough foetal red cells were to reach her circulation to elicit an immune response. This however does not occur as often as one would expect because of several factors. These include;[1-4]

1. Maternal inborn ability to respond to the rhesus antigenic stimulus. About two-thirds of rhesus negative women are responsive and one third are non-responders.
2. Protection from isoimmunisation by the ABO incompatibility of the foetus and mother. This reduces the incidence of rhesus isoimmunisation to about one tenth of that when they are ABO- compatible.
3. Variable antigenicity of the rhesus negative antigenic stimulus, depending on the rhesus group of the foetal blood e.g. the Cde/cde genotype seems to be relatively "strong."
4. Volume of foetal blood entering the maternal circulation whereby 0.25 mls represents a critical sensitizing volume and with the likelihood of severity of sensitization increasing with greater volumes.
5. Lack of transfer of antibody across the placenta from the mother to the foetus in sufficient amounts to affect the foetus

The patient presented had manual removal of the placenta thus had a higher likelihood of transplacental bleeding. A Kleihauer test was not done to assess and estimate fetomaternal haemorrhage if it occurred.
Chow in 1954 was the first to report fetomaternal haemorrhage [1]. Silent fetomaternal haemorrhage and foetal red blood cells are present in maternal circulation in 3% of women during the third trimester. Haemorrhage may occur in cases such as antepartum haemorrhage, cephalic version, pre-eclampsia, trauma, amniocentesis, chorionic villous sampling, abortion and termination of pregnancy. During labour and during the third stage, transpacental bleeding occurs and is more likely following manual removal of placenta, caesarean section and in multiple pregnancies [3,5].
The amount of foetal blood in maternal circulation is estimated using the acid denaturation test introduced by Kleihauer-Betke in 1957. Other tests are the erythrocyte rosette test, immunofluorescence tests and assays of maternal serum alpha-fetoprotein [3,4,5]. Our patient was not isoimmunised. She had manual removal of the placenta but Kleihauer test was not done as it is not usually done in our hospital.

This patient did not develop any features of hydrops foetalis during the pregnancy. Hippocrates was the first to describe the birth of a hydropic foetus in 400 BC and in 1641 it was also observed by Plater. Diamond et al in 1932 linked together the triad of hydrops foetalis, icterus gravis and neonatal anemia as rising from the single process of erythroblastosis foetalis while Levine et al in 1941 provided the final serological proof that this was due to rhesus isoimmunisation with paternally inherited foetal factors [1,4].

A foetus receives half its genetic component from its father and half from its mother. As in the case presented a rhesus negative mother can carry a rhesus positive baby if the father is rhesus positive [1,4]. If enough foetal red cells leak into the maternal blood, a maternal antibody response will be provoked [5]. The initial response is with a low level of IgM antibodies but within six weeks to six months IgG antibodies become detectable. IgG is capable of crossing the placenta in a subsequent pregnancy, enter foetal circulation and attach to antigenic sites on the surface of foetal red cells. These cells are destroyed in the reticuloendothelial system with release of bilirubin and haeme and resultant anaemia in the foetus. In utero bilirubin appears in amniotic fluid and is an important marker [1,3].

The foetus compensates by increased extramedullary erythropoiesis in the bone marrow, spleen and liver resulting in hepatosplenomegaly. There is compensatory placental hyperplasia to increase oxygen transfer. Excessive anaemia leads to congestive cardiac failure, subcutaneous oedema, ascites, pleural and pericardial effusion and the resultant picture of hydrops foetalis. After delivery bilirubin crosses the blood brain barrier and may get deposited in the basal ganglia to cause kernicterus [1,3,4,5].However while the foetus is in utero, the placenta effectively removes haeme and bilirubin and the mother metabolises them. The patient presented was not sensitized and did not develop these complications.

The overall risk of isoimmunisation for a rhesus positive ABO compatible infant with a rhesus negative mother is 16%. Of these, 1.5-2% of the isoimmunisation will occur intrapartum,7% within 6 months of delivery and the remaining 7% early in the next pregnancy most likely as a result of amnestic response. ABO incompatibility is protective and the overall risk is only 1.5-2% because cells are rapidly destroyed before eliciting antigenic response [3,4]. The first pregnancy of a rhesus negative mother is almost invariably unaffected if she has no previous sensitization. Occasionally there is sensitization through maternal foetal transfusion during her own birth if her mother was rhesus positive-the 'grandmother theory'. After delivery antibodies are detectable in about 8% of at risk mothers 6 months after delivery [2,3].By the end of the second delivery with a rhesus positive baby 17% of rhesus negative mothers will have detectable antibodies. Most will have resulted from immunization by the second pregnancy [1-3]. When antibodies become detectable in a first pregnancy it probably means that the mother is a strong responder in whom the foetus can become so severely affected that it is at risk of intrauterine foetal death [2,3]. Once a woman has developed rhesus antibodies they will become detectable from an early stage in a future pregnancy not only if the foetus is rhesus positive, but also if rhesus negative. This is the amnestic response [3,4,5].

In the case presented the mother was blood group B negative and the partner was A positive. ICT was negative at 28 weeks. ICT was not repeated as she could not afford the charges for the test.
All pregnant women should be screened for the ABO blood group and for the rhesus factor. If found to be rhesus negative, the father's blood group should also be checked. If negative, there is no need for further worry. If the father is rhesus positive, antibody screening mostly with indirect coomb's test (ICT) should be done. Even when antibodies are not found on initial screening, ICT should be repeated at 4-6 weeks intervals because they may develop later. Often this is done at 20-24 weeks, then at 28,34 and 38 weeks gestation [1,2,3,5].

In the case presented, the baby's group was B rhesus positive, DCT was negative, total bilirubin was 15.9mmol/l and haemoglobin was 19g/dl. Management of labour should be by a joint team of obstetrician and paediatrician. The patients are allowed to go to term and spontaneous labour awaited. Induction of labour is done selectively. In case of caesarean delivery there should be adequate packing and lavage of the peritoneal cavity. Immediately after expulsion of the foetus, 10mls of cord blood should be taken and

analysed for blood group, rhesus factor, direct coomb's test (DCT), haemoglobin and bilirubin levels [2,4,5]. Our patient had normal vaginal delivery.

After delivery, our patient was given a dose of 300ug of anti-D immunoglobulin intramuscularly.Passive immunization by intramuscular (IM) injection of anti-D antibodies has dramatically decreased the risk of isoimmunisation but not eliminated it completely. Use of anti-D was started by Finn in the U.K in 1961 and later Freda et al in the U.S.A in 1963 1,4. 300ug (500 i.u) of anti-D given by deep I.M injection within 72 hours of delivery will prevent isoimmunisation provided the mother has not already been immunized. The dose is enough to neutralize 4mls of foetal blood [1,4]. Some authours recommend that it can be given up to within 28 days postpartum. A similar dose should be given in case of antepartum haemorrhage, external cephalic version, amniocentesis, any intrauterine procedure and following blood transfusion. Half standard dose is given if these events occur before 20 weeks gestation or in case of abortion because fetomaternal haemorrhage is less than 20mls. [1,4,5].

In our patient, anti-D was given 24 hours after delivery.
Silent antepartum haemorrhage is frequent after 28 weeks. Immunisation may be prevented by giving anti-D at 28 weeks. A single dose given at 28 to 32 weeks may be adequate and is considered protective for about 12 weeks [1,2]. Other doses are usually not given in our unit due to cost.

Ultrasonography (U/S) when done early will give foetal measurements that will confirm dates or help adjust them in order to time delivery. The U/S aids biophysical profile, detects features of hydrops foetalis and guides amniocentesis and transfusion [1,2,3]. Our patient had U/S that confirmed the foetus was normal.

Patients who are sensitized need close follow up. Amniocentesis is done every 3-4 weeks under U/S guidance from 28-30 weeks (not before 22weeks). The fluid is analysed to determine optimal time for intervention. Bevis in 1956 showed a correlation between the severity of erythroblastosis foetalis and the amount of bilirubin in the amniotic fluid [1-5]. Liley in 1964 deviced use of spectrophotometry by measurement of the optical density at the peak wavelength of 450nm and developed a graph with three zones applicable after the second trimester. Based on the 3 zones a decision is made either to await labour, perform intrauterine transfusion or delivery [4-7]. In our patient ICT and subsequent U/S were normal and there was no need for amniocentesis.

The patient presented also developed a retained placenta, which increases the risk of fetomaternal haemorrhage especially when manual removal is done like in her case. A kleihauer test should be done to confirm the amount of fetomaternal haemorrhage and for adequate amount of Anti-D to be given. Failure of the placenta to deliver spontaneously is an important cause of postpartum haemorrhage[8]. Our patient had a loss of 300ml, which was not high. An oxytocin drip had been instituted. Manual removal is undertaken when spontaneous expulsion does not occur. After removal, the maternal and foetal sides are inspected to ensure the entire placenta has been removed and an oxytocin drip is administered to promote uterine contraction. This was done in the patient presented. She

was then covered with antibiotics as endometritis can follow manual removal of the placenta.
Counselling and contraception are an integral part of management. The patient should be adviced about the risks of future pregnancies [5]. Exact statistical prognosis is difficult but there is 4% mortality if no previous baby was affected and 6% when at least one was affected but had no transfusion, 10% when at least one needed exchange transfusion and 50% if at least one baby died of HDN [5].

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OBSTETRIC CASE 4

CARDIAC DISEASE GRADE II IN PREGNANCY-LIVE TERM DELIVERY

NAME:	F.A.H	L.MP.	13/02/04
AGE:	24 Years	E.D.D.	20/11/04
I.P NO:	0993582	Parity.	1 +0 Gravida 2
D.O.A:	14/11/04		
D.O.D:	28/11/04		

PRESENTING COMPLAINTS

She was admitted with 3-day history of dyspnoea on moderate exertion, easy fatiguability and lower limb swelling. She also had labour like pains for four hours.

HISTORY OF PRESENTING COMPLAINTS

She was referred from the Dadaab refugee camp where she resides after having complained of easy fatiguability, lower limb swelling and difficulty in breathing for three days prior to admission. She was examined and suspectedd to have cardiac disease and was at term gestation. She was airlifted to our unit due to inadequate facilities in that camp. The dyspnoea was worsened by moderate exertion beyond her normal chore. She would get tired on household chores. She also noted that her limbs were swelling at the ankles in the three days and she was having difficulty in breathing mainly on exertion but was relieved by rest. She had no known previous history of cardiac disease though she gave history of previous episodes of dyspnoea on exertion before the index pregnancy. She had been started on frusemide 40mg and digoxin 0.25mg daily at Dadaab and the symptoms had subsided by the time she was admitted at KNH three days later. By the time of admission she also had labour like pains for four hours. She did not have any vaginal bleeding and had not drained liquor. She also gave history of palpitations. She gave no history of antecedent febrile illness, cough or chest pain.

ANTENATAL CARE

She had attended the antenatal clinic at the refugee camp she lived in (GTZ clinic), having had two visits. A pansystolic murmur had been noted but she was asymptomatic before the above comlpaints. Only a haemoglobin test had been done which was 9.7g/dl. Other antenatal profile had not been done.

OBSTETRIC AND GYNAECOLOGY HISTORY

She had her menarche at 15 years. Her menstrual cycles were regular occurring every 27-28 days, with duration of 3-4 days and normal flow. She had no associated dysmenorrhoea. Her last menstrual period was on 13/2/04 and expected date of delivery on 20/11/04 though she was not very sure on the first day of her last period. She was thus at 39 weeks and 2 days gestation at the time of admission.
She had her first delivery in 2003 to a male infant who is alive and well. She had not used any contraceptive previously.

PAST MEDICAL HISTORY

She had no other chronic illnesses. She had never been admitted to hospital previously. She had no food or drug allergies.

FAMILY AND SOCIAL HISTORY

She was married and lived with her family at the Dadaab refugee camp after fleeing war in her home country of Somalia. She had no family history of chronic illnesses. She did not use alcohol or smoke cigarettes.

PHYSICAL EXAMINATION

She was a young lady in fair general condition, she was mildly pale, had no jaundice, no oedema, no finger clubbing or cyanosis, no lymphadenopathy. Her blood pressure was 133/89mmHg, pulse rate 90 beats/minute, respiratory rate 24/minute and temperature of 36.2°C. Her nervous system was normal.

CARDIOVASCULAR SYSTEM

Her pulse was 90/minute, it was regular and non-collapsing. The blood pressure was 133/89 mmHg. The jugular venous pressure was not elevated. The praecordium was not hyperactive but there was a palpable thrill. The apex beat was in the seventh intercostal space left of the midclavicular line. Both heart sounds were heard and normal. She had a pansystolic murmur at the mitral area.

RESPIRATORY SYSTEM

Her respiratory rate was 24 breadths/minute and had mild distress. There was equal air entry bilaterally, no crepitations or rhonchi.

ABDOMINAL EXAMINATION

The abdomen was uniformly distended. The fundal height was term, with longitudinal lie, cephalic presentation and only three-fifths of the presenting part were palpable above the pelvic brim. The foetal heart rate was 140/minute and regular. She was having two moderate contractions every ten minutes each lasting about 30seconds. The liver and spleen were not palpable.

PELVIC EXAMINATION

She had had clitoridectomy done. The cervix was 3cm dilated, central and partially effaced. The pelvis was adequate, the membranes were flat and she had show discharge.

DIAGNOSIS

Cardiac disease grade II in early labour.

MANAGEMENT

She was admitted into the labour ward acute room for labour monitoring and observations. She was put in the semi-fowler position, given oxygen by mask as necessary and started on parenteral antibiotics (crystalline penicillin 2MU 6 hourly and gentamicin 80mg 8 hourly). Labour was to be monitored with use of the partogram. She was also given intramuscular morphine 15mg stat for analgesia and to allay anxiety. Resuscitation drugs frusemide, aminophylline, digoxin, sodium bicarbonate were availed and kept ready for use if needed. The mode of delivery was anticipated to be assisted vaginal delivery with vacuum extraction.

The cardiologist consulted confirmed the above findings and adviced to continue with lasix 80mg daily for three days then 40mg and digoxin 0.25mg daily.
She was reviewed after four hours and found to be having 3 strong contractions every ten minutes; only two-fifths of presenting part was above the pelvic brim. Pelvic examination revealed she was 7cm, artificial rupture of membranes yielded clear liquor and she had no caput or moulding. She was given buscopan 40mg and was to be reviewed in 3 hours. Preparations for assisted vacuum delivery were done and intravenous frusemide and oxytocin kept ready. About two hours later, she reported a feeling of bearing down and it was confirmed on vaginal examination that she was fully dilated. She was taken to the delivery room and strongly adviced not to bear down or use any effort. She was kept in the propped up position and oxygen by mask continued. The perineum was draped and her bladder emptied. The head was a fifth up. A size six cup was placed on the vertex taking care not to include maternal tissue.This was ensured by running a finger around the cap when it was in place. She was given a mediolateral episiotomy and had assisted second stage by vacuum extraction as described in the introduction and successfully delivered a live female infant weighing 3kg and scored 8/1 and 10/5 minutes. She was given 80mg lasix and the placenta and membranes delivered by controlled cord traction. A drip of 10 IU oxytocin in 5% dextrose was set to run slowly, the uterus was massaged. The episiotomy was repaired under local anaesthesia and the. She did not develop postpartum haemorrhage. Her immediate postpartum observations were-blood pressure 119/71mmHg, respiratory rate 21/minute, pulse 91/minute.

She remained in the labour ward for 48 hours on observation after which she was transferred to the post-natal ward. While in the ward investigations were done to confirm the cardiac lesion she had.

INVESTIGATIONS

1. Haemogram – haemoglobin 9g/dl, white cell counts 12 $x10^9/l$
2. Chest x-ray- cardiomegaly. Both lung fields were clear.
3. VDRL- Negative
4. Blood group- O positive
5. Urea and electrolytes- Na+ 140mmol/l, K+ 3.5mmol/l, Urea 3.6mmol/l.
6. Urinalysis- protein nil, sugar nil, no growth on culture.
7. Echocardiography- mitral valve prolapse/regurgitation. Large left ventricle and left atrial activity. Prolapsed anterior mitral valve leaflet.
8. Electrocardiogram- Features of left ventricular hypertrophy and left atrial hypertrophy. Left axis shift. Tachycardia.

Diagnosis: mitral valve regurgitation

FOLLOW-UP

She was continued on the antibiotics for a total of 14 days. She was also put on haematinics (ranferon 10mls daily) and was continued on the lasix and digoxin as adviced by the cardiologist. Repeat haemogram showed haemoglobin of 10g/dl, white cell count $10x10^9/l$. After two weeks of antibiotics, she was still stable and the baby was well. Her temperature, pulse, blood pressure remained within normal limits. They were discharged home. She was to continue with lasix and digoxin and was for follow-up in the cardiology clinics. She was adviced to be referred to our hospital early in her next pregnancy for close monitoring to avoid coming late as had happened in this pregnancy. She was given a summary for follow up by the medical officers at the camp where she stayed and for referral to KNH whenever necessary.

DISCUSSION

The case presented is that of a 24 year old para 1+0 who presented to our unit with cardiac disease in early labour and proceeded to have a normal vertex vaginal delivery with vacuum assistance. The outcome was a live female infant with a good score. Both mother and baby did well post-natally and were discharged home in good condition.

She presented while in early labour as a referral from a refugge camp.Cardiac disease is a leading cause of indirect maternal mortality during pregnancy [1]. The marked haemodynamic changes during pregnancy have a profound effect on underlying heart disease. The resting pulse rate increases by about 10-15 beats per minute. The plasma volume increases by 40% and overall the cardiac output increase is due to increase in stroke volume. Additional workload on the heart starts around the 8th week and reaches a maximum between 28-32 weeks and is then maintained to term. It also increases during labour with each uterine contraction. The burden though reduced may persist for the first ten days of puerperium [2,4,5]. Ideally, the patient with known cardiac disease should consult her physician before becoming pregnant. This helps determine advisability and optimum timing for pregnancy and diagnostic procedures, prospect for corrective or palliative cardiac surgery and need for discontinuing certain medications during pregnancy. If the woman presents when already pregnant, as is often the case, the obstetrician must be able to recognize the presence of pre-existing cardiac disease, assess the degree of disability, and understand the impact of the added haemodynamic changes of pregnancy. Our patient presented at term gestation and with symptoms of cardiac disease. Confirmation of the lesions was actually done postpartum.

Our patient was found to have rheumatic heart disease after evaluation. Cardiac disease complicates about 1-2% of all pregnancies with maternal mortality of about 0.5/10000 deliveries. In Kenya, the incidence is about 0.2-0.66%[3].Rheumatic heart disease has historically been the most common type of heart disease in pregnant women but is currently rare in the developed world due to the increased use of antibiotics, declined virulence of strains of streptococcal infection and improved living standards [2,4]. In Kenya, rheumatic heart disease accounts for about 84% of heart disease in pregnancy. However, with improving medical services and advancement in cardiac surgery techniques, some women with congenital heart disease will soon be able to live long enough and conceive leading to successful term pregnancies as in the developed world.

The presented patient had mitral regurgitation and mitral prolapse.Mitral stenosis is the commonest lesion accounting for up to 90% of all cases of cardiac diseases [1,4,5]. Bhatt in 1978 found that the majority of his patients had combination of mitral regurgitation and mitral stenosis.

Our patient was found to be having functional class II cardiac disease.The likelihood of a favourable outcome for the mother with heart disease depends upon the functional capacity of the heart, the likelihood of other complications that further increase the cardiac load and the quality of medical care provided. The degree of functional disability is graded according to the New York Heart Association (NYHA) [7] classification. The

grading is clinical and depends on cardiac response to physical activity with no relationship to the extent of the heart lesion. The NYHA includes the following;

Class (Grade) I No symptoms on ordinary physical activity. Patient with cardiac disease no symptoms of cardiac insufficiency or experience any angina pains.

Class (Grade) II Slight limitation with mild to moderate activity but asymptomatic at rest. They are comfortable at rest but with activity, discomfort results in form of excessive fatigue, palpitation, dyspnoea or anginal pain.

Class (Grade) III Marked limitation with less than ordinary activity. Dyspnoea or pain on minimal activity.. These women are comfortable at rest, but less than ordinary activity causes discomfort in the form of excessive fatigue, palpitation and dyspnoea or anginal pain.

Class (Grade) IV Symptomatic at rest or minimal activity and symptoms of frank heart failure. They have inability to perform any physical activity without discomfort. Symptoms of cardiac insufficiency or angina may develop even at rest and if any physical activity is undertaken, discomfort is increased.
Patients with prosthetic valves, some cases of mitral stenosis, those with prior cardiac surgery, atrial fibrillations belong to class IV irrespective of clinical state.

The patient presented was in class II as she had dyspnoea on exertion.

The management calls for team approach involving the obstetrician, cardiologist and anaesthetist. The patient's cardiac grade should be decided during the first four months of pregnancy if possible and grading repeated again between 28-32 weeks and managed accordingly. The aim of management is to prevent cardiac failure. Proper antenatal, intrapartum and puerperium follow up is essential. Activity depends on her tolerance. Ample rest should be adviced (10 hours of sleep everyday and half hour rest after every meal) and exertion should not be such as to cause symptoms. Infections should be treated promptly and any tooth extractions covered with antibiotics. Patients should avoid alcohol and cigarette smoking is prohibited. They are adviced to see their doctor should they experience dyspnoea or cough. Pneumococcal and influenza vaccines are recommended in the west but we do not give it routinely in our unit. All these were not possible in our patient as she presented late to our unit.

Grade I and II patients are managed as out patients after clinical evaluation. Both cardiologists and obstetrician should see them frequently as their grades may change to higher ones as pregnancy progresses and/or may present with complications. At 36 weeks they are admitted into the ward to await delivery [8]. Infections must be avoided and treated aggressively when they occur. Rest is adviced for mild respiratory tract infections that may trigger cardiac failure [2]. Excessive weight gain is discouraged. Anaemia should be sought for and treated vigorously with haematinics and/or transfusion.

Grade III patients should be hospitalized if necessary throughout pregnancy and those with grade IV must be hospitalized with total bed rest. Restriction of maternal physical activity tends to avoid cardiovascular compromise and improves uteroplacental perfusion [10]. The supine position should be avoided and the patient should be propped up in the cardiac beds. Our patient was managed while in a propped up position. Pre-eclamptic toxaemia should be taken more seriously than usual because of the increased burden on the heart by hypertension and the resultant fluid retention.

Our patient was given intravenous frusemide at the time of delivery. Other drugs such as digoxin were available in case their use was necessary.Digitalis and diuretics may be necessary to prevent and treat congestive cardiac failure and atrial arrhythmia. Those with atrial fibrillation and prosthetic valves should be given anticoagulant therapy to protect against thromboembolism [2,4]. The hypercoagulability associated with pregnancy heightens the need for adequate anticoagulation.

The patient presented had vaginal delivery after she was admitted in active labour at term gestation. She was given morphine during labour and started on intravenous antibiotics.Patients with cardiac disease should be allowed to reach term and go into spontaneous labour. Labour induction is associated with significant haemodynamic changes that could precipitate cardiac failure and in case of failed induction need for operative delivery which carries its own added risk to the mother including pneumonia, infective endocarditis and pulmonary embolism [10]. Caesarean section should be done only for obstetric indications.Our patient had no indication for caesarean section. Intrapartum, the aim is to have a short, pain free labour. Apprehension and discomfort of labour should be reduced by liberally using sedatives and strong analgesics. Morphine is superior to all other drugs for this. It also reduces elevated respiratory rate and reduces reflex abdominal muscular activity in the second stage of labour. This was used for our patient. The mother should be in the semi-recumbent position in bed and oxygen given by mask. Parenteral antibiotics should be started and digoxin and frusemide administered. A fast acting diuretic such as frusemide is given to stimulate diuresis and relax capacitance vessels, which in turn decrease preload by reducing venous return. Patients with hypertension are given hydrallazine to reduce cardiac after load. Monitoring of vital signs, auscultation of lung bases are important in detecting early signs of congestive failure. A resuscitation tray with digoxin, frusemide, sodium bicarbonate, morphine, aminophylline should be kept ready. Second stage should be shortened by use of vacuum extraction as was done in our case. Where necessary, a generous episiotomy should be given to aid delivery. Bearing down should be discouraged. If delayed labour or poor progress is noted, caesarean section is done early enough as prolonged labour leads to dehydration, metabolic acidosis and infection of the genital tract and these may precipitate heart failure. This patient presented in labour was given morphine, and had assisted vacuum delivery.

The patient presented was given oxytocin and frusemide after delivery of the baby. Marked fluctuation of cardiac output can occur during labour and delivery. Cardiac output increases progressively from first stage of labour, in some cases reaching an additional 50% by late second stage. The potential for further dramatic volume shift is

heightened at the time of delivery, secondary to the physiologic transfusion which occurs with the release of vena cava obstruction and centralization of blood from the now contracted uterus, as well as postpartum haemorrhage. In the third stage, ergometrine should never be given as it may precipitate pulmonary oedema due to its sustained uterine contraction and its vasoconstrictive effect. Oxytocin is given since it does not cause vasoconstriction and also reduces peripheral resistance. Uterine massage is also sufficient. A bolus of frusemide 40-120 mg is given immediately after third stage to offset the anticipated increase in cardiac output from the placental bed on contraction of the uterus. As soon as delivery is complete and the episiotomy repaired, the patient is propped up given morphine and oxygen by mask [7-11].

Our patient stayed in the acute room for 48 hours and was ambulated early enough. She was on antibiotics for 14 days.The first 12 hours after delivery are most critical [2]. Signs of pulmonary congestion and oedema must be carefully watched for. Sedatives should be given liberally in the first few days to reduce tachycardia. The patient should not be allowed out of bed until the signs of cardiac failure if present have disappeared. Close obstetric and medical surveillance must continue particularly during the first 24-48 hours Mild infection should be treated vigorously. Infective endocarditis (an acute or subacute inflammatory process resulting from blood borne infection with streptococcus viridans or other streptococci) is suspected when symptoms like fever, chills, sweating and signs such as petechial haemorrhages, splenomegaly, Osler's nodes, appearance of new murmurs or change in existing murmurs occurs. Confirmation is by finding a positive blood culture or demonstration of vegetations on echocardiogram. In our unit parenteral antibiotics are given for two weeks after delivery. Early ambulation after the initial period is encouraged if the patient is not in failure.

Our patient was allowed to breastfeed soon after delivery.Patients with grade I or II disease should breastfeed as normal. However care should be taken allowing those with grade III and IV to breastfeed. Some authors(in the Western world) even think breastfeeding should be contraindicated in this group of patients [2]. However it should be remembered that artificial feeding might put the mother under more strain and trouble than breastfeeding.
Our patient did not come back for post natal evaluation where contraception would have been offered probably due to logistical reasons being unable to get transport to our hospital.Surgical sterilization is the contraceptive of choice in those with cardiac disease since the sequel of the next pregnancy is normally worse and the prognosis of cardiac mothers in pregnancy frequently worsens with increasing age and parity (the next pregnancies are poorly tolerated with worse outcomes both maternal and foetal). Barrier methods, depot progesterone or progesterone only contraceptives can also be used to achieve postnatal contraception. Oestrogen based contraceptives may increase the risk of thromboembolism and hypertension. Intrauterine devices may increase risks of infective endocarditis in patients with valvular lesions [1]. Ideally sterilization should be delayed until it is obvious that the mother has no infection and demonstrates that she can ambulate without evidence of stress. In our unit it is done 48 hours after delivery if the patient is stable.

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OBSTETRIC CASE 5

MALARIA IN PREGNANCY: REMISSION AFTER THERAPY- LIVE DELIVERY –C/SECTION DUE TO FOETAL DISTRESS

NAME: V.K. L.M.P: 25.10.03

AGE: 27 Years E.D.D: 1.08.04

I.P NO: 0957722 Parity: 1 + 0 Gravida 2

	1st admission		2nd admission
D.O.A:	8.05.2004	Readmission	4.7.04
D.O.D:	10.05.2004		9.7.04

PRESENTING COMPLAINT

She was admitted with complaints of fever, chills, joint pains and general body weakness for three days.

HISTORY OF PRESENTING COMPLAINTS

VK had travelled from Busia in western Kenya about a week prior to admission. Three days later she started feeling unwell having chills, fever and general body weakness. A day later she started experiencing generalized joint pains, the malaise was increasing and she was having loose stools but no vomiting. She also had associated headaches. It was then she visited a prison clinic where her spouse worked and was referred to the KNH. She had not received any malaria prophylaxis during her antenatal follow-up.

ANTENATAL CARE

Her last menstrual period was on 25.10.03 and expected date of delivery on 1.8.04; hence her gestation at admission was 28 weeks. She was attending antenatal care at the Mbagathi hospital from 18 weeks gestation. Profile done indicated the following; her blood group was A positive, haemoglobin 8.3g/dl, VDRL was negative. HIV test had not been done. She was started on haematinics (ferrous sulphate and folate).

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She attained her menarche at13 years. Her menstrual cycles were regular occurring every 28-30 days and flow 3-4 days. She had no dysmenorrhoea. She had previously used Depo-Provera injection after her last delivery
She was Para 1+0, her first delivery in hospital having been in 1999 vaginally to a term baby girl who weighed 3.5kg. The baby was alive and well.

PAST MEDICAL HISTORY

She had been admitted once before with typhoid fever in 2003. She did not have any chronic illnesses or allergies.

FAMILY AND SOCIAL HISTORY

She was married, lived in Busia but occasionally came to visit her husband who worked in Nairobi as a prison warder. She was a housewife. There were no major chronic illnesses in the family.

PHYSICAL EXAMINATION

She was in fair general condition, was mildly pale, and had no jaundice or lymphadenopathy. Her blood pressure was 100/60mmHg, pulse rate 112/minute, respiratory rate 22/minute and had temperature of 39.2^0C.
Her cardiovascular, respiratory and nervous systems were normal.

ABDOMINAL EXAMINATION

Her abdomen was uniformly distended; the fundal height was 28 weeks with longitudinal lie and cephalic presentation. The foetal heart tones were heard and regular at 138beats/minute. She had no contractions. The liver and spleen were not palpable. Pelvic examination was not done at this time.

INVESTIGATIONS

1. Blood slide for malaria revealed moderate parasitaemia with malarial parasites.

2. Haemogram; Haemoglobin 9.8g/dl with microcytic hypochromic picture.

3. Random blood sugar 5.8mmol/l

MANAGEMENT

VK was initially admitted to labour ward as the investigations were done, an intravenous line established. She was given intramuscular Aspergic acid 1g stat to control the fever. She was started on injectable Artenum once the blood slide result arrived at 300mg stat and was to continue with 100mg daily for four days. Once stable she was transferred to the lying-in ward to continue the artenum treatment and also paracetamol 1g three times daily for the fever. She recovered well and by the following day(9/5/04) was afebrile (36.8^0C) and was feeling much better. A repeat slide for malaria was negative. She was also started on iron therapy (Ranferon 10mls daily). She was to continue with treatment and observations but insisted on being discharged and was discharged against medical advice. However she was given oral artenum to continue taking at home for four days.

VK presented to hospital again on the 4.07.04 with labour pains. She was now at 36 weeks gestation. She had had two visits to the antenatal clinic at Mbagathi hospital after discharge from KNH. At one visit when she was at 33 weeks gestation, she was given malaria prophylaxis(fansidar three tablets).

Examination revealed a fundal height of 36 weeks gestation, longitudinal lie and cephalic presentation. She had moderate contractions, had an irregular and slow foetal heart at 90 beats/minute. The cervix was 2cm dilated and artificial rupture of membranes revealed meconium-staining grade II liquor. An impression of foetal distress was made and emergency caesarean section planned.She was informed of this and gave consent for caesarean section. An intravenous line was established and a drip of 5% dextrose fixed. She was placed in the left lateral position and oxygen administered by mask at 6L/min.A sample of blood for crossmatching was taken to the blood transfusion unit in case use of blood became necessary during the operation.

In theatre, aseptic catheterization was done, the abdomen then cleaned and draped. Anaesthesia was induced and at caesarean section, a live male baby was delivered who weighed 3600g and Apgar score of 8 in one and 10 in five minutes. He had a cord round the neck that was attributed as the cause of the foetal distress.

Postoperatively, observations remained normal and she was started on penicillin 2MU 6 hourly and gentamicin 80mg 8 hourly. Analgesia was provided initially with pethidine then followed by diclofenac 75mg three times daily. Both the mother and her baby remained well throughout and on the fourth day were discharged to be followed up in the postnatal clinic.

FOLLOW-UP

She did not honour her appointment.

DISCUSSION

The case presented is of a Para 1+0 who had malaria in pregnancy and was later delivered by caesarean section due to foetal distress.

Our patient developed malaria after travelling to western Kenya.
Malaria is a disease caused by a blood parasite of the genus plasmodia and is transmitted when a female anopheles mosquito carrying the parasite bites a person and inoculates the parasite. Malaria is a great problem in most parts of the world especially in the tropics and subtropics with 40% of the world's population being at risk. Presently, the disease is endemic in 101 territories and countries of which 45 countries are in Africa, 21 in the Americas, 4 in Europe, 14 in Mediterranean, 8 in south east Asia and 9 in the west pacific region. It is estimated that each year 110 million people are infected with malaria and of these 1.8-2 million die. Most deaths occur principally in children and pregnant mothers especially primigravidas [1]. In Kenya national statistics are scanty but malaria is the number one cause of morbidity in most hospitals accounting for 30% of outpatient attendances, especially in Western, Coast and Nyanza provinces. This patient was pregnant at the time of infection.

This patient had mild anaemia but none of the other complications of malaria.Brabin [2] who evaluated its risks and severity has extensively reviewed malaria in pregnancy. The diagnosis of malaria should be sought not only for the pregnant women residing in endemic areas but also for all those with a fever syndrome and have travelled through areas where there is a risk of transmission. Malaria causes serious complications in pregnant women especially those who have low levels of acquired immunity before pregnancy. In endemic areas, pregnant women are the main group of adults at risk for malaria. Plasmodium falciparum infection during pregnancy increases the chances of maternal anaemia, abortion, stillbirth, prematurity, intrauterine growth retardation and low birth weight (below 2500g), which is the greatest risk factor for neonatal morbidity and mortality. The effects of the other three malaria-causing parasites (P. vivax, P. malariae and P. ovale) are less clear [1-6].

Mechanisms underlying the adverse effects of malaria in pregnancy are not well understood. Low birth weight and miscarriages have been described as the effects on the foetus. In the hyperendemic areas, the association between malaria and the spontaneous abortions has not been well documented; on the other hand there is a close relationship with low birth weight. The causes leading to low birth weight may be related to the reduction of the oxygen and nutrients reaching the foetus, premature delivery, presence of parasitaemia during delivery, first pregnancy, mother below 20 years age, underweight mothers, maternal anaemia and active placental infection [7]. Various factors have been identified as important in the virulence of plasmodia including; the capacity to multiply, preference for certain life-stages of erythrocytes, ability to produce cytoadherence and rossetting, potential to induce the release of cytokines, antigenicity not recognised by the host and resistance to antimalarial drugs.

In most of Sub-Saharan Africa, stable transmission of P. falciparum is the norm and women therefore have a relatively high level of acquired immunity from repeated attacks of malaria. Parasites can often be found in peripheral films of an asymptomatic patient. This immunity declines if the individual leaves the endemic area. Maternal immunity declines during pregnancy, particularly in primigravidae probably due to transfer of IgG. Both parasite rates and densities are higher during pregnancy. This susceptibility appears to diminish with subsequent pregnancies[7, 8]. Malaria promotes placental insufficiency and can lead to intrauterine growth retardation, low birth weight and foetal death. The uteroplacental vascular spaces provide a relatively protected site for parasite sequestration and development. It is this parasite replication in the placenta, its potential to alter transplacental nutrient transport, the passage of parasitised red cells to the foetus and the anaemic state of the mother that may lead to serious compromise of the foetus. The density of placental parasitisation and delivery of low birth weight babies have been shown to be associated.[8, 9]. Early placental infection (less than 32 weeks) is rare and thought to be due to maternal factors leading to placental infection from 16th to 32nd week of gestation. This factor has been explained on the basis of a surge of immunity, which protects the early placenta[10].

The patient presented had mild anaemia thought to be due to the malaria.Malaria causes anaemia in various ways including; rupture of parasitised erythrocytes, sequestration of erythrocytes in tissue venules, destruction of parasitised cells by the monocyte-macrophage system, haemolysis due to presence of malaria antigen, antibodies and complement on erythrocytes and marrow suppression. Malaria is thought to stimulate prostaglandin metabolism due to pyrexia, initiating vigorous uterine contraction and thus premature labour[11, 12]. Mild malaria attacks have little effect on labour. Malaria is common in the puerperium but does not interfere with lactation unless the patient is gravely ill with severe anaemia. To prevent relapses prophylactic drugs used in pregnancy should be continued up to six weeks after delivery. Congenital malaria is likely only when the level of immunity passively transferred to the foetus is low. It's therefore rare in endemic areas. It is common in infants of unprotected susceptible immigrants from non-malaria areas and the indigenous mothers in areas of unstable malaria. This patient did not turn up for follow up thus may not have received post partum prophylaxis.

Our patient had received prophylaxis with SP once after the acute attack.
Malaria is a priority disease in Kenya, accounting for 30% of outpatient attendance nationally. Women in the first and second pregnancy are at greatest risk of the effects of malaria. The Ministry of health guidelines advice that all pregnant women in endemic areas should receive a dose of sulfadoxine/pyrimethamine (SP) combination at the beginning of the 2nd trimester, and a second dose at the beginning of the 3rd trimester (between 28 and 34 weeks) [11]. One problem with treatment during pregnancy is compliance. In a study in Kilifi, Kenya it was shown that intermittent treatment with SP combination during clinic attendance avoided non-compliance and was effective in reducing the frequency of severe anaemia in primigravidae living in areas with high prevalence of malaria infection [13].

Our patient was treated with artenum which was available in the unit as she could not afford the recommended lumefantrine/artemisinine combination.Malarial parasite resistance to common antimalaria agents has been a major therapeutic problem. The following should be considered before treatment is initiated with any agent; the place where infection occurred so as to know whether it's a resistant or multi-resistant strain, the stage of pregnancy which serves to guide the choice of drugs that are least toxic to the foetus, and whether it is severe disease. Malaria should be considered an emergency situation requiring immediate treatment. The drug chosen should be effective in the reduction of asexual forms not only in the blood but also in the placenta. These measures prevent or reduce the severe clinical forms. Quinine is the drug of choice in severe malaria but artemisinin derivatives are being preferred, as they are safe, have rapid onset of action and have fewer side effects. Primaquine, tetracycline and doxycycline should never be used in pregnancy. In Kenya, the ministry of health guidelines recommend the use of SP drugs as first line treatment for non-complicated malaria, while quinine is used for very sick patients. Lumefantrine/Artemisinine combinations have also been included in the guidelines as first line treatment. Due to widespread resistance to chloroquine it is no longer recommended in the prophylaxis or treatment of malaria in Kenya [9].

For prophylaxis, consideration should be taken of the resistance of P.falciparum and the contra-indications of anti-malarials during the different stages of pregnancy. Studies have been done on proguanil, chloroquine and mefloquine showing reduction of parasitaemia. Unfortunately chloroquine resistance is high in majority of countries where malaria is endemic [14,15,16]. The issues of cost of prophylaxis programs and compliance should also be taken into account. Other measures in place locally to reduce the malaria burden include use of insecticide-treated bed nets to prevent mosquito bites and other preventive measures such as use of ferrous sulphate and folic acid to prevent anaemia[9]. The Kenya health policy framework of 1994 proposed strengthening of activities geared towards the reduction of mortality and morbidity due to malaria. Pregnancy increases a woman's risk of illness by four and doubles her risk of death. In April 2001 the malaria control unit released a malaria control strategy that aims at decreasing malaria illness by 30% by 2006 and sustain the improved level of control up to 2010. It is also expected that 60% of pregnant women will have 2 doses intermittent preventive therapy with SP drugs in the second and third trimester at least four weeks apart and 80% of pregnant women with fever and anaemia to be approximately managed at antenatal services[11].

The patient presented did not develop any major complications except anaemia. Complications of malaria include multisystemic disease that may affect the brain, liver, kidneys and lungs. It may cause impaired consciousness, drowsiness, progressing to coma and convulsions. It may cause oliguria with raised urea and electrolytes, hamoglobinuria (black water fever). Involvement of the liver causes upper abdominal tenderness, marked jaundice with elevated bilirubin and aminotransferases. Lung involvement is indicated by dyspnoea, tachypnoea, cyanosis and scattered bilateral crackles. Intravascular haemolysis results in haemoglobinuria, tachycardia, low blood pressure, jaundice and oliguria[17].

This patient was delivered by caesarean section due to cord around the neck which was noted intraoperatively. Foetal distress indicates a critical response of the foetus to stress and implies metabolic derangements especially hypoxia and acidosis. In our unit, diagnosis is made using intermittent auscultation with the Pinnard foetoscope where irregularities of the foetal heart are noted as we do not yet have electronic monitors available. Meconium passage may be associated with foetal distress but is not diagnostic and in this case follows vagal stimulation from umbilical cord entrapment resulting in hypoxia. The best way to describe foetal distress is by assessing foetal acid-base status. A ph less than 7.2 indicates foetal distress. This was not done in our patient as the necessary equipment is not readily available. Our patient had bradycardia with a foetal heart rate of 90/minute. Hormonal response to hypoxaemia is through carotid artery chemoreceptors in the carotid sinus nerve, a parasympathetic nerve whose stimulation results in bradycardia [18,19].

Giving the mother oxygen as was done in our patient increases the oxygen delivery to the uteroplacental unit. Prevention of dehydration with intravenous fluids is helpful. Repositioning the mother to the left lateral position helps to increase blood flow with concomitant increased oxygen supply to the foetus. These were done to the patient presented.

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OBSTETRIC CASE 6

HIV IN PREGNANCY- PMTCT PROPHYLAXIS ELECTIVE CAESAREAN SECTION AT TERM- LIVE BABY.

NAME: A.A.O.

AGE: 21 Years

I.P NO: 0851476

D.O.A: 4.12.02

D.O.D: 8.12.02

L.M.P: 10.03.2002

E.D.D: 17.12.2002

Parity: 0 + 0 Gravida 1

PRESENTING COMPLAINT

She was at term gestation and was admitted from the KNH clinic for elective caesarean section.

HISTORY OF PRESENTING COMPLAINT

She had been attending the antenatal clinic at KNH where she was found to be HIV positive and was put under the prevention of mother to child transmission of HIV programme. She was scheduled for elective operation at term. She was admitted at 38 weeks gestation from the clinic for elective caesarean section.

ANTENATAL CARE

She attended antenatal care from 29 weeks gestation at KNH. Antenatal profile done showed; blood group B positive, haemoglobin 11.1g/dl, VDRL test negative and HIV I test positive. She was started on azidothymidine (AZT) 300mg twice daily from 34 weeks gestation after counseling about her condition and ways of protecting her baby from infection. This was continued throughout her pregnancy. Her fundal height corresponded to the dates throughout the six visits she attended. She was given haematinics (ranferon). She was adviced on safe sex (use of condoms), and the need to have her partner tested. Nutritional advice was also given with emphasis on balanced nutrition, and she was started on multivitamins.She was adviced to avoid uncooked or undercooked food except fruits. Her partner did not accompany her during her visits and was not tested for HIV. She had chosen not to breastfeed after delivery.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She had her menarche at 14 years. Her menstrual cycles were regular occurring every 28 days and flow 3-4 days. She had no dysmenorrhoea. She had not used any contraceptives previously. She gave no prior history of treatment for any sexually transmitted infection.

She was a primigravida, this being her first pregnancy. Her last menstrual period was on 10.03.02 and her expected date of delivery on 17.1.02 and hence at 38 weeks and one day maturity at admission. She had never had a Pap smear.

PAST MEDICAL HISTORY

She had never been admitted previously, had no history of allergies or previous blood transfusion.

FAMILY AND SOCIAL HISTORY

She was married and lived with her spouse at Kayole. She was a housewife while the spouse was a casual labourer. She was educated up to primary class seven. She did not use alcohol or smoke cigarettes. She had no family history of chronic illnesses.

PHYSICAL EXAMINATION

She was in fair general condition, not pale and had no jaundice or oedema. She had cervical and axillary discrete lymphadenopathy. Her blood pressure was 100/60mmHg, pulse rate 82/minute, respiratory rate 20/minute and temperature 36.2^0C.
Her respiratory, cardiovascular and nervous systems were normal.

ABDOMINAL EXAMINATION

Her abdomen was uniformly distended and had no surgical scars.She had evidence of healed rash on the abdomen. The fundal height was term with longitudinal lie, cephalic presentation and the foetal heart tones were heard regular at 136 beats/minute. She had no contractions and the presenting part was five fifths above the pelvic brim.

PELVIC EXAMINATION

She had normal external genitalia. The cervix was closed, posterior, thick and long about 2cm long. There was no vaginal discharge.

INVESTIGATIONS

1. Haemogram – Haemoglobin 10.3g/dl
   white cell count 4.5 x $10^3/mm^3$
   plaetelets 300x$10^3/mm^3$

2. Urea and electrolytes- Urea 1.4 mmol/l
   Na^+ 141 mmol/l
   K^+ 3.8 mmol/l
   Creatinine 78 umol/l

3. Blood group B positive and cross-match of two units

4. Liver function tests showed normal enzymes and bilirubin levels.

DIAGNOSIS

HIV in pregnancy at term gestation.

MANAGEMENT

In view of her HIV status, she had been counseled on various methods of reducing mother to child transmission of HIV and she had chosen on elective caesarean section. She was admitted and prepared for the same. She was further counselled to confirm the mode of management and she gave informed consent for elective c.section. She was starved from midnight on the night before surgery. On the morning of surgery she was given a last dose of AZT with a small sip of water, she was premedicated with atropine 0.6mg and an intravenous line with 5% dextrose drip set up. She was then wheeled into theatre.
In theatre, vulvo-vaginal toile was done, she was catheterised and clear urine was drained. The abdomen was then surgically cleaned and covered with sterile drapes.
A lower segment caesarean section was then done. A live female baby with Apgar score 8 in one and 10 in five minutes and who weighed 2700g was delivered. The baby was in good condition, her face was wiped immediately on extraction and minimal suction was done.

POSTOPERATIVE MANAGEMENT

She was observed until fully awake then taken back to the ward and observations continued four hourly. She was on intravenous fluids for 24 hours after which oral sips were started and gradually allowed on free diet by the third day. She was also on intravenous penicillin 2mu 6 hourly and gentamicin 80mg 8 hourly until the third day when she was started on oral amoxil 500mg three times daily. She was given mefenamic acid 500mg three times daily for analgesia. The baby was put on AZT syrup (5mg twice daily) for six weeks. The mother had opted not to breastfeed and was given bromocriptine to stop the lactation that had started.
By the fourth postoperative day her wound was healing well and they were both discharged home to be seen in the postnatal clinic in two weeks time. She was given formula milk for the baby and she was to continue with her oral drugs.

FOLLOW-UP

She was seen and had no complaints. Her wound had healed well, and the breasts were not active. The baby was doing well on infant formula milk. She was discharged to the comprehensive care clinic for follow-up of her condition and to the family planning clinic for contraception. The baby was also to be seen in the paediatric PMTCT program center for follow-up.

DISCUSSION

The patient presented was a 23 year-old primigravida found to be HIV infected during antenatal follow-up. She was given AZT from 34 weeks gestation and had elective caesarean section at 38 weeks. The baby then received AZT syrup for six weeks.
The human immunodeficiency virus (HIV) is the cause of acquired immune deficiency syndrome (AIDS) in humans. AIDS is characterized by profound immuno-incompetence, recurrent opportunistic infections and susceptibility to certain malignancies. The seroprevalence of HIV infection in pregnancy among screened antenatal mothers in East Africa is as high as 20-32%[1,2]. Seroprevalence as high as 41% has been reported from South Africa[3]. HIV/AIDS has been declared a national disaster in Kenya since November 1999 and is nationally responsible for over 700 deaths daily and over 50% bed occupancy in most public hospitals.

The first description of AIDS was in 1981. A viral aetiologic agent was demonstrated in 1985-the HIV. HIV1 and HIV2 have since been known to be the cause of AIDS. HIV infection continues to increase rapidly in the developing world, especially in Africa and Asia[4]. Current estimates are that there are over 11 million women infected worldwide, with a majority living in sub-Saharan Africa. More than 80% of infections in women occur in the reproductive age group and up to 90% acquire HIV heterosexually[5].

Our patient was first diagnosed to have HIV during pregnancy.The issue of screening for HIV in antenatal care clinics has been contentious. At KNH, overall acceptability of 99.4% was found when testing was offered during ANC[6]. Similar rates are reported in West Africa[7, 8]. However this testing has been found to interfere with continuance of care for these women [6,7,8]. Our patient had screening test in the clinic using the ELISA test. Other tests used for HIV infection include the Western blot, viral cultures, Polymerase chain reaction (PCR), and CD4 counts. Viral cultures and CD4 lymphocyte counts are important in assessing the severity of the disease and response to retroviral treatment.

This patient had term delivery to a normal baby and was well at discharge.Early repots from New York suggested that pregnancy might cause more rapid progression of HIV illness, resulting in the impairment of the immune[4]. However current consensus is that pregnancy in HIV infected women is a high-risk pregnancy. Increased rates of abortion and prematurity have been reported in these women; also increased rates of urinary tract infection, herpes zoster, low birth weight, intrauterine growth retardation, chorio-amnionitis, puerperal infection. No increase in congenital abnormalities has been noted in the HIV infected women over the general population[1, 10,11].
Our patient did not have CD4 counts during pregnancy and was on antiretrovirals for prevention of mother to child transmission(PMTCT) of HIV.Mother to child transmission contributes significantly to paediatric AIDS. It is the major route of infection in childhood and 300,000 children are infected in this way each year worldwide4. 20-40% of HIV infected mothers pass on the virus to their babies and about 80% of the infected children will die of AIDS before their 5th birthday. The likelihood of vertical transmission is related to the viral load. It is also increased in women with advanced disease, low CD4 cell count and P24 core antigenaemia, and in women in the seroconversion phase of the

disease [1,2,4]. Other factors include; pre-term delivery, premature rupture of membranes with over 4 hour period nearly doubling the transmission risk, vaginal delivery has a higher risk than elective caesarean section. Episiotomies, intrapartum haemorrhage, use of invasive foetal monitoring devices, instrumental deliveries and twin deliveries (the first twin has a higher risk of infection than the second twin) are factors also thought to increase transmission rates. Our patient had elective caesarean delivery at term and thus most of these risks were avoided.

Our patient was delivered by elective caesarean section as part of PMTCT.Vertical transmission is a major concern for the obstetrician. This can occur antepartum, intrapartum or postpartum. It's estimated that about 25-40% transmission occurs in late pregnancy, 60-75% during delivery. Breastfeeding carries an additional risk of transmission of approximately 14% (established infection) and as high as 29% risk with primary infection in the current pregnancy/breastfeeding period. Transmission from early breast milk is hypothesized to be considerable due to the high cellular content of early milk. In early pregnancy it is thought to occur by invasion of the virus through the villous stroma into the foetal circulation via Hofbauer cells or by invasion of trophoblast. Most of transmission occurs during labour and delivery with increased risk in prolonged rupture of membranes, prolonged labour, chorio-amnionitis and this seems to be related to the length of time during which the baby is in contact with cervical and vaginal secretions [1,4,11,12,13]. Since our patient did not go into labour, and had opted not to breastfeed her baby, the risk of vertical transmission was reduced.

This patient was on zidovudine as part of the PMTCT protocol in use in our unit at that time.Despite the availability of very potent anti-viral drugs, treatment of HIV has been characterized by phases of hope and of disappointment. Viral resistance to drugs is apparent within weeks of initiation of therapy. Combined therapy of zidovudine (AZT) with other agents such as ddC, 3TC or protease inhibitors results in greater and sustained fall in viraemia. Symptomatic treatment, nutritional support and treatment of opportunistic infections are important modalities of management in these patients. Important progress has been made in the area of PMTCT. Interventions that reduce the viral load may help reduce the transmission. The AIDS Clinical Trials Group Protocol 076(ACTOG076) comprising of antepartum AZT 100mg orally five times daily, intrapartum AZT 2mg/kg body weight given intravenously over a one hour period, then 1mg /kg body weight per hour until delivery, and AZT for the newborn at 2mg /kg body weight orally every 6 hours for six weeks reduced the risk of mother to child transmission by approximately twothirds[14]. However due to its complexity and costs, this regimen has not been implemented in many developing countries.

A simple and less expensive regimen of AZT consisting of 300mg orally twice daily from 36 weeks gestation until onset of labour, then 300mg 3 hourly until delivery, and no breastfeeding after delivery, was used in Thailand and reduced perinatal HIV transmission by approximately half [16]. This is the protocol our patient had been put on. Regimens combining multiple antiretroviral agents, protease inhibitors may decrease transmission further. Nevirapine (NVP) a non-nucleoside benzodiazepine derivative, a potent inhibitor of HIV replication has also been used as a single oral dose given to HIV

infected mothers during labour at 200mg, and a single 2mg/kg oral dose to the infant within 72 hours of delivery is thought superior to AZT [15] and this was shown in the HIVNET 012 trial in Uganda. Presently the two dose NVP is the least expensive and most easily administered and is likely to be implementable in most of developing countries. Use of HAART (Highly active antiretroviral therapy) in pregnancy should be the ultimate goal for PMTCT since it has been shown to lower the risk up to 2% in some centers in Europe.

Our patient had elective caesarean section.Recent studies have established that elective caesarean section delivery prior to labour and membrane rupture reduces perinatal transmission [12,17,18]. Caesarean delivery reduced the risk of perinatal transmission by around 50%. In addition, elective caesarean delivery combined with the three-part AZT prophylaxis regimen reduced transmission by about 85% compared with other modes of delivery[17, 18]. Elective caesarean section reduces the exposure of the baby to blood or cervical secretions and microtransfusion during uterine contractions. Other studies showed no justification for routine use of caesarean section for HIV infected women [19,20]. HIV positive mothers are at an increased risk of complications when delivered by caesarean section, particularly those with a CD4 count less than $200x10^6$/l. CD4 counts had not been done in our patient as she could not afford the cost of the test. Currently, this service is being offered at no cost to antenatal mothers in our unit.

Chlorhexidine has been shown to neutralize HIV in vitro. Vaginal cleansing with 0.25% chlorhexidine during labour combined with infant washing after birth has been used in the reduction of perinatal transmission[1,4]. During labour avoidance of invasive procedures like foetal scalp sampling and use of scalp electrodes is adviced. Delay in rupturing the membranes, less use of episiotomies unless indicated is also encouraged. Avoidance of breastfeeding reduces perinatal transmission by about half [1,4]. However in most developing countries, formula feeds are expensive, hygiene not up to standard, lack of water may lead to increased infant morbidity and mortality, thus the risk of not breastfeeding has to be weighed against advantages of avoiding higher mortalities from diarrhoeal and other childhood afflictions.

Other methods on trial include use of passive and active immunization to the mother and child with hyperimmune intravenous immunoglobulin (HIVIg).
Before the availability and affordability of HAART is globally realized, development of successful strategies for primary prevention in women and their unborn children must be made top health priorities. A lot has to be done especially in the area of sexual and behavioral change. Policies and practices to strengthen access to prenatal care, testing and counseling for HIV infection, and provision of antiretroviral therapy for HIV infected women should be developed. Fortunately this has been started at KNH but it needs to be extended to all public hospitals.

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OBSTETRIC SHORT CASE 7

PRETERM PREMATURE RUPTURE OF MEMBRANES AT 31 WEEKS- CONSERVATIVE MANAGEMENT AND LIVE PRETERM C/SECTION DELIVERY DUE TO CORD PROLAPSE.

NAME: M.N.B L.M.P. 13/08/03

AGE: 27 Years. E.D.D. 20/05/04

I.P. NO: 0814377 Parity. 2 + 6 Gravida 9

D.O.A: 11/03/04

D.O.D: 29/03/04

PRESENTING COMPLAINTS

MNB presented to labour ward with complaints of drainage of liquor for six hours without labour pains.

HISTORY OF PRESENTING COMPLAINTS

She had had a McDonald stitch inserted at KNH at 14 weeks gestation and was currently at 31 weeks gestation.
She had been well until the morning of admission, when she felt a sudden gush of fluid run down her legs. The fluid was clear, not foul smelling and of moderate quantity. She had changed two soaked pads by the time she arrived to hospital. She had no associated abdominal pain, dysuria, urine frequency or fever. She also gave no history of trauma or strenuous physical activity, no prior abnormal vaginal discharge before onset of draining.

ANTENATAL CARE

Her last menstrual was on 13/8/03, and her expected date of delivery was on 20/5/04. She was at a gestation of 30 weeks and five days on the date of admission. She had started antenatal care at KNH, her profile indicated blood group O positive, VDRL and HIV tests were negative and her haemoglobin was 11g/dl. She had a McDonald stitch inserted at 14 weeks gestation (20/11/03) due to cervical incompetence. The antenatal period had been uneventful until she came with the above complaints.

OBSTETRICS AND GYNAECOLOGY HISTORY

She was a para 2+6 gravida 9. Her menarche was at 14 years and her menses were regular, lasting four days and occurring every 28 days. She had no dysmenorrhoea. She had not used any contraceptives previously. Her first delivery was in 1995 when she delivered a 4kg male baby who is alive and well. In 1998 she had a premature delivery at

7 months gestation to a live female baby weighing 1.5kg who is alive and well. Since then she has had six pregnancy losses (in1996, 1997, 1999, 2001, 2002, and 2003) all at approximately six months gestation. Each was preceded by drainage of liquor then labour pains followed by expulsion of the foetus. They were all abortions.

PAST MEDICAL HISTORY

She had no previous admissions or chronic illnesses.

FAMILY AND SOCIAL HISTORY

She was a married lady who lived with her family in Kibera. She had two living children. She was a housewife, did not use alcohol or smoke cigarettes. There were no chronic illnesses in the family.

SYSTEMIC ENQUIRY.

Non-revealing.

PHYSICAL EXAMINATION

She was a young lady in good general condition, was not pale or jaundiced and had no fever. Her vital signs were; blood pressure 110/70mmHg, respiratory rate 20/minute, pulse rate 82/minute and temperature of 36.8^0 C.

Her respiratory, cardiovascular and central nervous systems were essentially normal.

ABDOMINAL EXAMINATION

The abdomen was uniformly distended and moving with respiration. The fundal height was at 30 weeks, the lie was longitudinal, cephalic presentation and the liquor felt adequate clinically. The presenting part was five-fifths above the pelvic brim. The foetal heart rate was heard at 140 beats/minute and was regular. There was no uterine tenderness or any contractions palpable.

SPECULUM EXAMINATION

Vulval toilet was done, the external genitalia were normal and there was clear fluid at the vulval opening. A sterile speculum was gently inserted into the vagina. The vaginal mucosa was normal. The cervix was visualized, it was normal and there was clear fluid draining from the closed cervical os and forming a pool in the posterior fornix. There was no cord prolapsing from the cervical opening. The McDonald stitch was removed.

DIAGNOSIS

Preterm premature rupture of membranes (PPROM) at 30 weeks with no features of infection was made.

MANAGEMENT

1st Admission 19/11/03 – 22/11/03

At 14 weeks gestation, she was admitted to the antenatal wards for cervical cerclage. An obstetric scan done then had shown a normally growing foetus. Pelvic examination revealed a short cervix. She consented for MacDonald stitch insertion.
In theatre and under general anaesthesia, she was placed in the lithotomy position. Vulvovaginal toilet was done and draping done. The urinary bladder was catheterized. Examination under anaesthesia revealed a short cervix about 1.5cm long and which was open 2cm. An Auvard speculum was inserted to expose the cervix and both the anterior and posterior lips of the cervix held with sponge holding forceps. No anatomical defects were noted. With gentle traction on the cervix, No. 2 silk suture on a round body needle was used to insert a purse string at the level of the internal os that was identified as the point where the smooth cervical mucosa meets the rugged vaginal wall mucosa. Four bites were taken at 1,4,7 and 11 o'clock and avoiding the vascular and nerve bundles at 3 and 9 o'clock. The knot was tied at 6 o'clock leaving about 2cm of the suture with the knot. The knot was tied tight to just allow space for the little finger.
Post operatively she was monitored with emphasis on checking on any drainage of liquor, vaginal bleeding, uterine contractions or fever. She did not develop any of these. She was discharged on the second day with advice to have bed rest, avoid sexual intercourse and avoid strenuous activity. She was to be followed up in the antenatal clinic. She had six visits by the time of the second admission.

2nd Admission 11/3/04 – 29/3/04

She was admitted with preterm premature rupture of membranes at 31 weeks gestation She was planned for conservative management which was explained to her and she agreed to this. She was put on bed rest, 4 hourly vital signs observations including temperature charting, maternal pulse rate and blood pressure. Temperature rise above 38^0C or pulse above 100/minute was to indicate probable amnionitis. The heart rate was also monitored and any tachycardia above 160 beats/minute to be noted.
The amount of liquor drainage was monitored using a perineal pad and she was to avoid douching. Foetal well being was also monitored with a kick chart. She was also put on antibiotics (erythromycin 500mg 6 hourly for one week) and was given dexamethasone 12mg two doses twelve hours apart. During the period of admission, she did not develop foul smell in the liquor, fever or abdominal tenderness but continued draining liquor. Baseline specimens were taken for haemogram with emphasis on white cell counts with repeat weekly, urine culture, microscopy and sensitivity patterns, endocervical swab was taken for microscopy, culture and sensitivity. The results are presented below:

INVESTIGATIONS

	12/3/04	17/4/04	21/3/04
Haemogram: Haemoglobin g/dl	10.3	10.4	10.2
White cell count (x 109/l)	10.3	10.6	10.6
Neutrophils(%)	70	70	70

Urinalysis: normal, no growth on culture was obtained.

Endocervical swab: culture was negative.

Obstetric scan: Showed a single foetus at 29 weeks gestation, amniotic fluid slightly reduced and biophysical profile of six out of eight.

MNB remained stable in the ward, the amount of liquor draining reduced. On the morning of the eleventh day after admission, she complained of feeling of something hanging in her perineum. She could perceive foetal movements. On examination, the fundal height was at 30 weeks, she had no contractions and the foetal heart tones were perceived at 138 beats/minute. On pelvic examination, the cord was seen prolapsing through the introitus, it was pulsatile. The cervic was 3 cm dilated on digital examination. A diagnosis of cord prolapse was made. She was immediately put in the knee chest position to try and relieve the pressure on the cord if any. The cord was replaced into the vagina and she was given oxygen by mask. An intravenous line was fixed, a blood sample taken for crossmatching blood and one unit asked to be availed.

She was immediately prepared for emergency caesarean section and transferred to labour ward where caesarean section was done. The outcome was a live female baby weighing 1500g and scored 8 in one and 9 in five minutes. The baby was taken to the newborn unit due to low birth weight and prematurity. The mother did well postoperatively. She was continued on antibiotics and endocervical cultures were still negative. On the seventh postoperative day, she was examined and found to be afebrile, the wound was well healed, the uterus was well involuted and the lochia was not foul smelling. She was discharged on antibiotics to await her baby while at the mother's hostels.

DISCUSSION

The case presented here is of a 27 years old para 2+6 who had preterm premature rupture of membranes at 30 weeks. She was managed conservatively for eleven days when she was delivered by caesarean section due to cord prolapse and had a live baby who was taken to the newborn unit. Her postnatal period was unremarkable.

The foetal membranes (amniochorial membranes) is composed of two layers derived from the amnion and chorion, which are closely adherent layers consisting of several cell types including epithelial cells, mesenchymal cells and trophoblast cells embedded in a collagenous matrix. The membranes are sealed and contain amniotic fluid by the twelfth week gestation. They retain amniotic fluid, secrete substances both into the amniotic fluid and toward the uterus and they guard the foetus against infection ascending the reproductive tract. The membranes normally rupture during labour.

This patient had premature rupture of memebranes at 31 weeks gestation.Premature rupture of membranes (PROM) refers to spontaneous rupture of foetal membranes with leakage of amniotic fluid more than 8 hours before the onset of labour regardless of gestation. Preterm premature rupture of membranes (PPROM) is rupture of membranes before 37 weeks. If more than 24 hours have elapsed between onset of rupture of membranes and the onset of labour then prolonged rupture of membranes has occurred. [1,2].The patient presented had PPROM.

Our patient delivered prematurely after she developed cord prolapse and caesarean section was done.Despite advances made in perinatal care, PPROM and PROM continue to be important obstetric complications. Both conditions are important causes of maternal and perinatal morbidity and mortality. At term, 8-10% of pregnant women present with PROM which increases the risk of intrauterine infection especially if the period between rupture and delivery of the baby is prolonged [3]. PPROM occurs in approximately 1% of all pregnancies and is associated with 30-40% of preterm deliveries. It is the leading identifiable cause of preterm delivery and its complications including respiratory distress syndrome, neonatal infection and intraventricular haemorrhage. It is the greatest cause (other than congenital anomalies) of neonatal morbidity and mortality. Even for those infants who survive the sequel of untimely birth, permanent disabilities often result such as cerebral palsy. After preterm premature rupture of membranes the latency period from membrane rupture to delivery decreases inversely with advancing gestation [4]. The rate of neonatal sepsis after PPROM ranges from 2-20% and the incidence of neonatal death caused by infection is approximately 5%. The global incidence of PROM ranges 2-18% and it contributes about 20% of all perinatal deaths. In up to 94% of these cases, the foetus is mature and only 6-10% of them are premature [1,2,5]. At Kenyatta hospital, Wanjala [6] reported an incidence of 8.2% in 1980 and the Nairobi birth survey found an incidence of 16% in 1982 [7]. Our patient's baby did not develop sepsis but was premature.

The patient presented had cervical incompetence as a risk factor for PROM.
In majority of cases, the cause of premature rupture of membranes is not known, but various risk factors have been identified. The final mechanism is the ultimate weakening

of the amniochorial membrane, which allows rupture. Factors associated with PROM include; changes in collagen content, structure and catabolism, connective tissues disorders such as in Ehlers-Danlos syndrome, nutritional deficiencies of vitamin C and E, copper and zinc deficiency. Increased collagen degradation as a result of altered balance between matrix metalloproteinases (MMP) and tissue inhibitor of matrix metalloproteinases (TIMP). Risk factors for PROM include; cervico-vaginitis, chorioamnionitis, cigarette smoking, cervical incompetence, polyhydramnios, multiple pregnancy, vaginal bleeding any time during the index pregnancy, urinary tract infection, and previous preterm delivery or PROM, prenatal diagnostic procedures such as amniocentesis and chorionic villous sampling and foetal malpresentation. Other risk factors are maternal injuries, coitus, digital pelvic examination, placental pathologies such as placenta praevia and abruptio placenta and inherent decreased tensile strength of foetal membranes as connective tissue disorders.

Our patient had McDonald stitch due to cervical incompetence, which is the technique commonly used in our unit.Cervical incompetence is characterized by repeated second trimester pregnancy losses due to weakness and inability of the cervix to maintain the pregnancy until term gestation. It is characterized by relatively painless dilation of the cervix in the second and early third trimester with prolapse and ballooning of membranes into the cervical canal followed by rupture of the membranes and rapid expulsion of an immature foetus[7].This patient had six previous abortions all preceded by drainage of liquor and followed by expulsion of the foetus. Treatment of incompetence involves strengthening of the weak uterocervical junction that is achieved by various cerclage technique that use a purse string suture passed around the internal cervical os[8,9].

Endocervical cultures done in our patient were negative for any organism growth. There is indirect evidence that genital tract infection precipitates rupture of membranes in animals and humans. Microbial invasion of amniotic cavity is involved in 30-40% of patients with PPROM and the lower the gestational age, the higher the association with intrauterine infection. Epidemiological data demonstrates an association between colonization of the genital tract by Group B streptococci, chlamydia trachomatis, neisseria gonorrhoea and the microorganisms causing bacterial vaginosis (vaginal anaerobes, gardnerella vaginalis, mobiluncus and genital mycoplasma) and an increased risk of premature preterm rupture of membranes. Further, in some studies, treatment of infected women with antibiotics decreased the rate of preterm premature rupture of membranes[3,5,9].

In our patient, the diagnosis was made from history and speculum examination visualized leaking from the cervical os forming a pool in the posterior fornix.Diagnosis of PPROM is made from history, physical examination and laboratory testing. History alone has an accuracy of 90%. There may be history of sudden gush of amniotic fluid vaginally or intermittent leakage of small amounts of amniotic fluid or increased perineal moisture. A most important step in accurate diagnosis is examination with a sterile speculum. This is conducted only after careful abdominal examination reveals no contraindication to vaginal examination. The posterior vaginal fornix is exposed by means of a speculum. Amniotic fluid can be observed flowing from the cervical os, forming a pool in the

posterior fornix. If the fluid cannot be visualized easily, application of some slight fundal pressure or asking the patient to cough or perform a valsalva manouvre may induce leakage of liquor. Presence of meconium staining often verifies the diagnosis. Hair and vernix in the pool of fluid are gross indicators that it is amniotic fluid. If doubt still exists about rupture of membranes, transabdominal instillation of 1ml of indigo carmine dye in 9mls of sterile saline (or Evans blue or pyridium) into the amniotic cavity under ultrasound guidance and observation of staining of a vaginal sanitary pad quarter hour later should alleviate any question of rupture of membrane. This is done after removal of fluid for physiologic maturity testing, analysis of white cell count or bacteria, and possible culture and sensitivity. During speculum examination, one should determine the cervical dilatation and effacement, check for cord prolapse and if no fluid is found give a pad and observe for subsequent leakage. These were checked in our patient and cord prolapse developed later while on conservative management.

PROM should be differentiated from vaginitis, increased vaginal fluid, semen and urinary incontinence. Various diagnostic tests can be done to confirm that the fluid leaking is actually amniotic fluid. Nitrazine paper test turns blue from yellow when exposed to amniotic fluid-amniotic fluid is alkaline (pH 7.0-7.25); arborisation fern test when the fluid is dried on glass slide; presence of alpha fetoproteins. Other tests that can be done on the fluid include culture and sensitivity, determination of L: S ratio, phosphatidyl glycerol or quick surfactant test. The ultrasound is used to determine the amount of liquor and assist in diagnosing oligohydramnios, foetal malpresentation, and abnormal placentation. Foetal cells in amniotic fluid also stain Nile blue and Sudan dyes red.

In our unit, all patients with PROM at term are delivered as soon as possible by inducing labour or augmenting mild labour. Our patient was on conservative management because at the gestation of 32 weeks when she presented, foetal maturity had not been achieved. Management of PPROM takes into consideration two important two aspects directed towards foetal lung maturity and possible chorioamnionitis. Assuming that no untoward prenatal outcome occurs due to entangled or prolapsed cord or from placental abruptio after drainage, the greatest concern with prolonged rupture is the risk of maternal or foetal infection. Fortunately, most infants born after 34 weeks gestation survive due to advances in neonatal care. Thus in our set-up, we aim reaching 34 weeks before we deliver in the conservative regime management. In our patient, conservative management was instituted but abandoned when she developed a cord prolapse.
Our patient had monitoring of her pulse, foetal heart rate and abdominal tenderness white cell counts were done weekly.Conservative management of PPROM before 34 weeks is aimed at prolongation of gestation to allow foetal lung maturity, and because prematurity is the greatest risk in these fetuses, the patient is hospitalized and adviced on strict bed rest. Maternal monitoring to elucidate the earliest signs and symptoms of chorioamnionitis are put in place and include;

- Maternal pulse, temperature and foetal heart rate are taken four hourly. Rise in maternal pulse above 100/minute or foetal tachycardia above 160/minute are some of the earliest signs of chorioamnionitis. Fever is the only reliable indicator for making diagnosis where a temperature of 38°C or high4er after membrane rupture means

infection. These vital signs must be recorded for over four hours apart and the Foetal tachycardia above 160/minute for over 10 minutes.

- White blood cell counts with differential counts should be monitored every 48 hours or at least twice weekly. Maternal leucocytosis by itself alone has been found to be unreliable by most investigators. A rising trend in the value or a single value of total white cell count of over 16x 10^9/l is an important sign.
- Non stress tests should be performed weekly
- Digital examination must be avoided until she goes into labour.
- Daily abdominal examinations (palpation) checking for tenderness and daily evaluation of the leakage of liquor should be observed in terms of amounts, colour and smell of the liquor. Endocervical swab and uterine cultures should be taken at least weekly.
- Ultrasound to evaluate amniotic fluid volume, intervene in the event of severe oligohydramnios such as maximal vertical amniotic fluid pocket less than 2cm or amniotic fluid index less than 3cm.

This patient had a scan that indicated she had reduced amniotic fluid.

Conservative management was abandoned whe our patient developed cord prolapse. Conservative management is abandoned and induction of labour instituted if bacterial cultures are positive of evident signs and symptoms of clinical chorioamnionitis or severe ologohydramnios set in. If the patient is over 34 weeks one is not justified in continuing conservative management due to improved neonatal care facilities.

Our patient was started on a week's course of antibiotics and was given a dose of steroids.The use of anti-microbials, tocolytics and steroids in conservative management of PPROM remains controversial. The prevalence of positive bacterial culture in preterm PROM ranges from 14.6%- 39% 9. Antibiotics eliminate bacterial by-products involved in initiating the cascade that leads to labour.. The use of corticosteroids and antibiotics has been associated with a reduced risk of respiratory distress as compared with use of corticosteroids alone. Broad-spectrum antibiotics such as amoxycillin or erythromycin and metronidazole are given both to prevent infection and to treat any infection that may have caused the PROM. They are given for a total of not more than seven days. Tocolytics have been shown not to provide any improvement in outcomes and they might shield labour, which is one of the signs of chorioamniotis. However they may be used when it is absolutely necessary to delay deliveries for up to 48 hours to allow steroids to act in lung maturity or for the patient to be transported to a centre with adequate neonatal care facilities. Steroids can suppress the host immune response thus placing the mother and foetus at risk of infection. However the use of steroids has been found to decrease the incidence of intracranial haemorrhage, necrotising enterocolitis and respiratory distress syndrome [1,2,9,10].

Cord prolapse is one of the complications of PROM. Our patient developed cord prolapse and had to be delivered by emergency caesarean section. When this happens, the aim is to deliver the foetus as quickly as possible before hypoxia and death develops from cord compression. If the cord is pulsating, then delivery is effected by caesarean section as

was done in the patient presented. When the patient is in second stage of labour, delivery can be expedited by use of vacuum extraction[11].

Other complications of PROM include increased incidence of perinatal mortality and morbidity, prematurity, chorioamnionitis, foetal distress, thick meconium aspiration, compression syndrome brought by severe oligohydramnios which if PROM occurred before 30 weeks gestation can also cause foetal lung hypoplasia. The syndrome includes amniotic band amputations, limb and skull malformations, wrinkled leathery skin and foetal distress resulting from cord compression. Prophylactic trans-abdominal amnioinfusion with 150-350mls of warmed saline at a rate of 25-50mls per minute given weekly has been shown to decrease the compression syndrome as well as delay the onset of labour [1,2,9,10].

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OBSTETRIC CASE 8

ANTEPARTUM HAEMORRHAGE DUE TO PLACENTA PRAEVIA TYPE IV- ELECTIVE CAESAREAN SECTION AT TERM-LIVE BABY

NAME: S.M. L.M.P: 13/03/02

AGE: 30 Years E.D.D: 20.12/02

I.P NO: 0845152

D.O.A: 3/11/02 Parity: 3 + 0 Gravida 4

D.O.D: 12/12/02

PRESENTING COMPLAINTS

SM was admitted with one-day history of painless vaginal bleeding at 8 months gestation.

HISTORY OF PRESENTING COMPLAINTS

She was well until the morning of admission. About 2 hours before admission, she noted a sudden gush of vaginal bleeding which was not associated with abdominal pain, trauma, and exertion. The bleeding consisted of fresh blood and she had no dizziness. She immediately went to seek advice at Rongai health center where she had started antenatal care. She was referred to Kenyatta hospital for review. She had changed 2 moderately soaked pads by the time she arrived in our labour ward. She reported that she could still perceive foetal movements. She had not drained any liquor.

ANTENATAL CARE

She had attended antenatal care at a clinic in Rongai town once at 24weeks gestation and no antenatal profiles had been done.

OBSTETRIC AND GYNAECOLOGY HISTORY

She was para 3+0, this being her fourth pregnancy. She had her menarche at 14 years, her cycles were regular occurring every 28 days with moderate flow for three days. She had no dysmenorrhoea.

She had had three spontaneous vertex deliveries in hospital. Her first delivery was in 1992 to a female baby who weighed 3.4kg. Her second delivery was in 1996 to a female baby weighing 3.1kg. Her third delivery was in 2000 to a live male baby weighing 2.9kg.They are all alive and well.

She gave history of use of combined oral contraceptives between her deliveries. Her last menstrual period was on 13/03/04 and expected date of delivery on 20/12/02. She was at a gestation of 34 weeks at the time of admission.

PAST MEDICAL HISTORY

She had not been admitted previously, had no known allergies to drugs or foods.

FAMILY AND SOCIAL HISTORY

She was married and lived with her family in Rongai town. She was a housewife while the husband worked with KEMRI. She did not smoke cigarettes or use alcohol.

PHYSICAL EXAMINATION

Her general condition was fair. She was mildly pale, afebrile, had no jaundice. Her blood pressure was 120/70mmHg, pulse 88 beats/minute, respiratory rate 22/minute and a temperature of 36°C. She was not cyanosed and had no pedal oedema.
The cardiovascular, respiratory and nervous systems were essentially normal.

ABDOMINAL EXAMINATION

The abdomen was uniformly distended. The fundal height was 34 weeks, with longitudinal lie and cephalic presentation. The foetal heart rate was heard at 142 beats/minute and was regular. The head was five-fifths above the pelvic brim. She had no uterine contractions and had no uterine tenderness or hardness. There were no other palpable masses.

SPECULUM EXAMINATION

The external genitalia were normal, vaginal walls were healthy with no discharge or lacerations. There was slight blood oozing from the cervix that was closed and bluish in colour. A digital examination was contraindicated in this patient.

DIAGNOSIS

Antepartum haemorrhage at 34 weeks gestation

INVESTIGATIONS

1. Urgent PCV at admission was 27%- Haemoglobin approximately 9g/dl
2. Blood group A positive. 2 units of cross-matched blood were kept ready in case it became necessary to transfuse her.
3. An urgent obstetric scan was done soon after admission showed a single foetus in cephalic presentation with a low-lying placenta completely covering the internal cervical os. The estimated maturity was 34 weeks corresponding to her dates. The amniotic fluid was adequate in amounts.
4. VDRL was negative
5. ELISA for HIV was negative.

6. Haemogram Done four days after admission- haemoglobin 10.0g/dl, platelet count 309 x 10^9/l, white cell count 9.7 x 10^9/l, normocytic normochromic picture on peripheral film.

MANAGEMENT

The patient had an intravenous wide bore gauge 16 branula inserted and plasma expander put up (normal saline). She was started on haematinics (ranferon), bed rest to avoid further bleeding. She was sedated with phenobarbitone 30mg three times daily. Blood was taken for cross matching and two units were available for her. A decision was made to manage conservatively to allow further foetal maturity and deliver by elective caesarean section at 37 complete weeks in case she had no further episodes of bleeding. She was to be delivered by emergency section if she bled at any time in excessive amounts. She was given dexamethasone 12mg twice daily for one day to assist lung maturity in case early delivery had to be effected. By the second day after admission, she reported very minimal bleeding. She was to stay in hospital until after delivery.

She remained stable in the ward and at 37 complete weeks, a haemogram done indicated haemoglobin of 11g/dl, urea and electrolytes were normal. She was scheduled for elective caesarean section that was done on 8/12/02.The uterine incision was made just above the upper margin of the placenta. The membranes were ruptured and the baby extracted. The outcome was a live male baby who weighed 3.3kg and had Apgar score of 9 in one and 10 in five minutes. The placenta and membranes separated spontaneously and were then extracted. Intravenous oxytocin drip was started immediately the baby was extracted. The placental site was cleaned, inspected and had minimal oozing. Warm packs were applied on these sites. The uterus was well contracted. The uterus and abdominal wall were then closed and the patient reversed from anaesthesia. The total blood loss was about 500mls. Postoperatively, she had an indwelling catheter for fluid chart, monitoring for any vaginal bleeding was done and other vital signs were taken every 2 hours on the first day. By the second day she was started on oral sips which were graduated to light diet and finally free diet. She had also been started on crystalline penicillin (2MU 6hourly), gentamicin (80mg 8 hourly) and pethidine (100mg 8 hourly). She was later changed to oral amoxil (500mg three times daily), mefenamic acid(500mg three times daily) when she started taking orally. She did not develop any bleeding and had been put on haematinics. A haemoglobin done on the third day showed her level to be 10.2g/dl.

On the fourth day after operation, both mother and baby were well and were discharged home to be reviewed in the postnatal clinic after six weeks.

POSTNATAL FOLLOW-UP

She came to the clinic as appointed, Her wound had healed well, and the uterus was well involuted. She had not received menses and she was still breastfeeding. She was discharged through the family planning clinic for contraception where she chose to use combined oral contraceptives.

DISCUSSION

A case is presented of a 30 year old para 3+0 who presented at 34 weeks with antepartum haemorrhage due to placenta praevia type IV. She was managed conservatively until term when she was delivered by elective caesarean section and had a live delivery with good score.

Antepartum haemorrhage (APH) refers to bleeding from the female genital tract at any time after 28 weeks of pregnancy but before delivery of the baby [1,2,3]. The 28th week is arbitrary, related to legal definition on the viability of a newborn. In western countries where technology has allowed premature babies of gestational age 24 weeks or birth weight over 500g to survive, the limit of APH has been dropped to that gestation or weight in their centers [1,2]. The haemorrhage can range from mild spotting to torrential bleeding which may be fatal to both the mother and/or foetus. The source of bleeding is not always discernible but one third of pregnancies in which bleeding occur remain at increased risk for poor maternal or foetal outcome after the bleeding stops. Obstetric causes of APH are more hazardous than non-obstetric causes. The major causes include abruptio placenta and placenta praevia. Abruptio placenta refers to haemorrhage from a normally situated placenta, which has partially or completely separated from the placental bed before delivery of the baby. Placenta praevia refers to haemorrhage from partial separation of an abnormally implanted placenta in the lower segment of the uterus over or very near the internal cervical os. Other causes of APH include trauma to the genital tract, cervicitis, vaginitis, varicose veins in the genital tract, cervical erosion, cervical polyps or cancer of the cervix and abnormal blood clotting mechanism from various disorders [1,2,3]. The patient presented had APH due to placenta praevia.

This patient had type IV placenta praevia. APH is estimated to complicate 2-3% of pregnancies and among these, placenta praevia contributes 0.3-0.5%. Of all praevias, complete coverage of the internal cervical os (placenta praevia type IV) account for 20%. In the western world, maternal mortality due to obstetric haemorrhage has been reduced considerably by easy availability of well-equipped blood banks, antibiotics, expertly administered anaesthesia and caesarean section. In developing countries, obstetric haemorrhage remains a leading cause of maternal mortality. At Kenyatta National Hospital, Ojwang[4] found in 1974 found APH to occur in 2% of pregnancies and placenta praevia contributed to 0.3%. APH is responsible for up to 13% of obstetric related maternal deaths. Placenta praevia exists when the placenta is wholly or in part into the lower segment of the uterus. If it encroaches the cervical os it is considered a major or complete praevia, if not then it is minor or partial placenta praevia. This diagnosis has evolved from the original I-IV grading system and is determined by ultrasonic imaging techniques, relating to the leading edge of the placenta to the cervical os and the leading foetal pole. The four degrees of placenta praevia are:-

Type I: Also known as low-lying or lateral placenta praevia, in which the placenta extends to the lower uterine segment but does not reach the margins of the internal cervical os.

Type II Marginal placenta praevia in which the edges of the placenta are at the margin of the internal os, but do not cover it. This is further divided into type II anterior and type II posterior if the placenta is anteriorly or posteriorly implanted respectively.

Type III Also known as accentric or partial placenta praevia in which the placenta covers the internal os but is not centrally placed.

Type IV Eccentric or total placenta praevia in which the placenta completely covers the internal cervical os

Other authors divide placenta praevia into major or minor types [3,5]. Minor placenta praevia refers to the type I stated above and it accounts for 20%. Major placenta praevia refers to types II, III and IV stated above and comprises the remaining 80%. The patient presented had placenta praevia type IV which is major placenta praevia. Some patients have low lying in second trimester but toward term, the placenta is found to be located in the upper segment or has changed from complete praevia to minor degree of placenta praevia. This arises because the placenta and uterus grows with advancing gestation and the placental site is likely to be pulled further up into the upper segment bringing a change in the relation between the edge of the placenta and the internal os[1,3].

The aetiology of placenta praevia is not well known but various theories have been advanced. One of these alludes that placenta praevia may arise due to low implantation of the zygote arising from rapid embryo transport which arrives before the endometrium is receptive. Other theories advanced postulate that the endometrium of the upper segment is deficient probably from previous endometrial damage, leading to poor vascularisation with the placenta spreading over more of the uterine wall cavity in effort to obtain adequate blood supply; partial development of the placenta from the chorion laeve in relation to the deciduas capsularis. Another factor is a large placenta that spreads over a large area of the uterus as seen with foetal erythroblastosis, multiple pregnancy.

Our patient's age(30 years) and parity-3 were the only notable risk factors for developing placenta praevia.Several factors have been associated with increased incidence of placenta praevia including; multiparity, advancing maternal age, previous caesarean section delivery, previous induced abortion, cigarette smoking, history of previous placenta praevia. Singh and associates in 1981 identified placenta praevia in 3.9% of women who had undergone caesarean delivery compared to 1.9% for the whole obstetrical population. Locally, Ojwang and Kirima found placenta praevia to be commonest in the age group 20-30 years and in women with parity of 3 and above[5]. It is thought that as women age, several biological mechanisms may explain the increasing frequency of uteroplacental bleeding. These include decrease in uterine blood flow, utero-placental underperfusion and large placental infarcts among older women. Vessels at the site of prior placental attachment undergo changes that may lead to decrease in utero-placental flow to the endometrium. Consequently more surface area may be required for placental attachment in order to maintain adequate blood flow. Presumably, each pregnancy damages the underlying implantation site reducing the area suitable for

implantation and increasing the likelihood of lower uterine segment implantation. Defective decidual vascularisation, the possible result of inflammatory or atrophic changes has also been implicated in the development of placenta praevia [1].

The patient presented was admitted with painless vaginal bleeding.Characteristically, placenta praevia presents with painless vaginal bleeding mostly near the end of the second trimester or early in the third trimester. The bleeding is often sudden, unprovoked and profuse, but in 50% of cases, the first episode is rarely severe to be fatal. Bleeding may also follow minor trauma as in coitus or defecation. The cause of the bleeding is formation of the lower segment and dilation of the internal os resulting in tearing of placental attachment and bleeding. The lower segment is less muscular than the upper segment and does not contract or retract effectively to stem such bleeding. The bleeding is bright red and is lost in the vagina early. It may cease completely but subsequently recur with torrential amounts. About 10% of patients report some initial pain and cramping due to co-existing abruptio placenta. About 25% of the patients go into spontaneous labour in the next few hours or days after the haemorrhage. Although all blood loss is maternal, some foetal blood loss is possible especially if the placenta is traumatized [1,2,3]. Features that aid in diagnosis include the fact that the uterus remains soft, relaxed and non-tender. Other signs include malpresentation (unstable lie), bleeding from the cervical os on speculum examination and a high presenting part that is slightly displaced from the midline. There is usually no foetal distress unless abruptio placenta or other complications compound placenta praevia. Our patient did not have any of these features.

In our patient, placenta praevia was confirmed by transabdominal ultrasound. Ultrasonography can be used to confirm placenta praevia and has an accuracy of 98%. Transvaginal ultrasound (TVS) is the diagnostic technique of choice for praevia. It is safe and more accurate than transabdominal ultrasound (TAS) in locating the placenta [6] During the second trimester, TAS is associated with high false positive diagnosis of placenta praevia with some series reporting a low lying placenta in up to 25% of women [7]. Although third trimester scanning can improve on this, they are still associated with significant errors. False positives can be due to bladder overdistension or placental migration while false negatives may be due to large fundal placenta that is not noted to be extending to the lower segment. TVS improves on TAS accuracy. MRI has been used for diagnosis of placenta praevia where TAS images have been unsatisfactory. Mbithi in a study at KNH found an accuracy of 86.4% when ultrasound was used to confirm placenta praevia [8]. Other investigations that can be done to locate the placenta include soft tissue placentography, arteriography, infrared thermography and angiography, displacement placentography, iodine labeled albumin uptake.

Our patient was admitted at her first presentation with bleeding and remained in hospital until delivery. A careful history and physical examination with vaginal speculum examination aids in the diagnosis. The patient must be admitted (A study by Mouer[9] did not find any apparent difference in the outcome of pregnancy between patients managed conservatively as in-patients and those managed at home. Factors influencing home management include amount of initial bleed, the home situation and the distance from

hospital) and evaluated in the hospital where facilities for appropriate management including operating theatre, blood bank and expert neonatal care are at hand. An intravenous line with wide bore canula is set up and blood for group and cross-match taken. Digital vaginal and rectal examination which may provoke uncontrollable catastrophic haemorrhage must not be undertaken until placenta praevia has been ruled out. Digital examination is done in theatre when a decision to deliver the patient has been made. Blood loss is estimated by visual inspection, blood pressure, pulse rate and urinary output. Usually the visual estimate is one third of the actual loss. Hypovolaemia must always corrected. Overt hypotension and tachycardia are ominous signs of hypovolemia but the converse is not necessarily true. Urine output should be 30-60mls per hour. Blood transfusion should be with screened whole blood rather than blood components [1,3]. Our patient did not develop severe haemorrhage to cause hypotension.

The patient presented was managed conservatively; with delivery being effected at term gestation.Management of placenta praevia can either be conservative or interventional. The choice is dependent on the amount of bleeding, foetal maturity and foetal viability, foetal condition such as distress, whether the mother is in labour or not. Patients with placenta praevia can be grouped into 4 clinical categories, namely:

1. Those in whom the foetus is premature but there is no pressing need for immediate delivery. In this group conservative management is indicated and delivery is effected when lung maturity is confirmed. Hospitalisation is ideal for these cases. Arias reported outstanding results from cervical cerclage done between 24 and 30 weeks in women with bleeding caused by placenta praevia [11].
2. Those whom the foetus is reasonably mature and confirmation of foetal maturity is followed by elective delivery or emergency delivery.
3. Those in whom labour is in progress
4. Those in whom haemorrhage is so severe as to necessitate evacuation of the uterus despite foetal maturity

If the foetus is already dead, the mode of delivery will depend on the type of praevia, clinical condition of the patient and the mode of delivery with minimal adverse effects to the patient. Our patient was in category 2.

Before delivery, vaginal examination under general anaesthesia (EUA) is done under a double set-up with all preparations made for caesarean section. This vaginal examination may provoke severe bleeding thus some clinicians abstain from EUA when diagnosis of major placenta praevia is certain. The mode of delivery will depend on the parity, cervical ripeness (Bishop score), amount of bleeding, presentation, placental location and the presence of obstetric complications [1,10]. In our patient, EUA was not done as placenta praevia had been ascertained. Patients with type 1 and type II anterior, with cephalic presentation and no active bleeding or those with intrauterine foetal death can deliver vaginally if there is no other obstetric indication for caesarean section. Artificial rupture of membranes is done and labour augmented with close monitoring for bleeding and foetal well being during labour. Release of some amniotic fluid allows descent of the presenting head to cause tamponade and reduces bleeding [1,2,10]. Caesarean section is

indicated in type II posterior, types III and IV or if there is severe haemorrhage, foetal distress, malpresentation, poor labour progress, and in other obstetric risks that indicate caesarean section such as multiple pregnancy, bad obstetric history. The choice of incision is not settled yet. Some advocate for the classical incision to make it easier to avoid the placenta. Others use a low vertical incision if the placenta is anterior and the low transverse incision if the placenta is posterior. For premature cases where the lower segment is not well formed, a vertical incision is more practical, when the placenta is complicated by degrees of placenta accreta, which renders control of bleeding from the placental site difficult by conservative means, total hysterectomy may be the procedure of choice [1,2]. In our patient, the placenta separated easily and severe haemorrhage did not occur.

Neither our patient nor her baby developed any untoward complications.Placenta praevia is associated with increased maternal and foetal complications. Maternal complications are related to haemorrhage, hypovolaemic shock, sepsis and an increased rate of operative deliveries. Placenta praevia may be associated with placenta accreta, abruptio placenta and postpartum haemorrhage may be so severe that if desperate measures such as hysterectomy or ligation of iliac vessels are not done on time, death may result. A rare complication arises when placenta percreta occurs at previous uterine scar site at the lower segment with the placenta penetrating into the urinary bladder. This also leads to severe postpartum haemorrhage, haematuria and predisposes to fistula formation. Maternal mortality can be reduced by universal adequate antenatal care with identification of high-risk mothers [1,2,10].

Foetal complications include intrauterine growth retardation, intrauterine asphyxia and intrauterine foetal death, prematurity. The perinatal mortality due to prematurity as a result of APH occurs in up to 60% of cases. Ojwang found a perinatal mortality rate of 39% in his series [5]. Other complications are abnormal lie in 38.3%, cord prolapse in 1.7%, premature rupture of membranes in 11% of cases. Our patient did not have any complications.

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OBSTETRIC SHORT CASE 9

POST TERM PREGNANCY –INDUCTION OF LABOUR-SVD – LIVE BABY

NAME: R. W. L.M.P. 27.12.04

AGE: 28 Years E.D.D. 03.10.04

I.P NO: 0975861 PARITY 0 + 0

D.O.A: 22/10/04

D.O.D: 28/10/04

PRESENTING COMPLAINT

She was admitted from home with complaints of having completed her 42nd week of gestation without going into labour. Foetal movements were still normal.

ANTENATAL HISTORY

She had attended antenatal clinic at Kasarani city council clinic from 20 weeks gestation. By the time of admission she had attended six visits. The fundal height corresponded well with her dates during these visits as per the clinic card. Antenatal profiles done showed an haemoglobin of 12.1g/dl , blood group A positive, VDRL negative and HIV negative. She could not remember well the time of quickening and a scan had not been done in early pregnancy but she was sure of her dates.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She was a Para 0 + 0 gravida 1 at a gestation of 42 weeks. Her last normal menstrual period was on 27/12/03 and the expected date of delivery was on 3/10/04 .She had had irregular menses prior to this pregnancy and had been treated with oral contraceptives after which she had regular periods every 28 days for the three months before conception.

PAST MEDICAL HISTORY

She had not been admitted previously in her life and had no known chronic illnesses.

FAMILY AND SOCIAL HISTORY

She was a housewife who lived with her husband at Kasarani. Her husband was a businessman. Her mother was asthmatic on bronchodilators. There were no other chronic illnesses in the family. She neither smoked cigarettes nor drank alcohol.

PHYSICAL EXAMINATION

She was in a good general condition, was not pale, and had no jaundice, no oedema.

VITAL SIGNS

Temperature 37.2° C, blood pressure 135/85mmHg, respiratory rate 18/min and a pulse rate of 74 beats/min.

CARDIORESPIRATORY AND NERVOUS SYSTEM

Her pulse was regular, of good volume and non-collapsing. Both heart sounds were audible and normal. No murmurs were heard.
She had normal vesicular breathing in both lung fields.
Her nervous system was normal.

ABDOMEN

The abdomen was uniformly distended, the fundal height corresponded to a term pregnancy. Foetal lie was longitudinal with a cephalic presentation. The station was 4/5 above the pelvic brim. The amount of liquor was felt reduced as evidenced by more easily palpable foetal parts. Foetal heart was normal at 134 /min. There were no palpable uterine contractions.

VAGINAL EXAMINATION

She had normal external genitalia, the cervix was closed, about 2 centimetres long, posterior and thick. The pelvis was clinically adequate. The bishop score was 3, thus unfavourable for induction.

MANAGEMENT

She was admitted and a decision made to induce labour after cervical ripening. She was explained about this and consented to induction. Cervical ripening was achieved with prostaglandin E_2 3mg tablet inserted in the posterior fornix of the vagina. After eight hours of the first pessary the cervix was still closed and thick. A second pessary was inserted. After about six hours of the second insertion she complained of having mild labour like pains. She was examined and found to have a cervical dilation of 3 cm. Artificial rupture of membranes was done revealing clear liquor, no cord was felt presenting or prolapsing. Labour was augmented with an oxytocin drip (5 IU in 500mls 5% dextrose started at 10 drops/min escalated at 10 drops/min up to a maximum of 60 drops/min or until the patient was having 3 strong contractions/10 min each lasting 20 – 40 seconds) and she was monitored with use of the partogram.Hyoscine butylbromide 40mg was given when she had developed good contractions. Tramadol 100mg was given

weighing 2600g and having an Apgar score of 9 in one and 10 in ten minutes.An episiotomy was not given because the perineum felt roomy enough for her baby to pass through. She was examined after delivery and found to have no tears on the cervix or perineumThe foetal skin was dry, scaly and had long finger and toe nails. Both mother and baby remained well and were discharged on 28/10/04.

POSTNATAL REVIEW

She was well at six weeks with no complaints. She was discharged through the family planning clinic and well baby clinic. She opted to use oral contraceptives.

DISCUSSION

The patient presented was a 28 years old primigravida with prolonged pregnancy and had a successful induction of labour using prostaglandin E_2 pessary. The outcome was a live female infant with a good Apgar score and weighing 2600g.

In the case presented, the patient had completed 42 weeks and five days at the time of admission from the first day of her last menstrual period.The definition of prolonged pregnancy or postterm pregnancy was endorsed by the American College of Obstetricians and Gynaecologists (1997) as a pregnancy lasting 42 completed weeks or more from the first day of the last menstrual period [1]. This is equivalent to 294 or more days. The term post-dates is often loosely used to mean prolonged pregnancy and is better avoided. The term post-maturity should be reserved for the infant with recognizable clinical features indicating a pathologically prolonged pregnancy [1]. There are large variations in menstrual cycles in normal women and many pregnancies thought to be prolonged are actually due to inaccurate dating criteria [2]. However there is no method of identifying pregnancies that are truly prolonged and all pregnancies judged to be 42 completed weeks should be managed as if they are abnormally prolonged

In our patient, diagnosis was made from her last menstrual period, quickening and extrapolation from her antenatal record card.The reported frequency of postterm pregnancy is 3-12% and it varies with the criteria used for diagnosis. The diagnosis is made through calculation from the last normal menstrual period, early pregnancy ultrasound scan, early antenatal booking examination and follow up, quickening and obstetric scan done on encountering the patient (to estimate foetal age and the amount of liquor) [2]. When using ultrasound criteria it is necessary to take into account the margin of error reported at various times during pregnancy. Most use the biparietal diameter, femur length and abdominal circumference to calculate gestational age. Transcerebellar diameter has also been used with the diameter corresponding to weeks of gestation up to 24 weeks; thereafter charts are available to calculate gestational age. Diagnosis of prolonged can therefore be difficult given all the errors in the conventional methods used. The patient presented for example did not have an ultrasound scan during the antenatal period and dating was used from her last menstrual period. Other parameters used were time of quickening, record of examination findings at date of first clinic attendance.

Our patient was sure of the dates of her last menstrual period and no other cause of her condition could be found though she was on oral contraceptives three months prior to conception. Causes of prolonged pregnancy are unknown though an obvious cause is inaccurate dating criteria especially in those unsure of their last menstrual period or those who were on contraception. Contradictory results have been found concerning the significance of a variety of maternal demographic factors such as parity, prior postterm birth, socio-economic class and age [1]. However the tendency for some mothers to repeat post term births suggests that some prolonged pregnancies are biologically determined. Prolonged pregnancy has also been shown to recur across generations in Swedish women such that when the mother had a prolonged pregnancy in delivering her daughter, the risk for postterm pregnancy in the daughter's pregnancy was increased two to three fold [1]. Conditions associated with post term pregnancy include; anencephaly, foetal adrenal hypoplasia, absence of foetal pituitary, placental sulfatase deficiency and extrauterine pregnancy [3].

A decision was reached at to induce labour in our patient when the diagnosis of prolonged pregnancy was made.The historical basis for the concept of having an upper limit for human pregnancy duration came from the observation that perinatal mortality increased after the expected due date was passed. The postterm pregnancy is at an increased risk for

- Intrapartum asphyxia and foetal distress
- Low Apgar score
- Abnormal heart tracing with persistent late and variable decelerations
- Unexplained foetal demise
- Oligohydramnios
- Meconium aspiration syndrome
- Foetal macrosomia and cephalo-pelvic disproportion
- Labour induction and caesarean delivery
- Shoulder dystocia
- Foetal growth restriction
- Placental dysfunction

Our patient did not develop any of these complications.

Clifford (1954) described a recognizable clinical syndrome in some infants delivered typically 300 or more days after the last menstruation. This syndrome he called Postmaturity syndrome, and divided it into three stages;

Stage 1- the amniotic fluid was clear
Stage 2 – the amniotic fluid was stained green
Stage 3 – skin discoloration was yellow-green

The post mature infant presents with a unique and characteristic appearance, which includes wrinkled skin, patchy peeling skin, a long thin body suggesting wasting, advanced maturity because the infant is open-eyed and unusually alert with an old and worried look. This patient's baby had dry, scaly skin with long nail but did not have other features of advanced maturity.

Once a diagnosis of postterm pregnancy has been made, then decision must be reached at on whether to deliver or to manage the pregnancy expectantly [1,4,5]. In our unit (KNH) all post term pregnancies have labour induced. In other institutions, the first decision made is whether to deliver or not. In certain cases such as non-reassuring surveillance, oligohydramnios, growth restriction, the decision is straightforward and induction of labour is done [6,7]. The delivery versus expectant management of pregnancies between 41 and 42 weeks gestation remains controversial though recent studies show that routine induction at 41 weeks gestation does not increase the caesarean delivery rate and may decrease it, without affecting perinatal morbidity or mortality [7].

Our patient had cervical ripening with prostaglandin E_2.Once the decision to deliver has been reached, the inducibility of the cervix has to be ascertained as it has considerable impact on the management. As many as 80% of patients who reach 42 weeks gestation have an unfavourable cervical examination (Bishop score less than 7) [8]. Many options are available for cervical ripening. These include; prostaglandin E_2 tablet which is the most commonly used in our set-up, prostaglandin E_2 gel for intracervical application, a vaginal insert containing 10mg of dinoprostone. Other methods include mechanical dilation by membrane stripping or balloon catheters placed in the cervix. Extramniotic saline infusion and laminaria tent have all been studied and shown to be effective [9,10]. Misoprostol (prostaglandin E_1) and RU486 (a progesterone antagonist) have also been used.

During labour, our patient was monitored with use of the partogram. She did not develop any complications during labour. Once induction of labour has begun, it is necessary to watch out for potential complications associated with induction beyond 41 weeks gestation which include;

- Uterine hyperstimulation
- Iatrogenic prematurity
- Increased rate of caesarean section (about 2%) due to failed induction
- Cord prolapse
- Malpresentation
- Intrauterine infection
- Water intoxication
- Increased neonatal morbidity and mortality
- Uterine rupture

Intrapartum management of the postterm pregnancy includes use of electronic foetal monitoring, uterine contraction monitoring, foetal scalp pH, foetal scalp electrode monitoring, frequent auscultation and cord blood sampling. Caesarean delivery is indicated if the obstetrician cannot find reassurance that the foetus is tolerating labour well. Our patient was monitored using intermittent auscultation and partograph by a well trained mid-wife and successfully delivered a live female infant with a good score.

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OBSTETRIC CASE 10

CERVICAL INCOMPETENCE: MACDONALD STITCH LIVE DELIVERY

NAME: M.N. L.M.P: 10/09/03

AGE: 34 Years E.D.D: 17/06/04

I.P NO. 0741295 PARITY: 3 + 2 Gravida 6

	1st Admission	2nd Admission
D.O.A:	5/2/04	30/06/04
D.O.D:	7/2/04	08/07/04

First Admission

She was first seen in the antenatal care clinic when at 13 weeks gestation. She had a history of having had two previous pregnancy losses at five and three months respectively. These had started with spontaneous rupture of membranes followed by relatively painless expulsion of the foetus. She also gave a history of having had a successful term delivery after McDonald stitch insertion subsequent to the above losses. Pelvic examination revealed she had a short cervix-1cm. She was adviced to have a McDonald stitch. She was admitted when at 17 weeks gestation as this was the time she had got enough funds for admission. An obstetric scan done showed she had a single viable foetus at 18 weeks gestation. The placenta was not low lying and the liquor was adequate. The internal cervical os appeared open and the cervix was short. The stitch was inserted on 6/2/04. She had been starved the night before and was premedicated with atropine 0.6mg half hour before theatre.

In theatre, under general anaesthesia she was placed in lithotomy position and vulvo-vaginal toilet done. The urinary bladder was catheterised and clear urine drained. Draping of the vulva area with sterile drapes was then done. Examination under anaesthesia was done; the fundal height was 18 weeks gestation, the cervix was open about 1cm and had no anatomical defect. She had no drainage of liquor and no cervicitis. An Auvard speculum was then inserted to expose the cervix and both the anterior and posterior lips of the cervix held with sponge holding forceps. With gentle traction on the cervix, No.2 silk suture on a round body needle was used to insert a purse string at the level of the internal os that was identified as the point where the smooth cervical mucosa meets the rugged vaginal wall mucosa. Four bites were taken at 4o'clock coming out at 2 o'clock, 1 o'clock through 11 o'clock, 10 o'clock through 8 o'clock and 7 o'clock through 5 o'clock avoiding the vascular and nerve bundles at 3 and 9 o'clock. The knot was tied at 6 o'clock leaving about 2cm of the suture with the knot. The knot was tied tight to just allow space for the little finger. There was minimal bleeding and she was reversed from anaesthesia.

Post-operative care.

She was observed continuously until fully awake then 4 hourly. She was adviced on bed rest, and was given phenobarbitone 30mg together with salbutamol 4mg three times daily. She remained stable and on the second postoperative day was discharged for follow-up in the antenatal clinic in two weeks. She was adviced to have bed rest, avoid sexual intercourse, and avoid strenuous activity. She was also adviced to immediately seek help in hospital in case she experienced severe low abdominal pain, drained liquor, or had vaginal bleeding.
She was followed up without any of these complications and the stitch was removed at 37 complete weeks (26/5/04). She was then to await spontaneous labour and undergo trial of scar as she had a previous caesarean section.

Re-admission.

PRESENTING COMPLAINT

She was admitted with history of being passed her due date and having one previous caesarean section and a bad obstetric history.

ANTENATAL CARE

She attended antenatal care at the KNH from 3 months gestation. Her profile was as follows; blood group O positive, VDRL and HIV tests were negative, haemoglobin 13.8g/dl.
When at 17 weeks gestation, she had a McDonald stitch inserted due to cervical incompetence. She was then followed up in the clinic. Her antenatal period was uneventful. She had six visits.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She was Para 3 + 2, with two living child. Her first delivery was in 1992 by caesarean section (due to cephalopelvic disproportion) to a baby weighing 3.7kg who is alive and well. In 1998, she had premature delivery at 8 months to a stillbirth weighing 2kg. Subsequently, in 1999 and 2000, she had spontaneous abortions at 5 and 4 months respectively. They both started with drainage of liquor and followed by mild low abdominal pain (uterine contractions) then expulsion of the foetus. No uterine curettage was done in both cases. She had no trauma or febrile illness preceding both abortions. In 2001, she delivered at term after McDonald stitch insertion. The baby was delivered vaginally after successful trial of scar and weighed 3.3kg and is alive and well.
Her menarche was at 14 years; her menstrual cycles were regular occurring every 27-30 days and with flow of 3-4 days. She had no associated dysmenorrhoea. She had not used any contraceptives previously. She had no history of gynaecological surgery previously.

Her last menstrual period was on 10.09.03 and expected date of delivery on 17.06.04, hence was at 42 weeks gestation at her latest admission.

PAST MEDICAL HISTORY

She had no chronic illness and had no previous admissions.

FAMILY AND SOCIAL HISTORY

She was married and lived with her family in Nairobi. She was a housewife. She did not use alcohol or smoke cigarettes. She gave no family history of chronic illnesses.

PHYSICAL EXAMINATION

She was in good general condition, well nourished, not pale and had no jaundice or oedema. Her vital signs were as follows; blood pressure 110/60mmHg, pulse rate 71/minute, respiratory rate 20/minute and temperature 36.9^0C.
Her cardiovascular, respiratory and nervous systems were normal.

ABDOMINAL EXAMINATION

Her abdomen was uniformly distended, the fundal height was term and with longitudinal lie. She had cephalic presentation and the foetal heart tones were heard and regular at 140 beats/minute. She had no contractions.

PELVIC EXAMINATION

She had normal external genitalia, her cervix was closed but short about 1cm long. She had no discharge or bleeding. No defect was noted on the cervix.

INVESTIGATIONS

1. Haemogram – haemoglobin 13.8g/dl
   white cells count 6.4 x 10^9/l
   platelets 240 x 10^9/l

2. Urea and electrolytes – Urea 6.7mmol/l
   Na^+ 140 mmol/l
   K^+ 4.7 mmol/l
   Creatinine 91.1 umol/l

3. Obstetric scan – was done when at 17 weeks by dates. Indicated she had a single viable normal formed intrauterine gestation at 18 weeks, with the placenta not low lying and having adequate liquor. Corresponded to her being postdates at the time of the second admission

4. Blood group O positive – 2 units of cross-matched blood were kept ready for her.

DIAGNOSIS

Bad obstetric history due to cervical incompetence with one previous scar at 42 weeks gestation.

MANAGEMENT

She was readmitted and elective caesarean section was planned due to postdates with one previous caesarean scar. She consented and the caesarean section was done. The outcome was a live male infant who weighed 3900g and had Apgar score 8 in one and 10 in five minutes. Postoperative period was uneventful for both mother and baby. By the fourth day both were well, the mother's wound was dry and healing well, her uterus was well contracted and she had no calf tenderness. She was discharged home and to be reviewed in the postnatal clinic in six weeks.

FOLLOW-UP

She was well having no complaints. Her wound was well healed and the baby was well. She was discharged through the family planning clinic for contraception.

DISCUSSION

The patient presented was a para 3+2 who had had 2 previous spontaneous early second trimester abortions. Subsequently she also had a successful pregnancy after insertion of McDonald stitch. During the index pregnancy a McDonald stitch was inserted and she delivered at term by elective caesarean section due to being post-term. The outcome was a live baby who scored well and both mother and baby went home in good condition.

Our patient presented with cervical incompetence at her first admission.
Cervical incompetence is a clinical condition characterized by repeated second trimester or early third trimester pregnancy losses due to weakness and inability of the cervix to maintain the pregnancy until term gestation. It is a discrete obstetrical entity also accompanied by cervical effacement and dilation in mid trimester without uterine contractions and uterine body anomalies [1,2,3]. Palmar and Laconne first described the condition in 1948, and again by Lash and Lash in 1950 and by Shirodkar in 1983. Cervical incompetence is characterized by relatively painless dilation of the cervix in the second and early third trimester with prolapse and ballooning of membranes into the vagina followed by rupture of membranes and rapid expulsion of an immature foetus. The cervix dilates and effaces which can be a result of weakness in the muscle of the cervix that cannot be voluntarily controlled [1,3]. This patient had presented similar history in her previous pregnancies. Unless effective treatment is given this sequence of events tend to repeat in each pregnancy.

The incidence of cervical incompetence globally ranges from 0.05 to 2% of all pregnancies and it accounts for about 16% of all mid trimester abortions[1, 2]. However it is the cause of 20-25% of miscarriages in the second as well as 10% of preterm deliveries [4]. Locally Njage [5] found an incidence of 1.1% at KNH. The actual cause of cervical incompetence is still obscure and only circumstantial evidence has been used to establish the aetiology. Previous cervical trauma sustained during previous delivery, from dilation and curettage, conisation, cauterization and cervical amputation has been found to be a major aetiological factor. The occurrence of cervical incompetence in primigravida suggests congenital weakness and investigations have pointed at collagen distribution in the cervix with abundance of muscle tissue and sparse connective tissue, the opposite of distribution found in the normal cervix. In this congenital type, physical examination reveals normal cervix but sudden pregnancy loss occurs at 18-20 weeks gestation. A physiological dysfunctional disorder has also been demonstrated in some cases. Prenatal exposure to diethylstilbestrol has been associated with increased incidence of cervical incompetence in the daughters [1,2,3,8].

The patient presented had two mid trimester abortions in the classical way with drainage then uterine contraction and expulsion and were of decreasing gestation. The classical presentation is history of recurrent mid trimester pregnancy losses that starts with painless drainage of liquor, then uterine contraction and finally expulsion of the products of conception. The gestational ages decrease with subsequent pregnancies.
Previous history of abortion especially if managed by dilation and curettage forms a significant aetiological association as in Kagia's series where a third of the cases of

cervical incompetence had prior abortion [5]. History of previous preterm births, cervical tears and repair, dilation and curettage, cone biopsies is collaborative. On examination cervical defects suggesting previous cervical trauma may be found. A very short cervix less than 2.5cm in length with dilation or bulging membranes may be observed. However in practice, some patients present with symptoms ranging from urinary frequency, lower abdominal pressure, a sensation of bearing down and bloody show or a watery discharge. Additionally menstrual like cramps and a pattern of uterine irritability or small amplitude contractions may occur as the membranes protrude, distend the cervix and activate the Ferguson's reflex [6]. In our patient, a short cervix was found but there were no bulging membranes. She did not have any vaginal discharge.

In the patient presented, the diagnosis of cervical incompetence was made from her clinical presentation and she had not had any tests previously to make this diagnosis. History and physical examination may be obscure such that confirmation of the diagnosis cannot be reliably made. Several diagnostic techniques have been employed in effort to make a diagnosis. Outside pregnancy, an incompetent cervix will easily allow passage of size 8 Hegar's dilator. Traction test of Bergman and Sverrund may be performed where a balloon catheter placed in the uterine cavity with an external traction of 600g is positive indicating incompetence if the balloon falls out. A hysterosalpingogram at antero-posterior view with the cervix pulled down may demonstrate funneling and a shortened endocervical canal [1,2,3,5,8]. In pregnancy, transvaginal ultrasound done at 14-30 weeks may demonstrate a short cervix with funneling of the internal os and protrusion of chorioamniotic membranes into the endocervical canal. Our patient had not had any of these tests.

Baden and Baden in 1960 suggested a grading system in which the severity of cervical incompetence can be shown [6].These were;
Grade I (mildest form)-premature delivery occurs at 30-36 weeks gestation
Grade II (moderate) Delivery occurs between 24 and 30 weeks gestation
Grade III (Severe)- abortion occurs between 18-24 weeks.
Grade IV (Very severe)-abortion occurs before 18 weeks.

They found 100% term pregnancy rates outcomes with cerclage of grade I and II compared to 25% for grade III and IV. Our patient had losses at gestation of up to five months thus could be placed in grade III. She attained term pregnancy after cerclage.

Treatment of cervical incompetence in pregnancy involves strengthening of the weak uterocervical junction achieved by various cerclage techniques. They all involve passing some kind of purse string suture around the internal cervical os. The commonest cerclage method is the McDonald stitch technique, which involves application of non-absorbable purse string submucosally at the level of the internal cervical os. It is technically simple and less traumatic than both the conventional and modified Shirodkar methods in application and removal. It does not require mucosal dissection unlike the Shirodkar. Howevere both have similar success rates of 85-90% foetal survival [1,7]. The placement of Shirodkar suture may be done using a strip of fascia lata or mersilene tape tied submuscularly around the isthmus of the cervix after vaginal mucosa and bladder have

been reflected. The suture is difficult to remove often leading to caesarean section as mode of delivery, which may be complicated by lochia outflow obstruction if the suture is left in situ. The conventional Shirodkar technique requires laparotomy for insertion while the modified one uses the transvaginal route with double needle ligature instead of mersilene tape allowing it to be performed even during pregnancy [1,3,7].

Other methods used to strengthen the cervix include

- The Lash procedure; requires laparotomy to perform and often requires caesarean section delivery like the conventional Shirodkar technique.
- Trachelorrhaphy
- Tracheloplasty

Both trachelorraphy and tracheloplasty have been associated with increased incidence of complications including cervical scarring and stenosis, which interfere with sperm transport to cause infertility. These three methods have fallen out of favour in preference to McDonald stitch and the modified Shirodkar that is used for cases where the McDonald stitch has failed. [1,3,7].

Cervical cerclage is best performed between 14th to 19th weeks of gestation by dates and before a dilation of 3cm is reached. After 19 weeks cerclage is technically difficult due to effacement of the cervix and is associated with increased complication rates especially preterm labour, rupture of membranes and chorioamnionitis. Cerclage is not done in the first trimester due to several reasons, namely

a) Ultrasound can be done to confirm viability and rule out major congenital anomalies in the foetus

b) First trimester causes of abortions (especially due to chromosomal abnormalities) are avoided

c) It has also been noted that before 15 weeks gestation, the amniotic fluid pressure and the weight of the products of conception exerted on the cervix are not able to cause abortion by mechanical means [1,3].

Our patient had McDonald stitch insertion at 17 weeks after a scan showed a normally growing foetus. She remained well until the time of delivery at term.

Results of cervical cerclage are difficult to assess because diagnosis of incompetence of the cervix may not be certain in every case. Worldwide rates of success are 85-90% with respect to foetal survival. MacDonald himself reported term pregnancy rates of 85.5% while Shirodkar reported 70-85% success. Locally Njagi reported 64% foetal survival rates and 53% term pregnancy rate in 1978. Ruminjo [10] reported 69.5% term pregnancy rates and 78.1 foetal survival rates in 1991. Block and Rahhal (1976)[9] proposed a scoring system, which was based on clinical and historical data of the patient to ascertain the diagnosis and the likely prognosis. Each of the five parameters considered scores one point if present and thus giving a maximum of five points and minimum of zero. The parameters considered include:

1. Previous premature delivery and mid trimester abortion without obvious cause.
2. History of painless premature labour and delivery.
3. Visual evidence of previous surgical or obstetric trauma to the cervix
4. Progressive dilatation greater than 2cm on initial examination during second trimester

5. Previous diagnosis of cervical incompetence with previous cerclage
A score of 3 or more points has statistically more successful outcome with cerclage. Our patient had a score of 3 and had a successful outcome after cerclage.

The patient presented did not have any contraindications to the insertion of the stitch. Contraindications to cerclage include uterine bleeding, uterine contractions, ruptured membranes, polyhydramnios, intra uterine foetal death, cervicitis and obvious foetal malformations such as hydrocephalous, anencephaly [1,3,7].
Before cerclage, foetal viability should be confirmed by ultrasound as was doen for the patient presented, which also rules out congenital malformations, multiple pregnancy and polyhydramnios. Other causes of abortion should be ruled out thus tests performed for diabetes mellitus, rhesus incompatibility, thyroid disease, uterine fibroids, syphilis, brucellosis, toxoplasmosis and HIV. Where possible a Pap smear and investigations to rule out autoimmune disease should be done [1,3,7]. In our patient, tests were done for HIV and syphilis, which were negative.

In our patient the stitch was removed at 37 complete weeks as she was term and had no complications during follow up.The McDonald stitch is removed after 37 completed week's gestation or at any time the patient presents in labour, has ruptured membranes, has uterine bleeding or intrauterine foetal death or she develops chorioamnionitis. Removal of the stitch would permit vaginal delivery. It can be left in situ if delivery will be by caesarean section.
Complications of cerclage include haemorrhage, rupture of membranes, chorioamnionitis, abscess formation, formation of haematomas and disseminated infection including septicaemia. Fibrosis of the cervix after cerclage may cause cervical dystocia during labour. If labour starts with the stitch in situ, cervical tears and amputation, vesicovaginal fistula and uterine rupture can occur. The inflammatory process in the cervix following cerclage may induce preterm labour.

This patient had one previous caesarean section scar in addition to cervical incompetence. She had had one successful birth after the caesarean section and had been scheduled for trial of scar in the index pregnancy. She was eventually delivered by caesarean section for having passed the due date by two weeks. In our unit, induction of labour and use of oxytocin are not done in patients with previous caesarean section scars due to lack of proper monitoring equipment such as pressure monitors and oxytocin pumps. Successful vaginal birth after caesarean section would have advantages including less operative and postoperative complications, shorter hospital stay and lower postpartum morbidity. The success rate of VBAC is approximately 75% though actual rates vary from centre to centre. Success also depends on other factors such as reason for primary caesarean section, history of previous vaginal births and the number of previous caesarean section scars[11]. Our patient had repeat caesarean section due to also having prolonged pregnancy. Prolonged pregnancy is one which is judged to be 42 completed weeks of gestation. The reported frequency is 3-12% and varies with the criteria used for diagnosi. Induction of labour at 41+ weeks significantly reduces the perinatal mortality rate and possibly the caesarean section rate. Nevertheless, some opt for conservative management with foetal surveillance[12].

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OBSTETRIC CASE 11

VAGINAL BIRTH AFTER CAESAREAN SECTION: SUCCESSFUL TRIAL OF SCAR- LIVE BABY

NAME: S.M. L.M.P: 6/10/03

AGE: 32 Years E.D.D: 13/7/04

I.P NO: 0969953 Parity: 1 + 0 Gravida 2

D.O.A: 8/7/04

D.O.D: 9/7/04

PRESENTING COMPLAINT

SM was admitted complaining of lower abdominal pains and backache for seven hours prior to admission.

HISTORY OF PRESENTING COMPLAINTS

She had low abdominal pains that started seven hours prior to arrival, were increasing in intensity radiated to the back. She had not drained any liquor and did not have any vaginal bleeding. The patient delayed arriving in hospital due to problems with transport from her home.

ANTENATAL CARE

She attended antenatal clinic at KNH from 27 weeks gestation. She was given two tetanus toxoid doses four weeks apart. The antenatal period was uneventful. She had six visits in total. Her antenatal profile done was as follows; blood group B positive, haemoglobin 11.1g/dl, HIV and VDRL tests were negative.At 36 weeks gestation, she had a pelvic examination. The sacral promontory was not easily tipped, the sacral curve was convex and the ischial spines were not prominent. The outlet could easily accommodate five knuckles. She had a clinically adequate pelvis. In addition, an erect lateral pelvimetry was done which showed an inlet of 11cm, midcavity 13.5cm and outlet 12.5cm. With these, she was adviced that a vaginal delivery could be allowed as she had a non recurring indication from previous c/section; but that there is a small risk (1%) of the uterus giving way and caesarean section may also be done in case of other indications while in labour. She was to get to hospital as soon as labour started.

OBSTETRIC AND GYNAECOLOGIC HISTORY

She was a Para 1+0 gravida 2, her last delivery being in 2000 at the KNH by caesarean section to a male baby who weighed 4kg and is alive and well. Her last menstrual period was on 6.10.03 and the expected date of delivery on 13.7.04, hence was at 39 weeks and two days gestation at admission.
She had attained menarche at 14 years, her cycles were regular occurring every 28 days and lasting 3-4 days with normal flow. She had no dysmenorrhoea. She had used Depo-Provera injection for two years after her first delivery.

PAST MEDICAL HISTORY

She had not been admitted before and had no known allergies to foods or drugs.

FAMILY AND SOCIAL HISTORY

SM was married and lived with her family in Nairobi's Eastlands. She was a housewife while the husband was a graphic designer. She did not use alcohol or cigarettes. She had no family history of chronic illnesses.

PHYSICAL EXAMINATION

She was in good general condition, was not pale, and had no jaundice or oedema. Her vital signs were as follows; blood pressure 110/60mmHg, respiratory rate 19/minute, pulse 80/minute and temperature 36.8^{0}C. Her cardiovascular, respiratory and nervous systems were normal.

ABDOMINAL EXAMINATION

She had a sub-umbilical midline scar from the previous surgery, and the abdomen was uniformly distended. The fundal height was term with a longitudinal lie and cephalic presentation. The presenting part was two fifths above the pelvic brim and the foetal heart tones were heard regular with a rate of 144beats/minute. She was having 3 strong contractions lasting 40 seconds every ten minutes. There was no tenderness in the lower uterine segment. The estimated foetal weight was 3500g.

PELVIC EXAMINATION

She had normal external genitalia, the cervix was 8 cm dilated and fully effaced. The membranes were bulging and artificial rupture (after ascertaining there was no cord presentation) revealed clear liquor. She had no moulding or caput on the presenting foetal head. Her pelvis was clinically adequate as the sacral promontory was not tipped, the ischial spines were not prominent and the outlet sub-pubic angle was obtuse.

INVESTIGATIONS

1. Blood group B positive

2. Haemogram haemoglobin 11g/dl
White cell counts 10.4 x 10^9/l
Platelets 342 x 10^9/l

DIAGNOSIS

Active labour in a patient with one previous caesarean scar for a non-recurring cause, and with adequate pelvis.

MANAGEMENT

She was scheduled for trial of scar.
Vital signs observations were confirmed normal again. She was informed of the mode of management and readily agreed as this had been explained well to her during antenatal follow-up. All the same consent was taken for operative delivery in case this became necessary. She was asked to press the alarm bell if she felt unusual pain at the lower abdomen on the scar site. She was then started on a partogram with strict one to one nursing to monitor her progress. She was started on an intravenous drip of 10% dextrose. A blood sample for cross matching was taken and she was to be reviewed in two hours time. She was asked to lie in the left lateral position. Blood pressure taken twice in labour was 100/60 and 110/70mmHg; pulse rates recorded were 76 and 82/minute.

She progressed well; about one and a half hours later she reported a feeling of bearing down. She was examined and found to be in second stage of labour and wheeled to the delivery room. She delivered by spontaneous vertex delivery, a live female baby who weighed 3600g and had Apgar score of 10 in one and 10 in five minutes. No episiotomy was required as the outlet felt roomy enough for easy delivery. The placenta was delivered by controlled cord traction and noted to be complete. She was given 0.5mg of ergometrine. The cervix, vaginal walls and perineum were examined and found to be intact only with very minimal bruising that was not bleeding. The uterus was well contracted and its cavity was not explored; rather she was to be observed for any excessive vaginal bleeding and signs of shock. Blood loss was estimated at 250mls.

One hour post delivery her blood pressure was 110/60mmHg, pulse 80/minute respiratory 20/minute and after four hours was discharged to the postnatal ward. All observations were normal over this period and no excess vaginal bleeding was evident.

Post-natal she remained stable, initiated breastfeeding and was discharged home on the second postnatal day. She was to be reviewed in the postnatal clinic after six weeks.

FOLLOW-UP

She did not honour her appointment.

DISCUSSION

The case presented was of a Para 1+0 who had previous caesarean section for a non-recurring indication-foetal macrosomia. She was admitted in active labour and had a successful trial of scar with a good outcome live baby. She was well postnatal and was discharged on the second day after delivery.

Our patient had successfu vaginal birth after caesarean section (VBAC). VBAC, also known as trial of scar (TOS) is the delivery of a baby by the vaginal route in a patient who has had previous delivery by caesarean section. It is considered successful if vaginal delivery is achieved and failed when a repeat caesarean section is done. Caesarean section rates continue to rise the world over. The reported incidence of caesarean section at KNH in 1980 was 17.8% with 59% of the cases being repeat sections[1]. In the USA the rate is up to 24% and about 30% are repeat caesarean deliveries[2,3]. At KNH, a success rate of VBAC is reported to be about 53.1%[4]. In the USA, the number of women receiving a trial of scar increased from 7% of in 1985 to 18.5% in 1989[5]. Current evidence suggests that up to 70% of women can achieve vaginal delivery following a previous caesarean section[6].

The increasing caesarean section rates have been attributed to various factors; use of electronic monitoring which detects even the slightest foetal distress, inadequate management of labour, concerns about malpractice litigation and elective repeat caesarean sections for those with one previous caesarean scar. Walton[3] found previous scar as the commonest indication for elective caesarean section in 53.3% of all sections followed by contracted pelvis in 37.4% of cases in our local set-up.

Our patient had prior section due to foetal macrosomia. The success rates for VBAC when all indications for the primary operation is considered is approximately 75%, approaching in many hospitals the likelihood of a vaginal birth for a primigravida though actual rates vary from centre to centre (67-85%) [7,8,9,10]. Success in trial is influenced by various factors that include; indication for primary caesarean section, history of previous vaginal delivery, number of previous caesarean sections and previous maternal and foetal outcomes. Success rate is highest if the previous scar was due to breech presentation and lowest if it was due to cephalopelvic disproportion (CPD). However recent literature indicates that diagnosis of CPD has no prognostic value from one pregnancy to the next and generally should not exclude a patient from trial of scar[11]. A woman who has had vaginal delivery at least once before or after caesarean section is more likely to have a successful trial of scar than one who has not had any vaginal delivery[12]. Our patient had not delivered vaginally before.

The criteria used at KNH for patient selection to undergo trial of scar initially set by Walton include[3]:

1. The patient should have a history of only one previous caesarean section, which was lower uterine segment incision.
2. The patient should have no adverse medical or obstetric conditions such as hypertension, diabetes, breech presentation, multiple pregnancy, bad obstetric history.
3. Non recurrent indication for the primary caesarean section such as foetaldistress,breech
4. Clinical and radiological pelvimetry should show adequacy of the pelvis as at 36

week's gestation. The true conjugate must be at least 10.5cm in erect lateral pelvimetry
5. The estimated foetal weight should not exceed 3500grams and the foetus should be in cephalic presentation.

Our patient satisfied all these criteria and was scheduled for trial of scar.
Elsewhere criteria includes; patient agreeing to the procedure, low transverse uterine incision was used, primary caesarean indication was non-recurring in subsequent pregnancies, benign postoperative course (non-septic), index pregnancy not complicated by macrosomia, malposition, multiple gestation[13].

The patient presented had previous scar due to macrosomia, and ELP done at 36 weeks showed an adequate pelvis. X-ray pelvimetry is of value in a patient with contracted pelvis to prevent them from unnecessary trial of scar. However radiological assessment of the pelvis should not be absolute indication. The measurement does not assess foetal size and presentation as it is a static examination, which does not take into account the changes in the pelvic and foetal dimensions during labour, it should not be used to solely to offer repeat caesarean section unless there is obvious pelvic inadequacy [14,15].

During labour, our patient had an intravenous line for fluid administration and a blood sample was taken for cross-matching. Vital signs were monitored closely during labour. The key to successful trial of scar is good intrapartum monitoring in a centre capable of performing caesarean section and managing possible uterine rupture. Management of labour should be according to a set protocol. A large bore intravenous canulla should be in place, and cross-matched blood must be available in case haemorrhage necessitating transfusion occurs[13, 16]. The use of oxytocin for induction or augmentation of labour is controversial. Judicious use in is not contraindicated provided the unit has facilities for very close monitoring including that for intrauterine pressure monitors and electronic foetal heart rate monitors. The use of intrauterine monitors is more sensitive for detection of impending uterine rupture than the sensation of pain or abdominal tenderness. Due to lack of monitoring facilities oxytocin is not used in our unit for induction or augmentation of labour in trial of scars. The use of analgesia remains controversial. Of concern is that analgesics may mask uterine rupture. It is now agreed that analgesia is not contraindicated provided proper monitoring and care is available [13,17]. Oxytocin was not used in our patient as there was no indication for its use.

Our patient did not develop any complications and did not have any exploration done.The most feared complication associated with trial of scar is scar dehiscence and subsequent uterine rupture. Scar dehiscence is the unsuspected and undiagnosed silent separation of a uterine scar from a previous caesarean section and is usually limited to the immediate area of the scar. Rupture is the sudden separation of the scar, usually with haemorrhage and the lacerations may be extensive. A rupture is complete if it communicates with the peritoneal cavity and incomplete if the visceral peritoneum is intact. Signs associated with rupture include abdominal tenderness, rising pulse rate of over 100/minute vaginal bleeding, pallor and hypotension. Pulse rate is the best indicator for impending rupture and should be taken quarter hourly [13,17]. In an attempt to discern scar integrity following successful VBAC, the practice of postpartum scar exploration has been advocated. If

dehiscence is found and does not open into the peritoneal cavity and it is small with minimal bleeding, repair is probably not necessary. Some authours are against uterine exploration since if it is a small tear one is bound to increase its depth with exploration, and also increase the risk of endometritis. Most now suggest constant observation of the patients haemodynamics and for severe per vaginal bleeding than exploration. If bleeding ensues and dynamics change then laparotomy has to be done. Vital signs remained normal in our patient.

Contraindications for a trial of scar include previous classical caesarean section scar, T-shaped scar as absolute contraindications. Foetal macrosomia (mild) does not highly increase the risks during TOS. Some authors now feel that the occurrence of 2 prior caesarean scars should no longer be an absolute contraindication [13]. Overall VBAC should be encouraged in cases satisfying trial criteria. Our patient had no contraindication to vaginal birth after caesarean section which was successful.

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OBSTETRIC CASE 12

MULTIPLE GESTATION: LIVE VAGINAL DELIVERY

NAME:	L.L	L.M.P:	29/01/04
AGE:	35 Years	E.D.D:	05/11/04
I.P NO:	0988176	PARITY	2 + 0 Gravida 3
D.O.A:	12.10.04		
D.O.D:	14.10.04		

PRESENTING COMPLAINT

LL presented to labour ward with a history of lower abdominal pain and drainage of liquor for three hours prior to arrival in hospital.

HISTORY OF PRESENTING COMPLAINT

She was well until the day of admission when she suddenly noted that her clothing had become wet and followed by low abdominal pain soon after. She noted the amount of liquor was increasing and was associated with backache; the abdominal pain was also increasing in intensity and frequency. She could appreciate foetal movements. It was then that she decided to seek advice in hospital, as she had not yet reached her due date of delivery. She had not experienced any vaginal bleeding. She delayed arrival in hospital due to lack of transport from her home to KNH.

ANTENATAL CARE

She had been attending antenatal clinic from 30 weeks gestation at the KNH. Her profile done found her blood group to be O positive, her haemoglobin 11.1g/dl, and her HIV and VDRL tests were negative. Her last menstrual period was on 29.01.04 and her expected date of delivery on 5.11.04, therefore being at a gestation of 36 weeks and 5 days at the time of admission. She had been treated once for vaginal candidiasis, and had also been given haematinics (ranferon). Her fundal height had been noted to be larger than the dates during her visits and an obstetric scan was done to confirm suspected multiple gestation (Done on 14/7/04 revealed triplet gestation with mean maturity of 25 weeks. Cardiac activities of all fetuses were normal, the liquor was adequate and there was a posterior placenta. By extrapolation she was at 37 weeks gestation corresponding with her dates).

OBSTETRIC AND GYNAECOLOGICAL HISTORY

LL attained her menarche at the age of 14 years, had regular periods occurring every 21 days and lasting 3-4 days. She had no dysmenorrhoea or menorrhagia. She was a Para 2+0, now gravida three. Her first pregnancy was in 1994 at term to a female infant who weighed 3.2kg. In 1997 she had her second delivery at term to another female infant who also weighed 3.2kg. Both children were born in hospital and were alive and well. She had used oral contraceptive pills between her first and second pregnancy. After the second pregnancy she was on Norplant implant for five years. No use of fertility drugs.

PAST MEDICAL HISTORY

She had never been admitted to hospital before, had no allergies or chronic illnesses.

FAMILY AND SOCIAL HISTORY

She was a married lady who lived with her family in Nairobi. She and her husband worked in a disinfectant manufacturing company. She did not have any history of chronic illnesses in her family. There was no family history of twinning, hypertension or diabetes.

PHYSICAL EXAMINATION

She was in a good general condition and of good nutritional status. She was not pale, not jaundiced and had mild pedal oedema. Her vital signs taken found her blood pressure to be 120/70mmHg, her pulse 70 beats/minute, respiratory rate 20 breaths/minute and her temperature was 37.2^{O} C. .Her central nervous, respiratory and cardiovascular systems were essentially normal on examination.

ABDOMINAL EXAMINATION

Her abdomen was grossly distended. She had a fundal height that corresponded to a term gestation. There were multiple foetal parts felt on her abdomen and the first triplet was in cephalic presentation. The lie was longitudinal and two distinct foetal heart tones were heard. She had three strong contractions every ten minutes each lasting 20-40 seconds.

PELVIC EXAMINATION

She had normal external genitalia. The cervix was 8cm dilated, soft and well effaced. She was draining clear liquor and the head of the first baby was felt with no caput or moulding.

INVESTIGATIONS

1. Haemoglobin 11.1g/dl

2. Blood group O positive

DIAGNOSIS

Triplet gestation at 36 weeks 5 days in preterm labour.

MANAGEMENT

LL was admitted into labour ward, and was to be delivered by emergency caesarean section to which she had accepted as she had been counselled during her antenatal visits. However as she was being prepared for theatre, she complained of an urge to defecate and a pelvic examination revealed that the first foetus was crowning at the vaginal introitus. She was immediately taken to the delivery room, the perineum draped and the first baby delivered in vertex position, being a female with an apgar score of 8 in one and 10 in five minutes and weighed 2.45kg. Episiotomy was not given as her outlet was roomy enough to allow spontaneous delivery. She was re-examined and both remaining fetuses found to be in longitudinal lie and both in breech presentation. The foetal hearts were normal. The second triplet was felt to have engaged with 2/5 part felt above the pelvic brim. On vaginal examination, membranes were bulging the cervic was fully dilated. The umbilical cord was not felt within the membranes. Artificial rupture of the membranes was done and she was encouraged to bear down with each contraction. Spontaneous delivery was allowed until the umbilicus was visible when assisted breech delivery done. Flexion of the knee by pressure on the popliteal fossa accompanied by abduction of the thigh was used to deliver both legs, each at a time. The legs were supported with a green towel. A loop of cord was brought down to ensure there was no undue traction on the cord. The rest of the abdomen emerged with minimal assistance. The foetus was allowed to hang from the perineum until the scapula was seen. The upper limbs were delivered by flexing the elbow and sweeping it across the body. The foetus was then allowed to hang from the vulva for a few seconds until the nape of the neck was visible. The foetal legs were then used to gently raise its body vertically above towards the mother's abdomen and the head was easily delivered. The perineum was supported to prevent trauma. The baby's face was wiped gently, the umbilical cord double clamped, ligated and cut and the baby covered and taken to the resuscitaire. The second baby weighed 2.05kg and scored 7 in one and 10 in five minutes.She was still having strong contractions. On palpation of the abdomen, the third triplet was found to be in longitudinal lie and breech presentation. Membranes were artificially ruptured and assisted breech delivery was done as for the second twin. The third triplet weighed 2.05kg and had a score of 7 in one and 8 in five minutes. They all had clear liquor. The third stage of labour was managed actively with the placenta being delivered by controlled cord traction. There was one huge placenta suggesting monochorionic gestation. Each triplet was in its own amniotic sac. An oxytocin drip had also been started to prevent post partum haemorrhage. Blood loss at delivery was about 300mls. The uterus remained well contracted. Her vital signs after delivery were; blood pressure110/70mmHg, pulse 74/minute.

She was taken to the postnatal ward where she remained stable, initiated breastfeeding well. Having delivered triplets, she was adviced to also have formula milk to supplement feeding of the babies. She was told to also make sure each baby has a chance for breastmilk by ensuring, preferably at each feed one baby who had not been first at the last feeding sessionwas now to be first. She was also to get extra help at home for assistance in feeding the babies. While in the ward assistance was offered by the nursing staff on duty. She was started on haematinics (ranferon 10mls daily) and was adviced to use this for at least six weeks. Nutritional advice was also offered. She was subsequently allowed home 48 hours after delivery.

POSTNATAL FOLLOW-UP

She was well when she returned to the clinic after six weeks. Her infants were breastfeeding well and she also was supplementing their feeding. She was discharged to the family planning clinic where she opted for bilateral tubal ligation.

DISCUSSION

The case presented is of a 35 years old para 2+0 with triplet gestation who ended having a successful vaginal delivery at 36 weeks gestation to three female infants with good Apgar scores.

Multiple pregnancy is the term used when more than one foetus is carried in the uterus at the same time. The incidence of twin and higher order multiple births has increased significantly over the last 15 years primarily due to the availability and increased use of ovulation inducing drugs and newly developed assisted reproductive technologies such as in-vitro fertilization [1,2].

Our patient had not used any fertility enhancing drugs.Twin pregnancy produced by fertilization of a single ovum that subsequently divides spontaneously is referred to as monozygotic or identical twins also sometimes called true twins. Monozygotic triplets result from repeated twinning of a single ovum. Twins produced from fertilization of two separate ova by two separate sperms are referred to as dizygotic or fraternal twins [3,4]. Monozygotic twins are genetically identical and have the same sex. Dizygotic twins are two different individuals. The outcome of the twinning process depends on when division occurs;

- If division occurs before the inner cell mass forms and the differentiation of the trophoblast (that is the first 72 hours), two chorions and two amnions will form resulting in dichorionic diamniotic monozygotic gestation.
- Division between the fourth and eighth day after the inner cell mass is formed but before formation of the amnion, the result is a common chorion and two amnions-diamniotioc, monochorionic gestation.
- Division after differentiation of the amnion-day 8to 13- results in a single placenta one chorion and one amnion- a monochorionic monoamnionic gestation
- Division later than day 13-15 may result in conjoined twins due to incomplete cleavage

Other forms of multiple gestation include superfecundation- the fertilization of two ova released at about the same time by sperm released at intercourse on two different occasions; and superfetation- the fertilization of two ova released in different menstrual cycles.

Our patient had triplet pregnancy. The incidence of triplets is approximately 1:6400. Monozygotic triplets occurence is random and occurs by chance. It results from repeated twinning of a single ovum. In contrast the incidence of naturally occurring dizygotic twins varies and is affected by maternal race, parity, age, nutritional status and height. This may be related to higher baseline levels of gonadotrophins in certain women groups [2,3,5]. One third of twins are monozygotic and they occur at a constant rate of 2.4-4 per 1000 deliveries in all races [5].The incidence of multiple pregnancy varies with racial characteristics. Those of Black race have higher twinning rates than the Caucasians and the Oriental people have the lowest rates [3,5]. The highest reported incidence of twinning is 1 in 22 deliveries found in Nigeria [6]. Locally Oyieke (1978) found an incidence of 1:58.8 deliveries at the Kenyatta national hospital (KNH) [7] while Mutungi found an incidence of 1:46 deliveries at the KNH and Pumwani maternity hospital [8].

Our patient did not develop any antenatal complications but the babies birth weight were on the lower normal scale.Multiple pregnancy is associated with an increased number of complications, both maternal and foetal which are of clinical importance. These include pregnancy induced or aggravated hypertension, maternal anaemia, preterm labour, premature rupture of membranes, malpresentations, cord accidents, postpartum haemorrhage, placenta praevia due too huge placenta, high perinatal morbidity and mortality, low birth weights and a high risk of operative delivery [1-8]. Perinatal morbidity and mortality rate is 3-4 times that of singleton pregnancies. Almost 15% of triplets deliver before 30 weeks gestation. Perinatal mortality rates are 37, 52 and 231 per 1000 births for twins, triplets and higher order births respectively [2,3,4,7,8]. Oyieke in his series reported a perinatal mortality rate of 22.9%, which he attributed largely to premature deliveries and low birth weight. Spontaneous abortion is more likely with multiple pregnancy. The incidence of congenital malformations is appreciably increased in twin and higher multiple gestations compared to singleton. Major malformations occur in 2% and minor malformations in 4%. Other unique complications found in multiple pregnancy includes conjoined twins, acardiac twins, twin-twin transfusion and those of the central nervous system.

Our patient was diagnosed to have multiple gestations after clinical suspicion because of between her dates and a larger fundal height and a scan confirmed the diagnosis.There are various aids to the diagnosis of multiple pregnancy. A maternal family history of twins, older maternal age, higher parity, larger maternal size and a previous history of twins provide weak clues, but knowledge of recent administration of clomiphene or gonadotrophins or pregnancy accomplished by assisted reproductive technology provide strong clues. The most common clinical signs or symptoms suggestive of multiple pregnancy include uterine size that is greater than the expected for dates, hyperemesis gravidarum, auscultation of two or more foetal heart rates, accelerated maternal weight gain and palpation of multiple foetal parts. Higher levels of biochemical pregnancy markers such as p-HCG, progesterone, oestriol, maternal serum alpha feto-protein than in singleton pregnancies can also be suggestive of multiple pregnancy, although none are specific enough to make the diagnosis of more than one foetus without ultrasonography [2]. Ultrasonography is usually used to confirm the diagnosis. In a number of occasions the diagnosis is missed altogether. Mutungi reported that the correct diagnosis was made in only 40.8% of cases antenatally while 44.1% were diagnosed either in labour or after delivery of the first twin. The diagnosis was completely missed in 54.9% of her patients[8]. Ultrasound can diagnose multiple gestations from as early as 5-6 weeks gestation by identification of multiple gestational sacs. Foetal cardiac activity can be identified between 6-7 weeks [2]. Biochemical tests were not done in our patient for diagnosis as the tests are not routinely done in our unit.

Early diagnosis and proper antenatal care is vital in the management of multiple gestation. Special consideration should be paid to nutritional requirements. It is recommended that dietary intake in multiple gestations be increased daily by approximately 300kcal above that for a singleton pregnancy [1]. Supplemental elemental iron 60-80mg and folate 1mg daily are also recommended [2]. These are given in our unit as a routine. Bed rest is important in reducing preterm delivery when started at 28-32

weeks of gestation. Bed rest also improves uteroplacental perfusion which increases birth weights. Routine administration of tocolytics and prophylactic cervical cerclage placement has been shown to be of little benefit [1,2,3,10].

All the triplets in our patient were normal. She had not had early pregnancy obstetric scan to check whether there were extra gestations at that time.Early ultrasound has shown higher incidence of twins and multiple gestations than actually found at delivery. This has been explained by the fact that some of the fetuses get resorbed (Vanishing twin). Foetal deaths as late as the end of the first trimester can be followed by complete foetal resorption, leaving no gross evidence at delivery that twins ever existed. The other foetus may also become mummified (foetus papyraceous) [3,4,5,11].

Intrauterine death of one twin with continuation of the pregnancy is a rare complication of multiple pregnancy. It is associated with increased morbidity and mortality for the surviving twin especially in monochorionic multiple gestations. Maternal consumptive coagulopathy with hypofibrinogenaemia may occasionally develop leading to troublesome bleeding [1,12]. Our patient did not have this complication.

The patient presented was scheduled for elective caesarean section at term but was admitted days prior to this in active labour. The elective delivery of higher order pregnancies, which is almost always by caesarean section, can be justified slightly earlier at 37 weeks. Ideally women with multiple pregnancy (twins) should undergo delivery at 40 weeks of gestation and if elective delivery is considered before 38 weeks gestation, foetal lung maturity should be assessed [1].The patient presented was for elective caesarean section but went into labour at 36 weeks and arrived at advanced dilation into the labour ward.

Our patient eventually delivered by the vaginal route, fortunately because she had delivered vaginally before and the babies were not very big in weight. In most instances, higher order multiple gestations (above two) are delivered by caesarean section.

1. Controversy surrounds the preferred route of delivery for some multiple gestations, especially twins. Deliveries should be based on individual needs and many depend on the clinician's practices and experience [1,2].

Our patient did not have any complications during delivery. Active management of third stage was done at delivery of the third triplet.Normally the interval between delivery of the first and second twin is 10-30 minutes [3]. Breech presentation of the second twin carries a higher morbidity and mortality. The first twins have a 3% greater chance of survival than the second twins. Hypoxia and trauma of operative delivery are the primary cause of death of second twins. Active management of the third stage of labour by administration of intravenous ergometrine or oxytocin at delivery of the anterior shoulder of the second twin is recommended in multiple pregnancy to avert postpartum haemorrhage [3,5].

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OBSTETRIC SHORT CASE 13

SICKLE CELL DISEASE IN PREGNANCY- LIVE PRETERM DELIVERY

NAME: B.A.O. Parity 0 + 0

AGE: 18 Years L.M.P 15/03/04

I.P NO: 0996095 E.D.D 22/12/04
D.O.A: 25/11/04
D.O.D: 25/11/04

PRESENTING COMPLAINT

She was admitted with labour pains for about 6 hours.

HISTORY OF PRESENTING COMPLAINTS

The pain was increasing in intensity and frequency for six hours before admission. It was accompanied by passage of mucoid discharge. She did not report any vaginal bleeding or drainage of liquor. Foetal movements were present and satisfactory to her even at admission. She did not have breathing difficulties, fever, urine frequency or any other complaints.

ANTENATAL CARE

She was a primigravida and had attended the antenatal clinic at Kenyatta hospital from 21 weeks gestation and had had three visits. She was known to have sickle cell disease. Her antenatal profile showed blood group B positive, VDRL and HIV tests were negative, the haemoglobin was 10.8g/dl.
She had received two tetanus toxoid injections, was on folate 5mg throughout pregnancy and was on proguanil 100mg daily. She was treated on one occasion for an upper respiratory tract infection. She was not admitted during the index pregnancy for any illness.

OBSTETRIC AND GYNAECOLOGY HISTORY

Her menarche was at 16 years and menses lasted 4-5 days and occurred every 21 days. The flow was moderate. She did not have dysmenorrhoea. She had never used any contraceptives before.

PAST MEDICAL HISTORY

BAO was known to have sickle cell disease from childhood and was on follow up in the haematology clinic. She had been maintained on folate and proguanil one tablet daily.

She had been admitted twice previously, the first time in 1989 at KNH with osteomyelitis of the right femur and twice at the Aga Khan (1995 and1998) hospital with painful crises. She was allergic to quinine. She did not have other allergies to foods or drugs.

FAMILY AND SOCIAL HISTORY

She was single, lived with her parents at upper hill in Nairobi. She was a form four student. She was second born in a family of five siblings. One of her brothers had sickle cell disease. There were no other chronic illnesses in her family.

PHYSICAL EXAMINATION

She was in a fair condition, mildly pale, had no jaundice, and was not dehydrated. She had no fever, no oedema, and no lymphadenopathy. Her vital signs were; pulse 90/minute regular and of good volume, respiratory rate 20/minute, blood pressure 120/8-mmHg, and temperature 37.1°C.
She had a healed scar on the right thigh lateral aspect and a healed sinus on the left thigh distal third medially.
Her cardiovascular, respiratory and nervous systems were essentially normal.

ABDOMINAL EXAMINATION

The abdomen was uniformly distended and moved with respiration. She had no surgical scars or therapeutic marks. The fundal height corresponded to a term gestation, the foetus was in longitudinal lie and cephalic presentation. The head was four-fifths above the pelvic brim. She had two mild contractions each lasting 20 seconds. The foetal heart tones were heard and normal at 140 beats/minute. The liver and spleen were not palpable.

PELVIC EXAMINATION

She had normal external genitalia, the cervix was central, partially effaced, short and 2cm dilated. The pelvis was clinically adequate. There was show on the examining finger.

DIAGNOSIS

Known sickler in latent labour at 36 weeks gestation (preterm labour).

INVESTIGATIONS

1. Haemogram – Haemoglobin 10.8g/dl
   white cell 11.5 x 103/l
   neutrophils 79%, lymphocytes 19%
   platelets 395 x103/l
2. Blood slide for malaria – negative.

3. Urea and electrolytes – Na+ 136mmol/l
K+ 4.0mmol/l
Creatinine 71umol/l
Urea 1.3mmol/l

4. Urinalysis – PH 6.5
Protein- nil
Sugar – nil; Leucocytes +

5. Haemoglobin electrophoresis – HbSS.

MANAGEMENT

A decision to allow labour to progress was made. She was admitted into the labour ward acute room, a large bore intravenous canula was inserted and hydration with normal saline started. She was given tramadol 100mg intramuscularly. She was to lie in a propped up angle of 45° with a left tilt. She was given oxygen by mask.A sample was taken for grouping and cross match in case delivery by caesarean section was necessary. Consultations were taken to the haematologist and the anaesthetist to review her. The haematologist agreed we continue with the current management while the anaesthetist considered her fit in case surgery was necessary. On review 3 hours later, the cervix was 4cm dilated and well effaced. Artificial rupture of membranes was done revealing clear liquor. There was no moulding, caput and the cord was not felt. Augmentation with escalating oxytocin 5i.u. in 500mls normal saline was started and she was given buscopan 40mg. She progressed well and 4 hours later she complained of an urge to pass stool. Examination revealed she was fully dilated. She was taken to the delivery room and had assisted vacuum delivery to a live male weighing 2700g and scored 10 in one and five minutes. Active management was done in third stage, the placenta was delivered by controlled cord traction.The episiotomy was reapired in layers. Total blood loss was estimated at 100mls.

Post delivery, hydration was well maintained, analgesics (tramadol and mefenamic acid) were continued. She was put on heparin prophylaxis 7500i.u. twice daily for three days and thereafter she was on junior aspirin one tablet dailiy. Prophylactic antibiotics (crystalline penicillin 2MU 6hourly) was started. Folate and proguanil were continued as in the antenatal period. She had initiated breastfeeding well.

By the third postnatal day, she was well, had no complaints. Her vital signs were normal, uterus was well contracted and she had no calf tenderness. She was discharged to be followed up in the postnatal clinic and haematology clinic.

FOLLOW UP

She was seen in the clinic at 6 weeks and was well with no complaints. Her uterus was not palpable, she was lactating well. She was adviced on contraception condoms as she was single and was planning to go back to school. She was discharged through the haematology clinic.

DISCUSSION

A case is presented of an 18 years old Para 0+0 with sickle cell disease since childhood and now in pregnancy and delivered a live infant at 36 weeks gestation without complications.

Sickle cell disease is a genetic disorder characterized by an abnormal haemoglobin, which causes red blood cells to become sickle shaped in certain conditions. Sickle cell haemoglobin results from a genetic substitution of valine for glutamic acid in the sixth position of the N-terminal of the beta chains. The resulting haemoglobin is called haemoglobin S. Other variants of the sickle cell include sickle cell haemoglobin C disease (HbSC disease-where lysine for glutamic acid is the substitution at the 6^{th} position of the N terminal of the beta chain) and sickle cell beta thalassaemia disease (HbS β thal) and HbD (glutamine for glutamic acid at the 6^{th} position on the N terminal of the beta chain) [1,2]. Our patient had HbSS disease.

This patient was homozygous for sickle cell disease, having inherited one gene from each of her parents.The sickle cell gene is inherited as autosomal recessive and can be passed on to both sexes. Because there are two beta globin genes, the sickle cell disorders can be heterozygous or homozygous. Heterozygous patients have HbAS and are said to have sickle cell trait. 25-45% of their haemoglobin is HbS. Their clinical course is benign. Patients with sickle cell anaemia are homozygous for Haemoglobin S (HbSS) which represents 75-100% of their haemoglobin. They are symptomatic and have reduced life expectancy. Severity of complications is related to the HbS concentration[1,2].

Our patient was from western Kenya and was diagnosed to have sickle cell disease in childhood.Sickle cell haemoglobinopathy is the most common inherited cause of haemolytic anaemia in the world. About 10% of blacks in the USA carry the sickle cell trait and 1 in 500 has sickle cell anaemia1. In Kenya, sickle cell disease is found mainly among young children from western Kenya and the coastal region. The distribution follows closely that of plasmodium falciparum malaria endemicity due to the less susceptibility of red cells with HbAS to affection by the malaria parasite. There is a worldwide increase in the cases of sickle cell anaemia in pregnancy due to the increasing survival of children with sickle cell towards adulthood and motherhood[2,4]. The prevalence of SS patients in obstetric practice among black population in the USA was variably reported as 1 in 806 to 1 in 1500 and it was noted that pregnancy in sickle cell disease accounted for 1 to 300 deliveries [1,5].

Our patient had mild anaemia at the time she was admitted. Patients with sickle cell disease have chronic anaemia because of shortened red cell survival due to circulation trauma and intravascular haemolysis or phagocytosis by the reticuloendothelial cells in the liver iand spleen. These patients are more prone to infections.The abnormal amino acid in the beta chain reduces both the stability and solubility of the haemoglobin. Under conditions of low oxygen tension, haemoglobin S polymerises producing distorted red blood cells that have the characteristic sickle shape. The deformed red cells in turn obstruct the microvasculature resulting in thrombosis. Usually the thrombosis only causes

pain but sometimes the thrombi damage organs like the kidneys, heart and lungs. Our patient did not have thrombotic phenomenon during the time of admission.

Our patient did not develop any other complications during this pregnancy.Infection, dehydration, cold, hypoxia and pregnancy precipitate vaso-occlusive episodes, also called sickle cell crises. Pain crises involve the bones and joints. Aplastic is characterized by rapidly developing anaemia due to cessation of red cell production by the bone marrow. Acute splenic sequestration crisis is associated with severe anaemia and hypovolaemic shock resulting from sudden massive red cell trapping within the splenic sinusoids[1]. Other manifestations include increased susceptibility to bacterial infection especially streptococcal pneumonia, pulmonary infarction, and myocardial damage, haematuria due to renal damage, cerebrovascular accidents, retinopathy and cholelithiasis.

The diagnosis in our patient was found to be HbSS on electrophoresis.Diagnosis of sickle cell is made by laboratory investigations. The patients have severe anaemia. On peripheral film sickle cells and macrocytes are seen. Reticulocyte count is increased due to increased red cell production. The sodium metabisulphite test involves using one drop of fresh 2% reagent mixed with one drop of blood on a slide, covered with a cover slip and the sides sealed with molten wax to prevent drying up and exclude air. Sickling occurs in both the sickle cell trait and sickle cell disease. Haemoglobin electrophoresis is done to confirm the major genotypes in the diagnosis of sickle cell disease [1].

Sickle cell disease is a recognizable risk factor in pregnancy and this is principally due to anaemia. There is increased risk of infection. Folate deficiency occurs due to increased erythropoiesis. The frequency of sickle cell crises increases especially in the third trimester [2, 4, 5]. There is also increased risk of PET, preterm delivery and caesarean section delivery. Maternal mortality is due to septicaemia, thromboembolic episodes and cardiac failure following haemolytic crisis [2,7]. Maternal mortality rate is 4-5% in the USA [1]. Otieno in his study at KNH reported a mortality of 36%[3]. The patient presented did not get a crisis while in the ward.

The patient presented did not have any foetal complications. Foetal complications include high abortion rates, intrauterine growth restriction, high stillbirth rates and prematurity. The overall foetal wastage is about 30% 2,. These are due to anaemia and the sickling process in the placenta causing placental insufficiency as a result of placental infarcts and increased blood viscosity [2,5]. Perinatal mortality rate is 180/1000 births in the United Kingdom. Our local study showed a rate of 450/1000 births. Perinatal mortality is due to low birth weight and intrapartum asphyxia secondary to chronic maternal anaemia and impaired placental function[3].

During follow up, our patient was on folate, and while in labour she was propped up and was started on intravenous antibiotics. Management of these patients antenatally requires a multidisciplinary approach involving the obstetrician, haemotologist, neonatologist and good laboratory back-up. Folic acid (5mg) given throughout pregnancy prevents megaloblastic changes. It is now recognized that patients with sickle cell disease have inadequate iron stores for the demands of pregnancy and should be given iron during

pregnancy and after delivery if peripheral blood picture is dimorphic suggestive of low iron stores[2]. Infections should be treated aggressively. These patients require weekly non-stress test together with serial ultrasound to monitor foetal growth and amniotic fluid from 32 weeks gestation. This was not done in our patient due to cost factors and she was well clinically-the symphysiofundal height corresponded to the dates and foetal movements were normal.

Episodes of bone pain are more common especially in third trimester and postpartum period. The mechanism of increased bone pain in late pregnancy in late pregnancy is unclear but hormonal and coagulation changes and effects of prostaglandins have been proposed[2,5]. Management of occlusive crises is by hydration, analgesia and warmth. Blood transfusion is reserved for those patients with Hb less than 6g/dl, severe vaso-occlusive crises, severe infection, acute splenic sequestration, and acute lung syndrome (pulmonary infection combined with pulmonary sickling).Malaria prophylaxis should be provided throughout pregnancy and even prenatally as infection can cause profound anaemia. The risks of heart failure and maternal death are reported to be proportionate to the haematocrit and prognosis is dramatically improved by exchange transfusion or by simple transfusion in association with diuretics [1,8]. Our patient did not require transfusion.

The patient presented had a vaginal delivery, and during labour adequate hydration was maintained. Most patients with sickle cell disease have normal vaginal delivery, though caesarean section is advocated for disproportion due to pelvic outlet deformity and is being used increasingly in an attempt to decrease the high foetal wastage in full term deliveries. In general, normal vaginal deliveries occur with relative ease due to low birth weight babies born to mothers with SS disease. During labour there is hypoxia in the placental bed with each uterine contraction. This can lead to a sickling crisis in the placental bed resulting in foetal demise. The patient should be adequately hydrated and a high oxygen tension maintained. Adequate pain relief must be given during labour. Caesarean section delivery should be done at the earliest sign of foetal distress and for other obstetric indications [1,2]. Following delivery, there should be active management of the third stage of labour to minimize blood loss. The patient should be kept warm, well hydrated and prophylactic antibiotics and heparin are given. Our patient was on oxygen by mask, pain relief was by tramadol injection and had vaginal delivery.

Reliable contraception should be offered. The combined pill is contraindicated due to increased risk of thromboembolism. The IUCD predisposes to endometritis. Barrrier methods can be used though they have a high failure rate. Tubal ligation offers a safe and permanent method for those with desired family size or if maternal complications are too life-threatening. Progestin only contraceptives can be used with caution as reversible methods[9]. Our patient chose to use condoms.

When there is a risk of sickle cell disease, partner testing for disease or trait can be done and appropriate counseling done prenatally. Prenatal tests such as chorionic villi sampling, amniocentesis or cordocentesis can be done where there is a risk of a fetus with sickle cell disease.

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OBSTETRIC SHORT CASE 14

FOETAL DISTRESS IN EARLY LABOUR – EMERGENCY CAESAREAN SECTION – LIVE BABY

NAME: W.C. K. L.M.P. 27.12.03

AGE : 25 Years E.D.D. 3.10.04

D.O.A. 10/10/04 PARITY 0 + 0

D.O.D 14/10/04

I.P NO. 0988133

PRESENTING COMPLAINT

She was admitted with a six-hour history of increasing low abdominal pain accompanied by backache.

HISTORY OF PRESENTING COMPLAINT

The pain was of increasing intensity, intermittent, radiated to the back and was also accompanied by mucoid vaginal discharge. There was no discharge of liquor or vaginal bleeding.

ANTENATAL CARE

She had attended the clinic at Huruma nursing home from three months gestation. Antenatal profile had shown the following; haemoglobin of10.5g/dl, VDRL and HIV tests were negative, her blood group was A positive. She had been given tetanus toxoid and ferrous sulphate plus folic acid tablets during the pregnancy follow up. The pregnancy was uneventful.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She was a primigravida. Her last menstrual period was on 27/12/03 giving a gestation of 41 weeks at the time of admission. She had her menarche at 17 years, her cycles were regular occurring every 28 days with flow lasting 4 days. She gave no history of dysmenorrhoea. She had not used any contraceptives previously.

PAST MEDICAL HISTORY

She had never been admitted to hospital before, she had no known chronic illnesses or any allergies.

FAMILY AND SOCIAL HISTORY

She was a married lady who lived in Bahati with her husband. She was employed, as a receptionist while the spouse was a businessman. She was the seventh born in a family of eight siblings, all alive and well. There were no chronic illnesses in the family. She neither smoked nor consumed alcohol.

SYSTEMIC ENQUIRY

This was nil significant.

PHYSICAL EXAMINATION

She was in a good general condition, was not pale and had no jaundice or oedema.

VITAL SIGNS

Temperature-36.7o C, pulse rate 62 beats/min, blood pressure 126/62mmHg, and a respiratory rate 20 breaths/minute.

CARDIOVASCULAR SYSTEM

Her pulse was regular, of good volume and non-collapsing. Both heart sounds were audible and normal. She had no murmurs.

RESPIRATORY SYSTEM

She had normal vesicular breathing bilaterally.

NERVOUS SYSTEM

She was well kempt, fully conscious and oriented. Her sensory and motor activities were normal.

ABDOMINAL EXAMINATION

The abdomen was uniformly distended and moved with respiration. The fundal height was equivalent to a term gestation, with the foetus in longitudinal lie, cephalic presentation and an irregular foetal heart rate at 170 beats/minute was heard. She had 3 strong contractions per ten minutes. The foetal head was ballotable. No other masses were palpable.

VAGINAL EXAMINATION

Her external genitalia were normal, the cervix was three centimetres dilated and soft. Artificial rupture of membranes revealed grade 3 meconium stained liquor. Her pelvis was clinically adequate. There was no cord prolapsing.

DIAGNOSIS

An impression of foetal distress in early labour was made.

MANAGEMENT

She was scheduled for an emergency caesarean section. The management was explained to her and she gave an informed consent. An IV line with 5% dextrose was set up, she was given oxygen by mask. The abdomen was shaved and a sample for group and cross-match was taken. Premedication with atropine 0.6mg IM was given, the paediatrician was called to receive the baby in view of the foetal distress and she was then wheeled to theatre.

In theatre the earlier findings were confirmed, vulvo-vaginal toilet was done and the bladder catheterised. The abdominal wall was surgically cleaned and draped using sterile drape towels. A caesarean section was then performed on W.C.K as described in the introduction. The outcome was a live female infant who weighed 3400gm and had an Apgar score of 4 in one, 7 in five and 8 in ten minutes. The baby had the cord entangled twice over the right lower limb and was taken to the newborn unit for observation after resuscitation by the paediatrician due to mild foetal distress. The liquor was thick meconium stained but not foul smelling. The placenta was normal. After uterine closure the abdomen was inspected and found normal, its wall was closed in layers and the wound dressed. Vulvovaginal toilet was done and clots expelled, anaesthesia was reversed successfully and she was taken to the recovery room.

Postoperatively, she had vital signs monitoring till fully awake, and was started on prophylactic antibiotics (crystalline penicillin and gentamicin). She was on pethidine initially and diclofenac for analgesia. By the second day she was started on oral feeds and ambulation while the treatment was continued. The baby was discharged from the NBU after one day and was brought to be with the mother. By the fourth day, her wound was healing well, the uterus was well contracted and lochia was alba. She was discharged home and was to be seen in the postnatal clinic in six weeks time.

FOLLOW-UP

She was seen in the clinic and had no complaints. Her wound was well healed, the uterus well involuted and had no calf tenderness. The baby was well and growing normally. She was discharged through the family planning clinic where she chose to use oral contraceptive pills.

DISCUSSION

The patient presented was a 25 year old primigravida who was admitted with foetal distress in labour and was delivered by emergency caesarean section. A live female infant weighing 3400g and with a score of 4/1, 7/5 and 8/10 was delivered. The baby was admitted to the NBU due to mild distress and eventually did well.

The term foetal distress may be defined as a complex of signs indicating a critical response in the foetus to stress. It implies mainly metabolic derangements-notably hypoxia and acidosis- that affect the functions of vital organs to the point of temporary or permanent injury or death. The term is too broad and vague to be applied with any precision to clinical situations. Uncertainty about the diagnosis of foetal distress based upon foetal heart rate patterns has given rise to the use of descriptions such as reassuring or nonreassuring patterns [1,2]. A nonreassuring foetal heart rate pattern suggests inability to remove doubt. Thus most obstetricians make a diagnosis of foetal distress using heart rate patterns when they lose confidence or cannot assuage doubts about foetal condition. Skillful monitoring will detect some degree of foetal compromise in at least 20% of all obstetric patients [1,2].

Foetal heart rate patterns can be measured in various ways including;

- Internal electronic foetal heart rate monitoring- a bipolar spiral electrode is attached directly to the foetus after membranes have been ruptured.
- External electronic monitoring- the foetal heart rate is detected through the maternal abdominal wall using the ultrasound Doppler principle.
- Intermittent auscultation- using the Pinnard foetoscope the foetal heart rate is heard through the maternal abdominal wall. This is the method commonly used in our set-up, as the electronic monitors are not yet available. It was used on the patient presented.

The diagnosis in our patient was made from non-reassuring foetal heart rate on auscultation, which was associated with meconium staining of the liquor. PH was not done, as the necessary equipment is not available in our unit.

Diagnosis of foetal distress based on heart rate patterns is tenuous because;

- The foetus is tethered by an umbilical cord where blood flow is constantly under jeopardy, which demands that the foetus has a strategy of survival
- These patterns are more of a reflection of foetal physiology than pathology. Physiological control of heart rate includes a variety of interconnected mechanisms that depend on blood flow as well as oxygenation.
- Normal labour is a process of increasing academia and thus a process of repeated hypoxic events resulting inevitably in acidosis.

According to the American College of Obstetricians and Gynaecologists, measurements of the pH in capillary scalp blood may help to identify the foetus in serious jeopardy. The pH of the foetal capillary scalp is normally lower than the umbilical venous blood and approaches that of the umbilical arterial blood. The following protocol has been recommended to try and confirm foetal distress[3]. If the pH is greater than 7.25, then labour is observed. If it is between 7.20 and 7.25, the pH measurement is repeated within thirty minutes. If the pH is less than 7.20, another sample is taken immediately and the

mother is taken to the operating room and prepared for surgery. Delivery is performed immediately if the low pH is confirmed, otherwise labour is allowed to continue. Foetal scalp sampling is not done in our setup thus it is difficult to objectively determine the infants who are actually in distress.

Our patient was admitted with meconium staining grade 3 and an irregular foetal heart rate. In the absence of any other foetal monitors, this is taken to be indicative of foetal distress. Obstetric teaching throughout the century has included the concept that meconium passage is a potential warning of foetal asphyxia. J Whitridge Williams observed in 1903- " a characteristic sign of impending asphyxia is the escape of meconium". He attributed meconium passage to 'relaxation of the sphincter ani muscle induced by faulty aeration of the foetal blood'. In a recent investigation from Parkland Hospital, meconium was found to be a 'low risk' obstetrical hazard because the perinatal mortality was 1 death per 1000 live births[4]. Meconium staining without foetal heart rate abnormalities and with labour progressing well seems to have no great significance. Meconium in the presence of a complicated labour with foetal heart rate abnormalities has a greater risk of foetal hypoxia than either meconium alone or foetal heart rate abnormalities alone[5]. Various theories have been suggested to explain foetal passage of meconium and may in part explain the tenuous connection between detection of meconium and infant mortality. These include;

- Intrauterine passage of meconium may represent normal gastrointestinal tract maturation under neural control[6].
- The pathological explanation proposes that the foetus pass meconium in response to hypoxia, and therefore it signifies foetal compromise[7].
- Meconium passage may also follow vagal stimulation from common but transient umbilical cord entrapment and resultant increased peristalsis[8].

Our patient's baby did not develop any features of meconium aspiration. A study at the Parkland Hospital involving almost 8000 pregnancies showed that meconium aspiration syndrome was significantly associated with foetal acidemia at birth[9]. This syndrome is more common with thick meconium staining (grade 3 meconium). Mechanisms of meconium aspiration are also poorly understood. Theories postulated include fetal gasping in-utero possibly when severely hypoxemic or with the onset of breathing following delivery. The first description of foetal heart sounds was in 1818. In 1821 it was observed that a slowing of the foetal heart rate was often associated with poor foetal outcome. In 1843 it was observed that that adding meconium staining in amniotic fluid is a further feature of foetal distress[2].

Delivery in the case presented was by caesarean section as she was still in the early stages of labour. Intrapartum foetal distress is an obstetric emergency traditionally managed by immediate delivery by either vaginal route or caesarean section. Studies have shown that babies delivered by caesarean section secondary to obstructed labour may have thick meconium but a healthy baby is delivered. Whereas meconium is rarely evident in cases of acute foetal distress such as abruptio placenta, ruptured uterus and prolapsed cord, the baby may suffer severe neurological damage due to hypoxia[1, 5].

Our patient was found to have cord entanglement intraoperatively and this could probably have been the cause of foetal distress. The best way to describe foetal distress is by assessing fetal acid-base status. These include blood pH, partial pressure off oxygen and partial pressure of carbon dioxide in foetal blood sample[10]. Foetal response to hypoxia is mediated through chemoreceptors at the carotid body for PCO2 and hormonal response to hypoxaemia is via carotid artery chemoreceptors in the carotid sinus nerve. This is a parasympathetic nerve its stimulation results in a bradycardia[2,5,10]. Prolonged hypoxaemia results in secretion of catecholamines from the adrenal cortex in proportion to the level of hypoxia. This raises the blood pressure and stimulates the heart, raising the heart rate. During each uterine contraction, there is reduced blood flow to the placenta due to occlusion of utero-placental arteries. However, uteroplacental circulation has enough reserve thus no hypoxia occurs. Some conditions have been identified to reduce perfusion of the intervillous space during labour resultant hypoxaemic foetal blood. These include: epidural anaesthesia, supine hypotension, frequent uterine contractions, placental insufficiency, ruptured uterus, abruptio placenta, umbilical cord compression, foetal haemorrhage as in vasa praevia, foetal anaemia in rhesus isoimmunisation[11]. Our patient did not have any of these conditions.

Managing our patient was hampered by the fact that continuos monitoring facilities are not available in our unit thus a decision to continue with labour while monitoring foetal heart rate patterns was difficult to make in her case. Continuous foetal monitoring with a cardiotocograph (CTG) has helped to pick up those babies at high risk or those in actual distress. It continuously records the foetal heart during contractions. Changes of foetal heart in relation to uterine contraction are useful in determining a foetus at risk. A rising foetal heart rate may be secondary to increasing catecholamines, particularly after interruption of oxygen supply to the foetus. Early deceleration of the foetal heart is described as harmless. It results from head compression during contractions. This raises vagal tone secondary to increased intracranial pressure. This normally recovers once uterine contraction is over. Variable deceleration is a transient fall in foetal heart rate after the onset of contraction and returns rapidly before the contraction ends. This is considered indicative of cord compression. Late deceleration starts after the onset of contraction and ends after the contraction finishes. This indicates interruption of oxygen delivery at the intervillous space or uteroplacental insufficiency. It indicates a greater risk for the development of foetal academia. Initially it causes respiratory acidosis in early stages then metabolic acidosis when oxygen delivery is severely reduced[2,5,10]. Since introduction of CTG in the early 1980's, it has helped to recognize these foetal heart rate changes early enough. Foetal blood sampling during labour was developed in the 1960's. Foetal acid-base changes during labour in conjunction with other parameters like foetal heart rate changes and meconium staining of liquor may give the exact condition of the foetus[2].

The progressive loss of foetal heart rate is indicative of a foetus slowly losing resource for labour thus prompt intervention is required. Metabolic acidemia during labour occurs in about 20% of high-risk pregnancies. Acidemia usually develops after the onset of abnormal foetal heart until late in second stage. A late deceleration, variable deceleration pattern is associated with low Apgar score, neonatal death, cerebral dysfunction and

delay in developmental milestones. Acidosis becomes statistically significant with pH value of 7.05[2,10]. This patient did not have foetal biochemical tests but her baby had a low Apgar score at delivery.

Where facilities allow, continuous monitoring of labour with CTG machine should be done to all patients with preterm labour, post term pregnancy, meconium staining of amniotic fluid, foetal growth retardation and babies who are small for dates.

Upon admission and evaluation, our patient was started on intravenous fluids, given oxygen by mask and was placed in the left lateral position.
Several methods have been shown to resuscitate a baby in-utero when in distress. Giving the mother oxygen increases oxygen delivery to the utero-placental unit. Prevention of dehydration and intravenous dextrose will help the baby in distress. Repositioning the mother to the left lateral position helps to increase blood flow to the utero-placental unit with increased oxygen supply to the foetus. These were done to the patient presented. Other methods like use of magnesium sulphate boluses given to the mother helps to resuscitate foetal distress in mothers awaiting caesarean section especially if there is evidence of increased uterine activity. Amnioinfusion using normal saline has been used to reduce cord compression. Tocolytics also help to silence the hyperactive uterus. Intravenous nitroglycerin has also been tried to relieve uterine hyperactivity[11,12]. To predict foetal distress, accurate measurement of inferior vena-caval diameter (IVCD) during late second trimester has been used. Non-reactive foetal heart rate pattern before labour is also a predictor of a foetus that may develop distress in labour.
After delivery, erythropoietin concentration in the cord arterial blood indicates a chronic foetal hypoxia and a longer duration of hypoxia before birth.

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OBSTETRIC CASE 15

RETAINED PLACENTA – SUCCESSFUL MANUAL REMOVAL

NAME: E.G. L.M.P: 24.04.2004

AGE: 27 Years E.D.D: 31.01.2005

I.P NO: 0990829 Parity 1+0

D.O.A: 30.10.04

D.O.D: 02.11.04

PRESENTING COMPLAINT

EG was admitted with a two-day history of lower abdominal pain and drainage of liquor for three hours prior to admission.

HISTORY OF PRESENTING COMPLAINT

She was first seen in a private clinic from where she was adviced to be seen at KNH. The abdominal pain was increasing in intensity and associated with drainage of liquor. It was radiating to the back. She did not give history of having fever previously. She had not had any trauma, frequency of urine or dysuria. She did not have any abnormal discharge prior to the onset of draining liquor.

ANTENATAL CARE

She had started antenatal clinic at St Mary's hospital at five months gestation. Her antenatal card was not available though she said antenatal profile had been done and was normal. The pregnancy had been uneventful until the present complaints.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She was a Para 1 + 0. She had her first delivery at 7 months gestation in 2001 by caesarean section to an infant weighing 1100g who died at six weeks of age due to neonatal sepsis. This was her second pregnancy.
She attained her menarche was 14 years, her menstrual cycles being regular occurring every 28 days and lasting 3-5 days with normal flow. She did not have dysmenorrhoea. She had not used any form of contraception previously. Her last menstrual period was on 20.4.04 and the expected date of delivery on 31.01.05. She was at maturity by dates of 27 weeks and 2 days at the time of admission.

PAST MEDICAL HISTORY

She had not been admitted to hospital before and had no known drug or food allergies.

FAMILY AND SOCIAL HISTORY

She was married and lived with her spouse in Nairobi. She worked as a sales lady in a milk factory while the spouse was a businessman. She did not use alcohol or smoke cigarettes. She gave no family history of chronic illnesses.

PHYSICAL EXAMINATION

She was in a fair general condition and of good nutritional status. She was not pale, had no jaundice or lymph node enlargement. Her vital signs were; blood pressure 110/60mmHg, respiratory rate 22/minute, pulse rate 102/minute and febrile with a temperature of 39^0 C. Her cardiovascular, respiratory and nervous systems were normal.

ABDOMINAL EXAMINATION

She had a midline subumbilical scar.
The uterus was well contracted corresponding to 26 weeks gestation size. She had two moderate contractions each lasting 20-40seconds. The foetus was in cephalic presentation and a foetal heart was heard that was regular at 132 beats/minute.

PELVIC EXAMINATION

She had normal external genitalia.The cervix was fully effaced and 5 cm dilated. The liquor had light staining with meconium and also had a foul smell. The foetal head was well applied to the cervix.

DIAGNOSIS

Preterm labour in Patient with one previous caesarean section scar.

MANAGEMENT

The conditioned was explained to her. She was started on metronidazole 500mg, augmentin 1.2g and gentamicin 80mg due to the suspected chorioamnionitis; all were given intravenously. Blood was taken for cross-match. Labour was allowed to progress and was monitored by use of the partogram. Three hours later, she complained of an urge to pass stool. Pelvic examination revealed that she was fully dilated and the foetal head was nearly crowning. She was taken to the delivery room, her perineum draped and the foetus delivered in vertex presentation, a live female infant weighing 850g and Apgar score 2 in one and 4 in 5 minutes. The baby was taken to the newborn unit. The placenta did not detach thereafter and she started having per vaginal bleeding. The urinary bladder was emptied by catheterization. She was started on 20 units of oxytocin in 500mls of normal saline. Controlled cord traction was attempted to try and deliver the placenta. This

was not successful. After 30 minutes of oxytocin and attempting cord traction, the placenta still felt adherent and had not separated. A diagnosis of retained placenta was made and the patient informed. Informed consent was obtained and she was given 0.6mg atropine intramuscularly. She was then taken to theatre for manual removal under anaesthesia.
Manual removal of placenta.

In theatre and under general anaesthesia, she was placed in the lithotomy position, vulva vaginal toilet was done and she was draped with sterile drapes. The bladder was catheterised and drained off clear urine. Examination revealed a uterus at 24 weeks, intact cervix and vagina. The right hand was inserted into the uterus identifying the placental site implantation site by the hand following the course of the umbilical cord. With the left hand supporting the uterus, the ulna aspect of the right hand was used to separate off the placenta from its attachment to get a line of cleavage. With this plane of cleavage separation was continued until the whole placenta was detached from the uterine wall. The hand was then removed delivering the placenta with it. The placenta was normal and weighed 200g. It was inspected and found to be complete including the membranes. The uterine cavity was then explored and found to be empty; uterine massage was done and the oxytocin drip continued. She had minimal bleeding and was taken to the recovery ward.

POSTOPERATIVE MANAGEMENT

She was continued the antibiotics that had been started, and the intravenous infusion of oxytocin of 20 units in 500mls saline ran at 20-drops/ minute. Her vital signs remained normal. Vaginal bleeding remained minimal. In the postnatal ward she remained stable and was discharged on oral augmentin and metronidazole for five days. She was to be reviewed in the postnatal clinic in four weeks time. The baby had died in the newborn unit due to severe prematurity and the mother was put on bromocriptine 2.5 mg twice daily to stop lactation that had set in.

FOLLOW-UP

She did not turn up in the clinic as scheduled.

DISCUSSION

Our patient was a Para 1+0 who had retained placenta after preterm delivery in the hospital. Manual removal of the placenta was done under general anaesthesia.

If the placenta is not expelled within the first ten minutes following completion of the second stage of labour it is considered abnormal[1]. However there is no general consensus regarding the time interval before manual removal is attempted. Some authours regard ten minutes for the third stage abnormal and recommend manual removal after this interval, whereas others consider 30 minutes to two hours expectant management especially if labour was induced [2,3,4]. Failure of the placenta to deliver spontaneously is one of the important causes of postpartum haemorrhage. However it has been shown that there is no increased risk until 30 minutes have elapsed suggesting that conservative management is appropriate during this interval[5]. In the presence of haemorrhage, it is obviously unreasonable to wait for spontaneous separation, and manual removal should be undertaken without delay. 30 minutes had elapsed in our patient when a diagnosis of retained placenta was made.

This patient had previous caesarean section, which could have contributed to her developing a retained placenta. The cause of prolonged third stage is often not identified. Likely mechanisms include uterine atony, abnormal placental implantation and inadequate efforts to express the placenta before spontaneous separation[5]. Several correlates of prolonged third stage that may be related to uterine atony include; prolonged labour, augmented labour, multiparity and induced labour, abnormalities of the placenta implantation, previous abortion and caesarean deliveries. There is also increased incidence of retained placenta among patients who had preterm labour[1, 5,6]. Our patient had been admitted with preterm labour.

In our unit the policy is for active management of the third stage of labour with controlled cord traction combined with upward pressure on the lower uterine segment; this ensures that the placenta can be removed as soon as it separates (Brandt-Andrews method). Cord traction is gentle to avoid uterine inversion. There is also routine use of ergometrine and oxytocin. This reduces the risk of postpartum haemorrhage. This was done in our patient but the placenta was noted not to be separate. A combination of ergometrine and oxytocin (syntometrine) is more effective in reducing postpartum haemorrhage but either may be used alone[8].
Spontaneous placental separation is impending if the uterus becomes round and firm, a sudden gush of blood comes from the vagina, the uterus seems to rise in the abdomen, and the umbilical cord moves down out of the vagina[1, 4,6,7].

The patient with retained placenta is often in shock due to haemorrhage. Adequate resuscitation is mandatory before attempting manual removal. This should include transfusion where necessary and the use of oxytocin[4, 6].Our patient did not have shock and only got oxytocin infusion.

The patient presented had manual removal of the placenta under anaesthesia. Manual removal is undertaken under general anaesthesia. With the patient in lithotomy position, the uterus is stabilized by grasping the fundus with a hand placed over the abdomen. The other hand traces the course of the umbilical cord through the vagina and cervix into the uterus to palpate the edge of the placenta. The hand is then inserted between the placenta and uterine wall, palmar side toward the placenta. The hand is then gently swept from side to side and up and down to peel the placenta from its attachments to the uterus. When the placenta is completely separated from the uterus, it is grasped and pulled from the uterus. The foetal and maternal sides of the placenta are then inspected to ensure that the entire placenta has been removed. If there is evidence of incomplete removal (haemorrhage due to interruption of vessels on the foetal side or missing cotyledons on the maternal side) the uterus is re-explored and any pieces of adherent placenta removed. Gentle curettage can also be done. The uterus is massaged until a firm myometrial tone is achieved; an oxytocin drip is administered to promote uterine contraction[1, 4,6]. All these were done for our patient.

Other methods of managing retained placenta remain controversial. The effect of intraumbilical oxytocin into the retained placenta has not been agreed upon. It has been proposed that this leads to a high concentration of oxytocin at the uterine wall[2, 9,10]. This method has not been tried in our unit.

Our patient was put on antibiotics and did not get any complications. Risks of manual removal include haemorrhage due to partial detachment, weakening of the uterine wall with subsequent rupture, endometritis and uterine inversion[1,4,6,7].

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OBSTETRIC LONG COMMENTARY

MANAGEMENT OF SEVERE PREECLAMPSIA AT KENYATTA NATIONAL HOSPITAL(KNH) – A FIVE YEAR REVIEW

ABSTRACT

BACKGROUND : Hypertensive disorders in pregnancy remain a major contributor to maternal and perinatal morbidity and mortality. Current data is needed in KNH on the treatment of severe preeclampsia in the obstetric unit. This can help to formulate protocols for management of this serious condition in the unit.

OBJECTIVE : To determine the management and outcomes of severe preeclampsia at the Kenyatta National Hospital.

STUDY DESIGN : This was a cross – sectional study based on the retrospective analysis of records of patients with severe preeclampsia.

STUDY SETTING : The study was conducted at the Kenyatta National Hospital.

STUDY SUBJECTS: All case notes of patients with blood pressures higher than 160mmHg systolic or 110 mmHg diastolic, other features of severe preeclampsia who were admitted between January 1, 1999 and December 31 2003 were studied.

MAIN OUTCOME MEASURES: Treatment regimens in use for preeclampsia. Maternal and fetal morbidity and mortality.

RESULTS : There were 33,661 deliveries at KNH during the study period. Severe hypertensive disease occurred in 1.14 % deliveries. Combination of hydralazine and methyldopa was the most commonly used antihypertensive regimen while diazepam was the most used anticonvulsant. The mean duration from admission to delivery was 5 days and the mean gestation at delivery was 34.5 weeks. Caesarean section was the main mode of delivery (64.3%) during the study period. The commonest reason for intervention was deteriorating foetal status as indicated by poor biophysical profile (<6/8) or reduced foetal movements. The perinatal mortality rate was 266/1000 births. The maternal mortality ratio was 29/100000 deliveries. Complications occurred in 24.2% of the women. The average hospital stay was 13.7 days. Perinatal outcomes were affected by gestation at delivery, birth weight, the mode of delivery.Logistic regression analysis including other factors revealed that the Apgar score, birth weight and caesarean delivery were the factors influencing perinatal outcome (0R 2.1, 1.7, and 3.3 respectively 95% CI).

CONCLUSION

Severe preeclampsia is a significant cause of maternal and perinatal morbidity and mortality at the KNH. There is no standard protocol currently in use for treatment in the KNH obstetric unit. There is need to develop protocols for its management in our unit.

INTRODUCTION

Normal pregnancy, childbirth and maternal satisfaction from the birth experience is fortunately the commonest outcome of pregnancy. However some pregnancies become complicated by illnesses and conditions that make the mothers fulfillment of their high expectations a real nightmare. These complications at times lead to maternal deaths. There are also attendant risks to the developing foetus. Maternal death is just the tip of the iceberg. Many women survive deaths but suffer from severe acute morbidity that results from conditions arising from and some peculiar to their pregnant state. Over half a million women die worldwide annually as a result of complications of pregnancy and childbirth[1].

Hypertensive diseases in pregnancy is one of the five major contributors to maternal morbidity and mortality, being third after sepsis and haemorrhage[2]. This is true in developing countries where in most cases there is still poor obstetric care, inadequate antenatal and delivery services, poor infrastructure and minimal knowledge in terms of recognition of risk factors among the ante-natal population. In the developed world, hypertensive disease in pregnancy is the most common disorder in pregnancy and the biggest cause of maternal morbidity and mortality[2,3].In the United States of America, hypertensive disorders of pregnancy run second only to embolism as a cause of maternal mortality and is directly responsible for 15% of maternal deaths in that country[3].

In Kenya, it has been reported that hypertension in pregnancy accounts for 9.4% of maternal deaths, third only to sepsis (42.9%) and haemorrhage (10.8%).Of these deaths, 84% are due to eclampsia and 16% due to severe preeclampsia[4]. Pre eclampsia is also an important contributor to perinatal morbidity and mortality, secondary to both direct foetal effects and iatrogenic preterm delivery for maternal indications.

Despite the importance of this condition peculiar to pregnancy, its origin remains obscure and the disease process is ultimately reversed only by delivery. The identification of the clinical entity of hypertension in pregnancy and its effective management play a significant role in the outcome of pregnancy for both the mother and the baby. Management of pre eclampsia poses one of the greatest

challenges in obstetrics related to the delicate balance between maternal safety and the fulfillment of her high expectations of a favourable outcome at the end of her pregnancy. Maternal benefits must be weighed against the neonatal risks of early preterm delivery e.g. prematurity and respiratory distress syndrome.

Management protocols differ widely in many centres across the world. Invariably the aim is control of blood pressure to avoid severe complications followed by safe delivery. The optimal time for delivery in the group with severe pre eclampsia is not known as there are no predictors for two of the commonest complications occurring in them, namely eclampsia and abruptio placenta. It was the aim of this study to determine the magnitude of severe pre eclampsia and eclampsia in pregnancy at the Kenyatta National Hospital obstetrics unit, their treatment, and the outcomes of both the mother and foetus and any complications arising out of severe pre eclampsia in pregnancy. This will help in formulating future protocols and making necessary changes in current management.

LITERATURE REVIEW

Hypertensive disorders in pregnancy remains a major cause of maternal and perinatal morbidity and mortality. Approximately 6.8 % of all pregnancies are complicated by hypertension[1,2,5,6,7].

Hypertension in pregnancy is defined by two blood pressure readings of 140/90 mmHg or more taken at least six hours apart occurring after 20weeks gestation. The working group on High blood Pressure in Pregnancy and the American College of Obstetricians and Gynaecologists have established guidelines for diagnosis of high blood pressure in pregnancy [2,5,8]. Five types of hypertensive disease in pregnancy are recognized; Gestational hypertension, preeclampsia, eclampsia, superimposed preeclampsia on chronic hypertension and chronic hypertension.

Severe preeclampsia is characterised by the following:-[2,5,8]

BP 160/110mmHg,Proteinuria 5.0g/24hrs or 2+ dipstick,

Serum creatinine 110 umo1/L unless known to be previously elevated. Oliguria (<500ml/24hours)

Cerebral or visual disturbance or persistent headache.

Epigastric pain (persistent), Elevated liver enzymes (ALT or AST > 70units/l), haemolysis (schistocytes on peripheral blood film)

Thrombocytopaenia (<100,000/mm3), Pulmonary Oedema, Foetal growth restriction

Retinal Haemorrhage, exudates, and papilloedema.

Convulsions. Occurs when seizures are present in a patient with preeclampsia that cannot be attributed to any other causes.

The difference between mild and severe disease may be misleading since apparently mild disease may rapidly progress to severe disease.

The presence of preeclampsia substantially increases the rate of maternal and perinatal morbidity. A study at Kenyatta hospital found hypertensive disease in pregnancy to account for 9.4% of maternal deaths, being third after sepsis and haemorrhage. Of these deaths, 84.2% were due to eclampsia and 15.8% due to severe preeclampsia[4].Another study in Garissa provincial hospital found hypertension being the leading cause of complications in the antenatal period (33% of the pregnancies with complications) while it was the second cause of mortality after haemorrhage causing 18.2% of the deaths reported [9]. The prevalence of hypertensive disorders has been reported by different investigators to range from 1.5-9%[10,12,14] for preeclampsia. Recently a study reported a prevalence of 15.8% among patients delivering at the Aga Khan Hospital in Nairobi[13]. A prevalence of 2.9% was reported at Pumwani Hospital with eclampsia occurring in 0.3% of the deliveries[14]. The incidence of eclampsia is reported as 0.2-0.5% of all deliveries[18]. Locally reported figures are 1.04-11 per 1000 deliveries [10,12,17,19]. Maternal hypertension also contributes to perinatal morbidity and mortality. Perinatal mortality rates for preeclampsia and eclampsia have been reported as 82.5 per 1000, 225 per 1000[2] and recently 3.3 per 1000 and 48 per 1000[15,17]. Maternal mortality rates reported are 46.5 per 1000[2], 3.25 per 1000 and 0.48 per 1000[17].The high figures could be due to poor timing of delivery, either too early leading to neonatal deaths or too late leading to maternal deaths.

Though still a major cause of maternal morbidity and mortality even in the developed world, and despite extensive research, the aetiology of preeclampsia remains elusive and largely unknown. Several risk factors have been identified as predisposing to the development of preeclampsia. These include nulliparity, multifetal gestation, family history of preeclampsia or eclampsia, non immune hydrops fetalis, molar pregnancy, antiphospholipid syndrome, diabetes mellitus, and renal vascular disease[2,6,8,18]. Older women who have an increasing incidence of chronic hypertension are also at a greater risk for superimposed preeclampsia. Obesity, black race and genetic predisposition have also been identified as possible risk factors. The familial factor in pathogenesis of preeclampsia has been recognised for many years. Chesley and Cooper referred to an account by Elliot in 1883 of

eclampsia occurring in a mother and her four daughters[19]. Maternal genotype alone was implicated by Sutherland et al [20].Chesley et al observed an increased frequency of preeclampsia in the daughters but not daughters in-law of women who themselves had history of eclampsia[19].A study in Nigeria found that 74% of multiparous eclamptics had a new partner for the current pregnancy, hence the likelihood of an immunological factor[21]. It has been suggested that preeclampsia/eclampsia may be genetically determined, susceptibility based on a single recessive gene with incomplete penetrance [20,22]. Preeclampsia/ eclampsia have also been found to vary significantly with the weather. The incidence has been found to increase with wet, humid weather and low temperatures. These are implicated through as yet unestablished mechanisms[23].Although maternal smoking causes a variety of adverse pregnancy outcomes, ironically smoking during pregnancy has consistently been associated with a reduced risk of hypertension during pregnancy[2,24]
Preeclampsia is now recognised as a multisystemic disease of unknown aetiology characterized by vasospasm, renal vascular damage and abnormal coagulation in the mother and varying degrees of intra uterine growth retardation in the fetus[2]. There is vascular endothelial cell dysfunction with generalised vasospasm, and ischaemia causing hypertension and necrosis[2,5,8,25].
The principle problem is poor placental perfusion from vasospasm attributed to abnormal implantation thought to be an immunologic disturbance or underlying maternal vascular disease. In normal pregnancy foetal trophoblasts invade maternal decidua in the first trimester and remodels the spiral arteries making them low resistance arteries. In pregnancies complicated by preeclampsia trophoblast invasion is shallow and maternal vascular conversion is shallow with subsequent development of atherosis and poor placental perfusion as pregnancy progresses and this has been postulated to produce placental hypoxia. This in turn results in release of a factor by the placenta which is responsible for endothelial cell damage in the mother[26]. The placenta is usually the unifying organ, removal of which results in reversal of the disease in most women within 48 hours of delivery. Clinical observation such as increased incidence of preeclampsia in those with hydatidiform mole[27] and persistence of the disease until the removal of the placenta three months after delivery emphasize the importance of the placenta in genesis of this disease [28].

Within the placenta, ischaemia, physical changes in vessels and increased enzymatic degradation of purines to uric acid may generate radicals which damage endothelial cells either directly or through lipid peroxidation [29,30]. In addition, cytokines from the placenta may stimulate lipolysis with a resultant increase in free fatty acids to levels beyond those seen in normal pregnancy, a change that

occurs weeks prior to clinical signs of disease[30,31]. The increased free radical production in combination with reduced antioxidative capacity and dyslipidaemia lead to formation of toxic lipid peroxidation products, which may be critical to vascular endothelial damage that underlies the pathogenesis of preeclamptic toxaemia. No single toxin is responsible for the systemic manifestations of the disease and the term "toxaemia" merely reinforces the idea of placental substances causing detriment to both mother and foetus. Vascular damage results in imbalances in substances that regulate vasodilation and constriction important in control of the haemodynamic changes of pregnancy.

Damage to vascular endothelium results in multiple organ system involvement in preeclampsia including the renal, cardiovascular, haematological, hepatic, pulmonary, and central nervous system[2,6,8]. The characteristic renal lesion is glomeruloendotheliosis. There is swelling of the endothelial cells leading to occlusion of the capillary lumen and apparent glomerular ischaemia. Impaired tubal function leads to inability of the kidneys to concentrate urine and a raised level of serum urea, creatinine, and uric acid[2]. There is an increase in the total body water especially if oedema is present. Liver disease ranges from subclinical involvement to HELLP Syndrome, an acronym for haemolysis, elevated liver enzymes and low platelet counts that occurs in severe preeclampsia [32]. A study at Kenyatta National Hospital found the HELLP syndrome to occur in 2.8% of patients with preeclampsia[33].

In the central nervous system, petechial haemorrhages may be seen. Sometimes massive haemorrhage occurs in the basal ganglia, pons, subarachnoid space and at times, seizures occurs. Eclampsia remains a complex and partially understood disease. The aetiology of eclamptic fits is unclear but the pathology finding is similar to those of hypertensive encephalopathy including fibrinoid necrosis, thrombosis of arterioles, microinfarcts and petechial haemorrhages. Fever has been postulated to be a trigger factor for eclampsia in various studies[34,35]. Seizures have been attributed to platelet thrombi, localized vasoconstriction and foci of haemorrhage in the brain cortex [2,6]. Vasospasm causes local ischaemia, arteriolar –necrosis, and disruption of the blood brain barrier. Forced dilatation has also been advanced as a theory to explain the pathogenesis of eclampsia. It is thought that as blood pressure rises above the limit of autoregulation, cerebral vasodilation occurs. Overdilatation of cerebral vessels results in necrosis of the medial muscle fibres and damage to the vessel walls. It is possible that both above mechanisms operate together[6]. CT scan studies indicate

vasospasm and cerebral oedema involving the main watershed areas and the parieto-occipital lobes of the brain[36]. Non specific electroencephalograph abnormalities can usually be demonstrated for sometime after a seizure[2,6] Most of these abnormalities subside within three months.

Management of severe preeclampsia poses one of the greatest challenges in obstetrics especially when the patient is remote from term. Management protocols for severe preeclampsia and eclampsia differs widely in many centres across the world. For those with severe preeclampsia options available include immediate delivery, pregnancy prolongation with elective delivery or a forty eight hours delay to facilitate steroid therapy, the aim being to improve neonatal outcome [2,6]. The optimal time for delivery is unknown as there are no predictors for two common complications of severe preeclampsia-eclampsia and abruptio placenta. Intervention is hampered by the fact that not all hypertensive disease follows orderly progression from mild to severe disease, some patients may be found suffering from any stage including eclampsia without apparently having passed through preceding stages [2,6,7,8]. Most protocols aim at control of blood pressure, prevention of convulsions to avoid severe complications, followed by prompt delivery. HELLP syndrome is a poor prognostic sign. It may occur remote from term and with no elevation of blood pressure[8,32].

For the patient with eclampsia, the objectives in treatment are control of fits, control of blood pressure, prevention of complications and prompt delivery [2,5,8]. There is usually no aura preceding the seizure and the patient may have many seizures. Unconsciousness lasts for a variable period of time. About 75% of eclamptic seizures occur before delivery. About 50% of postpartum seizures occur in the first 48 hours after delivery but they may occur as late as 6 weeks postpartum [6]. In rare instances, eclampsia develops before 20 weeks gestation like in cases of hydatidiform mole. Seizures after 48 hours require an evaluation for other aetiologic pathology [2,5-8] e.g. cerebral venous thrombosis, intracerebral haemorrhage, hypertensive encephalopathy, phaeochromocytoma, space occupying lesions of the central nervous system, metabolic disorders, epilepsy and infections (Meningitis and cerebral malaria). Hypoxia and the development of lactic acidemia following convulsions adversely affect the fetus as well. Fetal bradycardia lasting 3-5 minutes has been documented [2]. Development of pulmonary oedema following seizures in a primigravida is a grave prognostic sign [37]. A 2.9% incidence of pulmonary oedema has been reported with 70-80% occurring postpartum as the parturient begins to mobilize oedema fluid [50].

In the initial management of eclampsia, stabilization of the mother should receive most of the attention. Immediate control of fits and prevention of further convulsive activity needs to be established. This requires intravenous access and institution of anticonvulsant. MgSO4 therapy –a 4.0g intravenous loading dose followed by 1g every hour by infusion is the most appropriate. This is continued for at least twenty four hours postpartum. Correction of maternal and foetal hypoxia and acidemia is accomplished by giving oxygen supplementation by mask. Intubation to protect the airway should be performed if needed. Attention is then directed to blood pressure assessment and control. Antihypertensive agents are given to control the pressure and bring the diastolic pressure to below 110mmHg and the systolic to below 160mmHg. Once the antepartum mother with eclampsia is stabilised and the foetal status checked, only then can delivery be instituted. Vaginal delivery is preferred to caesarean section where possible as long as foetal presentation and status are appropriate and labour can progress in an orderly fashion. Where vaginal delivery will cause delay to delivery such as in the patient with an unfavourable cervix then caesarean section is better than a long induction of labour[2,5-8].

Various drugs are used across the world in the management of severe preeclampsia and eclampsia. These are mainly for controlling blood pressure and for the treatment and prevention of eclamptic fits. Drugs used are those that even if they cross the placenta have no deleterious effects on the fetus. The drugs used for controlling blood pressure are in many centres given if blood pressure exceeds a diastolic of 110mmHg. Treatment that causes large, precipitate drops in mean arterial pressure are avoided [2,6]. The goal is to maintain the diastolic blood pressure at the 90-100mmHg range. Drugs used include the following: [2,6,7,8]

-Hydrallazine, Labetalol, Nifedipine, Methyldopa

-Other drugs used depending on availability and centre protocols include Trimethophan (a ganglionic blocker), Nitroglycerin (a venodilator), and Sodium Nitroprusside (a vasodilator without autonomic or central nervous system effects).

Various drugs are used in the prevention and management of eclamptic fits. Anticonvulsants are given to women in the belief that they will prevent the onset of eclampsia and so improve the outcome of both mother and foetus. Eclampsia may be associated with poorer outcome than preeclampsia. But even if eclampsia is prevented it does not mean other serious complications such as renal failure, hepatic failure, and disseminated intravascular coagulation are avoided. Patients may develop eclampsia without hypertension or proteinuria ever having been documented[2,38].

Prediction of eclampsia is therefore fraught with difficulty. Eclampsia remains a complex and partially understood disease. Drugs used in prophylaxis and management of fits in eclampsia include the following: [2,5,39-44]

-Magnesium sulphate ($MgSO_4$). The most commonly used drug in the United States of America. Mg SO_4 has been found superior over other anticonvulsants.

- Diazepam, phenytoin. Other drugs that have been used for seizures or prophylaxis against seizures include Chlormethiazole, sodium thiopental, Phenobarbital, and the lytic cocktail of chlorpromazine, phenergan and pethidine.

Mg SO_4 has been found superior over other anticonvulsants. Diazepam crosses the placenta and may cause hypotonia,apnoeic attacks and hyperbilirubinaemia in the neonate.The collaborative eclampsia trial has provided irrefutable evidence of the superiority of $MgSO_4$ compared to other anticonvulsants in prevention of recurrent seizures in women with eclampsia.$MgSO_4$ is given in one of two protocols-intravenous(I.V) or intramuscular(I.M).In both, an initial loading dose of 4g is given I.V over 5-10minutes.This is followed in the IM regime by 5g in the upper outer quadrant of each buttock and a maintenance dose of 2.5g in each buttock every 4 hours. In the IV regime the loading dose is followed by an infusion of 1g per hour. In both treatment is continued for 24 hours after the last fit or delivery.$MgSO_4$ has no sedative effects but can depress neuromuscular junction activity thus can cause muscle paralysis as serum levels increase. Therapy monitoring is by close observation of patella reflexes, respiratory rate and urine output as excretion is renal. Quarter hourly observations are necessary if complications are to be minimised. Overdose can cause respiratory depression or arrest. Loss of patella reflexes precedes respiratory depression. Calcium gluconate is used as the antidote to magnesium toxicity together with oxygen by mask for ventilatory support. Intubation may be necessary for those with respiratory arrest. Further administration of $MgSO_4$ should be stopped in any patient who loses deep tendon reflexes, respiration is less than 16 breaths/minute and urine output below 25mls/hr.Treatment can be restarted at lower dose if needed once reflexes have returned.[38-40,44]

Severe preeclampsia and eclampsia is associated with various complications. These include: early delivery and complication due to prematurity, acute and chronic uteroplacental insufficiency resulting at times in foetal distress or still births, intrauterine growth retardation and oligohydramnios. In the mother pulmonary oedema may occur following seizures. Cerebrovascular haemorrhage also occurs more in the older women. Acute renal failure may result from renal

tubular necrosis. This is mostly induced by haemorrhage. The patient has oliguria or anuria and levels of urea and creatinine are high, some requiring dialysis. Retinal detachment or occipital lobe ischaemia may result in blindness. The HELLP syndrome sometimes occurs and is a poor prognostic sign. Other maternal complications include abruption placenta, disseminated intravascular coagulation cardiopulmonary failure, postpartum haemorrhage. The risk of preeclampsia recurring in subsequent pregnancies is 33% and when this was superimposed on chronic hypertension, the recurrence rate is about 70% [2,6,7,8,33] Increased maternal deaths are mainly related to eclampsia, accidental haemorrhage, renal failure, pulmonary oedema, disseminated intravascular coagulopathy and the HELLP syndrome. Perinatal mortality is related to early delivery, respiratory distress syndrome, hypothermia, hypoglycaemia and electrolyte imbalance.

From the foregoing it is clear that preeclampsia remains a major contributor to maternal and foetal morbidity and mortality, and that we are still struggling to understand its aetiology, offer prophylactic treatment or prevent its complications. Management protocols differ the world over in terms of drug use and timing of delivery. It was the aim of this study to determine the magnitude of severe preeclampsia and eclampsia in the KNH obstetric unit, its treatment and outcomes for the mother and foetus, the complications encountered arising out of severe hypertensive disease in pregnancy. It will provide information on the drug regimens in use and the outcomes and will assist in formulating future treatment protocols.

STUDY JUSTIFICATION

Severe preeclampsia and eclampsia are common obstetric emergencies and remain a major contributor to maternal and perinatal morbidity and mortality. Maternal mortality rates ranges from 1-20%, while perinatal mortality rates range between 130-300/1000 deliveries [2]. In Kenya, in 1980 preeclampsia/eclampsia was found to contribute to 3% of maternal mortality at the Kenyatta national Hospital [45]. By 1999, preeclampsia/eclampsia accounted for 9.4% of maternal deaths at the same hospital [5]. More recently (2002) it was found that hypertensive disease was the commonest cause for patients requiring critical care in our labour ward, with 67.5% of patients needing critical care presenting with preeclampsia or hypertensive related disease .37.5% of these patients required intensive care unit facilities [46]. Thus the increasing importance of this condition.
Standard protocols of management when used consistently and correctly can improve outcomes. In our unit at KNH, there is no standard protocol for the treatment of preeclampsia. This study aimed

to find out the various treatment approaches and outcomes in our unit with a view to recommending formulation and utilisation of standard guidelines in the management of this serious condition.

STUDY OBJECTIVES

Broad Objective

To determine the prevalence of severe preeclampsia/ eclampsia, its treatment, and outcomes at the Kenyatta National Hospital.

Specific Objectives

1. To determine the prevalence of severe preeclampsia/eclampsia in obstetric patients at the K.N.H.
2. To determine the socio- demographic characteristics of patients presenting with severe preeclampsia at K.N.H.
3. To determine the drug regimens in use at the K.N.H obstetric unit for severe P.E.T.
4. To determine the time interval between the diagnosis and delivery of patients with severe preeclampsia.
5. To determine foetal outcomes in patients with severe preeclampsia/eclampsia.
6. To determine the maternal complications
7. To determine the period of hospital stay in the patients with severe preeclampsia.

MATERIALS AND METHODS

1. STUDY DESIGN

This was a retrospective cross – sectional study.

2. STUDY AREA

This study was carried out at the Kenyatta National Hospital (K.N.H). The K.N.H is a national referral hospital and receives high risk, selected and many unbooked patients from Nairobi, its environs as well as from neighbouring hospitals, provincial and district public hospitals to the labour ward. The files of patients who have been admitted in K.N.H are available and can be retrieved with permission for study purposes.

3. STUDY PERIOD AND POPULATION

This comprised the records of patients who were admitted to the K.N.H obstetric unit with a diagnosis of severe preeclampsia and eclampsia between 1st January 1999 and 31st December 2003 both dates inclusive. This time interval was chosen because it is recent thus easier retrieval of files and various changes are being made in treatment of the disease over this period, like the introduction of magnesium sulphate.

This study was carried out between the months of January and March 2005.

4. SAMPLING

All the files meeting the inclusion criteria were used to collect data in the study.

The sample size was calculated from the formula

$$N = \frac{Z^2 P.(1-P)}{d^2}$$

Where Z- is the standard normal deviate (1.96)

P- Estimated prevalence of severe preeclampsia (it was assumed 50% of patients admitted with preeclampsia would have severe preeclampsia).

d- the level of precision with which to estimate P was taken as 5%.

Therefore $N = \frac{1.96 \times 1.96 \times 0.5 \times 0.5}{0.05 \times 0.05}$

$= 384$

All files retrieved were used for data collection.

5. INCLUSION CRITERIA

All case records of women admitted through labour ward with a diagnosis of severe preeclampsia or eclampsia during the period of admission were included in the study. This included all those with admission diastolic blood pressures 110mm Hg or systolic blood pressures 160mm Hg and other features of severe preeclampsia as described on page 141. Those who developed severe preeclampsia while in the ward were also included in the study.

6. EXCLUSION CRITERIA

All those who did not satisfy the inclusion criteria above were excluded from the study. Those admitted with convulsions that were not due to eclampsia were also excluded.

7. ETHICAL CONSIDERATIONS

This was a non-invasive study that involved review of files of patients who were managed with severe pre-eclampsia and eclampsia in K.N.H. Numbers rather than names were used to identify the questionnaires. The study was approved by the Kenyatta National Hospital Research and Ethics Committee before commencing the study.

8. STUDY LIMITATIONS

Difficulties were met where files retrieved were having inadequate information like where a patient would have been unconscious and thus difficult to get information such as period of gestation. These were included and documented as having inadequate information. The filing and retrieval method used in KNH is manual and thus it is likely that some patients with severe preeclampsia had not been entered in the register. Those who developed severe disease while in the ward are also registered with the initial diagnosis at admission and were likely to have been left out. These were overcome by the principal investigator going through all files coded as preeclampsia (ICD code 14.9) and extracting those who developed severe preeclampsia after admission. Some data entry books were missing in the labour ward records office. This was overcome by using the returns within the central records department.

9. DATA COLLECTION

A list of the in patient numbers of files of all the patients admitted to labour ward/antenatal wards with preeclampsia and eclampsia between 1st January 1999 and 31ST December 2003 was generated using the ICD coding within the records department. With the list of inpatient numbers, the files were retrieved from the registry in the records department by the records staff. The principal investigator reviewed the files within the registry. Each file was reviewed and relevant information entered into the data collection sheet under the following sections.

a) This included the socio demographic characteristics of the patient.

b) Past obstetrics and past medical history.

c) Information on the index pregnancy from antenatal period, treatment during the period of admission to delivery and the delivery outcomes plus foetal complications.

d) Maternal outcomes.

The data collection sheet was pre-tested before commencement of data collection to establish the suitability, practicability and reliability of the study questions. This was done within the K N H records department.

10. DATA MANAGEMENT

The completed data collection sheets were checked for completeness and correctness. The cleared sheets were entered into a computer for analysis using the SPSS software. Descriptive statistics were determined during the analysis. Comparison of qualitative variables was done using the chi-square test statistics to identify factors related to outcomes in patients with severe preeclampsia/eclampsia. All results will also be presented in tabular format.

RESULTS

During the study period, there were a total of 33,661 deliveries. 1692 of them had preeclampsia and of these, 384 were complicated by severe pre-eclampsia and eclampsia. The prevalence of preeclampsia was 5.02% and for severe preeclampsia was 1.14%.The socio-demographic characteristics of these mothers with severe hypertensive disease in pregnancy are as shown in table 1.

Table 1: Socio-demographic characteristics n-384

Age group	**Frequency(n)**	**% N**
15-19	23	6
20-24	96	25.1
25-29	118	30.7
30-34	95	24.7
35+	52	13.5
Parity		
0	151	39.3
1	106	27.6
2	59	15.4
3	43	11.2
4	15	3.9
≥ 5	10	2.6
Marital status		
Married	334	87
Single	45	12
Divorced	5	1
Education		
None	13	3.4
Primary	138	35.9
Secondary	154	40.1
College	79	20.6
Occupation		
None	63	16.4
Self employed	55	14.3
Employed(non-professional)	49	12.8
Employed (professional)	59	15.4
Housewife	158	41.1

The age range of the mothers was 15-46 years, the mean being 27.7 years. Only 6% of the patients were teenagers, with 80.5% being in the optimum reproductive age group of 20-34 years. Most of the patients were of low parity. Only 2.6 % were para 5 or higher. Most of the patients (87%) were married. Majority (96.6%) had formal education with only 3.4 % having never obtained any formal education. Over half of the mothers were not in formal employment, most being housewives. Only 15.4% did professional jobs.

Table 2 shows the mothers utilization of antenatal services

Table 2. Utilization of antenatal services

	Frequency	Percentage N
ANC Place	**N=384**	
City council	59	15.3
KNH	91	23.7
Government hospital	23	6
Private hospital	62	16.1
Rural health centre	29	7.6
None attended Not recorded	120	31.3
Gestation at booking in weeks	**N= 264**	
Less than (<) 20	49	18.6
21-28	118	44.7
29-36	44	16.7
>36	12	4.5
Not recorded	41	15.5
No of ANC visits	**N=264**	
1-4	137	51.9
5-10	54	20.4
> 10	6	2.3
No record	67	25.4

68.7% of the patients had attended some place for antenatal care, with 23.7% having attended the Kenyatta National Hospital antenatal clinic. However in 31.3% of cases there was no record or the patient had not attended any antenatal care.

About 63% of the patients who attended antenatal care had booked in by the 28th week of gestation and 4.5% booked beyond 36 weeks. Approximately half (51.9) of those who attended antenatal care

had 1-4 visits. The mean number made was 4 antenatal care visits before admission into the hospital. The mean gestation at diagnosis of hypertension was 32.41 weeks with a range of 11-41 weeks.

The patients prior medical history was also looked at. This illustrated in table 3.

Table 3 Previous Medical History. N= 384

	n (Yes)	% N
History of high blood pressure in previous pregnancy /ies	66	17.2
Prior history of eclampsia	9	2.3
Prior history of hypertension outside pregnancy	22	5.7
Prior history of renal disease	5	1.3
Prior history of diabetes mellitus	6	1.6

Positive history of hypertension in previous pregnancies was found in 17.2 % of the study population, while 2.3% had had eclampsia in previous pregnancies.

A small number (5.7%) had chronic hypertension while 1.3% and 1.6 % of the patients had renal disease and diabetes mellitus respectively. There were 35 (9.1%) of the patients who had family history of hypertension. Of these patients with family history of hypertension, it was mainly the mother who was hypertensive (60%).

Table 4. Findings at admission N-384

Finding	**Frequency n**	**% N**
Mean gestation(weeks) 33.88 (15-42)		
Oedema		
None	128	33.3
Pedal	198	51.6
Generalized	58	15.1
Proteinuria		
+	91	23.7
2+	143	37.2
≥3+	150	39.1
Diagnosis		
Severe PET	338	88
Eclampsia before delivery	38	9.9
Postpartum eclampsia	8	2.1

The mean gestation at admission was 33.88 weeks with a range of 15-42 weeks. Most of the patients (51.3%) had pedal oedema. Average proteinuria at admission was 2+. In 46 of the patients studied, eclampsia occured before admission.

Table 5. Blood pressure recording during the period from admission to delivery N-384

BP	Recordings mean (Range)
Highest recorded systolic	174.3 (120-250)
Highest recorded diastolic	118.4 (100-180)
Lowest recorded systolic	133.1 (80-180)
Lowest recorded diastolic	84.7 (50-170)

The highest recorded systolic pressure was 250mmHg while the highest diastolic was 180mmHg. Higher systolic pressures were associated with poorer neonatal outcomes (P value 0.021 using student's T-test). After controlling for other factors, systolic pressure did not independently affect perinatal outcome.

Table 6 illustrates the drug regimes in use during the study period.

Table 6 Drugs used for hypertension and as prophylactic anticonvulsants N-384

Treatment	n	% N
Antihypertensive alone	66	17.2
Antihypertensive and anticonvulsant	318	82.8
Antihypertensives		
Methyldopa only	41	10.7
Hydrallazine drip only	40	10.4
Methyldopa + Hydrallazine	244	63.5
Methyldopa + Nifedipine	21	5.5
Other (Combination of above drugs –such as 3 drugs)	38	9.9
Anticonvulsant/sedative		
Diazepam	85	22.1
Phenobarbitone	242	63
Magnesium sulphate	5	1.3
Other (Phenytoin)	2	0.6
No record	50	13

The drugs in use as antihypertensives included methyldopa, hydrallazine, and nifedipine. They were used alone or in combination. Anticonvulsants used included diazepam and magnesium sulphate in

order of reducing frequency. There were only 5 cases where magnesium sulphate was used. This was the time it was being introduced in the unit. Currently, it is the anticonvulsant of choice in use in KNH. Phenobarbitone was the most commonly used prophylactic anticonvulsant (72.5% of cases). In majority (82.6%) of cases, treatment was with combined use of antihypertensives and anticonvulsants.

Table 7 illustrates the indications for delivery

Table 7 Reasons for intervention N-384

Indication	n	%N
Intrauterine death	61	15.9
Reduced fetal movements/low biophysical profile	128	33.3
Uncontrollable blood pressure	58	15.1
Deteriorating renal function	5	1.3
Patient at term	79	20.6
Eclampsia	44	11.5
Other – Antepartum Hemorrhage, spontaneous labour etc	9	2.3

Most of the patients (33.3%) were delivered due to deteriorating fetal status as defined by reduced fetal movements or poor biophysical profile score <6/8. Intrauterine fetal death accounted for 15.9% of reasons for intervention and 20.6% of the patients were delivered as they had reached term gestation.

Table 8: Modes of intervention and delivery

	Frequency	Percentage
Gestation at intervention		
< 28	26	6.8
28-31	50	13
32-35	116	30.2
≥36	192	50
Mode of intervention		
Spontaneous labour	76	19.8
Induction of labour	108	28.1
Elective C/section	10	2.6
Emergency C/section	190	49.5
Mode of delivery		
SVD	130	33.9
Caesarean section	247	64.3
Vacuum extraction	5	1.3
Vaginal breech	2	0.5

The mean gestation at intervention was 34. 57 weeks with a range from 20 –42 weeks. About 30% were delivered between 32 and 35 weeks with 6.8 % being delivered below 28 weeks gestation, the earliest being at 20 weeks. Steroids were administered in 71 (18.5%) of the patients for lung maturation in those below 34 weeks of gestation.

The main mode of intervention was emergency caesarean section (49.5%) followed by induction of labour in 28.1%. Most patients were eventually delivered by caesarean section (64.3%). Assisted vacuum delivery was done in only 1.3 % while 33.9 % had spontaneous vertex deliveries.

As most deliveries were by caesarean section, the indications for the operations were looked into. These are shown in table 9.

Table 9: Indications for caesarean section. N-247

Reason	**n**	**%N**
Foetal distress	102	41.3
Failed induction	25	10.1
Previous C/Section scar	36	14.6
Malpresentation	11	4.5
Abruptio placenta	9	3.6
Other Eclampsia with poor, Bishop score, multiple pregnancy, CPD	64	25.9
Total	**247**	**100.0**

Most patients were done caesarean section due to foetal distress. 10% of the caesarian sections were due to failed induction of labour. Some of the patients on induction were also operated on for other reasons such as foetal distress. It is operation diagnosis that was used to enter the indication for caesarean section during data collection.

Table 10. Foetal outcomes

Birth weight N-384	**n**		**Percentage N**	
< 1000	40		10.4	
1001-1500	65		16.9	
1501 –2000	73		19	
2001- 2500	65		16.9	
2501-3000	78		20.4	
> 3000	63		16.4	
Apgar score N-384	**One Minute**		**5 Minutes**	
	Number	**%**	**Number**	**%**
0	89	23.2	89	23.3
1-4	26	6.8	10	2.6
5-7	93	24.2	57	14.7
8-10	176	45.8	228	59.4
Total	**384**	**100.0**	**384**	**100.0**
Foetal deaths N-102	**n**		**% N**	
Fresh still births	22		22.5	
Macerated still births	67		55.9	
Early Neonatal deaths	13		21.6	
Total	**102**		**100**	

The mean birth weight was 2235g with a range from 500g to 4600g. Approximately two thirds (63.2%) of the babies were of birth weight below 2500g. Most of the babies (45.8% and 59.4%) had good Apgar (>7) scores at 1 and 5 minutes respectively. However, 30 % and 25.9% of the babies had poor Apgar (4) scores at 1 and 5 minutes respectively. There were 102 perinatal deaths, giving a perinatal mortality rate of 266 per 1000 deliveries. Of these, 13 (12.7%) were early neonatal deaths.

Table 11: Complications in the newborns N-126

Complication	Frequency	%N
Respiratory distress	69	54.8
Asphyxia	7	5.6
Prematurity	46	36.5
Congenital Malformations	4	3.1
Total	**126**	**100.0**

Of the infants born alive, 32.8% (126) developed complications and among the babies with complications, most had respiratory distress (54.8%) and prematurity (36.5%)

Table 12 shows the outcomes according to the birth weight at 24 hrs

Table 12: Perinatal status at 24 hrs

Birth weight (g)		<1000	1001-1500	1501-2000	2001-2500	2501-3000	3001-3500	>3500	Total
Survived	n	2	35	53	59	74	44	15	**282**
	%	5	53.8	72.6	90.8	94.9	93.6	93.8	73.4
Died	n	38	30	20	6	4	3	1	**102**
	%	95	46.2	27.4	9.2	5.1	6.4	6.2	26.6
Total	n	40	65	73	65	78	47	16	**384**
	%	10.4	16.9	19	16.9	20.4	12.2	4.2	**100**

The birth weights ranged from 500 grams to 4600g. The neonatal survival increased with increasing birth weight, with over 93% of those weighing above 2500g surviving. The death rate increased with decreasing birth weight. The birth weight thus significantly influenced neonatal survival, with a P value < 0.001 using the pearsons chi-square. After controlling for other variables, the birth weight was found to significantly influence perinatal outcome, OR 1.7 (1.2-2.5,95%C.I).

The neonatal survival was also related to gestational age at delivery, with 50% of those born by the 31st week surviving, compared to 74% survival rate at 34 weeks and 90% after 36 weeks,

P value < 0.001.

Table 13: PERINATAL OUTCOME VERSUS APGAR SCORE AT 5 MINUTE AND THE MODE OF DELIVERY

	Survived		Died		Total	
Apgar Score At 5minutes	**No**	**%**	**No**	**%**	**No**	**%**
0-4	2	2.2	97	97.8	99	25.9
5-7	54	94.7	3	5.3	57	14.7
8-10	226	99.1	2	0.9	228	59.4
Total	**282**	**73.4**	**102**	**26.6**	**384**	**100**
Delivery-(route of)						
Vaginal	59	43.1	78	56.9	137	35.7
C/section	223	90.3	24	9.7	247	64.3
Total	**282**	**73.4**	**102**	**26.6**	**384**	**100**

The Apgar score at one minute significantly affected neonatal survival, with those scoring ≥ 8 at one minute having 99% survival compared to 2.2% for those scoring ≤ 4, P value < 0.001. Similar trends were found for the 1 minute Apgar scores.

Majority (64.3%) of the deliveries were conducted by caesarean section (C/S), compared to 35.7% conducted vaginally. 56.9% of adverse outcomes occurred in those delivered vaginally compared to 9.7% among the C/S delivery group. The mode of delivery significantly influenced the perinatal outcome, P < 0.001 using Pearsons Chi square.

Table 14: Logistic regression analysis for independent correlates of perinatal outcome

Independent correlates	Coefficient	P value	OR(Odds Ratio)	95% C.I for OR Lower Upper
Apgar score at 5 minutes	0.727	0.001	2.069	1.741 2.460
Birth weight	0.536	0.008	1.710	1.154 2.534
Caesarean delivery	1.214	0.038	3.368	1.068 10.621
Constant	-4.974	0.000	0.007	

As shown in table 14 above, live births were independently associated with Apgar score at 5 minutes OR 2.1 (1.7-2.5 95%CI), and higher birth weights 0R 1.7(1.2-2.5, 95%CI) and caesarean delivery OR 3.4(1.1-10.6 95%CI).

A larger number of the mothers (51.6%) attended antenatal care. About 35.3% of adverse outcomes occurred in those who did not attend ANC compared to 22% among those who attended ANC. Failure to attend ANC care was significantly associated with a poorer perinatal outcome. $P < 0.007$ and 0.008 but did not independently influence perinatal outcome.

Table 15: Types of maternal complications n=93

Complication	Frequency	% N
Renal failure	15	3.9
Cerebral Hemorrhage	2	0.5
Eclampsia (Intrapartum and Postpartum)	13	3.4
Abruptio placenta	9	2.3
Difficult reversal from anesthesia	12	3.1
Pulmonary Oedema	14	3.7
Sepsis	13	3.4
Post partum hemorrhage	4	1.0
Visual disturbance	3	0.8
Laryngeal Oedema	6	1.6
Anaemia	2	0.5
Total	**93**	**24.2**

NB: For purposes of the study, antenatal eclampsia was considered hypertensive disease while intrapartum and post partum eclampsia were considered a maternal complication.

About one quarter (24.2%) of the study population developed complications, with the most common complications being renal failure (3.9%), pulmonary oedema (3.7%) eclampsia 3.4%, poor reversal from anesthesia (3.1%) and sepsis (3,4%). Of the 93 patients who developed complications, 25 had to be admitted to the Intensive Care Unit (ICU) while the rest were managed in the labour ward acute room, lying –in wards and the renal unit. Table 17 shows the indications for ICU admission.

Table 16. Intensive Care Admissions n=25

Indication	**Frequency**	**% n**
Pulmonary Oedema	9	36
Poor reversal from anesthesia	12	48
Cerebral hemorrhage	2	8
Laryngeal oedema	2	8
Total	**25**	**100**
Duration of stay (days)		
1-2	20	80
3-4	2	8
≥ 5	3	12

Most of those admissions to ICU were due to poor reversal from anesthesia. Majority stayed In I.C.U for 2 days. Of the 15 patients who developed renal failure, 10 underwent dialysis while 5 were managed conservatively.

Table 17 Duration of stay in hospital

Duration (days)	Frequency	(%)N
≤ 10	87	22.7
11 – 15	131	34.1
16 – 20	88	22.9
21 – 25	53	13.8
≥ 25	25	6.5
Total	**384**	**100**

The mean duration of stay in hospital was 13.7 days (SD) with a range of 1 to 78 days. Most were hospitalized for 11 to 15 days.

Table 18 Duration on antihypertensives after delivery N=384

Duration (days)	Frequency	%N
≤ 2	68	17.7
3 – 6	176	45.8
7-14	87	22.7
> 14	53	13.8
Total	**384**	**100**

Most were on antihypertensive for between 3-6 days after delivery. 13.8 % were on treatment for more than 14 days.

There were 10 maternal deaths in this study giving a mortality rate of 26/1000 deliveries.

The causes of maternal mortality were :

Acute renal failure - 3 patients

Cerebral hemorrhage - 2 patients

Pulmonary Oedema/cardiopulmonary arrest- 5 patients

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DISCUSSION

In this study, 384 patients were seen with severe pre-eclampsia in the period from 1st January 1999 to 31st December 2003 both dates inclusive. In this period there were 33661 deliveries. Their files were reviewed and analysis conducted. Hypertensive disease is a major cause of maternal and perinatal morbidity and mortality. The prevalence of severe hypertension was found to be 1.14%.

Like in previous studies, the incidence of hypertensive disease was higher in primigravidae, accounting for 39.3% of all cases. Only 6.5% of the cases were para 4 and above. Majority of the patients were young, with 61.5% being below 30 years. Only 5.5% were teenagers. The mean age was 27.7 years. The majority (96.6%) had received formal education. About 68.7% of the patients had received antenatal care, with 74.9% having booked for antenatal care by the 28th week of gestation. Our study indicated an advantage among the mothers who had antenatal care compared to those who had none, though this was not an independent variable. This has also been found in other studies[12-15,17].

The drugs in use as anti-hypertensives include α-methyldopa, hydrallazine, and nifedipine. They were used singly or in combination. The anticonvulsants used included diazepam magnesium sulphate and phenobarbitone.

Reduced fetal movements was the most frequent indication for delivery (33.3%). Other common indications were patient at term, intrauterine foetal death and uncontrollable blood pressure. Emergency caesarean section was the commonest mode of intervention (49.5%). Induction of labour was done in 28.1% while 19.8% went into spontaneous labour. 64.3% of the patients were delivered by caesarean section. This rate is high compared to a figure of 17.3% reported by Karanja (1980) among the general obstetric patients at KNH. This high rate may be explained by the high number of patients with deteriorating fetal status (reduced movements or poor biophysical profile). It can also be justified by the fact that all these patients had severe disease to warrant early or immediate intervention. In addition, 23.1% of patients that were induced were eventually delivered by cesarean section.

The eventual mode of delivery was significantly associated with the perinatal outcome, with those delivered by cesarean section having better outcomes (P value 0.001, OR3.3).Logistic regression

analysis including other factors revealed that Apgar score at 5 minutes, birth weight and caesarean section delivery were the factors influencing perinatal outcome.

The mean gestation at intervention was 34.5 weeks with a range 20-42 weeks. The gestation at delivery was significantly associated with eventual neonatal survival, with survival increasing with increasing gestation and vice versa ($P < 0.001$). Neonatal survival increased with increasing birth weights, with 94% of those weighing above 2500g surviving. 63.2% of the babies weighed less than 2500g. Birth weight was independently associated with influencing the perinatal outcome, OR 1.7(1.5-2.5 95%CI)[13-15]. This is similar to other studies where hypertensive disease in pregnancy has been shown to contribute significantly to the rates of low birth weight babies and prematurity.

The Apgar scores significantly affected neonatal survival. The lower the score, the more adverse the outcomes, $P < 0.001$. Poor Apgar scores in hypertensive disease are thought to be due to chronic anoxia secondary to reduced utero – placental perfusion. The Apgar score at 1 and 5 minutes significantly influenced neonatal survival with 99% of those scoring ≥ 8 surviving, compared to 2.2% for those scoring 1-4, $P < 0.001$. The Apgar score independently influenced perinatal outcome with higher scores having favourable outcomes 0R 2.1(1.7-2.5 95%CI). Other studies have also found similar trends[13-15].

The perinatal mortality rate of 266/1000 deliveries in this study was higher than that reported by Kibaru at the KNH in 1990, and that found by Wangwe (2002) of 222/1000 at the Pumwani maternity hospital[12,14]. Hibbard suggested that with a good antenatal care, perinatal loss related to pre-eclampsia/eclampsia should not exceed 20/1000. Our study groups rate was above this. This may be related to failure to attend antenatal care as well as non focused antenatal care services.

The poor perinatal outcome in severe preeclampsia is usually due to poor placental perfusion.

The ultimate goal of any protocol for the management of pre-eclampsia must be maternal safety first, followed by delivery of a live mature newborn in optimal conditions. Short of immediate caesarean delivery for all women with severe pre-eclampsia, the optimal timing for delivery remains elusive, and thus some element of expectant management exists in all treatment protocols. The chief aim of expectant management is to be able to deliver the baby on the best day, using the best way and in the best place. In our study, the period of prolongation ranged from 0-61 days with most being delivered within 7 days of admission 42.9% were delivered within one day of admission. This period did not significantly affect perinatal outcome (Chi- square 0.529 P 0.768).

There were 10 deaths during the study period. They were due to acute renal failure, cerebral hemorrhage and cardiopulmonary, failure. This is comparable to other studies. For the patients who survived, the mean duration of stay in hospital was 13.7 days with the majority (31.5%) staying 11-15 days.

During the period of study, 24.3% of the patients developed complications. These included renal failure, pulmonary oedema, anesthetic complications, sepsis, cerebral hemorrhage and placental abruption.

CONCLUSIONS

Severe preeclampsia was more common in women of low parity.

The most commonly used drugs (antihypertensive) at KNH were methyldopa, hydrallazine and nifedipine. The Apgar score, birth weight, and caesarean delivery were independently influence perinatal outcome. Other factors found to be associated with influencing the outcome were gestational age at delivery and utilization of antenatal care services.

Fetal factors contribute significantly to preterm delivery of women with severe pre-eclampsia.

Severe preeclampsia contributes significantly to overall perinatal mortality and to maternal morbidity and mortality at the KNH.Severe pre-eclampsia also contributes significantly to prolonged hospital stay.

RECOMMENDATIONS

Promotion of health education to the reproductive age group will help to increase awareness of hypertensive disease and to also motivate antenatal care attendance. This will help in the early diagnosis and management of this disorder.

There is a need to have a standard protocol for the treatment of preeclampsia at the Kenyatta national hospital which can then be adapted countrywide. Prompt management of preeclamptic patients with timely intervention will help in reducing maternal morbidity and mortality.

Further research is required to evaluate the role of caesarean delivery in mothers with severe pre-eclampsia. There were better outcomes in those delivered by caesarean section. This is especially necessary for those patients remote from term gestation.

A study is needed in KNH to evaluate specifically the use of magnesium sulphate in the treatment of preeclampsia/eclampsia and find out the outcomes of both the mothers and babies since the introduction of magnesium sulphate.

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GYNAECOLOGY SHORT CASE 1

INCOMPLETE ABORTION –MANUAL VACUUM ASPIRATION

NAME : L.M. L.M.P. 24.3.04

AGE : 17 Years PARITY . 0 + 0

O.P NO. : 34956/04

D.O.A. : 1/7/04
D.O.D. : 1/7/04

PRESENTING COMPLAINTS.

The patient presented with low abdominal pain, backache and per vaginal bleeding for four hours before arrival to hospital.

HISTORY OF PRESENTING ILLNESS

The patient was well until the morning of consultation when she started experiencing low abdominal pains. This was accompanied by backache and soon after followed by vaginal bleeding that was of moderate amounts. She had changed three soaked pads by the time she arrived. She had missed her periods for the last three months. She did not have any fever and the blood was not foul smelling. She did not give a history of having interfered with this pregnancy.

OBSTETRIC AND GYNAECOLOGICAL HISTORY.

She attained her menarche at 13 years. Her last menstrual period was on 24.3.04 giving her a 14 weeks period of amenorrhoea at the time of presentation. Her menstrual cycles were regular occurring every 28-30 days and duration of bleeding of 3-4 days. She gave no history of dysmenorrhoea. She was a primigravida and had not used any contraceptives before. She had coitarche at 15 years and had had two sexual partners since then.

PAST MEDICAL HISTORY

She had never been admitted to hospital before and had no chronic illnesses. She had no known allergies to foods or drugs.

FAMILY AND SOCIAL HISTORY

She was the first born in a family of two siblings. There was no history of chronic illnesses in her family. Her parents live in Kakamega while she lived in Nairobi where she worked as a house- help. She was single.

SYSTEMIC REVIEW

This was nil contributory.

PHYSICAL EXAMINATION
She was in fair general condition, was not pale, not jaundiced and had no oedema. Her clothing was blood stained.

ABDOMINAL EXAMINATION
The abdomen was not distended and was moving well with respiration. The suprapubic area was tender though no masses were palpable. The rest of the abdomen was normal.

PELVIC EXAMINATION
The external genitalia was normal grossly though blood stained, active bleeding was evident. The products of conception were felt in the vaginal cavity and the cervix was three centimeters open. The uterus was bulky, soft and slightly tender on bimanual palpation. Both adnexae were free and had no palpable masses. The pouch of Douglas was not full. There was blood on the examining fingers with clots.

CARDIOVASCULAR SYSTEM
Her pulse rate was 80/minute, of normal volume. The blood pressure was 100/60mmHg.The apex beat was normal and there were no murmurs.

RESPIRATORY SYSTEM
The respiratory rate was 24/minute. She was not in distress. Air entry was equal in both lung fields. No crepitations were heard.

NERVOUS SYSTEM
She was conscious, well oriented in time, place and person. Her sensory and motor activity was normal.

IMPRESSION
An impression of incomplete abortion was made.

INVESTIGATION
1. PCV-31%

MANAGEMENT

An intravenous access canula was inserted and a drip of normal saline set up. She was planned for manual vacuum aspiration (MVA). She was informed of the procedure of which she consented to.

UTERINE EVACUATION BY MANUAL VACUUM ASPIRATION

In the procedure room, the MVA equipment was assembled and the procedure done using the following steps:
The patient was placed in lithotomy position. Vulval toilet was done with a swab mounted on a sponge holding forceps. She was then draped with sterile drapes. A cuscos speculum was inserted into the vagina and the cervix exposed. The cervix was cleaned and clots expelled, its anterior lip was then held with a tenaculum forceps. The cervix

was inspected for any lacerations or injuries, of which there were none. Karman's canula size 12 was aseptically introduced into the uterus up to the fundus and the double valve syringe with vacuum connected. Uterine curettage was done with in and out stroking motion of the canula until the procedure was judged complete by a gritting sound and uterine contraction tightening on the canula. Approximately 80mls of products of conception were evacuated which were not foul smelling. The Karman's canula was removed and the syringe disassembled. The cervix and vagina were cleared of any remaining clots and the cuscos speculum removed. The external genitalia were dried and the patient provided with a perineal pad to monitor for any bleeding.

POST MVA CARE

The patient was taken to the recovery place and was put on metronidazole 400mg 8 hourly, doxycycline 100mg twice daily, to cover any secondary infections, ranferon 10mls daily and mefenamic acid 500mg three times daily for pain. Her condition stabilized and she was discharged home on the same day. She was advised on modern contraceptive methods and booked to the adolescent clinic for counseling and follow up.

FOLLOW UP

She did not honour her appointment.

DISCUSSION

A case is presented of a 17 years old primigravida with spontaneous incomplete abortion that was managed by manual vacuum aspiration and discharged home on antibiotics.

She was at a gestation of 14 weeks when she developed vaginal bleeding. Abortion is defined as termination of pregnancy that is less than 20 weeks (139 days) gestation or foetal weight less than or 500g. The weight of 500g according to the World Health Organisation is the bare minimum that a foetus can survive [1]. Abortion can be classified as spontaneous or induced. Our patient had spontaneous abortion as there was no evidence of interference. Abortion can further be classified into complete, incomplete, inevitable and missed abortion. Our patient had incomplete abortion and did not have features of sepsis. An abortion can also be septic or non-septic. In legal terms abortion can be classified as therapeutic and illegal abortion. The larger the gestation at the time of termination, the higher the chances of serious complications such as haemorrhage, sepsis and perforation of the uterus or even death. Abortion has been and is still being used as a method of fertility regulation or control, or as a back-up method for contraceptive failure. It may also be related to inadequate family planning knowledge and services [2]. This patient had never used any contraceptive previously.

Incomplete abortion is defined as expulsion of some but not all of the products of conception before 20 weeks of gestation. Before the 20th week the foetus and placenta are likely to be expelled together in the abortion process [1]. The patient presented had a spontaneous incomplete abortion as some products of conception could still be felt through the cervix on pelvic examination.

Our patient had spontaneous abortion. But sometimes patients may not volunteer information on having procured abortion services due to legal implications.
It is estimated that about 50 million pregnancies are terminated each year around the world. About 20 million of these are unsafe abortions that contribute considerably to maternal morbidity and mortality, especially in the developing countries. Recent estimates suggest that 20-50% of pregnancy related mortality in East, Central and Southern Africa is due to unsafe abortion. Where it is legal, it is generally safe and where abortion is not legalized, complications are more common and approximately 150,000 women die every year from these complications. Induced abortion is illegal in Kenya unless being done on medical grounds and thus most patients presenting with complicated abortions do not volunteer information on interference. Legal abortion was introduced was introduced in South Africa in 1997, making it one of the few countries in Africa with such legislation [3-7].

No identifiable cause of the abortion was found in the patient presented though histological examination of evacuated material was not done because of high costs which had to be met by the patient. Causes of spontaneous abortion are varied. Chromosomal anomalies cause 50-60% of early abortion (first trimester). Autosomal trisomy is the most frequently identified chromosomal anomaly associated with abortion followed by monosomy (45X0). Abnormal zygote development, polysomy and triploidy are the other

abnormalities associated with abortion. Other causes include infection, anatomical defects, endocrine factors, immunologic factors, maternal systemic disease, emotional causes and trauma. Hyperthyroidism, hypothyroidism, uncontrolled diabetes mellitus and infections such as TORCHES (Toxoplasmosis, cytomegalovirus, herpes simplex, rubella and syphilis), brucellosis, mycoplasmosis and malaria are all known to cause abortion. Insufficient progesterone from the corpus luteum or the placenta also causes pregnancy loss. Environmental factors or habits such as excessive alcohol consumption, cigarette smoking also increase the risk of abortion. Antiphospholipd antibodies and connective tissue disorders such as systemic lupus erythematosis, rheumatoid arthritis have also been associated and found to increase the risk of abortion and recurrent foetal death. Uterine defects like fibroids, synaechiae, mullerian defects and incompetent cervix are associated with recurrent pregnancy losses [1,4,5,7,8,10].

In our patient, the diagnosis of incomplete abortion was made clinically from her diagnosis and physical examination. History and physical examination is sufficient to make a diagnosis of abortion. History of vaginal bleeding, low abdominal pain after a period of amenorrhoea is highly suspicious of abortion. The range of presentation is varied in that some patients are stable while others present with severe haemorrhage and severe haemodynamic derangement and shock. Our patient had vaginal bleeding but was not in shock. In spontaneous early abortion, haemorrhage, necrosis and inflammation appear in the region of implantation. Clinically, this presents as low abdominal pains and persistent vaginal bleeding especially in incomplete abortion. On pelvic examination, the cervix is usually dilated as found in our patient. Other diagnostic aids include pregnancy test which will indicate falling or abnormally low levels of beta human chorionic gonadotrophin, blood studies indicate anaemia, vaginal smears for karyotyping which will show a karyopyknotic index of less than 10 if there are chromosomal abnormalities. Ultrasonography demonstrates retained products of conception in the uterine cavity. Differential diagnoses of incomplete abortion include membranous dysmenorrhoea, hyperoestrogenism, hydatidiform mole, pedunculated myoma and cervical neoplasia [1,2].

Our patient was started on intravenous normal saline and had manual vacuum aspiration (MVA) upon diagnosis of incomplete abortion and was started on antibiotics immediately after the procedure.

Successful management of abortion depends on early diagnosis. Antibiotics should be started on admission before evacuation is done. Stabilisation of the patient is achieved by use of plasma expanders or blood transfusion where necessary. Uterine evacuation to stop the bleeding is the ultimate procedure in management of incomplete abortion. In our unit this is performed by use of manual vacuum aspiration (MVA) technique. MVA is safer and easier to perform than use of sharp curettes in managing incomplete abortion. Additionally increased awareness of the risks of general anaesthesia prompted the procedure to be moved from an operating room to outpatient facility with the patient under regional anaesthesia or sedation [8,9]. The technique of vacuum aspiration is simple and provides developing countries (Kenya included) with the simplest safe method of uterine evacuation even in cases of hydatidiform mole. The equipment for MVA is composed of a sterile syringe and a flexible Karman's canula. Both single and double valve syringes are available. The polythene flexible canulae are in sizes 4-12mm. Except

for size 12 canula that fits directly, an adapter is needed for the other canulae when used with a double valve syringe to provide airtight connection. A vacuum of 60mmHg created when the valves are locked and the plunger pulled up is used for the evacuation procedure. The evacuation is done on an ordinary examination table in the procedure room within the ward in our unit. Evacuation is judged complete when four or more of the following are noted during the procedure;

- Appearance of air bubbles within the syringe
- Gritty feeling of canula against the endometrial wall
- Tight gripping of canula by the uterus
- No further flow of tissue when syringe is not full or canula not blocked
- Only minimal blood flow
- Patient complaining of griping lower abdominal pains

During the procedure, our patient was continuously talked to by the doctor and assisting nurse. During the procedure, conversation is kept between the patient, surgeon and assisting nurse. Analgesia is given as cervical block. The canula is inserted once to minimize infection. Postoperatively the patients are observed in the ward for six hours then discharged home on oral broad-spectrum antibiotics (metronidazole and doxycycline in our unit). Kizza[9] at Kenyatta hospital found that Karman's canula and syringe was safe especially in early gestation. Complications of MVA are rare and mainly include sepsis and haemorrhage. Thus prophylactic antibiotics are instituted to prevent infections. Makokha observed that 22.2% of maternal deaths were due to post-abortal sepsis, 85% having evidence of interference and haemorrhage was second to sepsis as cause of maternal deaths.

This patient was adviced on contraception but unfortunately did not turn up for provision of a method. This was a missed opportunity to provision of comprehensive care post abortion. The concept of postabortal care (PAC) formulated by governments at the international conference for population and development in Cairo in 1994 and reaffirmed at the Cairo+5 meeting has gained wide acceptance as one way of improving services provided to women with complications from spontaneous or induced abortions. This would help to break the cycle of repeat abortion and reduce maternal morbidity and mortality. One of the elements of PAC is post abortion family planning counseling and services. All modern methods of contraception are appropriate for use after treatment of abortion. This includes hormonal methods as long as the provider gives adequate counseling and screens women for the standard precautions for use of the particular method [12]. In addition PAC offers training to mid-level health workers on post abortion care.

This patient did not develop any complications during or after the MVA. The complications of abortion include severe haemorrhage, sepsis, uterine perforation during induced abortion or evacuation, injury to the bowel and bladder during evacuation or criminal abortion and fistula formation. Other complications include developing choriocarcinoma, uterine synechiae after excessive curettage, infertility and death from shock (septic or hypovolaemic) [8,10,11,12].

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GYNAECOLOGY CASE 2

BARTHOLIN'S ABSCESS – MARSUPIALISATION

NAME: J.N.

AGE: 39 Years.

I.P NO. 0984181

D.O.A ; 17/10/2004.

D.O.D: 18/10/2004.

Parity: 2 + 0

Last delivery: 1996.

L.M.P: 3/10/04

PRESENTING COMPLAINTS

She presented with a three-day history of pain and swelling of the vulva.

HISTORY OF PRESENTING COMPLAINTS

JN had been well until 3 days prior to admission when she noted a painful swelling on her external genitalia. Due to the pain she was not able or walk properly. She had no history of vaginal discharge prior to the above complaints. She had no history of fever or chills.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She was a Para 2+0. Her first delivery was by caesarean section due to twin gestation with first twin in breech position. Her second delivery was in 1996 also by caesarean section due to cephalopelvic disproportion. All the children were alive and well.
Her menarche was at 15 years, her periods were regular occurring every 28 days and lasting 3 days. She had used Depo-Provera up to 1999 and currently was on natural family planning and barrier methods (condoms). She had no prior history of having had sexually transmitted infections.

PAST MEDICAL AND SURGICAL HISTORY

She had been treated for bartholin's abscess on the same side in 1997 at a private hospital.She had no known allergies to food or drugs.

FAMILY AND SOCIAL HISTORY

She was married and lived with her family at Kahawa West in Nairobi. She worked as a secretary with Survey of Kenya. She did not smoke cigarettes or take alcohol. She had no family history of chronic illnesses.

PHYSICAL EXAMINATION

She was in pain and could not sit comfortably on a chair. She was not pale and clinically afebrile. Her vital signs were blood pressure of 110/70mmHg, pulse rate 80/minute, respiratory rate 20/minute and temperature of 36°C.
Her respiratory, cardiac and nervous systems were normal.

ABDOMINAL EXAMINATION

Her abdomen was not distended and moved well with respiration. She had a sub umbilical midline scar. There were no areas of tenderness and no masses were felt.

VAGINAL EXAMINATION

There was a tender fluctuant swelling 4 by 2cm involving the Bartholin's gland and labia minora on the right side. The left vulva was normal. She had some whitish discharge at the introitus. Digital vaginal examination and bimanual pelvic examination were not done due to the tenderness.

DIAGNOSIS

A diagnosis of acute Bartholin's abscess was made.

INVESTIGATIONS

1. Packed cell volume 45 % (Hb 15g/dl).
   White cell counts 6.3 x 10^9/l

2. Urea and electrolytes- Na^+ 136 mmol/l
   K^+ 3.5 mmol/l
   Urea 2.5 mmol/l
   Creatinine 58umol/l

MANAGEMENT

She was informed of the diagnosis and mode of treatment with marsupialisation. She gave informed consent. The vulva was shaven clean. She was then premedicated with atropine 0.6mg and pethidine 100mg intramuscularly half hour before the operation. She was then wheeled to theatre for marsupialisation.

Marsupialisation

In theatre, general anaesthesia was induced and she was placed in the lithotomy position. A vulvo-vaginal toilet was then done, she was catheterised and bladder drained of clear urine. After sterile draping of the vulval area, examination under anaesthesia revealed a right bartholins abscess measuring 4cm by 2cm. The mass was fluctuant. The left labia

were normal. Bimanual examination revealed a normal sized uterus, adnexa and Pouch of Douglas were free. There was no abnormal discharge on the examining finger. The vagina was packed with sterile gauze to prevent contamination with pus. A longitudinal incision was then made over the abscess along the mucocutaneous junction just lateral to the hymenal ring.
About 10mls of foul smelling pus were drained (a sample for culture and sensitivity taken was not analysed as the laboratory involved was not functional on weekends-the operation was done on a weekend). The cavity of the abscess was explored and locules broken with a finger and all the pus drained. The cavity was cleaned thoroughly. The linings of the cavity left were then everted by stitching to the vulvovaginal mucosa using interrupted sutures of 2/0 vicryl. This created a new stoma for the secretions. There was minimal bleeding and a pack soaked with povidone iodine was left in the abscess cavity. Vulval toilet was done and she was then reversed from anaesthesia.

Postoperative care

She was observed until fully awake while in the theatre recovery room, then every 2 hours thereafter. She was given intravenous metronidazole 500mg, crystalline penicillin 2MU 6 hourly and gentamycin 80mg 8 hourly for the first 24 hours after operation. She was given ibuprofen 400mg three times daily for pain relief. The vulval pack was removed after 12 hours.
24 hours after operation, she was reviewed and found to be stable. She had no oozing from the operation site, she could walk and sit well. She only had minimal pain at the site of the procedure. She was taught on how to clean the area and to have salt-water sitz baths at least three times a day. She was started on oral metronidazole 400mg thrice daily, amoxicillin/clavulanic acid 1g twice daily for two weeks and ibuprofen 400mg three times daily for five days. She was discharged home on this treatment and was to be reviewed in the gynaecology out patient clinic after 2 weeks.

FOLLOW UP

She came as scheduled and had no complaints. The site of surgery had healed well.
She was advised on regular Pap smear and to continue visiting the family planning clinic.

DISCUSSION

The case presented is of a 39 years old para 2+0 who presented with recurrent Bartholin's abscess and was treated by marsupialisation. She was well at the follow up visit. Bartholin's glands are a pair of compound racemose glands lined by cuboidal or columnar epithelium, one on each side of the vaginal introitus. They are situated beneath the vestibule on either side of the introitus and measure 0.5 x 1.0cm in diameter. They are the major vestibular glands and lie under the constrictor muscle of the vagina, they are partially covered in the vestibular bulbs. The gland ducts are 1.5-2.0cm long and open on the sides of the vestibule just outside the lateral margin of the vaginal orifice at 5 and 7 o'clock positions. They secrete a clear, viscid and stringy mucoid, alkaline fluid during sexual arousal, which may act as a lubricant for coitus[1,2]. After about 30 years of age the glands undergo involution and become atrophic and shrunken. The patient presented was 39 years old.

Our patient had a recurrence of this abscess but did not give history of having a sexually transmitted infection previously. The recurrence could probably have beeen due to scarring after the initial procedure causing obstruction of the outflow tract for the gland sectretions. Bartholin's abscess results from obstruction of the duct by various causes. This results in retention of secretions and cystic dilation. Apart from infection, obstruction may also result from insipissated mucus, congenital narrowing of the duct, iatrogenic following medio-lateral episiotomy[3]. Little is known about the epidemiology of Bartholin's gland infection and its complications in women. It is commonly associated with women of age 20-29 years, multiparity and low socio-economic status[1]. Bartholin's abscess is not related to ethnicity or marital status. These women often have prior or concomitant sexually transmitted infection (STI) particularly gonorrhoea or chlamydial infection. The demographical characteristics have led to the conclusion that acute Bartholin's infection is probably an STI. However, recurrent infection or abscess may be the consequence of scarring of the gland duct damaged by prior infection[1].. The incidence of Bartholin's abscess at KNH was found to be 1.9% with mean occurrence at age 22.5 years. Approximately 1.7% of acute gynaecological admissions in KNH were found to be due to this condition and that 50% of patients were aged 18-23 years [4,5].

The microbiology of Bartholin's gland infection is similar to that of pelvic inflammatory disease[2,3]. Most abscesses have mixed aerobic and anaerobic pathogens. In up to one third of cases no pathogen is isolated by culture. Organisms that have been isolated include Neisseria gonorrhoea, E. coli, proteus, streptococci faecalis, staphylococcus aureus, trichomonas vaginalis, candida albicans and chlamydia[6]. Culture was not done for our patient due to closure of the laboratory at the time the procedure was done.
Our patient presented with a three day history of vulval pain, and associated swelling. Bartholin's abscess usually presents as an acute gynaecological problem with symptoms of severe pain, tenderness, difficulty in sitting or walking, dyspareunia and swelling of the vulva. There may be purulent discharge and obvious signs of local inflammation. Our patient had pain, swelling and difficulty in walking. Constitutional symptoms of general malaise, fever and headaches may also be present. Ductal infection alone is unusual. The abscess is generally unilocular and several centimeters in diameter. The surrounding

erythema, induration and tenderness may obscure actual abscess size. This patient did not have any constitutional symptoms.

This patient had marsupialisation of the abscess. In addition she was started on antibiotics and analgesics for pain relief. The principles of management of Bartholin's gland abscess includes pain relief and surgical drainage of the pus. Several surgical techniques have been proposed for its management. These include incision and drainage, marsupialisation and catheter drainage. None of these methods has undergone randomized prospective evaluation in sufficiently large trials to exclude differences in outcome[6]. Other surgical methods include aspiration, use of an inflated bulb type catheter, complete excision and lately by window operation and laser surgery [7,8,9].

There are four main goals for the surgical treatment of Bartholins abscess. First, there must be adequate surgical drainage of the infected gland and abscess. Second, the gland should be preserved so that it may continue with its secretory function. Thirdly, recurrences should be prevented by creation of a new gland ostium or fistula to replace the function of the presumed damaged or occluded duct. Fourth, complications of the infection such as necrotising fascitis and sepsis should be prevented. Our patient did not develop sepsis after the procedure.

Our patient had marsupialisation under general anaesthesia and was on antibiotics during the postoperative period. She was adviced to have saline sitz baths three times daily to ensure cleanliness around the operation site.
Marsupialisation is the commonly used technique involving making a new ductal orifice. The abscess contents are drained and wall opened. The lining of the wall is then everted and approximated to the vaginal mucosa with interrupted delayed absorbable sutures. This ensures that the new orifice remains open and patent postoperatively minimizing the risk of recurrence as well as blood loss. The procedure can be done under local, regional or general anaesthesia[3, 8,9]. The patient should be given appropriate antibiotics and analgesics. Saline sitz baths should be started on the third or fourth postoperative day.

Marsupialisation is associated with a recurrence rate of 10-15%. This probably results from healing with fibrosis resulting in closure of the duct. The patient may also develop dyspareunia due to the fibrosis. Our patient had a recurrence after similar procedure seven years prior to the index admission.

In some centers, the Word catheter is used for treatment. It accomplishes the same results as surgery with minimal or no trauma. Under local anaesthesia, a small incision is made at the appropriate position of the normal orifice. The Word catheter is inserted through this opening and the bulb is inflated with 2-3mls of normal saline. The nipple of the catheter is inserted into the vagina. There is essentially no discomfort to the patient and coitus may resume normally. After three to four weeks the catheter may be removed by deflating the bulbs. By this time epithelialisation of the orifice will have taken place such that reclosure of the duct is unlikely. This method has not been tried in our unit and marsupialisation is still the mainstay of treatment.

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GYNAECOLOGY CASE 3

PELVIC ABSCESS - LAPARATOMY AND DRAINAGE

NAME:	E.N.N	L.M.P	25/9/04
AGE:	29 Years	PARITY	2 + 1
I.P NO.	0918882		
D.O.A:	11/10/04		
D.O.D:	18/10/04		

PRESENTING COMPLAINT

ENN presented with a two-week history of lower abdominal pain, fever and vaginal discharge.

HISTORY OF PRESENTING ILLNESS

She had a two-week history of progressive lower abdominal pain that was accompanied by fever at times. She also gave history of having pus like and foul smelling vaginal discharge over the same period. She did not notice any abdominal swelling. She had no vomiting or diarrhoea. She had been seen in a private clinic but the treatment offered did not relieve her symptoms. When the pain worsened and became continuous she decided to seek treatment at Kenyatta national hospital (KNH).

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She attained menarche at the age of 13 years. She was a Para 2 + 1. Her first delivery was in 1994 and the second in 1998. Both were normal vertex deliveries in hospital and the children were alive and well. In 2003 she had an abortion at three months gestation and an evacuation was done at KNH. Her last menstrual period was on 25/09/04.Her periods were regular occurring every 28-30 days and flow of 3-4 days. She had no dysmenorrhoea or menorrhagia. She had never been treated for any sexually transmitted infection and had never used any form of contraception in her life.

PAST MEDICAL HISTORY

She had never been admitted to hospital before, had no known chronic illnesses or allergies.

FAMILY AND SOCIAL HISTORY

She was married in a monogamous relationship and lived with her family in Githurai. She was a bussinesslady, educated up to secondary school level. She had occasional use of alcohol but did not smoke cigarettes. She had no family history of any chronic illnesses.

PHYSICAL EXAMINATION

She was sick looking. She was not pale, had no jaundice or oedema. Her pulse was 84 beats/minute, blood pressure 110/70mmHg, respiratory rate 20/minute and her temperature was 37.5 ^{o}C. Her cardiovascular, central nervous and respiratory systems were normal.

ABDOMINAL EXAMINATION

Her abdomen was not distended and moved with respiration. She had marked tenderness in the suprapubic region and the iliac fossae. Her bowel sounds were normal.

PELVIC EXAMINATION

She had normal external genitalia. Her cervix was closed, firm, long, and posterior. The cervical motion test was positive bilaterally. She had fullness in both adnexae and in the pouch of Douglas, which felt tense and was tender. There was pus discharge on the examining finger.

DIAGNOSIS

A diagnosis of pelvic abscess was made.

INVESTIGATIONS

1. Haemogram Haemoglobin 12g/dl
   white cell count 15.7 x 109/l with neutrophilia of 84%

2. Urea and electrolytes
   Na + 134 mmol/l
   K+ 3.5 mmol/l
   Urea 2.5 mmol/l
   Creatinine 68 umol/l

3. Pelvic scan. Showed fluid collection in the pouch of Douglas, probable pelvic abscess. The ovaries and uterus were not well visualized due to fluid collection.

MANAGEMENT

She was started on intravenous metronidazole 500mg 8 hourly and amoxicillin/clavulanic acid 1.2g twelve hourly. She was also given diclofenac 75mg 8 hourly intramuscularly

for analgesia. She was counseled and consented to the need for surgical intervention. She was starved from midnight on the night prior to surgery. On the morning of her surgery she had one unit of cross-matched blood available
She was premedicated with atropine 0.6mg and pethidine 50mg given intramuscularly half hour before surgery.
In theatre she was given general anaesthesia. A vulvo vaginal toilet was done, her bladder was catheterised and clear urine obtained. Examination was done again and the earlier findings were confirmed. In the supine position her abdomen was cleaned with antiseptic solution and draped with sterile towels. Her abdomen was opened via a sub umbilical midline incision. Her peritoneum was found to be thickened and there were adhesions in the pelvis with all organs in the pelvis matted together. Her omentum was pulled down towards the pelvis, the intestines were normal. There was pus in the pouch of Douglas that was drained with an estimated volume of about 100mls. A specimen was taken for culture and sensitivity. All pockets of pus were released drained. The uterus and both ovaries were covered in the adhesions. Her fallopian tubes had no pyosalpinx. Peritoneal lavage was then done with warm normal saline and rifocin. A corrugated drain was left in the pouch of Douglas and the end brought out through the right iliac fossa. Haemostasis was achieved and the abdomen closed by mass closure technique using nylon # 1 suture after ascertaining a correct count of swabs and instruments. The skin was closed with nylon # 2/0 suture. She was then reversed from anaesthesia successfully and taken to the recovery room.

POSTOPERATIVE MANAGEMENT

She was observed until fully awake then transferred back to the ward. She was maintained on intravenous fluids for 24 hours until her bowel sounds were back to normal, after which she was started on oral sips and gradually on free diet as she could tolerate. She was continued on intravenous antibiotics as prescribed preoperatively, and was given pethidine 100mg 8 hourly for analgesia. By the third day her drain was no longer active and it was removed. The culture results were reported to have grown Escherichia coli that was sensitive to amoxicillin/clavulanic acid which she had already been started on. She was discharged on the seventh day after removal of stitches and the wound was healing well. She was to continue with the antibiotics to finish a course of fourteen days and she was adviced to be seen in the gynaecological out patient clinic in four weeks.

FOLLOW UP

She returned to the clinic after four weeks. She was well and her wound had healed well. She had no complaints.

DISCUSSION

A case is presented of ENN, a 29 year old para 2+1 who had a pelvic abscess managed by laparotomy and drainage with subsequent good recovery. Our patient may have had a pelvic infection before developing the abscess.

Pelvic abscess is a collection of pus in the pouch of Douglas [1,2]. It may occur as a sequel to acute pelvic infection, chronic or recurrent pelvic infection, abortion, puerperal sepsis, gynaecological surgery or gynaecological cancer. Abscess may also follow introduction of foreign material into the uterus such as with use of the intrauterine contraceptive device (IUCD), after hysterosalpingography or dilation and curettage. A perforated appendix or salpingitis may also lead to a pelvic infection and abscess. It appears the tail of the IUCD may enhance entrance of organisms into the upper genital tract [1,2]. This patient had not used any IUCD and had not undergone any pelvic procedures previously.

The causative organism in our patient was E. coli. The causative organisms include Neisseria gonorrhoea, chlamydia trachomatis, Mycobacteria tuberculosis and gram-negative bacilli, various anaerobic organisms including anaerobic positive cocci and bacteroides species. Most of these organisms are sexually transmitted. Carty found that in 75% of all cases of PID, gonococcus was isolated while Fomulu found anaerobes and aerobes with E. coli occurring in 50% of all cases at the KNH. [3,4]. Actinomyces have been found in tubo-ovarian abscesses associated with IUCD [4,6].

Our patient was para 2+1 and 29 years old. Patients who develop pelvic abscess are usually in their 20's to 30's (most commonly 15-25) and a large percentage (20-59) of these women are nulliparous while it is rare in those with a parity of four and above [2-4]. Our patient fitted into the above criteria. Pelvic abscees is also more likely in those with multiple sexual partners. Spermatozoa may carry organisms including gonococcus and other bacteria. In postmenopausal women a pelvic abscess is usually secondary to pathology in the intestinal tract.
Pelvic abscess is a major cause of morbidity and mortality among women in the reproductive age group. The incidence varies worldwide. In Kenya, pelvic abscess accounts for 40% of all acute gynaecological admissions to the KNH and Aga Khan hospitals[3].

Our patient presented with low abdominal pain with vaginal discharge and diagnosis was confirmed by ultrasound. The usual presentation may involve symptoms of acute or chronic pelvic inflammatory disease including abdominal pain, fever, rigors, nausea, vomiting, diarrhoea, dysuria and/or frequency of micturition. There may also be vaginal discharge that is foul smelling or blood stained. Examination usually reveals a patient who is sick looking and may have fever as in our case, the abdomen is tender, fluctuant mass filling the cul-de-sac and dissecting into the rectovaginal septum is felt. Pelvic examination elicits severe pain even on slight cervical excitation. Paracentesis may be positive of pus. Jaundice and anaemia may also be present. Anaemia is especially common in those patients who have had an abscess for several weeks [3,6,7,8]. Laboratory findings include a raised erythrocyte sedimentation rate, leucocytosis, raised C-reactive

protein and these add little to the information got from the history and physical examination. Urinalysis may demonstrate pyuria without bacteria. Ultrasound is an invaluable tool for confirming diagnosis. Laparoscopy is most accurate in diagnosis and can also be used for drainage. Our patient had a leucocytosis and ultrasound was suggestive of pelvic abscess.
The patient presented had drainage of the abscess through laparotomy. This was done after adequate hydration was ensured and antibiotics were started.
The initial management of a patient with pelvic abscess consists of supportive measures such as rehydration with intravenous fluids, nasogastric tube suction, blood transfusion and parenteral antibiotics. Those with IUCD should have the device removed after initiation of treatment [3,6]. Response is judged by absence of fever, decrease in white cell count by at least 3,000/mm3 and decrease in size of the mass as well as improvement in symptomatology. In case there is no adequate response or a ruptured abscess is suspected then surgical intervention is needed [3,6]. Where the diagnosis is certain, a posterior colpotomy and drainage can be done bur with extensive abscesses or uncertain diagnosis, drainage by laparotomy is preferred [6].At laparotomy, pelvic adhesions should be released by gentle blunt dissection and the pockets of pus drained. In case the abscess followed an induced abortion, the possibility of undiagnosed or improperly managed uterine perforation should be considered, looked for and repaired appropriately. Salpingo-oophorectomy may be necessary where a tubo-ovarian abscess is encountered with extensive damage to both the ovary and the tube [8]. Antibiotic regimes should cover for gram positive, gram negative and anaerobes. This was done in the patient presented. For women past reproductive age, the practice to perform total hysterectomy and bilateral salpingo-oophorectomy has been recommended [6]. Radical surgery may also be preferred in those patients who have had several exacerbations of tubo-ovarian abscess who remain more sick, yet the pelvic organ damage is already too extensive for future fertility considerations.

Other methods used for drainage include percutaneous drainage of intra-abdominal abscess guided by computed tomography or real time scanners, which may be successful in 75-85% of cases [9]. Transvaginal sonography guided drainage (culdotomy) of a tubo-ovarian abscess has also been shown to be successful[10]. These were not done for our patient due to limitation of availability of the radiological machines.

Our patient did not develop any complications after surgery.
Complications of untreated pelvic abscess include recurrence or persistence of abscess associated with significant morbidity, septicaemia, chronic ill-health, chronic pelvic pain, dyspareunia, dysmenorrhoea, bowel obstruction, infertility, ectopic pregnancy and septic shock. Acute renal failure and septic thromboembolism are early complications, which have a high incidence of morbidity and mortality [6,8].

All these complications may be prevented by adequate health education, adequate health programmes to control and prevent sexually transmitted infections and provision of comprehensive family planning services. Barrier contraceptives and avoiding IUCD use in those at high risk should be recommended.

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GYNAECOLOGY CASE 4

RUPTURED ECTOPIC – SALPINGECTOMY

NAME: J. A Parity: 2 + 0

AGE: 27 Years L.M.P: 25/11/2001.

I.P NO: 0789495

D.O.A. 6/02/2002

D.O.D. 11/02/2002

PRESENTING COMPLAINT

JA was admitted with a one-month history of vaginal bleeding and lower abdominal pain.

HISTORY OF PRESENTING COMPLAINT

She was well till about one month prior to admission when she started experiencing irregular vaginal bleeding which would at times be heavy and with clots. Soon after the bleeding had started, she started having low abdominal pain. Initially the pain was mild and tolerable but worsened about five days prior to admission. She complained of mild dizziness but no palpitations. She had no dysuria or frequency of micturition. She had no diarrhoea or vomiting. She had not sought any medical advice before admission because the pain was relatively bearable until this time.

OBSTETRIC AND GYNAECOLOGY HISTORY

She was Para 2+0. Her menarche was at 18 years of age. Her cycles occurred every 21 days with flow of 4 days. She gave no history of dysmenorrhoea. She had had two term hospital deliveries in 1994 and 1996. Both children were alive and well. She denied any previous history of treatment for sexually transmitted illness or any vaginal discharges. Her last normal menstrual period was on 25/11/01 giving a period of amenorrhoea of 10 weeks and three days. She was currently not on any contraceptive method though she had briefly used combined oral contraceptives after her last delivery for about 3 months.

PAST MEDICAL HISTORY

There was none of significance. She did not have any known allergies to drugs or food.

FAMILY AND SOCIAL HISTORY

She was a married housewife. The spouse was a casual labourer in Nairobi. She neither drank alcohol nor smoked cigarettes. She was educated up to secondary school. She gave no family history of any chronic illnesses.

PHYSICAL EXAMINATION

She was sick looking, pale and afebrile. She had no jaundice, oral thrush or pedal oedema. Her blood pressure was 90/50mmHg, pulse was 102/minute and respiratory rate 20/minute. Her respiratory, cardiac and nervous systems were normal.

ABDOMINAL EXAMINATION

The abdomen was slightly distended in the suprapubic region. There was marked tenderness with more on the left lower quadrant accompanied by guarding. There was no organomegaly. Paracentesis (in the area of maximal abdominal tenderness) done after pelvic examination revealed non-clotting haemoperitoneum.

PELVIC EXAMINATION

Her external genitalia was normal, speculum examination revealed closed cervix with dark blood oozing from the os and normal vaginal walls. On digital examination, the cervix was firm and closed. There was fullness and tenderness in the pouch of Douglas and left adnexa. The uterus was bulky.

DIAGNOSIS

A diagnosis of ruptured ectopic pregnancy was made.

MANAGEMENT

The diagnosis and management plan (laparotomy) was explained to the patient. An intravenous drip of 1 litre normal saline running fast was set up. At the same time blood for quick haemogram and grouping was taken. Two units of typed blood were immediately availed. Her haemoglobin was reported as 8.2g/dl.
An informed consent was obtained from her. The pubic region was shaven and she was premedicated with intramuscular atropine 0.6mg. She was then immediately wheeled to theatre that had been availed.

In theatre, general anaethesia was induced and maintained. The patient was placed in the semi-lithotomy position, vulval vaginal toilet was done and aseptic catheterization of the bladder done. Repeat pelvic examination revealed a full pouch of Douglas and a bulky uterus at about ten weeks. The patient was repositioned to supine position; the abdomen was cleaned, draped and opened in layers via a sub-umbilical midline incision. Blood poured out upon opening the peritoneum. The right hand was immediately passed down into the pelvis and the uterus identified. Using the uterus as a guide, the adnexa was felt and a mass felt on the left side was grasped and lifted through the abdominal incision. The mass was examined and found to be the site of the ectopic gestation on the left ampullary part of the tube. Left partial salpingectomy and evacuation of the haemoperitoneum were done. The left tube and its mesosalpinx was held up and two curved artery forceps placed parallel to the length of the tube. The dilated tube was

excised by cutting between the two forceps. The mesosalpinx was then ligated by passing a ligature round the forceps that was holding it. Haemostasis was ensured and the abdomen was cleaned with warm normal saline. Inspection of the abdomen and pelvis revealed normal ovaries but the right tube had terminal blockage. The uterus was bulky. She had flimsy adhesions in the pelvis which were released. The abdomen was then closed in layers and the wound dressed. The total blood loss was 800mls. Anaesthesia was reversed, the patient extubated and taken to the recovery room and later to the ward.

Post operatively her vital signs were monitored half hourly till she was fully awake and four hourly thereafter. She was started on intravenous antibiotics crystalline penicillin 2MU six hourly and gentamicin 80mg eight hourly for five days. Analgesia was given as pethidine 100mg 8 hourly for 48 hours then mefenamic acid 500mg three times daily for one week. Oral feeding was started 24 hours after surgery. Haematinics (ranferon 10mls twice daily) were started in addition to oral antibiotics. Her postoperative period was uneventful and she was discharged on the fifth day post operation. She was given an appointment to be seen in the gynaecology out patient clinic after one month.

FOLLOW UP

When she came after one month, she was noted to be well, was not pale and the wound had healed well. Histology of the mass taken at laparotomy confirmed the diagnosis of tubal pregnancy. The tubal mucosa was identified and the wall of the tube was thickened. Degenerating chorionic villi were seen in the mucosa of the tube.
She was adviced that she still stood a small chance of another ectopic pregnancy thus need for evaluation at the earliest during her next pregnancy. She was referred to the family planning clinic, as she wanted a break before conceiving again.

DISCUSSION

The patient presented had a ruptured ectopic pregnancy for which left partial salpingectomy was done. She was well at the follow up visit.

She had tubal ectopic pregnancy. Ectopic pregnancy refers to the implantation of a fertilized ovum in any site other than the endometrial lining of the uterine cavity. Implantation thus occurs in the absence of decidualised endometrium and endometrial glands. 95% of ectopic pregnancies involve the fallopian tubes. Other sites include ovary, broad ligament, and rudimentary horn of the uterus, the cervix or in the abdominal cavity.. Multiple ectopic pregnancies may also occur including both tubes and intrauterine gestation [1,2,3]. It is a condition of immense gynaecological significance in Africa and the whole world. The risk of death following an ectopic gestation is 10 times that of a vaginal delivery and 50 times that of induced abortion[1,2].The presented patient was discharged home in fair condition.

Ectopic pregnancy remains the second leading cause of maternal mortality in the USA. More than 1 in every 100 pregnancies in the USA is ectopic[1, 4,5]. At KNH Mwathe[6] (1984) reported the incidence of ectopic pregnancies as 4-5 per week or 1:15 deliveries. The age incidence was found to be 20-29 years (worldwide is 25-34). Our patient was 27 years old. The incidence is increasing due to increased pelvic infections, improved treatment of pelvic inflammatory disease, use of intrauterine devices especially those containing progesterone.

Our patient was found to have pelvic adhesions (at laparotomy) which could have contributed to the ectopic gestation. The aetiology of ectopic pregnancy is unknown but several factors have been implicated. These include the following[1-5]:

- Mechanical factors that prevent or slow the passage of fertilized ovum into the uterine cavity. This can be caused by many factors including
    - Endosalpingitis from pelvic infections
    - Peritubal adhesions
    - previous ectopic pregnancy. The risk is increased in women who have had ectopic and further in proportion to the number of ectopics. Risk of recurrence decreases with subsequent intrauterine pregnancies after the initial ectopic pregnancy.
    - Developmental abnormalities such as diverticula, accessory ostia, lengthy tube and hypoplasia or atresia
    - Previous operations on the tube-tuboplasty, previous surgical sterilization
    - Multiple previous induced abortions
    - Tumours that distort the tube such as myomas or adnexal masses
    - Previous caesarean section

- Functional factors (delay the passage of the fertilized ovum into the uterine cavity). These include:
    External migration of the ovum

Altered tubal motility following luteal phase defects
Oral contraceptive pills especially progestin only pills
Failed sterilization
Intrauterine contraceptive devices
In vitro fertilization, ovum transfer and ovulation induction
Gamete intrafallopian transfer (GIFT)
Menstrual reflux with endometriosis

- Other factors implicated include:
  Ovarian factors such as fertilization of unextruded ovum, transmigration of the ovum, post mid-cycle ovulation and fertilization and ovarian enlargement due to use of clomiphene (clomid) or menotropins (pergonal)[4]
  Zygote abnormalities- a number of chromosomal abnormalities, gross malformations and neural tube defects end in ectopic pregnancy
  Previous genital infections, cigarette smoking, multiple sexual partners, vaginal douching, early age at first intercourse (<18 years), in utero exposure to diethylstilbestrol, increasing age, chlamydia infection and tuberculous endosalpingitis.

In our patient, suggestive history and a positive paracentesis were used to make the diagnosis. Abdominal pain with amenorrhoea and a tender adnexal mass is the most common presentation. Diagnosis is made from history, physical examination and investigation. Women in reproductive age presenting with menstrual irregularity, pelvic pain and dizzy spells should be investigated for possibility of an ectopic pregnancy. Other presentations include vaginal bleeding (of uterine origin due to endometrial involution and sloughing. The Arias-Stella reaction consists of hyperchromatic, hypertrophic irregularly shaped nuclei and foamy, vacuolated cytoplasm), hypovolaemic shock with pallor, tachycardia, hypotension and gastrointestinal symptoms with signs of free peritoneal fluid or peritoneal irritation. Paracentesis yielding non-clotting blood is usually confirmatory. A negative paracentesis does not rule out ectopic pregnancy. Sinei and Okumu[7] (1987) reported negative paracentesis in 30% of cases and only 21% of their cases reported with shock and 57.4% had anaemia. Paracentesis was positive in the presented case.A mass may be palpable on either side of the cervix and there may be fullness of the cul-de-sac. There may be some vaginal bleeding. Culdocentesis may also yield non-clotting blood. Pelvic scan or transvaginal ultrasound, β-HCG levels> 1000miu/l and laparoscopy are other diagnostic aids that are used to confirm suspected ectopic pregnancy. However clinical diagnosis for ruptured ectopic is usually straightforward and unnecessary delays should not occur while awaiting investigations.

Our patient had ampullary ectopic pregnancy. Majority of ectopic pregnancies are tubal accounting for as high as 99% of cases. The ampulla is the common site of implantation accounting for 78%, 12% are in the isthmus, 5% fimbrial and 2% are cornual.
The clinical course of ectopic pregnancy will depend on the anatomical site and the stage of the pregnancy. Ectopic pregnancy may thus mimic many gynaecological and surgical conditions and the diagnosis delayed especially if unruptured ectopic is present.

A tubal pregnancy may terminate in spontaneous regression, abortion or rupture of the tube. Isthmic rupture occurs early at 6-8 weeks, ampullary rupture at 8-12 weeks. Spontaneous resorption of tubal pregnancy may occur especially when the embryo dies very early. It often goes undiagnosed and does not require surgery.

Our patient was treated by partial salpingectomy at laparotomy. Treatment of ectopic pregnancy depends on the site and whether it is ruptured or not. Surgery is the preferred treatment of ectopic pregnancy when there is rupture, hypotension or pain persisting beyond 24 hours. Blood products should be available as transfusion is often necessary. Blood was availed for our patient. Partial or total salpingectomy is done once the source of bleeding is identified. Partial salpingectomy is done when only a short section of the tube is involved and allows the chance of later tubal surgery to achieve function in the future. Recently more conservative approaches have been used to treat tubal pregnancies. Most try to conserve the tube. They include salpingotomy-incision and closure of the tube after evacuation, milking or suction of the ectopic followed by fimbrioplasty and salpingostomy-incision without closure of the tube, which heals by secondary intention. These procedures are not recommended as recurrence of ectopic is doubled as well as the rate of persistent haemorrhage. Our patient was treated with partial salpingectomy. Conservative surgery should be followed with weekly βHCG levels till they reach non-pregnant levels.
After the tubal operation, it is important to remove all blood from the peritoneal cavity, especially that which has tracked up into the paracolic gutters. This was done in the case presented. Interstitial pregnancies may require wedge resection with uterine reconstruction. Ovarian pregnancy requires oophorectomy and sometimes salpingectomy on the affected side. Abdominal pregnancy involves delivery of the foetus with ligation of the umbilical cord close to the placenta and the placenta usually left in place to avoid haemorrhage. Chronic ectopic pregnancies are difficult to diagnose and are often found incidentally during exploratory laparotomy. Approximately 3-20% of ectopic pregnancies are chronic. Dense adhesions and luminal abscess formation are surgical features characterizing chronic ectopic pregnancies[1, 4,5,8].

Laparoscopy is now used in many centers both for diagnosis and management[9]. The procedure has the distinct advantages of lesser morbidity, decreased blood loss, shorter hospital stay, lowered cost and early return to full activity for the patients. This was not done for our patient as laparoscopy is currently not available for emergency situations within the unit.

Medical management of ectopic pregnancy is by use of methotrexate either locally or intravenously with or without folinic rescue and is used for unruptured ectopic pregnancy. The success rate is higher if the gestation is below 6 weeks or the tubal mass is 3.5cm or less and the foetus is not alive. Methotrexate inhibits dihydrofolate reductase, the enzyme responsible for converting folic acid to reduced folate co-factors. It thus blocks DNA and partly RNA synthesis. Consequently tissue undergoing rapid cellular turnover such as trophoblast are most susceptible to its action. Intramuscular methotrexate for ectopic pregnancy is associated with few side effects and also preserves the potential of reproductive function. The patient requires monitoring of their β-HCG

titres. A fall by at least 15% of baseline value within 4-7 days after administration. Failure of methotrexate is suggested by persistent rising or plateau in β-HCG. This demands another dose of the drug or surgery. Other drugs used in management of ectopic pregnancy include the use of techniques like salpingocentesis where agents such as potassium chloride, prostaglandin E_2, hyper osmolar glucose and even methotrexate are injected into the ectopic pregnancy transvaginally under ultrasound guidance. Other access routes for delivering these agents are transcervical tubal canalization and laparoscopy. Advantages of salpingocentesis include a one-time injection with the potential avoidance of systemic side effects. RU-486 and anti-HCG are also under research as agents that can be used for salpingocentesis. Contraindications to medical therapy include breastfeeding, overt or laboratory evidence of immunosuppression, pre-existing blood dyscrasias, peptic ulcer disease and hepatic, renal or haematologic dysfunction and known sensitivity to methotrexate[4, 10].

Expectant management has sometimes been tried in some centers. However, distinguishing patients who are experiencing spontaneous resolution of their ectopic pregnancies from patients who have proliferating ectopic pregnancies and require intervention is a clinical dilemma. Expectant management has been used in the hope of avoiding therapy that may be otherwise unnecessary as many ectopic pregnancies resolve spontaneously. Candidates for expectant management must be willing to accept the potential risks of tubal rupture and haemorrhage. If the initial βHCG is less than 200miu/mlor decreasing and the risk of rupture is low, then expectant management can be used[4, 5, 10]. This method is not in use in our set-up.

The greatest complication of ectopic pregnancy is death accounting for 10% of all maternal mortality worldwide. Rakwach[11] reported that ectopic pregnancy accounted for 4.7% of all maternal deaths in the acute gynaecology ward in Kenyatta hospital. Therefore to avoid this complication and reduce maternal mortality arising as a result of ectopic pregnancy, a high index of suspicion, early diagnosis and appropriate management of ectopic pregnancies is extremely important. Our patient was treated, recovered and was discharged home in fairly good condition.

After treatment, contraception should be adviced as return to fertility is within 2-3 weeks. Our patient was counseled and also adviced to attend a tertiary antenatal care center during her next pregnancy to allow diagnosis and follow up in view of the increased risk of recurrent ectopic pregnancy.

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GYNAECOLOGY CASE 5

BURST ABDOMEN: SUCCESSFUL REPAIR

NAME: S.N. L.M.P. 28/06/02

AGE: 22 Years PARITY 0 +1

I.P NO: 0830973

D.O.A: 10/09/02

D.O.D: 26/09/02

PRESENTING COMPLAINT

She complained of feeling of something giving way at the site of the abdominal wound eight days after surgery. This was accompanied by oozing of fluid that soaked her dress.

HISTORY OF PRESENTING COMPLAINT

SN was well until nine days prior to admission when she developed per vaginal bleeding, severe low abdominal pain, headaches, and dizziness. These started a day after she procured an abortion at a private clinic in Buruburu. She had missed her periods for about two and a half months. Vaginal bleeding persisted after the abortion and was accompanied by increasing low abdominal pain which became persistent. She had associated dizziness but did not report having fainted. It was then that she decided to seek treatment at KNH. On arrival, she was found to be mildly pale and afebrile ($36^{o}C$). Her abdomen was very tender in the suprapubic region, had rebound tenderness with guarding and it was difficult to delineate any masses due to the tenderness. Speculum examination revealed an open cervix with inflamed edges. she had no active vaginal bleeding. Bimanual examination was difficult due to tenderness. There was no foul smelling discharge from the vagina. A diagnosis of acute abdomen with likely uterine perforation was made and she was then planned for emergency laparotomy. Cross matched blood was asked for and two units availed. At laparotomy uterine wall perforation was found on the anterior uterine wall. The uterine cavity was empty. The walls were freshened and the perforation repaired in two layers. Postoperatively, she was started on amoxicillin/ clavulanate 1.2g 12 hourly, metronidazole 500mg 8 hourly and gentamicin 80mg 8 hourly and this was given for seven days.

Seven days after the laparotomy, her wound was examined and found to be healing well. However it had an area of induration but was not oozing any fluid and there was no fluctuant mass along the incision. The stitches were removed and she was to be observed for a day before discharge. She did not have any cough or constipation. On the following morning, she complained that as she was getting out of bed, she felt like her lower abdomen had given way and that she felt her dress get wet. She did not notice any bleeding.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

SN attained her menarche at 14 years, her menstrual pattern before conception was regular with flow of 3-4 days occurring every 28-30 days. She had no dysmenorrhoea and had never used any form of contraception.
She was now a Para 0 + 1, having procured an abortion nine days prior to admission (She voluntarily gave this information). Her last menstrual period was on 28/6/02 giving a gestation period of ten weeks at the time of termination.
She had never used any form of contraception previously. She had never been treated for sexually transmitted infection

PAST MEDICAL HISTORY

She had no other admitted previously in 1989 after having suffered burns. She had not been admitted with any other illnesses and did not have any known drug or food allergies.

FAMILY AND SOCIAL HISTORY

She was single and had no family history of chronic illnesses. She was a university student and gave this as the reason for procuring termination as she felt pregnancy would interfere with her studies. She did not smoke or use alcohol.

PHYSICAL EXAMINATION

She was in fair general condition, not pale, had no jaundice or oedema. Her vital signs were; blood pressure of 110/60mmHg, pulse 80/minute, respiratory rate 18/minute and a temperature of 37°C
Her respiratory, cardiovascular and nervous systems were found to be normal.

ABDOMINAL EXAMINATION

The abdomen was not distended. The wound was found gaping and was oozing serous fluid. The subcutaneous tissue was infected and the rectus sheath had separated. The parietal peritoneum was partially open. Loops of gut were seen through the wound

DIAGNOSIS

Burst abdomen after laparotomy for perforated uterus

MANAGEMENT

She was informed of the diagnosis and consented for repair under general anaesthesia. A sterile gauze pack was placed on the wound and bandage applied. Investigations done showed a PCV (packed cell volume) of 31%, urea, creatinine and electrolytes were

normal. A unit of cross-matched blood was immediately made available for her. She was then premedicated with atropine 0.6mg and pethidine 50mg and taken to the operating theatre.
In theatre and under anaesthesia the incision was cleaned, draped and inspected. It was found to be open through all layers and the viscera was visible. The edges were friable, there was pus on the abdominal subcutaneous layer. The uterine repair incision was healing well and was not septic. There was no pus within the abdominal cavity. The abdomen was cleaned with rifocin, and freshening of the incision edges done. Three tension sutures were placed at both ends and the middle of the incision. This was done using silk suture No 2 passed through all layers of the abdominal wall then passed through a rubber tubing to prevent cutting through the skin upon tying a knot. Mass closure of the abdominal wall was then done using Nylon 2. The skin was closed using interrupted nylon 2/0 suture. The wound was then cleaned and dressed.

POST-OPERATIVE MANAGEMENT

She was put on intravenous cefuroxime 750mg 8 hourly, metronidazole 500mg 8 hourly. She was also given diclofenac 75mg 8 hourly for analgesia. She did well post-operatively and the wound was well apposed when it was opened on the fourth day after repair. Culture of the pus taken from the wound grew staphylococcus aureus that was sensitive to the cefuroxime that she was already on. She was started on oral antibiotics on the fifth postoperative day. Her vital signs including temperature remained normal throughout the postoperative period. The tension sutures were removed on the seventh day after operation. By the tenth day the wound was well healed and the stitches were removed. She was discharged home in good condition.

FOLLOW-UP

She was seen in four weeks and she was well. She had no complaints, the wound had healed well and she was in a good general condition.

DISCUSSION

The patient presented was admitted with a perforated uterus after induced abortion, emergency laparotomy was done and was healing well but on the eighth day developed a burst abdomen after removal of stitches. Secondary repair was done and she healed well and was allowed home in good condition.

Our patient developed burst abdomen 8 days after surgery. Burst abdomen is the separation of all the layers of the abdominal wall including the rectus sheath and the peritoneum with evisceration of intra-abdominal contents including protrusion of the intestines whereas wound dehiscence is defined as disruption or separation of the layers of the abdominal wall but the rectus sheath remains intact. It is a serious complication that carries a mortality of 15-30%. It usually occurs 6-10 days after operation and is often preceded by the "pink sign" [1]- a serosanguinous discharge appearing on the dressing as happened to the patient presented. The incidence of wound disruption after caesarean section is reported to be between 0.14% and 4.37 %[2]. This patient had burst abdomen after her wound became infected post surgery.. No single aetiological factor can account for all wound disruption. In most cases it is a combination of various factors that ultimately lead to this complication. There are four categories under which aetiological factors for wound dehiscence can be placed. These include; [3,4,5]

i) Specific type of suture used
ii) Type and location of incision
iii) Inherent strength of the tissues
iv) Mechanical factors.

Our patient had a midline incision.Midline incisions are more prone to disruption than transverse ones. Factors that increase intra abdominal pressure such as cough, cardiopulmonary disease, ascites and ileus are associated with higher rates of wound disruption. Inadequate haemostasis at the operation site results in formation of haematoma usually under the rectus sheath. Due to the haematoma formation the tissue edges are kept apart and healing is delayed. Our patient had no other factors that could have led to wound disruption.

The patient presented was found to have infection that could have led to the disruption of the wound. Infection causes accelerated dissolution of catgut suture material, granulation tissue will form and healing will not occur. Tight sutures cause areas of ischaemia with necrosis leading to wound disruption, as would inadequate haemostasis, excessive use of diathermy and dead space left in the abdominal wall.

Anaemia secondary to haemorrhage in our patient may have contributed to the poor healing and subsequent burst abdomen that occurred The inherent strength of the tissue has been related to various factors, including vitamin C deficiency, anaemia, protein deficiency and the age of the patient..

There was pus, a sign of infection in our patient though her temperature was within upper normal limit. Bacterial sepsis after caesarean section is still a major cause of maternal

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GYNAECOLOGY SHORT CASE 6

THIRD DEGREE GENITAL PROLAPSE- TOTAL VAGINAL HYSTERECTOMY

NAME: M .M. PARITY 5 + 0

AGE : 64 Years L.M.P Post menopause for 10 years

I.P NO: 0808671

D.O.A: 9/8/02
D.O.D: 17/9/02

PRESENTING COMPLAINT

MM was first seen at the gynaecology out patient clinic with a 2 year history of lower abdominal pain, back pain and a sensation of heaviness in the vagina .

HISTORY OF PRESENTING ILLNESS

She developed the above symptoms about two years before admission. Initially it was just the pain alone. She later noted a swelling protruding from her vulva, which would spontaneously retract. This was associated with occasional bleeding and a foul smelling discharge. She also complained of urinary urgency, a feeling of incomplete bladder emptying, constipation and incomplete stool emptying.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She could not recall her age at menarche. She was a Para 5 + 0 and was postmenopausal for ten years. Her last delivery was 20 years back, all deliveries being vaginal with no history of trauma at delivery. She had never used any modern contraceptive methods.

PAST MEDICAL HISTORY

She had never been hospitalized before, had no chronic illnesses, or any previous surgery, or genital tract repair.

FAMILY AND SOCIAL HISTORY

She was married and lived with her spouse in Kitui.They were both peasant farmers. She had two sisters and one brother. There was no family history of a similar or chronic illness.

PHYSICAL EXAMINATION

She was an elderly lady in fair general condition and good nutritional status. She was not pale or jaundiced. She had no lymphadenopathy. Her vital signs were normal. Her blood pressure was 110/60mmHg, respiratory rate 18 breaths/minute, temperature 36o C and her pulse was 70 beats/minute and regular.
She had normal findings in the cardiovascular central nervous and respiratory systems.

ABDOMINAL EXAMINATION

The abdomen was scaphoid, had no surgical scars and moving well with respiration. There were no palpable masses and the bowel sounds were normal.

PELVIC EXAMINATION

MM had been done clitoridectomy. Her cervix was visible outside the vaginal introitus. The cervix would move further out on valsalva manouvre and was inflamed. The fundus of the atrophic uterus was easily palpable within the lower vaginal canal. The cervix protruded further when she was examined in the standing position. There was anterior vaginal wall prolapse but had no rectocoele when examined with a Sims speculum. There was a bulge on the anterior vaginal wall that increased with asking her to cough. She had no bleeding or discharge.

INVESTIGATIONS

1 Haemogram Haemoglobin 12.5g/dl
white cell count 3.0 x 109/l
platelets 340 x 109/l

2. Urea and electrolytes
Na+ 144mmol/l
K+ 4.3 mmol/l
Urea 5.7 mmol/l
Creatinine 72umol/l

3. Urinalysis was normal and there was no growth of any organisms on culture.

4. PAP Smear. Was satisfactory with no abnormal cells seen

DIAGNOSIS

Third degree genital prolapse.

MANAGEMENT

She was admitted for vaginal hysterectomy, for which she was counseled and gave informed consent. On the night prior to theatre she was fasted from midnight and given

metronidazole 1gm and an enema for bowel sterility. On the day of surgery she was premedicated with atropine 0.6mg and pethidine 50mg half hour before surgery. Two units of cross-matched blood were availed in theatre. In theatre general anaesthesia was induced, vulvovaginal toilet done and a bimanual pelvic examination to confirm the earlier findings, assess uterine mobility and descent, to rule out other adnexal pathology was done. A bladder foley catheter gauge 16 was then inserted to drain the urine. A total vaginal hysterectomy was then performed using the following steps;
A weighted speculum was placed into the posterior vagina, a right angle retractor was positioned anterior to the cervix and then both the anterior and posterior lips of the cervix were grasped with a toothed tenaculum. Adrenaline (10-20mls diluted in 50mls normal saline) was injected into the cervical, paracervical and submucosal tissues to help in identifying tissue planes and reduce blood loss. Traction was placed on the cervix to expose the posterior vaginal mucosa. Using the Mayo scissors, the cul- de-sac was entered and the peritoneum identified. A figure of eight suture was then placed to attach the peritoneum to the posterior vaginal mucosa. An Auvard weighted speculum was then inserted into the cul-de-sac. The uterosacral ligaments were clamped, cut and ligated. The ligated stump was transfixed to the posterior lateral vaginal wall to provide support to the vagina. Downward traction was applied on the cervix to expose the anterior vaginal wall, which was incised at the level of the cervicovaginal junction. The peritoneum was exposed after careful dissection using an open moistened gauze swab all around the cervix anteriorly and posteriorly. A right-angled retractor was used to protect the bladder. The cardinal ligaments were identified, double clamped, cut and ligated. The uterine vessels were clamped, cut and ligated. The ovarian ligaments were also double clamped, cut and ligated. The now free uterus was removed through the vaginal cavity and a purse string suture placed on the vaginal vault after haemostasis was ascertained.
An anterior colporrhaphy was then done to correct the cystocoele. A midline incision was made in the vaginal epithelium from 1cm below the urethral meatus to the area of the cervico-vaginal junction. The cyctocoele was dissected off the overlying epithelium using sharp and blunt dissection. Interrupted sutures using vicryl 3/0 were placed to strengthen the base of the bladder. Three mattress sutures were placed at the urethrovesical junction to prevent stress incontinence. Redundant tissue was trimmed and the vaginal epithelium closed using interrupted 2/0 vicryl suture. A vaginal pack was left in situ and the bladder catheter also left in. MM was then reversed from anaesthesia successfully and taken to the recovery room.

POSTOPERATIVE MANAGEMENT

She was observed quarterly till fully awake then four hourly. She was put on crystalline penicillin 2MU 6 hourly, gentamicin 80mg 8 hourly and pethidine 100mg 8 hourly. Oral fluids and later full diet were started from the second postoperative day. She developed acute urine retention on removal of the catheter and was returned to theatre to release the urethrovesical junction. Under general anaesthesia, the vaginal epithelium was opened and two of the mattress sutures that had been placed at the urethrovesical junction were removed. The vaginal epithelium was then repaired with interrupted sutures. A Foley catheter was retained for a further three days before removal. Thereafter she was able to pass urine well and she was allowed home on oral broad-spectrum antibiotics (ampicillin

500mg 6hourly and metronidazole 400mg 8 hourly for one week) and analgesics (mefenamic acid 500mg three times daily for five days).
Histology of the specimen removed showed: Gross- atrophic uterus and fallopian tubes. On microscopy- a normal uterine endometrium and myometrium, the cervix shows hyperkeratosis and parakeratosis which was consistent with the clinical diagnosis.

FOLLOW UP

She returned to the gynaecological out patient clinic two weeks after discharge. She was well without any complaints. She was free of any discomfort and could void urine well.

DISCUSSION

A case is presented of a 68 year old lady admitted with third degree genital prolapse and cystocoele who underwent vaginal hysterectomy and anterior colporrhaphy. She recovered well after the surgery and had no symptoms at the follow up visit.

Pelvic organ prolapse refers to protrusions of the pelvic organs into or out of the vaginal canal. The word 'prolapse' is derived from the Latin word prolapsus, a slipping forth and refers to the falling out of place of a part of viscus. Prolapse is descent of the pelvic organs into the vagina, often accompanied by urinary, bowel, and sexual or local pelvic symptoms[1]. Prolapse is a common and disabling condition. The organs of the urinary, genital and intestinal systems share common anatomic supports which are[2];

- The bony pelvis-this provides little support by surrounding and protecting its contents
- Muscles of the pelvic floor-the levator ani muscles have the ability to contract and offset increases in intra-abdominal pressure that would otherwise force the pelvic contents downwards.
- Pelvic ligaments-these serve to keep structures in positions where they can be supported by muscular activity rather than weight bearing structures themselves.
- Connective tissue-this is binding substance in the body and is affected by factors such as stress, hormonal changes, exercise and nutrition. Connective tissue abnormalities have been shown to be a significant factor contributing to prolapse and related conditions.

The patient presented was 64 years old and multiparous having delivered five times. Genital prolapse is thought to be rare in the African population compared to the Asian and western population. The incidence of genital prolapse is difficult to determine, as many women do not seek medical help. It has been estimated that half of parous women

lose pelvic floor support, resulting in some degree of prolapse, and that of these women only 10-20% seek medical care[3]. In the United Kingdom genital prolapse accounts for 20% of women on the waiting list for major gynaecological surgery[4]. The chance of a woman having a prolapse increases with age. Thus the incidence of prolapse will rise as life expectancy increases. Local incidence is not known but admission rates to KNH show a probable rate of about 1 in 100 or more. Mwalali[5] reported an incidence of 0.1% at KNH with most patients being above 40 years.

Our patient had anterior compartment prolapse and presented with lower abdominal pain, with associated feeling of heaviness in the vagina. Prolapse can occur in the anterior, middle or posterior compartments and depending on the location, the presenting symptoms will vary. It may also be asymptomatic and be an incidental finding and clinical examination may not necessarily correlate with symptoms. The causes of genital prolapse are thought to be multiple but certain conditions predispose to prolapse. These may be divided into:

- Congenital-such as bladder extrophy and collagen defects.
- Race-the whites have a higher risk compared to other races.
- Anatomical defects such as congenitally short vagina may play a role
- Childbirth trauma and denervation may predispose to prolapse. Injury sustained during delivery inevitably stretches and sometimes tears the supports of the pelvic viscera.
- Raised intra-abdominal pressure as in patients with chronic obstructive airway disease, ascites, tumour such as large cervical polyp causing traction on the uterus and those who strain as in constipation and weight lifting
- Menopause may predispose to prolapse due to oestrogen deficiency
- Pelvic surgery such as hysterectomy, colposuspension may predispose to prolapse.
- Sacral neural disorders especially injury to S_{1-4} as in spina bifida, diabetic neuropathy, caudal anaesthesia accidents, and presacral tumour.

Our patient probably had inherent weakness that was worsened by the many deliveries she had and worsened after menopause.

Anatomically, prolapse can be divided into the following;

- Anterior compartment prolapse-prolapse into the vagina or the urethra (urethrocoele) or bladder (cystocoele) or both (cystourethrocoele)
- Middle compartment prolapse- uterine or vault descent and enterocoele (herniation of the pouch of Douglas).
- Posterior compartment prolapse-prolapse of the rectum into the vagina (rectocoele)

Our patient had anterior and middle compartment prolapse. Enterocoeles may contain small bowel and omentum. Cystourethrocoele is the commonest type of prolapse, followed by uterine descent and then rectocoele. Urethrocoeles are rare [1]. Traditionally uterine descent is graded as:

- 1st degree- descent is within the vagina and does not reach the level of the introitus.
- 2nd degree- descent to the level of the introitus. The cervix protrudes through the vulva when the woman strains.
- 3rd degree- descent outside the introitus. Also known as procidentia. The entire uterus prolapses outside the vulva. The whole vagina or at least the whole of its anterior wall is inverted. Our patient had 3rd degree prolapse of the uterus.

Currently the pelvic organ prolapse quantitative exam (POP-Q) is being used to provide clinical grading of prolapse. It uses the hymen as the fixed point of reference. It includes the following stages;

Stage 0 No prolapse

Stage 1 Most distal portion of prolapse is more than 1cm above the level of the Hymen.

Stage 2 The most distal portion of prolapse is less than 1cm proximal to or distal to the plane of the hymen.

Stage 3 Most distal portion of prolapse is more than 1cm below the plane of the hymen but protrudes no further than 2cm less than the total vaginal length in centimeters.

Stage 4 Complete eversion.

Our patient had stage 3 prolapse as the cervix was more than 1cm below the plane of the hymen.

She presented with a feeling of mass protruding and chronic backache, urinary urgency and incomplete bladder emptying.The presenting symptoms will depend on the extent and site of the prolapse. Symptoms common to all types are a feeling of dragging, or a lump in the vagina, or something coming down, heaviness in the pelvis, low backache and lower abdominal and inguinal pulling discomfort. Some of the specific symptoms are;

- Urinary- stress incontinence, frequency, urgency and urge incontinence. Hesitancy, poor or prolonged urinary stream and a feeling of incomplete emptying. Manual reduction to start or complete emptying or positional changes may be required in patients with cystourethrocoele. Accumulation of residual urine can lead to urinary tract infection. Obstructive uropathy can result in severe cases of prolapse.
- Bowel symptoms- may be difficulty in defecation, incontinence of flatus, liquid stool. There may also be urgency of defecation or digitations or splinting of vagina,

perineum, or anus to complete defecation. A feeling of incomplete evacuation or rectal protrusion during or after defecation due to rectal prolapse may be noted.

- Sexual symptoms- inability to have or infrequent coitus and dyspareunia or lack of satisfaction or orgasm may be experienced. There may also be incontinence during sexual activity.
- Other local symptoms- feeling of pressure in the vagina, low back pain relieved by lying down is due to traction on the uterosacral and cardinal ligaments. In complete procidentia bleeding from ulceration of the exposed cervix may occur.

The patient presented was examined with a Sim's speculum and found to have the anterior vaginal wall prolapsing. The patient with genital prolapse should be examined in the left lateral or standing position with a Sim's speculum, inserting it along the posterior vaginal wall to assess the anterior wall and vault and vice versa.
Investigations done in patients with genital prolapse include;

- Urinalysis to exclude urinary tract infections
- Urodynamic studies such as cystometry and uroflowmetry are also recommended.
- Imaging studies such as pelvic fluoroscopy with barium contrast in the vagina, small bowel and rectum may help. Intravenous urography may help to exclude obstructive uropathy[8]. This test was not done in our patient.

Since our patient was symptomatic and was menopausal, she was treated by vaginal hysterectomy and anterior colporrhaphy. The treatment of patients with genital prolapse can be either conservative or surgical. Prophylactic measures for preventing prolapse include diagnosis and treatment of chronic respiratory disease and metabolic disorders, correction of constipation and intraabdominal disorders that may cause chronic increases in intra abdominal pressure. Administration of oestrogen to postmenopausal women who have no contraindication to its use may also help. Weight control, proper nutrition, smoking cessation and avoidance of strenuous occupational and recreational stresses that could damage the pelvic support system should be encouraged. Pelvic muscle exercises as a method of strengthening the pelvic diaphragm and as prophylaxis against development of pelvic organ prolapse should be encouraged in all women. Exercise may limit progression of mild prolapse and alleviate symptoms such as low back pain and pelvic pressure[9].
Symptomatic pelvic organ prolapse can be managed by nonsurgical and surgical methods. Treatment is determined by;

- Age of the patient
- The desire for future fertility
- The desire for coital function
- The severity of symptoms and degree of disability
- The presence of medical complications.

Nonsurgical treatment involves the use of pessaries. Pessaries are used in

- Patients unfit, waiting for, or who have declined surgery.
- Women who may want to bear children
- In the management of prolapse in neonates, which can occur together with neural defects such as spina bifida.

Pessaries are available in a variety of sizes and shapes to suit different patients and are of two main types

- Support pessaries- these rest under the symphysis and sacrum and elevate the vagina. They include the Ring pessary, Gehrung pessary and the Hodge pessary.
- Space occupying pessaries including the Cube pessary, Doughnut pessary and the Gellhorn pessary

Pessaries press against the walls of the vagina and are retained within the vagina by tissues of the vaginal outlet. They may cause vaginal irritation and ulceration. They are well tolerated when the vaginal epithelium is well oestrogenised. When adequate follow up is not assured they should not be used as they can become impacted within the vagina and, rarely ulcerate into the bladder or bowel[1]. Periodically, vaginal pessaries should be removed, cleaned and reinserted failure of which can result in fistula formation.

Indications for surgery include;

- Desire for definitive surgical correction
- Failure of pessary or recurrent vaginal ulcerations with a pessary
- Genuine stress incontinence that the patient finds unacceptable

The goals of surgical correction for prolapse are to relieve symptoms, restore normal anatomic relationships, restore normal visceral function, and allow satisfactory coital function. Operations can be classified by compartment and by approach. It is important to enquire whether the woman is sexually active before considering vaginal surgery, as this may alter the choice of surgery. Other factors that may influence choice of surgery are the patients' fitness, and the surgeon's experience. There is lack of data on pregnancy outcomes and childbirth after surgery for prolapse. If the prolapse remains corrected and the patient conceives, delivery by elective caesarean section is advisable. Generally women should avoid lifting heavy weights and avoid sexual intercourse for 6-8 weeks[1].

- Operations in the anterior compartment- Anterior colporrhaphy. This operation rectifies a cystourethrocoele. It is no longer the first treatment of choice for major urethral sphincter incompetence; instead tension free vaginal tape (TVT) may be inserted with an anterior repair. Colposuspension is indicated for urethral sphincter incontinence associated with a 2nd or 3rd degree cystourethrocoele. Our patient was treated with anterior colporrhaphy.
- Operations in the middle compartment – Vaginal hysterectomy as done in our patient is now the treatment of choice rather than the Manchester repair. Vaginal hysterectomy can be combined with an anterior or posterior repair if a cystocoele is present. The Manchester repair involves anterior and posterior colporrhaphy combined with cervical amputation[2]. This operation is done in patients who are poor surgical risks. A sacrohysteropexy can be performed on women who wish to retain the uterus. It involves attaching the uterus to the anterior longitudinal ligament over the sacrum with a Y shaped graft[11]. Vault prolapse and enterocoele may be repaired using either sacrospinous fixation, ileococcygeal hitch or pubocervical rectovaginal repair. Abdominal or laparoscopic sacrocolpopexy are other alternative procedures.

- Operations in the posterior compartment – A rectocoele can be repaired by levator ani plication or by repair of discrete fascial defects. Posterior colporrhaphy may be combined with perineorrhaphy or perineoplasty if the perineal body is noted to be deficient and the patient has a good introitus[2].

Abdominal approach involving total abdominal hysterectomy and obliteration of any associated enterocoele can also be used to treat prolapse. However this method can be more morbid and time consuming and is optimal when combined with transvaginal repair of cystocoele and rectocoele. Vaginal obliterative operations (Le Fort's operation or vaginectomy) are rarely indicated in elderly women who are poor surgical candidates and who no longer desire coital function [2]. Complications of surgery include haemorrhage, sepsis and injury of contiguous organs, vessels and nerves. Possible long-term complications include postoperative urinary incontinence/retention and recurrent prolapse

Complications of untreated genital prolapse include[12]

- Decubitus ulceration of the vaginal epithelium or exposed cervix
- Leucorrhoea, abnormal uterine bleeding
- Hypertrophy of the cervix
- Keratinisation of the vagina
- Urinary tract infection
- Obstructive lesions of the urinary tract with hydronephrosis in procidentia
- Haemorrhoids result from straining to overcome constipation
- Small bowel obstruction from a deep enterocoele is rare.

The patient presented had a vaginal hysterectomy and anterior colporrhaphy, the commonest procedure in our set-up for patients with genital prolapse because most are beyond childbearing age.

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GYNAECOLOGY CASE 7

IMPERFORATE HYMEN WITH HAEMATOMETRA: CRUCIATE INCISION

NAME	:	E. W	D.O.A	:	18.4.2005
AGE	:	13 YEARS	D.O.D	:	21.4.2005
IPNO	:	1018484			
PARITY	:	O+O			

PRESENTING COMPLAINT

Cyclic abdominal pains and swelling for three months.

HISTORY OF PRESENTING COMPLAINTS

EW was well until three months prior to admission when she developed abdominal pain that was colicky in nature at the beginning of every month. The pain used to last between four and five days. She had also noticed some progressive abdominal swelling more at the suprapubic region that was associated with occasional difficulty in passing urine or stool. There was no history of vomiting or any febrile illness.

PAST MEDICAL HISTORY

This was not significant

OBSTETRIC AND GYNAECOLOGIC HISTORY

She was Para O+O and had not yet started getting her periods. She was not sexually active.

FAMILY AND SOCIAL HISTORY

She was a standard seven pupil at Muguga Primary School who lived with her parents. She neither smoked cigarettes nor drank alcohol. There was no family history of chronic illness in her family.

PHYSICAL EXAMINATION

She was in good general condition. There was no pallor, oedema, jaundice or lymphadenopathy. Her blood pressure was 100/60 mmHg, pulse rate 80 /minute regular, respiratory rate 20/minute and temperature 36.7^{0}C. The breasts were Tanner class 3.

Pubic and axillary hair was present with normal distribution (tanner class 3). The cardiovascular, respiratory and central nervous systems were essentially normal.

ABDOMINAL EXAMINATION

The abdomen moved with respiration and there was distension in the lower abdomen. There was a suprapubic mass corresponding to a 14-week gestation. It was firm with a regular outline, smooth surface and tender on palpation. The spleen and liver were not enlarged.

PELVIC EXAMINATION

The procedure and its importance were explained to the patient and her mother. They both gave verbal consent. She was put in lithotomy position. The external genitalia were normal with Tanner stage three growth of pubic hair. There was a bulging hymen obliterating the introitus. Hence digital pelvic or speculum examination was not done. The vulva was cleaned and the urinary bladder catheterized. Clear urine was drained but the mass persisted, on rectal examination the anal opening was normal. The rectal mucosa was freely mobile. A tender mass was felt filling the vagina and continuous with the suprapubic mass.

DIAGNOSIS

A diagnosis of imperforate hymen with cryptomenorrhoea was made.

INVESTIGATIONS DONE

1. Haemoglobin level 11.7g/dl
2. Urea and electrolytes

   Sodium 140 mmol/l Potassium 4.2 mmol/l

   Urea 5-mmol/l Creatinine 62 µmol/l
3. Ultrasound showed marked distension of the vagina and endometrial cavity filled with fluid. Both ovaries were normal in size and shape.

DEFINITIVE DIAGNOSIS

Haematometra/haematocolpos due to imperforate hymen.

MANAGEMENT

The diagnosis and mode of management was explained to the patient and her mother. Written consent for the operation of cruciate incision under general anaesthesia was obtained from the mother. The patient was starved overnight and premedicated with atropine 0.6 mg intramuscularly before being wheeled to theatre.

Cruciate incision

In theatre, general anaesthesia was induced and in lithotomy position vulval toilet was done and the perineum was draped with sterile towels. The bladder was catheterised and about 200 ml of clear urine drained. Examination under anaesthesia confirmed earlier findings.

An incision was made into the membrane starting 10 O'clock and extending to 4 O'clock. About 1000 ml of chocolate coloured fluid was drained. In the process the abdominal mass disappeared. Another incision was made from 8 O'clock to 2 O'clock. The four flaps created by the cruciate incision were trimmed off thus creating a round hole. Bleeding vessels were ligated and haemostasis achieved. A sterile pad was placed on the perineum and she patient reversed successfully from anaesthesia.

POSTOPERATIVE CARE

She was taken to the recovery ward and observed half hourly until fully awake. She was then transferred back to the ward. She was started on Amoxil 500 mg and Paracetamol 500 mg 8 hourly for 5 days. She remained stable and was for review in the gynaecology out patient clinic in two weeks time.

FOLLOW-UP

She was seen in the clinic and had no complaints. Examination showed she was in good general condition. She had no abdominal mass and no vaginal bleeding. She was discharged from follow-up.

DISCUSSION

The patient presented was a 13-year-old girl with imperforate hymen. Cruciate incision was made with satisfactory results.

The hymen is the junction between the sinovaginal bulbs and the urogenital sinus. It is a thin membrane sometimes cribriform in appearance composed of endoderm from the urogenital sinus epithelium[1]. It is usually perforated during embryonic life, but occasionally it may persist intact. Inflammatory occlusion after perforation can also lead to imperforate hymen[2].

With an imperforate hymen there is failure to establish a connection between the vagina and the vaginal vestibule[1,2]. The incidence of imperforate hymen in Kenya or at Kenyatta National Hospital is not known. Most cases occur sporadically with no evidence to suggest any genetic factor[3].

The patient had an imperforate hymen with no other associated anomaly. Imperforate hymen is almost always an isolated finding. However, associated urological abnormalities have been found in 20% cases. Associated congenital anomalies reported in these patients include urethral membrane, imperforate anus, bifid clitoris, hypoplastic kidneys with ectopic ureters and vascular anomalies[3].

Transverse vaginal septum results from faulty canalization of the embryonic vagina and when present at birth has similar presentation like imperforate hymen. Distinction between imperforate hymen and transverse vaginal septum is largely academic as their effects and treatment are identical[4].

If the uterus is absent or functionless imperforate hymen has no serious effects[4]. Even when the uterus is present, it is unusual to have symptoms in childhood. However due to stimulation of the uterus and cervix by placental hormones in utero, mucus may collect in the vagina causing mucocolpos at birth [4,5].

When menstruation starts at puberty, the vagina fills with blood and cervical secretions that remain fluid. As the amounts gradually increase with each cycle, first the vagina distends (haematocolpos), then the uterus (haematometra), and finally the tubes (haematosalpinx) [1,4]. This may continue until there is retrograde blood flow through the fallopian tubes and into the peritoneal cavity resulting in haemoperitoneum and chemical peritonitis[1,2,6]. In most cases the altered blood tends to set up an aseptic inflammation in the tubes that tend to close up the tubes and prevent or limit peritoneal spill. The enlarged vagina and uterus may displace the fundus of the bladder upward and thus causing urinary retention by interfering with the opening of the urethral sphincter[7].
The patient presented occasionally had difficulty in passing urine.

Our patient presented with low abdominal swelling and pain then on examination she was found to have imperforate hymen with haematometra. The diagnosis of imperforate hymen is mainly clinical and is made when an adolescent girl presents complaining of primary amenorrhoea and recurrent pelvic pain[2]. At times urinary retention is the first symptom. Other symptoms include lower abdominal pain, back pain and is aggravated by defaecation and urinary symptoms including urgency, frequency and dysuria. Overflow incontinence may eventually develop [1]. On examination a tender mass is palpable suprapubically. Vulval inspection reveals the bulging imperforate hymen, which may or may not be bluish in colour depending on its thickness. On rectal examination the vagina is palpable as a large cystic mass[2,5].

Our patient had a cruciate incision to relieve the haematocolpos.Imperforate hymen is corrected by surgical treatment, i.e. making a cruciate incision at position 2,4,8 and 10 O'clock and allowing the retained menstrual blood to drain freely. Digital vaginal examination is avoided so as not to introduce infection[1,2]; intercourse is also avoided for about 4 weeks; patient is started on antibiotics. If the uterine mass does not regress within 2-3 weeks, inspection and dilatation of the cervix should be performed to make certain that drainage from the uterus is satisfactory[1].
In the patient presented cruciate incision was done with good results.

Complications of imperforate hymen include psychological due to failure to receive periods, haematocolpos, haematometra, retrograde menstruation with haemoperitoneal peritonitis, salpingitis with pelvic adhesion formation, infection can occur due to the accumulated blood in the vaginal cavity. Infertility can occur due to salpingitis and endometritis[1,8].

Investigations like a pelvic ultrasound are done to rule out any other associated urogenital malformations e.g. mullerian agenesis, failure of vagina to canalize. It also helps to confirm or rule out the retention of menstrual blood, resulting in haematocolpos and haematometra[1,2].

The patient had a pelvic ultrasound that confirmed the presence of haematocolpos and haematometra secondary to an imperforate hymen.

An intravenous urogram (IVU) should also be done to investigate for urinary tract anomalies. Considering the simplicity of diagnosis and the relative ease of surgical correction, routine postnatal examination of such a condition is advisable as delayed diagnosis can lead to undesirable complications[3].

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GYNAECOLOGY SHORT CASE 8

CARCINOMA IN-SITU OF THE CERVIX -WERTHEIMS HYSTERECTOMY

NAME:	A. W.	L.M.P.	May 2003
AGE:	28 Years	PARITY.	2 + 0
I.P NO.	0968354		
D.O.A.	2/8/04		
D.O.D.	10/8/04		

PRESENTING COMPLAINTS

She was admitted from the colposcopy clinic with a diagnosis of carcinoma in situ of the cervix for wertheim's hysterectomy.

HISTORY OF PRESENTING COMPLAINT

She had been referred to Kenyatta National Hospital (KNH) from a family planning clinic in Kariobangi where she had been found to have abnormal cervical cytology on routine check up. The cytology showed she had high-grade squamous intraepithelial lesion (HGSIL). She had presented to her family planning clinic complaining of low abdominal pain. She had no abnormal vaginal bleeding or discharge. She did not have any post coital bleeding. At KNH, colposcopy was done on 30/6/04. The transformation zone was well visualized and after application of acetic acid, a biopsy was taken from the 6 o'clock position which appeared white. Other areas appeared normal. The histology report indicated cervical tissue with moderate dysplasia and a background of human papilloma virus (HPV) infection. The dysplastic portion extended beyond the biopsy section margins. On review with these findings, a decision to do a loop electro-excision procedure (LEEP) biopsy was made and this was done at KNH on 2/7/04. The histology of the specimen taken indicated that the transformation zone was visible, and there was carcinoma in situ extending to the endocervical glands with surrounding HPV infection. With these results, AW was admitted for surgery.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She attained her menarche at 14 years. She experienced her first intercourse at the age of 16 years. She has had 3 partners since then. She had regular menstrual cycles at every 28 days with flow duration of 3 days. However she had been amenorrhoeic for the last 16 months since she started Depo-Provera as her contraceptive of choice. She had also used combined oral contraceptives for three years prior to starting Depo-Provera. She was a para 2 +0 with 1 living child. Her last delivery was 8 years ago. She had not been treated for any sexually treated infection before.

PAST MEDICAL HISTORY

She had no prior admissions. She had no known drug or food allergies.

FAMILY AND SOCIAL HISTORY

She was a single business lady who lived at Kariobangi.She smoked about 3 cigar sticks every day for the last 3 years, and also took alcohol occasionally. She is the tenth born in a family of eleven siblings. She was educated up to standard 8.She had no family history of any chronic illnesses.

SYSTEMIC ENQUIRY

This was not contributory.

PHYSICAL EXAMINATION

She was in a good general condition, not pale, not jaundiced, had no lymphadenopathy or oedema. She was afebrile.

VITAL SIGNS

Temperature-37.2^{0}C, pulse rate 76 beats/min, blood pressure 120/70mmHg and respiratory rate 17/min.

CARDIORESPIRATORY SYSTEM

Her pulse was regular, of good volume. Both heart sounds were audible and normal, there were no murmurs. The respirations were normal vesicular in both lung fields.

NERVOUS SYSTEM

This was essentially normal.

ABDOMINAL EXAMINATION

The abdomen was scaphoid, moved well with respiration, was soft, had no masses or areas of tenderness. The bowel sounds were normal.

SPECULUM EXAMINATION

The vulva and vagina were normal, the cervix had scarring after LEEP was done.

DIGITAL EXAMINATION

The cervix felt firm, the uterus was mobile, anteverted, and normal size. There was no mass or tenderness in the adnexa. The pouch of Douglas was not full.

IMPRESSION

Carcinoma in situ of the cervix.

INVESTIGATIONS

1. Haemogram- haemoglobin -13.0g/dl
white cell count –9.7 x 10^9/mm3
platelet count - 334 x 10^9/l

2. Urea and electrolytes
Na^+ - 134 mmol/L
K^+ - 5 mmol/L
Urea –3.4 mmol/L
Creatinine – 58umol/l

3. Blood group – O positive

4. LEEP biopsy – moderate dysplasia with HPV effect extending beyond the margins of excision. Carcinoma in situ (C.I.S) extending to endocervical glands
Conclusion; CIS with HPV effect.

5. H I V - Negative

MANAGEMENT

After consideration of the colpocsopic and LEEP histology reports, and discussion with the patient, a decision was made to treat her by Wertheim's hysterectomy as their was a likelihood that some of the dysplastic areas left could have had microinvasive cancer. She was explained on the condition and mode of management to which she gave informed consent. On the night prior to operation she was given an enema and this was repeated on the morning of operation. She was starved from midnight and premedicated with 0.6mg atropine and 50mg pethidine ½ hour before theatre .Two units of blood had been availed and the patient had auto-donated one unit.

WERTHEIMS HYSTERECTOMY

In main theatre, the patient was put under general anaesthesia, vulvo vaginal toilet done. Examination under anaesthesia was done, revealing a normal uterus, free adnexa, and a normal pouch of Douglas. A decision was made to continue with the planned procedure. The abdomen was cleaned surgically and draped. The abdomen was opened via a midline incision and the gut packed away to expose the pelvic vessels and ureters.
The round and ovarian ligaments were divided between clamps. The broad ligament was opened and paravesical space opened. The ureter was dissected in the parametrium as far as the point of entry into the bladder. The ureter was retracted away from the field of operation to avoid injury. The nodes along the internal iliacs, common iliac and obturator

vessels were dissected and sampled. This procedure was repeated bilaterally. The uterine arteries were clamped and ligated at their origins from the internal iliacs. The vagina was double clamped, excised between the clamps and the vault closed with mattress suture No. 2/0 vicryl. Haemostasis was achieved.
The abdomen was lavaged with warm saline and rifocin, the abdominal wall was then closed in layers after ascertaining the correct swab and instrument count. Anaesthesia was reversed successfully and the patient taken to the recovery ward for observation. She was transfused one unit of blood intrao-peratively.

POSTOPERATIVE MANAGEMENT

The patient was observed ¼ hourly till fully awake then every two hours for two hours and four hourly thereafter .She was started on pethidine 100mg intramuscularly every 8 hours, Augmentin 1.2g 12 hourly and metronidazole 500mg 8 hourly. She was kept on intravenous fluids (normal saline alternating with 5% dextrose 500mls every 4 hours) until the time she was able to feed orally.
On the second postoperative day she was started on oral sips and by the third day she was put on oral medication and was on full diet. The urinary catheter was removed and she was advised to be ambulant. By the fifth day she had no complaints, the wound was dry and healing well. She was thus discharged home for follow up in the GOPC in six weeks time together with the histology results of the specimen taken at hysterectomy.

FOLLOW UP

She came to the clinic and had no complaints. She did not have any vaginal discharge or pelvic pain. Her wound had healed well, she had no lower limb oedema. The histology of the specimens removed at surgery were availed and reported as follows
Gross; specimen of uterus, tubes and lymph nodes.
Microscopy; Sections from the cervix show areas of severe dysplasia, and carcinoma in situ of the cervix. Sections from the myometrium showed foci of adenomyosis. The endometrium was normal
Lymph nodes microscopy showed a reactive hyperplasia. No malignancy was seen.

She was adviced to continue with regular vault smear evaluation to detect any cancer recurrence at an early stage.

DISCUSSION

A case is presented of a 28 years old lady who was diagnosed to have early carcinoma in situ of the cervix and was managed by wertheim's hysterectomy. She was well at the follow-up visit and was adviced on regular follow up.

Carcinoma in situ (CIS) of the cervix occurs when the full thickness of the cervical epithelium is encompassed by disordered growth and development of the lining. It is the most extreme form of cervical intraepithelial neoplasia. Left untreated, a certain number of patients with carcinoma in situ, especially those with high grade lesions progress to invasive cancer of the cervix[1]. The patient presented had carcinoma in situ.
Cervical cancer is the second most common malignancy in women worldwide and remains a leading cause of cancer related deaths and a major public health problem especially in developing countries. At present, there are nearly 1 million women each year getting cervical cancer globally. Three quarters of all women with cervical cancer are in the developing countries. The incidence in developed countries has been on the decline largely due to implementation of population based screening programmes and treatment of preinvasive disease in these countries[1, 2]. The true incidence of preinvasive disease and cancer in Kenya is not known although it is the most common gynaecologic malignancy encountered in clinical practice. Studies in Kenya show that the mean age of onset is for cancer of the cervix 42 years with peaks in incidence at 25, 30 and 35 years. The mean age at onset is 10 years earlier than what is seen in the developed countries where 2 peaks of incidence for cancer are described at 35 and 50-55 years [3-5]. The peak age worldwide is 48-55 years. The peak incidence of carcinoma in situ worldwide is in women 25-35 years[1]. Our patient was 28 years old.

She was found to have human papilloma infection effects on the cervical epithelium. The exact cause of carcinoma in situ and cervical cancer is not known but various aetiologic risk factors have been implicated. These include:

- Human papilloma virus infection. This has been identified as the most important factor responsible for the association of CIS and cervical cancer with sexual activity. HPV DNA is found in 95% of all squamous cell carcinoma and in over 90% of all adenocarcinomas. HPV types 16,18,45 and 56 are the most oncogenic types accounting for approximately 80% of HPV types in cervical cancer. Other high-risk types include types 31, 39,58,59 and 68. Other factors that have previously been associated with increased risk of cervical intraepithelial neoplasia such as increased lifetime number of sexual partners, high risk sexual partners (history of multiple partners, HPV infection, lower genital tract neoplasia or prior sexual exposure to partner with cervical neoplasia), a history of sexually transmitted infection, early age at first intercourse, lower level of education and lower socio-economic status have actually been shown to be a result of HPV infection[6-8]. HPV infection was noted in the patient presented and could have contributed to her condition.
- Smoking. Cigarette smoking is associated with a two to fourfold increase in the relative risk for developing cervical cancer. Cigarette smoke carcinogens (nicotine and cotinine) accumulate locally in the cervical mucus and the cumulative exposure is related to the risk of developing carcinoma. Men also concentrate these chemicals in

their genital secretions and can bathe the cervix with these chemicals during intercourse. Male partners of women with cervical dysplasia should thus be adviced against smoking[9]. The patient presented was smoking cigarettes daily.

- Nutritional deficiencies are also thought to be involved in occurrence of cervical dysplasia and cancer. The national cancer institute recommends that women should consume five servings of fresh vegetables or fruits every day[9]. If this is not possible, then they should consider taking a multivitamin everyday with antioxidants such as vitamin E or beta-carotene.
- Immunodeficiency. The incidence of cervical neoplasia and cancer is increased in HIV-infected women. HIV infection allows neoplastic proliferation due to deficient host regulatory mechanisms. Since 1993, invasive cervical cancer has been included as an AIDS-defining illness. Women with HIV have been noted to have more advanced disease at presentation, poorer response to therapy and higher recurrence and death rates. The presented patient was HIV-negative.
- Other associated factors include multiparity and long-term oral contraceptive pill use, protein antigen in the sperm head, smegma in uncircumcised males, herpes simplex type II.

The patient presented started sexual activity at 16 years of age, HPV was noted on Pap smear, had had 3 sexual partners and smoked cigarettes.

On cytologic examination, the cells in CIS are dysplastic characterized by anaplasia, an increased nuclear: cytoplastic ratio, hyperchromatism, multinucleation and abnormalities in differentiation[1,2].

Grossly, the ectocervix is the common site of the cancer lesion occurring in 80% of cases while in 20% the lesion is in the endocervix. The lesion may be exophytic (a proliferative lesion forming a friable mass almost filling up the upper vagina in late cases), ulcerative where it excavates the cervix and often involves the vaginal fornices, and the infiltrative or endophytic type that expands the lower uterine segment giving rise to the barrel shaped cervix.

Commonly cervical cancer arises from either the squamous epithelium of the ectocervix or the columnar epithelium of the endocervical canal. About 85-90% of invasive cervical cancer is squamous cell carcinoma. The sources of the squamous epithelium that turn into malignancy are- squamocolumnar junction, healing erosion, squamous metaplasia of the columnar epithelium and the squamous cell rests in the ectocervix. Squamous cell carcinoma is further divided into three groups histologically namely large cell keratinising, large cell non-keratinising and the small cell type. Those with small cell type have poorer prognosis. Adenocarcinoma accounts for 10-15% of cervical cancer and develops from the endocervix either from the lining epithelium or from the glands. Currently there is increase in the number of cervical adenocarcinomas especially in the younger age-group below 35 years reported to be 25%. Other types of cervical cancer include endometrioid, clear cell, adenosquamous and mixed types. Others are sarcoma, malignant melanoma metastatic cancer and small cell (neuroendocrine) type [1,7-9].

Carcinoma of the cervix spreads principally by direct local extension followed by lymphatic spread. The growth spreads directly to the adjacent structures-bladder and

rectum, the vagina and to the body of the uterus. Extension laterally is to the parametrium, paracervical and paravaginal tissues where the ureter may be compressed. The primary lymphatic groups involved in spread are parametrial nodes, internal iliac nodes, obturator, external iliac and sacral nodes. Nodes involved secondarily are the common iliac, the inguinal and para-aortic nodes. Haematogenous spread accounts for less than 5%, occurs late and is usually by veins rather than arteries. Lungs, liver or bone are usually involved. Direct implantation of the cancer cells at operation on the vaginal vault or abdominal wound is very rare.

There are usually no symptoms or signs of CIS, and diagnosis is often based on biopsy findings following an abnormal routine cervical cytology smear. This was the case in our patient. She was found to have a high grade lesion on Pap smear. Vaginal bleeding or discharge are the most common presenting symptoms in women with invasive cervical cancer. The duration of symptoms may not be proportionate to the stage of the disease. In the early stages of the disease, carcinoma of the cervix is asymptomatic. Menstrual abnormalities in the form of contact bleeding, bleeding on straining, intermenstrual bleeding are very much suspicious especially in women over the age of 35. Excessive discharge that may be at times offensive may be present. Other symptoms include pelvic pain, referred pain. Speculum examination may reveal erosion, nodular growth or an ulcer. The lesion bleeds on friction. Bimanual examination reveals the lesion that bleeds to touch. Rectal examination reveals the state of the parametrium and rectal wall.

It is important to estimate the disease extent not only as an aid to prognosis but also to plan treatment. The staging of carcinoma of the cervix is principally clinical. The staging process involves speculum, pelvic and bimanual examination, and rectal examination, which are done under general anaesthesia. Confirmation of diagnosis is by biopsy. Ancillary aids for confirmation of staging include cystoscopy, intravenous pyelography, proctoscopy and chest radiography. Routine haematological, liver and renal function tests aid in planning treatment. The fallacies in clinical staging include difficulty to assess lymph node involvement on clinical examination which adversely affects prognosis and the difficulty in differentiation of inflammatory and malignant induration of the parametrium. The classification adopted by the International Federation of Gynaecology and Obstetrics (FIGO- 1998) for staging of cervical cancer is as follows [1,8]:

Stage 0: Carcinoma in situ, intraepithelial carcinoma

Stage I: Carcinoma strictly confined to the cervix (extension to the corpus to be disregarded).

Stage IA: Microscopic disease confined to the cervix. Further divided into:

IA_1: minimal microscopically evident stromal invasion not exceeding 3mm in depth and not more than 7mm in width (horizontal spread).

IA_2: Microscopically measured invasion greater than 3mm but less than 5mm and no wider than 7mm in horizontal spread.

Stage IB: Lesions greater dimension than stage Ia_2 whether seen clinically or not and macroscopic disease confined to the cervix. Further divided into;

IB_1: Lesion not exceeding 4cm in size.
IB_2: Lesion exceeds 4cm in size.

Stage II: The carcinoma extends beyond the cervix but has not extended to the pelvic wall. The carcinoma involves the vagina, but not the lower third.
IIA: Vaginal involvement without parametrial involvement
IIB: Parametrial involvement.

Stage III: Tumour extends to pelvic sidewall and/or causes hydronephrosis and/or extends to lower third of vagina.

IIIA: Involvement of the lower third of vagina with no extension to the side
IIIB: Extension to pelvic sidewall and/or hydronephrosis.

Stage IV: Extension beyond the true pelvis or into mucosa of rectum or bladder.

IVA: Extension into adjacent organs.

IVB: Distant metastasis.

The patient presented was in class 0 of the FIGO staging system.
Pelvic node metastasis in different stages of cervical cancer are; 0.5% in stage IA_1, 8% in stage IA_2, 16% stage IB, 30% stage II, 44% for stage III and 55% in stage IV.
Prognosis depends on the following factors[1,8]

- Type of tumour. Endocervical tumour is diagnosed late and grows faster
- Stage of the disease. Early disease has better prognosis.
- Tumour size more than 4cm is associated with more lymph node metastasis.
- Lymph node involvement especially pelvic and para-aortic reduces survival rate.
- Depth of tumour invasion when <1cm associated with improved survival.
- Age. Young age is usually associated with poorly differentiated squamous cell carcinoma or adenocarcinoma and is prognostically poor.

Management of CIS depends on the degree of dysplasia, natural history of various degrees of CIS, the patients immune competence, smoking habits and other factors such as the inciting HPV type. HPV typing was not done in our patient as the facility is not readily available. She had been reported to have severe dysplasia and was smoking. The most common techniques used for the treatment of CIS include ablative techniques (cryotherapy or laser ablation), and three excisional techniques-cold knife conisation, laser cone excision and Loop electrosurgical excision procedure (LEEP). Cure depends on the size of the lesion, endocervical gland involvement, and margin status of any excisional specimen[1,2] Our patient had LEEP initially, which showed there was endocervical gland involvement and the dysplasia extended beyond the excisional margins and further surgery was then done.

Management of invasive cancer of the cervix depends on stage of the disease, patients' age and fertility desires among other factors. Surgery and radiotherapy are the main modalities of treatment. A combination of the two modalities may also be employed in some cases. Other modalities include chemotherapy, neoadjuvant approach where cytoreduction via cytotoxic chemotherapy is used prior to definitive therapy with radiation or radical surgery. Surgery is used only for early disease stage I and IIA. Our patient was treated with surgery as she was considered to have early stage disease. Advantages of surgery as a primary procedure includes more thorough assessment of the spread and extent of the disease, preservation of ovarian function if desired, retention of more pliable and functional vagina especially in the young woman and psychologic benefit to the patient that her cancer bearing organ has been removed. Types of surgery done depending on stage of disease are cervical conisation, simple hysterectomy and radical types such as Wertheim's hysterectomy. Complications of surgery include fistula formation, bleeding at time of surgery, lymphocyst formation, venous thrombosis and bladder dysfunction. Pelvic exenteration is an ultraradical type of surgery done in cases of recurrent carcinoma where radiation had been done. This patient did not develop any complications.

Radiotherapy may be used to treat all stages of the disease if there are no contraindications. Advantages of radiotherapy as primary treatment include wider applicability in all stages of disease, comparable result with that of surgery in early stages, less primary mortality and morbidity, individualization of dose distributions and requirement is possible. Brachytherapy technique is employed. Radiation sources include radium, cesium or cobalt. Contraindications of radiotherapy include younger patients, presence of pelvic inflammatory disease, uterine fibroids, ovarian tumour and vaginal stenosis [1,7,8,10]. Complications of radiotherapy include bowel perforation, fistula formation, vaginal stenosis and uterine perforation during introduction of the uterine tandem. Chemotherapeutic agents used include cisplatin, bleomycin, methotrexate and vincristine. Chemotherapeutic agents may additionally be employed as radiation sensitisers to enhance response in patients with advanced disease. The results of therapy is expressed in terms of 5-year survival rate. This is given for the various stages as 85% stage I, 55% for stage II, 38% for stage III and 15% for stage IV disease [1,2,8-13].

Prevention of CIS and cancer of the cervix involves proper health education on risk factors and screening, especially among the poor socio-economic group, removal of the cervix during routine hysterectomy to prevent stump carcinoma. Information about the association of HIV infection and smoking with development of cervical cancers is crucial and should be provided to all women. Universal cytologic screening of all post-pubertal women on a regular basis should be encouraged. Cervical cytology screening by means of Papanicolaou smear offers great hope for early diagnosis and treatment of premalignant conditions of the cervix. There are four main reasons for this: the ability of cervical cells to exfoliate from pre cancerous lesions, accessibility of the uterine cervix, existence of a spectrum of change from atypia to pre malignant to frankly malignant state and the prolonged natural history of cancer of the cervix.

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GYNAECOLOGY CASE 9

OVARIAN CANCER –TOTAL ABDOMINAL HYSTERECTOMY AND BILATERAL SALPINGO-OOPHORECTOMY

NAME: E. K. PARITY 8 + 0

AGE: 49 Years L.M.P 6 years postmenopausal.

I.P. NO: 0819246

D.O.A: 08/7/02
D.O.D: 16/8/02

PRESENTING COMPLAINT

She presented with a six-month history of progressive abdominal swelling.

HISTORY OF PRESENTING ILLNESS

She was well prior to the onset of abdominal swelling. She gradually noted that her clothes were becoming tighter and could not fit her well. The swelling was associated with general malaise and reduced appetite. She had not noted any weight loss. Two days prior to admission she had two episodes of fainting attacks associated with abdominal pain.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She had attained her menarche at 18 years, her parity was 8 + 0 but she had only four living children while the others were perinatal deaths. Her first delivery was in 1974 and her last one in 1985. Her last normal menstrual period was in 1996 and was therefore 6 years postmenopausal. She had an intra uterine contraceptive device (IUCD) since 1985 to the time of admission.

PAST MEDICAL HISTORY

She had been admitted once previously after having fallen from a moving bus and was found to only have soft tissue injury. She had no chronic illnesses or allergies.

FAMILY AND SOCIAL HISTORY

EK was married and lived with her family at Shauri Moyo. She was working with the ministry of public works in Nairobi and her husband was a businessman. Her mother was diabetic. She had no family history of thyroid, uterine, breast or ovarian disease.

PHYSICAL EXAMINATION

She was in a fair general, was mildly pale but had no jaundice or oedema. Her vital signs were normal; her blood pressure was 110/70mmHg, respiratory rate 18 breaths/minute, temperature 36° C. She had normal central nervous, respiratory and cardiovascular systems.

ABDOMINAL EXAMINATION

E K had gross abdominal distension with fullness of the flanks. She had an umbilical hernia about 4cm in diameter. She had a mobile mass that was firm, cystic, slightly tender equivalent to a fundal height of 34 weeks gestation. She had a moderate amount of ascites. She did not have any enlarged lymph nodes.

PELVIC EXAMINATION

She had normal external genitalia, the cervix was closed and an IUCD string was felt. The vaginal wall was normal. The uterine size was not easy to appreciate because of tenderness. Bimanual examination was also not done due to the tenderness.

INVESTIGATIONS

1. Haemoglobin 9.4g/dl

2. Urea and electrolytes
   Na^+ 130 mmol/l
   K^+ 3.7 mmol
   Urea 5.2 mmol/l
   Creatinine 77 umol/l
3. Liver function tests
   Total protein 64g/dl; Albumin 32g/dl
   Liver enzymes normal
   Bilirubin levels normal
4. Pap smear
   Satisfactory with no abnormal cells. No evidence of malignancy.

5. Abdominal scan
   Normal liver, spleen, kidneys. Had moderate ascites.

6. Pelvic scan
   Normal uterus and a huge multiseptate mass arising from the pelvis measuring 17.8cm x 17.4 cm.
7. Chest x-ray . Normal lung fields with no features suggestive of metastasis.

DIAGNOSIS

Ovarian tumour

MANAGEMENT

EK was transfused 2 units of blood before operation and this raised her haemoglobin to 11.1g/dl. She was counselled and consented to total abdominal hysterectomy and bilateral oophorectomy. The night prior to surgery she was starved from midnight, given 1g of metronidazole and an enema for bowel sterility. On the morning of surgery she was premedicated with pethidine 50mg and atropine 0.6mg half hour before being taken in for surgery.

In theatre, general anaesthesia was induced, she was catheterised, the abdomen cleaned and draped. The abdomen was then opened via an extended midline incision in layers. She was found to have moderate haemorrhagic ascites of 400mls which was drained and a large right ovarian tumour measuring 30 x 25x 25 cm. The tumour was necrotic, cystic in areas and solid in other parts. The mass was freely mobile. The left ovary was normal but the tumour extended to the pouch of Douglas and there were peritoneal deposits. The liver and diaphragm were free of tumour. There were flimsy adhesions on the side of the tumour which were released by blunt dissection to free the mass. The left tube was attached to the mass. The mass was then excised whole by double clamping its pedicles in stages-the infundibulopelvic and ovarian ligaments, cutting between the clamps and then ligating the remaining pedicle. A total abdominal hysterectomy was then done using the procedure as outlined in the introduction of this book. Right salpingo-oophorectomy was included in the hysterectomy by double clamping, cutting and ligating the infundibulopelvic ligament on that side. Infracolic omentectomy was then done by serial clamping, cutting and ligating close to its attachment to the gut. Haemostasis was ensured throughout the operation. The abdomen was then cleaned with warm saline and the wall closed in layers. She was then reversed from anaesthesia and taken to the recovery room.

POSTOPERATIVE MANAGEMENT

She was observed until fully awake and then four hourly. She was on intravenous fluids over 24 hours after which she was started on oral sips and gradually on light diet. She was also given penicillin 2MU 6 hourly and gentamicin 80mg 8 hourly. By the fourth day her wound was healing well and she was allowed home after removal of skin stitches on the eighth day. She was to be seen in the out patient clinic within two weeks with results of histology of the mass removed.

FOLLOW UP

She came to the clinic and had no complaints. Her wound had healed well and the abdomen was soft. Histology results indicated the following; borderline mucinous cystadenocarcinoma of ovary, simple endometrial hyperplasia, normal left ovary, normal cervix, inflamed peritoneum with no cancer cell deposits noted.

Due to this she was readmitted for chemotherapy and was started on cyclophosphamide 500mg daily for 5 days, cisplatin 50mg once, adriamycin 50mg once for every course of chemotherapy. This was repeated every three weeks after ascertaining normal haematological, renal and liver functions with appropriate laboratory tests (haemogram, urea and electrolytes, liver function tests). She received a total of six courses of chemotherapy. She did not develop any signs of tumour recurrence, abdominal masses or ascites. She still continues with the out patient clinic.

DISCUSSION

This is a case of a 49 years old para 8+0 lady who presented with carcinoma of the ovary and had total abdominal hysterectomy and bilateral salpingo-oophorectomy and omentectomy followed by chemotherapy.

Cancer of the ovary comprises 25% of all malignancies of the genital tract. It is the fourth leading cause of death due to cancer in the United States of America. It has low incidence in Japan, Spain and India. It is commonest in U.S.A and Western Europe[1]. At Kenyatta National Hospital, Njuki found that cancer of the ovary comprises 8% of all female genital malignancies. It ranked third in KNH as a cause of gynaecological malignant disease after cancer of the cervix and choriocarcinoma[2,4].

Our patient was 49 years and had not used oral contraceptives previuosly. Worldwide the prevalence is age related with increased risk after 45 years and peak between 60 and 75 years with the majority of cases between 40 and 60 years. The patient presented was 49 years. No definite aetiological factors have been established for ovarian cancer, but it is known that the risk increases with age. Women of low parity, decreased fertility and delayed child bearing are at a greater risk. There also seems to be a familial predisposition in some women. Our patient did not have family history of ovarian or other cancers. Other studies have shown that early age at first pregnancy confers greater protection against cancer than high parity. Breastfeeding has also been shown to be protective and an early menarche and late menopause are associated with increased risk. Use of oral contraceptives also is thought to decrease the risk of ovarian cancer in patients aged 40-59 years. This protection appears to persist as long as 10 years after discontinuing use of oral contraceptives. Tubal ligation is associated with a reduction in risk of developing ovarian cancer. Chronic anovulation, multiparity are also protective. Pregnancy decreases the risk of ovarian cancer by 30-60%, oral contraceptives by 30-60% depending on the duration of use. Other factors thought to be associated with ovarian cancer include; use of asbestos containing talc powder, previous pelvic irradiation and excessive dietary vitamin A, high fat diet and fertility drugs. Genetic factors also appear to play an important role in the development and progression of ovarian cancer. Although most cases of epithelial ovarian cancer are sporadic and exhibit no heritable tendencies, approximately 7% occur in women with suggestive family history. The most common pedigrees are sister/sister and mother/daughter patterns. Three heritable syndromes have been described; site specific ovarian cancer, familial cases of breast and ovarian cancer, cancer family syndrome characterized by the occurrence of colon cancer and adenocarcinoma of the ovary, breast or uterus or a combination also known as Lynch II syndrome (hereditary non-polyposis colon cancer syndrome). Molecular biologic studies suggest the presence of one or more tumour suppressor genes on chromosome 17 may play a role in the aetiology of ovarian cancer [3,5]. Our patient did not have any of these risk factors.

Our patient had mucinous cystadenocarcinoma. Ovarian cancer may be divided into three major categories based on the cell type of origin. The ovary may also be the site of metastatic disease by primary cancer from another organ site. Unlike cancers of the

cervix and endometrium, precursor lesions of ovarian carcinoma have not been defined. The major histopathologic categories of ovarian cancer are:

- Epithelial ovarian cancer; includes serous, mucinous, endometrioid, clear cell, transitional cell and undifferentiated cell types. Epithelial tumours account for over 60% of all ovarian neoplasms and more than 90% of malignant ovarian tumours. Ovarian serous cystadenocarcinoma is the most common malignant tumour of the ovary.
- Germ cell malignancies. These include dysgerminoma, endodermal sinus tumour, immature teratoma, embryonal carcinoma, choriocarcinoma, gonadoblastoma and mixed germ cell tumours.
- Sex cord stromal tumors including granulosa cell tumour, fibroma, thecoma and sertoli leydig tumours.
- Neoplasms metastatic to the ovary. Usually from the breast, colon, stomach and endometrium.

The degree of cellular differentiation of the epithelial ovarian neoplasms is used to further grade the tumour. Grade 0 tumours are also known as borderline malignancies or tumours of low grade potential. Carcinoma of the ovary can be staged at surgery as follows (International Federation of Gynaecologic and Obstetric-FIGO staging); Our patient had stage IIIB disease as she had peritoneal deposits. The stages are:

Stage I; Growth limited to the ovaries

IA; Growth limited to one ovary, no ascites containing malignant cells. No tumour on the external surfaces.

IB; Growth limited to both ovaries, no ascites containing malignant cells. No tumour on the external surfaces.

IC: Tumours either stage IA or IB but with tumour on the surface of one or both ovaries or with capsule ruptured, or with ascites present containing malignant cells or positive peritoneal washings.

Stage II: Growth involving one or both ovaries with pelvic extension.

IIA: Extension and/or metastasis to the uterus and/or tubes.

IIB: Extension to other pelvic tissues.

IIC: Tumour either stage IIA or IIB but with tumour on the surface of one or both ovaries, or with capsule(s) ruptured or with ascites present containing malignant cells or with positive peritoneal washings.

Stage III: Disease extension to the abdominal cavity.

IIIA: Abdominal peritoneal surfaces with microscopic metastases

IIIB: Tumour metastases less than 2cm in size.

IIIC: Tumour metastases more than 2cm in size or metastatic disease in the pelvic, para-aortic, or inguinal lymph nodes.

Stage IV: Distant metastatic disease

Malignant pleural effusion, metastases to the supraclavicular lymph nodes

or skin. Pulmonary parenchymal metastases, liver or splenic parenchymal disease (not surface implants).

The patient presented was admitted with abdominal swelling and abdominal discomfort. A pelvic scan done showed a huge septate mass arising in the pelvis. Diagnosis of ovarian cancer is usually done late due to lack of appropriate screening tests and early symptoms or signs. Patients ignorance due to lack of education and non-availability of health services in remote areas of the country also contribute to late diagnosis. Ovarian cancer typically develops as an insidious disease, with few warning signs or symptoms. Most neoplastic ovarian tumours produce few symptoms until the disease is widely disseminated throughout the abdominal cavity. Most presenting symptoms are those associated with increasing tumour mass, spread of tumour along the serosal surface of the bowel and ascites. Abdominal discomfort, upper abdominal fullness and early satiety are associated with cancer of the ovary. Other signs and symptoms include fatigue, increasing abdominal girth, urinary frequency and shortness of breadth caused by pleural effusion or massive ascites. Elevated oestrogen production in certain stromal tumours may cause abnormal uterine bleeding. On physical examination a pelvic mass that is bilateral, irregular, solid or fixed is suggestive. Other findings include thrombophlebitis and hypercalcaemia [1]. Constipation, sensation of pelvic weight or pressure and pain may be present. Androgen producing tumours e.g. sertoli-leydig cell tumours may cause virilisation or hirsutism. Granulosa cell tumours are clinically oestrogen-producing tumours that present with abnormal vaginal bleeding or precocious puberty in young girls. There were no features to suggest an oestrogen producing tumour in our patient.

No tumour marker tests were done for our patient before laparotomy. Diagnosis of ovarian cancer poses a great challenge to the oncologist. A concise history and physical examination is paramount. The prognosis of ovarian cancer is significantly improved when the disease is detected while still confined to the ovary. Unfortunately routine pelvic examination is a notoriously poor screening method with limited sensitivity and specificity. Aids to diagnosis include ultrasonography and laparoscopy. Computed tomography scans can also be done. Tumour markers are proving useful in diagnosis and management of several types of ovarian tumours. These include CA-125, NB 70K, LASAP for epithelial tumours), β-HCG for trophoblastic disease, LDH for dysgerminomas, alpha-fetoprotein for endodermal sinus tumour, testosterone for sertoli-leydig tumour. However tumour markers have largely been disappointing except for the rare germ cell tumours because they may also be elevated in numerous benign conditions including pregnancy, pelvic infection and other conditions such as liver disease, breast and lung tumours. All said, they are useful in monitoring effectiveness of chemotherapy[2,5,6,7]. In patients above 45 years of age, colonoscopy or barium enema and proctoscopy should be performed to rule out the presence of colonic involvement, colonic cancer or inflammatory disease of the bowel[3]. Intravenous urography may be done to define the course of the ureters and to exclude the rare pelvic kidney. Cystoscopy is indicated where involvement of the anterior pelvis is suspected to rule out bladder involvement. Chest X-ray should be done prior to surgery to detect pleural effusion or metastatic disease. Chest x-ray in our patient revealed no metastasis.

Our patient had surgery at which staging was done, and hysterectomy with bilateral oophorectomy plus omentectomy were done.The management of suspected ovarian malignancy includes investigations as mentioned above followed by surgery at which definitive treatment is commenced after confirmation of the disease. Surgery is the cornerstone of therapy for ovarian cancer, regardless of cell type or stage of disease. A gynaecologic oncologist should be consulted whenever this is planned. Surgical preparation and procedures for ovarian neoplasm include surgical staging, debulking of advanced disease, and secondary debulking of recurrent or progressive disease and palliative surgery for ovarian cancer induced intestinal obstruction. Surgical procedures may include hysterectomy and bilateral salpingo-oophorectomy, resection of fixed ovarian tumours, pelvic lymphadenectomy, omentectomy, splenectomy, small bowel resection or by-pass, ureteral resection. Our patient underwent hysterectomy and bilateral salpingo-oophorectomy. During laparotomy, there are 9 steps to be followed in managing surgical stage I and II ovarian cancer;

Step I
A vertical midline incision is made on the abdominal wall. Cautery is normally used on the subcutaneous tissue, fascia and peritoneum to ensure that no blood contaminates the peritoneal cavity and thus the peritoneal washings.

Step 2
Peritoneal washings are obtained from the pelvis and both the right and left paracolic gutters by instilling about 100mls saline into each area.

Step 3
Because of the large volume of the diaphragm, random biopsies of normal-appearing diaphragm are discouraged. Thorough cytologic sampling of the undersurface of the diaphragm can be obtained by cytobrush as used for endocervical Papanicolaou smears.

Step 4
The entire pelvis and abdomen are carefully explored and biopsy of any suspicious nodules is taken.

Step 5
Ovarian tumour is resected and frozen section is taken.

Step 6
If malignancy is confirmed, total abdominal hysterectomy and bilateral salpingo-oophorectomy are performed.

Step 7
Omentectomy is performed.

Step 8
Pelvic lymphadenectomy is performed on the side of the tumour. Sampling of enlarged pelvic lymph nodes is discouraged since metastases are frequently microscopic and

unrecognized. Therefore, lymph nodes should be removed from the common iliac artery and obturator fossa.

Step 9

Para-aortic lymphadenectomy is performed by removing the lymph nodes from the aorta and vena cava from the bifurcation of the aorta to just below the renal vessels.

At laparotomy, the incision used should provide maximum exposure of the pelvis and allow thorough evaluation of the abdomen. The operation aims at resecting as much tumour as is safely possible. The site of the mass is noted and whether it is cystic or solid. The surface is checked; it may be smooth and irregular. The size of the mass is noted, as is its adherence to surrounding structures. The presence of ascites is also noted. If present, it should be aspirated and taken for histology. If ascites is absent peritoneal washings with saline are obtained from the paracolic gutters, subhepatic space and the pelvis. The mass is cut and the inside noted whether it is solid or fluid filled. The fluid can either be mucinous, serous, gelatinous or haemorrhagic. The inside may be a single cyst or multiloculated. Optimal preparation for surgery including mechanical and antibiotic preparation of the bowel in the event that colon resection will be done is necessary. Prophylaxis against infection and venous thromboembolism may be beneficial. Nutritional support is also necessary.

Post operatively, our patient had chemotherapy with a combination of cisplatin, cyclophosphamide and adriamycin being used. Adjunctive chemotherapy or intraperitoneal chromium phosphate (32p) are used following surgery for epithelial tumours. Chemotherapy is indicated in all patients with ovarian cancer except in those with surgical-pathological stage I disease with low risk characteristics. Cisplatin based combination chemotherapy is administered. One common regimen includes cisplatin 50-100mg/m2 and cyclophosphamide 750-1000mg/m2 given every 3 weeks for 6-8 cycles. The potential toxicities for this regimen include alopecia, nephrotoxicity, ototoxicity and myelosuppression. Carboplatin is an analog of cisplatin that can be used on out patient basis. It is also minimally nephrotoxic. Currently most centers recommend combination therapy with platinum and paclitaxel (taxol)[8]. Results from randomized clinical trials suggest that in patients with optimally debulked disease, intraperitoneal administration of chemotherapy (cisplatin) is superior to intravenous administration[9]. Assessment of response to combination chemotherapy is based on physical examination, changes in size of the palpable mass or radiographically measurable lesions and changes in the CA-125 levels. Although the preoperative CA-125 level does not correlate with the tumour burden, changes in response to chemotherapy appears to be of some prognostic benefit. During therapy, patients are seen at least once a month. A full haemogram, hepatorenal profile and chest X-ray are obtained every month. A pelvic examination to assess disease status is done on a monthly basis. Patients who have completed therapy and who are free of disease are evaluated every 2-3 months for 2 years. Thereafter they are evaluated every six months. Our patient was still on follow up.

Most patients with ovarian cancer will have a recurrence. Based on the disease-free interval after completing chemotherapy, patients can be classified into two categories

(i) Platinum sensitive patients in whom relapse occurs more than 6 months after initial chemotherapy and

(ii) Platinum resistant patients

Patients with platinum-sensitive disease may exhibit a good response if rechallenged with a platinum agent. The probability of response increases with the duration of the disease-free interval. The several chemotherapy agents that elicit a response in platinum-resistant patients include liposomal doxorubicin, topotecan, oral etoposide, gemcitabine, docetaxel and vinorelbine. Other agents include ifosfamide, 5-fluorouracil with leucovorin, hexalen. Tamoxifen, an oral antioestrogen also exhibits modest activity but has a very favourable toxicity profile[8]. Our patient had not been noted to have any recurrence at the follow up visit.

The patient presented did not have a second look operation as this is not routinely done in our unit. Following the prescribed course of chemotherapy, "the second look" operation is useful to make judgement regarding the continuation of chemotherapy. Second look laparotomy is defined as re-exploration in patients with advanced stage III or IV ovarian cancer, in whom after standard course of chemotherapy, have no clinical, biochemical (CA-125), or radiological evidence of disease. The value of second look therefore is:

- To discontinue all chemotherapy if there is no evidence of disease.
- To determine the actual surgical and pathologic response to cisplatin based chemotherapy if cisplatin is to be used as part of second line chemotherapy
- If possible to deal with any residual disease to minimal or microscopic disease to achieve the same theoretic benefits described for primary debulking surgery.

Second look laparotomy is performed after 6-12 courses of chemotherapy. Our patient was started on combination chemotherapy.

Conservative therapy may be recommended for epithelial tumours of low malignant potential. Patients who desire preservation of fertility may be managed conservatively with unilateral salpingo-oophorectomy [1,3]. The uterus and contra lateral ovary may be preserved in women with stage IA, grade I disease who desire to preserve fertility. Prophylactic oophorectomy has been advocated in patients with family history of ovarian cancer or at the time of hysterectomy for non-ovarian indications. However, this remains controversial.

The prognosis for patients with ovarian cancer is primarily related to the stage of the disease at diagnosis. The five year survival rate for patients with stage I epithelial disease is approximately 80%, stage II 40-50%, stage III 30% and stage IV less than 10% [3,8].

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GYNAECOLOGY CASE 10

CHORIOCARCINOMA – CHEMOTHERAPY

NAME: M.N. L.M.P: 25/09/02

AGE: 30 Years Parity: 1 + 1

I.P NO: 0844127

D.O.A: 30/10/02
D.O.D: 17/02/03

PRESENTING COMPLAINT

She presented to KNH after being referred with persistently positive pregnancy test despite having had curettage.

HISTORY OF PRESENTING COMPLAINT

M.N. was referred from Chania maternity in Thika on 24/10/03 with a history of having undergone evacuation of molar pregnancy on 6/05/02. This was followed by sharp curettage three weeks later in Kitale. Follow up was not done as required and she presented five months later with a positive pregnancy test despite being sexually inactive since the mentioned evacuation of molar pregnancy. A pelvic scan done in early October at Thika showed a bulky uterus with multiple cystic areas within the endometrial cavity and myometrium. The posterior border of the uterus was bossed and irregular, features consistent with invasive mole or choriocarcinoma. She had been amenorrhoeic since the termination of the mole. She had no cough, and no abnormal bowel habits or abdominal pains. She had no headaches or convulsions.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

MN attained menarche at 16 years. Her parity was 1+1. Her last menstrual period was on 25/09/02. She had regular periods that lasted three days and occurred every 28 days. She gave no history of dysmenorrhoea. Her first pregnancy was in 1997 when she delivered a female baby who is alive and well. Her second pregnancy had ended in an abortion at 3 months in May 2002, manual vacuum aspiration was done. This was then followed by the history above. She had never used any family planning method and had never had a Pap smear.

PAST MEDICAL HISTORY

This was nil significant having had no prior hospital admissions and has no known allergies to foods or drugs.

FAMILY AND SOCIAL HISTORY

She was single and lived with her parents in Kitale. She did not work. She did not smoke cigarettes or take alcohol. Her father was diabetic. There was no family history of other chronic illnesses.

PHYSICAL EXAMINATION

She was in a good general condition, was not pale or jaundiced. Her blood pressure was 120/80mmHg, pulse 70/minute and regular, respiratory rate 18 breaths/minute and a temperature of 37°C.
Her central nervous system, cardiovascular and respiratory systems were essentially normal.

ABDOMINAL EXAMINATION

The abdomen was not distended. There was tenderness in the suprapubic region. The uterus was palpable at 12 weeks equivalent of a pregnant uterus. No other masses were palpable.

PELVIC EXAMINATION

She had normal external genitalia. On speculum examination, no lesions were noted on the vaginal mucosa or on the cervix. On digital examination, the cervix was closed, vaginal walls smooth. The uterus was bulky corresponding to a 12 weeks pregnant uterus. The adnexa was free, the pouch of Douglas was empty. There was no blood on the examining finger.

DIAGNOSIS

An impression of choriocarcinoma was made.

INVESTIGATIONS

1. Beta HCG - 58,751 miu/ml done on 28/10/02.
2. Pelvic ultrasound – bulky uterus with multiple cystic areas features suggestive of invasive mole to rule out choriocarcinoma. The liver, spleen and kidneys were normal.
3. Chest X-ray – Normal (26/10/02)
4. Full haemogram – Haemoglobin 13.2g/dl
   white cells count 8.9 x 10^9/l
   platelets 250 x 10^9/l
5. Blood group – A positive.
6. Urea and electrolytes – Na^+ 142mmol/l
   K^+ 4.5 mmol/l
   Creatininine 80umol/l

7. Liver function tests – Normal enzyme, protein and bilirubin levels.

MANAGEMENT

The diagnosis, mode of treatment, side effects of the drugs to be used and investigations during treatment and follow-up were explained to the patient and her relatives. She was admitted for chemotherapy.

She was scored as having low risk disease and was scheduled and commenced on methotrexate 50mg daily for five days with rescue leucovorin 15mg daily alternate days for five days. The dose of methotrexate was calculated using her body surface area from her weight and height.

This course of treatment was followed by a rest period of one week. At the end of the rest period, a repeat β-HCG, urea and electrolytes, liver function tests and full blood count were done. The next course of chemotherapy was given if all the test of renal, liver function and blood count were normal. She was also given haematinics during this period.

A summary of serial β-HCG levels before each course of chemotherapy was as shown below (reference range for normal 0-10 miu/ml);

28/10/02 - 58,751

30/11/02 - 42.14

7/01/03 - 7.24

01/02/03 - 2.31

08/03/03 - 2.39

04/04/03 - 2.0

14/05/03 - Less than 2

04/06/03 - Less than 2.

30/07/03 - Less than 2.

FOLLOW UP

MN received 2 more courses of methotrexate after the first negative β-HCG and was adviced on contraception to avoid conception during follow-up. She was adviced on combined oral contraceptives but chose abstinence as she was not married and had no sexual partner. She was adviced not to become pregnant for at least one year. For the six months that she came to the clinic consistently, the β-HCG levels remained negative.

DISCUSSION

The patient presented was a 30 years old para 1+1 who had choriocarcinoma following molar pregnancy. She was commenced on single agent chemotherapy and responded well to treatment.

Choriocarcinoma is part of a spectrum of gestational trophoblastic diseases (GTD) or tumours derived from the trophoblast following pregnancy. Others in the spectrum include hydatidiform mole, invasive mole and placental site trophoblastic tumour[1]. GTD'S are one of the few cancers that can be cured even in the presence of widespread metastasis. They have varying propensities for local invasion and metastasis. They also have properties of the placenta like invasion and elaboration of human chorionic gonadotrophin (HCG). Hydatidiform mole presents the benign end while choriocarinoma represents the malignant end of the spectrum of GTD's [1-4].

Our patient had hydatidiform mole preceding choriocarcinoma. In about half the cases of choriocarcinoma the antecedent gestational event is hydatidiform mole, one fourth follow term pregnancy and the remainder follows other forms of gestation (abortion, ectopic pregnancy) [1,2]. The incidence of trophoblastic disease is reported to be highest in S.E Asia with Taiwan reporting an incidence of 1:82 pregnancies. In Europe and North America, the reported incidence is between 1:1500 and 1:2500 pregnancies[1,2]. The actual incidence in Kenya is not known. In a study at the Kenyatta National Hospital an incidence of 1:1118 deliveries was found[5,6].

The patient presented was 30 years old, her blood group was A positive, she was from a low socio-economic background and her disease followed a molar pregnancy. The exact aetiology of trophoblastic disease is unknown. Several theories have been proposed about possible aetiologic factors but none of these have been proven. The incidence is higher in women under 20 and over 40 years. The incidence is also higher with nulliparity, multiparity, low socio-economic status, Blood group A or AB women married to a husband of blood group O, those whose diet is deficient in proteins, folic acid and carotene and women who have previously had molar pregnancy[1, 2,7].

Our patient had only local disease with no metastasis but karyotyping of the mole was not done. GTD arises from foetal tissue and is associated with 46 XX karyotypes, trisomic and triploid chromosomes that are paternal in origin. Histologically choriocarcinoma is composed entirely of chorionic epithelium, which is avillous, necrotic and haemorrhagic. It is characterized by sheets of anaplastic syncytiotrophoblast and cytotrophoblast without any chorionic villi. In choriocarcinoma, the predisposition of normal trophoblast to invasive growth and erosion of blood vessels is greatly exaggerated. The tumour is dark red or purple and rugged or friable[2,8]. The tumour invades the myometrium and metastasizes easily. The common sites are the lungs (80%), vagina (30%), pelvis (20%), liver (10%) and the brain (10%). The high incidence of lung metastases may be due to pulmonary deportation of the normal trophoblasts that undergo neoplastic changes in situ. Metastasis to the lungs has the best prognosis among the distant metastatic sites.

In our patient diagnosis was made from elevated HCG and pelvic ultrasound. The leading symptom in GTD's is irregular uterine bleeding after expulsion of the foetus, mole or normal pregnancy. Later, there may be an offensive discharge, pyrexia and cachexia [9]. Theca lutein cysts are identified in over one third of the cases. Some present with features of metastasis. Physical signs are evident according to the organ involved. Most patients may look ill, because vaginal bleeding may be considerable and prolonged. Many of the patients may have anaemia[2, 9]. The definitive diagnosis of choriocarcinoma is based on the histological examination of the tissue involved. While uterine curettage may be useful in the diagnosis, a negative curettage does not exclude its presence, as the tumour is often myometrial[6]. Diagnosis is also based on clinical presentation and the demonstration of elevated levels of HCG. Other useful diagnostic tests include pelvic ultrasound.

After diagnosis of GTD, various investigations will help in determining further management. These include β-HCG, blood group and rhesus factor, full blood count, renal function, radiographic evaluation including chest x-ray, CT scan of the pelvis, abdominal ultrasound, brain CT scan or MRI [1,2,9]. In our patient, CT and MRI scans were not done due to being costly for the patient.
Trophoblastic disease may be divided into metastatic and non-metastatic disease, which may further be divided into poor or good prognosis disease. The International Federation of Gynaecology and Obstetrics (FIGO) has adopted an anatomic staging system for GTD as below [2]:
Our patient had stage IC disease (her disease was confined to the uterus and had two risk factors- beta HCG above 40,000 and duration of five months after evacuation of molar pregnancy). The staging is as follows:

Stage I: Disease confined to the uterus
- IA Disease confined to the uterus with no risk factors
- IB Disease confined to the uterus with one risk factor
- IC Disease confined to the uterus with two risk factors

Stage II: Disease extending outside the uterus but limited to genital structures (adnexa, vagina, broad ligament)
- IIA Disease involving genital structures without risk factors
- IIB Disease extending outside uterus but limited to genital structures with one risk factor
- IIC Disease extending outside uterus but limited to genital structures with 2 risk Factors

Stage III: Disease extending to the lungs with/without known genital tract involvement
- IIIA Disease extending to the lungs with/without known genital tract involvement and no risk factor.
- IIIB Disease extends to the lungs with /without known genital tract involvement and with one risk factor
- IIIC Disease extends to the lungs with/without known genital tract involvement and with 2 risk factors.

Stage IV: All other metastasis sites
IVA All other metastasis sites without risk factors
IVB All other metastasis sites with one risk factor
IVC All other metastasis sites with two risk factors

The risk factors affecting the staging include;

-Serum β-HCG above 40,000miu/ml
- Duration of the disease longer than four months from termination of antecedent pregnancy.

In addition to the anatomic staging, it is important to consider other variables to predict the likelihood of drug resistance and to assist in selecting appropriate chemotherapy. A prognostic scoring system has been proposed by the World Health Organisation and reliably predicts the potential for resistance to chemotherapy[2]. It takes into account the following factors;

Factor/score	**0**	**1**	**2**	**4**
Age (years)	Less than 39	Above 39		
Antecedent pregnancy	Hydatidiform mole	Abortion	Term	
Interval between antecedent pregnancy and start of chemotherapy	Less than 4	4-6	7-12	Above 12
β-HCG (Miu/l)- Pre-treatment levels	Less than 10^3	10^3-10^4	10^4-10^5	Above 10^5
ABO group (female x male)		O x A, A x O	B or AB	
Largest tumour including uterine (cm)	Less than 3	3-5	More than 5	
Sites of metastasis		Spleen/Kidney	GIT/Liver	Brain
Number of metastasis		1-3	4-8	More than 8
Prior chemotherapy			Single drug	More than or 2

Interpretation: total score - less than or 4=low risk,
- 5-7 =middle risk
- more than 8= high risk.

Our patient scored 4 hence was low risk.

Our patient was started on single agent methotrexate as chemotherapy. Chemotherapy remains the mainstay of treatment for choriocarcinoma. Patients for chemotherapy need evaluation and investigations with emphasis on possible metastasis. These were done in our patient. In addition, lumbar puncture with CSF: Blood β-HCG ratio less than 1:60 is significant and indicates brain metastasis. Evaluation should also identify high risk factors as outlined in the prognostic scoring system above. CSF puncture was not done in our patient as she was having local disease in the uterus.
A protocol for the management of choriocarcinoma is presented here[2]:

Stage I

Initial: single agent chemotherapy or hysterectomy with adjunctive chemotherapy

Resistant: combination chemotherapy or hysterectomy with adjunctive chemotherapy

Stage II and III

Low risk: Initial –single agent chemotherapy
Resistant- combination chemotherapy

High risk: Initial-combination chemotherapy
Resistant- second line combination therapy.

Stage IV

Initial: combination chemotherapy
Brain: whole head irradiation (300cGy), craniotomy to manage complications
Liver: resection to manage complications
Resistant: Second line combination chemotherapy, hepatic arterial infusion.

Single agent chemotherapy with either actinomycin D (0.5mg daily for 5 days) or methotrexate (0.4mg.kg daily for 5 days) achieves comparable and excellent remission rates in low risk disease. The administration of folinic acid limits toxicity of methotrexate, which is mainly bone marrow suppression and erosion of the mucous membranes[10]. Cyclophosphamide causes marrow depression, alopecia and haemorrhagic cystitis. Treatment is given every 7-10 days till remission occurs as tumour relapse and re-growth is significant if there is treatment lapse of more than 2 weeks[2,9,10]. Remission is said to have occurred if 3 consecutive β-HCG levels are negative. On average, 3-4 courses are required for remission and a further 2-4 courses are given to avoid relapse (at least 1 course for low risk and 3 courses for high risk disease) [2,10]. In his study at KNH Fongoh reported that most patients had negative tests after the third course and that those with disease following molar pregnancy had remission rates of 92.9%. Patients on treatment require monitoring with weekly β-HCG until remission and weekly full blood counts (Hb above 10g/dl, white cell count above 2000/ul and platelets above 150/ul), urea and electrolytes, liver function tests before every treatment course. Treatment is withheld if toxicity is noted. Treatment is changed if HCG titres rise or plateau at high levels or if metastases appear.

Following remission, patients are put on a surveillance programme. HCG is assayed monthly for one year, then every 2 months for another year and six monthly indefinitely [8,10]. During the period of surveillance, effective contraceptive use is mandatory for a minimum period of one year. The combined oral contraceptive pill is recommended. It causes regular and predictable vaginal bleeding for which disease relapse cannot be confused. It also suppresses the LH surge so that only the pituitary levels of LH are detectable and thus no interference with β-HCG assays. The pill should be started after HCG titres are negative because trophoblast is destroyed more slowly in patients on steroids [2,9,10]. The patient must avoid pregnancy for at least 12 months following completion of treatment. This period allows full metabolism and excretion of mature ova affected by chemotherapy as cytotoxic drugs are teratogenic. It also allows monitoring for relapse following remission. Following this, many women treated with chemotherapy are able to have pregnancy with outcomes similar to that of the normal population [8,10,1].

Patients with high-risk disease require use of more than one cytotoxic. Methotrexate-0.4mg/kg, actinomycin D-0.5mg, and cyclophosphamide-3mg/kg (MAC) all daily for 5 days is the most commonly used regime in our unit. Monitoring and follow up is the same as that of patients with low risk disease. Our patient was on single agent chemotherapy as she had low risk disease. The EMACO (etoposide, methotrexate, actinomycin-D, cyclophosphamide and vincristine) regimen is now preferred as primary treatment of in-patients with metastasis and high-risk prognostic score. If the patient does not wish to preserve fertility, hysterectomy with adjuvant chemotherapy may be performed as primary treatment [2,9,10,12].

The role of surgery is limited in the management of choriocarcinoma. Surgical excision of an isolated metastatic lesion in the abdomen, lungs or brain may be considered in a patient with resistant disease. Indications for hysterectomy include;

- Drug resistance or toxicity especially in cases where the disease is confined to the uterus.
- Complications such as vaginal haemorrhage, uterine perforation with intraperitoneal bleeding.
- In patients with extensive disease to reduce tumour burden and thereby limit the need for multiple courses of chemotherapy [1].

Radiation therapy may be useful in cerebral metastasis where whole brain irradiation is done. Intrathecal injection of methotrexate has also been tried for those with nervous system metastases. Our patient did not develop any metastasis.

Prognosis is good with 100% remission for non-metastatic tumour. Metastatic low risk tumours have 87-100% remission rates while metastatic high-risk tumours have 70-80% remission rates[9, 10]. Cure is said to have occurred after a period of 5 years in remission. Monitoring in subsequent pregnancies is necessary as there is a small risk of recurrent GTD of 1-2%[1].

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GYNAECOLOGY CASE 11

UTERINE FIBROIDS – MYOMECTOMY

NAME: P.W.C. Parity: 0+0

AGE: 30 Years

I.P NO. 0816387

D.O.A. 4/09/02

D.O.D. 17/09/02

PRESENTING COMPLAINTS

PWC presented with a 5-month history of low abdominal pain, heaviness of lower abdomen and backache.

HISTORY OF PRESENTING COMPLAINTS

She was well until 5 months prior to admission when she started having backache and lower abdominal pain. This was associated with a feeling of heaviness in the lower abdomen. This became worse after strenuous work or standing for a long time. She also noted that her lower abdomen was swelling. She had no change in her menstrual flow. She had occasionally had episodes of vaginal discharge. She also noted frequency of micturition and urgency, which was relieved by emptying the bladder. Her bowel habits had not changed.

OBSTETRIC AND GYNAECOLOGY HISTORY

She was nulliparous. Her menarche was at 15 years with regular cycles occurring every 28 days with flow for 3-4 days. She had associated mild dysmenorrhoea. Her last menstrual period was on 29.08.02. She had not used any contraceptives previously and had not had any Pap smear done.

PAST MEDICAL HISTORY

She had never been admitted previously and had no known allergies to foods or drugs.

FAMILY AND SOCIAL HISTORY

She was a single lady working as a teacher. She lived in Githurai. She did not use alcohol or smoke cigarettes. She gave no family history of similar illness or any chronic illnesses.

PHYSICAL EXAMINATION

She was in a good general condition, was not pale, and had neither jaundice nor oedema. Her blood pressure was 120/70mmHg, pulse 80 beats/minute and regular, respiratory rate 17 breaths/minute and temperature of 37°C.
Her cardiovascular, respiratory and nervous systems were essentially normal.

ABDOMINAL EXAMINATION

Her abdomen was distended in the hypogastric region and moved with respiration. She had no scars. On palpation a mass was found arising from the pelvis corresponding to a 24 weeks pregnant uterus. The mass was firm, mobile, non-tender, had an irregular surface. The liver and spleen were not enlarged and the bowel sounds were normal.

PELVIC EXAMINATION

She had normal external genitalia. The vaginal mucosa was normal. The cervix was closed, posterior and firm with a smooth surface. The uterus was enlarged, mobile and the mass moved with the cervix. Cervical excitation was negative in both adnexa and the pouch of Douglas was free. There was no vaginal discharge.

DIAGNOSIS

A diagnosis of large symptomatic uterine fibroids was made.

INVESTIGATIONS

1. Haemogram – Haemoglobin 16.0g/dl
   white cells count 8.5 x 109/l
   platelets 210 x 109/l

2. Urea and electrolytes – Na+ 142 mmol/l
   K+ 3.8 mmol/l
   Creatininine 90 umol/l
   Urea 6.0 mmol/l

3. Pap smear – satisfactory smear, endocervical cells seen and no abnormal cells noted.

4. Pelvic ultrasound – reported a bulky uterus 112mm in diameter with multilobulated intrauterine mass consistent with uterine fibroids.

5. Hysterosalpingogram – multiple filling defects probable submucosal fibroids.

6. Blood group and cross matching. Her blood group was B positive. Two units of blood were requested for.

MANAGEMENT

PWC was informed of the diagnosis and the various modes of management. Because of being nulliparous and planning on getting married, myomectomy was chosen as the procedure of choice. She gave informed consent for this operation including total hysterectomy in case it became necessary at the time of operation. She then autodonated 1 unit of blood. She was starved from midnight on the night before operation. On the morning of surgery, she was premedicated with atropine 0.6mg and pethidine 50mg intramuscularly half hour before being taken to theatre. Abdominal wall and pubic hair was shaven.

In theatre and under general anaesthesia, she was placed in the semilithotomy position for vulvo vaginal toilet, aseptic catheterization and examination under anaesthesia. The catheter was left in situ to maintain continuous bladder drainage during the operation. Pelvic examination confirmed the earlier findings. The abdomen was then cleaned and draped with sterile towels. The abdominal wall was then opened in layers via a midline incision. Inspection revealed an enlarged uterus with multiple fibroids, some pedunculated while others subserosal mainly in the fundal region anteriorly. Both ovaries and fallopian tubes were normal and healthy. The rest of the abdomen and pelvis were normal. A self-retaining retractor was placed to expose the pelvis and gut packed away with warm moist packs. The uterus was delivered out of the abdomen, the size and position of the fibroids noted. A decision was made to continue with myomectomy as planned pre-operatively. A uterine clamp was applied in the lower uterine region to minimize bleeding. Incision was made in the anterior uterine wall over the fibroid and enucleation from the false capsule done. The process of myomectomy was done for other fibroids through the same incision. The largest fibroid measured 15cm in diameter. The pedunculated fibroids were also removed. The bed that remained after the removal of the fibroids was obliterated using a series of mattress sutures until haemostasis was achieved. The serosa was then closed using interrupted sutures with catgut. The uterine cavity was not entered. The uterine clamp was then released and haemostasis was confirmed to be satisfactory. The whole procedure took about half an hour. The abdomen was then cleaned and its wall closed in layers after ascertaining that instruments and swab counts were correct as at the beginning of the operation. She was then reversed from anaesthesia and taken to the recovery room.

POST OPERATIVE MANAGEMENT AND FOLLOW UP

Vital signs were observed until she was fully awake. Attention was paid to the pulse and blood pressure to note any hypovolaemic shock from bleeding. She also had no abdominal distension or pallor clinically. She was given intravenous crystalline penicillin and gentamicin as prophylactic antibiotics, pethidine (100mg three times daily) By the second day she was started on oral fluids and eventually free diet. On the third day she was started on oral antibiotics (Amoxicillin/clavulanate 625 mg twice daily) and diclofenac for pain. Her wound was exposed and found to be healing well. On the fourth day she was well, had no complaints and was discharged home. She was for review in the gynaecology outpatient clinic in four weeks time. At review she was well, had no complaints and the wound was well healed. Her histology result confirmed the presence of fibroids with their characteristic whorled pattern. She was adviced on close follow up during pregnancy.

DISCUSSION

A case is presented of a 30 years old nulliparous patient with low abdominal pain found to be due to uterine fibroids. She had myomectomy done and was discharged home in stable state and was well at the follow up visit.

Uterine fibroids are benign proliferations of smooth muscle cells and fibrous connective tissue arising from uterine smooth muscle. They may be single or multiple, may range in size from 1mm to over 20cm in diameter and are surrounded by a pseudocapsule of compressed muscle fibres [1]. They are also known by various names including leiomyoma uteri, fibromyoma, myofibroma, myoma and leiomyofibroma. The term leiomyoma is preferred as it emphasizes the origin of the tumour from smooth muscle cells and the predominance of the smooth muscle component. Fibroids are the most common tumours of the uterus and female pelvis. They have been estimated to be clinically apparent in 25% of reproductive age women. Careful evaluation of pathological specimens suggest that prevalence is as high as 77%[2]. The incidence at Kenyatta national hospital was found to be 66.7% with mean age occurrence ranging between 35-45 years[3]. There appears to be a racial distribution with fibroids being 3-9 times more common in Negroid than Caucasian women. This patient was 30 years old. Factors behind this racial distribution are not known.

No factor could be attributed as the cause of fibroids in our patient. The aetiology of fibroids is undetermined but their growth is influenced by oestrogen. They are noted to grow faster in pregnancy and regress in menopause. They are not detectable before puberty [5,7]. Continuous oestrogen stimulation is thought to be a risk factor in their development. The relative risk reduces to one fifth with five term pregnancies compared to nulliparous women. Cigarette smokers have 18% reduced risk compared to non-smokers. Obesity increases risk with 21% for every 10kg gain above appropriate weight for height. This patient did not smoke and was not obese. The prevailing hypothesis is that fibroids arise from a single neoplastic cell but the initial neoplastic transformation stimuli are not known though the following are implicated [2,5,6,7,8]:

- Chromosomal abnormality – found in about 37% of cases. Involves translocations and deletions in several chromosomes but mostly chromosome 12.
- The role of polypeptide growth factors- epidermal growth factors, insulin-like growth factor-1, and transforming growth factor has been suggested to stimulate the growth of leiomyoma either directly or via oestrogen.
- Oestrogens are implicated in the growth of fibroids. Fibroids have been shown to have oestrogen receptors in higher concentrations than the surrounding myometrium. They may contribute to tumour enlargement by increasing the production of extracellular matrix. Myomas usually decrease in size after menopause.
- Progesterone increases mitotic activity of myomas but the mechanisms and growth factors have not been characterized yet. Progesterone may also allow for tumour enlargement by down regulating apoptosis in the tumour.
- Other factors thought to cause myomas are mechanical stress, pelvic inflammatory disease, prolactin and infertility.

Fibroids originate from the smooth muscle cells of the myometrium and can be classified by anatomic location into three main groups. Submucous myomas lie just beneath the endometrium and tend to compress it as they grow towards the uterine lumen. These may develop pedicles and protrude into the uterine cavity or even prolapse through the cervical canal. Intramural or interstitial myomas lie within the uterine wall, giving it a variable consistency. Subserous or sub peritoneal myomas lie just at the serosal surface of the uterus or may bulge outward from the myometrium. These may become pedunculated and some may acquire extrauterine blood supply from omental vessels and thus become parasitic. Fibroids are usually multiple, discrete and either spherical or irregularly lobulated. The have a false capsular covering, and they are clearly demarcated from the surrounding myometrium, thus can be enucleated easily. More common sites include the uterine body and fundus than the uterine cervix. They are usually firm, whorled and grossly, have a lighter colour than the surrounding myometrium. Our patient had fibroids in the body and fundal region.

No degenerative changes were seen in our patient's fibroids at operation and after histology. Their blood supply enters from the periphery of the tumour and they are relatively avascular. As the tumour grows and becomes larger, fibrous tissue becomes more abundant than the smooth muscle and degenerative changes may be noted. These changes may create a different histological picture from the original tumour. Benign degeneration may be of the following types [2,7,8,9]:

- Atrophic-with signs and symptoms regressing as tumour size decreases
- Hyaline- the tumour contains soft, gelatinous areas.
- Cystic – liquefaction following extreme hyalinization results in cystic change.
- Calcific (calcareous)- due to precipitation of calcium carbonate and phosphate within the tumour.
- Septic- circulatory inadequacy may cause central necrosis of the tumour followed by infection.
- Myxomatous (fatty) usually uncommon and asymptomatic following cystic and hyaline changes.
- Carneous (red) degeneration- is due to haemorrhage after venous thrombosis and congestion with subsequent interstitial haemorrhage. Is more common during pregnancy and is accompanied by pain.

Malignant change (leiomyosarcoma) is rare and reported as occurring in 0.1-0.5% of fibroids.

The patient presented was admitted with low abdominal pain and backache. The majority of fibroids are asymptomatic. Symptoms are present in 35-50% of patients with fibroids. Symptoms from fibroids depend on their location, size, state of preservation and whether the patient is pregnant or not. The symptoms can be grouped into the following categories [2,7,8,9]:

- Abnormal uterine bleeding – is the most common and most important clinical manifestation of leiomyomas, being present in up to 30% of patients. Bleeding from submucous myoma may occur from interruption of the blood supply to the endometrium, distortion and congestion of the surrounding vessels, or ulceration of the overlying endometrium. Mostly the patient has menorrhagia (prolonged

heavy menstruation) or minor degrees of metrorrhagia though this should be investigated to rule out endometrial disease.

- Pelvic pain – occurs when vascular compromise is present. It may result from degeneration associated with vascular occlusion, infection, torsion of a pedunculated tumour or myometrial contractions to expel a subserous myoma from the uterine cavity. Acute pain associated with low-grade fever, localized uterine tenderness and leucocytosis may be observed with red degeneration in pregnancy (usually second trimester) or rarely with pedunculation. Both of these are self-limiting and can be managed conservatively with rest, sedation and analgesics. Large tumours may produce a sensation of heaviness in the pelvic area, a feeling of a mass in the pelvis or mass palpable through the abdominal wall. Tumours becoming impacted in the pelvis may press on nerves and create pain radiating to the back or lower extremities. Dyspareunia may result depending on the site of the tumour and the pressure they exert on the vaginal walls.
- Pressure effects- may result when the uterus size is increased. The size of the myomatous uterus is described in menstrual weeks as in the pregnant uterus. The uterus may be irregularly shaped and specific symptoms will depend on the location of the myomas. Pressure effects may lead to urinary frequency, urgency, and hydroureters. Other pressure effects include constipation, pelvic congestion from impaction, serosanguinous vaginal discharge from cervical tumour, lower extremity oedema or exercabation of varicose veins and haemorrhoids. Parasitic tumours may cause intestinal obstruction if they are large and or involve omentum or bowel.
- Reproductive symptoms – a variety of reproductive disorders including infertility, recurrent spontaneous abortion and preterm labour have been attributed to uterine fibroids. But there is as yet no conclusive data to support a causal association between uterine fibroids and adverse reproductive outcomes. Infertility is thought to be due to cornual obstruction, intermittent anovulation, abnormal uterine or tubal motility or interference with sperm transport or abnormal vascularisation preventing normal implantation. Infertility affects about 2-10% of the patients. Abortion is 2-3 times more common in pregnant women with fibroids.
- Leiomyomas during pregnancy- during the second and third trimesters of pregnancy, myomas may rapidly increase in size and undergo vascular deprivation and subsequent degeneration leading to pain and local tenderness. Rest, analgesics (narcotic analgesics) alleviate the pain. Tocolytics may sometimes be necessary to control the uterine contractions. During labour they may produce uterine inertia, foetal malpresentation or obstruction of the birth canal necessitating caesarean delivery. Postpartum haemorrhage should be anticipated from ineffective uterine contraction after delivery.

Complications of uterine fibroids include severe haemorrhage causing iron deficiency anaemia or hypovolaemic shock. Other complications include uterine inversion, infarction and necrosis causing pain, ulceration and infections. Pedunculated fibroids may undergo torsion to cause acute abdomen. Ascites has been reported as a consequence of torsion of a pedunculated subserosal or parasitic fibroid. Polycythemia associated with fibroids is believed to be due to autonomous erythropoietin production by some of the myoma cells or due to pressure on the kidneys causing excess release of erythropoietin.

kidneys causing excess release of erythropoietin. Distant benign (commonly pulmonary) metastases aptly named benign metastasizing leiomyomas have been reported rarely in women with fibroids. Similarly the rare diffuse leiomyomatous nodules involving visceral and peritoneal surfaces termed leiomyoma peritonalis disseminata have been described. Whether these conditions represent metastasis of uterine tumours or primary neoplasm is unknown.
The diagnosis of leiomyoma is usually made clinically and confirmed by ultrasonography Leiomyoma are suspected on the basis of a bimanual examination that reveals an enlarged, firm, non-tender, and irregularly contoured uterus. Other investigations that confirm or corroborate the main diagnosis include hysterosalpingogram (HSG), intravenous urography (IVU), computed tomography (CT) scan, MRI scan. Other tests are done to fully work up the patient for appropriate management and exclude malignancies e.g. Papanicolaou smear, endometrial biopsy, full blood count, renal function tests and liver function tests. In our patient the diagnosis was made clinically and she also had ultrasound and Pap smear done.
Our patient was managed by myomectomy as she still desired fertility. Uterine myomas may be treated using surgical as well as nonsurgical modalities. Nonsurgical methods include expectant management and medical management. Surgical methods include abdominal or hysteroscopic myomectomy or hysterectomy. The choice of treatment is dependent on the size and location of the myoma, the presenting symptoms, her age, parity, pregnancy status, general health, reproductive desires of the patient and sometimes the surgeons experience.
Expectant management is used for asymptomatic fibroids. It is mostly for the small fibroids (less than 12 weeks pregnant uterus size) and follow up is done by physical examination regularly to assess growth or regression.

Surgery can either be conservative (myomectomy, avulsion of pedunculated fibroid) or radical (hysterectomy). Surgery is indicated for large myomas greater than 12 weeks, complicated fibroids e.g. torsion, obstruction or degenerative changes, severe symptomatology such as severe menorrhagia leading to anaemia, severe pain.

Myomectomy as for our patient is done when there is desire for preservation of fertility and sometimes the patients wish to retain her uterus. Myomectomy is done for relatively small tumours and can be done either laparoscopically or abdominally. But one can never be certain before operation that myomectomy can be accomplished easily thus the patient should be informed of possible hysterectomy in case of excessive intraoperative blood loss or postoperative haemorrhage. Myomectomy is quite successful for control of chronic bleeding associated with leiomyoma. Incisions should be as few as possible and usually on the anterior uterus. Posterior incisions may have difficulty in reaching the bed left after removal of the fibroid. As many as possible fibroids should be enucleated through one incision. Haemostasis during the procedure is achieved by use of Bonneys myomectomy clamp and adequate obliteration of the cavity left after removal of the fibroids. The clamp should not be left in place for more than 30 minutes at once as this has been associated with later postoperative shock due to release of histamine like substances

from the uterus into the general circulation thus less substance is released with periodic release of the clamp. Myomectomy has the disadvantage of recurrence of fibroid growth, with rates as high as 15% reported. Other complications are haemorrhage necessitating hysterectomy and thus the patient losing her chance of a pregnancy, menorrhagia persisting if submucous fibroids are missed at myomectomy. The major concern for pregnancy after myomectomy is that the uterus will not rupture during the pregnancy or during labour. It is a prudent measure where the myomectomy has been extensive and the uterine cavity opened to have delivery by elective caesarean section. Myomectomy is contraindicated in pregnancy because it is associated with severe haemorrhage and induction of premature labour or abortion [7,9]. For women who have completed childbearing, and for whom bleeding is the primary problem and decline hysterectomy, endometrial ablation alone or in combination with hysteroscopic myomectomy will give relief. Our patient had myomectomy as she still desired fertility and she did not develop any complications. Laparoscopic myomectomy is still investigational.

Hysterectomy is the definitive treatment of choice for symptomatic uterine fibroids where the woman has completed childbearing. It eliminates both the symptoms and the chance of recurrence. Indications for hysterectomy include asymptomatic fibroids of such size that that they are palpable abdominally and are of concern to the patient, excessive uterine bleeding evidenced by either anemia, profuse bleeding in clots in repetitive periods, pelvic discomfort caused by myoma, bladder pressure with urinary frequency. The absence of cervical, endometrial malignancy is necessary before hysterectomy is done. Total vaginal hysterectomy is done for small fibroids associated with other pathology such as pelvic relaxation where repair of cystocoele, rectocoele and enterocoele is done at the same time. Total abdominal hysterectomy is done for big fibroids and when the mobility of the fibroids/uterus is limited. If the ovaries are diseased or if their blood supply is destroyed, oophorectomy is necessary. Otherwise the ovaries should be preserved. Complications of hysterectomy include haemorrhage, bladder and bowel injuries as well as injury to the ureters and postoperative sepsis.
Myolysis can also be done. Uterine artery embolisation is a new technique where embolic occlusion of the uterine arteries may successfully treat symptoms of leiomyomas, particularly menorrhagia. This is still under study.

Medical therapy involves symptomatic treatment with haematinics, analgesics and gonadotrophin releasing hormone (GnRH) agonists, progestagens, antioestrogens and antiprogesterones. Menorrhagia can be treated by use of nonsteroidal anti-inflammatory drugs that inhibit prostaglandin synthesis. Therapy is administered on a scheduled basis for the patients with menorrhagia. GnRH agonists which form the mainstay of medical therapy for uterine fibroids act by binding to GnRH receptors resulting in biphasic response, first increasing the levels of gonadotrophins temporarily, followed shortly by desensitization and down regulation resulting in reduction of the levels of gonadotrophins and gonadal steroid secretion within 1-3 weeks. This leads to a hypogonadotrophic hypogonadal state clinically resembling menopause or medical menopausal state [12,13]. The end result is

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treatment uterine size/volume. In addition, the severe hypo-oestrogenism that accompanies this GnRH agonist therapy cause significant bone loss that can lead to osteoporosis with time. Thus this therapy is used as a temporary measure to allow a woman prepare for surgery, await imminent onset of menopause, decrease the size and vascularity of the tumour making surgery easier and minimize blood loss. In our unit most patients do not afford it due to its prohibitive costs.

Low dose oral contraceptives and progestins may also be effective in the treatment of menorrhagia. New innovations to manipulate the actions of oestrogen and progesterone are being tried such as use of GnRH antagonists that provide a rapid onset of action [14].

The major steroid studied is Tibolone, which has oestrogenic, androgenic and progesterone activities. Tibolone was associated with higher rates of amenorrhoea in postmenopausal women with myomas than was conventional hormone replacement therapy. In premenopausal women with myomas treated with tibolone after GnRH down regulation, there was preserved bone density and lipid profiles without reversal of uterine shrinkage[14].

Steroid antagonists are another way to manipulate steroid hormone concentration. Mifepristone (RU486), which is a progesterone antagonist used for 3 months, decreases the volume of myomas by average of 49% and induction of amenorrhoea. The immunoreactivity of the progesterone and not oestrogen receptors in the myoma and myometrial tissue was decreased significantly by mifepristone treatment suggesting that regression of these tumours may be attained through direct antiprogesterone effect[15].

Anti-oestrogen therapy has also been tried with some success especially use of Ethinyl-nor-testosterone which also possesses some antiprogesterone properties [8,15].

On trial also is myolysis using N.YAG laser or bipolar needles, which penetrate myomas at multiple sites at right angle to the uterus[16]. This causes the myomas to eventually atrophy. When used with prior GnRH analogues, 30-50% reduction in size can be achieved. The main drawback with this method is damage to the uterine serosa by the process.

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GYNAECOLOGY CASE 12

PRIMARY INFERTILITY – LAPARASCOPIC TUBOPLASTY

NAME: J.M.M Parity: 0 + 0

AGE: 32 Years L.M.P: 11/9/04

I.P NO: 0973773

D.O.A: 21/09/04
D.O.D: 25/09/04

PRESENTING COMPLAINT

She was admitted with complaints of inability to conceive for ten years.

HISTORY OF PRESENTING COMPLAINT

JMM presented to the gynaecological outpatient clinic with history of being unable to conceive for ten years despite having normal regular coitus with her spouse; at least three times in a week of penetrative coitus. She had no past history of sexually transmitted infections, treatment for vaginal discharge or chronic low abdominal pain. She had regular menstrual periods and had no galactorrhoea. Her spouse had three children from a previous marriage.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She had her menarche at 13 years. Her parity was 0+0. Her last menstrual period was on 11/9/04. Her menses were regular occurring every 28 days and lasting 3 days with no dysmenorrhoea. She had never used any form of contraceptives.

PAST MEDICAL HISTORY

She had no prior admissions, no chronic illnesses or allergies.

FAMILY AND SOCIAL HISTORY

She was married and lived with her husband in Machakos. Both were small-scale business people. They neither smoked nor used alcohol. She had no family history of chronic illnesses.

PHYSICAL EXAMINATION

She was in a good general condition, was not pale, and had no jaundice or oedema. Her vital signs were normal with a blood pressure of 120/80mmHg, pulse of 72 beats/minute, respiratory rate of 18-breaths/ minute and a temperature of 36.8^{o} C.

She had normal findings in the central nervous, cardiovascular and respiratory systems. She had no goitre. She had normal female hair distribution and her breasts were well developed at Tanner 5 with no nipple discharge.

ABDOMINAL EXAMINATION

The abdomen was not distended, had no surgical scars. She had no palpable masses or areas of tenderness.

PELVIC EXAMINATION

She had normal external female genitalia, normal female pubic hair distribution. The cervix was closed, long and posterior. Her uterus was normal size, anteverted and freely mobile. Her adnexa were free and had no tenderness. The cervical motion test was negative. No discharge was noted on the examining fingers.

INVESTIGATIONS

1. Semenalysis - Revealed normozoospermia

2. Hysterosalpingogram – showed a normal uterine cavity. The left fallopian tube was blocked at the fimbrial end and the right had terminal hydrosalpinx with no dye spill bilaterally.

3. Pelvic scan - A normal anteverted uterus measuring 6.2 by 3.6 cm. The endometrium and myometrium were normal. The ovaries were normal and no free fluid in the pouch of Douglas.

4. Haemogram - Haemoblobin 13.2g/dl
white cells count 6 $x10^9$/l

5. Urea and electrolytes - Na^+ 144mmol/l
K^+ 4.3mmol/l
Urea 5.0mmol/l
Creatinine 76umol/l

6. Pap smear within normal limits; had no abnormal cells.

7. Elisa for HIV - Negative

DIAGNOSIS

Primary infertility due to bilateral tubal blockage.

MANAGEMENT

JMM was counseled and prepared for laparascopic surgery-diagnostic and therapeutic. On the night before surgery she was starved from midnight. On the morning of surgery she was premedicated with atropine 0.6mg and pethidine 50mg half hour before start of surgery. In theatre anaesthesia was induced and hysteroscopy was first done. Both fimbrial ostia were visualized and found normal. No tubal activity was noted and the tubal mucosa was noted to be flat and smooth. The cervix was held with a tenaculum and the dye canula introduced through the cervix. After cleaning and draping of the abdomen, a periumbilical stab wound was made and the veress needle pushed into the abdomen. Carbon dioxide gas was introduced up to to2.5 litres to create a pneumoperitoneum. The trochar was then introduced and the fibreoptic camera fixed in place. Two incisions were made in both iliac fossae for introducing the other instruments. Laparascopy done showed both tubes blocked with peri-tubal adhesions. Adhesiolysis was done and cuff salpingostomy was done bilaterally. Free spill was noted after the procedure bilaterally. Haemostasis was achieved and the pelvis cleaned with heparin in normal saline. The wounds were then closed. Anaesthesia was reversed successfully and her postoperative period was uneventful.

POSTOPERATIVE MANAGEMENT

She did well postoperatively and was discharged on the second day on analgesics and antibiotics for review in the gynaecological out patient clinic.

FOLLOW-UP

She returned to the clinic after four weeks. At the time she had no complaints and was well. She has since then come to the clinic twice lastly in March 2005 and had not conceived then.

DISCUSSION

The case presented is of a 32-year-old nulliparous patient with primary infertility due to bilateral tubal blockage and peritubal adhesions. She was managed by laparoscopic tuboplasty with adhesiolysis and patency achieved in both tubes. She had not conceived at the time of this write-up.

The patient presented had primary infertility. Infertility is failure to achieve a pregnancy after one year of regular and normal unprotected intercourse without use of contraception. Primary infertility refers to those who have never conceived while secondary infertility refers to those in whom at least one conception has occurred in one or both of the couple separately but have not achieved conception together. Most couples (80%) achieve conception within 1 year of unprotected intercourse, another 10% within the second year. [1-3].

In the patient presented, investigations found her to have bilateral tubal blockage while the husbands semenalysis was normal, thus the female factor as cause of infertility. The incidence of infertility ranges from 10-20% of all married couples worldwide. Exact statistics in Kenya are not known but it has been estimated that 60% of all patients attending the gynaecology clinic at KNH have infertility related complaints[4]. Both primary and secondary infertility were reported in equal proportions. Often the female patient is blamed for the infertility but the women contributes 40-50% to the causes and in 20-30% there is both a male and female factor, while the male contributes 30%[3]. In Africa the total female factor in infertility is 72% while in 35%there is both male and female factor and in 8% there is a pure male factor [5].

The aetiology of infertility is multifactorial; a world health organization (WHO) study between 1979 and 1984 found that up to 64% of females in Africa and 28-35% in other areas had infertility that could be traced to prior pelvic infection leading to tubal blockage. This extrapolated to about 49% bilateral tubal occlusion among African women and 11-15% among others[4]. In KNH, pelvic inflammatory disease has been blamed for tubal occlusion in over 70% of the cases of which gonorrhoea infection occurred in about 55% of the cases [3,7]. Chlamydial infections has now been found to be a major cause of tubal blockage accounting for even more cases than gonococcal infection especially in developed countries. It causes formation of 'violin string' adhesions in the pelvis. Genital tract tuberculosis is less common, occurring in about 2-6% of women in Kenya. Tubal blockage may also follow non-hygienic obstetric and abortion practices to pregnant women [3,7]. Although gonococcus and chlamydia may be responsible for acute salpingitis, residual chronic salpingitis and subsequent tubal damage is mainly due to the secondary invaders, both anaerobes and aerobic organisms [4]. These infections are associated with tubal damage adhesions. Oral contraceptives and barrier methods protect against pelvic infections whereas intrauterine contraceptive devices are known to predispose to pelvic infection. Our patient had not used any form of contraceptives.

Female fertility depends not only on an intact transport mechanism but also on regular ovulation. Ovulation disturbance contributes 20-25% of female infertility 2,3.

Anovulatory or irregular ovulatory cycles will result in subfertility or infertility. Disorders of ovulation are subdivided into clinical conditions including[8];

- Primary amenorrhoea – no spontaneous bleeding by the age of 20 years
- Secondary amenorrhoea – no spontaneous bleeding for a period of 6 months or more after menarche
- Oligomenorrhoea – cycles varying between 6 weeks and 6 months
- Anovulatory cycles
- Corpus luteum deficiency

The hormonal events associated with follicular maturation depend on well-controlled interrelations between the hypothalamus, the pituitary gland and the ovaries. Other causes of infertility are ovarian failure, pituitary failure, thyroid and endocrine disturbances, uterine and gonadal dysgenesis, uterine myomas and polyps, endometriosis, immunological factors as well as environmental factors (cigarette smoking, alcohol consumption). Our patient did not smoke or use caigarretes.

Semenalysis done for our patient's spouse showed normozoospermia. Male infertility can be due to several causes including abnormalities of sperm motility, morphology, count and ejaculatory problems. The commonest cause is due to oligospermia or azoospermia secondary to blockage in the vas deferens or varicocoele [6]. Inflammatory conditions involving the testis and its drainage such as epididymo-orchitis especially mumps orchitis has been implicated in causation of male infertility. A breach or break of the testis-blood barrier may lead to production of anti-sperm antibodies that can also lead to infertility.

The W.H.O. studied over 5000 women with infertility anf found no demonstrable cause of infertility in up to 27% of them4. Factors that affect the reproductive performance of a couple include their ages, frequency of coitus. There is an age related decline in fertility with advancement in age especially in women aged 35 years and over, with a sharp decline in their 40's. An average coital rate of 3 times a week has been found appropriate giving enough time for sperm maturation and covers all days of the fertile period. The patient presented gave a history of regular coitus.

The diagnostic evaluation of an infertile couple must involve both the man and the woman. In the woman, evaluation should begin with a thorough history and examination. The duration and type of infertility, coital function, menstrual history, pelvic disease, previous pelvic surgery and previous obstetric history and details of puerperal sepsis or abortion care. A history of deep dyspareunia or dysmenorrhoea especially secondary may suggest pelvic infection. Physical examination focuses on development of secondary sexual characteristics, body contour, hair distribution and the external and internal genitalia. For the male partner, a history of previous pelvic surgery or inguinal hernia especially during childhood is important as it may have been due to un-descended testis or may have caused damage to his spermatic cord hence testicular atrophy. Absence of one or both testis or presence of hydrocoele, varicocoele or testicular masses may point at suspected oligospermia or azoospermia. Previous history of sexually transmitted disease, or orchitis are important. A physical examination and semen analysis, which are both non-invasive and less expensive usually suffice..

The classic infertility evaluation of the infertile couple after complete history and physical examination consists of six basic steps:

- Evaluation of ovulation
- Evaluation of the male factors (semen analysis)
- Evaluation of the patency of the genital tract by hysterosalpingogram (HSG)
- Evaluation of the cervix mucus sperm interaction (post coital test)
- Evaluation of late luteal endometrial biopsy done during laparoscopy
- Evaluation of the peritoneal cavity during ultrasound and laparoscopy.

Semen analysis is done for the male to assay the sperm count, motility, morphology and vitality. The male factor is implicated in 30-40% of the cases of infertility[6, 9]. More invasive tests in the male include vasogram, testicular biopsy and blood specimens for anti-sperm antibodies and hormonal levels. This was done for our patient

In our patient, HSG showed bilateral tubal blockage that was confirmed at laparoscopy. Investigations in the female patient are invasive, expensive and time consuming therefore initial chosen tests should be cheap, simple and be able to provide information on tubal patency and surgical prognosis. Routinely done diagnostic tests include hysterosalpingography (HSG) and dye laparoscopy. These two are complementary tests in that the HSG will confirm patency while laparoscopy will show evidence of pelvic adhesions, ovarian cyst, endometriosis and thus evaluate possible success of further surgery [2,4,6]. HSG has been known to have fertility enhancing effects hence laparoscopy should be done 6 months following a normal HSG report. Conception rates have been known to increase 30% after HSG examination [10].

Several methods are used in evaluating ovulation. Basal body temperature charting is the least expensive and most valuable method. A biphasic temperature curve provides presumptive evidence of ovulation. A second indirect method of ovulation evaluation is the detection of the pre-ovulatory luteinising hormone (LH) surge. This can be detected in urine by available kits. A third method is mid-luteal increase in serum progesterone (peaks often 7 days after ovulation). The fourth method is the cervical mucous changes. Initially in the follicular phase the mucous is thick but thins out, becomes stretchable and profuse in mid-cycle as ovulation occurs. A trans vaginal ultrasound allows one to monitor follicular changes and confirms follicular collapse and presumably oocyte release. Secretory endometrium found in mid luteal phase by endometrial biopsy confirms ovulation occurred.

Therapy for infertility is aimed at identifiable problems. Pregnancy will occur without treatment in 15-20% of couples diagnosed infertile[3]. Problems of coital timing are tackled by counseling about the importance of adequate coital exposure during the woman's fertile period. Male azoospermia secondary to lack of gonadotrophin is remediable by use of human menopausal gonadotrophin (HMG), varicocoele are handled by varicocoelectomy done by the urologist, clomiphene may be effective in treatment of certain cases of oligo-asthenospermia. Those with oligospermia should avoid alcohol,

recreational drug use, tobacco and caffeine. They should also refrain from excessive coitus.

Anovulation and oligo-ovulation are treated by induction of ovulation using either clomiphene alone in increasing doses or in combination with HMG with success of ovulation occurring in 90% of cases. Corpus luteum insufficiency is treated with progesterone.

Tubal occlusion requires surgical management. Various methods of tubal surgery have been described including salpingolysis, salpingostomy, fimbrioplasty, end-to-end anastomosis and tubal re-implantation, which have been perfected by microsurgery. Success rates for tubal surgery have been promising in patients with distal tubal blockage with live births of 40-60% reported. Success generally depends on patient selection and is inversely proportional to the severity of tubal damage and to the extent and nature of the commonly associated adhesions. Mild flimsy adhesions carry better prognosis than thick broad and extensive pelvic adhesions. Presence of other pathologies like fibroids, endometriosis, infections and pelvic surgery that also predisposes to infertility and adhesion formation significantly reduce the success rates of tubal surgery. Female fecundibility decreases markedly after the age of 35 years hence a decline in success rates is expected after this age. Equally more than 60% of those who achieve extra-uterine pregnancy do so after the first year of follow up[12]. The role of repeat tubal surgery following a failed attempt remains controversial. It has been recommended only in selected patients with limited damage and has been found to have a better prognosis for overall term pregnancy rates than that achieved by in-vitro fertilization for patients with limited tubal damage[13].

Our patient had adhesiolysis and salpingostomy with free spill though delayed on the right side.The surgical techniques used in tubal reconstruction surgery are essentially the same irrespective of the access route. Whereas distal tubal disease appears very amenable to laparoscopic access, anastomosis procedures require open laparotomy. Tubo-cornual anastomosis is almost impossible to perform with laparoscopy while isthmic-isthmic and isthmic-ampullary is better and easier done by the open route. Salpingo-ovariolysis involves the excision of adhesions around the tubes and ovary to free the tube. It precedes most tubal reconstructive surgery. Broad adhesions are excised and removed from the pelvis, but shallow flimsy adhesions may be simply divided. Fimbrioplasty involves the reconstruction of fimbrial agglutination or pre-fimbrial phimosis. The fimbrial ends may be everted and secured onto adjacent ampullary serosa with No 7/0 or 8/0 vicryl sutures. Salpingostomy or salpingo-neostomy involves creation of a new ostium in a tube with a completely occluded distal end, such as with hydrosalpinx. Salpingostomy can be terminal, ampullary, isthmic or in form of a gutter along the avascular wall of the tube which is adjacent to the ovary.

Tubo-tubal anastomosis refers to anastomosis performed anywhere along the tube either to treat occlusion resulting from tubal disease process or to reverse prior sterilization. Tubo-cornual anastomosis is the process used to repair and anastomose the proximal or corneal tubal disease, which occludes the tube at the utero-tubal junction. Some rare and

technically difficult procedures may be performed for patients with exceptional circumstances such as in women with a single unicornuate uterus whose ipsilateral tube and ovary are damaged or non-functional. Tubo-ovarian anastomosis may be performed by microsurgical transposition of the opposite side ovary and functioning tube.

The success rates of salpingo-ovariolysis are reported as 41-57% achieving pregnancy, with 5-8% being ectopic pregnancy. In salpingostomy pregnancy rates of 20-37% and ectopic rates of 5-18% has been reported. Tubo-tubal anastomosis has variable results depending on the site on the tube, technique used, cause of tubal damage and the experience of the surgeon, but ectopic pregnancy rates were higher at 13-22%. In tubo-cornual anastomosis rates range at 33-58% with ectopic pregnancy rates ranging 5-7%. The Weltz criteria for selection is associated with better outcome [4,5,9,11].

Microsurgery, which has been defined as surgery under magnification embraces a broad concept of tissue care designed to minimize tissue damage[4]. In addition to delicate handling of tissue and judicious use of electrical or laser energy, frequent intraoperative irrigation with heparinised Ringer's lactate solution is performed to keep serosal surfaces moist and to prevent desiccation. Meticulous pinpoint haemostasis is achieved while minimizing the adjacent tissue damage. Microsurgery also allows precise alignment and approximation of tissue planes with use of magnification, which permits prompt identification of abnormal morphologic changes, recognition and avoidance of surgical injury [4,9,11].

Complications of tuboplasty include formation of adhesions, which worsens the tubal damage and kinking and a higher incidence of ectopic pregnancy arising from irregular tubal healing process or from the adhesions which kink the tube. More than 60% of those who achieve extra-uterine pregnancy do so during the first year after surgery. Our patient had not conceived a year after surgery.

Prevention of sexually transmitted diseases, pelvic infection including puerperal sepsis will decrease the incidence of infertility because about two-thirds to three quarters of tubal blockage cases follow an infective process. Early appropriate treatment of infections will slow disease progression to adhesion formation and subsequent tubal damage. Strategies for treating infertility should encompass management of the frustration and sense of personal loss that accompany the process of investigation and treatment[2,3,8]

In couples for whom evaluation and therapy has been unsuccessful and all options of treatment have been exhausted, adoption may be adviced. In-vitro fertilization where practiced offers an alternative mode of management though success rate is 20% per treatment at best.

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GYNAECOLOGY CASE 13

DESIRED FAMILY SIZE – BILATERAL TUBAL LIGATION

NAME: T.N. L.M.P 14.12.03

AGE: 36 Years PARITY 2 + 0

I.P NO. 0989176

D.O.A: 26.10.04

D.O.D: 30.10.04

PRESENTING COMPLAINT

She presented to labour ward in labour pains and was for permanent surgical contraception during scheduled caesarean section as she had attained her desired family size.

HISTORY OF PRESENTING COMPLAINT

She came having labour like pains for 12 six at 37 weeks gestation. During her clinic follow up she had requested for and was counseled on available contraception methods and opted for tubal ligation. She felt this was good for her as she had previous uterine surgery for myomectomy and she was thus to be delivered by elective caesarean section. She and her spouse had been explained on the permanence of the procedure, all their worries clarified and both expressed satisfaction and gave informed consent on the necessary forms during one of their antenatal clinic visits. She was to have the procedure done at the time of caesarean section. But she went into active labour before the scheduled operation day and was admitted for emergency operation.

ANTENATAL CARE

TN had started antenatal care at six months gestation at a private clinic then continued at KNH from 34 weeks. Antenatal profiles done indicated blood group O positive, haemoglobin level 11g/dl, and her HIV and VDRL tests were negative. The antenatal period had been uneventful. Her L.M.P was on 14.12.04 and was thus at 37 weeks gestation at the time of admission.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She was a Para 2+0 being in her third pregnancy. Her first pregnancy was in 1978 to a 2.8kg female baby. Her second delivery was in 1990 to a 2.9kg male baby. Both were hospital deliveries and the children were alive and well. She attained her menarche at 13 years, her cycles were regular occurring every 28 days and lasting 3-4 days. She had no

MANAGEMENT

She was scheduled for emergency caesarean section and bilateral tubal ligation under general anaesthesia. She was further counseled on the procedure and consented readily. She was prepared by shaving the abdominal wall and premedication with atropine 0.6mg; then taken to the operating theatre.

In theatre, vulvo vaginal toilet was done, she was catheterised and the urinary bladder drained. Under general anaesthesia, the abdomen was opened in layers through a sub-umbilical midline incision after surgical cleaning and draping. A lower segment caesarean section was then done and the uterine wall closed in layers and haemostasis was achieved. The outcome was a live male infant who weighed 2.5kg and scored 7 in one and 9 in five minutes.

With the uterus exteriorized, the fallopian tubes were then identified up to their fimbrial ends. The right tube was then held with a Babcock tissue forceps in its middle to form a loop, which was ligated using chromic No. 2 by Pomeroy's technique-a section of the tube in the loop was cut after ligation. Haemostasis was then ascertained. The left tube was then identified and the same procedure as done on the right side repeated. The abdomen was then closed in layers, the wound dressed and the patient reversed from anaesthesia and taken to the recovery room.

POSTOPERATIVE MANAGEMENT

She was on intravenous fluids over the first 24 hours after operation. She was started on oral sips on the second day and gradually to free fluids and solid diet by the third day. She was also on crystalline penicillin 2MU 6 hourly and gentamicin 80mg 8 hourly, and pethidine 100mg 8 hourly for analgesia. By the fourth day the wound was opened and found to be dry and healing well. She was discharged on amoxicillin 500mg and mefenamic acid 500mg three times daily for five days. She was to be seen in the postnatal clinic in six weeks time.

FOLLOW-UP

When she turned up she had no complaint. Both her and the baby were well. The wound had healed well. She was discharged from the clinic and adviced on regular follow up with Pap smear in the family welfare clinic. She was also adviced about protection against sexually transmitted infection (STI) as the BTL only protected her from pregnancy. Use of barrier methods (condoms) was adviced as protection against STI.

DISCUSSION

A case is presented of a 36-year-old lady with her desired family size and underwent voluntary bilateral tubal ligation using Pomeroy's method.

Our patient had tubal ligation at the time of caesarean section. Bilateral tubal ligation (BTL) is a permanent surgical method of contraception and is also known as tubal sterilization. It involves mechanical occlusion of and/or resection of the fallopian tube to prevent conception [1,2]. It can be done at the time of caesarean section, shortly (48 hours) after delivery, shortly after abortion or at a time unrelated to pregnancy when it is called interval sterilization. The case presented was done BTL at caesarean section. Tubal sterilization confers permanent contraception that is effective immediately. Those done after vaginal delivery are mostly via minilaparotomy with sub umbilical incision and partial salpingectomy. Postpartum minilaparotomy is usually done within 48 hours before the onset of uterine involution and colonization of bacteria ascending from the vagina through the uterine cavity. This period is convenient because the uterine fundus is near the umbilicus and the tubes easily accessible. Interval BTL may be performed by minilaparotomy with partial salpingectomy or by laparoscopy with use of coagulators, silicon rubber band application, or spring clips application [1-3].

Our patient had reached her desired family size of three children. Tubal ligation was first performed in 1823 to prevent pregnancy in women who would need repeated caesarean sections during childbirth. Since then, many modifications have taken place and it is offered to women who have desired family size and wish to have a permanent method of contraception. Other indications include women in whom a pregnancy could represent a significant clinical and medical risk, such as patients with diabetes with severe complications and those with severe cardiac disease.

The patient presented had previously used combined oral contraceptives. The contraceptive prevalence rate in Kenya is 39% and most current users of contraceptives use a modern method. It is not clear when tubal sterilization was introduced in the country but it has gained popularity alongside other modern methods of contraception. Injectables and the pill are the most commonly used contraceptive methods, 12% and 9% of married women using them respectively. 6% of married women have been sterilized, 3% are using IUD, 1% are each using condoms and implants. The use of male sterilization and vaginal methods is rare. Periodic abstinence is used by 6%, withdrawal 1% and other traditional methods by 1%. Contraceptive use, especially modern methods has risen sharply since 1980's and is probably the principal cause of decline in fertility. The total fertility rate has declined from 8.1 in mid 1970's to 4.7 children per woman, a decline of 42% over a 20-year period [4].

In our patient, BTL was done at caesarean section. Surgical approaches for BTL include laparoscopy, minilaparotomy, laparotomy (concomitant with caesarean section), and vaginal approach. Local anaesthesia is mainly used during minilaparotomy. Few cases may be performed under para cervical block or general anaesthesia such as in the obese, those with previous abdominal/pelvic surgery and chronic pelvic inflammatory disease.

Operating under local anaesthesia has possible disadvantages such as increased patient anxiety and incomplete analgesia. The surgeon has to be gentle and talk to the patient during the procedure [1,5]. In KNH, minilaparotomy and laparotomy at caesarean section are the common approaches used but laparoscopy is also being done. The patient presented had BTL at caesarean section.

Informed consent had been given by our patient before labour and delivery. Preoperatively, informed consent and counseling are very important. The discussion should include the available alternatives, explaining the procedure together with its permanence and irreversibility, failure rate 0.2-0.4% and the complications that may arise during the procedure. The relative likelihood of ectopic pregnancy where failure exists should be explained. A careful history should be taken to find out about previous pelvic infections, previous abdominal or pelvic surgery, medical disorders, allergies and recent infections. It is also important to determine the last menstrual period to ensure she is not pregnant or perform a pregnancy test [3,5,6]. Screening her for risk indicators for regret including young age, low parity, single parent status or marital instability. Stress the need for condom use for protection against sexually transmitted diseases and HIV infection if the patient is at risk of exposure. A general examination and assessment of heart, lungs and abdomen is done followed by pelvic evaluation paying attention to uterine position, its mobility and any infection or adnexal masses. These help the surgeon judge the difficulty that may be encountered if the patient is obese, the uterus is immobile or if the tubes have adhesions from previous surgery or infection. Current infection increases the incidence of postoperative sepsis as well as making the procedure difficult due to pain and pelvic organ hyperaemia [1-3]. Our patient was not obese and other examination had found her fit for the procedure.

The patient presented did not have any contraindications for BTL. Contraindications for tubal sterilization are few. The relative contraindications include obesity, pelvic adhesions, previous abdominal or pelvic surgery, and chronic pelvic inflammatory disease. Each of these factors should be assessed to reach a safe decision. Absolute contraindications include current pelvic infection or peritonitis. Abnormal Pap smear may be an indication for sterilization under local anaesthesia [1,3].
Our patient had the procedure under general anaesthesia. Preoperative medications serve to reduce fear and anxiety. It can provide analgesia, prevent postoperative nausea and vomiting and induce amnesia. The goal for anaesthesia should be to minimize psychological and emotional distress and trauma to the client and free her from pain and discomfort.

The Pomeroy's method was used to tie the fallopian tubes in the case presented. Many surgical techniques for accomplishing tubal ligation have been described. The Pomeroy technique is the simplest and most commonly performed puerperal tubal sterilization. The mid-portion of the tube is grasped with a Babcock's clamp creating a loop, which is tied with 2-0 or 0 plain catgut suture, and each limb of the tubal knuckle is cut separately. Specimens are submitted for histology. The endosalpinx at the cut ends may be cauterized (optional). The ligation sutures are held while the tube is cut to prevent retraction of the cut tubal stumps into the peritoneal cavity before they can be adequately

examined for haemostasis [1-3]. Failure rates are reported to be 1 in 300-500 patients [7]. Other techniques for BTL include The Parkland technique, a mid-segmental resection similar to Pomeroy's except that each leg of the loop is tied separately. Other techniques include the Uchida technique, Irving's technique, electrocoagulation, use of bipolar and unipolar currents. Mechanical techniques of sterilization include Falope (Yoon) ring technique, the Hulka-Clemens clip technique and the Filshie clip technique[8]. These mechanical methods are used mainly in laparoscopy. Irving and Uchida methods are designed in attempt to reduce the risk of tuboperitoneal fistula or ectopic pregnancy. The proximal ends of the tubes are extensively dissected, ligated and buried in a deep pocket created in myometrium on the posterior uterus in Irving or buried in surrounding mesosalpinx in the Uchida method. The major disadvantage with these techniques is that they carry a potential for greater blood loss as well as difficulty in performing them through a minilaparotomy incision. The Madlener technique of crashing and ligating the tube without excising any part is unreliable and is prone to recanalisation. It carries a failure rate of 3% and is rarely used. Similar fate has befallen the Kroener technique in which fimbriectomy including part of the ampulla is resected. In our patient, the Pomeroy method was used.

Laparoscopic sterilization is suitable for interval or post-abortal cases and not postpartum sterilization due to likely injury of the highly vascularised and bulky uterus. Laparoscopy involves actual disruption of tubal continuity using silicon rubber bands, spring clips or electrocoagulation with or without tubal transection. Laparoscopy requires highly skilled and trained personnel and equipment, which is costly. Tubal adhesions, thickened tubes can make this procedure very difficult. Vaginal sterilization via colpotomy can also be done but is associated with higher risk of complications such as infection[2].

Follow-up care both in the immediate and long term is important. The follow up visit is usually 1-2 weeks postoperatively. In KNH it is usually after 10 days. The patient is instructed to notify the health care provider if she develops fever (38°C or 100.4°F, dizziness with fainting, persistent or increasing abdominal pain, or bleeding/fluid coming from the incision. She should also be aware of the signs of pregnancy amenorrhoea, vaginal bleeding/spotting, and abdominal pain as ectopic pregnancy may occur thus early diagnosis necessary [8].

Complications can occur during sterilization. They usually occur in less than 1% of all sterilization cases and include wound infection, uterine perforation with the uterine elevator, bladder injury, intestinal injury, anaesthesia complications and sterilization failure. The risk of death during sterilization is 1-2 cases per 100,000 procedures. Most of these are complications of general anaesthesia during laparoscopic BTL. The most common cause of death during laparoscopic BTL appears to be hypoventilation related to anaesthesia, cardiopulmonary arrest, sepsis, bowel perforation or electrical bowel burns. Vessel injuries can also occur with laparoscopic BTL. Unintended laparotomy occurs in 1-2% of laparoscopic procedures. Most of these conversions are attributable to technical inability to complete the laparoscopic procedure rather than to complications of the procedure [8]. Bowel injury occurs during insertion of the insufflation needles or trochar or during electro coagulation [9]. Vascular injury can occur during insertion of trochar or

insufflation needle. Injury to a large vessel can lead to life-threatening haemorrhage. Overall mortality rate for the minilaparotomy procedure is 4-9 deaths per 100,000 procedures and is usually from anaesthesia complications or sepsis. Long-term complications after tubal sterilization include ectopic pregnancy after sterilization failure, menstrual pattern changes and regret. Most of these complications can be prevented by careful screening, local anaesthesia with light sedation, monitoring vital signs, good asepsis, and careful surgical technique. The seriousness of complications can often be minimized by early recognition and aggressive management. Our patient did not develop any of these complications.

Abnormal menstrual patterns have been thought to occur following sterilisation and a controversial " post tubal ligation syndrome" has been proposed with a constellation of symptoms including pelvic discomfort, ovarian cystic changes and menorrhagia that are suggested to occur as a result of disruption of the utero-ovarian blood supply. However, a specific and describable pattern of bleeding after sterilisation has not been convincingly demonstrated. Often these patients have history of similar problems before BTL or have been taking birth control pills, which masked their symptoms. After extensive study, BTL apparently causes few, if any menstrual abnormalities within several years after sterilisation, regardless of the method of tubal occlusion used. Data from the CREST study found that women who have had sterilisation were more likely to develop inter menstrual bleeding or changes in menstrual cycle length than those not sterilized. However, sterilized women were more likely to report reduced bleeding duration, less overall bleeding amount of flow and reduced menstrual pain. BTL also seemed to help reduce blood flow in women who reported very heavy menstrual bleeding at baseline. Our patient was still on follow up and had lactational amenorrhoea at the last visit.

BTL failure (pregnancy or ectopic pregnancy) can sometimes occur. Although sterilization is highly effective and considered the definitive form of contraception, it has a failure rate during the first year of 0.1-0.8 %. At least one-third of these are ectopic pregnancies. Recent findings suggest that pregnancy is somewhat common than previously estimated; that the risk of pregnancy persists for many years after sterilization and the risk varies by method and age of patient at sterilization [10,11]. Sterilisation failures can be categorized as:

- Luteal phase pregnancy defined as pregnancy in which conception occurs before the BTL but the pregnancy is diagnosed after the interval tubal occlusion. This was unlikely in our patient as BTL was done at the time of caesarean section.

- Misidentification of the tubes due to poor visualization from inadequate exposure. Adhesions, adnexal pathology, or poor lighting may result in mistakenly ligating the round ligament, ovarian ligament, infundibular ligament or dilated broad ligament blood vessels instead of the fallopian tubes. This can be avoided by following the tube to its fimbrial ends.

- Incomplete occlusion of the oviduct occurs because of poorly placed mechanical clips or the use of mechanical devices on oedematous or dilated tubes. Incomplete tubal

occlusion with electrocoagulation is generally associated with too brief application of current or with the use of modulated coagulation current instead of unmodulated cutting current.

Our patient was on follow up and failure was not expected. The main causes of sterilization failure include spontaneous re-anastomosis, fistula formation, surgical error, equipment failure in laparoscopic sterilization and luteal pregnancy [1,2,12]. In Kenya, Ruminjo [12] reported a crude failure rate of 0.4% and 50% of these failures were due to luteal phase pregnancies while 20% were in patients who had previous ectopic pregnancies and the ectopic site tube turned out to be patent. Timing the procedure to be done during the follicular (proliferative) phase of the cycle can reduce the risk of luteal phase pregnancies.

Sterilisation is intended to be permanent, but patient regret is not rare. Post-sterilisation regret is a complex condition often caused by unpredictable life events. Risk factors for regret that may be useful in pre-sterilisation counseling include young age, low parity, single marital status or being in an unstable relationship. As many as 6% of women who are sterilized report regret or request information about tubal reversal within five years of the procedure. Follow up interviews 14 years post procedure demonstrated that regrets were expressed by 20.3% of women aged 30 years or younger at the time of BTL and by 5.9% of women older than 30 years at the time of the procedure [13,14]. About 1% of all women who undergo sterilisation request reversal. The success rate of the reversal procedure depends on the extent of tubal damage removal at the time of sterilisation. However, careful microsurgical techniques can reverse the sterilisation procedure in up to 50% of cases. Success rates are usually higher for occlusive procedures. Isthmico-isthmic anastomoses are most likely to be successful.

BTL also confers some non-contraceptive benefits such as decreased risk of ovarian cancers. Some studies report a protective effect of BTL against ovarian cancer, with a relative risk ranging from 0.2-0.8. Protection is hypothesized to result from reduced exposure of the ovaries to potential environmental carcinogens and infective sources of malignant transformation (e.g. oncogenic viruses) BTL does not protect against sexually transmitted infections but the incidence of pelvic inflammatory disease (PID) is reduced since sterilisation has been demonstrated to reduce the spread of organisms from the lower to the peritoneal cavity and thus protect against PID. [15,16]. Our patient was adviced against protection from STI'S.

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GYNAECOLOGY CASE 14

VESICOVAGINAL FISTULA – SUCCESSFUL REPAIR

NAME: E.N Parity: 1 + 0

AGE: 25 Years

I.P No. 0775017

D.O.A: 03/04/2002

D.O.D: 15/04/2002

PRESENTING COMPLAINTS

EN was admitted with over 4 months complaints of continuous involuntary leakage of urine.

HISTORY OF PRESENTING COMPLAINTS

EN had delivered through emergency caesarean section five months prior to admission (November 2001) at Kenyatta national hospital. She had not attended antenatal care during that pregnancy and had been in labour at home for 3 days without any assistance. At admission, she was diagnosed to have obstructed labour and was delivered by emergency caesarean section. The outcome was a live baby weighing 2900g, with Apgar score 1 at one minute and 3 at 5 minutes. The baby was admitted to the intensive care unit with asphyxia and died after 5 days. At operation, the urine was noted to be blood stained and the bladder was oedematous. There was no injury to the bladder at operation. She developed postoperative wound sepsis. She had a catheter retained for 14 days upon whose removal she was noted to have leakage of urine. Another catheter was reinserted and she was discharged and to be reviewed in the VVF clinic after 8 weeks. At review she was noted to have a vesicovaginal fistula and was readmitted on 3/04/02 for fistula repair. She also had not received her menstrual period since the operation despite the baby having succumbed in the newborn unit days after birth. She reported no stool incontinence. She felt rejected by family and friends and thus avoided social gatherings due to persistent smell of urine. She had no other major complaints.

OBSTETRIC AND GYNAECOLOGY HISTORY

EN attained menarche at 16 years and her cycles prior to the first delivery were regular occurring every 28-30 days and lasting 4 days. She was para 1+0, had no living child the baby having died after caesarean section mentioned above due to severe foetal distress. Her last menstrual period was on 15/03/2002. She had not resumed her menses after the last delivery. She had never used any form of contraceptives and had never had a Pap smear.

PAST MEDICAL HISTORY

She had no other admissions except the above. She had no chronic illnesses or allergies.

FAMILY AND SOCIAL HISTORY

She was a single lady who lived in the Mukuru slums with a friend. She was the second born in a family of 8 siblings and worked as a casual labourer in a plastic industry. She gave no family history of chronic illnesses. She did not use alcohol or smoke cigarettes.

PHYSICAL EXAMINATION

She was in a good general condition, was not pale, and had no jaundice or oedema or lymphadenopathy. Her blood pressure was 110/60mmHg, pulse rate 82 beats/minute, respiratory rate 16/minute and temperature 36.7°C. Her height was 145cm.
Her cardiovascular, respiratory systems and nervous systems were normal.

ABDOMINAL EXAMINATION

She had a lower midline surgical scar. The abdomen was soft and had no palpable masses or any areas of tenderness.

SPECULUM EXAMINATION

Using the Sims speculum and the patient in lithotomy position, the anterior vaginal wall was visualized and inspected. Fibrosis was noted in the anterior vaginal wall. A fistula was noted about 3cm from the external urethral meatus and about 1cm from the cervix.

PELVIC EXAMINATION

She had excoriation of the vulva and the inner aspects of the upper thighs. An odour of ammonia was evident and clear urine was flowing from the vagina. The cervix was posterior, firm smooth and closed. A defect could be felt in the anterior vaginal wall. The uterus was normal size. The adnexa and pouch of Douglas were free of enlarged masses.

DIAGNOSIS

Vesico-vaginal fistula type II secondary to obstructed labour.

INVESTIGATIONS

1. Haemogram- haemoglobin 10.4g/dl
   white cells count 4.5x 10^9/l, platelets 295 x 10^9/l
2. Urea and electrolytes- Na^+ 130mmol/l, K^+ 4.0mmol/l, urea 2.8mmol/l

MANAGEMENT

The patient was admitted and planned for examination under anaesthesia and repair of fistula at the same sitting. The diagnosis and mode of management were explained to the patient and she gave informed consent for the procedure.
On the day before surgery, she was adviced to fluid diet only. An enema was given on the evening and morning prior to operation. She was fasted from midnight on the night before surgery. Her pubic hair was shaven. On the morning of surgery, a drip of 5% dextrose was started and premedication with atropine 0.6mg was given intramuscularly half hour before she was wheeled to theatre.
In theatre and under spinal anaesthesia, she was placed in the lithotomy position. Vulvovaginal toilet was done and examination confirmed a high vesicovaginal fistula. The diameter was 1-2cm. The fistula was about 3cm from the external urethral opening, and was 1cm from the cervix. The tissues around the fistula were free and mobile. Measurements were done and the fistula graded as type IIAa.
The labia were then held laterally with a stitch. A mediolateral episiotomy was done to create more space for operation. Jungle juice was infiltrated around the fistula to reduce bleeding and to ease dissection by making the tissue planes easier to identify. Gauze was used to cover the anal opening to prevent soiling of the operation site with faeces. Dissection around the fistula with bilateral extension was then done. The bladder was then mobilized. The fistula was then closed transversely with interrupted vicryl 2/0 sutures in one layer while avoiding tension. Methylene blue dye was instilled into the bladder and no leakage was noted. The vaginal mucosa anteriorly was closed transversely. The episiotomy was then repaired. A Foley's catheter gauge 16 was left in situ draining well. A vaseline gauze pack was left in the vagina.
The patient was then taken to the recovery room for observation and to the ward when she was stable enough.

POSTOPERATIVE MANAGEMENT

She was given intravenous fluids 500mls every four hours of normal saline alternating with 5% dextrose. She was also put on diclofenac for analgesia. She was also adviced on taking plenty of oral fluids at least 6-8litres per day. The vaginal pack was removed after 24 hours. No leakage was observed while in the ward. She was discharged on the fifth postoperative and adviced to be seen in the clinic on the 14th postoperative day. She was also adviced on pelvic muscle exercise (Kegel's isometric exercises)

FOLLOW UP

She came to the clinic on the 14th day and dye test was done which showed no leakage of urine. The patient had also not noted any leakage of urine while at home. She was instructed to abstain from sexual intercourse for four months, pass urine frequently and wake up at least once during the night to pass urine. She was also to continue with taking plenty of fluids and the pelvic exercises. At three months she was seen and still had no leakage and was quite happy. She was discharged with instructions that in case of conception, early antenatal care is necessary and future deliveries will be by elective caesarean section.

DISCUSSION

A case is presented of a 25 years old para 1+ 0 who had a vesicovaginal fistula following obstructed labour that was successfully repaired vaginally. At review 3 months after repair, she had no leakage and repair was considered successful.

A vesico-vaginal fistula (VVF) is an abnormal communication between the vaginal epithelium and the bladder epithelium. A genito-urinary fistula is an abnormal communication between the genital and urinary tracts either acquired or congenital leading to involuntary leakage of urine through the vagina. It is a socially embarrassing condition that makes the patient reclusive and depressed as she often smells of urine and becomes an outcast. Malnutrition and neglect may also supervene [1,2]. The patient presented had VVF and felt neglected by her relatives.

Our patient was 25 years and had VVF during her first pregnancy.The incidence of genitourinary fistula varies from region to region. Harrison suggested an incidence of 95 per 100,000. The fistulas are more in developing countries with incidence of 0.5-3% in the developed world. The estimated prevalence of genital fistula in Africa is 1-2 per 1000 deliveries. The incidence peaks in late teenage and in mothers with low parity and often in the first or second pregnancy [1,2,4,7].

The patient presented had fistula after difficult labour. The cause of fistulae varies. Obstetric causes account for 87-92% of all VVF at Kenyatta national hospital whereas in the west, 5-10% of VVF are secondary to obstetric causes. In the United States, the most common cause of fistulae is pelvic surgery accounting for 45%, cancer with or without radiotherapy 34% and obstetric cause 15%. Obstetric causes have become less important in the developed world due to improved obstetric care methods. The other causes of fistula which contribute less than 10% include cervical cancer, trauma on vagina or cervix and bladder, anterior colporrhaphy, foreign bodies, congenital anomalies and infections such as tuberculosis, schistosomiasis and syphilis[2,3,4,7].

Our patient had obstructed labour, relieved by caesarean section and later developed urine leakage. Obstetric causes of fistula include prolonged labour, obstructed labour, ruptured uterus difficult instrumental deliveries e.g. vacuum, and destructive deliveries. In obstructed labour, pressure necrosis causes sloughing off of the affected area with or without sepsis. Pressure necrosis occurs due to compression of the bladder or urethra between the presenting part and the pubic symphysis during labour. This leads to devitalisation of tissue due to ischaemia, then sloughing off occurs between the second and fifth postpartum days. The area usually affected is the anterior vaginal wall and the underlying bladder neck. Occasionally the trauma may involve the upper vagina and sometimes the anterior lip of the cervix. Although prolonged and obstructed labour is the direct leading cause of obstetric fistula, it is invariably a result of neglected or unmanaged cephalopelvic disproportion. In Nairobi, it has been shown that the average true conjugate among women sustaining fistula was less than 9cm [10]. It has been pointed out that cephalopelvic disproportion is common and very important obstetric complication in Africa [5,6]. It has been suggested that the small pelvis is due to

malnutrition in childhood and improvement in childhood diet has been recognized as one of the major contributors to the reduction of cephalopelvic disproportion in developed countries.

Our patient's baby had low Apgar score at delivery and died days later in the newborn unit. Obstetric fistulae are associated with high perinatal mortality rates. Neonatal death of 63% and perinatal mortality of 80% has been reported [2,3,4]. At the KNH, still birth rate of 63.7% was observed [5,6] with Orwenyo reporting perinatal mortality rate of 80.4%. Average peak incidence age was 20-24 years. Cultural factors also add to the risk of complications of childbirth. Even when skilled maternity care is available, utilization may be poor, confidence in its value is hard to win so prenatal clinics are poorly attended and elderly relatives or unskilled traditional birth attendants usually conduct deliveries at home.

Our patient presented with amenorrhoea and excoriation.Obstetric fistula is associated with other complications including rectovaginal fistula in 10% of cases[6]. This patient did not have stool incontinence.Oligomenorrhoea or secondary amenorrhoea occurs in 40% of cases[5]. Others are obstetric palsy, severe vaginal stenosis. Other features that may be present with fistula are redness of the vulva and perineum from excoriation and ulceration, itchiness, anaemia and vesicle calculi [2,7]. Amenorrhoea is due to among other factors, inhibition of the hypothalamo-ovarian axis due to stress, chemical endometritis or psychogenic disturbances.

Diagnosis in our patient was not difficult as urine leakage was obvious on examination.The diagnosis of VVF is usually not difficult. When the fistula is large, it is palpable during vaginal examination. Smaller fistulae are difficult to detect. Pooling of urine in the upper vagina occurs and on speculum one can see a small-reddened area on the anterior vaginal wall from which urine escapes. The three-swab test involves placing three swabs in the vagina and instilling dye into the bladder. If the last swab only stains then the fistula is urethral; if the middle and upper swabs stains then its vesical fistula; if only the uppermost swab has wetness without staining, then the fistula is ureteric [2].Intravenous urogram is essential to rule out concurrent ureteric fistula. It may also show abnormalities of the ureter and calyces. Cystoscopy can be used to identify bladder fistulae.

The fistula in our patient was noted to be about 3cm from the external urethral orifice. Fistulae may be located at any point along the anterior vaginal wall and may include any part or all of the bladder base and urethra. They may be single or multiple and may involve the ureter. They may be classified as juxta-cervical, midvaginal or juxta-urethral. Orwenyo found juxtacervical to be the commonest accounting for 34% of cases at KNH5. Depending on communication level, fistulae may also be classified into utero-vesical, vesico-vaginal, utero-vaginal, urethro-vaginal and vault fistulae. They may also be classified as small (less than 2cm), medium (2-3cm), large (4-5cm) and extensive (more than 6cm). A classification according to their anatomic and physiologic location is also currently in use. Various fistulae fall into one of three types using this classification. This

classification helps to determine those types of fistula that may develop urinary incontinence after repair. These include;

Type I Fistulas not involving the closing mechanism. They are usually more than 5cm above the urethral opening

Type II Fistulas involving the closing mechanism. Usually within 5cm from the urethral opening

A Without (sub) total involvement of the urethra

a. Without a circumferential defect
b. With a circumferential defect

B With (sub) total involvement of the urethra

a. Without a circumferential defect.
b. With a circumferential defect

Type III Miscellaneous e.g. ureterovaginal and other exceptional fistulas.

Our patient had type IIAa fistula using this classification.It did not close spontaneously even after a catheter was inserted for 8 weeks after delivery.Urinary fistulas have a tendency to close spontaneously by granulation, fibrosis and contraction but this process may be interfered with by sepsis and continuous flow of urine. Once the fistula is formed, continuous drainage of the bladder by use of catheter and control of infection with antibiotics have been shown to reduce the size of the fistula and may even lead to closure of the fistula[4]. Waaldjik reported that insertion of an indwelling catheter immediately obstructed labour is detected promotes the spontaneous healing of smaller fistulae up to 2cm in size. These may occur in about 60% of patients in 2-3 months[8]. Most authours maintain that repair of fistula should not be attempted until about 3 months after the causative labour. This can reduce the size of fistula by spontaneous healing and allows for inflammation to subside, infection to clear and tissue necrosis to heal so that enough surrounding normal tissue forms. With complete re-epithelialisation and revascularisation of the fistula tract, the size of fistula reduces in 20% of cases. During this time, infections are managed, nutrition improved and labial excoriation managed by zinc oxide or castor oil/lotion application.
The waiting period is uncomfortable, depressive embarrassing and restrictive to the patients both socially and even in marital obligations. Orwenyo found separation rates of 87% among couples affected and in 65% of cases had not had sexual intercourse since the fistula formed.

In our patient, repair was done 8 weeks after the VVF developed and was successful at first attempt. Currently early (after 6-8 weeks) full repair is being advocated as soon as the slough has disappeared. The recommendation is that any woman who develops an obstetric fistula should have a catheter inserted immediately and as soon as the slough has disappeared, fistula is clean, there is no infection, and then repair can be done early[8].

Preoperatively, examination under anaesthesia (EUA) is done to determine the number, site and size of fistula, the amount of fibrosis, and fixity around the fistula and to see other associated genital lesions that may be present. EUA also helps to determine the best route, position and mode of repair[4, 7,8]. In our patient EUA and repair was done at one sitting.

The vaginal route was used to approach the fistula in the case presented. An indwelling catheter was left in situ for the first 14 days post repair.The surgical approach is dependent upon the position of the fistula and the surgeon's preference. Many prefer the vaginal route. Positioning is important (dorsal lithotomy, Sims or knee chest) and the preferred position should give adequate exposure and accessibility. Episiotomy or Schuchardts incisions are used to aid in exposure. The suture material used should be small caliber, strong, absorbable after long time and on atraumatic needle. In our patient, vicryl 3/0 was used. Excellent success rates are achieved if the following general principles are followed: complete pre-operative diagnosis, adequate exposure, good haemostasis, mobilization of tissues, tissue closure without tension, water tight closure of the bladder, timing to avoid infection and inflammation of tissue, adequate blood supply at area of repair and continuous catheter drainage post-operatively.

Postoperative care is very important. An indwelling catheter is retained. The aim is to have high volume uninterrupted urine flow as this prevents ascending urinary tract infection and prevents blockage of the catheter in order to enable the bladder stay empty and avoid placing tension whatsoever on the suture line. Therefore the patient is adviced to take enough fluids with minimum intake of 4 litres but preferably 5-6 litres every 24 hours in order to produce a minimum of 4000mls of urine per 24 hours. The catheter is maintained for 14 days. If the catheter blocks, it must either be flushed out or changed as any delay will result in tension of the sutures and breakdown of the repair. Antibiotic use is currently not favoured as the fistula is caused by pressure necrosis and not infection. On day 14 after operation, dye test is done to determine the integrity of the repair and check for any leakage. If no leakage is noted, bladder-training exercises are introduced and the catheter removed. If there is leakage, a fresh catheter is inserted and retained for a further 14-21 days to give more time for spontaneous healing. On discharge after successful repair, the patients are adviced to void frequently, to take plenty of fluids, and abstain from sexual intercourse for at least 3 months to allow healing. Pregnancy is avoided for one year and future pregnancies are delivered by elective caesarean section.

Successful repair is gauged by whether the patient is continent of urine or not. The site and extent of the fistula and postoperative care may affect the outcome. Lawson stated that an experienced surgeon supported by a competent nursing staff should be able to achieve overall success rates of 75% at first attempt, a further 15% at second attempt, since each successive repair produces more scar tissue. Successful first attempt is thus the goal[4]. Our patient had successful repair at first attempt.

Complications of VVF repair include failure of repair, infection, catheter blockage and breakdown of repair, urinary retention, vaginal stenosis with resultant dyspareunia, and stress incontinence. Complicated fistulaes include those involving the bladder neck and ureters; those associated with intestinal fistulae, very large fistulas and failed repairs. Our patient did not develop any complications post repair.

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GYNAECOLOGY CASE 15

SEXUAL ASSAULT – MANAGEMENT

NAME: P.W. Parity: 0+0

AGE: 18 Years

O.P. No. 47865/05

D.O.A: 20/09/05
D.O.D: 20/09/05

PRESENTING COMPLAINTS

She was seen at the gynaecology out patient with complaints of having been sexually assaulted by a person known to her a day prior to arriving in hospital.

HISTORY OF PRESENTING COMPLAINTS

She was at her workplace when the husband to her employer accosted her and raped her. He forcefully had intercourse with her. No condom was used during this episode and the attacker achieved penetration and ejaculation. She was threatened with being relieved of her job if she revealed the assault to anyone. She did not note any vaginal bleeding after the assault and had no loss of consciousness. She reported the matter a day later to a neighbour friend who took her to a clinic from where she was taken by Samaritans from a non-governmental organization. It was they who brought her to Kenyatta hospital after reporting the matter to the police. She had already taken a bath, douched the vagina and changed clothing by the time she arrived in hospital.

OBSTETRICS AND GYNAECOLOGY HISTORY

She attained menarche at 13 years. Her periods were regular, lasting three days every 21 days. She gave no history of dysmenorrhoea. She was para 0+0. Her last menstrual period was on 14/09/05. She had never used any contraceptives. She was sexually active, her last consensual intercourse being about one month prior to the assault.

PAST MEDICAL HISTORY

She had not been admitted nor had any surgeries previously. She did not have any known allergies.

FAMILY AND SOCIAL HISTORY

She was single and worked as a househelp in Ngumba estate in Nairobi. She was the first born in a family of four siblings. Her parents lived in Muranga. She had dropped out of

school after class 8 of primary school due to lack of money (school-fees). There were no chronic illnesses in her family. She did not drink alcohol nor smoke cigarettes.

PHYSICAL EXAMINATION

PW was a young lady in good condition, not pale, not jaundiced, and had no lymphadenopathy. Her pulse was 76/minute and regular, the respiratory rate was 16/minute. Her blood pressure was 110/70mmHg and temperature was 36.8°C. The cardiovascular, respiratory and nervous systems were normal.

ABDOMINAL EXAMINATION

The abdomen was soft, not distended and there were no areas of tenderness. There were no palpable masses.

PELVIC EXAMINATION

In the procedure room, she was put in lithotomy position. There was bruising on the inner thighs and at the introitus around the 5 o'clock position. Speculum examination revealed normal vaginal walls, smooth cervix. A high vaginal swab was taken for microscopy and culture. Betadine douche was then done.

IMPRESSION

Sexual assault

MANAGEMENT

PW was reassured and explained on the need for further laboratory examinations. Blood was drawn for investigations including those for hepatitis, syphilis and HIV. A urine specimen was taken for a pregnancy test and for microscopy and culture.
Prophylactic antibiotics were given (Norfloxacin 800mg stat, Doxycycline 100mg twice daily and metronidazole 400mg thrice daily for five days). She was started on Postinor II, 1 tablet immediately and a repeat 12 hours later for emergency contraception. She was to be started on antiretrovirals (Lamivudine150mg/zidovudine300mg combination 1 tablet twice daily). She was counseled and emotionally encourage by the counselor from the adolescent clinic.

LABORATORY RESULTS

1. VDRL – Negative
2. ELISA for HIV – Negative
3. HVS Culture – No growth obtained.
   -Wet preparation-no pus cell seen. No yeast cells or T. vaginalis seen.
   - No spermatozoa seen.
4. Pregnancy test – Negative
5. Urinalysis – PH 6.2, protein nil, sugar nil, no deposits. No organisms on microscopy and culture.
6. Hepatitis B surface antigen – Negative.

Follow up
She was seen in the out patient again 3 days later when the above results were available. She was well. Further counseling was given and was adviced to take the antiretrovirals (a supply of 28 days was issued) and to be seen in 3 months for repeat HIV and VDRL tests.

DISCUSSION

A case is presented of a 20 year old Para 0 + 0 who was sexually assaulted. She was started on prophylaxis against HIV, emergency contraception offered and counseling was initiated.

The patient presented was forced to have intercourse without her consent. Sexual assault may be defined as any sexual act performed by one person on another without the persons consent. Despite this, legal definitions vary between countries and states. Sexual assault includes genital, anal, or oral penetration by a part of the acccused's body or by an object. It may result from force, the threat of force either on the victim or the victim's inability to give appropriate consent. Sexual assault involves the penetration of the vulva or beyond by the male genitalia without consent of the woman or when she lacks the ability to consent owing to physical or mental incapacity. Statutory rape is coitus with a female below the age of consent (16 years) and constitutes an offence by itself. The laws of Kenya state that any person who unlawfully and carnally knows any girl under the age of 14 years is guilty of a felony and is liable to imprisonment with hard labour for fourteen years together with corporal punishment. Although most definitions centre on forced carnal knowledge without consent of a woman by a man, newer definitions are often gender neutral [1-4].

Our patient was raped, full penetration and ejaculation was achieved. The attacker was known to her being her male employer. It is estimated that 9-24 % of the women will be raped at least once in their lifetime. The incidence in Kenya is unknown. Hospital records are often underestimates as most are not reported. Recently the incidence has increased due to public awareness and women's rights being highlighted especially the crusade of violence against women. Sexual abuse is the most frequent form of child abuse. Specific groups of women are more vulnerable to abuse including the very young (13-24), the mentally and physically handicapped, women who live nearby the accused, unaccompanied, intoxicated women, small sized women and the elderly. The commonest reported age is 8-11 years. Approximately one half of all reported child sexual abuse is the result of incestuous relationships. Most abusers are male adults and if known to the victim is likely to be a member of the victims own childhood. If unknown, the abusers are likely to be involved in another form of criminal activity such as gangs during robbery. Our patient was 18 years and was raped by a known male adult.

Almost 50% of rapists are under the age of 25 years with 30% being 18-24 years. Most are repeat offenders and average more than 10 rapes before they are apprehended. Careful retrospective evaluation reveals emotional and sociopathic disturbances of rapists.

Motivation for the assault seems to be degradation, terrorization and humiliation of the victim rather than sexual gratification. The view is reinforced by the fact that most rapists who are unable to ejaculate during a sexual assault do so without difficulty during consensual sexual intercourse. The age of the attacker in our case was not known.

The term rape is derived from the Latin word "rapere" which means to steal, seize or carry away. Sexual abuse is one of the most pervasive of human right violations, denying women and girls' equality, security, dignity, self-worth and their right to enjoy fundamental freedoms. This violence is present in every country, cutting across boundaries of culture, class, education, income, ethnicity and age. Psychiatrists describe three types of rape:

- Power rape which is the commonest. Here the rapist seeks to demonstrate power through sexual assault rather than physical injury. The assailant usually has feelings of sexual inadequacy. This type accounts for about 50% of rapes.

- Anger rape. This accounts for 40% of the cases. The assailant is usually impulsive and episodic. The psychological objective is humiliation and degradation of the victim.

- Sadistic rape. Is rare, accounting for about 5% of rape. It is usually characterized by extensive violent injury that may result in death. These assaults are premeditated and may involve ritualistic torture of the victim.

This patient likely had power rape as the employer had all authority over her.

Rape victims are exposed to serious medical, physical and psychological consequences. Few of the victims have serious physical injuries but all victims suffer psychological trauma. After the assault, a "rape-trauma syndrome" often occurs. The immediate response (acute phase) may last for hours or days and is characterized by a distortion or paralysis of the individuals coping mechanisms. Signs may include generalized pain throughout the body, headache, chronic pelvic pain, physical symptoms such as vaginal discharge. The next phase (the delayed or organizational phase) is characterized by flashbacks, nightmares and phobias in addition to gynaecologic and menstrual complaints. This phase often occurs months or years after the event and may involve major life adjustments.

In the case presented, condoms were not used but investigations did not reveal any bacterial infection. Health consequences after sexual assault include physical health outcomes, injury from lacerations to fractures and internal organ injuries, unwanted pregnancies, gynaecological problems, sexually transmitted infections and even HIV/AIDS. 3% of rape victims develop sexually transmitted infections. Condoms are rarely used during non-consented intercourse. These were not used in the case of PW. Child victims of sexual abuse may present with physical findings that can include anogenital problems, enuresis and behavioral changes such as sexual acting out, aggression and depression.

Rape victims may be too emotionally traumatized to wish to report the crime at the time they present to the hospital. The role of the physician is both medical and legal. Provision of a quiet private area equipped for pelvic examination is necessary. Informed consent must be obtained before examination of a sexual assault victim as it helps the victim participate in regaining control of her body and her life. General physical examination is necessary since 75% of sexually abused people, especially children show evidence of other abuse. Bite marks should be looked for in all regions of the body, particularly around the genitalia and breasts. Occasionally foreign objects may be found in the vagina, urethra or rectum. If oral penetration has occurred, injuries to the mouth and pharynx may be noted [2, 3,5].

The extent of injuries in our patient was assessed by visual inspection. The patient presented had already changed clothing but had minimal injuries on physical examination. Underwear should be removed carefully and kept in a plastic bag for examination for semen, blood or pubic hair of possible assailants. Pelvic examination is done with a Pederson's speculum. If necessary, examination can be done under anaesthesia. Evidence of trauma, dilatation or disruption of tissues is sought and documented. Accurate measurements of the genital anatomy and injuries are necessary. This is best achieved by use of a colposcope.

Sterile swabs were collected at the time our patient had a pelvic examination.Sterile swabs are used to collect materials from the vulva, vaginal walls and the cervical portio. The swabs are examined for the presence of semen and infections such as gonorrhoea. Blood is drawn for VDRL test for syphilis and ELISA for HIV and hepatitis. Urine test for pregnancy should also be done. All these were negative in our patient. Spermatozoa were not seen likely due to her having douched the vagina upon the assault. The tests should be repeated after three months of the assault to rule out infection [2,3,5].

After complete examination, methods of prevention of venereal diseases and pregnancy should be offered to the patient including emergency (post coital) contraception and post exposure prophylaxis for HIV infection. Immunisation against hepatitis is also offered. Our patient was covered with broad spectrum antibiotics, postinor for emergency contraception and went to a centre that would offer her anti-retrovirals at no cost. Continuous psychological support should be offered[7]. Our patient was referred to the patient support centre for follow up.

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GYNAECOLOGY LONG COMMENTARY

KNOWLEDGE ON REPRODUCTIVE PHYSIOLOGY,

CONTRACEPTION AND STI's/HIV/AIDS

AMONG RURAL TEENAGE

SCHOOL GIRLS IN NYAHURURU REGION

ABSTRACT

Background

Adolescent sexual and reproductive health has not been considered a major health priority as they suffer less morbidity than other age groups. With increase in urbanisation, there is a need to increase their awareness to be able to cope with the changes occurring in their bodies and environment around them and so that they can make informed choices in their future lives.

Study objectives

This study was to assess the adolescents' knowledge on basic aspects of reproductive physiology, and their awareness of the existence of modern contraception. It was also aimed to assess their knowledge on sexually transmitted infections(STI'S) especially HIV/AIDS among rural teenage schoolgirls in Nyahururu region.

Study setting

The study was carried out in Rumuruti/Nyahururu divisions between March and June 2005. It involved in-school teenage girls in five secondary schools in this region.

Study design and methodology

This was a prospective descriptive study in which 380 teenage girls from selected secondary schools in Nyahururu were interviewed.

Main outcomes measures

The girls knowledge on reproductive physiology and contraception was assessed. Their knowledge on sexually transmitted infections including HIV/AIDS was to be assessed.

Results

A total of 380 girls were interviewed during the study period. The mean age of the girls was 16.5 years. Majority(69.5%) of them lived with both parents. Over half (58.2%) of them reported the school teacher or their mother as the most important source of information on puberty and reproduction and 64.7% felt they were adequately informed about menses by the time they attained menarche. The most commonly heard of contraceptives were condoms (81.1%), the injection (by 60.5%) and pills (by 52.1%).The least heard of methods was implants (10.1%).Commonly known STI's were HIV (by 93.7%), gonorrhoea (84.7%) and syphilis (80.8%). Least heard of among the STI's were herpes (15.3%) and genital warts (6.6%).The most frequently known STI health effect was pain during urination by 65.8% of the girls. Over 90% understand transmission methods, associated risky behaviours leading to infection and infection prevention methods of HIV.

Conclusions

Knowledge on reproductive physiology and relationship of the menstrual cycle to pregnancy timing was average (68%). Knowledge of contraceptives was poor. Most known STI's were HIV, gonorrhoea and syphilis. The girls did not demonstrate extensive knowledge of the health effects of sexually transmitted infections. The girls demonstrated good knowledge of HIV, its transmission modes, risky behaviours and prevention methods.

Recommendations

There is a need to provide adolescents with information on reproductive physiology, contraception and STI'S to enable them make informed choices and to develop responsible patterns of sexual and reproductive behaviour.

INTRODUCTION

Kenya, like most other developing countries in Sub – Saharan Africa and the world over is striving to educate the majority of its young generation. Majority of secondary school girls are in their adolescent age. Adolescent sexual and reproductive health is very important, as what happens to these girls in this period often will endure into later life. Despite their numbers (they form a major group in developing countries), they have not traditionally been considered a health priority since they have lower morbidity and mortality than the older and younger groups. But in areas such as sexual health, they suffer disproportionately. Consequences of poor health at this age will stretch into the future, affecting their prospects and those of their children. Pregnancy and childbirth in adolescence carry major risks causing many deaths. Further, early pregnancy inhibits the young woman's chances of obtaining an education and may diminish their social status. Consequently most births to adolescents are unwanted and mistimed, a large proportion ending in unsafe abortion.

Adolescence is a period describing an age range from childhood into adulthood. It also describes a distinct period of life, characterised by changing roles and increased freedom from a state of total socio-economic dependence to one of relative independence.

As the world changes under the effects of globalisation traditional ways of life are breaking down, resulting in increased urbanisation, migratory working, and changing opportunities for education and employment. Coupled with the influence of media and

peers, these factors are shaping the adolescent period, often leading to greater sexual freedom. Thus sexually transmitted infections pose a serious threat to adolescent health. As men continue choosing younger sexual partners, infection rates for HIV/AIDS and other STD's keep rising among adolescent girls. The young people's ability to deal with these negative consequences of adolescent sexuality is severely constrained by inadequate information and poor access to education and health services.

Traditional values continue playing a part in shaping adolescents. Cultural expectations and gender roles determine many of the choices young people make. For example, in macho societies young men sometimes have difficulty in responding to information about sex and women lack the status to refuse sex.

Adolescents clearly have little choice relating to their sexual and reproductive health, lacking the information, skills and services necessary to make informed decisions. The adolescent period offers enormous opportunities to adjust health-related behaviours before they become established. However, to do so requires overcoming barriers at several levels.

Accurate relevant information is an obvious requirement for making decisions.

Life-skills properly targeted to the needs of young people will help establish safer sexual behaviour. Schools provide a good opportunity for this. Education increases young women's status and economic opportunities thus eliminating exploitation due to low socio-economic status.

Adolescent sexual health needs to be taken as a priority at all levels of policy making.

This study aimed at adding to the information on adolescents' knowledge of sexual and reproductive health that may be necessary for formulating future policies.

LITERATURE REVIEW

The Kenyan population pyramid has a broad base thus a majority of the population are young; up to 50 percent being at or below the age of 20years [1]. Those between 15-19 years constitute about 23 percent while the 20-24 age group is about 20 percent. The total fertility rate (TFR) is 5.0 and this is higher in the rural (5.6) than in urban areas (3.3) [2].

The age specific fertility is highest between 20-30 years and also between 15-19 years [3]. The figures are higher for rural than urban populations e.g., for the 20-24 age group, the TFR for rural girls is 280 per 1000 compared to 161 per 1000 women in urban areas. Figures for the 15-19 age group are 123 per 1000 and 85 per 1000 for rural and urban areas respectively. Contraceptive prevalence has been low approximately 7.5 in 1977 [1], 14.0 in 1984, 17.0 in 1989 [2], 15.2 percent in 1998 [2] for those aged 15-19 years.

The World Health Organization (WHO) considers the health of young people a priority. Four fifths of the youth in the world live in developing countries [4]. Adolescence is the transitional period separating childhood and adulthood. According to W.H.O adolescents are individuals aged 10-19 years and over one billion people in the world are in this age group, accounting for almost 30 percent of the world's total population[5,6]. It is projected that by the year 2025 their numbers will reach nearly two billion. Approximately 15 million young women aged 15-19 years give birth every year, accounting for over 10 percent of all births worldwide. In the least developed countries this age group accounts for 17 percent of all births [5,6].

The adolescent population in Kenya in 1985 was approximately 3.9 million. Currently the 15-19 age group makes up about 23 percent of the population (6.9 million), doubling compared to that in 1985[3]. As in the rest of Africa, adolescents constitute an important segment of the population. In Africa, adolescents constitute approximately 40 percent of people aged 15 – 49 years and they contribute to 10 percent of Africa's overall fertility. Very few of African countries have comprehensive policies that deal with issues related to adolescent sexual and reproductive health [4,7].

In addition to describing an age range, adolescence also describes a distinct period of life characterised by changing roles and increased freedom. The adolescents are reacting to changes within their bodies as well as to social changes within their environments. Hormone changes characteristic of the onset of adolescence lead to both physical and emotional awareness that lead to changes in personality. For the first time the individual is seen not only as a person but also as a sexual being [7]. The adolescent period offers enormous opportunities to adjust health-related behaviours before they become established. Traditional ways of life are breaking down, resulting in increased urbanisation and migratory working. Many adolescents are experiencing social turmoil

resulting from conflicting values as the country becomes more urbanised and industrialised. Coupled with the influence of the media and peers, these factors are shaping the adolescent period, often leading to greater sexual freedom. Parents, religious leaders and schoolteachers shy away from guiding the adolescent on sexuality issues as they are not or feel not adequately equipped to tackle various issues related to adolescent sexual and reproductive health (ASRH) [8].

Several studies have been conducted on adolescents in Kenya and other countries all over the world. Sexual activity begins at a younger age, with both men and women starting sexual activity at a younger age [2,3]. In most instances, the adolescents beginning sexual relations choose not to or lack people to consult. They receive little if any formal sex education [9]. Premarital sexual activity is common among adolescents. This is more in the developed countries and in Africa and the Caribbean than in Latin America, Asia or the Middle East. Between 26 and 60 percent of unmarried teens aged 15-19 in Ghana, Kenya, Liberia, and Togo report being sexually active [2,10]. In Kenya, Lema (1987) [10] found a mean age at first coitus of 15 years with the youngest being 5 years and the peak age at 16 years. According to the recent Kenya demographic and health survey (KDHS 2003) the mean age of starting sexual activity is 16.7 years [3]. Majority (72.6 percent) of all sexually experienced girls had started coitus between 14-17 years. Maggwa (1988) [11] in a study among rural girls found an exposure rate of 42 percent among them compared to 24 percent among the urban as found by Lema. Magoha (1992) [12] found mean age at first coitus of 16.4 years. In a study by the Kaiser foundation in the USA (1996), 29 per cent of teenagers between 12 – 18 years had had sex[13]. Between 20 and 36 percent of adolescents 15 – 19 years in aforementioned African countries were married thus also exposed to the risks of early child bearing. Obongo (1989) found 37.6 percent of girls had had sex by age 15 years [14] while Agullo (1999) [15]found similar figures – 37 percent – among teenage rural girls.

Menarche is occuring at an earlier age than in previous years. This has been attributed to better nutrition and quicker accumulation of body fat [16]. This decrease has occurred gradually over the last half century. Menarche has receded from the age of 16 to 17 years in the nineteenth century to the average of 12 – 14 years currently [10,17,18]. In Lema's study

the lowest age at menarche was 10 years. Many (75.2 percent) of all the sexually active had attained menarche between 13 and 15 years, 62.3 percent had started sex within 1 – 2 years of menarche. Wangwe (1995) [19] found similar figures with a median age of attaining menarche of 14.1 years, 63 percent of the sexually active in that study had their first experience by age 15 and 28.8 percent by age 14. Muroki (1987) [20] in a study among rural girls found a mean age at menarche of 14. 3 +/- 1.4 years whereas Bwibo et al (1982) [21] had found mean age at menarche of 13.2 +/- 2.3 years and 15.3 +/-2.2 years in studies done in Nairobi and Kitui respectively. The difference in the two places was attributed to the inverse relationship between age at menarche and socio-economic status.

Discussion of adolescent sexuality and reproductive health provokes a lot of controversy. Whether sex education should be introduced in schools is a topic that is still charged with emotions in many societies, yet studies have indicated that this group is sexually active and has little knowledge about their reproductive physiology and even fewer discuss any sexuality issues with their parents [10,22]. The erosion of traditional values and means of regulating adolescent sexuality has resulted in lack of appropriate guidance to the adolescent who is left groping in the mirk and mirth of Westernisation that is in the society. Increased westernisation and urbanisation of our society has led to the break – up of extended families and abandoning of many traditional cultural norms and practices, which has led to increased sexual activity among adolescence. Previously uncles, aunts, grandparents were responsible for sex education and supervision of adolescents' transition from childhood to adulthood. Life cycle changes were marked with celebrations and ceremonies with corresponding rewards as well as punishment for those who did not conform to the social norms. A period of seclusion with a selected tutor explained their role in society to them and taught them about sexual behaviour and pregnancy. This conferred peer – group identity and promoted a social and personal sense of belonging. For most, adolescence commenced with circumcision, which was a rite of passage. Many African cultures had attributes that did not place sexual behaviour at the centre of the moral or social systems. For example, the Kikuyu allowed unmarried youth to engage in a non – penetrative form of sex called *Ngwiko*. The Akamba in contrast allowed premarital sex for both boys and girls after circumcision. Among the Egoji clan of Meru young people were taught about periodic abstinence, non-penetrative sex and

"the wrong time of the moon" when a girl might get pregnant. However some ethnic groups were highly restrictive, especially of females. The Luo, for example, expected proof of female virginity at marriage demonstrated by some amount of blood on the consummation bed. The Maragoli ululated for a girl's virginity at marriage but tolerated prepubertal sexual activity among boys.The Maasai did not tolerate girls to have premarital sex in any form [23].

The consequences of this current new found freedom for the adolescent girl becomes a national problem as it results in unwanted and mistimed pregnancies. In many developing countries a large proportion of young women become pregnant during adolescence. Pregnancy and childbirth at this age carry major risks and according to WHO constitute the main cause of death in 15-19 years old girls worldwide, with about 60,000 young women dying each year [24]. Furthermore, early pregnancy inhibits young women's chances of obtaining an education and may diminish their social status. Most are forced to drop out of school. Most births to both married and unmarried adolescents are mistimed [25]. In the 1998 KDHS, 3.5 percent of girls 15-19 years were currently pregnant and 17.3 percent were already mothers [2]. The numbers are higher for rural than urban populations (3.8 versus 2.7 percent for those currently pregnant). Antenatal and obstetric complications are more prevalent among teenage mothers due to unfavourable socio – economic factors and lack of adequate prenatal care and health facilities [8,26]. Girls aged between 15-19 are up to twice as likely to die during pregnancy or delivery as women aged 20-34 [27], and those under 15 are five times as likely to die [28]. In Nairobi, the incidence of teenage pregnancy was 18.6 percent, the youngest mother being 10 years and two months [29].

A large proportion of these unwanted pregnancies end up in unsafe abortions because even where it is legal, poor access and high cost affects adolescents disproportionately. Induced abortion in Africa accounts for 20 percent of all maternal mortality. Hospital statistics from cities in Liberia, Mali, Nigeria, Ghana and Zaire show that 38 to 68 percent of women hospitalised with abortion complication were 19 years or younger [19-21,30]. Each year girls aged 15-19 undergo at least 5 million induced abortions [31]. Since most are unsafe, adolescent girls therefore suffer a significant and disproportionate share

of death and disability from unsafe abortion [25]. Most of the adolescents are unaware of ways of getting rid of unplanned pregnancy and carry the pregnancy to term with all their unpreparedness. In countries like Kenya where termination is illegal and a subject that provokes huge emotions, the pregnant adolescent has to opt between two grim choices: childbirth or illegal abortion [32]. At the KNH in Nairobi, Aggarwal et al (1982) [33] found that 28 percent of abortion patients in their study were aged less than 20 years. They also found 62.3 percent of the total number had interfered with their pregnancies and of these 79 percent were single girls. Unwanted pregnancy is a major reason for illicit abortion. Rogo estimated that about 1.5 million induced abortions occur in Africa alone [9]. Muliro [34] (2003) found 64 percent of women freely volunteering information of having procured abortion. In that study comparing 1997 and 2002, women aged 15-24 years comprised 70 percent of women presenting with induced abortion in 2002 whereas in 1997 they made up less than 50 percent.

Sexually transmitted infections are a consequence of unprotected sexual activity. These pose a major problem among the adolescents. According to sessional paper IV (1997) of the Kenya Government, the highest AIDS infection is among women aged 20-24 years. Those between 15-19 years are twice as likely to be infected as males in the same age group (probably due to having older partners). About 18 per cent of women are infected within two years of becoming sexually active [35]. According to the 1998 KDHS, sexual behaviour of teens puts them at risk of HIV. Up to 25 percent of teens lose their virginity because they are forced in unwilling encounters thus higher risk of infection for girls – due to abrasion of their developing vaginal mucosa and cuts making viral entry easy. Condom use is unlikely in these situations where force is used. Infection with other sexually transmitted infections is also easy during these encounters. The case of HIV powerfully illustrates the serious threat that sexually transmitted infections pose to adolescent health. It is estimated that more than 7000 young people become infected with HIV every day accounting for at least half of all new cases of infection[36]. Early initiation of sexual intercourse has been shown to consequently lead to a higher prevalence of carcinoma of the cervix, tubal infertility compared to those starting late. It is difficult to change sex behaviour pattern once it has become a habit. Thus it is necessary to equip the adolescent with knowledge, skills and attitudes that keep them safe from infection before

they are or become sexually active. In the Kenya Government sessional paper on AIDS [35], the government commits itself to protect young people, integrate HIV/AIDS education in existing curricula in schools and counselling by churches. In the schools studied, HIV/AIDS education was not included in the teaching curriculum.

While sexual activity is high, knowledge and use of contraceptives has been shown to be low [2,10,11,19-22]. Sanghvi (1983) found that only 2.2 percent of adolescents delivering in Nairobi had used any method of contraception [32]. Lema (1987) found that of all the sexually active girls in his study only 9.5 percent were using or had used any method at all – mainly withdrawal and safe days. Maggwa [11] (1985) found a contraceptive prevalence rate of 2.2 percent. Wangwe (1995) found only 10.7 percent of the sexually active had ever used a contraceptive method [22]. This low use of contraceptive which is evident in the whole of Sub – Saharan Africa is equally low among married adolescent, for example, percentages of married teens who have ever used contraception range from a high of 13 percent in Ghana to lows of 1 percent in Benin and Nigeria [21]. The available clinics are also not adolescent "user friendly". Some religions are against provision of this service to the adolescents. Sex education without provision of contraception increases conception rates. Parents, teachers and providers believe that sexual activity among adolescents is wrong or inappropriate and are therefore reluctant to offer counsel on such matters [8]. Regular use among adolescents of contraceptives is inhibited by their sporadic and unplanned nature and circumstances of their sexual activity. This is particularly true at the first coitus [8]. They lack and have title choice relating to their sexual and reproductive health, lacking the information and services necessary to make informed decisions.

Adolescents need to know the facts about their reproductive physiology, sexual development, planned parenthood, sexual relations, pregnancy and contraception. They need to know much more about transmission and prevention of STI's especially HIV/AIDS. They will then be able to make informed decisions and have bargaining power to make use of available options. These life skills can only be inculcated in the adolescents before they are sexually active. Accurate, relevant information will help them establish safer sexual behaviours. It was the aim of this study to provide additional baseline data on information the adolescents already have so that future policies can be

more oriented to improve on what they know and correct the misinformation that may be present among them.

STUDY JUSTIFICATION

It has been shown that the age of initiating sexual activity is low[10-12]. A confluence of social prejudice, repugnant cultural rites, and the grinding poverty keep pushing girls out of school making them a very vulnerable group. Beyond this, most girls get exposed to HIV/AIDS and other S.T.I's. Moreover there is the agony of having to bear children and possible death at childbirth or procuring illicit abortions.

It is difficult to change sex behaviour pattern once it becomes a habit, thus the teenager should be equipped with knowledge, skill and attitudes that keep them safe from infection before they are sexually active.

This study aimed to provide a baseline survey on young girls in a rural area assessing their knowledge on sexual and reproductive health. It aimed to seek their knowledge on S.T.I's especially HIV/AIDS, an area that other studies have not touched on. Previous studies have mainly dealt with attitudes and perceptions of adolescents to sex and contraception. One of the millennium development goals is to combat, halt and begin to reverse the spread and prevalence of HIV/AIDS among adolescents and young women by 2015. We need to have more data on their knowledge of this epidemic upon which we can then formulate further policies to help achieve the goals thus the need for this survey.

STUDY OBJECTIVES

1. **Broad objectives**

(i) To determine the knowledge of basic aspects of reproductive biology among adolescent secondary school girls.

(ii) To survey their awareness of the existence of various types of contraceptive methods.

(iii) To determine the knowledge of sexually transmitted infections among adolescent secondary school girls.

2. **Specific objectives**

(i) To determine information on menstruation and fertility.

(ii) To determine awareness and use of modern methods of contraception.

(iii) To determine their awareness of transmission and assess awareness of prevention methods of STI,s and HIV/AIDS.

(iv) To determine their knowledge on STIS /HIV/AIDS.

METHODS AND MATERIALS

1. STUDY DESIGN

This was a descriptive cross – sectional survey

2. STUDY AREA AND POPULATION

The study was conducted in the Nyahururu region. This area is partly in Laikipia and Nyandarua districts in Kenya. This is a rural region around 200-kilometre North West of Nairobi. Most families here reside in their farms. Both districts cover approximately 12,500 square kilometres in surface area and have a population of around 802,089 people [37]. It is a multiethnic region. Those studied were adolescent girls in five secondary schools Thiru secondary, Gatero girls school, Rumuruti secondary, Marmanet secondary and Gituamba secondary school whose head teachers allowed entry for the study to be carried out. Two of the schools were girls only boarding schools while the others were mixed day schools. Girls were chosen as it is they who suffer more from the consequences of lack of knowledge due to cultural, socio-economic disadvantages and also the effects of unwanted pregnancies.

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3. STUDY PERIOD

The study was conducted between March and June 2005.

4. SAMPLING

All those meeting the inclusion criteria in the selected schools were enrolled into the study.

The sample size was calculated from the formula.

$$N = \frac{Z^2 P. (1-P)}{d^2}$$

Where Z – standard Normal deviate (1.96)

P – Estimated prevalence reported of sexual activity among schoolgirls

d – Level of precision with which to estimate P

N – Desired minimum sample size

Thus P = 38%, taken as the mean prevalence of sexual activity in schoolgirls in the studies done by Maggwa [11], Agullo [17], Obongo [16] and AMREF [22].

d – Degree of accuracy with which P is determined taken as 0.05 (5 percent)

Therefore $N = \frac{1.96 \text{ X } 1.96 \text{ X } 0.38 \text{ X } 0.62}{0.05 \text{ X } 0.05}$

$= 362$

A total of 380 girls were interviewed. Simple random sampling was used to select the schools and girls that were interviewed. The limitation was that there was likelihood of over presentation when considering the level of education reached.

Schools selected were easily accessible and were at least 20 kilometres of each other. Two of the schools were girls only schools and the rest were mixed schools.

5. INCLUSION CRITERIA

(i) All teenage (13-19) girls in selected secondary schools who voluntarily accepted to participate in the study.

6. EXCLUSION CRITERIA

(i) Those who refused to participate in the study after explanation of the aims of the study. Fortunately for this study, none of the girls requested to participate declined to be included in the study.

(ii) Teenage girls who were not formally in school.

7. ETHICAL CONSIDERATION

There were no major ethical issues involved in the study as no invasive procedures or physical examinations were to be done. However permission was sought from responsible authorities before commencing the study. These included:

a) Ethical and Research committee at K.N.H.

b) Research/Ethical committee of the Ministry of Education, Science and Technology.

c) Head teachers of schools participating in the study gave verbal consent for entry into their schools.

Issues of voluntary participation and confidentiality were explained to the participants and adhered to. The names of participants were not included in the data collection tool and confidentiality was guaranteed. There was to be no victimisation and disclosure of whatever information they had given. Codes rather than actual names were used for each of them.

8. CONSENT

Consent was sought voluntarily from all girls participating in the study. Verbal consent was taken voluntarily from all girls who participated in the study. The adolescent girls are generally considered minors and may not give express consent by themselves. Consent in this case was sought from their guardians, the Ministry of Education and school headteachers as the study was conducted in school settings. The participants were assured of confidentiality, anonymity and voluntary participation.

9. CONSTRAINTS

Failure by the girls to volunteer correct information due to fear of victimisation. This was overcome by the investigator spending time with them explaining the need for the study, and the fact that anonymity was guaranteed. Teachers were not allowed near the area where questionnaires were being filled except for introducing the principal investigator and his team.

10. DATA COLLECTION

The principal investigator and selected assistants administered an anonymous pre-tested questionnaire. Pre-testing was done in Nairobi (Moi Nairobi girls school) All questions were in English – the language medium of instruction in all the schools.

For each particular school, the questionnaires were filled on the same day.

The assistants were trained to familiarize them on how the survey was to be conducted, discussion on survey objectives and the questionnaire.

The questionnaire consisted of four main sections.

A – General information on their socio demographic backgrounds.

B - This included sexual and reproductive health information, physical maturation, and biological aspects of sexual and reproductive systems, their sources of knowledge and preferred sources of knowledge on above issues.

C - This section sought to know the respondents' awareness of contraceptives and sources of the knowledge on contraceptives.

D - This section was to assess their knowledge of STI's especially HIV/AIDS.

11. **DATA MANAGEMENT**

Completed questionnaires were checked for completeness. All cleared questionnaires were entered into a computer database using SSPS software. Comparison of qualitative variables was done using chi – square test statistics, while the student's t-test and analysis of variance were used to compare the quantitative variables. Data analysis was conducted in SPSS for windows.

RESULTS

A total of 380 teenagers were interviewed. They were all included in the analysis.
The age distribution ranged from 13 to 19 years with a mean of 16.5 years. This is shown in figure 1.

Figure I – Bar graph showing age distribution

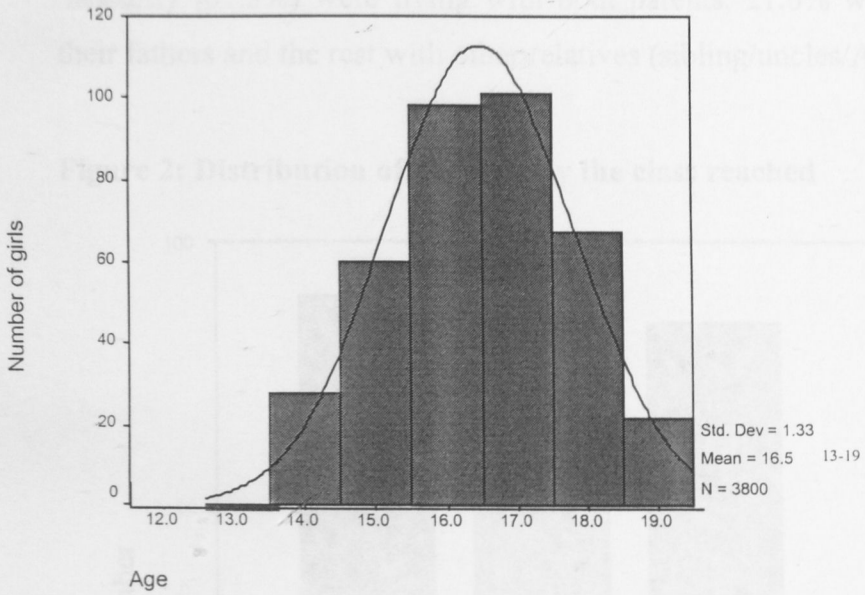


Table 1 shows the religious affiliations of the teenagers.

Table 1 Distribution of the girls by religious affiliation

Religion	frequency	%
Catholic	141	37.1
Protestant	230	60.5
Muslim	5	1.3
Other/None	4	1.1

Protestants formed 60.5 of the study group, 37.1% were Catholics, 1.3% were Muslims and other religions accounted for only 1.1%. Only 2 of the students were married at the time of the study.

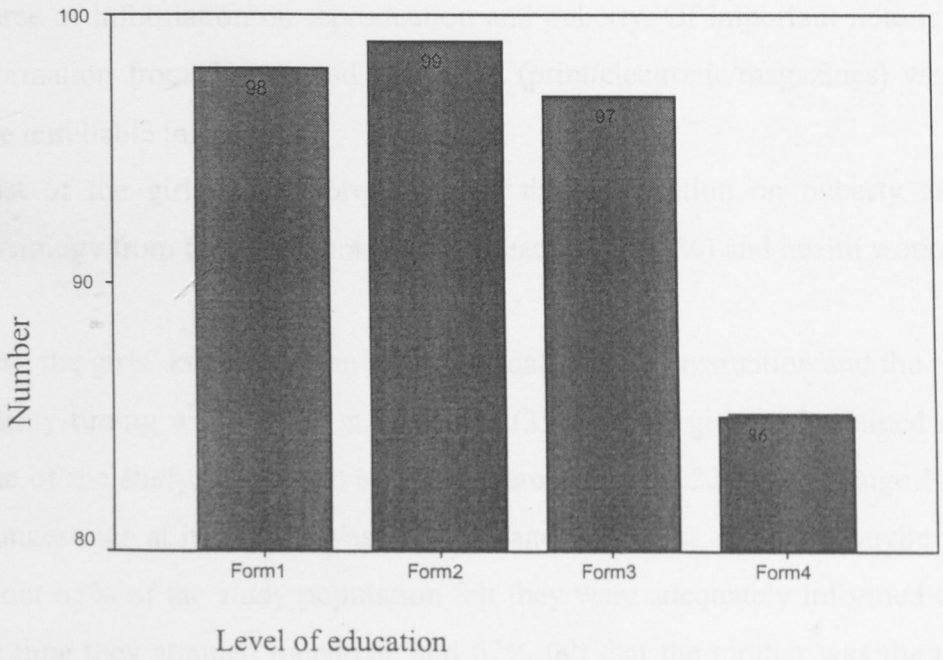
Table 2 shows with whom the students were living with as their primary care givers.

Table 2 Distribution of the girls according to the people they were living with.

Lives with	Frequency	%
Father	5	1.1
Mother	82	21.6
Father and Mother	264	69.5
Brother or Sister	10	2.6
Uncle/Aunt	5	1.3
Grandparent	14	3.7

Majority (69.5%) were living with both parents, 21.6% with their mother, 1.1% with their fathers and the rest with other relatives (sibling/uncles/Aunts or grandparents).

Figure 2: Distribution of the girls by the class reached



The teenagers' level of education at the time of the study is shown in Figure 2. It was nearly uniformly distributed in all classes of secondary school with 25.8% in form I, 26.1% in Form II, 25.5% in form III and 22.6% in Form IV.

Table 3 shows the important sources of information on reproductive biology.

Table 3: The source of information on reproductive biology

Source	frequency	%
School teacher	120	31.6
Mother	101	26.6
Father	3	0.8
Sister	16	4.2
Friends	32	8.4
Other family members	5	1.3
Doctors	6	1.6
Books/Magazines	74	19.5
Films/Movies	2	0.3
Radio/TV	6	1.6
Religious leaders	15	3.9

Most of the girls (58.2%) gave the schoolteacher or their mothers as the most important source of information on reproduction and puberty. Of important note is that 29.8% get information from friends and the media (print/electronic/magazines) who may at times give unreliable information.

Most of the girls would prefer to get the information on puberty and reproductive physiology from their mothers (33.9%), teachers (22.1%) and health workers (16.8%).

Next, the girls' knowledge on the menstrual cycle, menstruation and the relationship with fertility timing was looked at. Majority (354) of the girls had attained menarche at the time of the study. The mean age at menarche was 14.22 years (Range 10-19 years). The youngest age at menarche was 10 years and the oldest age at menarche was at 19 years. About 65% of the study population felt they were adequately informed about menses by the time they attained menarche and 67% felt that the mother was the most appropriate person to prepare a girl for her first menstrual period.

Table 4 overleaf shows the teenage girls knowledge on menstruation.

Table 4: Knowledge on Menstruation

	Correct knowledge n	%N
Source of menstrual period	98	25.8
Menstruation is normal in women	357	93.9
Menstruation occurs once a month in normal women	319	83.9
Menstruation is a sign of beginning of fertility in women	299	78.7
The menstrual cycle lasts for 28 days in every woman	211	55.5
Women always bleed for 5 days	268	70.5

Only 25.8% of the girls could correctly identify the uterus as the source of menstrual blood. Majority mentioned the ovaries (48.4%) as its source, 15.8% did not know and the rest 10% reported the vagina or fallopian tubes as the source.

In general, knowledge on menstruation was good. When given a score of 1 for correct response and 0 for incorrect response, the average percent score for knowledge on menstruation was 68.2% (Range 0-85.7%). Many (78.7%) of the girls correctly said that menstruation is a sign of the beginning of fertility in normal women. About 44.5% of the girls thought that the menstrual cycle lasts for 28 days in every woman while.

Table 5 shows the results of the girls' knowledge on reproductive physiology.

Table 5: Knowledge on reproductive physiology

	Correct knowledge(n)	%N
A woman can get pregnant on the 1st sexual intercourse	210	55.3
A woman stops growing after having sex for the first time	262	68.9
Masturbation causes serious damage to health	44	11.6
A woman can get pregnant if she has sexual intercourse in between (midpoint) her periods	178	46.8
If a man does not push his penis deep in to the vagina, pregnancy will not occur.	161	42.4
Having sex once a while does not result in pregnancy	246	64.7
A woman can become pregnant if she has sex during her periods	124	32.6
Boys between 13-15 cannot impregnate girls	263	69.2
A woman can become pregnant if she has sex before her periods begins (3 days)	94	24.7
A woman is safe from pregnancy a week after periods (mid point of cycle)	85	22.4
Pregnancy can only occur if a woman has sex with a man	242	63.7

Over half the girls know that a girl can get pregnant even on the first intercourse. Combining the percentages of those who gave the wrong response and those who did not know, 77.6% of the girls did not know that it was unsafe to engage in sex at the mid point of a woman's menstrual cycle.

Only 32.6% of girls knew that pregnancy would not occur if a woman has sexual intercourse during her periods.

Questions were also asked about what the girls knew of the sexual maturation of boys. About 56.8% girls said that wet dreams are a sign, that a boy has started producing sperms and 69.2% girls know that boys of between 13-15 years can make girls pregnant.

Only 16.6% (63) of the girls had ever had sex at the time of the study. The mean age at first sexual debut was 14.4 years. The youngest sexual debut age was 9 years. The range of coitarche was 9-18 years. In the 2 months preceding the study, 65.1% of the sexually active had not had coitus. The rest 34.9% had been active with 20.6% having had coitus in the month prior to the study. About 70% had only one partner over the last year, 12.7% had had two partners and 1.5% had three or more sexual partners in the last one year. In 58.7% of the episodes, the sexual encounters were unplanned and 65% (41) of the sexually active said they did something to avoid getting pregnant. Only 25.3% of the sexually active use a method always to avoid pregnancy while 39.6% use a method sometimes.

Seven of the study population had ever been pregnant, this being 1.8% of the girls. None was pregnant at the time of the study. Of the 7 girls, 1 had been pregnant twice, 5 had been pregnant once and 1 declined to say how many times she had been pregnant. The mean age at first pregnancy was 15.7 years, the range being 12-18. Of the 6 reported pregnancies, 2 procured an abortion while 4 ended in live births.

Table 6: Source of pressure to engage in sexual activity n=130

	Response	Frequency	%N
Has pressure from others to engage in sex	Yes	130	34.2
Magnitude of pressure	Great deal	71	18.7
	A little	59	15.5
Source of pressure	Partner/Friends	85	22.4
	Relatives	12	3.2
	Radio/TV	33	8.7

Of note is that 34.2% of the girls feel they have pressure from others to have sex. About 18.7% felt they were under a great deal of pressure to indulge in sexual activity. Most of the pressure was from friends and the girls' boyfriends while 8.7% felt the pressure was through electronic media.

From the foregoing, it was thus necessary to assess the girls' knowledge on contraception.Table 7 shows percentages of those who had ever heard of various modern methods of contraception.

Table 7: Contraceptive methods ever heard of among the girls

Contraceptives	Yes	%
Oral pills	198	52.1
Condoms	308	81.1
Spermicide	43	11.3
Coil (IUCD)	120	31.6
Injections	230	60.5
Abstinence	139	36.6
Calendar	149	39.2
Morning after pill/Postinor	87	22.9
Withdrawal	68	17.9
Implants - Norplant	41	10.8
Female/Male sterilization	119	31.3
Other(herbs)	7	1.8

The most commonly heard of methods were condoms (81.1%), injection (60.5%) and oral pills (52.1%). Least heard of methods included implants/Norplant (10.8%) spermicides (11.3%) and the withdrawal method (17.9% of the girls).

The school teacher is the source of information on contraception in 35.5% of cases, while other information sources are print and electronic media (34.2%), and the mother-31.1%.The preferred source of information is the mother 36.8%, health workers (31.6%) school teacher 23-9% and the media 17.1%.

Table 8: Knowledge on contraceptives

	Correct knowledge-n	%N
Contraceptives should only be used by women	101	26.6
Family planning means spacing the number of children one has	266	70
Contraception means taking pills only	188	49.5
Contraceptives for men are not available	116	30.5
Using contraceptives leads to having no children in future	86	22.6
Contraceptives interfere with sexual pleasure	65	17.1
Contraceptives increase premarital sex	31	8.2
Contraceptives are not dangerous to the health of women	37	9.7
Condoms are effective in preventing pregnancy	137	36.1
Condoms can be used more than once	236	62.1

In general, knowledge on contraception was poor among these girls.

Knowledge of sexually transmitted infections (STI's) was also looked into. The results are shown in table 9.

Table 9: STI's ever heard of among the girls

STI	Yes	%
Herpes simplex	58	15.3
Gonorrhoea	322	84.7
Warts	25	6.6
Syphilis	307	80.8
Hepatitis	87	22.9
HIV/AIDS	358	94.2
Trichomoniasis	47	12.4
Candidiasis	19	5.0
Pubic lice	35	9.2
Scabies	99	26.1
Chancroid (ulcer)	39	10.3

Many more of the girls had heard of HIV/AIDS (93.7%), gonorrhoea (84.7%) and syphilis (80.8%) as compared to other STI's such as Herpes simplex (15-3%), warts (6.6%), Hepatitis (22.9%) and trichomoniasis (12.4%).

The girls' knowledge of the health effects of STI's is shown in table 10.

Table 10: Knowledge of health effects of STI's

Health effects	n(Yes)	%N
Vaginal discharge	215	56.6
Pain during urination	250	65.8
Weight loss	138	36.3
Rash	140	36.8
Ulcer/Sore in genital area	145	38.2
Infertility	118	31.1
Pain during intercourse	155	40.8
Still birth/ Miscarriage	113	29.7
Abdominal pain	150	39.5
Swollen glands in the groin	164	43.2

The most frequently known STI health effect was pain during urination (65.8%), vaginal discharge (56.6%), swollen groin glands (43.2%) and painful intercourse (40.8%). Overall, the respondents did not demonstrate very extensive knowledge of the health effects of STI's.

In this study, the girls were asked whether they knew where one would go for STI treatment. The various places mentioned are as listed in table 11.

Table 11: Knowledge of sources of treatment for STI's

Source	N(Yes)	%
Shop	164	43.2
Pharmacy	53	13.9
GOVT hospital / Dispensary	273	71.8
Private doctor / Nurse / Clinic	182	47.9
Private hospital	173	45.5
Herbalist	71	18.7
VCT centre	155	40.8

The most frequently mentioned place was the government hospital/ dispensary as the source of STI treatment. VCT centre was listed as a source by 40.8% of the girls.

The next section looked closer at the girls' knowledge on HIV/AIDS. Table 12 overleaf summarises the results.

As shown in the table, there is a high level of knowledge of HIV/AIDS among this population. The majority of respondents had correct information about the epidemic, the main modes of transmission and whether a healthy looking person can have AIDS.

Table 12: Knowledge of HIV/AIDS among the girls

	Correct knowledge n	%N
Ever heard of HIV/AIDS (Yes)	358	94.2
AIDS is caused by a virus	358	94.2
HIV means Human Immunodeficiency Virus	326	85.8
It is possible to cure AIDS	296	77.9
A person with HIV always looks emaciated or unhealthy in some way	156	41.1
People can take a simple test to find out if they have HIV	230	60.5
A person can get HIV from having sexual intercourse	341	89.7
A person can get HIV by working next to a person with HIV/AIDS or casual contact (pecking, hugging).	357	93.9
An unborn child can get HIV from an infected pregnant woman or breastfeeding after birth.	267	70.3
A healthy looking person can be having HIV	330	86.8
Sharing sharp needles with infected person can lead to AIDS	354	93.2
You can get HIV/AIDS by being transfused with infected blood	360	94.7
Immediately you are infected with HIV, you become AIDS patient	167	43.9

Nevertheless, there was misconception in some areas. Less than half (43.9%) correctly knew that one does not have AIDS immediately at infection with the HIV virus. Also, 41.1% of the girls believe a person with HIV always looks emaciated or unhealthy in some way. Only 39.5% did not know that a simple test can be taken to find out one's HIV status and 22.1% do not know that AIDS cannot be cured.

Table 13: Knowledge of high risk behaviours and methods of preventing transmission of HIV/AIDS

	Responses	Yes- n	%N
Something can be done to prevent one from contracting HIV infection	Yes	358	94.2
Known risky behaviours that can lead to HIV/AIDS infection	Multiple partners	264	69.5
	Unprotected Intercourse	320	84.2
	Untreated STD	195	51.3
	Drunkenness (alcohol)	142	37.4
	Drug abuse	155	40.8
	Other risk behaviours that can lead to transmission of HIV	5	1.3
Known methods to avoid contracting AIDS or STI	Abstinence (No sex)	311	81.8
	Monogamy (being faithful to one partner)	250	65.8
	Condoms	230	60.5
	Medications	30	7.9

The girls are well informed about the major risk factors in the transmission of HIV/AIDS. About eight in ten (84.2%) correctly identified unprotected sexual intercourse as constituting high risk factors followed by multiple sexual partners (69.5%) and untreated STI's (51.3%). Only 40.8% appreciate drug abuse as a risk factor and 37.4% know that alcoholism can be related to increased risk of HIV/AIDS transmission.

There was generally a good level of awareness of prevention methods with the majority knowing of abstinence as a method of prevention (81.8%), followed by mutual monogamy (65.8%) and condom use (60.5%).

As many as 75% of the girls knew a place they could go for HIV testing. The VCT center was the most commonly mentioned place (73.9%) followed by hospitals and dispensaries (14.3%). Only 10% of the girls had ever been tested. Of the remaining, 65.5% of the total study population would have liked to be tested for HIV.

DISCUSSION

The results of a survey among teenage girls on reproductive physiology and STI's have been presented.

There was approximately equal distribution of representation from form I to IV. They were aged between 13 and 19 years with a mean 0f 16.5 years. About 97% were Christians (catholic or Protestants) and 1 % Muslims. Majority (69.5%) were living with both their parents as their primary caregivers.

Knowledge on the menstrual cycle and the relationship with pregnancy timing and sexual intercourse was varied. Knowledge on menstruation was average (68%) but the relationship with reproductive physiology was poor. This may reflect their source of information where there may be misinformation about their reproductive biology. For example, about half (48.4%) think of the ovaries as source of menstrual blood, 73% do not know when to avoid pregnancy in the menstrual cycle confirming their ignorance in anatomy and physiology. Other studies have shown these girls to be ignorant about reproductive physiology [10-15].

Menarche and sexual activity are occurring at a lower age. In this study the youngest reported age at menarche was 10 years. The average age at menarche was 14.2 years. This was comparable to other studies locally and elsewhere[10,19,20]. This would mean many girls attain menarche before entry into secondary school. 16.6% the total number of girls had been sexually active by the time the survey was carried out. There was a rising trend with age. Other studies had higher proportions of the population found to be sexually active though not with large differences[11,14,15]. The difference could be attributed to the study being in a different geographical region and or the permissiveness in the different study areas. The rising trend with age is expected as the more mature the girl is, the more likely she is to have a boyfriend and therefore the higher the chances of having sexual intercourse. The youngest age at start of sexual activity was 9 years. This is disturbing as start of sexual activity at early age is related to other serious effects- teenage pregnancy, sexually transmitted infection, and cancer of the cervix.

The peak age at first coitus was 14.4yrs. This is earlier than found in previous studies and the median given by the KDHS of 16.7yrs[3,11,14,15,19]. This means that sexual activity is starting earlier and within 1-2 yrs of menarche. There must be a significant proportion starting sexual activity prior to menarche. In the majority of cases the sexual acts are not planned (59%). This makes contraception less likely and chances of pregnancy high indeed. These teenagers knowledge on reproductive physiology still leaves a lot to be desired. The young people do not understand the basic facts of reproduction. In this survey, many respondents were misinformed or did not know about the fertile period in a woman's menstrual cycle. This was in keeping with findings from other surveys in the country and other region of Africa. [10-12, 27]. This reflects that the knowledge they have about fertility is incomplete, misleading or outrightly wrong. Many parents, teachers and most clergy have opposed introduction of sex education in schools in our country for the fear that it encourages promiscuity amongst the adolescents. But studies show that sex education in fact is a deterrent to early sexual activity and even that for those sexually active it promotes the use of contraception [5, 20, 27]. May be the findings of this survey would have been different had this education been done in that region.

The girls' knowledge on contraception was low. Only a small number (12%) of the sexually active were using an effective method. Considering that most of the sexual activity among this population is mostly spontaneous and unplanned, this puts them at high risk of getting unwanted pregnancy. The most commonly mentioned methods were condoms, the injection and oral pills. It is likely that the poor knowledge on contraception is a result of negative attitudes promoted by the opponents of contraceptive use by adolescents. Currently, we have a national policy regarding the use of contraceptives by adolescents[38]. It would be prudent to inform the teenage girls about contraception early since we have seen they are initiating sexual activity very early and pregnancy prevention should thus be a priority. This has also been recommended by other researchers. [11, 17,

Knowledge of sexually transmitted infections other than H.I.V was limited. The most known infections were gonorrhoea and syphilis, probably the infection that have been prevalent in most areas. Vaginal discharge and painful micturation were the commonly mentioned health effects of STI's. Overall, the respondents did not demonstrate very

extensive knowledge of the health effects of STI's. This probably is because they are not involved in forums where such teachings are offered, indicating a need to create awareness in this area. Other studies have found similar results [25,28,29,40]. This would help to have the adolescent seek treatment early in case they get infected.

Knowledge of HIV/AIDS was generally good among the study population. The majority of the respondents have correct information about the epidemic, and the main modes of transmissions. At least 8 in 10 of them could identify unprotected sexual contact, blood transfusion and mother to child transmission as modes of transmission. However, there are areas where they seemed not to have adequate knowledge, probably due to myths circulating in the communities. At least half of them thought one immediately becomes an AIDS patient at infection with HIV. The good knowledge on transmission routes may be attributed to the publicity that this epidemic has received at local and national level.

The respondents were also well informed on the major risk factors and risky behaviours that can lead to transmission of HIV/AIDS. At least half of the population could individually identify multiple sexual partners, unprotected intercourse and untreated STI's as constituting high risk factors for HIV transmission. The data show that these girls appreciate the risks involved in sexual relations and recognize that these could lead to transmission of HIV/AIDS. They were not sure of drunkenness (alcohol use) and other drug abuse as risk factors that may lead to activities culminating in HIV infection. This indicates need for continued provision of information to the adolescents especially considering the fact that significant number of them (30%) did not think they were at risk of HIV infection. Similar findings have been found elsewhere[40].

There was good level of awareness of prevention methods with majority saying something can be done to prevent infection (94.2%).Abstinence was mentioned as a method of prevention by 82%. Mutual monogamy and condom use are also known in this population. However 7.9% of the girls claim to have knowledge of protective medications against HIV/AIDS, probably a misconception about what they have heard of anti- retrovirals. Another study had similar findings[40].

The knowledge can only be beneficial if it is translated into behaviour change. Behaviour changes in the studied population included delay in starting sexual activity, stopping of

sexual activity and "asking partners to be faithful". It is hoped that this is actually true and will be carried on in their future life as it will lead to lower HIV infection rates and eventually to realization of millennium development goal combating HIV/AIDS and reducing HIV prevalence among adolescents and young women by 2015[39]. At least three quarters of them know of a place where they could go for HIV testing and the voluntary counselling and testing centre named by majority (74%) as a place for testing.

CONCLUSIONS.

This was an adolescent sexuality and reproductive health study in Nyahururu. It explored a range of sexual and reproductive health issues among in-school adolescent girls. The following conclusions could be drawn from the study:-

1. The girls do not have adequate information on reproductive physiology.
2. Knowledge on contraception is low among these girls.
3. There is low level of awareness of STI's besides HIV, gonorrhoea and syphilis. Furthermore, many adolescents did not know of specific health effects of STI's.
4. There is a high level of knowledge of HIV/AIDS transmission modes, high-risk behaviours and also acknowledgement that there are means of preventing HIV transmission.

RECOMMENDATIONS.

Adolescent health is critical for development. This study shows that there is an urgent need for more aggressive adolescent sexual and reproductive health interventions as there is not much difference in findings from surveys conducted earlier and in different regions of the country.

The challenge is how to create a situation where adolescents have access to more accurate sources of sexual and reproductive health information and services that are accessible, available, affordable and acceptable to them. The challenges are definitely daunting, but with determination, will and community plus stakeholders engagement, a difference can be made.

From this study findings, various recommendations can be made. These would include the following:-

1. There is a need to provide adolescents with information to enable them develop responsible patterns of sexual and reproductive behaviour. This can be done through schools, churches, community organisation and other appropriate forum.

2. There is need to strengthen the capacity of parents, teachers as they were mentioned as the main sources of information so that they have appropriate information and skills to impart accurate and appropriate sex education.
3. Efforts are needed to promote appropriate STI/HIV/AIDS education programmes for adolescents. This will help address existing misconceptions, give more information about STI's, their symptoms, health effects and sources of treatment. The present lack of awareness has important implications for treatment and counselling.

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APPENDICES

APPENDIX A

QUESTIONNAIRE

STUDY NO: ☐☐☐

PATIENTS HOSPITAL NO----------

SECTION A: SOCIODEMOGRAPHIC CHARACTERISTICS

Insert code in the box where applicable

1. AGE (In Years) ☐☐
2. MARITAL STATUS (Insert code in the box) ☐
   1. Single
   2. Married
   3. Separated /Divorced
   4. Windowed
3. LEVEL OF EDUCATION (Inset code in box) ☐
   1. None
   2. Primary
   3. Secondary
   4. College/University
4. Occupation (Insert code in box) ☐
   1. None
   2. Self employed
   3. Employed (Non – professional)
   4. Employed professional
   6. Housewife

SECTION B: PAST OBSTETRIC / MEDICAL HISTORY

5. Number of Deliveries ☐
6. Number of Abortions ☐

7. Number of living children ☐

8. If she has delivered before, history of high BP in prior pregnancies ☐
   1. Yes
   2. No
9. Prior history of eclampsia ☐
   1. Yes
   2. No.
10. Prior history of hypertension outside pregnancy ☐
    1. Yes
    2. No.
11. Prior history of Renal disease ☐
    1. Yes
    2. No.
12. Prior history of Diabetes mellitus ☐
    1. Yes
    2. No. ☐
13. Family history of hypertension ☐
    1. Yes
    2. No.

    If yes in whom ? ☐
    1. Mother
    2. Sister
    3. Other (specify) ------- ☐

SECTION C; INDEX PREGNANCY

14. Antenatal Care

a) Place
   1. None attended
   2. Rural health centre
   3. City council of Nairobi
   4. Public hospital
   5. Private hospital
   6. K.N.H

b) Gestation at booking
   1. Less than 20 weeks
   2. 21-28 weeks
   3. 29-36 weeks
   4. More than 36 weeks

c) Number of visits

15 a) Gestation at diagnosis of hypertension

   b) Diagnosis at admission
      1. Severe P.E.T
      2. Eclampsia before delivery
      3. Postpartum eclampsia

16. At admission
   a) Gestation period in weeks
   b) Oedema
      1. None
      2. Pedal
      3. Generalised

   c) Proteinuria
      1. +
      2. 2 +
      3. 3 + or more
   d) Fits 1. Yes
      2. No.
   e) Occurrence of fits
      1. Before Admission
      2. Intrapartum
      3. After delivery
      4. After admission but before delivery

17. During period of Admission to delivery

   a) Highest recorded Systolic pressure Mm Hg

   b) Highest recorded Diastolic pressure Mm Hg

   c) Lowest recorded systolic pressure Mm Hg

   d) Lowest recorded diastolic pressure Mm Hg
   e) Maximal proteinuria
      1. +
      2. 2 +
      3. 3 +
      4. 4 +
   f) Treatment
      1. Antihypertensive only
      2. Antihypertensive + Anticonvulsant

g) Antihypertensive given

1. Methyldopa (Aldomet) only
2. Hydrallazine drip only
3. Aldomet + Hydrallazine
4. Aldomet + Nifedipine (Adalat)
5. Other (specify)

h) Anticonvulsant used

1. Diazepam
2. Phenobaibitone
3. Mg So4
4. Phenytoin
5. Other (specify)

18. Labour and Delivery

a) Gestation at intervention (in weeks)

b) Fundal height at intervention (in weeks)

e) Reason for intervention

1. Intrauterine death
2. Reduced foetal movements
3. Uncontrollable BP
4. Deteriorating renal function
5. Patient at term
6. Other (specify) ----------

d) Steroids administered ?

1. Yes
2. No.

e) Mode of intervention

1. Spontaneous labour
2. Induction of labour
3. Elective c/section
4. Emergency c/section

f) Duration of labour

1. Less than 8 hours
2. 8-12 hours
3. More than 12 hrs

g) Mode of delivery

1. S.V.D
2. C/section
3. Vacuum extraction
4. Other (specify) ----------

h) For c/s deliveries , what was the indication

1. Fetal Distress
2. Previous section scar
3. C.P.D
4. Malpresentation
5. Abruptio placenta
6. Failed induction
7. Other (specify).....

19. Foetus

a) Sex

1. Female
2. Male

b) Weight

1 <1000g
2 1001-1500g
3 1501-2000g
4 2001-2500g
5 2501-3000g
6 3001-3500g
7 More than 3500g

c) Apgar score at

1 min

5 min

10min

d) Baby admitted to Nursery

1. Yes
2. No.

f) If yes to (d) Reason for admission to NBU

1. Congenital Malformations
2. R.D.S
3. Asphyxia
4. Birth trauma
5. Normal baby for observation
6. Other (specify) ----------

g) State of baby in NBU after 24hrs

1. Alive
2. Dead

h) Duration of NBU stay

1. <2 days
2. 3-7 days
3. 8-14 days
4. 15-21 days
5. 22-28 days
6. 28 days

i) In case the baby was dead

1. FSB
2. MSB
3. Early Neonatal Death

j) Reason for Neonatal Death

1. R.D.S
2. Birth Asphyxia
3. Prematurity
4. Birth trauma
5. Infection/sepsis
6. Other (specify) ----------

j) Placenta weight grams

20. Mother

a) Days in hospital

b) Complications

1. Yes
2. No.

c) If yes to (b) which complications (TICK AGAINST ALL THAT APPLY)

1. Renal failure
2. C.V.A
3. Sepsis
4. Eclampsia
5. Abruptio placenta
6. Pulmonary oedema
7. D.I.C
8. Other (specify)----------

d) (i) Admitted to I.C.U 1.Yes.
2. No

If yes

(ii) Length of stay in I.C.U in days
(iii) Indication

1. Pulmonary Oedema
2. Cerebral Haemorrhage
3. Difficult Reversal from Anaesthesia
4. Other (Specify)----------

e) (I) Renal failure?

1.Yes
2. No

(II) Dialysis

1. Yes
2. No

(III) Renal function at discharge

(i) Proteinuria 1.) Nil 2.) 1-2+ 3) equal or more than 3+

(ii) Urea 1) Normal 2) Elevated 3) Not done

(iii) Creatinine 1) Normal 2) Elevated 3) Not done

f) Duration on anti hypertensives after delivery

1. Less than 2 days
2. 3-6 days
3. 7-14 days
4. > 14 days

g) Maternal death 1. Yes
2. No

If yes, i) What was the cause of death?

1. Acute Renal failure
2. Pulmonary Oedema
3. Cerebral Haemorrhage
4. Haemorrhage shock 2^0 DIC
5. Sepsis
6. Cardiopulmonary failure
7. Others (Specify)----------

ii) Post-mortem done 1. Yes
2. No

APPENDIX B: GYNAECOLOGY QUESTIONNAIRE

DATE:_____/_____/2005

INTERVIEW NO. _____.

SECTION A:

1. Age (How old were you at your last birthday)? ☐☐ years
2. What day, month and year were you born? ☐☐ Day ☐☐ Month ☐☐ Year

3. Which form are you in? ☐ Form I ☐ II ☐ III ☐ IV

4. What is your religion?

None	01
Catholic	02
Protestant	03
Muslim	04
Hindu	05
Jew	06
Other (specify)	07

5. In the household you live, who do you live with?

Father	01
Mother	02
Father and Mother	03
Brother or sister	04
Uncle/Aunt	05
Grandmother/ Grandfather	06
Friend	07
Other (specify).....	08

6. Are you married?

1. Yes 2. No. ☐

SECTION B

Now some questions on sex and reproduction

B1: What has been your **most** important source of information on puberty and the reproductive systems of men and women?

School teacher	01
Mother	02
Father	03
Brother	04
Sister	05
Friends	06
Other family members	07
Doctors	08
Books/ magazines	09
Films/Videos	10
Radio/TV	11
Religious leaders	12
Others(Specify..... .....)	13

B2: What would be your **preferred** source of information on Puberty and the reproductive systems of men and women?

School teacher	01
Mother	02
Father	03
Brother	04
Sister	05
Friends	06
Other family members	07
Doctors	08
Books/ magazines	09
Films/Videos	10
Radio/TV	11
Religious leaders	12
Others(Specify.....)	13

3. Please tell me whether you think the following statement is true, false or whether you don't know. TICK IN THE APPROPRIATE COLUMN.

	True	False	Don't know
(a) A woman can get pregnant on the very first time that she has sexual intercourse	1	2	3
(b) A woman stops growing after she has had sex (intercourse) for the first time	1	2	3
(c) Masturbation causes serious damage to health	1	2	3
(d) A woman is most likely to get pregnant if she has sexual intercourse mid point between her periods.	1	2	3
(e) Periods mean sexual maturity	1	2	3
(f) A woman can become pregnant if she has sex during her periods	1	2	3
(g) Boys of 13 – 15 years cannot make girls pregnant	1	2	3
(h) A woman can become pregnant if she has sex just before her periods begin.	1	2	3
(i) A woman is safe from pregnancy a week after her periods.	1	2	3
(j) Pregnancy can only occur if a woman has sex with a man	1	2	3
(k) Kissing can result in pregnancy	1	2	3
(l) Having sex once a while does not result in pregnancy	1	2	3
(m)If a man does not put his penis deep in a woman's vagina ,pregnancy will not result	1	2	3
(n)Wet dreams are a sign that a boy has started producing sperms	1	2	3
(o)Boys produce sperms only after growing beards	1	2	3
(p)Sex can only take place between people of opposite sexes.	1	2	3
(q)sexual intercourse can help in spread of infections.	1	2	3
(r)A normal pregnancy lasts for 9 months before a baby is born.	1	2	3
(s)Condoms if used properly can protect a woman from becoming pregnant.	1	2	3

4. The following questions are about menstruation.

a)Which of the following do you think is the source of menstrual blood?

TICK AGAINST THE APPROPRIATE ANSWER

1. The Vagina
2. The uterus
3. The fallopian tubes
4. The ovaries
5. Other, specify ---------

b)Please tell me whether you think the following statement is true,false or whether you don't know.TICK IN THE APPROPRIATE COLUMN.

	True	False	Don't know
a)Menstruation is normal in women	1	2	3
b)Menstruation occurs in men also	1	2	3
c)Menstruation occurs once a month in normal women	1	2	3
d)Menstruation is a sign of beginning of fertility in women	1	2	3
e)The menstrual cycle lasts for 28 days in every woman	1	2	3
f)Women always bleed for 5 days	1	2	3
g)The safest point to avoid pregnancy is midpoint of a woman's menstrual cycle	1	2	3

5. Have you ever had your menstrual periods?

1.Yes 2. No ☐

6. How old were you when you had your first period? .....years

7. Do you think you were adequately informed about the periods by the time you started having them?

1. Yes 2. No ☐

8.Please tell me who you think is the **most** appropriate person among the following to prepare a girl for her first menstrual period.

1.Mother
2.Father
3.Elder sister
4.Aunties
5.Medical person(doctor/nurse)
6.Religious person
7.School teacher
8.Nobody

☐

SECTION C

Now some question about sexual behaviour and family planning methods

1. Have you ever had sex? 1.Yes 2.No ☐

If No, go to No. 9.

2. If yes to above, how old were you when you first had sex? ☐☐

Years

3. How long ago did you last have sex?
   1. Less than one month
   2. 1 –2 Months
   3. > 2 months

4. How many sexual partners have you had in the last1year?
   1. One
   2. Two
   3. ≥ Three

5. Do you or your partner do anything to avoid getting pregnant on these occasions? If yes Always or sometimes
   1. Always 2. Sometimes

6. The last time you had sex, was it planned or not planned?
   1. planned
   2. Not planned

7. Have you ever been pregnant? 1.Yes 2. No

   (b) If YES how many times ?
   1 One
   2 Two
   3 Three or more

   (c) How old were you during your first pregnancy? ---------- years

8. If yes to No. 7 what happened to the last pregnancy?
   1. Currently pregnant
   2. Abortion
   3. Miscarriage
   4. Live – birth
   5. Not sure

   * Go to No. 11.

9. People may have mixed reasons for not having sex (intercourse) please tell me for each of the following reasons whether it applies to you or not.

	Applies	**Not apply**
(a)I don't feel ready to have sex	1	2
(b)I have not had the opportunity	1	2
(c)I think sex before marriage is wrong	1	2
(d)I am afraid of getting pregnant	1	2
(e) I am afraid of getting HIV/AIDS or another sexually transmitted infection.	1	2

10. Which of these statements **best** describes your future plans about sex?
1) I plan to wait until marriage
2) I plan to wait until I am engaged to be married
3) I plan to wait until I find someone I love
4) I plan to wait until I finish my education and working
5) I plan to have sex when an opportunity comes along

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11.(a) Do you feel any pressure from others to have sexual intercourse

1. Yes 2. No ☐

(b) If yes, a great deal or a little

1. A great deal
2. Alittle
3. None ☐

12. From whom do you feel the pressure?
CIRCLE ALL THAT APPLY

1. Friends
2. Relatives
3. Partner/specialFriend
4. Radio/TV
5. Others.....

13. Have you ever received money or gifts in return for offering sex? Yes 1 No 2 ☐

14. Do you think it is proper for schoolgirls to engage in sex? Yes 1 No 2 ☐

a) If yes answer the following.

i) At what age should they start engaging in sex?

_____

ii) Why should they have sex?

_____

iii) With whom should they have sex?

_____

b) If No, why?

_____

15.Have you ever been forced to have sex ? Yes 1 No 2

16. Some people use various methods to prevent or delay having a pregnancy. This is called family planning or contraception.

a) Which method/s have you ever heard of? Tick against the method you have heard of.

METHOD	
Oral pills	
Condoms	
Spermicide	
Coil (intra uterine device)	
Injections	
Abstinence	
Calendar (Rhythm method) periodic Abstinence	
Morning after pill/postinor	
Withdrawal	
Implants – Norplant	
Female/Male Sterilization	
Other (Specify).....	

b) What was the most important source of your information

Source	(1) Most Important
School teacher	1
Father	2
Mother	3
Sibling –Brother	4
Sister	5
Doctor/Nurse	6
Hospital worker	7
Television/Radio/Magazine/film	8
Religious leaders	9
Chemist	10
Other Specify……..	11

c) What would be your preferred source of information.

Source	(1) Preferred
School teacher	1
Father	2
Mother	3
Sibling –Brother	4
Sister	5
Doctor/Nurse	6
Hospital worker	7
Television/Radio/Magazine/film	8
Religious leaders	9
Chemist	10
Other Specify……..	11

17. Do you know any place or person where young people could obtain family planning methods. Name some.

Source	Most Important	Preferred
Pharmacy/chemist.	1	1
Government Dispensary	2	2
VCT Centre	3	3
Market shop	4	4
Private doctors clinic	5	5
Other specify……..	6	6

18. Which method of family planning have you ever used.
CIRCLE ALL THAT APPLY.

Pill	1
Injection	2
Condom	3
Emergency pill	4
Withdrawal	5
Calender (Rhythm)	6
Other Specify.....	7

19. What do you think about the following with regards to contraceptive methods?

	1 Yes	2 No	3 Don't know/Not sure
a) They are not easily available to school girls.	1	2	3
b) They should be made easily available to school girls.	1	2	3
c) If made easily available, they will make girls more careless in their sexual behaviuor	1	2	3
d) parents should encourage their sexually active daughters to use them	1	2	3

20.Please tell me whether the following statements are true ,false or whether you don't know
TICK IN THE APPROPRIATE COLUMN

	True	False	Don't know
a)Contraceptives should be used by women only	1	2	3
b)Family planning means limiting the number of children one should have	1	2	3
c)Contraception means taking pills only	1	2	3
d)Contraception methods for men are not available	1	2	3
e)Using contraceptives results in failure to get children in future	1	2	3
f)Contraceptive use interferes with pleasure of sex	1	2	3
g)Contraceptive use increases premarital sex	1	2	3
h)Contraceptives are not dangerous to the health of a woman	1	2	3
i)Condoms are effective in preventing pregnancy	1	2	3
j)Condoms can be used more than once	1	2	3

SECTION D.

1. Sexually transmitted infections are those that are spread by people having unprotected intercourse. Which of the following have you ever heard of ?
   TICK AGAINST EACH ONE THAT YOU KNOW

1. Herpes simplex	
2. Gonorrhoea	
3. Warts	
4. Syphillis	
5. Hepatitis	
6. HIV/AIDS	
7. Trichomoniasis	
8. Candidiasis (Yeast)	
9. Pubic lice	
10. Scabies	
11. Chancroid (ulcer)	
12. Other (specify) -----	

2. What are the health effects of sexually transmitted infections?
   TICK AGAINST EACH ONE THAT YOU KNOW.

Health effect	**Yes**
1. Vaginal discharge	
2. Pain during urination	
3. Weight loss	
4. Rash	
5. Ulcer/ sore in the genital area	
6. Infertility (inability to have children)	
7. Pain during intercourse	
8. Still birth/Miscarriage	
9. Abdominal pain	
10. Swollen glands in the groin	
11. Other specify ----------	

3. Where could a person receive treatment for sexually transmitted infection?
   TICK/CIRCLE AGAINST THE PLACE/S YOU KNOW.

Place	
1. Shop	
2. Pharmacy	
3. Government Hospital/ Dispensary/ health centre	
4. Private Doctor/Nurse/ clinic	
5. Private hospital	
6. Herbalist	
7. VCT Centre	
8. Other Specify	

4 Have you ever heard of HIV/AIDS 1. Yes ☐

2. No

5 Of the following statements about HIV/AIDS tell me whether you think the statement is true or false or whether you don't know.
TICK IN THE APPROPRIATE COLUMN.

	True	False	Don't know
a) AIDS is caused by a virus	1	2	3
b) HIV means Human Immunodeficiency virus	1	2	3
c) It is possible to cure AIDS	1	2	3
d) A person with HIV always looks emaciated or unhealthy in some way	1	2	3
e) People can take a simple test to find out whether they have HIV	1	2	3
f) A person can get HIV from having sexual intercourse	1	2	3
g) A person can get HIV by working next to a person with HIV/AIDS	1	2	3
h) An unborn child can get HIV from an infected pregnant mother	1	2	3
i) A healthy looking person can be having HIV	1	2	3
j) A person can get AIDS from sharing sharp instruments like needles with an infected person	1	2	3
k) You can get HIV/AIDS by being transfused with infected blood	1	2	3
l) As soon as a person gets infected with HIV, he/she becomes an AIDS patient	1	2	3
M) Having unprotected sex for the first time with an infected partner can spread HIV	1	2	3
n) One can get AIDS by staying in an overcrowded place.	1	2	3
o) A baby can get HIV through breastfeeding from the mother.	1	2	3
p) A person can get HIV from casual body contact e.g hugging, pecking.	1	2	3

6 Do you think you can get AIDS ? 1.Yes ☐

2. No

7 Is there anything you can do to avoid getting HIV/AIDs?

1. Yes ☐

2. No

8 Which of the following are risky behaviours that can lead to HIV/AIDS infection?
FILL IN COLUMN FOR EACH.

HIV Transmission sexual Risk behaviours	Yes	No	Don't know
1) Multiple partners	1	2	3
2) Unprotected Intercourse	1	2	3
3) Untreated sexually transmitted diseases	1	2	3
4) Drunkeness (alcohol)	1	2	3
5) Drug abuse	1	2	3
6) Other specify.....			

9. Some people use various ways to avoid contracting AIDS or an S.T.I. Which ways have you heard of?
TICK AGAINST EACH ONE THAT YOU KNOW.

Safe Sex Method	**Yes**
1) Abstinence (No sex)	
2) Monogamy (Being faithful to one partner)	
3) Condoms	
4) Medications (specify.....)	
5) Other specify.....	

10. a) Since you heard of HIV/AIDS, have you changed your behaviour to prevent AIDS?
1. Yes
2. No ☐

b) If yes what have you done: Tick WHERE APPROPRIATE

i) I have not started having sex	
ii) Stopped all sex	
iii) Started using condoms	
iv) Restricted sex to one partner	
v) Reduced my number of sex partners	
vi) Asked my partner to be faithful	
vii) Stopped drug injections (shared)	
viii) Other (specify).....	

11. a) Do you know a place you can go for HIV testing? 1. Yes 2. No ☐

b) If yes Name the places you know.

_____

12. a) Have you ever been tested if you have the HIV virus? 1. Yes 2. No ☐

b) If No, would you like to be tested ? 1. Yes 2. No. ☐

KENYATTA NATIONAL HOSPITAL
Hospital Rd. along, Ngong Rd.
P.O. Box 20723, Nairobi.

Tel: 726300-9
Fax: 725272
Telegrams: "MEDSUP", Nairobi.

Ref: KNH-ERC/01/2433

Date: 4th November 2004

Dr. Kariuki David Gicheru
Department of Obs/Gynae
University of Nairobi

Dear Dr. Gicher.

RESEARCH PROPOSAL: " MANAGEMENT OF SEVERE PREECLAMPSIA AT KENYATTA NATIONAL HOSPITAL" (P119/9/2004)

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 4th November 2004 – 3rd November 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
CMRO
Supervisors: Dr. Waithaka Njoroge, Dept.of Obs/Gynae, UON
Dr. Magoha. O.B., Dept.of Obs/Gynae. UON

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Ref: KNH-ERC/01/2451

Date: 12th November, 2004

Dr. David Kariuki Gicheru
Dept.of Obs/Gynae
University of Nairobi

Dear Dr. Gicheru,

RE: KNOWLEDGE ON REPRODUCTIVE PHYSIOLOGY, CONTRACEPTION AND S.T.I's ESPECIALLY HIV/AIDS AMONG RURAL TEENAGE SCHOOL GIRLS IN NYAHURURU" (P96/7/2004)

This is to inform you that the Kenyatta National Hospital Ethics and Research Committee has reviewed and **approved** revised version of your above cited research proposal for the period 12th November 2004 – 11th November 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 Medicine, UON
CMRO
Supervisor: Dr. Waithaka Njoroge, Dept.of Obs/Gynae, KNH
Dr. Magoha O B, Dept.of Obs/Gynae, KNH

MINISTRY OF EDUCATION, SCIENCE AND TECHNOLOGY

ıs: "EDUCATION", Nairobi
e: Nairobi 334411
plying please quote

..... and date

JOGOO HOUSE "B"
HARAMBEE AVENUE
P.O. Box 30040–00100
NAIROBI

....., 20.....

MOEST 13/001/35C 96/2

16th March, 2005

Dr. David Gicheru Kariuki
P.O. BOX 6167-00200
Kenyatta National Hospital
NAIROBI

Dear Sir

RE: RESEARCH AUTHORISATION

Following your application for authority to conduct research on "Knowledge on reproductive Physiology contraception and S.T.I.'S especially HIV/AIDS among rural teenage school girls", I am pleased to inform you that you have been authorised to conduct research in Laikipia and Nyandarua Districts for a period ending 30th June, 2005. You are advised to report to the District Education Officers and the Medical Officers of Health of the respective Districts before embarking on your research project.

Upon completion of your research projects you are expected to submit two copies of your research report to this Office.

Yours faithfully

B. O. ADEWA
FOR: PERMANENT SECRETARY

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Cc
The District Commissioner
Laikipia
Nyandarua Districts

The District Education Officer
Laikipia
Nyandarua Districts

The Medical Officer of Health
Laikipia
Nyandarua Districts