CASE REPORTS AND COMMENTARIES

IN

OBSTETRICS AND GYNAECOLOGY

SUBMITTED BY

DR. FREDRICK ODHIAMBO OBAGO

IN PART FULFILMENT FOR THE DEGREE OF MASTER OF MEDICINE

IN

OBSTETRICS AND GYNAECOLOGY

OF THE

UNIVERSITY OF NAIROBI

JUNE 2009

TABLE OF CONTENTS

Gynaecology short cases

DEDICATION

To all the women of Africa, whose lives I seek to make a difference in.

ACKNOWLEDGEMENT

I thank almighty God for enabling me to reach this far and complete this book against so many odds.

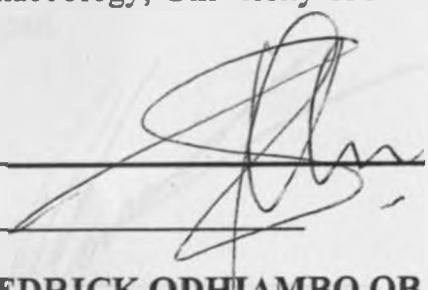
I would like to thank the Ministry of Health for having sponsored my training at the university of Nairobi.

I would like to thank all the consultants, lecturers and senior registrars in the Department of Obstetrics and Gynaecology for their dedication and commitment to my acquisition of knowledge and necessary skills during my training at the University of Nairobi.

I am sincerely grateful to my supervisors Prof. Ndavi, Dr. Wanjala and Dr Odawa for their efforts to see that my proposals and long commentaries were properly written by offering expert advice and guidance and also making very meaningful critique of the long commentaries.

DECLARATION

This is to certify that the case records and commentaries presented in this book are my original work and have not been presented for a degree course in any other university. I further certify that all the cases were managed by me under supervision of the senior members of the Department of Obstetrics and Gynaecology, University of Nairobi

Signed 

Date 11/11/09

DR FREDRICK ODHIAMBO OBAGO

CERTIFICATION OF AUTHENTICITY OF THE THESIS

I Dr. Fredric O. Obago, hereby declare that these case reports, commentaries and research work done in part fulfillment for the Degree of Masters of Medicine in Obstetrics and Gynaecology in the University of Nairobi are my original work conducted in the Department of Obstetrics and Gynaecology at the university of Nairobi under the Chairmanship of the under signed.

Signed _____

Date 14/11/09

PROF. KAMAU KOIGI

PROFESSOR IN OBSTETRICS AND GYNAECOLOGY,

CHAIRMAN,

DEPARTMENT OF OBSTETRIC AND GYNAECOLOGY,

COLLEGE OF HEALTH SCIENCES

UNIVERSITY OF NAIROBI.

LEAD SUPERVISOR

PROF. KOIGI KAMAU,

PROFESSOR IN OBSTETRICS AND GYNAECOLOGY,

CHAIRMAN,

DEPARTMENT OF OBSTETRIC AND GYNAECOLOGY,

COLLEGE OF HEALTH SCIENCES

UNIVERSITY OF NAIROBI.

SIGNED..... DATE..14th.... DAY OF...Nov.....2009

SUPERVISORS

DR.SAMSON WANJALA

SENIOR LECTURER

DEPT OF OBS/GYN,

COLLEGE OF HEALTH SCIENCES

UNIVERSITY OF NAIROBI

SIGNED..... DATE..12TH DAY OF..NOV.....2009

DR. F. ODAWA

MBCHB, MMED, NAIROBI

LECTURER

DEPT. OF OBS/GYN

COLLEGE OF HEALTH SCIENCES

UNIVERSITY OF NAIROBI

SIGNED.....DATE..12TH.....DAY OF..NOV.....2009

CERTIFICATION OF SUPERVISION

This is to certify that the Dr. Fredrick Odhiambo Obago researched upon the long commentaries in this book under my guidance and supervision and that this book is submitted with my approval.

Signed W Wanjala

Date 14/11/09

DR. WANJALA

MBCHB, MMED,

SPECIALIST OBSTETRICIAN AND GYNECOLOGIST

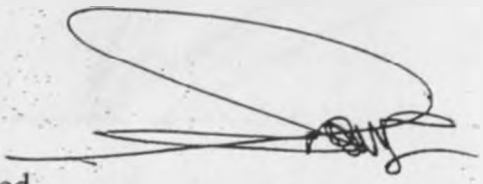
SENIOR LECTURER

DEPARTMENT OF OBSTETRICS AND GYNECOLOGY

UNIVERSITY OF NAIROBI

CERTIFICATION OF SUPERVISION

This is to certify that the Dr. Fredrick Odhiambo Obago researched upon the long commentaries in this book under my guidance and supervision and that this book is submitted with my approval.



Signed_____

Date 14.11.09

DR. ODAWA

MBCHB, MMED,

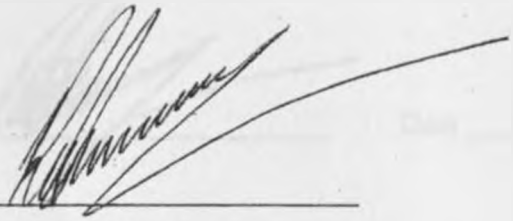
SPECIALIST OBSTETRICIAN AND GYNECOLOGIST

DEPARTMENT OF OBSTETRICS AND GYNECOLOGY

UNIVERSITY OF NAIROBI

CERTIFICATION OF SUPERVISION

This is to certify that **obstetrics** cases **no. 11,12,13,15** were managed by Dr. Fredrick O. Obago under my guidance and supervision at Kenyatta National Hospital.

Signed 

Date 14/11/09

PROF. KAMAU KOIGI

PROFESSOR IN OBSTETRICS AND GYNAECOLOGY,

CHAIRMAN,

DEPARTMENT OF OBSTETRIC AND GYNAECOLOGY,

COLLEGE OF HEALTH SCIENCES

UNIVERSITY OF NAIROBI.

SIGNED.....

CERTIFICATION

This is to certify that **gynecology** cases no. **6, 7,** and **9** and **obstetrics** cases no. **1, 3,** and **6** were managed by Dr. Fredrick O. Obago under my guidance and supervision at Kenyatta National Hospital.

Signed _____ Date 12/11/08

PROFESSOR KOIGI KAMAU,

PROFFESSOR AND CHAIRMAN,

DEPARTMENT OF OBSTETRICS AND GYNAECOLOGIST

UNIVERSITY OF NAIROBI

CERTIFICATION

This is to certify that gynecology cases no. **11, 12, 13, 14 and 15** and obstetrics cases no. **7 and 11** were managed by Dr Fredrick O. Obago under my guidance and supervision at Kenyatta National Hospital.

Signed Wanjala Date 3/11/08

DR SAMSON WANJALA, M.B.Ch.B, M.MED. (OBS/GYN)

Senior Consultant Obstetrician and Gynecologist

Senior Lecturer, Department of Obstetrics and Gynecology

University Of Nairobi

CERTIFICATION

This is to certify that gynecology cases no. 3, 4 and 5 **and** obstetrics cases no. 2 and 4 were managed by Dr. Fredrick O. Obago under my guidance and supervision at Kenyatta National Hospital.

Signed _____ Date 17/10/08

DR. NJOROGE WAITHAKA, M.B.Ch.B, M.MED. (OBS/GYN), HOD
Consultant Obstetrician and Gynecologist
Chief Medical Specialist
Kenyatta National Hospital
Nairobi

CERTIFICATION

This is to certify that gynaecology cases no. **1, 2, 8, 10** and obstetrics cases no. **1 and 3** were managed by Fredrick O. Obago under my guidance and supervision at Kenyatta National Hospital.

Signed _____ Date 14/5/09

DATE:.....DAY OF 2009

DR JALDESA GUYO
M.B.Ch.B, M.MED. (OBS/GYN)
CONSULTANT OBSTETRICIAN AND GYNAECOLOGIST

KENYATTA NATIONAL HOSPITAL

INTRODUCTION

KENYATTA NATIONAL HOSPITAL

The obstetric and gynaecology short cases presented in this book were managed at the Kenyatta National Hospital (KNH). Similarly, the obstetric and gynecology long commentaries were researched in the same hospital.

Kenyatta National Hospital is situated about 4km from the Nairobi central business district along Ngong Road. It serves as a national referral and teaching hospital as well as serving the population within and around the city. It provides curative, preventive and rehabilitative services in all medical specialties. It is a training centre for undergraduate and postgraduate students of the College of Health Sciences of the University of Nairobi and for nurses, clinical officers and other paramedics of the Kenya Medical Training College (KMTC) Nairobi.

OBSTETRIC AND GYNAECOLOGY SERVICES

The out-patient services are provided at casualty department, antenatal clinics, post-natal clinic, gynaecology outpatient clinics and the family welfare clinic (FWC). In-patient services are provided in labor ward, acute gynaecology ward, cold gynaecology wards and the antenatal/postnatal wards.

The unit is divided into three firms, each headed by a senior consultant obstetrician/gynecologist, with a team of consultant obstetricians /gynecologists, senior registrars, senior house officers, interns, nurses and paramedical staff. The senior medical staffs are from both the University of Nairobi and KNH.

Laboratory services are provided by both the hospital and the University of Nairobi. The department of Obstetrics and Gynaecology of the University

of Nairobi offers the following laboratory services for the hospital: semen analysis, hormonal radio-immunoassay, cytology, chromosome analysis, bilirubin spectrophotometry, surfactant test and glucose tolerance test. Radiological examinations such as ultrasound scans are provided in the radiology department of hospital and also at the department of Diagnostic Radiology of the University of Nairobi.

CASUALTY DEPARTMENT

The gynaecological casualty services are currently offered at the acute gynaecological ward (1D). Patients seen in this casualty are first screened at the main casualty. The patients are attended to by a senior house officer assisted by clinical and medical officers on internship training. Most patients are treated and discharged or referred to the gynaecology or obstetric clinics. Patients requiring admission are admitted to the labor ward, antenatal ward or to the acute gynaecological wards.

Obstetric cases involving mothers not booked at the KNH antenatal clinic are first seen in the main casualty and then sent to the labor ward where they are attended to by a senior house officer assisted by medial officer interns.

ANTENATAL CARE (ANC)

Initially this used to be for high- risk patients. However, currently any antenatal mother can be followed up in the clinic. The booking is done every Monday, Tuesday, Wednesday and thursday morning. During the booking visit, a detailed history of past obstetrical and gynaecological, medical, family and social history is taken. The weight, height, blood pressure and urinalysis are taken. A complete physical examination is done. Those needing admission are admitted to the labour ward or to the admitting antenatal ward. The rest are given appointments for their next visits. The clients receive health education talks from midwifes and nutritionists. Blood specimens are taken for haemogram, serology for syphilis and HIV and grouping for ABO and Rhesus factor.

ANTENATAL FOLLOW-UP

The follow-up is adapted to the focused antenatal care model At each visit health education about pregnancy, breast care, puerperium, baby care,

nutrition and HIV is given. The clients are then examined with particular attention to blood pressure, proteinuria, weight gain and oedema. Abdominal examination is done to determine fundal height, the foetal lie, presentation, engagement and foetal heart rate. The findings are recorded on the antenatal card which is kept in the hospital. For first pregnancies or with pregnancy interval of more than 5 years, two tetanus toxoid injections are given 4 weeks apart. Where the birth interval is less than 5 years, only a booster T.T. is given during the second trimester.

At 36 weeks of gestation, clinical pelvic assessment is done on all primigravidae and both clinical and radiological pelvimetry on patients with one previous scar with cephalic presentation. Amniocentesis for surfactant test used to be done at 38 weeks for mothers scheduled for elective caesarean delivery to assess foetal lung maturity. This is rarely done nowadays. Patients scheduled for elective caesarean delivery are admitted at 38 weeks of gestation with results of haemoglobin level and blood urea and electrolytes.

HOSPITAL ADMISSIONS

These fall into three categories namely; Booked patients from our antenatal clinic, referrals from other hospitals or health centres and those without prior antenatal care. The last two categories constitute the majority of admissions.

Booked patients report straight to labor ward when in labor or whenever they have a problem outside their date of appointment. Unbooked patients are first seen in casualty before being sent to labor ward admission area. In labor ward all patients are seen and those requiring immediate delivery remain in the labor ward until after delivery and patients not due for delivery are discharged through the antenatal clinic (ANC). Very sick are admitted to the acute room in labor ward and managed accordingly.

Patients in labor ward are seen by the House Officers and Senior House Officers. Difficult cases are managed in consultation with the specialist obstetrician/gynecologist and/or consultant obstetrician/ gynecologist.

COMMON OBSTETRIC PROCEDURES

The following procedures are performed frequently within the obstetric unit. The description of the procedures given in this book refers to the standard or preferred method(s) as performed and taught within the department.

Vaginal Examination

This includes speculum and digital examinations. The description below refers to digital examination. Vaginal examination is an aseptic procedure and is done on admission during the initial assessment of labor. The examiner washes his/her hands and wears sterile surgical gloves. Explanation is made to the patient on the nature of the procedure. After consent has been obtained, the patient is placed in dorsal position with knees drawn.

The vulva is inspected and any abnormalities noted. The vulva is then cleaned using five swabs soaked with antiseptic solutions as follows: a swab is picked by the right hand and transferred to the left hand, using the left hand, the left labia majora is swabbed once anteroposteriorly and the swab is discarded. Another swab is picked again and the same procedure is repeated on the right side. The procedure is repeated on the left side. The left hand now separates the labia using the index finger and thumb and the introitus gently swabbed anteroposteriorly. The right index and middle fingers are gently introduced into the vagina. Next the position, consistency, effacement and dilatation of the cervix are noted. The status of the membranes, presenting part, presence or absence of caput and moulding are noted. The

colour, smell and quantity of liquor and presence or absence of cord are also noted.

Speculum Examination

In obstetric speculum examinations, the bivalve Cusco's speculum is frequently used. Indications include antepartum haemorrhage, premature rupture of membranes, vaginal discharge and removal of a McDonald stitch.

The procedure and reasons for it are explained to the patient and verbal consent obtained. The patient is placed in dorsal or lithotomy position on the examination couch. The surgeon scrubs and wears sterile gloves. The vulva is swabbed as described above. The labia are then separated with the index finger and thumb of one hand to expose the vaginal introitus. The Cusco's speculum is then gently introduced into the vagina with the width of its blades transverse. The blades are then opened and the lateral walls are exposed and observed for any abnormality. The cervix is observed for the dilatation, bleeding, drainage of liquor or bulging of membranes. If the speculum examination is done to confirm premature rupture of membranes and does not show any liquor, the patient is asked to cough or fundal pressure is applied. The speculum is withdrawn in the same way it was introduced. During the procedure the patient is kept informed of each step as this makes the examination easy.

MANAGEMENT OF LABOUR

The main objective of labour management in our unit is to achieve delivery within 12 hours of admission for every mother admitted in active phase of labor.

First Stage of Labor

Patients in active or latent phase of labor are admitted in the first stage. Progress of labor is recorded graphically on a partogram where uterine contractions, fetal heart rate, maternal pulse rate and blood pressure are recorded every half hour. Vaginal examination to assess the cervical dilatation in cms, presence and degree of moulding and colour of draining liquor is done and recorded every 4 hours. Urine testing for proteinuria, ketones and glycosuria is performed each time the patient passes urine. An intramuscular injection of Buscopan 40mg is given to hasten cervical dilatation. In patients at cervical dilatation of 4 to 6cm an intramuscular injection of pethidine is given for analgesia.

The partogram has two parallel lines: the "alert line" and "the action line". The action line is 4 hours to the right of the alert line. At admission, for a patient in active phase of labour cervical dilatation is marked on the alert line and the time noted. Cervical dilatation of at least 1cm per hour is expected. Any deviation of cervical dilatation curve towards the action line is an indication of some abnormality in the progress of labour and corrective measures are instituted accordingly. Corrective measures may involve augmentation of labour if contractions are poor or caesarean section delivery if there is cephalopelvic disproportion (CPD). Augmentation of labour with syntocinon is done in those patients without a previous uterine scar, maternal or fetal distress and those who are not grand multipara. Induction of labour routinely starts in the morning and invariably done by artificial rupture of membranes followed by the syntocinon drip.

Management of Second Stage

When the patient is confirmed to be in second stage by vaginal examination and abdominal examination and also has the urge to bear own, she is transferred to the delivery room and placed on a delivery couch.

Normal deliveries are usually conducted by a midwife, student midwife or a medical student under instruction. High risk cases like multiple pregnancy, premature deliveries and breech presentations are delivered by the registrar in attendance. Strict asepsis is observed during the deliveries; sterile gowns and towels are used. The vulva and perineum are cleaned with antiseptic solutions (commonly savlon) and then the patient is encouraged to bear down with each contraction and to take deep breaths between contractions. Fetal heart rate is monitored every 15 minutes.

As the head distends the perineum, the left hand of the midwife maintains flexion of the fetal head and if episiotomy is indicated, 5-10mls of lignocaine are infiltrated on one side of the vulva and a mediolateral episiotomy performed using a blunt tipped Mayo's scissors. The perineum is supported by the right hand with sterile pad.

Once the delivery of the head has occurred, the mouth and nose are wiped with gauze to prevent aspiration of blood or amniotic fluid. A finger is swept around the fetal neck for the cord. If the cord is too tight around the neck it is divided between clamps and if it is loose it is slipped over the head. The anterior shoulder is delivered followed by the posterior shoulder and trunk. If the umbilical cord was not clamped, it is done so and the baby shown to the mother for sex identification before handing over to another midwife who carries out oropharyngeal suction if necessary. In high risk

cases, a paediatrician is usually in attendance. At delivery of anterior shoulder 10 units of syntocinon is given intramuscularly to the mother.

Management of Third Stage

Controlled cord traction is performed for delivery of the placenta after which delivery of the placenta, the perineum, vagina and cervix are inspected for any tear. The blood loss is estimated and the placenta and membranes are examined for completeness. If however, the placenta is not delivered by 30 minutes and there is no active bleeding then infusion of 20-30 units of syntocinon in 5% dextrose is used. If this measure fails, then the patient is prepared for manual removal of the placenta in theatre under general anaesthesia.

Repair of Episiotomy

This is carried out in three layers using chronic catgut suture No. 2/0. The apex of the incision is identified and from here repair of the vaginal mucosa is carried out in continuous suture while the muscle layer is approximated with interrupted sutures.The skin is apposed using interrupted or continuous chronic catgut No. 2/0 burying the knots and starting from the lateral edge. After repair the patient is advised on perineal hygiene and saline sitz baths.

The Fourth Stage

After delivery and repair of episiotomy, blood pressure, pulse rate, uterine contraction and lochia loss are observed and recorded. The patient is encouraged to empty the bladder. The patient is then observed for one hour

and then transferred to the postnatal ward for subsequent observations. Patients with normal delivery are discharged home after 24 hours due to pressure of bed space.

OPERATIVE DELIVERY

Vacuum Extraction

Vacuum extractor is used to accomplish delivery in prolonged second stage due to poor maternal effort or where bearing down is contraindicated as in cardiac disease or to expedite delivery in fetal distress occurring in the second stage of labour.

The procedure and its indication are explained to the patient and a verbal consent obtained. The patient is placed in lithotomy position. The vulva and perineum are cleaned with antiseptic solution and draped. Aseptic catheterization of the bladder is done and repeat vaginal examination performed to rule out any contraindication to vacuum delivery such as cephalo-pelvic disproportion and malpresentation. A mediolateral incision (episiotomy) is made under local anaesthesia. The largest suitable vacuum cap is passed against the fetal scalp taking care not to include maternal soft tissues by running a finger round the cap.

Suction pressure is then built up slowly upto a maximum of $0.8kg/cm^2$. This allows for formation of an artificial caput within the cap that holds firmly and allows adequate traction.

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 fetal head the pressure is released. The rest of the delivery is completed as described above.

Caesarean Section

The commonest caesarean section is the lower uterine segment caesarean section. Classical caesarean section is rarely done except for cases of transverse lie with ruptured membranes.

Pre-Operative Care

Caesarean section operations are either emergency or elective. For elective caesarean section, baseline investigations like haemoglobin level and urea and electrolytes are done, blood is taken for grouping and cross-matching and two units of blood are reserved and an informed consent for general anaesthesia and operation is taken. The patient is starved for at least six hours before the operation. The abdominal wall is shaved before theatre. Premedication with atropine 0.6mg is given intramuscularly half hour before theatre. For emergency caesarean section, blood is taken for grouping and cross-match and an informed consent for general anaesthesia and operation is taken. The abdominal wall preparation is similar to that of elective operation. The patient is premedicated with atropine 0.6mg intramuscularly before being wheeled to theatre.

Operation

In theatre the patient is placed in supine position with the legs separated, the vulva and perineum are cleaned with antiseptic solution such as savlon. Aseptic catheterization is done and the catheter is left in situ after draining all the urine. A repeat vaginal examination is done.

The anterior abdominal walls is cleaned with antiseptic solution and painted with iodine. The patient is then drapped with sterile towels. Anaesthesia is

induced with intravenous sodium thiopental. Succinylcholine 50-80mg is also given for temporary muscle relaxation to enable endotracheal intubation. Anaesthesia is then maintained with nitrous oxide, oxygen and halothane.

The abdomen is opened in layers either through a lower midline incision or through a Pfannenstiel incision depending on the surgeon's and/or patient's preference. After opening the skin, the rectus sheath is opened with curved Mayo's scissors. One side of the divided rectus sheath is elevated with two artery forceps and the rectus muscle separated from their attachment to it, using a surgical blade and then drawn to one side to expose the peritoneum. The latter is held in between two long artery forceps and opened. The incision is extended up and down to the incision limits taking care not to injure the bladder.

Wet abdominal packs are placed on either side of the uterus to prevent blood and liquor from running into the general peritoneal cavity. A Doyen's retractor is applied to retract the bladder away as well as expose the uterovesical fold of peritoneum.

The utero-vesical peritoneum is lifted up with a pair of dissecting forceps and incised. The incision is extended in an elliptical fashion downwards. The peritoneum is stripped off the lower uterine segment with mounted swab. The Doyen's retractor is shifted to include the lower part of the peritoneal fold in retraction of the bladder away 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 peritoneal fold. Once the membranes are reached or uterine cavity opened the incision is extended laterally on either side using curved scissors directed by two fingers of the

left hand. The incision is enlarged enough to allow delivery of the head and trunk. The Doyen retractor is then removed and the right hand is introduced into the uterine cavity under baby's head which is delivered gently out through uterine incision. Delivery is aided by gentle transabdominal fundal pressure. After delivery of the head, the mouth and nostrils are wiped with soft gauze. The shoulders are then delivered using gentle traction and still with some fundal pressure. The trunk follows readily. The umbilical cord is divided between clamps and the baby is handed over to a midwife or paediatrician. The placenta is delivered by either controlled cord traction or manually. The inside of the uterus is wiped with a swab on a holder. Bleeding margins of the incision are held by Green Armitage clamps. In transverse lie or breech presentation, the baby is delivered by breech extraction.

The uterine incision is then repaired in 2 layers with chronic catgut stitch number 2 or vicryl number 1 on atraumatic needle. The utero-vesical peritoneum is then closed with a continuous chromic catgut stitch number 1/0.

The abdomen is mopped and the abdominal packs are 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 abdomen is closed in layers. The peritoneum is closed with continuous No 1/0 chromic catgut stitch, rectus sheath is similarly closed with number 1 vicryl stitch and skin with interrupted silk or nylon. The wound is cleaned and then dressed. The catheter is checked for the urine draining and if clear the catheter is removed and the uterus is massaged and clots evacuated from the vagina.

General anaesthesia is reversed with 1.2mg of atropine and 2.5mg of neostigmine intravenously. Extubation is done and oropharyngeal suctioning done.

Post-Caesarean Care

The vital signs: blood pressure, pulse rate, respiration and body temperature are observed half hourly until the patient is fully awake then 4 hourly. Intravenous fluids are given until bowel sounds return after which she is started on oral sips. Pethidine 100mg is given every 8 hours for the first 24 hours to relieve the pain. She is also given antibiotics, Xpen 2mu 6 hourly and Gentamicin 80mg.8-hourly intravenously. Flagyl is added to those at risk of sepsis. On the first post-operative day the patient is ambulated and oral sips started if bowel sounds present. When she starts taking orally, medications are converted to oral medicines. On the third post-operative day haemoglobin is checked. The stitches are removed after seven days of operation. The patient is discharged home with a case summary and having been explained on the nature and findings of the operation and wound care. She is booked to be seen in the post-natal clinic after six 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 a paediatric registrar. Those having problems or who may develop problems are admited to New Born Unit (N.B.U). Premature babies are managed in NBU until a weight of 1900 g after which they are discharged. All babies are immunized with BCG before discharge. Normal mothers who have babies in NBU are lodged in the mother's hostel.

Post-Natal Follow-Up

The clinic is held every Friday. Only those patients who had complications or operative delivery are seen. Patients with normal deliveries are followed up in their nearest health facility.

Blood pressure and weight is taken and urinalysis performed. 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 contraception.

THE GYNAECOLOGY UNIT

The Gynaecology unit consists of the outpatient clinic (clinic 18) and two gynaecological wards 1B and 1D on the first floor of the tower block. Ward 1D is the acute gynaecological ward while ward 1B caters for non-emergency cases.

Gynaecological Outpatient Services

There are three outpatient clinics per week. Specific firms run the clinics on different days; Firm I on Tuesdays, Firm II on Thursdays and Firm III on Wednesdays. The clinics are run by consultants, senior registrars and registrars. Medical students are also taught during the clinics. There is also a colposcopy and oncology clinic on Friday morning.

The majority of the patients attending the gynaecology clinic are referred from casualty and emergency gynaecology ward after emergency management. Post-operative patients also attend this clinic. Some other patients are referred from other specialized clinics in Kenyatta National Hospital. The rest of the patients are referred from the district and provincial hospitals.

Infertility patients constitute about two thirds of the gynaecology consultation followed by uterine fibroids, abnormal uterine bleeding and oncology patients. In the clinic a thorough history and physical examination is conducted and most of the diagnostic investigations done. The investigations ordered depend on the impression after history and physical examination. Some of the investigations include: pelvic ultrasound, semen analysis, hysterosalpingogram, Pap smear and pregnancy tests among others. Patients requiring operative management have pre-operative investigations done from the clinic

Family Planning Clinic

The clinic is situated at the Family Welfare Centre (clinic 66). Here oral and injectable contraceptives, intrauterine contraceptive devices, implants (Jadelle) and barrier methods 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 minilaparotomy. Patients with infertility secondary to tubal factor are referred to this clinic for dye laparoscopy after HSG. However, currently the family welfare clinic is undergoing renovations and most of the functions of this clinic are run from clinic 18.

GYNAECOLOGY IN-PATIENT SERVICES

Acute Gynaecological Admissions - Ward 1D

This is an emergency ward with a bed capacity of 32 but usually has about 60 patients. It caters for all gynaecological emergencies seen and admitted at the Kenyatta National Hospital. About 15 patients are admitted daily and more than two thirds of these cases are abortion related. Patients are mainly admitted from casualty.

All patients for admission are clerked by the houseman and reviewed by the senior house officer (registrar) who undertakes the management in consultation with senior members of the department. Patients in the ward are reviewed daily by a registrar, senior registrar and consultant.

Apart from abortions, pelvic inflammatory disease and ectopic pregnancies are the next most common cases admitted into this ward. Uncomplicated cases of incomplete abortion have uterine evacuation done in the procedure room in 1D using Karman's cannula and syringe. They are discharged immediately after being counseled about contraception. Patients who have undergone emergency laparotomy for pelvic abscesses, ectopic pregnancy or pelvic masses are discarged home at least four days post-operatively.

Patients with carcinoma of the cervix undergo examination under anaesthesia (EUA), staging and biopsy as outpatients. After histology results are available, the patients are either admitted to the cold gynaecology ward for Wertheim's hysterectomy or referred for radiotherapy. Patients with carcinoma of the cervix are only admitted to the acute gynaecology ward with complications such as bleeding, severe anaemia or dehydration.

Cold Gynaecology Admissions - Ward 1B

Ward 1B is the non-emergency gynaecology ward where patients are admitted from the clinic or transferred from acute gynaecology ward for further management. The ward has a bed capacity of 33 beds. The beds are shared equally among the three firms. The patients commonly admitted in this ward are those for elective gynaecology operations or for chemotherapy for gynaecological malignancies. Uterine fibroids, vesico-vaginal fistulae (VVF), tubal infertility and gynaecological malignancies are among the conditions necessitating patients to be admitted to this ward.

GYNAECOLOGICAL OPERATIONS

Emergency gynaecological operations are perfomed daily. Laparotomy for ectopic pregnancies (ruptured and non-ruptured), pelvic abscesses; ovarian cysts and other tubo-ovarian masses are some of the operations perfomermed. Smaller procedures like diagnostic dilatation and curettage of the uterus, removal of misplaced intra-uterine contraceptive devices, marsupialization of bartholin's abscess and suction curettage are also performed here.

Elective operations are performed on Mondays for firm II while firms I and III operate on Thursdays. The operations are done from 8.00 a.m. to 5.00 p.m. The operations are usually performed under general anaesthesia as outlined below:-

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

Pre-Operative Preparations

Patients for emergency laparotomy are prepared for theatre on admission. Ruptured ectopic pregnancies are the most common indications for emergency laparotomy. In this case blood is urgently cross-matched and an intravenous drip of N/saline started. The abdomen is cleaned and shaved. An informed written consent is taken and premedication provided by atropine 0.6mg intramuscularly half before theatre.

For elective operations, baseline investigations such as haemoglobin level, urea and electrolyte levels are done before admission. The nature and purpose of the operation is explained to the patient and an informed written consent for the operation is obtained. Blood is ordered and reserved for the day of the operation. For most operations gut preparations is done by enema at 6.00 p.m. on the day before the operation and repeated at 6.00 a.m. on the operation day. The patient starves from midnight to morning of the day of operation. The skin over the area of operation is cleaned and shaved. Premedication is provided by atropine 0.6mg and pethidine 50-100mg intramuscularly half hour before theatre.

Post-Operative Management

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

Patients who have had uncomplicated laparotomy for hysterectomy, ectopic pregnancy, ovarian cyst etc are usually kept in the ward for 4 days. For the first 24 hours the patients are maintained on intravenous fluids. Oral fluids are given when bowel sounds are established. Blood transfusion is given when indicated. Pethidine 100mg 8 hourly for 24 to 48 hours is routinely given for analgesia. Prophylactic antibiotics are given routinely. A check hemoglobin level is determined on the third post-operative day.

Before discharge, the patient is informed about the findings at operation and a discharge summary issued. Patients are reviewed in the gynecology clinic after six weeks or earlier when indicated.

The most common acute gynaecological operation is laparotomy due to ruptured ectopic pregnancy while total abdominal hysterectomy (T.A.H) is one of the common cold gynaecological operations done in this unit. Total abdominal hysterectomy is described below.

TOTAL ABDOMINAL HYSTERECTOMY

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

The abdomen is opened in layers either through a pfanniestel incision or through a lower midline incision. The intestines are packed away from the intestines with wet gauze packs and a self-retaining retractor applied. The round ligaments are identified and beginning on either side using straight long artery forceps the round ligament is double clamped and divided between the two forceps. The lateral stump is transfixed with no.0 or no 1 vicryl. This procedure opens the anterior leaf of the broad ligament, which is pushed forwards through this opening with a surgeon's finger and incised with fingers. The same is done for the opposite side.

The next step depends on whether the tube and the ovary are to be saved or removed. If they are to be saved, the tube and the ovarian ligament are double clamped en masse and cut using a scalpel. The distal clamp holds the ovarian vessels as they approach the anastomosis with the uterine vessels. This stump is ligated using a transfixed vicryl no. 1 or no. 0. The same is

done for the opposite side. If the tube and ovary are to be removed with the uterus, the infundibulo pelvic portion of the broad ligament is double clamped with long curved artery forceps with the tips reaching the open window in the broad ligament. The ligament together with ovarian vessels is divided between clamps and ligated using vicryl no. 1. or 0. The same is done for the opposite side.

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

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

The uterus is retracted forward 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 uterosacral ligaments. The peritoneum is then incised with the scalpel and reflected, mobilizing it past the cervix to the posterior vaginal fornix. Care is taken not to dissect extensively laterally where the haemorrhoidal vessels are inserted into the rectum. Each uterosacral ligament is double clamped, cut and ligated with a No.1 vicryl suture. Here particular care is exercised to avoid the pelvic portion of the ureter as it courses along the base of the broad ligament. Next the cardinal ligaments on either side of the uterus are clamped, cut and ligated.

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

Suspension of the vaginal vault is done by tying the peritonization suture to the lateral and mid sutures of the vault. Peritonization is accomplished by means of a continuous No. 1. chromic catgut suture, that first pierces the vaginal walls near the midline and passes through the posterior leaf of the broad ligament, the free margin of the uterosacral ligament, then through the infundibulopelvic ligament, the free margin of round ligament and the anterior bladder peritoneum. The suture is tied at the center. The same is done for the opposite side with the suture being tied at the midline and

lateral angles. If the ovaries have been preserved an alternative suspension may be used in which the tip of the broad ligament is stitched separately with a purse string of No. 2/0 chromic catgut. The free margin of the pedicle is left high against the pelvic wall and is not anchored to the vaginal vault. This is advised in order to avoid subsequent dyspareunia and avoid stretching of the ovarian vessels with possible thrombosis, ischaemia and cystic changes of the ovary. After this abdominal viscera are well inspected. If haemostasis has been achieved, and instruments and swabs count are normal, the abdomen is closed in anatomical layers. Post-operative management is as described above.

COUNSELLING CLINICS

There are three such clinics in the hospital, which offer counselling to obstetrics and gynaecology patients. These are the Patient Support Centre, GOPC, High Risk Clinic and the Nairobi Hospice.

THE PATIENT SUPPORT CENTRE

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

THE HIGH RISK CLINIC (HRC)

This clinic is situated on the ground floor next to the maternity wards. It deals with young single mothers who have had an abortion, those who have delivered babies and even those who do not want to rear up their children.

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

THE NAIROBI HOSPICE

Workers here also offer counselling 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. Most of their patients have cancer of the cervix.

COLPOSCOPY

The gynecology clinic offers colposcopy training and services to both medical doctors and patients respectively. Patients with abnormal pap smears are referred to the clinic and colposcopic directed biopsies are performed or ablation of the abnormal zones using loop electrosurgical excision procedure is done. Cone biopsies can also be performed in the theatre as a therapy for patients who desire future fertility. Some patients with carcinoma opt to have a hysterectomy. Unfortunately many patients seen at the clinic present with cancer of the cervix that is too advanced for colposcopy.They are prepared at the clinic for EUA and biopsy as day cases in preparation for radiotherapy.

LAPARASCOPY

Diagnostic laparoscopy and laparoscopic surgery is available and operations such as tuboplasty, removal of ovarian cysts, hysterectomies and ectopic pregnancies are being performed.

THE MOTHER'S HOSTEL

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

THE HOSPITAL CHAPEL

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

OBSTETRICS CASE No. 1

RETAINED PLACENTA AFTER SPONTANUOUS VERTEX DELIVERY - MANUAL REMOVAL WITH GOOD OUTCOME.

NAME: M.O

AGE: 25 YEARS

IP NO: 0886891

D.O.A: 21.05.06 PARITY: 2 + 0

D.O.D:28.05.06

Presenting complaint

The patient was admitted as a referral from a peripheral clinic with 24 hour history of retained placenta following normal delivery to a 4kg live male infant. Attempts at removal in the clinic had been unsuccessful. She had a history of lots of per vaginal bleeding, and had been transfused 4 units of blood.

Obstetric and gynaecologic history

She was now para 2 + 0. 1st delivery 2000 , SVD to live female infant who is alive and well.

Her present pregnancy had been uneventful. She had attended her antenatal clinic at the hospital she had delivered. The antenatal profile were as follows:

Blood group O positive

Hb 11.5 g/dl

VDRL was negative

HIV had not been done.

Prior to conception, her periods were regular, having attained menarche at age 16 years. No history of use of contraception.

Past Medical and Surgical History

No previous history of admission or chronic disease.

Family and Social History

She was a housewife, who lived in Kayole with her child and husband. She did not smoke cigarettes or drink alcohol.

PHYSICAL EXAMINATION

She was in poor general condition, pale, afebrile, no jaundice, not cyanosed.

Her vital signs were as follows:

Blood pressure 100/60

Pulse rate 100/ min

Respiratory rate 25/min

Temperature 37.5^{0}C.

Abdominal Examination

There was a lower abdominal distension and the abdomen was moving with respirations. The liver and the spleen were not palpable. The fundal height corresponded to 24 weeks gestation.

Vaginal examination

The external genitalia were normal. The umbilical cord was seen dangling from the introitus with its distal end tied with a string. She was having minimal bleeding. The digital examination revealed an open cervix, about 4

cm, with placenta still attached in the uterine cavity. An attempt at delivery of the placenta by controlled cord traction was unsucceful.

Diagnosis

An impression of retained placenta was made.

MANAGEMENT

She was explained about the condition. An intravenous line was established and blood for grouping and cross- match drawn. She was started on 20 units of oxytocin in 500ml of 5% dextrose. She gave an informed consent in writing for manual removal of the placenta under general anesthesia. She received atropine 0.6mg intramuscularly and within 30 minutes, she was wheeled to theatre.

In the theatre she was given general anesthesia. She was placed in lithotomy position; vulva-vaginal toilet was done and then draped. She was catheterized and 200ml of clear urine obtained. Examination under anesthesia revealed a fundal height of 20 weeks gestation size. There were no tears or lacerations in the vagina or cervix. The cervix was about 4cm dilated and the placenta was still not separated. The left hand was placed on the abdomen to stabilize the uterus while the right hand was introduced into the uterus through the cervix following the cord. The lateral border of the placenta was identified and the hand insinuated under it, shearing it off its attachments on the uterus. The placenta was then delivered by the cord traction. It came out in one unit and on inspection found to be complete. It weighed 600gm. The uterine cavity was explored and found empty. Uterine massage was done and oxytocin drip continued, achieving good uterine contraction. The estimated blood loss was about 250ml. Anaesthesia was reversed successfully and the patient wheeled out of theatre for observation.

Post Operative Management

Intravenous infusion of oxytocin 20 units in 500ml of 5% dextrose was continued running at 40 drops per minute. She was started on metronidazole 500mg 8 hourly, crystalline penicillin 2MU 6 hourly and intravenous gentamycin 80mg 8 hourly, all intravenously. Her vital signs were observed ½ hourly until she was fully awake. Once fully awake, she was transferred to the postnatal ward where she continued with the antibiotics and monitoring of vital signs 4 hourly. She was also given mefenamic acid 500mg 8-hourly orally and started on ranferon syrup 10ml once daily. She remained stable all through. Vaginal bleeding remained minimal as expected of lochia loss. On the first postnatal day, she was found to be stable. She was moderately pale and the vital signs were normal. Check Hb was done and was found to be 7.6g/dl but asymptomatic. The patient was advised to continue with the hematinics started earlier. Her breasts were active and the uterus was well contracted, about 16 weeks by fundal height. The lochia loss was rubra and not malodorous. Meanwhile, her baby was brought from nursery and she was able to initiate breastfeeding. She remained stable in the second postnatal day. She was therefore discharged home on oral metronidazole, amoxicillin and mefenamic acid, to attend the maternal child health clinic nearest to her home, or to come back in the event she developed any complication.

Discussion

The patient presented was a para 2 + 0 after this delivery. She had delivered at home and brought into hospital following retention of the placenta for which manual removal was done. Twenty four hours had passed since the delivery of the baby to the time she presented to us. The placenta was

partially attached and she had bled a lot with history of being transfused 4 units of blood.

Delivery of the placenta constitutes the 3rd stage of labor. When the placenta is not expelled within the first 10 minutes after completion of the 2nd stage of labor, the situation is regarded as abnormal[1]. Whereas many authors regard a 3rd stage of labor longer than 10 minutes as abnormal and recommend manual removal after this interval, others wait for as long as 2 hours, more so if labor had been induced before term, before intervening[2,3]. Failure of the placenta to deliver spontaneously is an important cause of the postpartum hermorrhage. However, it has been shown that there is no increased risk until 30 minutes have elapsed and suggest that conservative management is appropriate during this interval[4,5].

The cause of prolonged 3rd stage is often not identified. Likely mechanisms include uterine atony, abnormal placental implantation and inadequate efforts to express the placenta. Several correlates of prolonged labor, augmented labor, induced labor and nulliparity. Abnormalities of the placental implantation such as placenta accreta might be expected in patients with previous endometrial curettage or caesarean deliveries. There is increased incidence of retained placenta among patients who had preterm labor. This patient did not have a clear etiological factor. Normally, the uterus should contract shearing of the placenta from the uterine wall and is spontaneously expelled. Spontaneous placenta separation is indicated when the umbilical cord lengthens and there is a gush of blood. In our unit, the policy is for active management of the third stage of labor. There is routine use of ergometrine as soon as the anterior shoulder is delivered. The use of oxytocic drugs routinely reduces the risk of post partum hemorrhage by

---

4,5

about 40%. A combination of oxytocin and ergometrine (syntometrine) is more effective in reducing the risk of postpartum hemorrhage than either used alone. In addition to use of oxytocic drugs in active management of third stage, pressure is applied to the body of the uterus and the umbilical cord kept slightly taut. The uterus is lifted cephalad with the abdominal hand. This is repeated until the placenta reaches the introitus after which it is lifted with the right hand. This is referred to as controlled cord traction. Mild traction on the cord is emphasized in fear of uterine inversion.

The patient who has had a retained placenta, with partial or complete separation of the placenta, is often in shock secondary to post-partum hemorrhage. Adequate resuscitation is mandatory, before attempting manual removal. This should include transfusion if the patient is bleeding and administration of oxytocin. The patient presented had post-partum hemorrhage as the placenta was partially separated from the uterus by the time she was being taken to theatre.

Manual removal is performed under anesthesia. The patient is placed in lithotomy position. One hand is placed on the abdomen, both to stabilize the uterus and stimulate it to contract and a last attempt is made to deliver the placenta with the Brandt- Andrews method of controlled cord traction. If this fails, the other hand should be insinuated through the cervical os and the retraction ring, if one is present, to the upper uterine segment following the cord to its insertion. The lower end of the placenta is then located from the uterus. When there is a total separation of the placenta, it is removed. Meanwhile an intravenous oxytocin drip is administered to promote uterine contraction ---. This is what was done for our patient.

Other methods of managing retained placenta remain controversial. The effect of intraumblical oxytocin on the retained placenta has not been agreed

upon. Some authors have found the method to be advantageous whereas others have not. It has been proposed that injection of intra-umblical oxytocin leads to a high concentration of oxytocin at the uterine wall.

Risk of manual removal of placenta include post-partum hemorrhage following partial detachment of a morbidly adherent placenta, weakening of the wall with subsequent rupture, uterine infection and inversion. The patient presented did not develop any of the complications. She was put on antibiotic prophylaxis.

REFERENCES

1. Zuspan F.P. Quilligan E.J retained placenta, placental cotyledons and membranes in: Strommes Operative obstetrics. Appleton and Lange (publishers) 5th ed.pp587, 1988
2. Golan A., Lidor A.L, Wexeter S.D A new method of management of the retained placenta. Am J. Obstet. Gynecol 146, 708, 1983
3. Kirz D. S., Hagg M.K. Management of the third stageof labor in pregnancies by prostaglandin E. Am. J. Obstet. Gynecol 160:416, 1989.
4. Combos C.A., Laros R.K. Prolonged third stage of labor: Morbidity and Risk factors Obstet. Gynecol 77 (6), 1991
5. Ratnam S.S. Rauff M. Post- partum hemorrhage and abnormalities of 3rd stage of labor In: Obstertics: Churchill Livingston (publishers) Ch 59; pp 872, 1989.

OBSTETRIC CASE NO. 2

MALARIA IN PREGNANCY – REMISSION WITH THERAPY

Name: N.A.A

DOA: 29/09/06

Age: 30 years

DOD: 02/10/06

Parity: 2+0 gravida 3

File No: 0985507

LMP: 29/01/064

EDD: 05/11/06

GBD: 34^{+}/40 weeks

Presenting Complaint

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

History of presenting complaints

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

Obstetric and Gynanaecologic History

She was para 2+0 gravida 3. Her last normal menstrual period was 29/01/04, the expected date of confinement was 05/11/04 and gestation by dates was 34 weeks plus 6 days. She had had antenatal care (ANC) at the Makadara city council health center from 26 weeks. The antenatal profile done then was Hb of 11.2 g/dl, blood group B+ve and negative HIV and VDRL serological tests. Hitherto the pregnancy had been uneventful. She had received the first dose of 3 tablets of sulphadoxine-pyrimethamine (SP) comination. She had had two uneventful spontaneous vertex vaginal deliveries to two male infants both at term gestation and in a health facility. The first was in 2000 and the infant weight 3000 grams. The second was exactly one year later in 2001. During the second pregnancy she had symptomatic anaemia and was treated with haematinics. The infant weight was 2.9 kg. Both children were alive and well. She attained he menarche at the age of 15 years and since then her menses flowed for 3-4days every 28days without any menstrual disorder. She had used three monthly injections for medroxyprogesterone acetate from 2001 to June during which she became ammenorrhoeic. No pap smear test had been donead neither had she contracted any sexually transmitted disease.

Past medical history

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

Family and social history.

She was a married housewife who had attained secondary education, drank no alcohol and did not smoke cigaretted. She lived with her

children and husband in Kibera, Nairobi. The husband worked with a plastic manufacturing factory in industrial area, Nairobi, and did not consume alcohol or tobacco. There was no history of chronic illness in the family.

PHYSICAL EXAMINATION

General examination

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

Pelvic examination

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

The nervous system

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

The respiratory and the cardiovascular systems were essentially normal

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

DIAGNOSIS

Malaria in pregnancy with mild anaemia at 34+week's gestation in a 30year old

Para 2+0.

MANAGEMENT

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

- Haemogram: Hb:10.1g/dl, platelets:230x10^9/L, WBC count: 6.7x10^9/L
- Widal test: Negative
- Urine analysis, culture and sensitivity: No abnormality noted.
- Repeat blood smear test for malaria parasites was negative.

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

Follow up

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

DISCUSSION.

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

Derived from the words mal (for bad) and air, Malaria is caused by protozoa of the plasmodium species that parasitize the red blood cells and the liver after finding their way into the blood circulation through the bite of an infective female Anopheles mosquito. There are four species of plasmodium that can infect man, namely P.falciparum, P.ovale, P.malariae and P.vivax of the four, P.falciparum is associated with the most severe forms of malaria and the worst disease outcome.[1, 2] P.falciparum is also the predominat species that causes malaria in most parts of Kenya as well as in the rest of WEastern and Southern Africa where it is responsible for 90% of cases. The other species cause the remaining cases although P.vivax is very rare [2]. Our patient had P. falciparum malaria.

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

region while Nyamogo reported a prevalence of 42% in Kisumu in the Lake Victoria region [6, 7]

Areas where there is a constant repeated infection are said to be hyperholoedemic. This includes the Kenyan coastal and lake regions. The populations in these areas have high immunity and epidemics do not occur here (stable malaria). [2] In the regions like Aberdare ranges and Mount Kenya areas, transmission is intermittent as there is poor community immunity and epidemics do occur (unstable malaria).[2] Our patient lived and was brought up in Nairobi (unstable transmission) and has traveled to Nyanza where she contracted malaria. Pregnant women resident in areas of unstable malaria are at 2-3 fold risk of developing severe disease as a result of malaria infection than are non-pregnant adults living in the same region.[4]

Both humoral and cellular immunity are involved in the development of immunity against malaria. This immunity is maintained by intermittent parasitaemia. Cellular immunity is in the form of phagocytosis by macrophages while humoral factors involve the production of specific antibodies.[8] Individuals living in endemic areas are therefore usually less susceptible to infection except during periods when the immunity is impaired. Pregnancy impairs immunity against malaria so that even in the hyper-endemic regions where tolerance to the parasites has previously been acquired, infection readily occurs. The increased propensity to malaria may be as a result of high cortical levels found in pregnancy as well as the decreased cellular immunity especially seen in the third trimester. The glycoproteins of pregnancy have also been implicated by their inhibition of the transformation of monocytes into macrophages. Additionally, sequestration of the parasites in the placenta shield them

from destruction by maternal effector cells.[9] Multiparity appears to conver some protection against this increased susceptibility during pregnancy such that the breakdown in immunity is most marked during the first pregnancy. However, this only holds true for those who have developed immunity.[1,2] Our patient was para 2+0 in the 3rd trimester and was not exposed to persistent malaria challenge necessary for her to mount the semi-immunity found in those living in endemic areas.

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

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

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

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

Fetal complications due to plasmodium falciparum malaria during pregnancy include increased chances of abortion, prematurity, intra-uterine growth restriction and infant low birth weight, which is the single risk factor for death in the 1st month of life.[12] Malaria has been estimated to cause 8% to14% of all low birth weight babies and 3% to 8% of all infant deaths in areas of Africa with stable malaria transmission.[3] Impaired foetal growth results from reduced placental blood circulation

in the intervillus space that develops from placental parasitization. The foetus is usually protected from acquiring malaria in the uterus by the placental barrier, circulating maternal antibodies and the fact that the foetal haemoglobin (HbF) is more resistant to the parasite. However, congenital malaria in endemic areas is estimated to be <1%. A study in Malawi detected parasites in 35% of cord blood of infants whose mothers were infected with malaria.[14] In Africa, however, clinical disease is rarely seen in neonates.

Management of malaria is both specific and supportive. The treatment is dependent on the severity of the disease the geographical area and the local pattern of drug resistance. Chloroquine is the drug of choice in chloroquine-sensitive areas. The aim of treatment in malaria is to reduce pyrexia and stop the attack as quickly as possible.[10] Patient with severe malaria are hospitalized and given parenteral quinine treatment. Those with milder forms of malaria are given 4-amnioquinolenes, chloroquine and amodiaquine as drugs of choice in areas where there is no resistance to these drugs. The Quinhosa derivatives such as artemesinin or artemether may also be used after the first trimester as was the case with our patient.[1,6,7,10] WHO recommends that the following drugs should not be used in pregnancy: Halofantrine, Primaquine, Tetracycline and Doxycycline.[15]

Anaemia responds rapidly in most patients following anti-malarial therapy and folic acid, but the haemotocrit does not rise in patients with hyperactive malarious splenomegaly.[10] Blood transfusion is indicated only if the patient is in incipient or established cardiac failure or if the patient is approaching delivery with an Hb<7g/dl. Mothers with severe forms of malaria in labour may need shortening of the second stage of

labour by assisted vacuum delivery. Care is taken to avoid postpartum haemorrhage or cardiac failure that may occur in moderately or severely anemic mothers after delivery. Active management of third stage is recommended.[2, 10] After successful treatment; malaria chemoprophylaxis is necessary thoughout the remaining period of pregnancy including peurperium as happened with our patient. This clears and prevents placental parasitization.

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

The objective of IPT is to provide all pregnant women with at least two preventive treatments of an effective anti-malaria drug; one in the 2nd trimester and another in the 3rd trimester. This approach has been shown to be safe, inexpensive and effective. One study in Malawi evaluating IPT showed a decline in placental infection (32%to23%) and in the number of low birth weight babies (23%to10%). It also found that 75% of all pregnant women took advantage of IPT when offered.[15] Insecticide-Treated Nets (ITN) decreases both the number of malaria cases and

malaria death rates in pregnant women and their children. A study in an area of high malaria transmission in Kenya has shown that women protected by ITNs every night during their first four pregnancies produce 25% fewer underweight or premature babies.[15] In addition, ITN use also benefits the infant who sleep under the net with the mother by decreasing exposure to malaria infection.

REFERENCES

1. Matheus D., Cunnl P.Plamodium Facliparum in pregnancy: A review. Brit.j.Obstet and Gynaecol 1992;99:118

2. National guidelines for diagnosis, treatment and prevention for malaria for health workers, MOH, Republic of Kenya, 1998, Revised in 2003.

3. World Health Organization. Expert committee on malaria, 20th report. Technical report series No.892. Geneva 2000

4. An Analysis of malaria in pregnancy in Africa. Bulletin of the World Heath Organization. 2002, 80(5)

5. Guyatt. H.L. Abdalla JS. Snow R.W. The impact of malaria in pregnancy in Kenya. 21st African Health Sciences Congress 28th -29th Aoril 2000. KEMRI headquaters, Nairobi.

6. Rukaria K., Ojwang S.B.O., Oiyeke J.B. Resestance to chloroquine therapy in pregnant women with malaria parasitaemia. Int. J.Gynaecology and obstetrics, 1996; June; 53(3): 235-41

7. Nyamongo M.A. Malaria in pregnancy in an endemic area in Kenya.M.Med thesis UoN 1992.

8. White N.J., Brewer J.G Malaria and other red blood cells parasites: in Fanci A.S., Braum W.G., Issabele K.J. et al. (editors). Harrison's principles of international medicine 14th Edition. Volume 1. McGraw Hill Comp Inc. New York. 1998, p.1180.

9. McGregor I.A., Wilson M.E. Malaria infection of the placenta in the Gambia, West Africa: its incidence, relationship to still birth, birth weight and placenta weight. Trans R. Soc Trop Med. Hyg. 1982; 7721:232.

10. Nicholas J.W.Mlaria in: Gordon C. (Ed) Manson's Tropical Disease. 20th edition. 1996, p.114-115, 393-394, 1109.1143.

11. Looreaguwen S., Philip R.E., White N.J. et al., Quinine and severe malaria iin pregnancy. Lancet 1998; 2:4.

12. Sullivan A., Nyirenda T., Malraia infection during pregnancy. Intrauterine growth retardation and pre-term delivery in malaria J.infect. Dis 1999; 179(6): 1580-3.

13. Steketee R.W., Bradly B. In vivo response of P. falciparum to chloroquine in pregnant wimen in Siaya District, Kenya. Bull WHO. 1987; 5(6) 889

14. Redd S.C., William J.J. Transplacental transmission of P.Falciparum in rural Malawi. Am. J. Trop. Med. Hyg. 1996; 55:57-60.

15. World Health Organization Roll Back Malaria. Strategic framework for malaria control during pregnancy in the WHO Africa region, Nov 2002.

16. Division of malaria control National malaria strategy. Ministry of Health, Nairobi; April 2001.

OBSTETRIC CASE NO.3

CORD PROLAPSE, EMERGENCY CAESAREAN SECTION: LIVE BABY

Name: V.W.R DOA: 30/1/07

Age: 31 years DOD: 3/2/07

Parity: para 1+0 gravida 2 File No: 1127039

LMP: 18/4/06

EDD: 25/1/07

GBD: 40 weeks + 5days

History of presenting complaint

V.W.R was admitted to the Kenyatta National Hospital labour ward with pains for 6 hours. She had been well until she developed lower abdominal pains that were intermittent and increased in frequency and intensity and radiated to the lower back. There was an associated show but no drainage of liquor or urinary symptoms. Fetal movements prior to this had been unaltered.

Past Obstetric and Gynaecology History

She was a para 1+0 gravida 2 at 40 weeks +5days. Her LMP was 18/4/06 and her EDD was 25/1/07. In 1997 she had spontaneous vertex delivery of a life infant who weigh 3500 grams and had an Apgar of 8 and 10 in 1 and 5munites, respectively and was alive and well. Pre-intra and post-partum periods were uneventful then. She had used the combined contraceptive pills until 2005 when she stopped them in order to conceive. A pap smear done in 1998 was normal. She attained menarche at 14 years and had normal menstrual flow for 3-4 days every 28days.

History of the present pregnancy

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

Past medical history was insignificant.

Family and social history

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

PHYSICAL EXAMINATION

General Examination

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

Abdominal examination

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

Pelvic examination

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

Diagnosis

Cord Proplapse at term in a 31 year-old para 1+0.

MANAGEMENT

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

The theatre staff was ready for the emergency C/S. the surgeon withdrew his hand while a nurse inserted her hand into the vagina. While the surgeon scrubbed general anaesthesia was administered. The usual catheterization was not done. Before the nurse withdrew his hand he confirmed that the cord was still pulsating and auscultation with the fetosope showed fetal heart tones at 126 beats per minute that were beginning to be irregular. Quick abdominal cleaning and draping

proceded abdominal opening via a sub-umbilical incision. Without the usual packing of the paracolic gutters, a lower uterine section c/s and cephalic extraction delivered a live male infant who weigh 3550 gram and anApgar score of 7 at 1 minute and 10 at 5 minute. Meconeum staining liquor grade I was noted. The placenta the cord and the membranes were delivered by continous cord traction. There were grossly normal. Uterine repair in two layers achieved haemostasis. After receiving a report of correct account of instruments, gauzes and needles, the abdomen was closed in layers, the skin with 3/0 monocryl. The reversal of the general anesthesia was smooth.

Post operative care

She was transferred to the recovery room where vital signs were observed ½ hourly. One hour later she was fully awake and was therefore transferred to the postnatal ward. Recovery was good. On the 1st postoperative day, she had passed urine twice, was in good general condition and had normal vital signs. Breasts, abdominal, pelvic and calf examination were all essentially normal. Ambulation, oral sips of warm water to graduate to light diet by midday and oral antibiotics were started. She opened bowels on the 2nd day and was put on normal diet by midday and oral antibiotics were started.

She opened bowels on the 2nd day and was put on normal diet. Her check Hb on the 3rd day was 10g/dl and breastfeeding had been established. She was ready for discharge home but she opted to stay an extra one-day. The baby and herself were doing well when they were discharged home on the 4th day on oral antibiotics and haematinics. She was to visit the postnatal clinic 1 week later.

Follow up

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

DISCUSSION

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

Umbilical cord anomalies could be divided into developmental and accidental disorders. Developmental disorders include anomalies of length (normal range 30-100cm) vascular number (2 arteries+ 1vein) and insertion (usually singly at the centre of placenta). Accidental abnormalities consist of true knots, loops round parts of the body mainly the neck, torsion, prolapses and strictures.[1] The "accidents" occur as a result of fetal movement. Umbilical cord prolapse (UCP) is defined as descent of the umbilical cord into the lower uterine segment to the level of or beyond the presenting part when the membranes are ruptured. It should be distinguished from cord presentation where the descent is the

same but the membranes are intact. There are two types of UCP: Occult (descent at the level of the presenting part) and OverT (cord lies below the presenting part). Umbilical cord prolapse is one of the most serious obstetric emergencies because of the very high perinatal morbidity and mortality caused by fetal exsanguinations by compression of the cord between the foetus and the uterus, cervix, or pelvic inlet.[2]

The incidence of UCP reported in the literature ranges from 0.14 to 0.62 percent and has not changed in years[3]. However, in the USA, perinatal mortality related to UCP, which was as high as 375 per 1000 births in the early 1900s, has fallen to between 36and 162 per 1000 births within the past few decades[4]. The causes or predisposing factors are those which lead to inadequate application of the presenting part to the cervix and those due to obstetric interventions. Usta et al in 1999 reported that obstetric interventions contributes to nearly half of cases of UCP[5]. These interventions include amniotomy, scalp electrode application, intrauterine cathether insertion and external cephalic version. Our patient had UCP secondary to artificial rupture of the membranes (amniotomy). It is possible however that occult presentation existed before amniotomy. Factors that lead to inadequate filling of the cervix by the presenting part are prematurity, malpresenttaion, malposition and minor degree of placenta praevia and cephalopelvic disproportion. Others are hydramnios, multiple gestation and premature rupture of the membranes. Malpresentation is one of the leading causes of UCP. It is present in to 41% of UCP[6]. The incidence of overt cord prolapse in cephalic presentation in the USA is 0.5%; in frank breech0.5% in complete breech 5% in footling breech 15% and in transverse lie 20% however the majority of UCPs occur with the vertex presentation because of the low

frequency of malpresentation[2]. None of these risk factors were obviously present in our patient

Preterm deliveries have a higher rate of UCP, probably due to smaller size of the presenting fetal parts and the increased frequency of malpresentation among premature fetuses. Babies with birth weight less than 1250grms, for example, had a 19-fold increased risk of UCP in one series while the risk of UCP in a term twin pregnancy is confined to the second born twin, in whom there is an increased probability of malpresentation [6 7] Multiparous women have a higher risk of UCP maybe due to increased likelihood of rupture of membranes (ROM) prior to engagement of the presenting part, since engagement in multiparas often occurs after labour has begun and later than in nulliparas [5] On the other hand the risk of cord prolapse during expectant management of patients with premature rupture of memberanes (PROM) is small, but should be considered due to the potential for an adverse outcome. As an example, a review of nine studies that included 731 patients with PROM reported a 1.9 percent incidence of cord prolapse.[5] Polyhydramnios is often associated with an unstable lie. Rupture of membranes may be followed by a forceful gush of fluid that carries the cord ahead of the unengaged foetus and through the cervical os. Mean cervical dilation and station at the time of UCP are 5.8cm and 1.6, respectively, although the ranges of values are broad.[9] Although there was no evidence of this in our patient, there was a gush of liquor on amniotomy at 6cm cervical dialation. There was no evidence of other risk factors.

The diagnosis of UCP is by inspection of an already prolapsed cord beyond the vaginal introitus, digital palpation and having index of suspicion based on the fetal presentation and cardiotographic findings

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

A protruding overtly prolapsed cord must be placed into the vagina to minimize vasospasm and desiccation but where this is not possible warm packs are gently wrapped around it. Other methods which have been used to displace the fetal head from compressing the cord include rapid filling of the urinary bladder with 500 to 700mls for normal saline and concomitant intravenous administration of tocolytics such salbutamol or

ritodrine and funic (cord) reduction by lifting the fetal head form the vagina and digitally elevating the cord above the widest part of the vertex so as to place in the nuchal area. The patient should be put in oxygen. These measures are important especially if delay on delivery is inevitable as when the patient is to be transferred to another centre for delivery.[3, 9]

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

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

REFERENCES

1. Benirschke K., Kaufman P. Pathology of the human placenta, 4th edition. New york, springer, 2000
2. Karen K., Collea J. Malpresentation and cord prolapse. N: Current Obstetrics and Gynmaecologic Diagnosis and treatment: 9yh edition, Appleton and lange publications, USA 2003.pp382-386
3. Kooning P et al. Umbilical cord prolapse- A contemporary outlook, journal of reproductive Medicine: 35-690-92:1990
4. Panther M.R., Hannah M.E. Umbilical cord prolapse: so far so good? Lancet; 1996 347:74.
5. Usta I.M., Mercer B.M., Sibai B.M. Current obstretrical practice and umbilical cord prolapse American Journal of Perinatology, 1999; 16:479?
6. Critchlow CW, Leet TL, Benedett, TJ, Daling JR Risk factors and infant outcomes associated with umbilical cord prolapse: a population based case contro study among births in Washington State. Am J Obstet Gynaecol 1994; 170:613?
7. Altars M, Potasnik G, Ben Adereth N, Leventha, H. the use of vacuum extraction in cases of cord prolapse during labor. A J Obstet Gynaecol 1974; 118 824.
8. Prabulos AM, Philipson EH. Umbilical cord prolapse. Is the time from diagnosis to delivery critical? Reprod Med 1998; 43:129.
9. Dutta D.C Malposition Malpresentation and cord prolapse. 3rd Edition. New Central Book Agency India. Pp 411-41: 1997.

OBSTETRIC CASE NO.4

ANTE-PARTUM HAEMORRHAGE:PLACENTA PRAEVIA. EMERGENCY CEASAREAN SECTION: LIVE BABY.

Name: J.W.N LMP: 10/10/05

Age: 28years EDD: 17/07/06

Parity: 1+0 Gravida 2 GBD: 29+Weeks

File No: 095960 DOA: 05/05/06

DOD: 03/07/06

Presenting Complaint

J.W.N presented with sudden onset of painless vaginal bleeding for 2 hours.

History of presenting complaint

Our client had been well until she developed the vaginal bleeding at about 5.30am on 5/5/06 soon after coitus with her husband. The bleeding was without associated abdominal pain, drainage of liquor or external trauma. There was no prior per vaginal discharge or urinary symptoms. The initial blood was frank red, fresh, profuse with clots and tracked down her lower limbs despite use of several pads. She still perceived foetal movements and had no history of any bleeding disorder. She had visited Huruma Nursing Home in Nairobi, thrice from about 20 week's gestation. Antenatal profile indicted Hb of 12.0g/dl, blood group O+ve and negative HIV and VDRL serology tests. Her pregnancy had been hitherto uneventful.

Past Obstetric and Gynaecologic History

J.W.N was a para 1+0 gravida 2 with no living child. In 1999 she had a premature spontaneous vertex delivery at about 61/2 months to a live female

infant who weighed 1.6kgs and survived respiratory distress syndrome but died of pneumonia at 1year of age. The predisposing factor(s) of preterm labour were unknown. Her menarche was at 15years, had regular menstrual periods each occurring every 28days and had had no sexually transmitted infections. She had used depot medroxy-progestrone acetate injections for three years after her 1999 delivery during which her periods were scant but remained regular.

Past medical history

This was insignificant.

Family and social history

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

PHYSICAL EXAMINATION

General Examination

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

Abdominal Examination

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

Speculum Examination

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

Respiratory, Cardiovascular and Central Nervous Systems.

These were essentially normal.

Diagnosis

A diagnosis of Ante-partum Haemorrhage (APH) probably due to placenta praevia at 29+ week's gestation was made.

Investigations.

They included:

- Urgent blood grouping and cross match of 2 units and a haemogram that showed Hb of 11.0g/dl, WBC of $6.5x10^9$/L and adequate platelets.
- An emergeny obstetric ultrasound showed a single intrauterine pregnancy in cephalic presentation at an average computed gestation age of 29 weeks+5days. The placenta was on the left lower uterine segment and covered the uterine internal cervical os. This was a major type of placenta praevia.

MANAGEMENT

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

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

In theatre general aneathsia was induced then quick aseptic catheterization was done and 150 mls of clear urine drained and per vaginal bleeding noted o be mild.The abdomen as cleaned and draped and opened through a pfannenstiel incision. Lower uterine segment caesarean section was performed via an elliptical incision on the upper margin of the LUS. A fast but cautious incision was made through the placenta and the amniotic membranes. By cephalic extraction, a live male infant was delivered and the cord quickly clamped to prevent fetal blood loss through the incised and partially separated placenta. The birth weight was 3000g and the Apgar score was 8 at 1minute and 10 at 5minutes. The placenta (which was located on the left anterolateral site of the lower segment of the uterus, covering the

entire internal cervical os) and the umbilical cord were delivered manually. It appeared grossly normal. Heavier than usual uterine bleeding was quickly arrested with double layer uterine repair, uterine massage and 40units of syntocinon infusion. The estimated blood loss was 750mls. She had smooth reversal of general anaesthesia after routine abdominal closure and vulvo-vaginal toilet as described for caesarean sectioin under 'introduction'.

Post –operative care

Recovery from surgery was good. She was put on nil per os, intravenous fluids and antibiotics and intra-muscular pethidine. On her first post-operative day oral amoxicillin, mefenamic acid, haematinics and oral sips to graduate to light diet were commenced. She had mild pallor and had passed urine three times. She had normal vital signs and was, therefore, mobilized and breast feeding initiated. Wound dressing was removed on her 3rd post – operative day and the wound was clean and dry. Her check Hb was 9.2g/dl. She was discharged home on the 4th post operative day.

Follow-up

She was reviewed in the postnatal clinic on the 2nd post-operative week. The breasts were active without abnormality. The wound had healed well and the uterus was involuting well and corresponded to 14 weeks. Lochia was serosal and not foul smelling. Advice on proper breastfeeding and family planning options was given and the patient asked to revisit the c linic on her 6th postnatal week. On the 6th postnatal week the patient was no longer pale and had normal vital signs. The uterus was no longer palpable and lochia loss had stopped. She opted for oral contraceptives and progestin only (microlut) was prescribed for her. Advice was given to her to report to the Family Welfare Clinic for the combined oral contraceptive pill and Pap smear after 6months.

DISCUSSION

J.W.N was a 28year old para 1+0 who was admitted with antepartum haemorrhage due to major placenta praevia (type III) at 29+ weeks gestation. By emergency caesarean st 37 weeks she delivered a live male infant with birth weight of 3000g and a good Apgar score. The mother and her infant did well postoperatively.

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

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

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

These are:

Type 1: (also called lateral pp, low lying placenta). The placenta is implanted in the lower uterine segment but does not reach the cervical os.

Type 11: (marginal placenta praevia). The placenta extends up to the margin of the internal cervical os.

Type 111: (partial placenta praevia). The placenta partially covers the internal cervical os.

Type 1V: (total or complete placenta praeavia). The placenta completely covers the internal cervical os and is centrally placed.

Placenta praevia has also been classified into major (requires delivery by caesarean section types 11b-1V) and minor (vaginal delivery is possible; type 1). Our patient had type III or major type of placenta praevia.

In literature, the overall incidence of placenta praevia ranges from 0.25% to 1%[1, 2, 5, 6, 7, 8]. Thirty years ago Ojwang [5] in 1974 found an incidence of 0.25% at the Kenyatta National Hospital while Kirima and Mbithi found similar incidence of 0.9% and 1% in 1981 and 1983, respectively in the same hospital [6, 7]. Mbithi used ultrasonography and perhaps that explains the higher prevalence relative to the other incidence rates. In the USA similar incidence rates have been documented. Overall 1 in 200 births (0.5%) is associated with placenta praevia in the USA, though the reported incidence rates range from 0.26% to 0.55%[1, 2, 7]. Although the aetiology of placenta praevia is unknown, it is postulated that defective vascularization probably resulting from inflammation or atrophic changes could be the cause. Certain factors, however, have been associated with increased risk of placenta praevia. These include previous (lower uterine segment) caesarean section,

multiparity, advanced maternal age, anaemia, erythroblastosis, succenturiate lobe or placentae membraneceae, cigarette smoking and increased placenta area due to multiple gestation.[1,2,3] Frederiksen and associates in 1999, attributed the increase of preavia from 0.3% in 1976 to 0.7% in 1997 to a shift to an older obstetrical population while Babinszki et alreported an incidence of 2.2% for para 5 or a greater which was significantly increased compared to women of lower parity[7,8]. In Sweden, Nielsen and colleagues found a 5 fold increased incidence of placenta previa in women with previous caesarean deliveries[9]. William and associates found the relative risk of placenta previa to be increased 2 fold in women who smoked cigarettes [10]. Our patient had no apparent risk facto though coitus apparently provoked the bleeding.

The cardinal sign of placenta praevia in 90% of patients is painless vaginal bleeding, which occurs rarely at the end of the second trimester but commonly in the third trimester, as was the case in our patient. Initial cramping occurs in 10% of cases. Spotting during the first and second trimester or just before a torrential bleeding is not uncommon. Our patient had no history of spotting but had an episode of profuse bleeding of frank fresh blood with clots at 29^{+} weeks gestation and again at 37^{+} weeks gestation. Bleeding from placenta praevia may be caused by mechanical separation of its implantation site, either during the formation of the lower uterine segment (by Braxton Hick contraction) or during dilation and effacement in labor, or during intravaginal examination. Our patient started bleeding after coitus with her husband. Placentitis and rupture of poorly supported venous lakes in the deciduas basalis are other possible causes of the bleeding. Inlike abruption placenta, the abdomen is usually soft, the uterus is soft, non-tender and the fetal parts are easily palpable with a high

presenting part. There is a high prevalence of malpresentation in women with previa due to the low-lying placenta displacing the presenting part. Oblique or transverse lie is found in 16%of patients. [1, 2] A deeply engaged presenting part, therefore, highly suggests a minor degree of placenta praevia. Cusco's speculum (and not digital) vaginal examination is used to confirm intra-uterine bleeding and/or rule out non-obstetric causes of APH.

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

The management of placenta praevia depends on the fetal maturity, the type f praevia, the local infrastructure and the degree of haemorrhage. Initial admission is mandatory for all cases where blood is taken for grouping and cross match and comprehensive evaluation made. Women with preterm fetuses and no active bleeding are managed conservatively. Bed rest analgesia and tocolytics if there are cramps and or signs of labor, transfusion (if the initial bleeding was heavy) and haematinics are prescribed. If

maturity is between 24 and 34weeks as was the case in our patient, intramuscular corticosteroids such as betamethasone or dexamethasone are given. The current recommended protocol is either intramuscular betamethasone12mg 24 hours apart (total 48mg) or intramuscular dexamethasone 6mg 12 hours apart for 2 days (total 48mg). This regiment is not repeated as a repeat administration of corticosteroids is associated with increased maternal sepsis (endometritis) and fetal growth (liver and brain) restriction, adrenal insufficiency, sepsis and placental infarction and neonatal necrotizing enterocolitis. Thus our weekly corticosteroid administration, as earlier practiced, was inappropriate. The aim of conservative management is to attain 37 complete weeks. Studies in the USA indicate that there is no significant difference in perinatal and maternal morbidity and mortality between carefully selected patients managed at home and those who are hospitalized. [1, 12]

Patients with access to immediate transportation to hospital in the event of haemorrhage could be allowed home and be re-admitted at about term for delivery. Expectant management is abandoned if there is active labour, uncontrolled bleeding and premature rupture of membranes or fetal death.

Though the delivery method of choice for all placenta praevia is caesarean section, type 1 and type11 anterior could be delivered vaginaly.[1,2] The two types require EUA and amniotomy in a "double set up" situation and subsequent induction of labor with syntocinon. This has been abandoned in the USA[1]. Emergency or elective caesarean section is performed for types II posterior, III, IV and even type I where bleeding is excessive. One should be prepared for post partum haemorrhage (PPH) with blood and equipment and staff for hysterectomy. PPH may occur due to poor contraction of the less mascular and more fibrous lower uterine segment and/or placenta increta

that is common in patients with previa. In such cases, mattress suturing, oxytocin, prostaglandins or methylergonovine should be used. Hysterectomy is the last resort. Although low uterine incision is commonly used, classical incision may be required to secure sufficient room in case of poor lower uterine development and to avoid incision through the placenta and the potential infant anaemia.[1, 2]

Malpresentation in up to 38.3% of cases, premature rupture of membranes (11%), cord prolapse (1.7%), increased perinatal and maternal morbidity and mortality in the USA[1,2]. intrauterine fetal restriction and unexplained fetal anomalies have been reported in 20% and 2.5% respectively.[1,2]. none of these complications occurred in our patient.

REFERENCES

1. Claydon C., Pernoll M. Third Trimester vaginal bleeding. In: DeCherney A., Nathan L (eds) Current Obstetric and Gynaecologic dianosis and treatment; 9th edition; Appleton and Lange, 2003 pp 354-66
2. Cunningham F et al. Obstetric Haemorrhage. In: Williams Obstetrics, 21st edition; The McGrow Hill Comp. 2001.pp 630-35
3. World Heath Organization: clinical management of abortion complications. WHO/FHE/SMS/94.1.
4. Barron SL. Bleeding in pregnancy: placenta praevia. In: Chamberlain G.(ed) Turnbull Obstetrics, 2nd edition. Churchil Livingstone, Edinbergh, 1996p789.

5. Ojwang's S.B.O. placenta praevia in KNH. M.Med Thesis, University of Nairobi, 1974.

6. Kirima J. Characteristics of patients with antepartum haemorrhage. M.Med Thesis, 1981.

7. Mbithi D.L.K. Gray scale ultrasound in the diagnosis of placenta praevia at Kenyatta National Hospital M.Med. Thesis University of Nairobi, 1983.

8. Fredericksen M.et al. placenta praevia of 22 year analysis. American journal of Obstetrics and Gynaecology. 1999; 180:1432.

9. Babinszki A. et al. Perinatal outcome in grand and great –grand multiparity: effects of parity on obstetric risk factors. Am J Obstet Gynaecol. 1999; 181:669.

10. Nielsen T.F. et al. placenta praevia and antepartum haemorrhage after previous caesarean section. Gynacol Obstet Invest. 1989; 27:88.

11. Laing F.C. Ultrasound evaluation of obstetric problems relating to lower uterine segment and cervix. Sonography in obstertrics and gynecology. In: Principles 5th edition. Appleton and Lange. 1996, p720.

12. Smith R.S. et al Transvaginal ultrasonography for all placentas that appear to be low-lying or over the internal cervical os. Ultrasound Obstet gynaecol 9:22, 1997.

13. Drost S.Keil K.Expextant management OD placenta praevia: Cost benefits analysis of outpatient treatement. Am J Obstet Gynaecol 170: 1254, 1994.

OBSTETRIC CASE NO. 5

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

Name: J.N.K DOA: 16/09/06

Age: 21 years DOD: 19/09/06

Parity: 0+0 gravida 1 File No: 0983627

LMP: 13/12/05

EDD: 20/09/06

GBD: 39 weeks+3 days.

Presenting complaint

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

History of presenting complaint

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

Antenatal care

Her antenatal care had been at the Riruta Health Centre from 20 weeks gestation. An obstetric ultrasound done then showed that she had a twin gestation. Both twins were without any anomaly and appeared binovular. Her antenatal profile was Hb 11.3g/dl, blood group; O+ve and HIV and VDRL tests were negative. Her prenatal period was uneventful save for

the pressure symptoms she experienced during her late 3rd trimester. Her blood pressure remained within normal limits and her weight gain was 11.3kg. In view of the twin gestation she was to undergo elective caesarean section at 38 completed weeks and was to report to a hospital with a functional operative theatre as soon as she developed labor pains. She had initially opted to undergo the caesarean section at Pumwani Maternity Hospital.

Post obstetric history

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

Past medical history was not significant.

Family and social history

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

PHYSICAL EXAMINATION

General examination

JNK was in good general condition. She had no pallor, pedal edema, jaundice or lymphadenopathy. Her height was 155cm. her BP was 120/75mmHg, PR; 80minute, temperature 36.7^{0}c and RR was 18 minute.

Abdominal examination

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

Pelvic examination

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

Diagnosis

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

Management

She was admitted to labor ward and preparation for emergemcy caesarean section (C/S) instituted. Her pubic hair was shaved, informed consent obtained, blood take for grouping and cross match, theatre team alerted and she was not to eat or drink anything before the operation. Intramuscular atropine was administered before she was wheeled to theatre. While being catheterized, she had spontaneous rupture of the

membranes with subsequent prolapse umbilical cord. Quick abdominal toilet and opening via a sub-umbilical midline incision and lower uterine segment caesarean section as described under introduction, led to the delivery of the twins. The first twin was a live female infant delivered by breech extraction with a birth weight of 2800grams and an Apgar score of 6.9 and 10 at 1,5 and 10 minutes respectively. The second twin was a live male infant delivered by cephalic extraction with a birth weight of 2300grams and an Apgar score of 7, 9 and 10 at 1, 5 and 10minutes respectively. They had distinct placentas, cords, chorions and amnions. The senior house officer in the pediatrics department reviewed both twins. They had no anomaly and were to join the mother as soon as she was able to take care of them the rest of the c/s was as per the description under introduction. The estimated blood loss was 500 mililiters.

Postoperative care

Her recovery from both the general anaesthesia and the surgery was good. She received intravenous crystalin pencilin and gentamicin and intramuscular pethidine for 24 hours. Ambulation and oral sips were started 8 hours after surgery followed by light diet 6 hours later. On the 2nd pos-operative day she and her infants were doing well. She was mildly pale with normal vital signs. She was commenced on oral antibiotics, analgesics haematinics and breast feeding advice given. The breast-feeding advice included sitting on firm sit with back support while breast feeding, taking plenty of warm fluids such porridge and tea, a balanced diet based on the available and acceptable foodstuffs locally, proper breast feeding technique and breast care. The lach-on technique whereby the entire nipple and most of the areola are placed in the baby's mouth was demonstrated. On the 3rd day she was in good general

condition. Lactation had been established, the uterin size corresponded to 18weeks' gestation, the wound was clean and dry, lochia was scanty rubra and the calves were soft and non-tender. A check Hb done then was 9.8g/dl. She was discharged home with the oral medicines and advice to attend the postnatal clinic in 6 weeks or earlier if there was a problem. She was also advised on contraception and asked to discuss the same with her spouse.

Follow up

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

DISCUSSION

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

Multiple or multifetal pregnancy is the occurance of two or more embryos/fetuses in one pregnancy. Twin-gestation therefore is the presence of two fetuses in one pregnancy. Twins occur in 1 of 100 pregnancies of white women, 1 of 80 pregnancies of black women and in only 1 of 155 pregnancies in Asian women.[1] Monozygotic twining

occurs in 1 in 250 births and is independent of race, heredity, age and parity. Dizygotic twinning is affected by these factors and by use of fertility drugs. In general the frequency of dizygotic twins is low in Asians, intermediate in whites and high in blacks. The Yorubas of western Nigeria have a frequency of 45 twins per 1000births and about 90% are dizygotic twins.[2] Oyieke found a high twinning rate at Kenyatta National Hospital of one in 59 births (1.7%)[3]. In a later study Mutungi found an incidence of 2.2% of all deliveries at KNH and Pumwani Maternity Hospital[4].

Worldwide, the incidence of twin and higher order multiple gestations has increased significantly over the last 15 years primarily due to the availability andincreased use of ovulation inducing drugs and assisted reproductive technology (ART). In USA, multiple gestation now comprises 3% of all pregnancies and twin comprise 25-30% of deliveries resulting from ART.[5] Monozygotic twins result from a single fertilized ovum and comprise slightly more than 30% of all twins. Dizygotic twinning is the result of fertilization of two separate ova. The two ova are released from separate follicles or rarely form the same follicle at approximately the same time. They comprise nearly 70% of all twins. The timing of monozygotic division has important implications. Division within the first 72 hours after conception results in a diamniotic, dichorionic monozygotic twin pregnancy. This occurs in 30% of twins with a mortality rate of 9%[1]. Division 4-8 days after fertilization results in a diamniotic, monochorionic twin pregnancy. It's the most frequent of the monozygotic twining about 68%, with mortality as high as 35% due to complications of vascular anastomoses within the placenta[1]. Division during days 8-13 results in monoamniotic, monochronomic twins. These

monozygotic twins occur least often (2%) but with a high mortality rate of up to 50%. Division at two weeks of fertilization after the amniotic sac and embryonic disk have been formed results in conjoined twins.[1] Our patient had dizygotic twins in view of the different sexes and less importantly, different aniotic sacs and separate placentas. Monozygotic twins without chromosomal anomalies are always of the same sex and usually (but not always) share the same physical characteristics; same genetic features and could be mirror images of one another. However, because monozygotic twins result from 'teratogenic' division of one ovum, the incidence of chromosomal and therefore, phenotypic inequalities in monozygotic twins is higher. They are therefore less identical than the dizygotic twins and their fingerprints differ[5] Chromosomal anomalies can result in heterokaryotypic monozygotes for instance; where one twin has Downs Syndrome and the other twin is normal.

The caused of monzygotic twinning is unclear. However, conventional ovulation induction methods and use of other ART [6, 7] Methods have resulted in an increase on monozygotic twinning. In addition, evidence suggests that delayed transport through the tuibe increase the risk twinning. Progestational agents and the combined contraceptives were shown by Bressers and associated (1987) to increase twinning in pregnancies conceived in close proximity with contraceptive use [8]. Dizygotic (fraternal) twins maybe of the same or different gender. About 75% of dizygotic twins are of the same sex with both twins being males' .Dizygotic twinning occurs most often in black, than whites and least common in Asians. Dizygotic multiple pregnancies tends to be recurrent and women who have borne dizygotic twins have a 10-fold increased

chance of subsequent multiple pregnancies. Age also increased the rate of twining with advancing age of over 35 years having an increased risk. Parity doesn't influence the incidence twinning. Dizygotic twinning is also more common among women who become pregnant soon after cessation of long-term oral contraception. The use of ART and ovulation induction methods increases multiple pregnancies with higher order pregnancies being more common. Besides the black race and the use of combined oral contraceptives, our patient had no other risk to twinning.

Early diagnosis of multiple gestations in the ante partum period is important for specialized care. Undiagnosed multiple gestation presents special problems of management and ultimately contributes significantly to higher perinatal and maternal morbidity. Oyieke reported that in 38% of twins seen at KNH, the diagnosis was made either in labour or after delivery of the first baby [3]. Multiple gestations should be suspected whenever the uterus seems lager than dates, hydramnios or unexplained maternal anaemia develops, auscultation of more than one fetal heart is suspected or when pregnancy has occurred following ovulation, induction or in-vitro fertilization. Ultrasound diagnosis is a simple, safe and effective tool in diagnosis of multiple gestations. Cetrulo (1980) argued that universal ultrasound screening for all pregnant women during the second trimester would result in early diagnosis of multiple gestations with almost 100% accuracy[9]. This had been validated by the Results of the Routine Antenatal Diagnosis Imaging with Ultrasound (RADIUS) study from which the separate gestation sacs can be identified ultrasonographically as early as six weeks from first day of last menstrual period. It's important however to visualize separate fetuses with independent cardiac activities. Ultrasound can help determine the

zygotism. If fetal gender can be identified on ultrasound, twins of the opposite sex are almost always dizygotic. Separate placentas and a thick separating membrane greater than 2mm between the twins also probably points towards dizygosity. In the absence of these findings, monozygosity is likely [1,5] In our patient diagnosis of twins was made by ultrasonograpy by the 20th week of gestation.

Maternal serum alfa-fetoprotein (MSAFP) screening at 14-20 weeks has been used to screen for multiple gestation. The median MSAFP level will be 2.5 times that of the medial level for singleton pregnancies at a similar gestation.[1,5] It is three times and four times higher in triplets and quadruplets respectively.[1,5] The hematocrit and haemoglobin values and red cell count are considerably reduced in direct relationship to the increased blood volume. Maternal hypochromic normocytic anaemia occurs so frequently in multifetal pregnancy that it has been suggested that all patients with the process be suspected of having a multiple gestation.[5]

Glucose tolerance tests show that gestational diabetes mellitus and gestational hypoglycaemia are much higher in multiple gestations compared to singleton of the same gestational age. [10] After 32 weeks, the combined weight gain of both twins is approximately equivalent to that gained by a singleton for the remaining portion of the pregnancy[10] The median weights of twins at birth is just over 2270 grams in the USA.[5] Our patient delivered low birth weight babies weighing 2300 and 2800 grams at 39 + weeks.

Antepartum care for twin pregnancy includes an early diagnosis of twin pregnancy, which is associated with improved perinatal outcome.[11] Ultrasound diagnosis may be used as early as the 4th week of pregnancy.

The mother should make frequent antenatal visits at least every fortnight from 20-36 weeks.[1] She will need diet supplementation with additional calories at least 300calories per day above the normal pregnancy requirements, protein intake of up to 80grams per day and falote at least 1 milligram per day. She will require extra bed rest in hospital is controversial.[12, 13, 14] Preterm labour should be anticipated and recognized early and aggressively trated if it occurs. Prophylactic administration of tocolytics to these women with twin pregnancy has been tried with varying degrees of success. Since an increased incidence of maternal cardiovascular complications has been reported in women with multiple gestations treated with B-agonist, it seems prudent to restrict the use of these agents to women who are confirmed to be in latent labour.[15]. Results of studies using prophylactic cervical cerclage in these women with multiple gestations have been disappointing. [16] It's now recommended that cervical cerclage placement should be restricted to women with documented cervical incompetence. Cervical assessments score based on the length of cervical canal minus the dilation of the internal os in centimeters, ultrasonogrphic assessment of cervical length and fetal fibronectin have been validated as useful adjuvant tests in the prediction of pre-term labour in multiple gestation. [17]

Fetal growth should be monitored by serial ultrasound examinations to detect discordant fetal growth and intrauterine growth restriction. The serial ultrasound should include monitoring fetal well-being by use of non stress tests. Blake and others confirmed that antepartum nonstress testing is a highly reliable and predictive test in assessment of multiple gestations[18]. It is recommended that ultra sound follow-up be done four weekly during the 3rd trimester and more frequently if intrauterine growth

restrictions is encountered [5].Pregnancy induced hypertension and pre-eclampsia, should aggressively be looked for and managed effectively to reduce both maternal and fetal morbidity. Other less common complications such as polyhdraminios, twin to win transfusion should be looked for and managed effectively. The parents should also be advised to make plans for two babies at home. Our patient did not have any of the complications associated with multiple gestation such as polyhydramnios, pregnancy induced hypertension and anterpartum hemorrhage among others.

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

delivery.[19] Twin A non vertex with twin B having any presentation should be delivered primarily by caesarean section [19] Locked twins may occur if twin A is breech and twin B is vertex. Our patient had first twin in breech presentation and hence the decision to deliver the mother primarily by caesarean section.

REFERENCES

1. Nina M. Boc M. John G, McFee. Twin pregnancy in: Obsygyn secrets, 2nd edition, 1997. edited by Helen L., Frederickson and Louise Wilkins-Haug pages 235-240.
2. Marivate M., Norman R.J. Twins. Clinical Obstet Gynaecol, 1982; 9:723.
3. Oyieke J.B.O. A two and half year review of some aspects of twin delivery at Kenyatta National Hospital. M.Med Thesis, Obstetric long commentary, University of Nairobi, 1978.
4. Mutungi A.K. A prospective study of twin deliveries at Kenyatta National Hospital and Pumwani Maternity Hospital- The antenatal care and delivery. M.Med Thesis, Obstetric long commentary, University of Nairobi, 1990.
5. Melisa B., Martin L.D. Multiple pregnancy in: Current obstetric and gynaecologic diagnosis and treatment, 9th edition, 2003. Edited by Alan H. Dercheny and Lauren Nathan pages 315-325.
6. Deron C., Deron R., Vlieneth R. Increase monozygotic twinning rate after ovulation induction. Lancet 1987; 2:1236.
7. Wernstrom K.D., Syrup H., Hammit D.G. Increased risk of monochromic twinning associated with assisted reproductive. Fertile steril, 1993; 60:510.
8. Bressers W.M.et al. increasing trend in the monozygotic twinning rate. Acta Genet. Med. Gemellol (Roma) 36:397, 1987.
9. Cetrulo C.I., Ingardia C.J., Sharra A.J. Management of multiple gestation. Clin Obst Gynaecol, 1980; 23:533.

10. Lefeure M.I.., Bain R.P., Favigman B.G. Arandomized trial of prenatal ultrasonographic screening. Impact on maternal management and outcome. Am J. Obstet Gynaecol, 1993; 169:483?

11. McKeownT. Recod R.A.G. Observation of a fetal growth in multiple pregnancy in man. J. Endocrinol, 1952; 8:386.

12. Daw E., Waclter J. Growth difference in twin pregnancy. Br. J. Clin pract 1975; 29:150.

13. Hartikainen-Sorri A.L., Joupilla P. is routine hospitalization needed in antenatal care of twin pregnancy, J. Perinat Med; 1984; 12:31.

14. Saunders M.C., Dick J.S., Brown I.M. The effects of hospital admission for bed rest on the duration of twin pregnancy. A randomized trial. Lancet 1985; 2:793.

15. Katz, Robertson P.A., Creasy K. Cardiovascular complications associated with terbulatine treatment of preterm labour. Am. J. Obstet Gynaecol, 1981; 139:605

16. Cameron A.H. The Birmingham twin survey. Proc. R.Soc. Med. 1968;61:229

17. Houlton N.C.C., Marivate M., Philpoh R.H. Factors associated with preterm labour and changes in the cervix before labour in twin pregnancy. Br. J. Obstet Gyneacol, 1982; 89:90.

18. Goldenberg R.L., Iams J.D., Miodvonik M. The preterm prediction study: Risky factors in twin gestations. National Institute of child health and development maternal fetal medicine units Network. Am.J.Obstet Gynaecol, 1996; 175:1047.

19. Blickstein I et al. Delivery of the second twin in breech presentation. Obstet. Gynecol. 67:774, 1987.

OBSTETRICS CASE NO.6

CARDIAC DISEASE IN PREGNANCY GRADE IV. SUCCESSFUL VAGINAL DELIVERY.

Name L.M

Age 36 years

Para 2 + 3

IP NO. 1051956

LMP 14. 06. 06

EDD 21. 03. 07

D.O.A 04. 10. 06

D.O.D 06. 04. 07

Presenting Complaints

Admiited as referral from Kitui district hospital for cardiac disease management in pregnancy. She was a known cardiac patient since August 2004, being followed up at Machakos for ? cardiomyopathy. She was not in failure and was on digoxin, captopril and inderal.

History of Present Illness

Patient was diagnosed as having cardiomyopathy in August 2004. She had presented with history of dizziness, palpitations and bilateral pedal oedema. Chest x-ray and ECG done then showed features of cardiomyopathy. She was put on captopril, digoxin and inderal which she had been on till time of admission at KNH. The diagnosis of cardiomyopathy was later changed to rheumatic valvular disease with pulmonary hypertension after echo on cardiologist review.

Past Medical History

Other than the cardiac problem she had been admitted with, the patient had had no other chronic disease or surgery.

Obstetric and Gynaecological History

She was para 1 + 3. Had 3 successive abortions before getting a normal delivery in 1999 who is alive and well. She had not started to attend ANC at time of admission.

She attained menarche at 15 years and her menses lasted for 3 days in a 28 to 30 day regular cycle. She had used oral contraceptive pills for three months prior to conception. Her last menstrual period was on 14th /06/06 and her expected date of delivery on 21st/03/07. She was at a gestation of 16 weeks on admission

Family and Social History

She was a married mother who had a small business of selling clothes in a boutique at Kitui. She did not smoke cigarettes or drink alcohol. There was no family history of heart disease or other chronic illnesses.

PHYSICAL EXAMINATION

She was sick-looking, had no pallor, cyanosis, pedal oedema or finger clubbing. She had a blood pressure of 134/70 mmHg, respiratory rate of 20/min, pulse rate of 80/min that was regular and a temperature of 36.8^{O} C.

Cardiovascular system

She had a regular pulse of 80 beats per minute that was of good volume. The jugular venous pressure was raised and the precordium hyperactive with a

tapping apex beat at the 5th intercostal space on the anterior axillary line. The first and second heart sounds were heard and there was a pan-systolic murmur best heard at the apex.

Respiratory system

She had good breath sounds bilaterally with no crepitations or rhonchi.

Abdominal examination

She had a lower abdominal distension. The abdomen was moving with respirations, she had no hepatomegally or splenomegally. The fundal height was corresponding to 18 weeks of gestation. There were no areas of tenderness.

Pelvic Examination

This was not performed as there was no immediate indication for it.

Investigations

1. Echocardiogram showed moderate to severe mitral stenosis and regurgitation with mild pulmonary hypertension.
2. Electrocardiogram showed a left axis deviation
3. Chest radiograph showed cardiomegally with features of mitral valve disease.
4. Urine microscopy culture and sensitivity – no microscopic haematuria. *Staphylococcus epidermidis* was grown on two separate occasions
5. Haemogram – WBC 6.5 x 10^9/l
   Hb 11.3 g/dl Platelets 265x10^9/l
6. VDRL was negative
7. HIV test was negative

DIAGNOSIS

A diagnosis of cardiac disease in pregnancy grade 4 but functionally grade 3 was made.

MANAGEMENT

She was propped up in bed and managed in consultation with cardiologists. She was continued on monthly intra-muscular benzathine penicillin 2.4 mega units, digoxin 0.25 mg once daily, lasix 40 mg once daily, atenolol 50 mg once daily, spironolactone 25 mg once daily and haematinics (Ranferon®) 10 ml three times daily.

The patient remained stable. Twice she had urine culture growth of Staphylococcus epidermidis which was sensitive to Augmentin. She was successfully treated with Augmentin on both occasions.

On 24. 03.07 at 38 weeks gestation it was decided that she be induced with PGE_1 pessaries. She was then transferred to labour ward where the pessary was inserted and shortly thereafter went into spontaneous labour.

Management in Labour Ward

She was propped up and given oxygen by mask. She was given pethidine 100mg intramuscularly for analgesia and started on prophylactic antibiotics, benzyl-penicillin G 2 mega units 6-hourly and Gentamicin 80mg 8-hourly intravenously.

On vaginal examination, the external genitalia were normal. The cervix was fully effaced, soft, and central and 4 cm dilated. There was no moulding or caput formation and the umbilical cord was not palpable. There was clear liquor draining.

An emergency tray containing aminophylline, digoxin, frusemide, sodium bicarbonate and calcium gluconate was prepared and kept ready. A vacuum extractor was also kept in readiness for use during the second stage of labour.

Vital signs, foetal heart rate and contractions were monitored half hourly and charted on a partograph. She progressed well in labour and 6 hours later was fully dilated and transferred to the delivery room. She was placed in semi fowler position with legs supported by stirrups. Vulvovaginal toilet and aseptic catheterization was done and clear urine obtained. Local anaesthesia (10 ml of lignocaine 2%) was infiltrated into the left side of the vulva. A left mediolateral episiotomy was performed. The medium (50mm) ventouse cap was applied to the foetal vertex and gently, a vacuum created; by gentle traction with the first uterine contraction, the baby was easily delivered. A live female infant who scored 4/1, 7/5 and 9/10 and weighed 2200 g was delivered.

The placenta was delivered by controlled cord traction and weighed 420g and was normal and complete. The uterus was messaged until well

contracted with minimal blood loss. There was no need for oxytocin. The episiotomy was repaired in layers after inspection revealed no cervical or vaginal tears. Estimated blood loss was 200mls. Post delivery, the patient was given 80mg of intravenous frusemide. Her respiratory rate was 24 per minute, pulse rate 82 per minute and blood pressure of 100/70mmHg. She was taken to labour ward acute room for observations.

Post Partum Management

The vital signs were observed half hourly for the next 24 hours. They remained within normal. After 24 hours she was transferred to the postnatal ward where 4 hourly observations were continued. She was put on intravenous Augmentin 1.2 grams 8 hourly for one week and continued on Digoxin 0.25mg daily and Lasix 40mg orally once daily.

She was reviewed by the cardiologists on 1st April 2007 and found to be doing well with no complaints. She was advised to continue with Lasix and Digoxin. She was discharged home on 6th April 2007 through the cardiac and postnatal clinic in two weeks.

Postnatal Review

She was seen in the postnatal clinic and found to have no complaints. She was in good general condition and not in cardiac failure and the uterus had involuted well. She was counselled on family planning and advised on tubal ligation but she opted to abstain for the time being. She was discharged from the postnatal clinic through the cardiac clinic.

DISCUSSION

The patient presented was a 38 year old married mother known to have cardiac disease admitted at 18 weeks gestation and managed as an inpatient until after delivery. She had an assisted vacuum delivery at 38 weeks of

gestation to a live female infant who had a good Apgar score and weighed 2200 grams.

Cardiac disease complicates 1-2% of all pregnancies and is the most important non-obstetric causes of maternal death [1, 2, 3]. Sequeira and Ojiambo in 1969 at Kenyatta National Hospital found an incidence of 0.5% with 95% of cases being of rheumatic heart disease (RHD) in origin, 35% of the RHD cases had mitral stenosis.[3]

In a later study Ngotho reported an incidence of 0.99% [4] again with 86.4% due to Rheumatic heart disease and 12.9% congenital heart disease [4]. These results are similar to other studies from the African region where Rheumatic heart disease predominates. [5]

Rheumatic heart disease is the commonest heart disease in pregnancy in our set up in contrast to the developed world where congenital heart disease predominates. However, with improved medical services and advancement in cardiac surgery, some women with congenital heart abnormalities will not only survive to reach the age of childbearing but also carry a pregnancy to term successfully [5].

Pregnancy is associated with major haemodynamic changes in the cardiovascular system that can contribute to greater morbidity and mortality in women with underlying heart disease. Therefore, the management of these disorders in the pregnant patients requires understanding of cardiovascular physiology during pregnancy, labour, delivery and the puerperium.

The management of heart disease in pregnancy is dictated by functional capacity of the heart and special emphasis should be placed on prevention and early detection of heart failure. The degree of functional disability is

graded according to the following New York Heart Association classification:

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

Grade II: Slightly compromised. The patients have cardiac disease and slight limitation of physical activity. The patients are comfortable at rest but ordinary physical activity causes symptoms.

Grade III: Markedly compromised. Patients have cardiac disease with marked limitation of activity. The patients are comfortable at rest but symptoms occur with less than ordinary physical activity.

Grade IV: Severely compromised. Patients have cardiac disease with symptoms even at rest.

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

The management calls for team approach involving obstetrician, cardiologist and anaesthesiologist [1,2,6,7].

Grade I and II patients are managed as outpatient after initial clinical evaluation. They are seen frequently by both the cardiologist and obstetrician as their grades may change to higher grades and present with complications. At 36 weeks they are admitted to await delivery. Grades III and IV patients are usually confined in the wards until after delivery [1,2,6,8]. Our patient was confined to the wards until after delivery.

Restriction of maternal physical activity tends to avoid cardiovascular compromise and improves uteroplacental perfusion [9]. The supine position should be avoided as pressure on the inferior vena cava reduces venous

return. Haematinics are recommended for the prophylaxis against anaemia or its vigorous treatment when it occurs.

Respiratory infections must be treated with antibiotics and oxygen liberally given if respiratory difficulties develop.

It is imperative to await the spontaneous onset of labour since induction is associated with significant haemodynamic changes that could precipitate cardiac failure and in case of failed induction caesarean section carries an added risk of pneumonia, infective endocarditis and pulmonary oedema and embolism [8]. However caesarean section should still be performed if there is an obstetric indication [5].

Relief from pain and apprehension without undue depression of the infant or mother is especially important during labour and delivery. Epidural anaesthesia and narcotic analgesics are preferable. The mother should be kept in a semi recumbent position in bed and oxygen given by mask if need be. The patient should be started on parenteral antibiotics and for grade III and IV patients Digoxin and frusemide administered. Monitoring of vital signs, auscultation of lung bases are important to detect early signs of congestive cardiac failure. A tray containing aminophylline, digoxin, morphine, sodium bicarbonate and frusemide is kept, ready for use if need arises. Vaginal delivery should be aimed at with shortening of the second stage of labour by use of assisted vacuum delivery. Active management of the third stage with ergometrine should be avoided and oxytocin used if bleeding is excessive otherwise uterine massage minus drugs is encouraged.

A bolus intravenous injection of frusemide 40 to100mg is given late in first stage to offset the anticipated cardiac output increase from the placental bed.

Close obstetric and medical surveillance must continue particularly during the first 24-48 hours and infection guarded against especially infective endocarditis. Early ambulation is necessary to prevent deep venous thrombosis and the attended risk of pulmonary embolism. A period of 10 to 14 days postnatal observation is recommended [8].

Before discharge contraception should be discussed with the patient. Barrier methods and progesterone only pill is advisable for those who desire another child or do not opt for tubal ligation [1, 2, 6]. When the family size is complete tubal ligation is the optimal choice. Alternatively vasectomy can be offered to the spouse if desired.

REFERENCES

1. Cunningham FG, Gant NF, Leveno KJ, Gilstrap LC, Hauth JC, Wenstrom KD. (Eds). Cardiovascular Diseases. In: Williams Obstetrics, 21st Edition, McGraw Hill, 2001:1181 – 1203.
2. Grewal M, Biswas MK, Perloff D. Cardiac, Haematologic, Pulmonary, Renal and Urinary Tract; Disorders in Pregnancy. In: DeCherney AH, Nathan L. Current Obstetric and Gynaecologic Diagnosis and Treatment 9th Edition McGraw-Hill Companies 2003; 387-409.
3. Sequiera R. F.; Ojiambo H.P. The heart in pregnancy. EAMJ 46 (1); 18, 1969.
4. Ngotho D. K. Cardiac disease at Kenyatta National Hospital. Mmed Thesis University of Nairobi, 1982.
5. Schoon M.G; Bann R. H.; Wolmarams L. Heart disease during pregnancy. S. Afri. Med. J. Feb 1997; 87 Suppl. IPC 1922.

6. Thomas R.E, Catherine O. Heart Disease. Obstetrics - Normal and Problem pregnancies 4th Edition, Churchill Livingstone 2002; 1005-1030.

7. Whitfield C. R. Heart Disease in pregnancy. Dewhurst's textbook of Obstetrics and Gynaecology for postgraduates 4th Edition. Blackwell Scientific publications ELBS Lincoln. 1986; 242.

8. Rush R.W; Fraser P.J; Coomeford J. Management of heart disease in pregnancy. S. Afr. Med. J., 61: 192, 1982.

9. Veland K, Novy M.J., Metcalfe J. Haemodynamic responses in patients with heart disease due to pregnancy and exercise. Am. J. Obstet. Gynecol. 113: 46, 1972.

OBSTETRICS CASE NO. 7

GESTATIONAL DIABETES MELLITUS AND IATROGENIC HYPOCALCAEMIA, CAESERIAN SECTION AT TERM, LIVE BIRTH

Name: T.S.S DOA: 16/03/06

Age: 35 years DOD: 16/04/06

Parity: 2 + 0 Gr 3 File No: 0873952

LMP: 06/07/05

EDD: 13/04/06

Presenting Complaints

T.S.S was admitted from the antenatal clinic at 36 weeks with poor control of blood sugar on diet alone. She had been found to have a random blood sugar of 8.1mmol/1 at booking at 29 weeks gestation. An oral glucose tolerance test showed she had impaired glucose tolerance in pregnancy. A dietician helped her develop a diet that would enable her control the blood sugar. She was seen weekly at the clinic for 4 visits; her pre-prandial blood sugar was 6 mmol/1. On the seventh visit, her fasting blood sugar was 8.1 mmol/1 and random pre-prandial, 10.2mmol/1. She had no polyuria, polydipsia or polyphagia. She had no dysuria and was taking calcium supplements

Antenatal care

TSS was booked at 29 weeks gestation at KNH and was found to have impaired glucose tolerance. She had 6 clinic visits prior to admission.

Antenatal profile

Booking Hb: 11.4g/dl, VDRL: Negative, HIV: Negative, Blood group: A+

Urinalysis: Glucosuria

Obstetric and Gynecology history

She was Para 2 + 0 gravida 3.

1st delivery: 2000, SVD, female 3600g who is alive and well

2nd delivery: 2003, C/S due to macrosomia, male 4350g, alive and well

The patient had 6 year period of secondary sub fertility characterized by hyperprolactinaemia, hypothyroid goiter, hypercortisolaemia and

acromegaly. She was put on bromocriptine for 2 years and was able to conceive.

Medical and surgical history

TSS was diagnosed with multiple endocrinopathy in the year 1999 characterized by elevated TSH, prolactin, growth hormone and cortisol. She had acromegalic features and a hypothyroid goitre. She was put on prolactin to enable her conceive. She underwent a partial thyroidectomy in 2000 for the goiter. After the surgery, she developed hypoparathyroidism for which she was put on calcium and vitamin D supplements. She was still on supplementation.

Social History

She was married and worked as a clerk in a local company. Her spouse was a technician with the National Telephone Company. They lived in Kayole in Nairobi. She did not smoke nor take alcohol. There was no family history of diabetes mellitus or hypertension. There was no family history of thyroid disease.

Examination findings

TSS was in good general condition, without pallor or jaundice or oedema. She had thickening of the noise and coarse moist skin. She was obese and weighed 108 kg at admission. She also had large hands and couldn't wear her ring anymore. Her pressure was 130/80mmHg, pulse rate 74/minute and respiration 18/minute. The cardiovascular system and the respiratory systems were normal

Abdominal exam

The abdomen was uniformly distended and moving with respiration. There was a pfannensteil incision scar. The fundal height was term, and the foetus

was in longitudinal lie, cephalic presentation and the presenting part was not engaged. The foetal heart rate was 148/minute and regular.

Vaginal Exam

She had normal external genitalia and vaginal walls. The cervix was firm, posterior and cervical os was closed. There was no evidence of candidiasis.

DIAGNOSIS

Gestational diabetes Mellitus in a patient with multiple endocrinopathy at 36 weeks gestation.

MANAGEMENT

TSS was admitted for blood sugar control by fasting blood sugar and serial post- prandial blood glucose measurements. She was also to have regular foetal monitoring with a plan to deliver her by elective caesarean section at 39 – 40 weeks with good blood sugar control. She was given a foetal kick chart to chart daily foetal movement.

Blood glucose and insulin chart

DATE	FBS mmol/1	RBS (AM)	RBS (PM	INSULIN DOSE	COMMENTS
21/03/06	6.2	11.9	10.6	None	
22/03/06	6.4	13.6	9.6	Sol insulin:	Only soluble
23/03/06	7.6	10.8		10 iu 8	Insulin

24/03/06	6.6	9.3	15.7	Hourly	available
25/03/06	6.3	6.9	11.2		
26/03/06	6.0	7.1	5.8	Sol: 14 iu	
28/03/06	6.8	8.7	5.8	am, Mid,	
29/03/06	6.8	8.7	9.2	10 iu pm	Diet advice
30/03/06	7.4	6.3	6.2		
31/03/06	5.3		7.3		
01/04/06	5.9	7.2	6.4		
02/04/06	5.4		7.7		
03/04/06	5.3	5.9	5.7		
04/04/06	5.5	5.5	6.2		
05/04/06		5.3	5.8		C/S done
06/04/06					
07/04/06		8.6	8.4		
08/04/06	7.2	8.7	7.6	Sliding scale	Diet
09/04/06		6.8	6.9		
10/04/06	6.0	7.0	8.1		
11/04/06	6.3	7.2	8.3	Insulin	
12/04/06				stopped	
					Discharged on diet

Serum Calcium (2.12 – 2.65 mmol/1)

04/03/06 1.65mmol/1

15/03/06 2.4mmol/1

26/03/06 2.1mmol/1

09/03/06 1.57mmol/1 (tetany, 20ml of 10% Calcium gluconate in infusion given).

RFTS

Date	Na (mmol/1)	K (mmol/1)	Urea (mmol/1)	Creat (umol/l)
23/03/06	132	3.6	2.9	60
26/03/06	138	4.1	2.6	78
02/04/06	133	4.2	3.1	57
04/04/06	133	4.0	4.2	63

Haemogram 26/03/06: Hb 11.5g/dl, WBC 9.0 x 10^9/l, Platelets 227 x 10^9/l

Normocytic normochromic

Thyroid Function: TSH 5.47mlU/l (0.3 – 5.7), Free T 8.22pmol/1 (9 – 20)

Delivery

On 04/04/06 at 38 weeks and 5 days gestation, SS complained of reduced fetal movements. A decision was made to prepare and deliver the baby by emergency caesarean section. A live female infant weighing 4000g who scored 8 at 1 and 10 at 5 minutes was delivered. She had a bilateral tubal ligation performed at surgery. The baby was taken to the new born unit for observation and was discharged after 4 days. She did not develop

hypoglycemia. SS continued to have insulin on a sliding scale with intravenous dextrose till she was able to take oral feeds after 12 hours. Insulin was stopped on third postoperative day. On the 7th postoperative day she developed paraesthesiae and spasms of the extremities.Serum calcium was 1.57mmol/1. She was given an infusion of calcium gluconate and advised to take her calcium tablets which she had stopped taking pre-operatively. On the 8th postoperative day, she was allowed home and booked for the follow-up in the endocrinology clinic. She was to be seen in the postnatal clinic in 5 weeks.

Postnatal Visit

SS was seen at the clinic 6 weeks after delivery. She was doing well and had also attended the endocrinology clinic. She was still on oral calcium and vitamin D supplements. Her baby was growing well and was exclusively breastfeeding. Her incision had healed well and the uterus was normal size. She had minimal non-foul smelling normal vaginal discharge. Her random blood sugar was 5.6 mmol/1. She was advised to continue with the endocrinology clinic follow up.

DISCUSSION

This was a patient with multiple endocrinopathy who developed gestational diabetes mellitus during the index pregnancy. She was initially managed on diet, then on insulin and was delivered by emergency caesarean section at 38 weeks.

Diabetes mellitus is a clinical syndrome characterized by hyperglycemia due to absolute or relative deficiency of insulin. Diabetes is classified as type I where the patient requires exogenous insulin to prevent ketoacidosis and type II where there is abnormal insulin secretion from the pancreas islets

cells. Diabetes is the most common complication during pregnancy and patients are classified into two groups: Those with overt diabetes before pregnancy and those with diabetes diagnosed during pregnancy. Depending on the specific population, abnormal maternal glucose regulation occurs in 3 – 10% of pregnancies. Although 80% or more of this glucose intolerance occurs in patient with gestation diabetes mellitus (GDM), the associated foetal and newborn morbidity rates are disproportionate. Infants of diabetic mothers (IDMs) experience double the risk of serious injury at birth, triple the likelihood of caesarean delivery, and quadruple the incidence of newborn intensive care unit admission. During pregnancy diabetes is classified depending on whether it is gestational (A) or overt (B, C, D, F, R and H) diabetes and end organ derangements corresponding to the white classification. Gestational diabetes is classified depending on the degree of glycaemia.

GDM only occurs during pregnancy. The diagnosis is established by glucose tolerance testing. Obesity and age are common risk factors. The best method for diagnosing GDM continues to be controversial. The WHO recommendation is the 75g, 2 hour oral glucose tolerance test.

Table 1: World Health Organization Diagnostic Criteria for Diabetes Mellitus

(Venous Plasma Glucose)

Diagnosis	*Fasting*	*2 Hour*
Healthy	<140mg/dL(<7.8 mmol/L)	<200 mg/dL (<11.1 mmol/L)

Impaired glucose tolerance	>140mg/dL(>7.8 mmol/L)	<200 mg/dL (<11.1 mmol/L)
Diabetes	>140mg/dL(>7.8 mmol/L)	<200 mg/dL (>11.1 mmol/L)

Screening for GDM during pregnancy is recommended because fewer than 20% of women with sufficient glucose intolerance during pregnancy exhibit glucosuria or other symptoms during pregnancy. However, whether universal screening of all pregnant women or targeted screening of patients with risk factors is most efficacious continues to be controversial. Typical risk factors include material age older than 35 years, previous infant weighing more than 4000g, previous unexplained foetal demise, previous pregnancy with GDM, strong immediate family history of type II or GDM, obesity (>90kg) and fasting glucose value greater than 7.8 mmol/1 or random glucose value greater than 11.1% mmol/l

SS had screening for GDM at the booking visit because of the previous history of macrosomic infant, incidental glycosuria and her booking weight which was 87kg. The random blood sugar done then was 8.1mmol/l and fasting sugar 6.0mmol/l. A 75g 2 hour oral glucose tolerance test was done which diagnosed impaired glucose tolerance. She was managed on diet until she went out of control and GDM diagnosed at 36 weeks. She was then admitted for blood sugar control.

GDM in our patient could have developed due to many factors. Pregnancy with production of human placental lactogen – an insulin antagonist could have precipitated GDM. She was also obese and had features of excess production of growth hormone and glucocorticoids. These factors could have caused the impaired glucose tolerance she developed during this

pregnancy; she did not have evidence of GDM then. She had episodic hypocalcaemia that began after thyroidectomy. She had been on calcium supplementation since then and was regularly monitored during this pregnancy. Thyroid function tests were also performed which showed features of hypothyroidism. The endocrinologist did not think she required thyroid hormone supplementation

Poor glycaemic control in pregnant diabetes is associated with spontaneous abortions in the first trimester, preterm deliveries and an increased incidence of congenital malformations with most involving the central nervous system and the cardiovascular system. No increase in birth defects occurs in infants of diabetic men, women with impaired glucose tolerance and women who develop GDM after the first trimester. These defects occur in overt diabetics with poor glycaemic control preconception and during the first trimester. Diabetes is pregnancy associated with foetal macrosomia and incidences of unexplained intrauterine foetal demise. Neonates are at increased risk of respiratory distress syndrome, hypoglycaemia, hypocalcaemia, hyperbiliubinaemia, hypomagnesaemia, transient tachypnoea, hypertrophic cardiomyopathy and polycythaemia. Maternal effects include nephropathy, retinopathy, pre-eclampsia, ketoacidosis, urinary tract infections and in the puerperium. She had a 4000g infant delivery by caesarean section. The infant did not develop respiratory distress or hypoglycaemia. She was admitted to the neonatal unit for 4 days to monitor for the development of other complications.

Control for blood sugar for our patient in the ward was satisfactory. The aim was to deliver SS between 39 and 40 weeks since a caesarean section had been planned. The test of foetal being was reassuring though some like the biophysical profile could not be done weekly as recommended. The

biophysical profile was done once and the daily foetal kick chart and intermittent auscultation provided bulk of foetal monitoring. In patients with GDM and superb glycaemia control, continued foetal testing and expectant management can be considered until 41 weeks gestation. Foetus with an abdominal circumference measurably larger than the head circumference, induction should be considered. After 40 or more weeks, the benefits of continued conservative management are likely to be less than the danger of foetal compromise. Induction of labor before 42 weeks gestation in pregnant women with diabetes, regardless of the readiness of the cervix, is prudent. Thus, an optimal time for delivery of most diabetic pregnancies is typically on or after the 39th week. Delivery of a patient with diabetes before 39 weeks gestation without documented foetal lung maturity should only be done for compelling maternal or foetal indications. In such cases, foetal lung maturity can be confirmed via amniocentesis.

Our patient was delivered by emergency caesarean section at week 38 and 5 days after she complained of reduced foetal movements. She would have been delivered at 40 weeks by elective caesarean section. Insulin was given on a sliding scale on the day of delivery and stopped 24 hours later. The blood sugar improved gradually on diet control postnatally. She did not develop puerperal sepsis but had carpo-pedal spasm due to hypocalcaemia on the 5th postnatal day which was treated with intravenous calcium gluconate. Her oral supplement was restarted and she was discharged on the 7th postnatal day with her baby in good general condition. After the puerperium, the diabetes had resolved. She was to continue long term follow up with an endocrinologist due to the multiple endocrinopathy she had.

Macrosomia and polyhydramnios usually complicate plans for delivery of diabetic mothers. Planned caesarean section may be better unless

macrosomia and polyhydramnios have been ruled out, in which case labor may be induced at term. Patients with GDM will require insulin which will be stopped after delivery. These women require careful lifelong screening as it has been shown that 40 – 60% develop diabetes in the long run. The recurrence risk of GDM in subsequent pregnancies is 40 – 70%

Our patient felt she had her desired family size and for contraception, chose to have tubal ligation at caesarean section. There is no single contraceptive that is appropriate for all women with overt diabetes. Those with GDM may use any contraceptive of choice with regular cardiovascular monitoring. Low dose combined oral contraceptive pills do not increase the risk of type II diabetes mellitus in the postpartum women with recent GDM. Progestin only contraceptive can be used because of their minimal effects on carbohydrate metabolism. Many overtly diabetic women chose to have sterilization and this should be made available.

REFERENCES

1. Frier BM et al. Diabetes Mellitus and nutritional and metabolic disorders In: Haslett C, Chilvers ER et al (editors). Davidson's principles and practice of medicine 18th edition. Churchchill Livingstone publisher 1999; 472
2. Diabetes In: Cunningham HG et al (editors) Williams Obstetrics 21st edition. Mcgraw Hill 2001; 1360 – 1381
3. Moore TR. Diabetes Mellitus and Pregnancy Continuous Medical Education article for eMedicine, October 2002
4. World Health Organization: Diabetes Mellitus: Report of WHO study Group. Geneva: WHO; 1985. Technical Report series No. 727
5. Carpenter MW, Coustan DR: Criteria for screening tests for gestational diabetes.American Journal of Obstetrics and Gynecology 1982 December; 144(7): 768 – 73
6. Lucas MJ, Leveno KJ, Williams ML, et al: Early pregnancy glycosylated hemoglobin, severity of diabetes and fetal malformations. American Journal of Obstetrics and gynecology 1989 August; 161(2): 426 – 31
7. James Dk, Johnson IR, McEwen A. An Obstetrics and gynecology Vade-Mecum. Arnold Publishers, London 2000; 51, 74
8. Kjos SL, Shoup D, Donyou S, Friedman RL, Bernstein Gs, et al. Effect of low dose oral contraceptives on carbohydrate and lipid metabolism in women with recent gestational diabetes; results for prospective randomized control study. American Journal of Obstetrics and Gynecology, 1990; 163: 1822

OBSTETRICS CASE NO. 8

CERVICAL INCOMPETENCE, MACDONALD STITCH INSERTED WITH GOOD OUTCOME

NAME: G.N L.M.P:20/1/04

AGE: 31 YEARS E.D.D:27/10/04

IP NO: 0883990

D.O.A: 29.04.04 PARITY:PARA 1 +3 GRAVIDA 5

D.O.D: 03. 05.04

Presenting complaint

She was admitted through the antenatal clinic for Macdonald stitch insertion. She had presented with a history of 3 successive 2nd trimester pregnancy losses.

Obstetric and gynaecologic history

She attained her menarche at 15 years. Her menstrual cycles were regular, the menstrual flow occurring for 4-5 days in a 28-day cycle. She had no dysmenorrhoea.

She was attending antenatal clinic at KNH since 8 weeks of pregnancy and antenatal profiles had been done. The results were as follows:

Blood group O positive

Hb 13.7 g/dl

VDRL negative

HIV negative

Obstetric scan showed single viable fetus at 10 weeks gestation.

She was a para 1 + 3 gravida 5. Her past pregnancies were as follows:

1996:- normal SVD at term to LFI

1999:- spontaneous abortion at 16/40

2000:- spontaneous abortion at 16/40

2001:- spontaneous abortion at 20/40

The 3 spontaneous abortions started with painless drainage of liquor followed by expulsion of the products of conception. Evacuation was done for the first two abortions but not the 3rd as it was a complete abortion.

Past Medical and Surgical History

This was nil significant.

Family and Social History

She was a married business lady . She did not smoke cigarettes or drink alcohol.

PHYSICAL EXAMINATION

She was in fair general condition, had no pallor, jaundice, cyanosis or dehydration. She had a blood pressure of 110/60 mmHg, a pulse rate of 80/ min, respiratory rate of 20/min, and a temperature of 36.8^{O}C.

Abdominal Examination

The abdomen was moving with respirations. The liver and the spleen were not palpable, and there were no obvious masses palpable. There was no area of tenderness. The fundal height was not palpable.

Vaginal examination

Her external genitalia were normal. A speculum examination revealed normal vaginal and cervical mucosa. The cervix was about 1 cm long and had an old tear about 0.5 cm running vertically but not reaching the level of the internal os at 7 o,clock. The external os was closed. There was no discharge noted.

Impression

An impression of cervical incompetence was made

MANAGEMENT

She had already been informed of the diagnosis and the planned form of management at the ANC clinic.The impression was explained to her again and the mode of management, its benefits, possible complications and the options available and their consequences explained in details. She opted to have the stitch inserted. She gave a written consent to that effect.

Renal function test was done and the results were as follows:-

Na+ -139mmol/l

K+ -4.4mmol/l

Crat -46mmol/l

BUN –2.9mmol/l

She was starved from midnight till after the stitch insertion.On the following morning she was given atropine 0.6mg intramascularly, put on 10% dextrose intravenously and then wheeled to theater. In theater,she was put on the operation table in supine position and general anesthesia initiated and maintained as described in the introduction.

She was then placed in lithotomy position,perinium cleaned and draped.On catheterization, about 150ml of clear urine was then drained.

An Auvard's speculum was then inserted into the vagina and the cervix exposed..The earlier findings were confirmed.The cervical lips were held with two sponge forceps and gently pulled down.

The junction of the rugose vaginal epithelium and the smooth vaginal part of the cervix identified.This was approximated to be the level of the internal os and a silk suture size 2 was then placed round the cervix at this level,with bites at 7,5,1 and 11 O'clock, and the sponge forceps being advanced round the cervix as neccesary to stabilize it . There was insignificant bleeding. She was then placed back to supine position and the general anaesthesia reversed successfully. Her vital signs were monitored ¼ hourly until she was fully a wake. Once fully a wake she was taken back to the ward, where the vitals signs were observed 6-hourly. She was started on ibuprofen 400mg 8-hourly for analgesia. Three days after the insertion of the stitch, she was allowed home, with advice to avoid strenuous work and avoid coitus, and to report to hospital for follow up after 2 weeks or in case she had any labour –like pains or drainage of liquor. Subsequently she had several antenatal visits. They were all normal.

On 29th October 2004 at a gestation of 40 weeks and two days, she was reviewed at the antenatal clinic. She had no complaints. She was not pale and the blood pressure was 110/65mmHg.The fundal height was term and the foetus was in cephalic presentation and longitudinal lie. The fetal heart tones were regular at 134 B/min.The presenting part was 4/5 the above the pelvic brim. She had a stitch removed at our labor ward and was allowed home. That same day at 9.00pm, almost 12 hours after the removal of the stitch, she came back to labor ward complaining of lower abdominal pains that were radiating to the back and associated with

mucoid vaginal discharge for 3 hours prior to admission. Abdominal examination revealed same findings as during the day, but the presenting part was 3/5th above the pelvic brim. Three uterine contractions each lasting 30 seconds were palpated. Vaginal examination showed a cervix that was fully effaced and an os that was 5 cm dilated. The membranes were bulging and were ruptured to yield clear liquor. The cord was not palpated .The pelvis felt adequate.

She was started on oxytocin infusion 5 units in 500mls of 5% dextrose running at 10 drops /min and increased by 10 drops ½ hourly. She was also given tramadol 100mg intramuscularly and a partogram started. She progressed well in labor and 3 hours later, at 12 midnight delivered a live female infant who scored 9/1and 10/5 and weighed 3000g. Third stage was completed by controlled cord traction. She had no perineal tears. Estimated blood loss was 250ml. Fourth stage was uneventful, and she was encouraged to initiate breast-feeding. On the following day, she had no complaints. The breasts were lactating and the uterus was well contracted, corresponding to 18 weeks gestation. Lochia loss was rubra, normal flow and not malodorous. Her baby was stable. She was allowed home with advice to be reviewed at the postnatal clinic after 6 weeks. She never turned up.

DISCUSSION

Presented here is a 31-year old who presented with three pregnancy wastages following a normal delivery. She had a MacDonald stitch inserted at 10 weeks successfully.

Cervical incompetence is characterised by painless dilatation of the cervix in the second or early third trimester of pregnancy, with prolapse of the membranes through the cervix and ballooning of membranes onto the vagina. Rupture of this membranes then follows with expulsion of the foetus. This is however not always the case, as the cervical dilatation may be accompanied by painful uterine contractions as the products of conception are being expelled.

The incidence of cervical incompetence varies, with rates ranging from 0.5 to 10 per 1000 pregnancies. Between 20% and 30% of all second trimester abortions are thought to be due to incompetent cervices.

Cervical incompetence may be congenital or acquired. Congenitally acquired incompetence may be associated with mullerian duct abnormalities such as uterus didelphis or bicornuate uterus. Patients who were exposed to diethylstilbestrol in utero have been shown to have an increased incidence of cervical incompetence.

Excessive dilatation of the cervix during curettage or induced abortion is the commonest cause of acquired incompetence. This is thought to result in structural damage to the internal os. Cone biopsy of the cervix, precipitate labour or cervical lacerations during delivery may result in an incompetent cervix. The patient presented here had no history of induced abortion but duration of labour of her 1st pregnancy was unknown. If precipitate this could have resulted in damage to the cervix as she had a

tear at about 7 o'clock. Still, the incompetence could have been congenital for she had a short cervix of about 1 cm in length.

Diagnosis of cervical incompetence can be made from the reproductive history of 2 or more previous mid trimester pregnancy losses preceded by painless cervical dilatation, rupture of membranes and expulsion of conceptus, or a precipitate labor. A short cervix with or without any defect is most likely to be incompetent. In non-pregnant woman, several diagnostic aids are applied to confirm diagnosis. These include:-

i. Easy passage of size 8 (8mm) Hegars dilator into the uterus.

ii. An increased isthmic diameter from an average of 2.63mm to 6.09mm in the hysterosalpingography.

iii. Ability of a Foleys catheter placed in the uterine cavity and balloned with about 1cc of water to be pulled through the cervix using a force of 600g. This is called the test of Bergman and Svenerund.

In their research, Brooks et al concluded that an internal os that was 1.9cm or less in width was highly suggestive of incompetence.

In 1976, Block and Rahall devised a prognostic scoring system for incompetent cervices. This system takes into consideration 5 factors as follows:-

1. Previous premature delivery

2. Progressive dilatation of 2 cm or more during pregnancy or a cervix dilated 2cm at the initial assessment in the second trimester.

3. Previous diagnosis of cervical incompetence and stitch insertion.

4. Visual evidence of previous trauma to the cervix.

5. History of painless premature labor or rapid delivery

Patient with 3 or more of the above factors has a better prognosis with stitch insertion when compared with those who never had the stitch.

The aim of management of a patient with cervical incompetence is to retain the foetus in utero until it is mature. Three methods have been employed with a variable success. The use of Hodge pessary has been shown to be effective in the management of incompetence. Use of progestin therapy has also been tried in the hope of maintaining the uterus in a quiescent state. These methods are hardly ever used in our setting. Surgical approach is the most commonly employed mode of management of the incopetent cervix. This is in the form of Shirodkar, the MacDonald or modified Shirodkar procedures. In KNH, the MacDonald procedure is employed. This involves placing a non-absorbable suture like silk, round the cervix at the level of the internal os, with bites at 1, 5, 7 and 11 o'clock. It was the procedure employed on the patient presented here.

In our set up, the earliest time for insertion of the stitch is at 14 weeks. This is to allow any abortions due to chromosomal abnormalities to occur. The upper limit is controversial, with most preferring to put the stitch before 18 weeks; that is before appreciable cervical dilatation has occurred. The stitch is hardly ever inserted after 24-26 weeks, bed rest being preferred instead. Patients are advised to abstain from sex for at least 1 week before the procedure and throughout the pregnancy after the procedure. Use of prophylactic antibiotics and tocolytics is of proven value after cerclage. The patient presented was only given analgesia for a few days.

Complications following cervical cerclage include rupture of membranes, chorio-amnionitis and haemorrhage. Further damage to the cervix following labour with the stitch in situ may also occur.

REFERENCES

1. Cunningham F.G, MacDonald P.C, Gant N.F. Abortion in: Williams obstetrics,19th ed. Prentice-Hall Int.,1991,pg661-689
2. Tindal V.R Abortion in : Jeffcoat's principles of Gynaecology, 5th ed.1987, pg191-211
3. Cousins L. Cervical incompetence; a time for re-appraisal. Clinical obstetrics and Gynaecology, 1980
4. Sromme W.B, Haywas E.W Intra uterine fetal death in second trimester Am.J. Obstet gynaecol
5. Mann. E.C et al. The philosophy and clinical significance of the uterine isthmus. Am.J.Obstet Gynae,1966,28;198-205.
6. Luis A,Gibil S incompetent cervical os. Surgical disease in pregnancy,1990.pg 1-21.
7. Asplun F. The uterine cervical isthmus under normal and pathological conditions. A clinical and roenteonological study. Acta radio,1952;91:3-76
8. Brook et al. Ultrasonography in the diagnosis of cervical incompetence in pregnancy. A new diagnostic approach. Brt, J. Obstet Gynaecol,1981;88;640-643
9. Block M.F, Rahal D.K. Incompetence in the cervix J.Obstet gynae,1976 47;279
10. Sherman A.I. Hormonal therapy for control of the incompetent os in pregnancy. Obst. and Gynaecol.1966,28,198-205

11. Cunningham F.G, Paul C.M. Gant F.N Reproductive success and failure in: Williams Obstetrics 20th ed.Pentice-Hallint.inc.,1997, pg588-91.

12. Mattingly R.F, Thompson J.D. surgical management of abortion. In operative Gynecology,6th ed, pg, 513-519

OBSTETRICS CASE NO. 9

PRETERM PROM AT 34 WEEKS GESTATION – PRETERM LABOUR, LIVE BIRTH

NAME: R.N.

IPNO: 1079390

AGE: 26 YEARS

PARITY: 1 + 0 GR 2

LMP: 22. 07. 05

EDD: 29. 04. 06

MATURITY: 28 WEEKS

DOA: 13. 03. 06

DOD: 22. 03.06

Presenting Complaint

She presented with a 5 hours history of drainage of liquor.

History of the presenting Complaint

She was well until 5 hours prior to admission when she had a sudden gush of fluid from the vagina while standing doing her household chores. The fluid was colourless and trickled down the legs to the floor. There was no associated vaginal discharge or bleeding. There were no associated abdominal pains at the time of admission. There was no history of trauma, dysuria or frequency of micturition.

Obstetric and Gynaecological History

She was a para 1+ 0 gravida 2. Her last menstrual period was on 22/ 7/05 and her expected date of delivery on 29/04/06; she was at 34 weeks gestation by dates. Her antenatal profiles were as follows:-

Hb 12.8g/dl

Blood group O positive

VDRL negative

HIV non reactive.

Her last delivery was SVD to a live male infant at a local clinic in 1998.

She attained menarche at 15 years; she had a regular menstrual cycle of 30 days with duration of flow of 4 days. She had not used any contraceptive method.

Past Medical History

She had no significant medical or surgical history.

Family and Social History

She was a single businesswoman. She did not smoke cigarettes or drink alcohol. Her mother was diabetic.

PHYSICAL EXAMINATION

She was in good general condition, not pale, afebrile, not jaundiced, and without oedema or lymphadenopathy. She had a pulse rate of 84 beats per minute, a respiratory rate of 18 breaths per minute, a blood pressure of 120/70 mmHg and a temperature of 36.8^{0}C.

Systemic Examination

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

Abdominal Examination

The abdomen was distended and moved with respiration. There were no areas of tenderness. The liver and spleen were not palpable. The uterus corresponded to 34 weeks gestation. The foetus was in longitudinal lie and cephalic presentation and the descent was 5/5. The foetal heart tones were heard and regular at 144 bets per minute.

Speculum Examination

She had normal external genitalia. The vaginal walls and the cervix were healthy. There was a pool of clear fluid in the posterior fornix and active drainage of liquor was noted from the cervical os. The cervical os was closed and the umbilical cord was not seen.

Diagnosis

A diagnosis of preterm premature rupture of membranes at 34 weeks gestation was made.

Investigations

1. Obstetric ultrasound scan showed a single intrauterine pregnancy at 33 weeks gestation with reduced amount of liquor.
2. Haemogram WBC 8.5 x 10^9/l
   Hb 12.3 g/dl
   Platelets 295x10^9/l
3. Blood group O positive
4. VDRL negative
5. HIV test negative

6. Urine microscopy culture and sensitivity was normal with no growth obtained.
7. Endocervical swab culture grew no organisms.

MANAGEMENT

She was admitted to the admitting antenatal ward for conservative management. She was put on bed rest and oral erythromycin 500mg 8 hourly and metronidazole 400 mg 8 hourly. Intramuscular dexamethasone 6mg twice daily for a total of four doses was also given to enhance foetal lung maturity. An endocervical swab was taken for culture and sensitivity and specimens taken for haemogram and urine microscopy culture and sensitivity. Vital sign were taken every 4 hours. Daily patient examinations were done including abdominal palpation for tenderness and examining the liquor for smell and the colour. No signs of sepsis were noted during the conservative management.

Two days after admision, she went into spontaneous labour and had a spontaneous vertex delivery to a live female infant with an Apgar score of 10 and 10 in 1 and 5 minutes respectively and weighing 2450g. The baby was was allowed to join the mother. The mother continued with oral erythromycin 500mg 8 hourly and metronidazole 400 mg 8 hourly for five days.

She remained stable and was discharged home on 2nd postnatal day for review in PNC after two weeks.

DISCUSSION

The case presented is of a 26 year old para 1 + 0 admitted with preterm premature rupture of membranes at 34 weeks gestation. She had failed conservative management and delivered two days after admission to a preterm infant weighing 2450g who was allowed to join the mother after getting good apgar score.

Premature rupture of membranes (PROM) is defined as rupture of the amniotic membranes before the onset of labour, regardless of gestational age. Preterm PROM (PPROM) is defined as PROM that occurs before 37 weeks of gestation.[1]

PROM occurs in approximately 10.7% of all pregnancies. In approximately 94% of cases, the fetus is mature. Premature foetuses (1000-2500g) account for about 5% of the total number of cases while immature foetuses (<1000g) account for less than 0.5% [(2)]. The incidence of PROM at Kenyatta National Hospital has been cited as 6.2 to 9.3%. [3, 4, 5]

Eighty five percent of neonatal morbidity and mortality is as a result of prematurity. Preterm PROM is associated with 30-40% of preterm deliveries and is the leading identifiable cause of preterm delivery as was the case for our patient.[6]

Foetal membranes are made of a thin layer of amnion and a thicker outer layer of chorion that is directly apposed to maternal decidual tissue. Interspersed between the two layers is a collagen rich connective tissue zone that serves in part to replenish the amnion. The amnion by 26 wks gestation is composed of one layer of cuboidal cells while the chorion is 4-6 cells layer thick. The amnion has greater tensile strength than the

chorion though combined can withstand greater bursting pressures. The amount of physical stress tolerated by the membranes decreases as the pregnancy progresses and membranes supported by a closed cervix require much greater pressure to rupture than membranes with an open cervix [7]. The amount of collagen decreases relatively as gestational age advances [8]. These factors help maintain the integrity of membranes throughout pregnancy, yet allow rupture of membranes at term [8]. Collagen maintenance and degradation are regulated in foetal membranes by the interaction of matrix metalloproteinases and tissue inhibitors of matrix metalloproteinases. Collagen and proteinase activity are increased in women with PROM [9].

The exact cause of rupture is not known, although there are many associated conditions. These include maternal infection (e.g. urinary tract infections, lower genital tract infection, sexually transmitted diseases), intrauterine infection, cervical incompetence, multiple previous pregnancies, hydramnios, decreased tensile strength of membranes and familial history of premature rupture of membranes [2], sexual activity, smoking, dietary deficiencies in ascorbic acid, zinc or copper deficiency [10].

Pregnancies affected by PROM are at increased risk of umbilical cord prolapse, chorioamnionitis, and postpartum endometritis. Patients with PROM are also at increased risk of abruptio placentae. When the amniotic fluid volume in PPROM remains significantly reduced for any length of time the foetus is at risk of developing a foetal compression syndrome. Pulmonary hypoplasia is a significant sequel of prolonged PPROM occurring in 25% - 30% of patients whose membranes rupture before 22 weeks. [1, 2, 10]

Diagnosis of PROM is based on a history of a gush of watery fluid, or persistent wetness, demonstration of pooled amniotic fluid in the posterior vaginal vault, or fluid leaking from the cervical canal on examination with a sterile speculum. Laboratory tests may show a fern pattern in dried amniotic fluid on a slide and a PH of the fluid of more than 7 as seen by colour change of Nitrazine paper from yellow to blue [2, 10]. Other confirmatory tests for PROM include observed loss of fluid from the cervical os when the patient coughs or performs a valsalva manoeuvre during speculum examination and oligohydramnios on ultrasound examination. It may sometimes be necessary to perform amniocentesis and inject a dilute solution of Evans blue or indigo carmine dye. After 15-20 minutes, insertion of a speculum will reveal blue dye in the vagina if the membranes are ruptured [1, 2, 10]. Our patient presented with history of gush of watery fluid and on speculum examination there was pooling of amniotic fluid in the posterior vaginal fornix and fluid was also observed draining from the cervical os.

Initial laboratory studies should include a complete blood count with differential. In preterm pregnancies, urinalysis culture and sensitivity should be done. Ultrasounds scan for foetal size and amniotic fluid index and amniocentesis in some cases to determine foetal lung maturity and the presence of infection may be done [1, 2].

The management PROM depends on several factors, including gestational age and the presence or absence of amnionitis. If amnionitis is present, the patient should be actively delivered regardless of gestational age. Broad-spectrum antibiotics should be started to treat the amnionitis. Labour should be induced to expedite delivery. The most common organisms causing amnionitis are those that ascend from the

vagina (e.g. streptococci B and D, and anaerobes). Signs of infection include fever, maternal leucocytosis (WBCs > 16000/cmm), uterine tenderness, tachycardia - either maternal pulse >100/min or foetal heart rate > 160/min, foul smelling liquor [1, 2, 3]. Unfortunately fever is the only reliable indicator in making a diagnosis of amnionitis. [11]

A term pregnancy (>37 wks) with PROM in the absence of amnionitis can be managed expectantly or actively. Expectant management entails non-intervention while waiting for the patient to go into labour spontaneously, whereas active management entails induction of labour. If a patient on expectant management does not go into labour within 6-12 hours after PROM, labour should be induced to minimize the risk of infection [1, 2]

Pregnancies beyond 33-34 weeks can be managed as a term pregnancy because there is no evidence that antibiotics, corticosteroids, or tocolysis improve outcome in these patients. As long as these patients show no sign of amnionitis, they can be managed expectantly. Our patient was managed conservatively for 2 days then went into spontaneous labour. She had initially been given corticosteroids.

Pregnancies prior to 24 weeks of gestation have extremely low rates of foetal salvage with considerable maternal risk. Furthermore, at this early gestational age, steroids, tocolysis and antibiotics have no proven benefit. These patients should be managed with expectant management or active termination [1, 2].

For pregnancies with PROM between 24 and 32 weeks of gestation, several interventions have been shown to prolong pregnancy and improve outcome. In the absence of amnionitis, management should consist of antibiotics, corticosteroids and tocolysis. The role of tocolysis in

PPROM should be limited to 48 hrs duration to permit administration of corticosteroids and antibiotics [1, 2].

REFERENCES

1. Heyl PS. Preterm Delivery. In: Evans AT, Niswander KR (Eds). Manual of Obstetrics. 6th Edition, Lippincott Williams and Wilkins, Philadelphia, 2000:448-457.
2. Roman A.S., Pernoll ML. Late Pregnancy Complications In: DeCherney A.H., Nathan L. (Eds): Current Obstetric and Gynaecologic Diagnosis and Treatment, 9th edition, McGraw-Hill Companies, 2003:292-294.
3. Orina M. Maternal and Neonatal Outcome in Patients Presenting with Preterm Premature Rupture of Membranes (PPROM) at Kenyatta National Hospital. Mmed Thesis, University of Nairobi, 2003.
4. Wanjala S. Premature Rupture of Membranes at Kenyatta National Hospital. Mmed Thesis, University of Nairobi 1980.
5. Otieno JA. Spontaneous Rupture of Membranes at Kenyatta National Hospital, Mmed Thesis, University of Nairobi, 1981.
6. Wilkes PT, Galan H. Premature Rupture of Membranes. http://ww.emedicine.com.Inc. 2004.
7. Kitz MJ. Preterm Premature Rupture of Membranes. In: Fuchs F; Slubblefield P. (Eds): Preterm Birth Causes, Prevention and Management, McMillan, New York, 1984:298.
8. Athayde N., Edwin SS. Pomero R. A Role for Matrix Metalloproteinases - 9 in Spontaneous Rupture of Fetal Membranes. Am. J. Obstet Gynecol, 1998; 179:1248.

9. Skinner S; Campos G; Liggins G. Collagen Contact of Human Amniotic Membranes. Effects of Gestational Length and Premature Rupture. Obstet. Gynecol. 1981; 57: 487.

10. Blide AG. Premature Rupture of Membranes. In: Krishna U, Tank DK, Daftary S. (Eds). Pregnancy at Risk Current Concepts, 4th Edition Jaypee Brothers, New Delhi, 2000:362 - 364.

11. Cunningham FG, Gant NF, Leveno KJ, Gilstrap III. LC, Hauth JC. Wenstrom KD (Eds). Preterm Birth In: Williams Obstetrics 21st Edition McGraw-Hill Comp. , 2001:704 - 718

OBSTETRIC CASE NO. 10

SEVERE PRE-ECLAMPSIA AT 35 WEEKS: EMERGENCY CAESAREAN SECTION DONE WITH GOOD OUTCOME.

Name: A.M.M DOA: 06. 04. 06

Age: 25 years DOD: 12. 04. 06

File No: 1085698

LMP ? / 06. 08.05

EDD ? / 13. 05. 06

Presenting illness

A.M.K was admitted as a referral from Matuu cottage hospital with diagnosis of severe PET at 35 weeks gestation. She complained of headache and swelling of face and lower limbs.She gave no history of blurring of vision or epigastric pain.She reported history of reduced fetal movements. Blood pressure on admission was 170/110 proteinuria of 3+.

She had been on Aldomet 250 mg and nifedipine 20 mg once daily.

Past medical history

Was nil significant

Obstetric and Gynaecological History

She was a Para 2 + 0 gravida 3

LMP? / 06. 08.05

EDD? / 13. 05. 06

She attended her antenatal clinic at Matuu cottage hospital. Her antenatal profile was as follows: HIV negative, Hb 12g/dl, Blood Group O positive, VDRL negative.

Past obstetric histories were all SVD and uneventful.

Menarche was at 15 years. Periods were regular, lasting 3 to 4 days and coming after every 28 days. No history of use of contraception.

Family and Social History

She was a married business lady who did not smoke cigarettes or drink alcohol.

PHYSICAL EXAMINATION

General examination

She was in fair general condition, oedematous, not pale or jaundiced and afebrile. BP 170/110 mmHg. Proteinuria ++++.

Abdominal examination

She had a FH of 28/40, oblique lie and breech presentation. Fetal heart beats were noted to be low at 100/ min and irregular.

Vaginal examination

The external genitalia were normal. The cervix os was closed, posterior, firm not effaced and 2cm long.Other systems were essentially normal.

Investigations

Full Haemogram: Hb 12.3 g/dl, WBC 6.15 x 10/l, Platelets 200 x 10/l

LFTs normal,

RFTS: All normal

Obstetric Ultrasound Scan

The scan reported a single intrauterine pregnancy in breech presentation. It had a regular heart beat of 141/ min. the fetus showed no obvious abnormality. The placenta was fundo-anterior and not low lying.Amniotic fluid was adequate.Biometric profile corresponded to 30/40 gestation.Biophysical profile was 3/8

DIAGNOSIS

An impression of severe Pre-eclampsia with severe intra-uterine foetal growth retardation was made and patient prepared for delivery.

MANAGEMENT

She was given dexamethasone and as the Bishop score was poor she was prepared for emergency caesarean section. She was told of the likely poor outcome due to the poor condition of the baby, the growth retardation and the prematurity.

Caesarean section

Patient was placed in semi-lithotomy position, aseptically catheterized draining clear urine. In supine position, cleaned draped and put under GA. Abdomen was opened via Joel Cohen incision. Lower uterine segment was identified and transverse incision made.

A LFI was delivered who scored 7, 9 and 9 at 1, 5 and 10 minutes respectively and weight 1550gms.Placenta was extracted by CCT. Uterus was repaired in layers. Abdomen was closed in layers after swab and instrument was found correct.Patient was reversed from GA and taken to recovery room in good condition.Postoperatively patient did well and was discharged on 6th post operative day to mothers hostel to await her child in the NBU who was doing well but had not gained necessary weight for discharge.

DISCUSSION

Pre-eclampsia is a triad of oedema, hypertension and proteinuria occurring primarily in nulliparas after the 20th gestational week and most frequently near term [1, 2]. Eclampsia is the occurrence of seizures that cannot be attributed to other causes in a pre-eclamptic patient.Hypertension is defined as blood pressure equal to or greater than 140/90 mmHg and proteinuria as excretion of 300mg or more in a 24 hour specimen of urine.

The characteristic renal lesion in pre-eclampsia is Glomeruloendotheliosis.

Incidence

Pre-eclampsia occurs in about 6 % of the general population, its incidence varying with geographic populaton.Predisposing factors are nulliparity, black race, maternal age below 20 or over 35 years, low socio-economic status, multiple gestation, hydatidiform mole, polyhydramnios, non-immune foetal hydrops, diabetes, chronic hypertension and underlying renal disease.

Classification

Pre-eclampsia can be mild or severe. Severe pre-eclampsia has the following features:

a) blood pressure greater than 160 mmHg systolic or 110 mmHg diastolic

b) proteinuria exceeding 5g in a 24 hour period or 3-4+ on dipstick testing

c) increased serum creatinine (>1.2 mg/dl unless previously elevated)

d) cerebral or visual disturbances

e) epigastric pain

f) elevated liver enzymes

g) thrombocytopenia (platelet count < 100,000/ mm3)

h) retinal haemorrhages, exudates or papilloedema.

i) Pulmonary oedema

Pathogenesis

The current hypothesis of the pathogenesis of pre-eclampsia is that an immunologic disturbance causes abnormal placental implantation resulting in reduced placental perfusion. The abnormal perfusion stimulates the production of substances in the blood that activate or injure endothelial cells. The vascular endothelium provides a single target for these blood borne products which explains the multiple organ involvement in pre-eclampsia.

Pre-eclampsia, particularly when associated with proteinuria (proteinuric pre-eclampsia) poses risk to both mother and foetus [1]. While the cause of pre-eclampsia remains elusive, its origins are found in early pregnancy with hypertension, with or without proteinuria, being a late manifestation of the syndrome [2]. It is known to be associated with inadequate placentation and reduced blood supply to the intravillous space in the developing placenta.Abnormal morphological changes, such as narrowing of the spiral arteries, have been demonstrated by the end of the first trimester [3, 10] Despite the fact that symptoms rarely develop until the third trimester. Changes to the vascular reactivity as assessed by an increased sensitivity to infused Angiotensin 2 can be demonstrated in women who develop the syndrome well before a rise in blood pressure occurs [4, 5, 6.]

Clinical features

1. Oedema: Dependent oedema is a normal finding in pregnancy but non-dependent oedema of the hands and face present in the morning is pathologic. Sudden weight gain should raise suspicion of preeclampsia.
2. Hypertension: Hypertension is the most important criteria for diagnosis of preeclampsia. A rise of 15 mmHg in the diastolic or 30 mmHg in the systolic pressure is considered ominous.

3. Proteinuria: This is the last sign to develop though eclampsia may occur without proteinuria. Most patients with proteinuria will have glomerulo-endotheliosis on kidney biopsy. Proteinuria is an indicator of foetal jeopardy.

Laboratory findings

1. The haemoglobin and haematocrit may be elevated due to haemoconcentration. In severe cases there may be anaemia due to haemolysis.
2. Thrombocytopenia [12]

1. Fibrin split products and decreased coagulation factors may be detected.
2. Uric acid is usually elevated.
3. Creatinine may be elevated in severe preeclampsia.
4. Abnormal liver functions: Alkaline phosphatase and Lactate dehydrogenase are usually quite high.

Complications

Like in our patient, preeclampsia may be associated with early delivery and foetal complications due to prematurity. There may be acute and chronic uteroplacental insufficiency and this may result in intrapartum foetal distress or stillbirth.Chronic uteroplacental insufficiency may result in asymmetric or symmetric growth restriction.

Complications that can occur in the mother include eclampsia, renal failure, coagulation disorders, HELLP syndrome and cortical blindness [7].

Prevention

1. Calcium supplementation: Calcium supplementation reduces the incidence of transient hypertension and preeclampsia [11]
2. Aspirin: The place of aspirin in preeclampsia prevention is uncertain and benefits may be confined to high risk women.

Treatment

The treatment of preeclampsia is bed rest and delivery. Women with mild preeclampsia who can be relied on to follow the doctor's instructions can be managed as out patients [9] Patients must be warned of danger signals such as severe headache, epigastric pain or visual disturbances.

For hospitalized patients blood pressure is measured every 4 hours and patients are weighed daily. Urine dipstick testing for protein is done daily. Liver function, uric acid electrolytes and serum albumin are determined on admission and weekly. The

coagulation profile should be measured in patients with severe preeclampsia.

Assessment of gestational age and foetal weight are performed by ultrasound on admission and as indicated thereafter.

Antihypertensive medication is usually withheld unless diastolic blood pressure exceeds 100 mmHg and the gestational age is less than 30 weeks. Sedatives may also be used [8].

Like in our case corticosteroids may be used to accelerate lung maturity.

REFERENCES

1. Douglas KA, Redman CWG. Eclampsia in the United Kingdom. BMJ 1994; 309: 1395-1400.
2. Redman CWG, Jefferies M. Revised definition of pre-eclampsia. Lancet 1998; 1:809-812.
3. Brosens IA, Robertson WB, Dixon HB. The role of the spinal arteries in the pathogenesis of pre-eclampsia. In: Wynn RM, editor. Obstetrics and Gynaecology Annual. New York: Appleton-Century-Crofts, 1972: 177-191.
4. Chesley LC. Vascular reactivity in normal and toxaemic pregnancy. Clin Obstet Gynaecol 1966;9: 871.
5. Gant NF, Daley GC, Chand S, Whalley PJ, MacDonald PC. A study of Angiotensin 2 pressor response throughout primigravid pregnancy. J Clin Invest 1973; 52: 2682-2689. From BJOG Vol 103 Number 5 May 1996 p 421.
6. Abdul-Karim R, Assali NS: Pressor response to angiotonin in pregnant and non-pregnant women. Am J Obstet Gynecol 82:246, 1961?
7. Apollon KM, Robinson JN, Schwartz RB, Norwitz ER: Cortical blindness in severe preeclampsia. Obstet Gynecol 95:1017, 2000.
8. Atkinson MW, Guinn D, Owen J, Hauth JC: Does magnesium sulphate affect the length of labour induction in women with pregnancy-induced hypertension. Am J Obstet Gynecol 173:1219, 1995.

9. Barton JR. Stanziano G, Sibai BM: Monitored outpatient management of mild gestational hypertension remote from term. Am J Obstet Gynecol 170:765, 1994.

10. Brosens IA, Robertson WB, Dixon HG: The role of the spiral arteries in the pathogenesis of preeclampsia. Obstet Gynecol Ann 1:177, 1972.

11. Chappell LC, Seed PT et al: Effect of antioxidants on the occurrence of preeclampsia in women at increased risk: A randomized trial. Lancet 354:810, 1999.

12. Pritchard JA, Cunningham FG, and Mason RA: Coagulation changes in eclampsia: Their frequency and pathogenesis. Am J Obstet Gynecol 124:855, 1976.

OBSTETRIC CASE NO. 11

RHESUS NEGATIVE MOTHER WITH BAD OBSTETRIC HISTORY –SUCESSFUL INDUCTION OF LABOUR, LIVE BIRTH.

Name: H.W.M DOA: 21/07/06

Age: 26 years DOD: 24/07/06

Parity: 2+0 gravida 3 File No: 0901913

LMP: 26/10/05

EDD: 04/08/06

GDB: 38 weeks

Presenting Complaint

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

History Presenting Complaint

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

Obstetric and Gynaecologic History

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

Her last menstrual period was on 26/10/05 and the expected date of delivery was 4/08/06. Gestation by date was therefore 38 weeks. She had attended

Airport Medical Clinic for ANC once at 30 weeks and later went to Kitengela health centre where she was seen three times from 32 weeks. At 38 weeks she was referred to Kenyatta National Hospital for delivery. She had no other test for antenatal profile except blood group, which was B Rhesus negative. She had attained menarche at 16 years. Her menses were regular occurring every 28 days and lasting 5 days. She had no history of use of contraceptives.

The past medical history was insignificant

Family and Social History

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

PHYSICAL EXAMINATION

General Examination

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

Abdominal examination

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

Pelvic examination

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

DIAGNOSIS

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

Investigations

- Full Haemogram: Hb:10.8g/dl ,WBC-7.1x10^9/1,Platelets: 176x10^{12}/1
- RFTS: Na+ 141mmol/1, K+ 3.9mmol/1,Urrea2.6mmol/1,Creatinine 63 umol/1
- Blood group Rhesus negative
- Indirect Coomb's test: Negative

MANAGMENT

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

The results were:

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

The mother was given Anti-D globulin 300ug on the second day after delivery. The baby remained well and did not develop jaundice. Both baby

and mother were discharged 48 hours after delivery for follow up at the postnatal clinic after 6 weeks.

Follow up

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

DISCUSSION

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

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

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

Although there is no significant dfference in the distribution of the rhesus antigen with regard to gender, there exist important racial differences. The incidence of rhesus negativity ranges from zero in the Mongoloids to the highest incidence rate of 30-35% among the Basque people. Caucasians have an incidence of 15-16%, African blacks4% indocaucasians 2% and Northe American Indians 1%.[4] In Kenya Mulandi found a prevalence of 4.1% in antenatal women at Kenyatta National Hospital (KNH) compared to 3% in the general population as found by Kingo in Tanzania and noted by Mulandi [5.6].

Rhesus isoimmunization occurs when sufficient numbers of foetal rhesus positive erythrocytes gain access to the maternal circulation and the mother has rhesus negative erythrocytes and the capacity to produce antibodies against the D antigen. Fetal RBCs enter maternal circulation during pregnancy and the immediate postpartum period. The amount of fetal haemrrhage necessary to cause isoimmunization varies from patient to

patient. As little as 0.1mls of rhesus positive RBCs have been showed to cause sensitization.[7] However, during normal pregnancies and deliveries, over 50% have fetomaternal haemorrhage or less.[8,9] Risk factors that increase fetomaternal haemorrhage include abortion, caesarean delivery, manual removal of the placenta, multiple gestation, antepartum haemorrhage and intra uterine manipulation. The majority of the feomaternal haemorrhage occur in patients without risk factors who have uncomplicated vaginal deliveries[8]

The amount of fetal blood in the maternal blood can be calculated using many tests, but the commonest used method is the Keihauer-Betke test. This test is based on the fact that fetal RBCs are less likely to get haemolysed than maternal RBCs because of the difference in the haemoglobulin, about 16% of rhesus negative mothers will become isoimmunized by their first rhesus incompatible, ABO compatible pregnancy with no apparent predisposing factor studies have detected fetal blood in maternal blood in 6.7% women during the first trimester, 16% during the second and 29% during the third trimester[4, 8, 9]. This contrasts from finding by Kizza's study at KNH Nairobi that showed fetomaternal haemorrhage in15.4% of women during the first trimester, 29.5% during the second and 38% during the third trimester. Antepartum sensitization, however, rarely occurs before thrd trimester. This is because o fthe cellular fetomaternal blood barrier reduces to two cells: the syncytiotrophobloast and the capillary endothelium – so called placenta underwent induction of labour at 38 weeks to pre-empt both the higher incidence of isoimmunization in the late 3rd trimester and the unknown cases(s) of her two previous fetuses in the early 3rd trimester.

The management of women with regard to rhesus isoimmunizatio begins with blood typing. Any pregnant woman, therefore shouls have her blood

typed. Husbands of rhesus negative women should have their blood typed too. By direct Coomb's test, antibody screening is performed at 28 weeks gestation in unsensitized rhesus negative mothers. If negative 300ug of Rhesus Immunoglobulin (RhIgG) is given. If positive the patient should be managed as Rh-sensitized. Rh-negative mothers should receive intramuscular 300ug of rhesus immunoglobulin ('ant i-D') any time there is a risk of fetomaternal haemorrhage (abortion, antepartum haemorrhage, amniocentecis, etc). Ideally the dosage of RhIgG should be 10ug per milliliter of whole fetal blood. However, this is rarely done due to the cost of the test and the fact that fetomaternal haemorrhage greater than 30mls (the volume covered by 300ug) occurs in only 0.4-1%.[4, 10] The life span of anti-D is 12 weeks. Mothers who are given the drug at 28weeks should therefore receive another 300ug at 40 weeks. However, to avoid this they are delivered instead. As happened with our patient, upon delivery, cord blood is taken for haemoglobin and bilirubin levels, direct Coomb's test (DCT), blood typing and if necessary cross match. If the baby's RBC is rhesus positive, the mother is given 300ug of anti-D. Our patient received the 300ug of anti-D because the baby was Rhesus positive and the negative DCT test and baby's haemoglobin level of 16g/dl indicated no prior isoimmunuzation.

In those who have no history of affected fetus, the patient is followed up by antibody titres at booking at 20 weeks and then every 4 weeks. As long as titres remain less than 1:32, no further intervention is required. But once antibodies reach 1:32 amniocentesis should be performed 4-8 weeks earlier than the gestational age in the previous pregnancy when rhesus associated morbidity was first identified. Cordocentsis, amniocenteses and ultra sound with Doppler velocimetry have been used in the follow up and management of these patients. The essence of amniocentesis is that liquor has bilirubin

and other blood breakdown products the concentration of which is directly proportional to the haemolysis of fetal blood. The amniotic fluid is analysed by spectrophotometry and the amount of light (wavelength 450) absorbed by the blood breakdown products (bilirubin) and plotted on semilogarithmic scale versus gestational age.

This forms the liley's chart, which is divided into three zones: zone1, Zone 3 and zone 3 in severity of fetal affliction.[11] The unaffected or mildly affected fetus falls into zone 1 Amniocentesis is repeated every 2-3 weeks and the fetus dwelivered near term. The moderately affected fetus falls into zone 2 where amniocentesis is repeated every 1-2 weeks. Delivery is generally before term as soon as pulmonary maturity is achieved. In the severely affected fetus in zone 3, intervention is usually needed to allow the fetus to reach a gestational age at which delivery and neonatal risks are fewer than the risks of uterotherapy. Intrauterine transfusion may be necessary to prevent the fetus from dying. This may be done by the old fashion intraperitoneal or the new, prevered intravascular methods.[4] In conjuction with Doppler velocimetry score and as a predictor for significant prehydropic fetal anaemia.[9]

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

REFERENCES

1. Levine P., Stetson R.E. An unsual case of intragroup agglunation journal of American Medical Association. 1939; 113:126
2. Landstaner K., Weiver A.S. An agglutinable factor in human blod recognized by immuned sera for rhesus blood. Proc soc exp bio med; 1940; 43:223.
3. Bergstorm H et al. Demonstration of Rh antigens I a 38 day fetus. American Journal of Obstetrics an Gynaecology, 1967; 99:130
4. Ashley S., Pernoll M.Rh. Isoimmunization and other blood group incompatibilities. Late pregnancycomplications. In current obstetrics and gynaecologic diagnosis and treatment, 9th edition 2003. Edited by Alan H. Dercheney and Lauren Nathan.
5. Mulandi T.N.A2year prospective study to show the effectiveness of anti-D immunoglobulin in prevention of Rh. Isoimmunization in rhesus negative pregnant (African) women at Kenyatta National Hospital. M.Med Thesis, University of Nairobi
6. Kango J.S Rhesus immunization in African women in Dar-es –Salaam. East African Medical Journal, 1981; 58:456
7. Zipursky A., Israels L.G. The pathogenesis and prevention of Rh. Immunization. Can Med Assoc J, 1967; 97:1245
8. Stedman C.M. et al use of erythrocyte rosette testto screen for excessive fetomaternal haemorrhage in the Rh. Negative women. American Journal of Obstetrics and Gynaecology, 1986; 154:1363.
9. Bowman J.M Controversies in Rhesus prophylaxis. Who needs Rh. Immune globulin and when should it be used? Am J. Obstet Gynaecology 1985; 151:289?

10. Standards committee, American association of blood banks. Standards for blood banks and transfusion services. 19th edition. Washington D.C American Association of blood banks, 1999.
11. Liley A.W. Liuor amnii analysis in the management of pregnancy complicated by rhesus sensitization. Am J. Obstet Gynaecol 182:1359 1961.

OBSTETRICS CASE NO. 12

INTRAPARTUM FOETAL DISTRESS – EMERGENCY CAESAREAN SECTION DONE: LIVE BIRTH

NAME: C.M. PARITY: 1+0 GRAVIDA 2

AGE: 21 YEARS LMP: 28. 07. 06

IPNO: 1021039 EDD: 04. 05. 07

DOA: 14. 04. 07 GESTATION: 37+ WEEKS

DOD: 19.04. 07

Presenting Complaints

She presented with a two days history of intermittent lower abdominal pains at 37+ weeks.

History of the Presenting Complaints

She was well till 2/7 prior to admission when she developed intermittent lower abdominal pains that was increasing in frequency and intensity. There was no history of per vaginal bleeding or drainage of liquor.

Obstetrics and Gynaecological history

She was para 1+0. Her first delivery was a spontaneous vaginal delivery in 2003 to an infant who weighed 3.5 kg who was alive and well. Her last normal menstrual period was on 28th July 2006 and her expected date of delivery on 4th May 2007. She was at a gestation of 37 weeks.

She had antenatal care at Kenyatta National Hospital from 23 weeks of gestation and had made 7 visits. Her blood group was 'O' Rhesus 'D" positive, VDRL and HIV test were negative and had a haemoglobin level of

12.1 g/dl. She had two tetanus toxoid injections and had no complications during the pregnancy.

She attained menarche at 16 years and prior to conception, she had a regular menstrual cycle of 30 days with duration of flow of 4 days. She had used no contraceptives.

Past Medical History

She had no significant medical or surgical history.

Family and social history

She was a housewife married to a businessman. She lived with her family at Karura in Nairobi. She did not smoke cigarettes or drink alcohol and had no history of chronic illness in the family.

PHYSICAL EXAMINATION

She was in fair general condition, was not pale, jaundiced or oedematous. She had a blood pressure of 117/79 mmHg, a pulse rate of 72/min, respiratory rate of 20/min and a temperature of 36^{O} C.

The central nervous system, respiratory system and the cardiovascular system were essentially normal.

Abdominal Examination

The abdomen was symmetrically distended and moving with respiration. The liver and the spleen were not palpable. The fundal height corresponded to a term gestation, the foetus was in longitudinal lie and cephalic presentation and the foetal heart tones were heard and regular with a rate of 134 beats per minute. Descend was 3/5. Three moderate contractions in 10 minute each lasting 20 seconds were palpated.

Pelvic Examination

She had normal external genitalia. The cervix was central in position, 1cm long. soft and 4 cm dilated. The membranes were intact.

Diagnosis

A diagnosis of active phase of labour was made.

Management

She was admitted to a first stage room in labour ward where her labour was monitored on a partograph. She was reviewed 4 hours later and found to have an irregular foetal heart rate of 108 to 128 beats per minute. Descend was 2/5 and had 3 contractions in 10 minutes each lasting 35 seconds. The cervix was fully effaced, soft, 7 cm dilated with no caput or excessive moulding. Artificial rupture of the membranes yielded thick meconium stained liquor. The umbilical cord was not palpated.

A diagnosis of foetal distress was made. The patient was counselled on the diagnosis and prepared for an emergency caesarean section. She was nursed in a left lateral position, put on intravenous dextrose infusion and given oxygen by mask. She gave an informed consent for emergency caesarean section and was premeditated with atropine 0.6 mg intramuscularly before being wheeled to the operating room.

An emergency caesarean section was performed though a lower midline abdominal incision and a transverse incision in the lower uterine segment. The outcome was a live female infant with an Apgar score of 6 in 1 minute and 8 in 5 minutes with a birth weight of 3450 grams. There was no cord entanglement or knots and the placenta looked grossly normal. There was thick meconium stained liquor. The infant was reviewed by the paediatrician

who admitted her to the newborn unit with suspected meconium aspiration syndrome.

Post- operative Management

The vital signs were observed half hourly until she was fully conscious, one hourly for 4 hours and then 4-hourly. She was put on intravenous fluids, 1 litre of dextrose alternating with normal saline 8-hourly for 24 hours. Analgesia was through intramuscular pethidine 100 mg 8-hourly for the first 24 hours then oral ibuprofen 400 mg 8-hourly. She was also put on intravenous benzyl-penicillin G 2 mega units and intravenous Gentamicin 80 mg 8-hourly for two days.

She did well postoperatively; the baby was discharged from the newborn unit on the third day. They were discharged home through the postnatal clinic in six weeks.

Post-natal Follow-up

She was seen in the post-natal clinic after 6 weeks. She had no complaints and the infant was well and exclusively breastfeeding. She was counselled on family planning and referred to the family planning clinic.

DISCUSSION

The patient presented was a para 1+0 diagnosed with intrapartum foetal distress who was delivered by caesarean section to a live female infant who was admitted to the newborn unit with suspected meconium aspiration syndrome.

Foetal distress may be defined as a complex of signs indicating a critical response in the foetus to stress. It implies metabolic derangement, notably hypoxia and acidosis that affect the functions of vital organs to the point of temporary or permanent injury or death. Foetal distress may be acute or chronic. Skilful monitoring will detect some degree of foetal compromise in at least 20% of all obstetric patients.[1]

The oxygen supply to the foetus depends principally on the adequacy of uterine perfusion, placental gas transfers and foetal circulation. The physiology of foetal circulation in terms of the higher percentage of foetal haemoglobin, the higher haemoglobin concentration and the higher affinity of foetal haemoglobin for oxygen enabling more efficient delivery of oxygen to the tissues renders the foetus relatively resistant to mild to moderate hypoxia. However, in severe hypoxia foetal tissue requirements for aerobic metabolism exceeds the oxygen supply and foetal tissue hypoxia results. The foetus as a result turns to anaerobic metabolism, which causes a rise in lactic acid levels and a consequent fall in the tissue and blood pH. Hypoxia for the foetus may arise from factors affecting intervillous space perfusion, placental oxygen transfer or umbilical blood flow.[2]

Maternal position, exercise or chronic maternal medical conditions such as hypertension and diabetes may be associated with decreased placental blood flow. During a normal uterine contraction, there is an acute interruption of blood flow through the intervillous space. If the contractions are prolonged

or tetanic the uteroplacental reserve may be exceeded and foetal hypoxia produced. Maternal hypotension, placental infarcts or premature placental separation may also affect oxygen delivery to the foetus. Oxygen transfer can actually be increased by administration of high oxygen concentration to the mother, which increases the maternal-foetal oxygen gradient and can significantly increase the foetal blood oxygen contact. [3]

The objective of monitoring the foetus in labour is to detect foetal abnormalities at a stage when they are reversible. The current modalities for the monitoring of the foetus are intermittent auscultation, cardiotocography, colour and quality of amniotic fluid and foetal blood sampling. Biophysical profile, moulding of the foetal head and caput formation serve as accessories to monitor the foetus.

No foetal compromise is present when there is absence of any abnormality of foetal heart rate (FHR) or rhythm and no response to uterine contractions other than early decelerations.[1]

Foetal heart rate patterns are reflections of underlying pathophysiologic mechanisms. Abnormal patterns may be indicative of hypoxia with or without acidosis. Evaluation of the foetal ability to tolerate decelerations requires examination of all components of the foetal heart rate pattern. Concomitant loss of variability or baseline tachycardia suggests the possibility of hypoxia and acidosis. If the return to baseline is gradual rather than abrupt the clinician should be suspicious of progressively worsening hypoxia and possibly progressive foetal depression. If variability and baseline are normal, the foetus can be assumed to be tolerating the intermittent stress, changes in foetal heart rate may indicate early hypoxia without acidosis.[1]

Measurement of 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 umbilical venous blood and approaches that of umbilical arterial blood. The following protocol has been recommended to try and confirm foetal distress. If pH is greater than 7.25, then labour is observed. If the pH is between 7.20 and 7.25, pH measurement is repeated within 30 minutes. If the pH is less than 7.20, another scalp blood measurement is taken immediately and the mother taken to the operating room for preparation for surgery. If the pH is still low immediate caesarean delivery is performed. [5] In our setup, foetal scalp blood is not done and hence it is difficult to objectively determine the foetus that is actually having foetal distress.

The significance of the presence of meconium in labour is controversial and likewise the necessity of rupturing the membranes in an attempt to detect the presence of meconium. The presence of thick meconium in labour particularly in association with post-term pregnancy, oligohydramnios and poor foetal growth has been associated with increased risk of acidemia which then increases the risk of meconium aspiration.[2] Meconium staining without foetal heart abnormalities or foetal scalp abnormalities and with labour progressing well seems to have no great significance. Meconium in the presence of complicated labour with foetal heart rate abnormalities has a greater risk of foetal hypoxia than either meconium alone or foetal heart rate abnormalities alone. [6] Our patient had thick meconium stained liquor with foetal heart rate irregularity.

In cases of possible foetal compromise, vaginal examination should be done to assess for rapid progression or cord prolapse. Intrauterine resuscitation will help improve the condition of the foetus and may help avoid unnecessary intervention. Intrauterine resuscitation measures include:

1. Maternal position change often alleviates cord compression - turning the patient on her side improves uterine blood flow by relieving uterine pressure on the maternal aorta and vena cava. A good rule in the labour room is that all patients should recline as much as possible in a semi lateral position [1,2,3,4]

2. Maternal hypotension should be corrected by intravenous hydration, position change or vasopressor treatment if the cause is conduction anaesthesia related vasodilatation. Ephedrine is the drug of choice in this situation.

3. Administration of supplemental oxygen to the mother results in improved foetal oxygenation, assuming that placental exchange is adequate and umbilical cord circulation is unobstructed.

4. Oxytocin should be discontinued until foetal heart rate and uterine activity return to acceptable levels.

5. Vibroacoustic stimulation (VAS) or foetal scalp stimulation may be used to induce accelerations in foetal heart rate that indicate the absence of acidosis.

6. Tocolytic agents such as Beta-adrenergic agonists can be administered to decrease uterine activity in the presence of uterine hypertonus with non reassuring foetal heart rate patterns.

7. Therapeutic amnioinfusion should be considered for repetitive, non reassuring variable decelerations. [4] The therapeutic goal is expansion of the amniotic fluid volume. Amnioinfusion has been shown to be useful in the management of variable decelerations in foetuses with oligohydramnios and subsequent cord compression. Amnioinfusion has also been used for dilution and lavage of meconium.[4]

8. In the past, bolus doses of hypertonic dextrose were used for the management of foetal distress. But the use of dextrose has been shown to be of little value.[1]

9. If maternal acidosis is the cause of foetal acidosis, administering bicarbonate to the mother may benefit both patients.

Labour may be continued in the presence of reassuring signs of foetal status through foetal acoustic stimulation, scalp stimulation, or sampling. If foetal well-being cannot be documented, if the situation worsens, if the signs of probable foetal distress persist for 30 minutes, or if there is continued foetal distress despite conservative treatment, immediate delivery is indicated. If caesarean section is chosen, it must be done rapidly.[1] in our patient, we relied mainly on foetal heart rate and grade of meconium staining to monitor the foetal well being and the decisions made thereof.

REFERENCES

1. Polo A., Chang L, Herrera E., Pernoll M.L. Complications of labour and delivery. In: DeCherney AH, Nathan L. (Eds): Current Obstetric and Gynaecologic Diagnosis and Treatment, 9th edition, McGraw Hill, 2003:474 - 476.

2. Tank JD. Intrapartum Foetal Distress. In: Krishna U, Tank DK, Daftary S (Eds) Pregnancy at Risk Current Concepts, 4th edition, Jaypee Brothers, New Delhi, 2001: 426 - 430.

3. Robinson E., Bienstock J. Fetal Assessment, Normal Labour and Delivery. In: Lambrou NC, Morse AN, Wallach EE. The John Hopkins Manual of Gynaecology and Obstetrics, Lippincott Williams and Wilkins, Philadelphia 1999:28-29.

4. Berkowitz K, Nageotte P. Intrapartum Fetal monitoring. In: Evans AT, Niswander KR. (Eds). Manual of Obstetrics 6th edition, Lippincott Williams and Wilkins, Philadelphia 2000: 313 - 329.

5. Zallar RW, Quilligan E.J. The influence of scalp sampling on caesarean section rate for fetal distress. Am J. Obstet. Gynecol., 1979; 135: 239.

6. Spencer JA. Induction and Augmentation of labour. In: Dewhurst's Textbook of Obstetric and Gynaecology; 6th edition,. Blackwell Scientific Publishers 1999:259 - 275.

OBSTETRICS CASE NO. 13

DEEP VENOUS THROMBOSIS IN PREGNANCY

NAME: T.N.K L.M.P:20/6/02

AGE: 32 YEARS E.D.D:27/03/03

IP NO: 0860412

D.O.A: 22.01.06 PARITY: 2 +0

D.O.D: 09. 04.06

Presenting complaint

She presented with painful swelling of the left lower limb for one week.

History of presenting complaint

She was admitted through casualty with a one-week history of a painful left lower limb swelling. There was no history of trauma prior to the onset of the swelling. She however gave a history of having had wounds on the left leg for which she had received a tetanus toxoid a month prior to the onset of the swelling. There was history of fever. She had no history of cough, chest pain or difficulty in breathing.

She had not begun her antenatal clinic. She reported quickening in October.

Obstetric and gynaecologic history

She was a para 2 + 0 with 2 living children. Both were vaginal deliveries. The first was in 1993 in hospital and the outcome was a 2900g female infant. The second was in 1997 in hospital and the outcome was a 3900g male.

She gave no history of similar symptoms in her previous pregnancies.

She attained menarche at 15 yrs. prior to pregnancy, her periods were regular, coming every 28 days and lasting for 3 days. It was of moderate

flow with no associated dysmenorrhoea. She had used an IUCD from 1993 to 1996. She gave no history of use of oral contraceptive pills.

Past Medical and Surgical History

She had no previous history of thrombo-embolism and had no previous history of hospitalisation.

Family and Social History

She was a housewife, who lived in Murang'a with her 2 children and husband. She did not smoke cigarettes or drink alcohol.

PHYSICAL EXAMINATION

She was in fair general condition, had no pallor, jaundice, cyanosis or dehydration. She had a blood pressure of 110/60 mmHg, a pulse rate of 80/ min, respiratory rate of 20/min, and a temperature of 36.8^{O} C.

Abdominal Examination

There was uniform abdominal distension and the abdomen was moving with respirations. The liver and the spleen were not palpable. The fundal height corresponded to 32 weeks gestation which was in keeping with the dates. The foetus was in longitudinal lie and cephalic presentation and the foetal heart tones were heard and regular at 132 beats per minute.

Cardiovascular and respiratory systems

She had a regular pulse of 80 beats per minute and the jugular venous pulse was not elevated. The apex beat was in the 5^{th} intercostal space anterior axillary line, the first and second heart sounds were heard and were normal and she had no murmurs.

She was not in respiratory distress; she had good breath sounds bilaterally with no crepitations or rhonchi.

Vaginal Examination

Vaginal examination was not done at this time as it was not indicated.

Local examination

The left lower limb was swollen from the thigh to the foot. It was warm and shiny over the thigh and no varicosities were noted. Measurements were taken using a tape measure 25 cm below the anterior superior iliac spine for the thigh and 10cm below the tibial tuberosity for the leg.The circumferences of the thighs were 60 cm on the left and 56 cm on the right and the circumferences of the legs were 38 cm on the left and 34 cm on the right. The difference in thigh and leg circumference was significant.

IMPRESSION

An impression of deep venous thrombosis at 32 weeks gestation was made with cellulitis as a differential diagnosis

Investigations

1) left lower limb velocimetry

There was sluggish flow in the common femoral vein with poor compression. Response to augmentation was not adequate. There was increased flow noted in the greater saphenous vein. There was flow in the popliteal veins which showed adequate compression and augmentation response.

Conclusion: thrombosis of the left common femoral vein.

2) Haemogram

WBCC : 5.5×10^9/l.

Hb : 10.5g/dl

Platelets : 148 x 10^9/l

3) VDRL negative

4) Eliza for HIV I and II negative

5) Obstetric scan : a single, viable fetus in cephalic presentation. Fetal heart rate was 136 bpm. Average u/sound gestation was corresponding to 34 weeks . biophysical profile was 8/8.

MANAGEMENT

She was admitted to the antenatal ward and started on intravenous infusion of heparin 10,000 units in normal saline 6 hourly. She was also put on intravenous Augmentin® 1.2g 8 hourly to cover for possible cellulitis and Ibuprofen 400mg 8 hourly for pain relief. The affected limb was elevated on the bed with the aid of pillows.

Progress was monitored by daily measurement of leg and thigh circumferences. Heparin therapy was monitored using activated partial thromboplastin time (aPTT or KCCT). The dose was readjusted to 7500 units 8 hourly after aPTT results.

APTT was done 4 days after commencement of intravenous infusion of heparin 10,000 units 8 hourly.

Test 111 sec.

Control 36 sec

Ratio - approximately 3.

This reflected an over anti-coagulation with heparin.

Repeat APTT after adjusting intravenous infusion of heparin to 7500 units 8 hourly

Test :- 61 sec

Control:- 34 sec

Ratio:- 1.79

Follow up

The infusion was continued for one week. The acute symptoms of pain and swelling were seen to have reduced and the medication was changed to subcutaneous heparin. APTT was monitored weekly. Subsequent ratios ranged from 1.5 to 2. The calf and thigh circumferences were measured frequently and revealed progressive reduction of the thigh circumference from the original 60 cm to 56 cm and the calf from 38 cm to 34 cm.

At 40 weeks, a decision to induce labor was made. This was discussed with the patient and reasons for the decision were explained to her. She said she understood and gave verbal consent for the induction.

Labour ward

She was assessed in labour ward . The FH was term with a cephalic presentation and longitudinal lie. The fetal heart was heard and regular at 140 bpm.

The fetal head was 3/5th up. She had no contractions

The cervical assessment indicated a bishop score of 3 out of 13. cervical ripening was done using misoprostal 50 micrograms placed in the posterior fornix. The subsequent dose of heparin was withheld. Blood was grouped and cross matched and 2 units of fresh blood reserved for her. Protamine sulphate was also kept ready for her. The haematologist who was assisting in

the management was alerted. After 6 hours she developed labour like pains. Cervical assessment showed that she was 4 cm dilated. Sweeping of the lower segment was done followed by ARM. Clear liquor was obtained. No cord was felt. Partogram recording was commenced.

Labour progressed well and was uneventful. She had a spontaneous vertex delivery to a LMI who had an apgar score of 6, 8, & 10 at 1, 5 & 10 minutes respectively. There was no perineal or cervical tear. Estimated blood loss was 200mls.

Postpartum

Heparin was restarted afer 8 hours. She did well post partum. She was mobilised on the 3rd post partum day. Lactation was well established, uterus was well contracted and lochia loss was normal. Calf muscles were non tender and equal in circumference bilaterally. She was also commenced on wafarin 5 mg which was gven concurrently with heparin for 3 days after which heparin was stopped. She continued with the warfarin at the same dosage. PTI indicated good control INR – 2.4.

She was advised against use of hormonal contraceptives especially the oestrogen based. She was advised to use IUCD which she was inserted on the 6th post natal week. She was advised to start ANC early in case of another pregnancy.

She was discharged home on the 10th postnatal day through the postnatal and haematology clinic.

DISCUSSION

The case presented is of a 32 year old para 2+ 0 gravida 3 admitted with left lower limb deep venous thrombosis at 32 weeks of gestation who was managed with heparin and warfarin.

The risk of venous thromboembolism is five times higher among pregnant women as among non-pregnant women of similar age [1-3]. Estimates of the incidence of pregnancy associated venous thromboembolism vary from 1 in 1000 to 1 in 200 deliveries [4]. Thromboembolic complications, including pulmonary embolism, are major causes of death among women during pregnancy and the puerperium [5]. Thus venous thromboembolism is an uncommon but leading cause of morbidity and mortality among women during this period.

The incidence of thromboembolism is 0.2% in the antepartum period and 0.6% in the postpartum period. Caesarean section increases the incidence to 1-2% [6] At Kenyatta National Hospital (KNH) an incidence of 1.6% has been reported [7]

Vascular clotting develops mainly due to circulatory stasis, infection, vascular damage, or increased coagulability of blood. All the elements of Virchow's triad (circulatory stasis, vascular damage and hypercoagulability of blood) are present during pregnancy. Increase in calibre of capacitance vessels produce vascular stasis, and blood hypercoagulability is due to increased amounts of factors VII, VIII, and X. Thrombin- mediated fibrin generation is increased many times during pregnancy. Significant vascular damage occurs during delivery. Venous return from the lower extremities is reduced by the pressure of the gravid uterus on both the iliac veins and the inferior vena cava. Other important predisposing factors include heavy cigarette smoking , obesity, previous thromboembolism, anaemia, haemorrhage, heart disease, hypertensive disorders, prolonged labour, operative delivery and postpartum endomyometritis.

Almost 90% of DVT affect the left side among pregnant women with 55% among women who are not pregnant [8]. This difference may reflect

compression of the left common iliac vein by the right common iliac artery and the left ovarian artery which cross the vein on the left side only. Furthermore in pregnancy most cases are ileofemoral rather than calf vein thrombosis (7.2% vs 9%) and ileofemoral DVT is more likely than calf vein thrombosis to lead to pulmonary thrombo-embolism.

DVT in pregnancy can present or be associated with, lower abdominal pain due to peri-ovarian collateral circulation or thrombosis. When coupled with the mild pyrexia and leucocytosis of thromboembolism, this pain can be mistaken for other intraabdominal disorders such as urinary tract infection or appendicitis.[8]

Clinical evidence of DVT of the legs precedes pulmonary thromboembolism in only about half the cases. Importantly 40% of asymptomatic patients with DVT are found to have a concomitant pulmonary embolism. Chest-discomfort, shortness of breath, air hunger, tachypnoea and obvious apprehension are signs and symptoms that should alert the physician to a strong likelihood of pulmonary embolism. The most reliable symptom is breathlessness [9]

The presently available techniques for the objective diagnosis of DVT include contrast venography or phlebography, non-invasive methods, and biochemical assays. Venous ultrasonographic imaging is most widely used and has largely replaced venography. Proximal veins are compressed under gentle pressure with the ultrasound transducer (compression ultrasonography) the inability to compress a vein indicates presence of DVT [9, 10]. Impedance plethysmography, real time B-mode ultrasonography, magnetic resonance imaging and computed tomography are some of the other tests that can be used. Venography remains the standard for confirmation but it has the risk of inducing thrombosis itself [9]. The

sensitivity of real-time ultrasonography has been shown to be100% with a specificity of 99% using contrast venography as the standard[11] Magnetic resonance imaging is reserved for specific cases in which ultrasound findings are equivocal. Computed tomographic scanning may also be used to assess the lower extremities.

Treatment options in venous thrombosis include anticoagulation, caval filters, fibrinolytic therapy, and surgical thrombectomy [10]. The last three therapeutic approaches have been assessed less extensively and are not routinely used. A combination of anticoagulation, analgesics and bed rest is usually adequate in the management of antepartum DVT; the gestational age should be taken into account in order to avoid warfarin in the first trimester and after 36 weeks. Anticoagulation is initially achieved with heparin such that the activated partial thromboplastin time (aPTT) is prolonged to 1.5 to 2.5 times the laboratory control value. Heparin may be given by several regimes:

- The dosage schedule recommended by the American College of Obstetricians and Gynaecologist include a loading dose of 80U/Kg (Minimum 5000U). This is followed by a continuous infusion of heparin, at a dose of 15-25 U/kg/Hr. After 4hrs, the activated partial thromboplastin time (aPTT) is determined and adjusted accordingly. Once a steady state is achieved, aPTT is measured daily.
- Subcutaneous adjusted dose heparin may also be used in the treatment of DVT to maintain the aPTT at 1.5 times the control value as determined at 6 hrs after the last injection.
- Alternatively treatment with one of the low molecular weight heparin compounds is suitable.

If IV heparin is introduced at the same time as oral warfarin, then heparin can be safely discontinued after 5 days [9]. The ideal duration of therapy for pregnant women is undetermined.

The American College of Obstetricians and Gynaecologists recommend therapeutic subcutaneous heparin throughout pregnancy and 6 to 12 weeks postpartum.

Warfarin crosses the placenta and is teratogenic and if given in the first 8 weeks of pregnancy may result in congenital abnormalities, which include nasal hypoplasia, ophthalmological abnormalities and retarded development. Central nervous system abnormalities have also been reported with second and third trimester coumarin explosure [9].

During delivery, heparin does not cross the placenta. Its effects on blood loss at delivery will depend upon a number of variables including the dose, route and time of administration, the magnitude of incisions and lacerations; the intensity of postpartum myometrial contractions and retraction; the presence of other coagulation defects. In general, therapeutic heparin therapy should be stopped during the time of labour and delivery. If the uterus is well contracted and there has been negligible trauma to the lower genital tract, it can be restarted within several hours. Otherwise, a delay of 1 or 2 days may be prudent. Protamine sulphate 1 mg per 100U of heparin administered slowly IV will generally relieve the effects of heparin promptly and effectively. Protamine sulphate should not be given in excess of the amount needed to neutralise heparin because it has an anticoagulant effect [9].

After delivery, thromboprophylaxis should be continued for a minimum of 6 weeks, but in patients with severe thrombotic problems, for 3 months [8]. Many patients with underlying congenital or acquired thrombophilia will require antenatal prophylaxis, the timing of which will depend on the

patient's history and thrombophilic disorder. The practise in our set up is to continue warfarin treatment for a period of 6 months postpartum from the time of diagnosis. It is also recommended that heparin prophylaxis be given in subsequent pregnancies beginning a month before the gestation date of onset of DVT in the previous pregnancy.

The indications for preventive therapy include previous documented DVT or pulmonary embolism or antithrombin III deficiency. Heparin is the drug of choice given 5000-7500 units subcutaneous twice daily during the first and second trimester. Around the beginning of the third trimester, the dosage increased by approximately one-third to provide additional anticoagulation for the increased coagulation factors in late pregnancy. Prophylaxis should be stopped with the outset of labour and started again following delivery and continued for at least 2 weeks [6].

REFERENCES

1. Prevention of Venous Thrombosis and Pulmonary Embolism: NIH Consensus Development JAMA 1986; 256: 74-9.
2. Kiekegaard A; Incidence and Diagnosis of Deep Vein Thrombosis associated with Pregnancy; Acta Obstet Gynaecol Scad 1983: 62:239-43.
3. Treffers PE, Huidecoper PL, Weenink GH, Kloosterman GJ. Epidemiological Observations of Thromboembolic Disease during Pregnancy and in the Puerperium in 56, 022 Women. Int J. Gynaecol Obstet 1983; 2:32-31.
4. Rutherford S, Montora M, McGehee W, Strong T. Thromboembolic Disease Associated With Pregnancy, an 11 Year Review. Am J Obstet Gynaecol 1991: 164: Suppl : 286

5. Hellgren M, Sensson PJ, Dahlback B, Resistance to Activated Protein C as a Basis for Venous Thromboembolism Associated With Pregnancy And Oral Contraceptives. Am J. Obstet Gynaecol 1995; 73:210-13

6. Grewal M, Biswas MK, Perloff D. Thromboembolization. In. DeCherney AH, Nathan L (Eds). Current Obstetric and Gynaecologic Diagnosis and Treatment; 9th Edition McGraw Hill Companies, New York, 2003:416-419.

7. Waweru JM. Deep Venous. Thrombosis. Mmed Thesis, University of Nairobi, 1981.

8. Greer IA: Thrombosis in Pregnancy: Maternal and Fetal Issues. Lancet 1999: 353: 1258 –1265.

9. Cunningham FG, Gant NF, Leveno KJ et al. Pulmonary Disorders. In: Williams Obstetrics 21st Edition, McGraw Hill, New York City, 2001:234-1240.

10. Anthonie LWA, Prandoni P, Martin HP, Buller HR. Deep Vein Thrombosis. Lancet 1999; 353: 479-85.

11. Lensing AWA, Prandoni P, Brandjes D, et al. Detection of Deep Vein Thrombosis by Real Time B-Mode Ultrasonography. N Eng J Med 1989. 320:342.

OBSTETRIC CASE NO.14

CEPHALO-PELVIC DISPROPOTION IN LABOUR-EMERGENCY CEASAREAN SECTION: LIVE BABY.

Name: S.I.O

DOA: 22/09/06

Age: 28 years

DOD: 25/09/06

Parity: 1+0 gravida

File No: 0985002

LMP: 13/12/05

EDD: 20/09/06

GBD: 40 + weeks

Presenting Complaint

S.I.O was admitted to the Kenyatta National Hospital on 22/09/06 at 8.40p.m as a referral from St. Luke's surgery nursing home with complaints of labour pains for 25 hours.

History of presenting complaints

She had been well until she developed intermittent lower abdominal pains that increased in frequency and intensity on 21/09/06 at 7pm. She had no urinary symptoms or change in bowel habits. She was admitted to St. Luke's Nursing home at 11.20p.m and was found to be labour with cervical dilation of 2cm. At about 9am on 22/09/06, a drip of syntocinon was started, but by 4p.m, the cervix remained 7cm dilated and the head was still high above the pelvic brim, hence the referrel. The referreal note indicated that she had had 3 contractions in 10 minutes each lasting 45 seconds for 5 hours in active labour without progress of labor.

Obstetric and Gynaecologic history

She was a para 1+0 gravida 2. Her last menstrual period (LMP) was on 13/12/05, the expected date of delivery (EDD) was on 20/09/06 and therefore the gestation by dates (GBD) was 40weeks plus 2days. She had started antenatal care (ANC) at the St Luke's surgery nursing home from 24 weeks gestation. Then and throughout the follow-up the fundal height corresponded to the gestation by dates.

The antenatal profile done at 28 weeks gestation was: Hb; 13.0gm/dl, blood group B+ve and serological tests for VDRL and HIV were negative. She received 2 tetanus toxoid vaccines at monthly interval. Her blood pressure remained normal throughout the pregnancy. Urinalysis was done only once at 24 weeks and had no abnormality. The last 4 weeks she developed frequent urination without burning sensation .Her first delivery was in 2000, a hospital delivery at term by spontaneous vertex to a live male infant with a birth weight of 3600 grams. He cried immediately after birth and was alive and well. Pre-intra-and postpartum periods were uneventful. She attained menarche at the age of 15 years and since then menses for 3 days every 26to 28 days without any menstrual anomaly. She used the combined oral contraceptive pill six months after the first delivery in 2000 to October 2003 when she stopped to conceive. She had no pap smear done.

Past medical history

This was insignificant

Family and social history

She was a married lady who sold second hand clothes and smoked no tobacco and drank no alcohol. She had attained only secondary education. She lived with her husband in Dagoreti corner, Nairobi. The

husband was a counselor and worked with Dagoreti community voluntary counseling and testing (VCT) for HIV. There was no history of chronic illness such as diabetes mellitus in the family.

PHYSICAL EXAMINATION

General examination

She was in fair general and nutritional condition, not of short stature, mildly anxious and dehydrated but without pallor, pedal edema, jaundice or lymphadenopathy. The BP was 130/80mmHg, the pulse was of good volume at 84/minute, the temperature was 36.5^{0}c and the RR was 22/minute. Urinalysis showed moderate ketonuria.

Abdominal examination

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

Pelvic examination

She had external genitalia and healthy vaginal mucsa. The cervix was 8cm dilated, fully effaced and slightly edematous. There was mild caput succedaneum and no moulding. The sacral promontory could not be reached, the pelvic contours were not suggestive of a contracted pelvis and the ischial spines were not prominent. Meconium staining liquor grade 1 was present. The knuckles of a clenched fist could easily fit in the intertuberous diameter of the outlet.

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

Impression

A diagnosis of Cephalo-Pelvic Disproportion (CPD) was made.

MANAGEMENT

The clinical findings and the diagnosis were explained to the patient. She was to undergo an emergency caesarean section. An informed consent was obtained and blood taken for grouping and cross match. She was shaved and premedicated with 0.6mg of intramuscular atropine. In theatre vulvo-vaginal toilet and catheterization drained 100mls of

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

Post operative care

The immediate postoperative care was explained in the introduction. Intravenous fluids and antibiotics (crystalline penicillin, gentamicin and metronidazoe) and intramuscular pethidine were started. On the first postoperative day she was ambulant, had passed urine three times, had normal vital signs and was not pale. The breasts were soft and not yet active, the chest was clear, the abdomen was soft and moved with respiration and had normal bowel sounds. The uterine fundal height corresponded to 20 weeks gestation and was well contracted. The lochia loss was normal and the calves were soft and non-tender. In view of the large for gestational age infant and the history suggestive of gestational diabetes, a random blood sugar test was done and was 6.7mmol/L. a dipstick urinalysis showed glycosuria of 1+. Oral slips preceded gradual introduction of light diet by evening. On the 2nd day, oral medications were started and light diet encouraged. The baby had been found to be

without abnormal hypoglycaemia or any other anomaly. Breastfeeding was encouraged. A fasting blood sugar done then was 5.8-mmol/L. she was advised to undergo oral glucose tolerance test (OGGT) in the 6th postpartum week. On the 3rd day, she was ready to go home and was discharged with advice to attend the antennal clinic in the 6th postpartum week or any time she deceloped any complications. She was to do the OGtt a day or two prior to attending the clinic.

Follow – up

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

DISCUSSION

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

Cephalopelvic disproportional (CPD) is defined as the disparity in relation between the feta head (cephalo) and the pelvis (pelvic).[1, 2, 3] It

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

Cephalo-pelvic disproportion should be distinguished from Contracted pelvis. Whereas CPD commonly occurs in contracted pelvises, it does also occur in normal pelvises when the fetal head is bigger than the pelvic passage. Besides, although clinical pelvimetry can be used to

diagnose a contracted pelvis, it is usually confirmed by radiological pelvimetry and unlike CPD is not dependant on the size of the fetus and is always recurrent. CPD can be recurrent if there is a contracted pelvis. Although our patient had no clinical signs of a contracted pelvis, the fetus was large for gestational age (LGA-4100 grams), the fetal head remained unengaged despite adequate contractions and vaginal examination revealed caput succedaneum and moulding, hence the diagnosis of CPD.

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

1. Pelvic inlet: a) anterior-posterior (AP) diameter-11cm

   b) Transverse diameter – 13.5cm

2. Pelvic mid-cavity: both transverse and antero-posterior diameters are 12 cm

3. Pelvic outlet: a) anterior-posterior diameter- 13.5cm

   b) Transverse diameter – 11.0cm

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

The widest diameter of the fetal head, the biparietal diameter, in vertex presentation is on average is 9.5cm. The pelvis is contracted if on radiological pelvimetry one or all of pelvic dimensions (the inlet, the mid-cavity and the pelvic outlet) are diminished. A contracted pelvis by obstetric definition is the alteration of the size and/or the shape of the pelvis of sufficient degree so as to alter the normal mechanism of labour in an average size baby [5] The pelvic inlet is contracted if the AP diameter is less than 10cm and/or if the transverse diameter is less than

12cm. Clinically, the inlet could be estimated to be contracted if the mother is of short stature (less than 150cm), puts on shoe size 4 and below, the sacral promontory could easily be tipped, the head floats easily above the pelvic brim despite uterine contractions or on performing the Muller-Munro-Kerr test. [5] Other factors associated with inlet CPD include malpresentation and early rupture of the membranes. In women with contracted pelvis, face and shoulder presentation are encountered three times more frequently and cord prolapse occurs 4-6 times more frequently.[1, 2]

According to Cheng and Huang the pelvic mid cavity is contracted when the sum of the interspinous and posterior sagittal diameters (normal 10.5 and 5 cm respectively) is 13.5 and below[6]. Clinical features of contracted mid pelvis include prominent ischial spines, converging pelvic sidewalls, flat and long sacrum and narrow sacrosciatic notch. The pelvic outlet is usually defined as diminution of the interischial tuberous diameter to 8cm or less. [1, 2, 5] Clinically the sub-pubic angle is acute and the knuckles of a clenched fist cannot enter the intertuberous diameter.[5] There is no definitive fetal size and/or fetal head biparietal diameter that is indicative of CPD.[1, 2, 3] Although the American College of Obestetricians and Gynaecologists has recommended an estimated fetal weight of 4250-4500 grams as the cut off point for caesarean section, this is usually for the diabetic mothers where shoulder dystocia is to be pre-empted. [7]

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

1. Malnutrition: rickets in childhood and severe prolonged osteomalacia in adult multipara women, stunted growth due to severe under-nutrition.

2. Disease or injury of bones: a) pelvic tumours, tubercular arthritis, traumatic and pathologic fracture, b) spinal deformities: kyphosis, scoliosis, spolilolisthesis, coccygeal deformity, c) lower limbs: congenital dislocation of the hip, hip joint disease,

3. Development defects: Naegele's and Roberts pelves, sacralization or lumberization of the vertebrae.

4. Endocrine disorders: diabetes mellitus (causing fetal macrosomia), precocious puberty and early fusion of epiphysial plates.

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

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

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

of the head despite strong uterine contractions are all suggestive of CPD.[1, 2, 5] All these were present in our patient.

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

1. Premature induction of labour
2. Elective ceasarean section at term or
3. Trial of labour

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

Trial of labour is the conduction of spontaneous labour in a moderate degree of CPD in an institution trained personnel and facilities of fetal and maternal wellbeing monitoring and for performing emergency caesarean section it aims at avoiding unnecessary cesarean section and achieving good maternal and fetal outcome[2, 5]. The patient should tentatively be prepared for, counseled on and consent obtained for

emergency cesarean section. Ideally labour should be monitored with a cardiotocogram but otherwise the progress of labour is meticulously monitored using the partogram and if there is inadequate uterine contractions labour is augmented with oxytocin. This requires that cautious amniotomy be performed when the cervix is at least 3cm dilated and cord prolapse be anticipated if the head is not yet fixed. The presence and the degree of meconium in the liquor should also be looked for once amniotomy has been done.

The length of trial of labour is individualized and depends on the progress of labour, the degree of CPD and the maternal and fetal well-being and the second stage of labour should not last more than ½ an hour.[1, 5] In India the outcome of trial of labor is spontaneous vaginal delivery in 30%, ventouse or forceps delivery in 30% and caesarean section in 40% the complication of CPD varies from center to center and depends on the level of obstetric care the patient gets[5]. They include a high caesarean section rate including repeat, obstructed labour and its attendant maternal and fetal morbidity and mortality. These are feta lintracranial haemorrhages, severe caput succedaneum, asphyxia or death, uterine rupture, genital tract injuries including fistulations and lacerations and fetal and/or maternal sepsis and death[1, 2,3,5] Fortunately our patient had none of these complications except the caesarean section which had a good outcome.

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

Our patient was fortunate to have been referred early hence the good outcome.

REFERENCES

1. Cunningham F.G. et al. Abnormal labor: fetopelvic disproportion in: Wiliams Obstetrics, 21st edition; 2001 McGraw Hill Companies. Pp 436-450.
2. Stuart C., Christopher L. Anatomy of the female pelvis and fetus relevant to labor. In: Obstetrics by Ten Teachers, 17th Edition; Arnold Publishers; 2000:pp104-108
3. Steven W., Ainbinder M.D. Operative delivery: Cephalopelvic disproportion. In: Current Obstetric and Gynaecology Diagnosis and Treatment, 9th Edition; Mcgraw Hill Companies. Pp518-520.2003.
4. Karanja S.K. Prospective study on the patter of caesarean section at Pumwani Maternal Hospital M.Med Thesis, 1991, University of Nairobi.
5. Dutta D.C. Contracted pelvis. In: Textbook of Obstetrics, 5th edition; 2001. New Central Book Agency, Calcuta, India: pp 366-381.
6. Chen H.Y., Huang S.C. Evaluation of midpelvic contraction. Int Surg 67:516,1982
7. American College of Obstetricians and Gynaecologists: Dystocia. Technical Bulletin No.137, December 1989.

OBSTETRIC CASE NO.15

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

Name: M.K DOA: 13/7/04

Age: 27 years DOD: 3/8/04

Parity: 3+0 gravida 4 File No: 0975033

LMP: 14/10/03

EDD: 22/07/04

GBD: 38+ Weeks

Presenting Complaint

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

History of presenting complaint

She had been well until 10p.m on 12/07/04 when she developed labor pains. She and her husband could not get means of transport immediately and it was not until 3a.m on 13/07/04 that she reported at PMH. Being a mother with three previous caesarean section deliveries in established labor by then, she was prepared for emergency caesarean section. Unfortunately this was not possible since there was an ongoing operation in the only operational theatre in PMH. While she was waiting for her turn to be operated, she developed progressive persistent lower abdominal pain associated with very bad feeling. At about 5a.m the persistent pain suddenly ceased but the bad feeling persisted and she developed heavy vaginal bleeding associated with

dizziness, headache, blurred vision and fainting tendency. She was referred to Kenyatta National Hospital (KNH) but again lack of immediate transport led to her being admitted to KNH labor ward at 7:45am, virtually three hours later.

Antenatal History

She had her antenatal care in a health centre in Kisii, her rural home. She had menses only once six weeks after her last delivery in June 2003. She breastfed exclusively for only one month and did not use any contraceptive method despite being advised to.She first noted pregnancy symptoms in December. Quickening was about late March or early April 2004, giving an estimated gestation of 36-40 weeks by extrapolation of the quickening date. She had visited the health centre only three times from about 26 weeks. Blood and urine had been taken for antennal profile. The ANC card was unavailable but she said the results had been all right. Despite being unsure of her dates, an obstetric scan had not been done. The prenatal period had been uneventful and she had planned to deliver at PMH.

Past Obstetric and Gynecology History

She was a para 3+0 gravida 4 at 38 weeks gestation by dates. All her three deliveries had been by emergency cesarean section and all her children were alive and well. The first baby, a life female infant, 3.5 kg was delivered in 1996 by emergency cesarean section due to malposition in labor at term. The second baby delivered in 2000 by emergency cesarean section due to foetal distress with one cesarean section scar, was a live female infant with a birth weight of 3kg. The third baby, a life male infant with a birth weight of 3.4kg was delivered in June 2003 also by emergency cesarean section due to 2

previous cesarean section scars in labor at term. All the caesarean sections were uneventful. She had not used any contraceptive method despite being advised to use an intrauterine contraceptive device (IUCD) since she had goiter.

Past Medical History

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

Family and Social History

She was a married woman who had schooled up to form 2. She did not smoke tobacco or drink alcohol. Her husband and her were small scale grocers in Nairobi and Kisii respectively. The husband also worked with Keroka bus services limited and lived in Limuru 40km from PMH in Nairobi.

PHYSICAL EXAMINATION

General examination

She was sick looking, moderately pale, anxious and moderately dehydrated. She had no jaundice lymphadenopathy or obvious cyanosis but had mild pedal oedema. Her BP was 142/94mmHg. Pulse was of satisfactory volume and regular at 92/min. temperature was 36.5^{0}C. Two pints of blood ran through 2 cannulae on her forearms.

Thyroid examination

There was diffuse enlargement of the whole gland. It had a smooth surface and moved up with deglutition. Either of the lobes measured about 3x4x5cm.

Abdominal examination

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

Pelvic examination

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

Cardiovascular system

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

Respiratory and central nervous system

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

DIAGNOSIS

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

MANAGEMENT

Immediate preparation for emergency laparotomy was instituted. Blood transfusion was maintained, blood taken for urgent grouping and cross-matching, atropine given and informed consent obtained. The surgical team including a consultant obstetrician and gynaecologist was assembled.

Laparotomy revealed haemoperitoneum of about 1500mls, and an extruded male fresh stillbirth with a birth weight of 3100 grams. An extensive T-shaped uterine rupture was found. The transverse tear was annular along the previous scar while the vertical one extended up to the cervix and led to avulsion and vertical tear of the urinary bladder from the fundus to the level of the apex of the trigone. The tear was about 4cm long. The ureteric orifices were patent. This necessitated subtotal hysterectomy. The bladder tear was repaired in 2 layers with absorbable (Vicryl) size 2/0 and haemostasis achieved. However, on doing vulvovaginal toilet, bloody urine was noted draining per the urethral catheter. This was expected and the catheter was left in situ for 14days. The reversal of general anesthesia was fairly smooth.

Post operative care

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

haematocrit was 23%. Appraisal on the outcome of surgery and counseling was provided.

The patient did very well. The breasts remained unengorged, the chest was clear and exposure of the wound on the 3rd postoperative day revealed a well-healed dry wound. Blood was taken for haemogram and plenty of oral fluids advised. However on the 5th postoperative day she developed fever associated with chills, headache and general malaise. Her breasts were soft and non-tender, the chest was clear, the wound was clean and dry and the abdomen was soft and non-tender. Her scanty lochia was non-foul smelling, the catheter urine for culture and sensitivity and blood smear of deep venous thrombosis. A repeat haemogram, urine for culture and sensitivity and a blood smear for malaria parasites were ordered. The combined results were as follows:

13/7/04: a) Haematocrit: 23%

b) RFTSs: Normal

16/7/04: Full Haemogram: Hb: 7.5g/dl, WBC: $8.2x10^9$/L

18/7/04: Full Haemogram: Hb: 8.0g/dl, WBC: $6.2x10^9$/L

B.S for malaria parasites: negative

Urine: a) Urinalysis: No abnormality

b) Culture and sensitivity: No growth obtained.

The interrupted stitches were removed on the 8th post operative day. Her urine cleared. No incontinence occurred. On the 9th postoperative day the vesico-vaginal team who removed the foleys catheter reviewed her. She had normal voiding thereafter and was subsequently discharged home on the 20th postoperative day through the postnatal clinic.

Follow-up

M.K was reviewed two weeks following discharge in the postnatal clinic. She was in good general condition, not pale and systematic examination revealed no abnormality. She was advised that she still had a chance of developing cervical cancer since only subtotal hysterectomy was done. A request for Pap smear was given to her and was to have the smear taken in six weeks post surgery. Because of massive transfusion, she was advised on testing for HIV, Hepatitis B and C viruses in three months time. She was also advised to seek surgical evaluation of the goiter and that the IUCD is not contraindicated in patients with asymptomatic goiter or even those with hyperthyroidism unless they have dysfunctional uterine bleeding secondary to the disease.

DISCUSSION.

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

One of the most dreaded obstetric catastrophes is uterine rupture because it is associated with high incidence of maternal and foetal morbidity and mortality rates and reflects poor obstetric care. The incidence of uterine rapture varies from region to region and from institution to institution. The incidence in Pumwani Maternity Hospital, Nairobi as found by Wanyonyi in the period 1996 to2001 was 1:219 deliveries.[1] This compared to the

incidences of 1:192 by Webala in his 1999 study at Kenyatta Nation Hospital.[2] These local incidences are quite high compared to the incidence of 1 in 1280 deliveries in a Western Institution in 1950 and 1 in 18500 in another one in 1994.[3,4] Uterine rupture is classified into complete (rupture communicates directly with peritoneal cavity) or incomplete (rupture separated from it by the visceral peritoneum). It should be distinguished from dehiscence of uterine scar which is partial separation of the scar with intact overlying peritonium, unextruded fetus and no or minimal bleeding.[4] Our patient's uterine rupture was complete.

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

In developing countries such as Kenya, however studies have shown that socio-economic factors such as inadequate means of transport facilities for operative delivery, qualified and motivated personnel and maternal ignorance play a significant role in putting the parturient at risk of uterine rupture.[2, 5] Our patient suffered the tragedy of having multiple risks. She went into labor at night with 3 previous c/s scars, 40 kilometers away from her preferred hospital of delivery to which she arrived 5 hours later only to find the only theatre occupied!

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

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

Treatment depends on the degree of rupture. Non-bleeding small rents as occurs after some normal vaginal deliveries after successful trial of scar are best managed conservatively. However, complete rupture requires emergent laparotomy preceded by intensive resuscitative measures such as correction

of hypovalaemia with colloids and blood transfusion. Operative management depends on the state, age and family size of the patient; they type, and site and extend of the rupture and the surgeon skills. At laparotomy three surgical operations are available; repair of the rupture only, repair and bilateral tubal ligation and hysterectomy.

Either subtotal (mostly) or total (rarely) hysterectomy is preferred for complete extensive ruptures. Subtotal hysterectomy is preferred to total because it is faster and safer. Total hysterectomy is reserved for extensive tear involving the cervix and vagina and then a highly skilled surgeon should perform the surgery. Lateral rupture involving the uterine artery with attendant broad ligament haematoma renders the ipsilateral ureter at risk of ligation or other injury. Excessive haemorrhage will, therefore require ligation of hypogastric artery to clear the operating field of blood.[6] Other pelvic organs must be inspected for possible injury, as was the case in our patient who underwent subtotal hysterectomy and bladder repair. Possible complications of uterine rupture include haemorrhage with resultant hypovolaemic shock and possible prerenal failure, postoperative sepsis, ureteral injury, thrombophlebitis, amniotic fluid embolism, disseminated intravascular coagulopathy, pituitary failure (Sheehan syndrome) infertility/ sterility and death.

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

REFERENCES

1. Wanyonyi C.H. W.Review of the management of uterine rupture in Pumwani Maternity Hospital, 1996-2001 M.Med. Thesis, 2003 University of Nairobi.

2. Webala G.S.R. The management uterine rupture in Kenyatta National Hospital. M.Med Thesis, 1979, University of Nairobi.

3. Eden R etal. Rupture of the pregnant uterus: a 53-year review. Obstretic and Gynaecology 68:678,1986.

4. Cunningham F.G. etal. Obstretrical haemorrhage. In Williams Obstetrics, 21st edition. Lippincot Williams and Wilkins; 1997:66-652.

5. National council for population and development. The Kenya demographic health survey, 1998-97-110.

Labour Induction at Kenyatta National Hospital

ABBREVIATIONS

CPD	:	Cephalo-pelvic-disproportion
CI	:	Confidence Index
EPNMR	:	Early Perinatal Mortality
KNH	:	Kenyatta National Hospital
NBU	:	New Born Unit
KNH/UON-ERC	:	Kenyatta National Hospital/ University Of Nairobi Ethics and Research Committee
WHO	:	World Health Organisation
IUFD	:	Intrauterine foetal death
PG	:	Prostaglandin
RR	:	Relative Risks
SVD	:	Spontaneous Vaginal Delivery
APH	:	Antepartum Haemorrhage

OBSTETRIC LONG COMMENTARY

TITLE: OUTCOME OF LABOUR INDUCTIONS AT KENYATTA NATIONAL HOSPITAL.

SUMMARY

Objective – The aim of the study was to determine the outcome of labour inductions at Kenyatta National Hospital with regard to the socio-demographic characteristics of the clients induced, indications for the inductions, different methods used, and their outcomes, at various gestation periods.

Study Design: - This was a prospective descriptive study.

Setting: - Kenyatta National Hospital – Nairobi , this is a referral and teachinh hospital which also serves as the population within and around the city. It provides curative, preventive and rehabilitation services in all medical specialities.

Study Population: - A total of 185 expectant mothers who were admitted in labour ward in Kenyatta National Hospital between 1st June 2008 to 30th March 2009 and underwent labour induction within the study duration and who fulfilled the study criteria.

Study Period: - The study was done between June 1st 2008 to March 30th 2009. This being the period when the data was collected.

Recruitment: -
Women admitted at KNH for induction of labour during the study period were recruited to the required population number of 185 by the principle investigator. Recruitment was done from Monday to Sunday on a 24-hour basis. In the absence of the principle investigator, two research assistants, who had earlier been trained by the principal investigator and were acquainted with the questionare, were available in labour ward to recruit participants into the study. The study objectives were explained to the clients and formal consent was obtained.

Inclusion Criteria:-

- Women who were admitted in labour ward and had induction of labour as a management plan
- All mothers admitted for other reasons but for whom induction of labour was agreed upon as part of the management plan in the ward.

Exclusion Criteria:-

- Any mother who was unwilling to participate in the study
- Any mother who went into spontaneous labour
- Any pregnant lady with diagnosis for which induction of labour was contraindicated.

Main outcome measures: -

The main outcome was success rate for each method employed and was defined as spontaneous vaginal delivery and labour duration of less than 12 hours for both preterm inductions and term inductions.

Secondary outcomes were failure rates for example caeserian section as a result of failure of induction of labour.
Others were duration of labour lasting more than twelve hours, post partum haemorrage and vacuum exraction as a method of delivery.

Results: -

A majority of the study participants (40.5%) were between the age of 26-30 years, with over 98% aged above 20 years. Most of them (63.2%)were married. Over 95% had atleast primary education, with over 65% having secondary and post-secondary education. Over 50% were in gainful employment.
Use of Prostaglandins was the most frequent method employed (45.4%), followed by use of oxytocin alone (23.2%). The use of amniotomy only was the least employed at (6.6%).

The leading indication for labour induction, was premature rapture of membranes (36.8%), followed by hypertensive diseases in pregnancy- pre-eclapmsia/ eclapmsia at 35.6% .

The most common gestation of mothers at induction was 37-40 (37.2%).

Over 70% of the induced clients were of gestations of 34 weeks and above.

A majority of the clients delivered within 12 hours (80.9%), while (19.1%) had prolonged labour.

For all labour induction methods combined, 71.3% had spontaneous vaginal deliveries, 2.2% had assisted vacuum deliveries and 26.5% emergency caeserian sections.

Post partum haemorrage occurred in 13.2 %.

Analysis: - Was done using EPI- INFO computer package. Data from fully filled questionnaires were entered into the computer for analysis.

Conclusions / Recommendations: -

Labour induction is a safe obstetric procedure if done at the right gestation for the right indication and with the right method.It often results in favourable method of delivery, thus spontaneous vaginal delivery, and takes appropriate duration of labour, with minimal blood loss after the delivery.Good outcomes of labour inductions are achieved when combination of methods are used e.g. prostaglandins, and later amniotomy and use of oxytocin

DEFINITIONS

Labour

True labour is defined as a coordinated effective sequence of involuntary uterine contractions resulting in descent of the presenting part, cervical effacement and dilatation culminating in voluntary bearing down leading to delivery of the foetus and the placenta.
For the purpose of this study the term "labour" was used to denote true labour. False labour is characterized by irregular brief uterine contractions evoking back or abdominal pains without effacement and dilation of the cervix. The contractions of false labour are inconsistent in duration and strength and cause no change in the cervix.

Parturient: A patient in labour

Nulliparous: has not delivered an offspring weighing 500g or more or of 24 weeks duration or more

Primiparous: given birth to more than once fetuses weighing 500g or more or of 24weeks gestation or more

Gravidity: The total number of pregnancies, including abortions, hydatidiform moles, ectopic pregnancies and normal intrauterine pregnancies

Nulligravida: has never been pregnant

Primigravida: currently pregnant for the first time

Multigravida: Currently pregnant and has been pregnant once or more before.

Post datism: pregnancy gestation of more than 40 completed weeks (for this study)

PPH: post partum haemorrage, which means excessive loss of blood per vagina after delivery of the placenta. For this study, it means any estimated loss of more than 500 mls of blood.

Amniotomy: artificial rupture of the amniotic membrane with subsequent sweeping of the lower uterine segment aimed at initiating or potentiating labour.

PROM: premature rupture of membranes for more than 12 hours before the onset of labour. It's considered as pre-term prom if the gestation is below 36+ weeks and term-prom if more.

Prostaglandins: pharmacological agent for ripening cervix and subsequently inducing labour, commonly used types are: PGE1-misoprostol, PGE2: dinoprostone, PGF2∝. For this study, the word prostaglandin will refer to any of the three.

INTRODUCTION AND LITERATURE REVIEW

Despite decades of research, the events leading to the initiation of labour in humans remain unclear. It is suspected that biochemical substances produced by the fetus induce labour. In addition, the timing of the production of these substances and their interaction with placental and maternal biochemical factors appear to influence this process. Among the most studied of these biochemical substances are fetal hormones such as oxytocin and placental inflammatory molecules[1]. Increased placental and maternal production of inflammatory molecules in late pregnancy has been strongly linked to the initiation of labour. Hormone like substances called prostaglandins, which are produced by the placenta in response to various biochemical signals, can induce inflammation and are present in increased levels during labour. Several factors that increase the production of prostaglandins include oxytocin, which stimulates the force and frequency of uterine contractions, and a fetal lung protein called surfactant protein A (SP-A). Surfactant production in the fetal lung does not begin until the last stages of gestation, when the fetus prepares for air breathing; this transition may act as an important labour switch[2].

The withdrawal of progesterone is known to precipitatelabour and it has been suggested that sudden involution of the corpus luteum of pregnancy might cause a sudden fall in the blood progesterone level, but *Short and Eton*[3]' were unable to demonstrate any correlation between the blood progesterone levels and the onset of labour. Oxytocin has little action on the non-pregnant uterus, and only acts on uterine muscle that has been subjected to the action of progesterone. As with progesterone levels, there is no evidence of any abrupt change in blood oxytocin levelsimmediately prior to the onset of labour. Work by Riad and Scandrett' on the levels of oxytocinase similarly failed to reveal any marked change prior to the onset of labour[4]. The optimum pH for the activity of oxytocinase is 7.3,' and interestingly enough the pH of the amniotic fluid is of this magnitude for the greater part of pregnancy,'only falling to a level of pH 7.0 or less at 39

[1] 31-32
[2] 31-32

[4] 19,21,31

weeks' gestation. At a level of pH 7.0 or less the activity of oxytocinase is markedly reduced,' and therefore the possibility exists that although there is no marked change in the level of oxytocinase in the amniotic fluid as term is approached, there is a reduction in the biological activity of this enzyme due to the fall in pH. The oxytocinase in the amniotic fluid may be presumed to inactivate the oxytocin that reaches the amniotic fluid and thus prevent it from diffusing through the decidua to reach the myometrium[5]. The amniotic acidosis occurring as term is approached is brought about by a foetal metabolic acidosis caused by increased activity on the part of the foetus.' This amniotic acidosis, by causing a reduction in the activity of oxytocinase, may result in an increase in the amount of oxytocin in the amniotic fluid, and this in turn might result in an increased diffusion of oxytocin across the decidua to the myometrium, thus giving rise to contraction of the uterine muscle. It is therefore suggested that increasing foetal activity as term is approached may result in acidotic inhibition of oxytocinase in the amniotic fluid[6] with a consequent rise in the amount of oxytocin in the fluid. Because of this rise diffusion of oxytocin across the decidua may occur and myometrial stimulation follow. The concept of a pH change in the amniotic fluid being responsiblefor the onset of labour would not be incompatible with the observation of Bengtsson and Csapo,' that a solution of hypertonic saline administered into the amniotic fluid can produce termination of pregnancy. The diffusion of oxytocin across the decidua might not be in uniform amounts over the whole of its surface, and this would explain the existence of " functional asymmetry " of the uterus observed by Csapo.' The fact that women with diabetes insipidus go into labour normally has been advanced as an argument against oxytocin having an important part to play in the onset of labour. However, polypeptides
other than oxytocin produced at sites elsewhere than the posterior pituitary might be of importance in these cases, and these " alternatives " to oxytocin may well be destroyed by oxytocinase in the same manner. The fact that labour not infrequently ensues after the intrauterine death of a foetus does not invalidate the hypothesis, as in this instance there will also be a pH change[7].

The exact mechanism, which triggers the onset of labour is not fully understood, however, research involving animal models and humans has

---

[5] 32
[6] 32
[7] 31,32

shown that it is the fetus which initiates a train of physiological interactions which initiate labour. However, sometimes pregnancy is prolonged and induction of labour required. This study examines the methods utilized in the induction of labour. Knowledge of physiology is important as the midwife not only provides information and care for women regarding the onset of spontaneous labour, but also when induction of labour is required[8].

Induction of labour is the process of initiating labour pains by artificial means. Around 20% of all deliveries are preceded with labour induction, a proportion which has not varied dramatically over recent years [1].

Fetal death, prolonged pregnancy and maternal risks e.g. maternal hypertensive disorders have taken center stage as the major indication for the last 50-60 years [2, 3].

Techniques for inducing labour have changed from dietary delicacies, verbal threats and enema (emptying of the rectum) giving way to physical stimulation, mainly achieved by cervical stretching, amniotomy and more recently sophisticated pharmacological manipulation using oxytoxin and prostaglandin. Relaxin, antiprogestines, nitric oxides have been explored in the recent years[22].

Successful induction is however, still not guaranteed and there has been increasing emphasis during the last decade on exploring strategies for identifying the probability of success. Measurement of fetal fibronectin in the cervical mucus, maternal serum nitrite/nitrate concentrations, ultrasound deneation of the cervical form and electrical impedance measurements across the cervix are all being investigated.

Safety, success and patient satisfaction continue to be major objectives with economic evaluation now becoming a significant factor in the search for the ideal induction method. The need for a healthy delivery has been recognized and practiced for centuries; the indications have clearly changed during the past 20 years from the need to expel a dead fetus to pre-emptying action, to reduce the threat to fetal or maternal health. Effective and safe methods of achieving delivery must have always been the primary objective to achieve.

---

[1] 8.33

Most methods of inducing labour before the last half of the century which were invented with precedent manipulations involve; Calvinism, repeated pressurized douches, extra-amniotic aqua picea, tents, bougies and catheters. Lately, castor oil, quinine and posterior pituitary extract are also utilized. Among the more common old approaches are frequent walking, vaginal intercourse, participating in heavy exercises, consumption of laxatives, spicy foods or herbal tea, nipple stimulation and administration of enema. During the 1980s and 1990s, patients acceptance, when and how delivery was achieved became significant consideration[19.26.27].

The cervix is essential in maintaining uterine stability during pregnancy. To achieve this, the maintenance of cervical shape and consistency is imperative since cervical ripening is a physiological process occurring throughout the later weeks of the pregnancy and is complete with onset of labour. When delivery is necessary and ripening has not had time to occur or has failed to be initiated, there is need for the natural process to be accelerated. With the advance of pregnancy, increased vascularity is seen and the fibroblasts become secretory, white cells and macrophages migrate out of vessel walls into cervical stroma with an increase in water content. Enzymatic breakdown of collagen fibrils by collagenases/ matrix metallo-proteinase produces fibroblasts and polymorph nuclear leukocytes alongside leukocyte elastase, which catabolises elastin, leads to increase cervical compliance. Cytokines notably IL-8 or platelets activating factor (PAF) and monocyte chemotactic protein-1 (MCP) have been proposed as possible interactants in the remodeling process involved in cervical ripening as has nitric oxides synthesized by macrophages myometrium and the cervix[8.11.29 8]

Indication of labour induction:

This can be divided into maternal, fetal, social or a combination of these and may be also as anticipated. Specific definitions and the relative importance of the various indications of labour inductions vary between obstetricians, obstetric unit and country [4. 5]. As an example, post term, post dates or prolonged pregnancy is probably the commonest indication in many units but definitions may include any gestation beyond 40, 41, 0r 42 completed weeks of gestation. Some obstetricians consider that cervical state should

19.26.27
8.11.29

determine the timing of delivery, particularly when post date is the indication for labour induction [6].
It must be remembered, however that there is often a poor relationship between cervical favuorability and gestational age. It has been difficult to compare rates between obstetric units, states, because of differences in definitions for induced or augmented labour [7].

e.g:

- An oxytocin given following spontaneous rupture of membranes without labour, is variously called labour induction
- Labour that follows prostaglandins given to ripen the cervix is variously called spontaneous or induced labour
- Attempts to ripen the cervix that fails is called failed induction of labour

Generally, doctors may suggest an induction of labour for any of the following

- Post dates (which varies, most doctors will recommend an induction of labour at around 7-14days past the patient's due date according to their own options and/or beliefs)[2]
- Where continuing the pregnancy poses a threat to the mothers health (mental or physical)
- Pre-eclampsia/Hypertension/Eclampsia
- Diabetes
- Rhesus incompatibility
- Fetal and/or maternal compromise (Poor biophysical profile)
- IUGR (intra-uterine growth restriction)
- Fetal abnormality
- Chronic renal disease
- Polyhydramnios
- Abnormal liver function tests
- Blood dyscrasias
- Previous stillbirth
- Fetal death in utero
- Poor past obstetric history (complications and/or lost or damaged babies)
- Membranes rapture but no labour after 12hours, (premature rupture of membranes at term)

- Where continuing the pregnancy is detrimental to the baby i.e. is not growing or is unwell & would be safe out than in
- Large for dates infant
- Placental insufficiency[8.10.13]

Cases where an induction of labour is not clinically advisable

- Abnormal lie (e.g. transverse)
- Fetal distress
- Placenta previa
- Cord prolapse
- Vasa previa
- Cephalopelvic Disproportion[2,4,19]

In recent analysis, Kirby 2004 reviewed data on induction of labour in the USA from 1990-2002 and found a marked discrepancy between the rates quoted by the national center for health statistics and the persistent 3% level rates quoted by birth certification Analysis. Most notable however, was the increase in inductions from around 10-15% of deliveries in 1990 to around 17-21% by 2002. During the early part of this period, the National rate for cesarean section in the USA was relatively static around 21-22%, followed by a sudden escalation to 26% in 2002.This was thought to be related to a gathering momentum against encouraging labor in women previously delivered by caesarean section [9] and that failed induction leads ultimately to caesarean section [9, 10]

Methods of Labour induction

It is important to prescribe different methods of labour induction when the cervix is unripe and unfavourable and those used when the cervix is favorable. In reality, more than 12 different pelvic scoring schemes have been described during the past 70 years, but semi-quantitative cervical scoring systems described by Bishops (1964) is the most widely commonly referred to as Bishop Score[7,12].

8.10.13
2.4.19
7.12.

Table A. Bishops scores:

	0	1	2
Dilation of cervix	<1	1-2	>2
Length of cervix	2cm	1cm	Effaced
Consistency of cervix	Firm	Soft	Soft/Stretchy
Position of cervix	Posterior	Mid	Anterior
Station of presenting part	-4 to –3	-2 to –1	0 to +1

Score 8-10 – Favourable cervix
Score 0-7 --- Unfavourable cervix[13].

Current methods of inducing labour:

Two approaches are used, often in combination; one relies upon pharmacological agents to modify cervical form with or without stimulating uterine contractions and the other uses mechanical stimulation to provoke cervical effacement, dilation and ultimately uterine contractions [14,15].

Prostaglandins

By mid 1980s, the prostaglandins had become established as the most effective pharmacological agent for inducing labour when the cervix is unripe. Variety of administration routes had been explored which include oral, intravenous, sublingual, rectal, intra-amniotic, extra-amniotic, intra-cervix, endocervical and vaginal. The vaginal route was found to be the most acceptable, providing good efficiency and acceptability for the parturient and is now the preferred choice. PGE come in two forms:

- PGE_1 _ misoprostol: synthetic vaginal analog insert found to be safe and in expensive
- PGE_2 – dinoprostone: a vaginal insert that increases the activity of collagenase in the cervix which facilitates dilation

Prostaglandins act on the cervix to enable ripening by a number of different mechanisms. They alter the extra cellular ground substance of the cervix, and PGE_2 increases the activity of collagenase in the cervix. They cause an increase in elastase, gylcosaminoglycan, dermatan sulfate, and hyaluronic

acid levels in the cervix. A relaxation of cervical smooth muscle facilitates dilation. Finally, prostaglandins allow for an increase in intracellular calcium levels, causing contraction of myometrial muscle. Prostaglandin E_2 (PGE_2) and prostaglandin F_{2a} (PGF_{2a}) are synthesized in the endometrium and myometrium to cause contractions of the uterus. Administration of PGE_2 or PGF_{2a} by various routes induces labour or abortion at any stage of gestation. Risks associated with the use of prostaglandins include uterine hyper stimulation and maternal side effects such as nausea, vomiting, diarrhea and fever[16].

Oxytoxin

Since Du vigneaud synthesized oxytocin from the nona peptide oxytoxin in the 1950s, it has been used by intravenous infusions for the majority of women having their labour induced. Although used as the primary induction agent occasionally, it is more frequently given to assist the induction process using prostaglandins when the cervix is unfavourable or as an adjunct to low amniotomy in more favorable cases. Originally given as a constant dose, infusion at less than 10mU/min has been replaced by titrated doses, determined by intensity and frequency of uterine contractions[17, 18, 19, 20].

Membrane sweeping and amniotomy

The amniotic membrane is ruptured by an amnion or corker to drain out some of the amniotic fluid and the cervical membranes sweeping. This may be enough to start labour on its own, but usually syntocinone intravenous infusion maybe required. Sweeping performed during vaginal examination has been shown to result in established labour in 70% of cases if repeated daily over 30days. Striping of membranes causes an increase in the activity of phospholipase A_2 and prostaglandin F_{2a} 9 PGF_{2a} as well as causing mechanical dilation of the cervix that releases prostaglandins. By inserting the examination finger through the internal cervical os and moving it in a circular direction to detach the inferior pole of the membranes from the lower uterine segment. Amniotomy increases the production of prostaglandins locally but are all with associated risks of infection, bleeding, accidental rupture of membranes and patient discomfort. One must then ensure there are no contraindications to rupture of membranes such as active herpes, HIV-which would increase chances of maternal to child

16

transmission, vasa previa, funic presentation, IUFD or any obvious reason that would necessitate caesarean delivery [21].

Hygroscopic dilators

They absorb endocervical and local tissue fluids causing the device to expand within the endocervix and providing controlled mechanical pressure. The products available include dilators (e.g. laminaria japonicum) and synthetic dilators e.g. lamicel) [22].

A Foley catheter or specifically designed balloon device can be used. Using the balloon device will exert mechanical pressure directly in the cervix as the balloon is filled with air causing release of prostaglandin locally on mechanically causing dilatation of the cervix.

Recently explored labour induction methods

- **Antiprogestins**

Epostane, the 3B-hydroxy dehydrogenase inhibitor, and subsequently mifepristone, the progesterone receptor blocker, were shown to have a dramatic effect upon reducing induction to abortion interval during second trimester therapeutic abortion, hence remains a possibility that this drug could be used to improve the outcome of labour induction in future [23].

- **Oestrogens**

With ever increasing concentrations of oestrogen in the maternal circulations leading to term pregnancy, the belief that this could be a trigger for the onset of spontaneous labour led to studies exploring oestrogens for the induction of labour. Oestradiol gel given extra-amniotically, endocervically or vaginally must produce some improved cervical favorability with minimal myometrial stimulation [24].

- **Dehydroepianndrsterone sulphate (DHEAS)**

DHEAS, which is transformed into oestrogens in the fetoplacental unit, has been explored as a possible cervical ripening agent, achieving effacement without inducing uterine contractions [25].

- **Relaxin**

This polypeptide has been explored in humans using purified porcine relaxin 1-4mg in a viscous gel vaginally or endocervically to ripen the unfavourable cervix but unfortunately there appear to be little evidence of any therapeutic effect [26].

OUTCOME OF LABOUR INDUCTION

Induction of labour is increasing in the USA from 9.5% in 1990 of to 22% of births in 2004.whether induction of labour improves outcomes or simply leads to greater complications and health care costs has been a common debate.compared to waiting for labour to occur,labour induction after 41weeks is associated with reduced perinatal deaths and helps in reducing the needs for further and additional monitoring of women and decrease of hospitalization especially in seetings where women need to be hospitalized early to avoid situations in which they are unable to reach hospitals in an emergency.(17)Despite the would be advantage induction of labour also has less preferred outcomes like,in the event of failure induction of labour ultimately leads to ceaserean section.It is debatable whether increase in labour induction ultimately results in increase in number of ceaserean sections.(14).

This study will appraise the indications for labour induction and the frequency with which it is currently used in KNH.

STUDY HYPOTHESIS

Can the use of various methods of inductions, of labour, as practised in KNH result in acceptable promotion of maternal reproductive health?

STUDY RATIONALE

Labour induction is indicated mainly when it is probable that the mother or foetus will benefit from earlier onset of labour than if birth is delayed, thus any intervention to initiate the same is meant to achieve this probability advantage.

Induction of labor is common in obstetric practice. According to the most current studies, the rate varies from 9.5 to 33.7 percent of all pregnancies annually. In the absence of a ripe or favorable cervix, a successful vaginal birth is less likely. Therefore, cervical ripening or preparedness for induction should be assessed before a regimen is selected. Assessment is accomplished by calculating a Bishop score. When the Bishop score is less than 6, it is recommended that a cervical ripening agent be used before labor induction. Nonpharmacologic approaches to cervical ripening and labor induction have included herbal compounds, castor oil, hot baths, enemas, sexual intercourse, breast stimulation, acupuncture, acupressure, transcutaneous

nerve stimulation, and mechanical and surgical modalities. Of these nonpharmacologic methods, only the mechanical and surgical methods have proven efficacy for cervical ripening or induction of labor. Pharmacologic agents available for cervical ripening and labor induction include prostaglandins, misoprostol, mifepristone, and relaxin. When the Bishop score is favorable, the preferred pharmacologic agent is oxytocin, (17).

Some literatures are quoting an induction rate of as high as 40% in the obstetric units denoting the fact that this is a common obstetric procedure, whose benefits and demerits should well be understood. It is worthwhile to note that when induction fails, the next option is still caeserian section. An option, which stood if induction was omitted. Hence no clinical loss, but rather again due to the high costs, high morbidity and mortality of caeserian sections, hence, will reduce the social and financial strain, not only to the individual, but to the community and healthcare facilities at large.

The study was to reveal the characteristics of clients most likely to be induced, reasons for their induction and the possible outcomes. When success is defined as spontenous vagianl delivery, the success of induction would mean a cheaper mode of delivery in women and a convenient method for health care systems than other methods for ensuring earlier delivery like caeserian section.

The various methods of induction at KNH were to be audited to establish the most effective in terms of vaginal delivery and of least maternal complications with regard to post partum haemorrage.

The timing of the gestation period would reveal the clinicians reasons for intervention like chances of foetal survival, in KNH set-up.

The study will establish the most likely mode of delivery and duration of labour after induction.

Since labour induction enhanced the propbability of vaginal spontenous delivery in our study, this study forms a foundation for further studies, where the labour induction would reduce the incidences of emergency caeserian sections in our set-up.

STUDY OBJECTIVES

Broad objective

To evaluate the procedures and outcome of labour induction in Kenyatta National Hospital.

Specific objectives

1. To determine gestation at labour induction.
2. To determine indications of labour induction.
3. To determine the methods used for labour induction.
4. To determine the outcomes of labour induction.
5. To determine the relation between labour induction and outcome.

STUDY METHODOLOGY

Study Design

This was a prospective descriptive study in which patients admitted at KNH who required induction of labour were recruited and the outcome of the inductions analysed. The patients who presented and had induction of labour in the management plan as opted for by clinicians were recruited and those who met the study criteria and consented were recruited.Those recruited had a questioner filled and completed as labour progressed.

Study location

The study was carried out at the Kenyatta National Hospital, in Nairobi city, Kenya. It serves the population within and around the city and receives patients from other countries in East and Central Africa for specialized treatment and care. It has a bed capacity of 1500. It also serves as the university teaching Hospital for the college of health science of the University of Nairobi. Several medical specialities and sub specialities are catered for by the hospital and the department of obstetrics and gynecology is one of them. The department is divided into 3 firms headed by consultants. The labour ward is just one of the units and serves as an entry point to all obstetric cases of patients. Before being filtered and undersigned to various procedures and specific wards. Patient who require induction

belong to this unit and are either admitted from the clinics for the procedure as a management plan or are identified in the labour ward and assigned labour induction as a management plan. On admissionm the patients undergo an admission process, which includes opening of files. This encompasses listing of their socio-demographic characteristics prior to being admitted. The patients are then evaluated by the nursing staff and then reviewed and clinically evaluated by the senior house officer or registrar, who stipulates the management plan. Those who require induction, are then started on the various different methods of induction as the registrar deems fit and are either managed in labour ward or taken to the general wards to be reviewed later and ease congestion. But the ultimate ward is the labour ward where delivery occurs.

Study period

The study period was 1st JUNE 2008 to 30th MARCH. 2009.This being the period during which the data was collected..

Study participants

This involved women who were admitted in labor ward while not in labour and in whom vaginal delivery was felt to be the appropriate route of delivery and whom to initiate labour, decision was made to induce them. Presumably for the well being of both the baby and mother and where there was foetal demise, for the well being of the mother.

Inclusion Criteria

- Women who are admitted in labour ward and have induction of labour as management plan
- Women who are admitted for other reasons but for whom induction of labor was agreed upon as part of the management plan in the ward.
- Only women who qualified for the exercise and consented to participate were recruited and filled the questionare.

Exclusion Criteria: -

- Any woman who was unwilling to participate in the study
- Any woman who went into spontaneous labor
- Any woman in labor ward with diagnosis for which induction of labor was contraindicated.

Sample size

A sample size to effect data collection by random sampling was obtained by the formula below:-

$$N = \frac{Z^2 \times p\,(1-p)}{}$$

C^2

N= Sample size

Z^2= number of standard errors away from the mean

P= prevalence of labour induction (P being the prevalence of labour induction in KNH as determined in early studies,Khisa MMed thesis 1999, p=14%)

C^2= confidence interval 90.05

(For confidence level of 95%,) Za=1.96

$$= \frac{1.96^2 \times 0.14\ (1-0.14)}{0.05^2}$$

$$= 185.01$$

= 185 Cases

Sampling Method

A systematic random sampling technique was employed. The sampling frame was that of all mothers eligible for the study. Every second mother who met the inclusion criteria was identified and recruited for the study to ensure random sampling.

Data collection materials

Only those who qualified for the exercise & consented to participate were recruited.The data was collected and filled in an already availed questionnaire and consent form by the already trained staff.

Data collection and Study tool

The principal Investigator identified eligible clients with the assistance of the labour ward staff on a systematic random basis which involved all the legible even number clients received in labour ward. Their suitability was reassessed and those who consent to participate in the study were followed up. All the information about the clients was entered in the client record form where only serial numbers were used. The labour ward clinical team on duty decided upon the desired mode of induction of labour. The principle investigator to fill the questionnaire and interview the patients perused patient records from time to time. Postnatal interviews were done when the mothers were fairly stable. All complete questionnaires were kept safely by the principal investigator waiting for data analysis at the completion of the entire sample size of the study.

Data quality control

It was insisted that only those who qualified and consent were given a chance to fill the questionnaires and without influence from anybody. All questions were answered to make it easier to analyze data.Only valid and completed questionnaires which were all numerically numbered were analysed.

Study limitations

- ❖ Some clients did not afford the chosen mode of induction of labour and had to wait for the second option from the clinical team.
- ❖ Most of the outcome e.g blood loss is a quantitative estimated analysis and its likely to have errors.
- ❖ Most of the socio-demographic were verbally received and not subject to any verification.. e.g age
- ❖ Some clients had more than one indication for induction e.g. PROM at term with pre-eclampsia and so difficult to tabulate the indication for induction. In such a case, the more fatal condition was picked as the reason for induction.

DATA ANALYSIS

The data collected was entered into the computer and the analysis done using the EPI-INFO package.

Ethical considerations

Before the study commenced, approval from the Kenyatta National Hospital Research and Ethics Committee was sought. Mothers participating in the study were explained to and those who consented were interviewed and questionnaires filled those who declined, were managed to their full satisfaction and not included in the study. Information collected from the clients was treated with utmost confidentiality. No names were written anywhere on the questionnaires which were serialy numbered and the information later used for analysis.Prior permission had been sought from the staff and management of labour ward to allow for this study.

17.1 RESULTS: A total of 185 study participants were recruited into the study over this period , and a total of 2562 deliveries were recorded over the same period.

17.2 The results are: -Table 1. The Socio-demographic characteristics of the study population (*n=185*)

CHARACTERISTICS (%)	NUMBER	PERCENTAGE
Age (Years)		
< 20	2	1.1
20-25	28	15.1
26-30	75	40.5
31-35	63	34.1
36-40	17	9.2
Marital Status		
Single	57	30.8
Married	117	63.2
Divorced/Separated	5	2.7
Widowed	6	3.3
Parity		
0-1	69	37.3
2-3	63	34.1
4-5	31	16.7
over 5	22	11.9
Level of Education		
None	9	4.9
Primary	50	27.0
Secondary	86	46.5
Post Secondary	40	21.6
Religion		
Catholic	82	44.3
Protestant	93	50.3
Muslim	7	3.8
Other	3	1.6
Occupation		
Employed	78	42.2
Self Employed	90	48.6
Unemployed	17	9.2

Table 1 shows that modal age group was 26-30 years and 90% of the clients were within 20-35 years with 1.1% below 20 years and 9.2 above 36 years. About Two thirds were married. Most (71.4%) were para 3 or less.Majority (68.1%) had atleast secondary education.About 91% were in gainful employment, and most of them were Christians.

Table 2
Gestation at labour induction (*n=185*)

Gestation (in weeks)	Number	Percentage (%)
<28	34	18.4
28-33^{+}	20	10.8
34-36^{+}	48	26.0
37-40^{+}	69	37.2
≥ 41	14	7.6
Total	185	100%

The most common gestations at labour induction were at 37-40 weeks (37.2%), followed by moderate preterm labour induction (26.0%). Severe preterm labour induction constituted 10.8%, but in overview, preterm labour induction (less than 37 weeks gestation) was more common, constituting 55.2%. Post-term labour (more then 40 weeks) constituted only 7.6 %.(Table 2)

Table 3

Primary indications for labour induction (n=185)

Indication	Number	Percentage (%)
Post dates (≥ 41 weeks)	14	7.6
Premature rupture of membranes	68	36.8
Intra uterine fetal death	23	12.4
Pre-eclampsia/Eclampsia	66	35.6
Other Medical diseases in pregna	3	1.6
Bad obstetric history	9	4.9
Others	2	1.1
Total	185	100%

As shown in table 3, the two leading primary indications for labour inductions were premature rupture of membranes (36.8%), followed by hypertensive diseases in pregnancies (pre-eclampsia/eclampsia) at 35.6%. Intra uterine fetal death constituted 12.4%.

Table 4. Methods used in Labor Induction

Method (%)	Number	Percentage
Prostaglandin +amniotomy +oxytocin	84	45.4
oxytocin only	43	23.2
Amniotomy +oxytocin	28	15.1
Prostaglandin only	18	9.7
Amniotomy only	12	6.6
Total	185	100%

The most common used method for labour induction was the use of prostaglandin, and later amniotomy with subsequent augementation with oxytocin infusion (45.4%). This was followed by oxytocin (23.2%)

primarily due to rupture of membranes,and amniotomy and oxytocin (15.1%)Amniotomy alone was only used in 12 clients(6.6%.)(table 4)

Table 5. Mode of Delivery after labour induction (n=185)

Mode of Delivery	Number	Percentage (%)
Spontaneous vaginal delivery	132	71.3
Assisted Vacuum delivery	4	2.2
Emergency Cesarean delivery	49	26.5
Total	185	100%

Vaginal delivery was the most common outcome (71.3%.) Delivery by emergency caesarean section was in 26.5% instances while assisted vacuum delivery was required in 2.2% of cases. (Table 5)

Table 6. Duration of labour (n=136)

Duration	Number	Percentage (%)
< 12 hours	110	80.9
12-24 hours	19	14.0
>24 hours	7	5.1
Total	136	100%

Following labour inductions, 80.9% of the total clients had spontaneous vaginal delivery within 12 hours.Of the total, 5.1% were in labour for over 24 hours. This could have been due to improper partogramming as to when the active labour set in, especially where induction was done with a very poor Bishops Score, or where amniotomy alone was used in a very favourable Bishops Score as seen in the clinical notes.
It also happened that 49 of the initial 185 clients had emergency cesarean section as interventions.

Table 7. Estimated blood loss after vaginal delivery (n=136)

Estimated blood loss (mls)	Number	Percentage (%)
< 500	118	86.8
>500	18	13.2
Total	136	100%

Estimated blood loss of more than or equal to 500mls was witnessed in 13.2% thus had post partum haemorrage.(Table 7)

Table 8: Vaginal delivery by labour induction

Methods of induction	Number (vaginal deliveries)	Percentage (%)
Prostaglandin + amniotomy + Oxytocin n= 84	78	92.8
Oxytocin only n=43	35	81.3
Prostaglandin only n=18	5	27.7
Amniotomy and Oxytocin n= 28	13	46.4
Amniotomy only n=12	5	41.6
Total n=185	136	73.5

Majority of those who were induced by prostaglandin and later amniotomy and oxytocin had SVD (92.8%), while oxytocin only had 81.3%.Use of amniotoy and oxytocin are 46.4% and amniotomy only had 41%. For all patienta induced 73.5% had SVD.(Table 8)

Table 9:Association between Vaginal Delivery and Method of Labour Induction (n=185)

od of Induction	N	Vaginal Delivery		% Success	OR (95% CI)	P-Va
		YES	NO			
aglandin niotomy +Oxytocin	(n=84)	78	6	92.8	-Ref	-
tocin only	(n=43)	35	8	81.3	4.2(1.2-14.8)	0.00
aglandin only	(n=18)	5	13	27.7	48.0(3.8-1343.7)	0.00
iotomy +Oxytocin	(n=28)	13	15	46.4	3.6(0.6-20.5)	0.11

iotomy only	(n=12)	5	7	41.6	48.0(3.8-1343.9)	0.001
AL	185	136	49	73.5	-	-

As shown in table 8, those who had prostaglandin and later amniotomy and oxytocin as amethod of induction, had the highest success rate at 92.8%

Use of oxytocin had 4.2 times less chances of having a viginal delivery, while prostanglatin only had 48 times less chances.

Table 10: Association of blood loss by method of inductioin (n=136)

of Induction		Blood Loss		OR (95%CI)	P-Value
		< 500	> 500ml		
ndin mniotomy n	(n=78)	73 (93.6%)	5 (6.4%)	Ref	-
only	(n=35)	34 (97.1%)	1 (2.9%)	0.4(0.0-4.1)	0.664
ndin only	(n=5)	2 (40%)	3 (60%)	21.9(2.2-261.9)	0.001
my +Oxytocin	(n=13)	7 (87.5%)	6 (12.5%)	12.5(25-66.5)	0.001
my only	(n=5)	2 (40%)	3 (60%)	21.9(2.2-261.9)	0.001
	136	**120**	**18**	-	-

With the use of prostaglandin, amniotomy and oxytocin combined as a reference, clients who used prostaglandin only had 21.9 times more chances of blood loss more than 500mls, while amniotomy and oxytocin had 12.5% . Those who used Oxytocin only, showed no significance correlation in blood loss. Of the 78, who used prostaglandin, amniotomy and Oxytocin, only 6.4% had likehood of PPH but prostaglandin alone and amniotomy alone had 60%. With the p valus shown, and assuming that third stage labor management was standard both in method and personnel, the table shows significant blood loss in the last three methods hence likelihood of PPH.

Table 11:Association between Duration of Labour and Method of Induction (n=136)

ethod of Induction	Duration (Hours)		OR (95% CI)	P-Value
	≤ 12	> 12		
staglandin + Amniotomy ytocin 78)	76 (97.4%)	6 (2.6%)	Ref	
tocin only (n=35)	26(74.3%)	9(25.7%)	4.2(1.2-14.8)	0.009
staglandin only (n=5)	1(20%)	4(80%)	48.0(3.8-1343.7)	0.001
niotomy + oxytocin (n=13)	10(76.9%)	3(23.1%)	3.6(0.6-20.5)	0.116
niotomy only (n=5)	1(20%)	4(80%)	48.0(3.8-1343.7)	0.001

From table 11,the use of acombination of prostaglandin and amniotomy and oxytocin had 94.4% likehood of labour lasting ≤ 12 hours. While amniotomy and oxytocin had 76.9% , oxytocin only 74.3%.

DISCUSSION

This was a prospective descriptive study done at Kenyatta National Hospital to determine the various methods of induction of labour, their indications and some of their outcomes. A total of 185 participants were recruited in the study, a period during which a total of 1322 deliveries were recorded resulting in a labour induction rate of 14% in this institution. This compares favorably with 9.8% induction rate in the USA noted in the year 2000. [17] The induction of labour rate in England for 1985 (Department of health, 1988) was 17.5% and a survey of constructive obstetrics units in the UK in1989-1990 revealed the rate at 17%. Rates for individual units varied from 4% -37% [7, 10, 14, 22]. In our study, with the data collected over a period of nine months, the centre recorded, 3607 deliveries, out of which 185 deliveries were induced, giving an induction rate of 2.3%, a value still below levels recored in developed countries.

Induction rates were not related to cesarean section rates in these units as have been suggested, but was a consequence of high induction rates in the

1970s [11]. The rate remained relatively constant from 1995-2004 at around 20% with an increase in planned cesarean section from 7-10% over the same period (Government statistical services for the department of health 2005). Khisa in 1999, in his study found the induction rate at the Aga Khan Hospital, Nairobi to be 14% with Prostaglandin alone or in combination with artificial rupture of membranes and/or oxytocin used in 48% of induction of labour cases [12].

Labour induction rates have relatively remained high despite the fact that many women do not primarily intend to undergo the labour induction process and prefer spontaneous delivery. With improving health services, labour induction has become imminent service vital for the well being of both mother and the foetus, but this noble aim has been hampered by induction failures. This prospective descriptive study was to look at the major indications of labour induction, various methods of labour induction used, some maternal outcomes, success and failure rates of labour induction and social demographic characteristics of women admitted for induction of labour to determine the rightful management parameters of labour induction with an aim to identify chances of labour induction failures at the Kenyatta National Hospital. Failure of induction of labour ultimately leads to increase in caesarian sections[23].

The majority of the study participants were aged between 26 and 30 years (40.5%) with 90% being in the active reproductive age of 20-35 years.

Majority were married, (63.2 %,) with single mothers being 30.8% .
Those who were self employed constituted 48.6%, while the employed ones were 42%, showing those with gainful employment to be over 90%.
The majority 68.1% had at least secondary education while those who had no education at all were only 4.9%, showing predilection of choice for this type of implant for the educated. Most of them were Christians (94.6%). While Muslims constituted only 3.8%.
Most of the participants were induced at term, thus 37 - 40+ weeks (37.2%), followed by moderate preterm gestation of 34-36+ weeks at 26.0% then severe preterm (28-33+) at 10.8%. Post-term labour inductions constituted 7.6% showing a general trend of doing induction with both maternal and fetal survival as a priority.

23

The commonest indication for labour induction was premature rupture of membranes at 36.8%, closely followed by pre-eclampsia/ eclampsia at 35.6%. Intra uterine foetal death was third at 12.4%. Khisa[12] found that the commonest indication for induction of labour was medical reasons, which formed 28% while Van Gemund[16] found the leading cause for induction to be post datism at 31.2%. Though the values differ, the most likely reason could be difference in set ups and environment. The chances or factors which lead to premature rapture of membranes e.g uterine tract infection are likely to be higher in a developing country set up like Kenyatta National Hospital, and with hypertensive diseases in pregnancies being more common in low social economic status, the incidences are likely to be more in my area of study.

A study by Jose Et Al, showed that in Latin America, 12 of the 19 Latin American countries had above 15% incidenceof labour induction ranging from 16.8%-40%[11], generally induction of labour is a common obstetric practice and according to the most current studies, the rate ranges from 9.5% to 33.7% of all pregnancies annually. During the study period, a total of 2562 deliveries wre recorded in Kenyatta National Hospital during which we had 185 inductions giving an induction rate of 7.2%, a figure slightly below those in Latin America and the world at large.

A majority of the participants 71.3% had spontaneous vaginal delivery and only 26.5% had caeserian section. Well below incidence of cesarean sections at KNH which stand at 44 %(Omengo Mmed thesis, 2003). But with prostaglandin, amniotomy and oxytocin as amethod of induction 92.8% had SVD while with oxytocin only 81.4% had SVD.Other meethods of induction produced less than 50% each for SVD.As can be seen in table 9 using prostagladin+amniotomy+oxytocin as a reference, use of prostagladin only had 48.0 times less chances of getting SVD while oxytocin only had 4.2 times, given odds ratio (OR) and the P-values stated.

In the study prostaglandin combined with Amniotomy and Oxytocin was the commonest method of induction of labour at 45.4% while Amniotomy alone was least common at 6.6%. This is comparable to Kurup[17] and Hadi[15] who both found prostaglandin combined with Amniotomy and Oxytocin as aprocedure for induction of labour at 38% and 41% respectively.

Using prostaglandin followed by Amniotomy and Oxytocin as standard exposure the study found that prostaglandin alone had 21.9 times more

(12)
(16)
11.

chances of having blood loss of more than 500mls comparatively at an odds ratio of 95% Confidence Index (C.I). Oxytocin alone had 0.4 times less chances of developing the same hence the sense of exogenous myometrial contractile factors in post partum haemorrage. Amniotomy alone had 21.9 times more chances of having blood loss of more than 500ml with a p value of 0.001.The use of Prostaglandin amniotomy and oxytocin had 6.4% chances of post partum hemorrhage, a value much in keeping with 8% incidence of PPH as noted by Khisa[12]

The outcome of labour following induction with SVD as a success was 71.3% with a duration of labour of less than 12 hours. Chamberlin G & Zander in Australia found success of 66.3% thus less, but probably due to higher efficiency in Labour monitoring and intervention. This can also be attributed to the variation in "passage" pelvic types. Sasaki K et al 2003 found a rate of 68.3% in Malaysia thus comparable.

Induction of labour is in overall aimed at the well being of both the mother and baby and when a compromise arises, priority is given to the mother[19]. This seems to be a major factor in determining the gestation at which induction is done. In our set-up, foetal survival, when induction is done at less than 28 weeks is minimal. In the study, 34 clients (18.4%) were induced at less than 28 weeks. This was primarily for the sake of the mothers health, e.g in IUFD, severe pre-eclampsia/ eclampsia. At between 28 and 33 weeks, still foetal survival, is not guaranteed and only 20 clients (10.8%) were induced. At between 34-40 weeks, a period characterized by better survival for both mother and baby, a total of 117 constituting, 63.2% were induced, the most preferred gestations for labour inductions. Lopez – Zeno, J A et Al[5] found an induction rate of this period (34-40 weeks), to be only 51%. This was most likely due to better paediatric care and foetal survival in their set-up.

Premature rupture of membranes remains the leading indication for induction in the study, with 68 in number (36.8%). This could be due to the vague definition of induction e.g. a mother who comes with a term PROM and started on Oxytocin was considered to have undergone induction. Pre-eclampsia and eclampsia followed close at 66 in number thus 35.6%, indicating induction of labour as an integral part in the management of this higly morbid condition. Other leading indications for induction were post

19.

datism, here defined as gestations greater or equal to 41 weeks (7.6%), inter uterine foetal death at 12.4%, bad obstetric history (BOH) at 4.9%. Stubbs TM, in his study, found no change in the incidence of caeserian sections with increase in labour inductions. This study showed a relatively less incidence of emergence caeserian sections (26.5%, 49 in number) compared to the incidence rate at KNH of about 44%[12].

12

CONCLUSION

1. The commonest gestation at which labour induction is done is 37 – 40+, thus term gestation.

2. The leading indication for labour induction is premature rapture of membranes followed by hypertensive diseases in pregnancy mainly eclampsia/ pre-eclampsia.

3. The commonest method used for labour induction is use of protaglandin and later, amniotomy and xytocin infusion.

4. The modal age of the client was 26 – 30.Most of them were married and of good education background.

5. Labour induction done using the right method leads to lower incidences of blood loss, appropriate duration of labour and higher incidence of vaginal delivery most of the times.

6. Good outcomes of labour induction are achieved when combinations of methods are employed, rather than when single ones are used.

Recommendation

a) Induction of labour is reccommendable medical procedure that should be encouraged when indicated. It not only ensures safety for both mothers and child but also reduces the incidences of ceaserean sections.

b) Clinicians and medical staff in general, should be encouraged to employ combination of methods e.g prostaglandin, amniotomy and oxytocin in practice for labour induction.

c) Practice should be instituted as to lead toearly recognition of cases which would need labour induction through good ante-natal care to facilitate diagnosis, control and treatment as to reduce the incidences of labour induction and its complications to ensure better reproductive health practices.

d) With better antenatal care the indications for labour induction can be recognised early, controlled and treated so as to reduce the incidences of labour induction and its complications.

e) More data and further studies are required to prove wether labour induction reduces the incidences of PPH and / or emergency cesearean sections.

REFERENCES

1. Norwitz E, Robinson J, Repke J. Labour and delivery. In: Gabbe SG, Niebyl JR, Simpson JL, eds. Obstetrics: Normal and problem pregnancies. 4th Ed. New York: Churchill Living stone, 2002:353-94

2. American College of Obstetricians and Gynaecologist. Induction of Labour. Practice bulletin No. 10. Washington DC. ACOG, 2004

3. Declercq E et al. Listening to Mothers: Report of the first U.S. National Survey on women's childbearing Experiences. New York: Maternity Center Association, October 2002

4. O'Driscoll K, Jackson RJ, Gallagher JT; Prevention of Prolonged Labour. Br Med J 2000 24th;2 (655):477-80

5. Lopez-Zeno JA et al. A controlled trial of a program for active management of labour. N Engl J Med 1992 Feb 13;326(7)450-4

6. Thornton JG. Active management of labour. BMJ 1996 Aug 17;313 (7054):378

7. Turnbull AC & Anderson AB 2002 introduction of labour 3 results with amniotomy and oxytocin titration .Jornal of Obstetrics nad gynaecology of the British common wealth 1999 pages 32 - 41

8. Chamberlain G & Zander L. ABC of Labour Care: Induction. BMJ 1999 April 10, 318 (7189):995-8

9. Kirby RS 2004. Trends in Labour Induction in the United States: Is it true that what goes up must come down? Birth 31;148-151

10. Mackenzie IZ & Boland J 2002 therapeutic uses of prostaglandins in obstetrics in the United Kingdom. Contemporary reviews in Obstetrics and gynaecology 5;9-15

11. BMJ: 1999,Nov. 27th, 332 (1397-1402), Jose Et Al.

12. Khisa W. W. Review of risk factors associated with induction of labour at the Aga Khan Hospital Nairobi. MMED Thesis, UoN pp194 - 247, 1999

13. Bishop EH 1964. Pelvic scoring for elective induction. Obstetrics and Gynaecology 24:266-268

14. Terore JL. Methods of cervical ripening and induction of labour. Am Fam Physician 2003;3;67:2123-8

15. Hadi H. Cervical ripening and labour induction. Clinical guidelines. C1in Obstet & Gynaecol 2000;43:524-36

16. van Gemund et al 2004. A randomized trial comparing low dose vaginal misoprostol and dinoprostone for labour induction. Br J of Obstet and Gynaecol 111:42-49

17. Am Family Physician 2003;67:2123-8. Copyright© 2003 American Academy of Family Physicians.

18. sa/amaleksi et al 2000. A randomized trial of pulsatile vs continuous oxytocin infusion for labour induction. Clinical and experimental Obs & Gyn 27:21-23

19. Friedman EA, Sachtleben MR. Effect of Oxytocin and oral prostaglandin E2 on uterine contactility and fetal heart rate patterns. Am J Obstet & Gynaecol 2001 43 (3) : 489:94

20. Stubbs TM. Oxytocin for Labour Induction. C!in Obstet & Gynaecol 2000;43(3):489-94

21. Swann RO 1998. Induction of labour by striping membranes 11: 7478

22. Adair CD. Non-pharmacological approaches to cervical priming and labour induction. C1in Obstet & Gynaecol 2000;43:447-54

23. Hill NC et al 1990. Physiological and Clinical effects of progesterone inhibition with RU38486 (mifepristone) in the 2nd Trimester. Br] Obstet & Gynaecol 97:487-92

24. Managni M & Cabrol 0 1986. Declenchement artificel du travial par la prostaglandine E2 apre maturation du col par I' estradiol. Inserm 151: 109-118

25. Sasaki K et al 1982. Cervical ripening with dehydroepiandrosterone sulphate. Br J Obstet & Gyn 89:195-198

26. Brennand JE et al 1995. Recombinant human relaxin as a cervical ripening agent.
Br] Obs & Gyn 104:775-80

27.Chwalisz K et al 1997. Cervical ripeng after a local application of nitric oxide.
Huan reproduction 12:2093-101

28.Finkel RS & Zarlengo KM 2004. Blue cohosh and perinatal stroke. New England Journal of Medicine 351:302-303

29.Kavangah J et al 2001. Breast stimulation for cervical ripening and induction of labour. Cochrane Database of Systematic Reviews CD003392

30. KavangahJ et al 2001. sexual intercourse for cervical ripening and induction of labour.Cochrane Database of Systematic Reviews CD003393

31.Britannica.com / EB/ checked/ 44527/ parturition and initiation of labour.

32. BMJ 02092-007,page 122

33.BJ, Vol.8,Iss.05, 03 May 2007, pgs 281-286

34.Julie M Harison Journal, Obs; Issue 135; Pages 35-93.

References:

GYNAECOLOGY CASE 1

IMPERFORATE HYMEN: CRUCIATE INCISION

NAME: A. L IP.NO: 1034990

AGE: 15 YEARS DOA: 22/6/06

PARITY: 0+0 D.OD: 23/6/06

PRESENTING COMPLAINT

The patient presented with history of low abdominal pain and swelling for 1week.

HISTORY OF PRESENTING ILLNESS

The low abdominal pain was colicky in nature, often so intense that she could not stand upright. The pain was reduced by iboprufen and hyocine butylbromide (buscopan) and aggravated by passing urine or stool. She experienced urgency and frequency but no dysuria or difficulties in passing urine. She had had the pain twice previously but the previous episodes were not as severe. She had noted a low abdominal swelling which she thought was progressively increasing.

PAST MEDICAL HISTORY

She had neither been diagnosed with any chronic illness nor been hospitalized before. She did not seek medical help during the first episode of pain and in the second episode. Her mother bought brufen and buscopan for her from a chemist shop on advice from a friend. This last episode however, iboprufen and buscopan were ineffective in relieving her pain.

OBSTETRIC AND GYNAECOLOGIC HISTORY

She was para 0+0 with no history of sexual intercourse or contraceptive use. She had not started menstruation

FAMILY AND SOCIAL HISTORY

She had just finished standard eight and lived with her parents. She was the second born in a family of four 2 boys and 2 girls. The other siblings were alive and well. There was no history of chronic or similar illness in 1st and 2nd degree relatives.

PHYSICAL EXAMINATION

She was in fair general condition, not pale or jaundiced and was afebrile but was in pain. Her blood pressure was 100/60mmHg, pulse rate 94/minute, respiratory rate 20/minute and temperature 37.1°C. She had Tanner stage four breasts and pubic hair and coarse axillary hair.

Respiratory, cardiovascular and central nervous systems were essentially normal.

ABDOMINAL EXAMINATION

Abdomen was slightly distended, with a tender suprapubic mass measuring up to 16 weeks. It was mobile with a smooth surface and firm with no fluctuance. There was no renal angle tenderness or swelling.

VAGINAL EXAMINATION

The labia, mons pubis and perineum were normal. There was a bluish mildly tender fluctuant swelling bulging at the vestibule. Vaginal canal was

obliterated by a smooth membrane. Rectal examination revealed a boggy mass that filled the vagina.

INVESTIGATIONS

- Pelvic ultrasound showed a large fluid collection with floating debris in the vagina and the endometrial cavity. The uterus was enlarged with no intrinsic lesions. The adnexae were normal and there was no fluid in the Pouch of Douglas.
- Haemoglobin level was 11.7g/dl, WBC 5.2 x 10^9/l and platelets 250 x 10^9/l
- Urea and Electrolytes were as follows: Na^+ was 136 mmol/l, K^+ 4.5 mmol/l, Urea 5.2 mmol/l and creatinine 52mmol/l.

DIAGNOSIS

A diagnosis of imperforate hymen with haematocolpos and haematometra was made.

MANAGEMENT

The patient and the mother were informed of the diagnosis and the intended management. An informed consent was obtained from the mother and the patient was admitted in the acute gynaecology ward (ID) for surgery.

She was starved from midnight on the day before surgery. During the day of surgery, pubic hair was shaven and premedication with atropine given before she was wheeled to theatre.

In theatre she was put under General anaesthesia. She was put in lithotomy position before vulval scrubbing and draping was done. The urinary bladder was catheterized and about 150ml of clear urine obtained. A cruciate incision was made on the bulging hymen from 2 to 8o'clock and from 4 to 10 o'clock. About 700mls of chocolate brown - coloured haematocolpos and haematometra was drained. The hymenal tissue was trimmed leaving a ring of tissue near the vaginal wall. There was no bleeding noted and stitching was not done. General anaesthesia was then reversed successfully and the patient was taken to the recovery room.

POST-OPERATIVE CARE

The patient was observed continuously in the recovery room till fully awake then transferred back to the acute gynaecology ward. She was put on doxycycline and brufen. She did well post-operative and was found in good condition with the uterus about 12 weeks, non-tender and with minimal vaginal drainage of menstrual flow. She was discharged home the following day on the same drugs for review in the gynaecology outpatient clinic (GOPC). She was advised to use sitz births and dilate the vagina with the small finger painted with vaseline daily.

FOLLOW UP

She was seen in the Gynaecology Clinic after four weeks and was found to have healed well. The incision edges had healed well, vaginal canal was patent and menstrual flow, which had started the previous day, was occurring freely. Menstruation was also painless. Digital examination revealed a normal vaginal canal and uterus with no adnexal tenderness.

DISCUSSION

A.L was a 15year old girl who presented with cryptomenorrhoea secondary to imperforate hymen. Cruciate incision was made with good outcome.

The hymen is a thin often cribriform mucous membrane that forms the sinovaginal bulbs and the urogenital sinus. The hymen perforates by degeneration of the centrally placed cell in embryonic life to establish a connection between the vaginal canal and the vestibule. When this does not occur imperforate hymen is said to exist [1, 2].

Imperforate hymen is often associated with urological anomalies. Usta reported minor anomalies including urethral membrane, imperforate anus, bifid clitoris, hypoplastic kidneys with ectopic ureters and vascular anomalies in 20% of the patients who also had familial history of imperforate hymen[3]. The patient presented had imperforate hymen without any other external anomalies and normal renal function.

Imperforate hymen is rarely diagnosed before puberty. Most patients present with symptoms of cryptomenorrhoea. Blood accumulates in the vagina and the uterus resulting in what is referred to haematocolpos and haematometra respectively. Other causes of cryptomenorrhoea are transverse vaginal septum and vagina agenesis or atresia [1, 2, 4].

The symptoms include low abdominal pain and distension. The pain may radiate to the back and is aggravated by urge to pass stool or urine. Failure to start menstruation in presence or normal secondary sexual characteristics

is universal. Urinary pressure symptoms like frequency and urgency or obstruction may be present. A tender suprapubic mass is usually palpable at presentation as is protrusion of the hymen. Per rectal examination usually reveals distention of the vagina by a cystic mass. [1, 2]. The patient presented had low abdominal pain and swelling as well as urgency and frequency but no urinary obstruction. She also had distention of the vagina on rectal examination.

Treatment of imperforate hymen involves making a cruciate incision between 2 and 8oclock and between 4 and 10oclock. The hymenal tissue is then excised but not too close to the vaginal wall to reduce the risk of scarring and stenosis, with subsequent dyspereunia. Haematocolpos and haematometra then drains spontaneously. Vacuum aspiration is only done when drainage is incomplete. Instrumentation should otherwise be avoided due to risk of perforating the fragile wall of the uterus. Prophylactic antibiotics are administered for a week and unnecessary vaginal examination avoided to avoid infection [5].

Late complications of imperforate hymen and the surgery are rare but may include dyspereunia due to stenosis and endometriosis. Chronic pelvic pain and infertility may result from endometriosis [2, 4].

Our patient underwent cruciate incision with complete spontaneous drainage of the haematolcolpos and haematometra and subsequently healed without apparent stenosis. Menstrual flow preceding her review in the clinic had been painless suggesting absence of endometriosis.

REFERENCES

1. Rock J.A., Horowitz I.R., Dominguez C.E. Surgical conditions of the vagina and vulva. Te Lindes Gynecologic Surgery 9th Ed. 2001; Lippincot-Raven Publishers, Philadelphia, **34**:893 - 924

2. Chang L., Muram D. Pediatric and Adolescent Gynecology. Current Obstetric and Gynaecologic, diagnosis and treatment, 9th Ed. 2003; McGraw-Hill, New York, **31**:595 - 622.

3. Usta I.M., Auward J.T., Usta J.A. Imperforate Hymen: Report of unusual familial occurrences. Obstet Gynecol 1993; **82**: 665.

4. Dewhurst L. Normal and abnormal development of genital organs. Dewhurst textbook obstetrics and gynecology for postgraduate students 4th Ed. 1988; Blackwell publishers, Philadelphia **1**:1 - 14

5. Varma T.R. Formation of urogenital system, imperforate Hymen. Clinical Gynecology 1st Ed 1991; **4**: 96

GYNAECOLOGY CASE 2

RUPTURED ECTOPIC – SALPINGECTOMY

NAME R.A. PARITY 3+0

AGE 28 L.M.P. 25-12-06

I.P NO. 1149091

D.O.A.1-3-2007

D.O.D. 4-3-2007

PRESENTING COMPLAINT

R.A. was admitted with a two day history of vaginal bleeding and lower abdominal pain.

HISTORY OF PRESENTING COMPLAINT

She was well until two days prior to admission when she started experiencing vaginal bleeding. There was associated lower abdominal pain which progressively worsened. She complained of mild dizziness and general body weakness but no palpitations. She had no dysuria or frequency of micturition. She had no diarrhea or vomiting. She had not sought any medical advise before admission.

OBSTETRIC AND GYNAECOLOGY HISTORY

She was para 3+0. She had had three term hospital deliveries in 2005, 2003 and 2001. all children were alive and well. Her menarche was at 16 years and her cycles occurred every 28 days with flow of 3 days. She gave no history of dysmenorrhoea. She denied any previous treatment for sexually transmitted illnesses or any vaginal discharge. She had never used any contraceptive method. Her last normal period was on 25-12-2006 giving a period of amenorrhoea of 9 weeks.

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 mechanic in Nairobi industrial area and they lived in Huruma. She neither smoked cigarettes nor drank alcohol. 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 90/50 mmHg, pulse was 102/minute and respiratory rate 20/minute. Her respiratory, cardiac and nervous systems were normal.

ABDOMINAL EXAMINATION

The abdomen was distended in the suprapubic region.there was marked tenderness with more on the left lower quadrant accompanied by guarding. There was no organomegally. Paracentesis done after the pelvic examination revealed non-clotting haemoperitoneum.

PELVIC EXAMINATION

Her external genitalia were 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

R.A was explained to the diagnosis and the plan for laparatomy. An intravenous drip of 1 liter normal saline running fast was set up. At the same time blood for haematocrit and blood grouping was taken. Two units of typed blood were availed. Her haematocrit was 25% (Hb 8.3g/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.

In theatre, general anaesthesia was induced and maintained. The patient was placed in semi-lithotomy position, vulval vaginal toilet was done and aseptic catheterization was 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 pfanelstel 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 portion of the tube. Left partial salpingectomy was and evacuation of the haemoperitoneum was done. The left tube and its mesosalpinx were 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 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 1 liter. Anaesthesia was reversed, the patient was 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. Analgaesia was given as pethidine 100mg 8 hourly for 48 hours

then ibuprofen 500mg three times daily for one week. Oral feeding was started 24 hours after surgery. Haematinics (Ranferron 10mls twice daily) were started in addition to oral antibiotics. Her postoperative period was uneventful and she was discharged on the third post operative day. She was given an appointment to be seen in the gynaecology outpatient clinic after one month.

FOLLOW UP

When she came after two weeks she was noted to be well, was not pale and the wound had healed well. Histology of the mass taken at laparatomy 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 chance of another ectopic pregnancy hence the need for evaluation at the earliest opportunity 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 left tubal ectopic 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 that the decidualized endometrial lining of the uterine cavity.implantation thus occurs in the absence of decidualised endometrium and endometrial glands. Ninety five percent of ectopic pregnancies affect the fallopian tubes. other sites include ovary, cervix, broad ligament, rudimentary horn of the uterus and the abdominal cavity, multiple ectopic pregnancies can also occur involving 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 patient presented was discharged home in good 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] reported the incidence of ectopic pregnancy as 4-5 per week or 1:15 deliveries. The age incidence was found to be 20-29 years (worldwide 25-34). Our patient was 28 years old. The incidence is increased due to increased pelvic infections, improved treatment of pelvic infections ad use of intrauterine devices especially those that contain progesterone.

Our patient was found to have pelvic adhesions at laparatomy 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 fertilize ovum into the uterine cavity. This can be caused by many factors including
    - Endosalpingitis from pelvic infections
    - Peritubal adhesions
    - Previous ectopic pregnancy
    - Developmental abnormalities such as diverticula, accessory ostia, lengthy tube and hypoplasia or atresia.
    - Previous tubal surgery such as tuboplasty or reversal of surgical sterilization
    - Multiple previous induced abortions
    - Tumours that distort the tube such as myomas or adnexal masses
    - Previous caesarian section
- Functional factors that 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, transmigratin of the ovum, post mid-cycle ovulation and fertilization and ovarian enlargement due to use of clomiphene or menotropins[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, in utero exposure to diethylstilbestrol, increased age, Chlamydia infection and tuberculous endsalpingitis.

In our patient suggestive history and physical examination were used to make the diagnosis. Abdominal pain with amenorrhea 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 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] 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 either trans-abdominal or trans-vaginal, β-HCG levels and laparoscopy are other diagnostic aids that are useful 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 if ectopic pregnancies are tubal accounting for as high as 99% of cases. The ampulla is the most common site of implantation accounting for 78%, 12% are isthmic, 5% fimbrial and 2% are cornual. The clinical course will depend on the anatomical site and the stage of 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 laparatomy. Treatment of ectopic pregnancy depends on the site of the ectopic and wether it is ruptured or not. Surgery is the preferred treatment of ectopic when there is rupture, hypotension or pain persisting for more than 24 hours. Blood

products should be availed as tansfusion is often necessary. Partial or total salpingectomy is done once the source of bleeding is identified. Partial salpingectomy is done when only a small portion of the tube is affected to allow for later tubal surgery to achieve function in the future. Recently more conservative surgery has been used in attempts to conserve the tube. These 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 associated with high rate of persistent haemorrhage and high recurrence rate of ectopic.

Interstitial pregnancies may require wedge resection with uterine reconstruction. Ovarian pregnancy requires oophrectomy 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 left in place to avoid haemorrhage. Chronic ectopic pregnancies are difficult to diagnose and are often found incidentally during exploratory laparatomy. Approximately 3-20% of ectopic pregnancies are chronic. Dense adhesions and luminal abscesses are surgical features characterizing chronic ectopic pregnancies[1.4.5.8].

Laparoscopy is now used in many centres 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 patients. Currently, laparoscopy is not available for emergency situations in our unit.

Medical management of ectopic pregnancy is by use of methotrexate either locally or intravenously with or without folinic rescue is used for unruptured ectopic pregnancy. The success rate is higher if the fetus is less than six weeks or the tubal mass is 3.5cm or less and the fetus 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, tissues 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 βHCG titres. Failure of methotrexate is suggested by persistent rising or plateau in βHCG. 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, hyperosmolar glucose and even methotrexate are injected into the ectopic pregnanacy transvaginally under ultrasound guidance. Other access routes for delivery of 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. Contrindications to medical therapy include breastfeeding, overt or laboratory evidence of immunosuppresion, pre-exsisting blood dyscrasias, peptic ulcer disease and hepatic, renal or hematological 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 are having proliferating ectopic pregnancies and require intervention is a clinical dilemma. Expectant management has been used in the hope of avoiding therapy that may otherwise be unnecessary as many ectopic pregnancies resolve spontaneously. Candidates for expectant management must be willing to accept the potential risks of tuba rupture and haemorrhage. If the initial βHCG is less than 200miu/ml or decreasing and the risk for rupure is low,

then expectant management can be used[4,5,10]. this method is not used 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 National 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.

After treatment, contraception should be advised as return to fertility is within 2-3 weeks. Our patient was counseled and advised 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.

REFERENCES

1. Cunningham, MacDonald, Grant, Leveno et al. ectopic pregnancy; in Williams obstetrics 22nd edition 2005.
2. Russel J.B. the aetiology of ectopic pregnancy. Obstet Gynaecol. 1987; 30:181-190.
3. Monaghan A. common gynaecological emergencies 1st edition. Wright London 1988; 35-43.
4. DeCherney A.H, Nthan L. early pregnancy risks; In current obstetric & gynecologic diagnosis & treatment. McGraw Hill, 9th edition 2003 pp 278-283.
5. Thomas S. early pregnancy loss and ectopic pregnancy. In Novaks gynaecology 13th edition pp 507-535.
6. Mwathe E.G. Patterns of ectopic pregnancies at KNH. MMed thesis, UON.
7. Sinei S.K. Okumu S. Ectopic pregnancy at KNh, Nairobi. J. Obstet. Gynecol E.Central Africa 1987; 6:9.

8. Monaghan J.M. Management of extrauterine gestation; in Bonney's gynaecological surgery 9th edition 9th edition 2002 pp 168-172.
9. Murphy A.H, Najer C.W, Wujeck J.J, Kettel L.M. Operative laparoscopy for management of ectopic pregnancy. Fertil. Steril. 1992; 57:1180-85.
10. Steven J. Medical management of tubal pregnancy. A.C.O.G practice bulletin No. 3, 1998.

Rakwach P.O. review of mortality in emergency gynaecological ward at Kenyatta National Hospital. M.Med thesis U.O.N. 1987

GYNAECOLOGY CASE 3

BILATERAL TUBAL LIGATION FOR DESIRED FAMILY SIZE

NAME L.W.

IPNO 1027251

AGE 33 YEARS

PARITY 4+0

DOA 13-3-2007

DOD 17-3-2007

PRESENTING HISTORY

She delivered on 14-3-2007 by spontaneous vetex delivery to her fourth child. She decided to have a BTL as she had now got her desired family size. She had expressed her desire during her antenatal clinic attendance at KNH and had been counseled along with her husband and had given consent to the procedure.

OBSTETRIC AND GYNAECOLOGY HISTORY

She was para 4+0. her first delivery was in 1993 at term in hospital, she had a 3.5 kg baby girl. In 1994 she had a hospital delivery to a 3.8 kg baby boy. In 2002 she had a 3.2 kg baby girl in hospital. Her last child was born during this admission SVD to 3.2 kg boy who scored 9 at 1 minute and 10 at 5 minutes.

Her menarche was at 14 years and had regular 28 day cycle with 3 day flow. She had previously use depo provera on and off between her other children.

PAST MEDICAL HISTORY

She is asthmatic on budecort and was last admitted in 2005 with an acute asthmatic attack.

GYNAECOLOGY CASE 3

BILATERAL TUBAL LIGATION FOR DESIRED FAMILY SIZE

NAME L.W.

IPNO 1027251

AGE 33 YEARS

PARITY 4+0

DOA 13-3-2007

DOD 17-3-2007

PRESENTING HISTORY

She delivered on 14-3-2007 by spontaneous vetex delivery to her fourth child. She decided to have a BTL as she had now got her desired family size. She had expressed her desire during her antenatal clinic attendance at KNH and had been counseled along with her husband and had given consent to the procedure.

OBSTETRIC AND GYNAECOLOGY HISTORY

She was para 4+0. her first delivery was in 1993 at term in hospital, she had a 3.5 kg baby girl. In 1994 she had a hospital delivery to a 3.8 kg baby boy. In 2002 she had a 3.2 kg baby girl in hospital. Her last child was born during this admission SVD to 3.2 kg boy who scored 9 at 1 minute and 10 at 5 minutes.

Her menarche was at 14 years and had regular 28 day cycle with 3 day flow. She had previously use depo provera on and off between her other children.

PAST MEDICAL HISTORY

She is asthmatic on budecort and was last admitted in 2005 with an acute asthmatic attack.

FAMILY AND SOCIAL HISTORY

She is a housewife married to an electrician. They live in Eastleigh. There is no family history of

chronic illness. She neither drinks alcohol nor smokes cigarettes.

PHYSICAL EXAMINATION

She was in fair general condition. She was not pale or jaundiced and she had slight pedal pitting oedema. Her temperature was 36.8°C pulse 84/min, blood pressure 120/80 and respiratory rate of 16/minute.

The cardiovascular, respiratory and central nervous system were normal.

On abdominal examination, the abdomen was slightly distended and moved normally with respiration. There were no surgical scars. She had a uterine size corresponding to 16 weeks of gestation. The uterus was non tender and well contracted. There were no other masses palpable.

Vaginal examination revealed she had an episiotomy wound that was sutured and clean. No other perineal laceration was seen. The cervix was parous and there was lochia serosa on the examining finger which was not foul smelling.

Diagnosis

Multipara with desired family size in the immediate post partum period was made.

INVESTIGATIONS

1. ANP Blood group A+ve, VDRL negative, HIV negative, Hb 10.0
2. PCV 48
3. Na 145, K 3.7, BUN 2.5, creatinine 60

MANAGEMENT

She was prepared for minilaparatomy in theatre. The consent form she had signed while in the antenatal clinic was checked and she was starved from midnight on the day of the procedure. She was also given pre-medication with atropine 0.6mg 30 minutes before the operation which was done on 16th march 2007.

MINILAP FOR BTL

In theatre she was put under general anaesthesia. She was then catheterized asceptically. Vaginal examination was done which showed her to have a well healing episiotomy, closed cervical os and non-foul smelling lochhia serosa. She was then cleaned and draped.

The uterine fundus was palpated and a transverse incisin 2 cm long was made 2 cm below the uterine fundus. The incision was opened and extended downwards to the rectus sheath and the peritoneum to gain access to the peritoneal cavity. Th head of the table was then lowered to move the gut and omentum away from the operation site and the uterus was thus identified.

The right fallopian tube was identified and lifted using the hook and with the help of a babcock forceps.this was ligated and cut using pomeroys method for tubal ligation. The left tube was identified and the same process repeated. The cut segments were identified to confirm they were indeed tubes.

The peritoneum was left unsutured and the adomen closed in layers. She was reversed and wheeled to the recovery section and subsequently to the ward.

FOLLOWUP

She was discharged the next day and given an appointment to attend post natal clinic after one week. She did not honour her appointment.

DISCUSSION

Our patient was a multiparous patient with desired family size who chose bilateral tubal ligation as a mode of family planning.

Tubal ligation is permanent method of contraception which involves the mechanical occlusion and/or resection of a portion of the reproductive passages. It is one of the surgical methods of contraception available today[1].

Voluntary sterilization of either partner is the most frequently used method of fertility regulation in the world. This method was first described in the late 1800s but was not used for contraception until early to mid 1900's.

Four procedures are commonly used:

1. Tubal sterilization at the time of laparatomy for a caesarian delivery or other abdominal operation.
2. Postpartum minilaparatomy soon after vaginal delivery.
3. Interval minilaparatomy.
4. Laparascopy.

Globally contraception prevalence has risen from less than 10% in the early 1960's to an estimated 55% in china, threefold in Europe and India and fourfold in the usa. The rapid changes are due in part to acceptance of sterilization by the community and the interplay between religious and cultural beliefs and family planning. According to the Kenya contraceptive prevalence survey of 1994, surgical sterilization forms the third most commonly used method after oral contraceptions and the intrauterine contraceptive device, with an estimated 400 tubal ligations done at the Kenyatta national hospital yearly. In Kenya, it accounts for 5% of contraception prevalence. In Burkina faso, voluntary female sterilization

accounts for only 9% of the contraceptive methods used and is mainly done as a post partum procedure[2,3,4].

There are two broad classifications of tubal ligation; post partum and interval. Post partum BTL is carried out in the immediate post partum state as was the case in our patient. Interval BTL is carried out remote from pregnancy. Postpartum minilaparatomy 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 uteine fundus is near the umbilicus and the tubes easily accessible.

The indications for tubal ligation include; multiparity, completed family size, socio-economic reasons, medical reasons like diabetes, chronic pulmonary disease, heart disease[5]. our patient had BTL due to desired family size.

There are various methods of female sterilization. Those used with the minilaparatomy approach include madlener method, pomeroy method, Irving, Kroener and Uchida methods. Laparascopic sterilization is accomplished by any of four techniques: bipolar electrical coagulation, application of silastatic rubber band (falope ring)[8], the plastic and metal Hulka clips[9] or the Filshie clip[10].

In the case presented, the Pomeroy method was used. In the classic Pomeroy method, a loop of tube is excised after ligating the base with a single absorbable suture. This method is easy to perform given the size of incision and gives a low failure rate. It is the method recommended in Kenya today. The modified pomeroy method varies in that the mid portion of the tube is excised after ligation of the segment with two separate absorbable sutures. Pomeroy and partial salpingectomy procedures have failure rates of 1 to 4 per 1000 cases[11]. the failure rate may be due to defective materials or

technical error[1,2]. in the Madlener technique now abandoned due to too many failures, a loop of tube is crushed by cross-clamping its base, ligated with permanent suture and excised. In the Uchida method, the serosa of the tube is opened and separated from the muscular tube by injecting a saline epinephrine solution beneath the mid-portion of the tube. The mucosa is incised along the anti-mesenteric border of the tube and about 5 cm of tube is cut out. The part of the tube from the uterus is retracted and buried in the serosa while the fimbriated end and serosa are closed and tied together. In the Irving method, the mid portion of the tube is excised and the proximal stump of each tube is turned back and led into a small stab wound in the wall of the uterus and sutured in place creating a blind loop[12]. Pregnancy is almost uneard of in the Uchida and Irving methods[7,11].

Utbal sterilization is remarkably safe. In 1983, the total complication rate in a large series from several institutions was 1.7 per 100[13]. Complications were increased by use of general anaesthesia, previous pelvic otr abdominal surgery, history of PID, obesity and diabetes mellitus. Complications included psychiatric disturbances, wound infections, failure of operation, unintended laparatomy, regrets and dissatisfaction. Evens gave rates of regrets or dissatisfaction following surgical sterilization as 3.3% for interval and 8.7% for postpartum.

Complications can be minimized by carefully screening for contraindications including postpartum haemorrhage which increases the risk of infection. In postpartum tubal ligation, the abdominal wall is thin at the umbilicus. Dissection into the abdomen must therefore be cautious to minimize the risk of damage to the intestines. Haemorrhage a common complication of postpartum BTL can be avoided by gentle handling of the tube and secure application of the ligatures. To reduce the potential for bacteria ascending th tubes, the procedure should be performed within 48

hours of delivery and prophylactic antibiotics given if the procedure is performed on the 3rd to the 7th day postpartum. If the procedure cannot be done within 7 days after delivery, it is often advisable to wait until 4 to 6 weeks post partum. Ruminjo in his series reported an overall 30.2% of complications of which 98.7% were minor out of 1999 tubal ligations. The main complication was wound abscesses and haematoma. Our patient had no complications by the time of discharge.

In addition to providing excellent contraception, tubal ligation is associated with reduced risk for ovarian cancer that persists for as long as 20 years after surgery.

Reversal of sterilization is more successful after mechanical occlusion than after electrocoagulation because the latter method destroys much more of the tube. With modern microsurgical techniques and an isthmus-to-isthmus anastomosis, pregnancy follows in about 75% of cases[14]. a substantial risk of ectopic pregnancy exsists after reversal. Henderson reported pregnancy rates of 42% after tubal diathermy compared with 67% following pomeroy or ring sterilization. The number of patients who request for reversal is between 0.1 to 10% of these approximately 30-70% may be suitable for reversal. Remarriage is the most common cause for request for reversal of sterilization and it is more likely to occur in young women who are more often contraceptive users, have spent less time in school and have had more abortions and fewer live children. In this group, counseling is vital.

REFERENCES

1. Dao B. et al. voluntary female sterilization via minilaparatomy. Report for Burkina faso. E.A.M.J. Vol:74 no.2 february 1997.
2. Douglas Huber M.D. voluntary surgical contraception in contraceptive technology international edition 1989 page 209-236.

3. Gomel V. microsurgical reversal of female sterilization. Reappraisal fertile steril 33:587,1980.

4. Newton J.R. sterilization in contraceptive update. Clinics in obstetric and gynaecology vol II No. 3 December 1994 pages 603-636.

5. ruminyo J.K. et al. A comparative study of users of long term contraceptive subdermal implants and female surgical contraception in Kenya. E.A.M.J. Vol 70; No. 9 september 1994.

6. Susu B. et al. family planning practices before and after childbirth in Lusaka, Zambia. E.A.M.J. vol 73, No 11 november 1999.

7. Uchida H. Uchida tubal sterilization. Am J Obstet Gynecol 1975;121:153-159.

8. Yoon IB King TM Parmley TH A two year experience with the Falope ring sterilization procedure. Am J Obstet Gynecol 1977;127:109-112.

9. Hulka J.F. metods of female sterilization in Nichols DH ed Gynecologic and obstetric surgery. St. Louis; Mosby 1993;640-651.

10. Filshie GM, Pogmore JR, Dutton AG et al. The titanium/Silicon rubber clip for female sterilization. Br J Obstet Gynaecol 1981;88:655-662.

11. Shepard MK. Female contraceptive sterilization. Obstet Gynecol Surv 1974;29:739-787.

12. Irving FC. A new method of insuring sterility following caesarian section Am J Obstet Gynecol 1924;8:335-337.

13. Desatefano F, Greenspan JR, Dicker RC et al. complications of interval laparascopic tubal sterilization. Obstet Gynecol 1983;61:153-158.

14. Corson Sl. Female sterilization reversal. In Corson Sl, Derman RJ Tyrere LB eds. Fertility control Boston: little, Brown 1985:107-118.

GYNAECOLOGY CASE 4

SYMPTOMATIC UTERINE FIBROIDS: TAH DONE

NAME A.B.

IPNO 1038930

AGE 40YRS

DOA 11-3-07

DOD 18-3-07

PRESENTING COMPLAINTS

Abdominal swelling for 3 years

Heavy periods for 3 years

HISTORY OF PRESENTING ILLNESS

A.B. was well until three years ago when she developed abdominal swelling that was increasing slowly in size with time. Later she developed heavy periods. She would use 6 to 9 pads up from her usual 3 to 4 pads. The periods would last 7 to 10 days up form her usual 3 days. She had no history of dizziness or palpitations.

PAST MEDICAL HISTORY

She was HIV positive and was on anti retroviral treatment with combivir and nevirapine.

OBSTETRIC AND GYNAECOLOGIC HISTORY

She was para 2+0. Her first child was a boy born vaginally in 1983 at home. Her second child was female born in 1985 at home also vaginally. Both children were alive and well. She had used oral contraceptive pills for a year after her first delivery. Between 1986 and 1988 she had used

injectable contraception. She stopped contraception due to desire to conceive.

FAMILY SOCIAL HISTORY

She was unemployed and a widow. Her husband had died in 2002 due to meningitis. She lived at Mlango Kubwa in Nairobi. She neither smoked cigarettes nor drank alcohol.

PHYSICAL EXAMINATION

She was in fair general condition was not pale or jaundiced and was afebrile.

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

ABDOMINAL EXAMINATION

The abdomen was distended in the suprpubic region and moving normally with respiration. There was a mass arising from the pelvis corresponding to a 20 week gestation. It was firm, mobile, nodular and non-tender. There was no other organomegaly or area of tenderness.

VAGINAL EXAMINATION

The external genitalia were normal. The cervical os was parous and the uterus was bimanually palpable and mobile. The adnexa were free.

INVESTIGATIONS

Pelvic ultrasound report (20.09.06)

The uterus is massively enlarged by multiple fibroids. No adnexal lesions are seen. The pouch of Douglas is seen.

Haemogram: WBC 5.7 x 109/L

HB 12.3 g/dl

PLT 236.1 x 109/L

Urea and creatinine Urea 2.4mmol/l

Creatinine 102 umol/l

Pap Smear (17.12.06) Normal cervical cytology.

OPERATION NOTES

General anaesthesia was induced. In semi-lithotomy position the perineum was cleaned and the bladder catheterized aseptically draining clear urine. The vagina was then cleansed with an iodine solution. The patient's legs were straightened. She was then cleaned and draped.

The abdomen was opened via a pfannenstiel incision. The uterus was noted to be enlarged with multiple fibroids. She was also noted to have pelvic adhesions. A stay suture was placed on the uterine fundus for traction during the operation.

The distal portion of the left round ligament was clamped cut and ligated. The anterior leaf of the broad ligament was incised separating the peritoneal reflection of the bladder from the lower uterine segment. A window was created in the posterior leaf of the broad ligament under the utero-ovarian ligament and fallopian tube. A clamp was placed medial to the ovaries and the fallopian tube cut and ligated preserving the ovaries. The above procedures were repeated on the right side. The bladder was then dissected away from the lower uterine segment.

The uterine vessels were identified clamped cut and ligated on both sides. The cardinal ligaments on both sides were clamped cut and ligated until the junction of the cervix and vagina was reached. The uterus was then removed

by cutting round the cervix. The vaginal cuff was then closed. The peritoneum was closed and the abdomen closed in layers after ensuring correct swab and instrument count and hemostasis. Blood loss was estimated at 500ml.

General anaesthesia was reversed and the patient wheeled to recovery room.

POST OPERATIVE MANAGEMENT

She was put on broad spectrum antibiotics and analgesics. She recovered well and was discharged home on the fourth day post operatively.

FOLLOW-UP

The patient was seen in the clinic at two weeks and six weeks and had fully recovered. Histology confirmed the diagnosis of uterine leiomyomata with normal cervix and endometrium.

DISCUSSION

A.B. presented with symptomatic uterine fibroids and underwent total abdominal hysterectomy and was discharged home well.

Uterine fibroids, also known as leiomyomata are benign tumours which arise in the myometrium. They are composed predominantly of smooth muscle with a variable amount of connective tissue and have a characteristic whorled appearance on cross section[1].

Fibroids are frequently asymptomatic and it is therefore not possible to give a precise figure of incidence since many remain undiagnosed. They are however the commonest tumour of the reproductive tract. In the western countries, the figure is quoted as 50%. The incidence of fibroids is affected by age and race[2]. They are more common in the black population and towards the end of the child bearing period between 35 and 45 years. A.B. was 40 years old. Wanjala (1980) in his survey noted that 66.8% of total

abdominal hysterectomies done in Kenyatta National Hospital were due to uterine fibroids[3].

Fibroids are not seen in any other animal species except humans. The aetiology of fibroids is unknown but it is associated with high oestrogen levels, which exerts its effect through the epidermal growth factor, since they do not occur before puberty and regress in size after menopause. Prolactin and growth hormone promote their growth while progesterone inhibits their growth. Women with fibroids have been found to have other endocrine abnormalities. They have low follicle stimulating hormone and a diminished follicle stimulating hormone response to thyrotropin releasing hormone. Uterine fibroids are common in nulliparous women. Wanjala reported that 85% of women with fibroids had not had a pregnancy in the last 6 years. A.B. was para 2+0 and had her last delivery in 1985. Several studies suggest that each leiomyoma arises from a single neoplastic cell within the smooth muscle of the myometrium[4]. There appears to be an increased familial incidence[5].

There are three main groups of uterine fibroids. Submucous myomas lie just beneath the endometrium and tend to compress it as they grow towards the uterine lumen. Intramural myomas lie within the uterine wall while subserous tumours lie just at the serosal surface of the uterus. External tumours tend to become pedunculated and may acquire extra-uterine blood supply from omental vessels hence become parasitic. The patient presented had intramural fibroids.

Uterine fibroids can undergo benign or malignant secondary changes. Degenerative changes occur in approximately two-thirds of all specimens[6]. Benign changes include atrophy, hyaline changes, cystic changes, calcification, septic changes, carneous and myxomatous (fatty) changes.

Malignant transformation is a neoplastic phenomenon and its incidence is 0.1-0.5%.

Menorrhagia is the most common initial symptom associated with fibroids and is the one that most commonly leads to surgical intervention. Menorrhagia is thought to be due to ulceration of submucous leiomyoma, anovulation associated with leiomyomata leading to endometrial hyperplasia, increased bleeding surface area and venous congestion due to compression of the venous plexus of the adjacent myometrium and endometrium.

Chronic pelvic pain may also be present and it is often characterized as dysmenorrheal, dyspareunia or pelvic pressure. Acute pain may result from torsion of a pedunculated leiomyoma or infection and degeneration.

Urinary symptoms include frequency due to extrinsic pressure on the bladder. Partial ureteral obstruction may occur. Some reports suggest some degree of ureteral obstruction in 30% to 70% of tumours above the pelvic brim. Ureteral compression is 3 to 4 times more common on the right because the left ureter is protected by the sigmoid colon. Rarely the ureters may obstruct completely.

Pregnancy loss or complications can occur in women with leiomyomas although most patients have uncomplicated pregnancies and deliveries: one study found a rate of 10%[7]. Although growth of leiomyomas may occur in pregnancy, no demonstrable change in size based on ultrasound has been noted in 70% to 80% of patients[8,9]. Leiomyoma have been reported as a sole cause in a small percentage of infertile patients[10]. Infertility coud be due to unovulation, intereference with sperm transport, abnormal vascularization preventing implantation, cervical and cornual obstruction and the associated pelvic inflammatory disease.

Uterine fibroids are usually diagnosed cinically but a number of techniques are of value in furter assessment. Laboratory findings may reveal anemia as a result of excessive uterine bleeding and depletion of iron stores. In certain cases polycythemia may be present and this is attributed to compression of the ureters to cause ureteral back pressure and thus induce erythropoietin. Leucocytosis and elevated erythrocyte sedimentation rate may be present with acute degeneration or infection.

X-rays are useful in diagnosis especially if the tumour has undergone calcification. Pelvic ultrasound is now routinely performed to evaluate the nature of the mass and especially to differentiate an ovarian lesion and exclude pregnancy. Other diagnostic work up includes hysterography, hysteroscopy and laparascopy. Summer and co-workers in a study described a syndrome in black women consisting of hypertension, obesity and myomas.

When considering differential diagnosis, all other causes of irregular bleeding in women approaching menopause have to be considered, in particular malignant disease of the genital tract. Ovarian dysfunction may co-exsist and this explains the delay in menopause which occurs frequently in women with uterine fibroids. Abdominal pain may be due to pelvic inflammatory disease. Other differentials include a gravid uterus, bowel tumours, mesenteric and retroperitoneal tumours and a full bladder[12].

The management of women with uterine fibroids depends on the associated symptoms, the age, reproductive status of the individual and the rate of growth of the tumour. Small asymptomatic fibroids do not require intervention. When the tumour is small and the patient desires more children, myomectomy is performed, although myoma as the sole cause of infertility accounts for only 2-10% of the patients.

Myomectomy carries a higher morbidity than hysterectomy and it is more difficult to perform. Myomectomy in women with menorrhagia has been followed by subjective relief of this symptom in between 40 and 91% of cases. In Kenyatta National Hospital myomectomy as an indication for infertility has a success rate of less than 20%.

When the tumour is large and the symptoms severe, total abdominal hysterectomy is performed. Newer surgical methods include the removal of subserous and intramural fibroids by laparascopy, peduculated submucous fibroids by hysteroscopy or ablation of submucous fibroids with neodymium-yttrium aluminum laser.

Medical treatment includes the use of gonadotrophin releasing hormone analogues. This can be used as a hemostatic and to reduce the tumour size in pre-operative patients with uterine fibroids. Progestogens, prostaglandin synthetase inhibitors, oral contraceptives, androgens and LHRH analogues have also variously been tried[1,11,12,13].

REFERENCES:

1. Bailliere's clinical obstetrics and gynaecology. Dysfuctional uterine bleeding and menorrhagia. 1989 page 357.
2. Martin L. Pernoll and Ralph C Benson. Current obstetric and gynaecologic diagnosis and treatment. 7th edition pag e 657-661.
3. Wanjala S. Uterine fibroids at Kenyatta National Hospital. MMed thesis U.O.N. 1980.
4. Tindal V.R. Tumours of the corpus uteri In Jeffcoates principles of gynaecology page 417, 5th edition 1987.

5. Howard W.J. Anne C.W and Lonnie S.B. Novaks textbook of gynaecology 11th edition page 443.

6. Spellacy E.N. et al. Growth hormone and estradiol levels in women with uterine myomas. Journal of obstetrics and gynaecology.

7. Townsend DE, Sparkes RS, Baluda MC et al. Unicellular histogenesis of uterine leiomyomas as determined by electrophoresis by glucose -6-phosphate dehydrogenase. Am. J. Obstet Gynecol 1970;107:1168-1173.

8. Marshall LM, Speigelman D, Barbieri RL et al. variation in the incidence of uterine leiomyoma among pre-menopausal women by age and race. Obstet. Gynecol. 1997;90:967-973.

9. Persaud V, Arjoon PD. Uterine leiomyoma. Incidence of degenerative change and correlation of associated symptoms. Obstet Gynecol 1970;35:432-436.

10. Katz VL, Dotters DJ, Droegemeuller W. complications of uterine leiomyomas in pregnancy. Obstet Gynecol 1989;73:593-596.

11. Rosati P, Exacoustos C, Mancuso S. Longitudinal evaluation of uteruine leiomyoma growth during pregnancy. A sonographic study. J Ultrsound Med. 1992;11:511-515.

12. Aharoni A, Reiter A, Golan D et al. Patterns of uterine leiomyomas during pregnancy. A prospective study. Br j Obstet Gynecol 1988;95:510-513.

13. Lumsden M.A, Wallace E.M. Clinical presentation of uterine fibroids. Ballieres clin. Obstet gynecol 1998;12:177-195.

GYNAECOLOGY CASE 5

BARTHOLINS ABSCESS: MARSUPIALIZATION DONE

Name: E.G.K

Age: 46 years Parity 2+0

Ip No: 1149154 Last delivery 1990

DOA 12-3-2007 LMP 1-3-2007

DOD 14-3-2007

Presenting complaint.

She presented with four day history of pain and swelling of the vulva.

History of presenting illness

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

Past medical and Surgical history

She had been treated for bartholins abscess on the same side in 1994 at a private clinic. She had no known drug or food allergies.

Family and social history

She was married and lived with her family in Buruburu in Nairobi. She was a business woman and her husband a clerk with a government ministry. Her mother was hypertensive and her father had a cardiac condition.

Obstetric and gynaecology history

She was para 2+0.

Her first delivery was by caesarean section due to cord prolapse in 1987, the baby was alive and well. Her second baby was born by spontaneous vertex delivery in 1990 and was also alive and well. She did not remember the birth weights of her two children.

Her menarche was at 14 years and her cycle was regular occurring every 28 days with 5 days flow. She gave history of having been treated for vaginal discharge 3 years prior to admission. She was currently using combined oral contraception having used an intrauterine contraceptive device for 13 years prior.

Systemic inquiry.

This was non-contributory.

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 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 4x2 cm involving the bartholins gland and labia minora on the right side. The left vulva was normal. She had some whitish discharge at the introitus. Digital examination and bimanual 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).

2. urea and electrolytes-Na 136 mmol/l

K 3.5 mmol/l

Urea 2.5 mmol/l

Creatinine 58 umol/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 0.6 mg and pethidine 100mg intramuscularly half an hour before the operation. She was then wheeled to theatre for marsupialisation.

Marsupialisation.

In theatre, general anesthesia was induced and she was placed in the lithotomy position. A vulvo-vaginal toilet was done, then she was catheterized and bladder drained of clear urine. After sterile draping of the vulval area, examination under anesthesia 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 made over the abscess along the mucocutaneous junction just lateral to the hymenal ring.

About 10mls of foul smelling pus was drained (a sample for culture and sensitivity was taken.) the cavity of the abscess was explored and locules

broken with a finger and all the pus was 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 reversed from ananesthesia.

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 gentamicin 80mg 8 hourly for the first 24 hours after the operation. She was given ibuprofen 400mg three times daily for pain relief. The vulval pack was removed after 12 hours.

24 hours after the 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 three times a day, amoxicillin/clavulanic acid 1 g twice daily for two weeks and ibuprofen 400mg 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 smears and to continue visiting the family planning clinic.

Discussion

The case is presented of a 46 years old para 2+0 who presented with ecurrent bartholi's abscess and was treated by marsupialisation. She was well at 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 0.5x1.0 cm in diameter. They are the major vesibular glands and lie under the constrictor muscle of the vagina. They are partially covered by the vestibular bulbs. The gland ducts are 1.5-2.0 cm 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 46 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 have probably have been due to scarring after the initial procedure causing obstruction of the outflow tract for the gland secretions. Bartholins 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 Bartholins gland infection and its complications in women. It is commonly associated with women 20-29 years of age, multiparity an dlow socio-economic status[1]. Bartholins abscess is not related to ethnicity or marital status. These women often have prior or concomitant sexually transmitted infection (STI) particularly gonorrhea and chlamydial infection. The demographic characteristics have led to the conclusion that acute

bartholins 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 bartholins abscess at KNH was found to be 1.9% with mean occurrence at age 22.5 years. Approximately 1.7% of acute gynaecology 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 Bartholins 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 gonorrhea, E.Coli, proteus, streptococci faecalis, staphylococcus aureus trichomonas vaginalis candida albicans and chlamydia[6]. Culture results were not gotten back for our patient due to logistical issues. Our patient presented with four day history of vulval pain and associated swelling. Bartholins abscess usually presents as an acute gynaecological problem with symptoms of severe pain, tenderness, difficulty in sitting and walking, dyspareunia and swelling of the vulva. There may be purulent discharge and obvious signs of local inflammation. Our patient had pain, swelling and difficuty 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 constitutiona symptoms.

This patient had marsupialization of the abscess. In addition she was started on antibiotics and analgesics for pain relief. The principles of management of bartholins abscess include 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 have 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 window operation and laser surgery[7,8,9].

There are four main goals for the surgical treatment of Bartholins abscess. First there must be surgical drainage of the infected gland and abscess. Secondly 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 necrotizing 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 advised to have saline sitz bath three times daily to ensure cleanliness around the operation site.

Marsupialisation is the commonly used techniques involving making a new ductal orifice. The abscess contents are drained and the walll 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 analgaesics. 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 a similar procedure 13 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 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 bulb. 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 remains the mainstay of treatment.

References

1. Aghajamian A, Bernstein L, Grimes D.A. Bartholins duct abscess and cyst; a case control study. South-Med J. 1994; 87:26.
2. Brook I. Anaerobic and aerobic microbiology of Bartholins abscess. Surg. Gynaecol Obstetrics 1989; 169:32.
3. Lee V.H. RanKen J.J, Alpert et al. Microbiological investigations of Bartholins abscess and cyst. Am J. Obstet Gynaecol. 1977; 129:150.
4. Mumia J.A. Bartholins abscess at Kenyatta N. Hospital. M.Med thesis U.O.N. 1981.
5. Ndede F. A six months survey of Bartholin's abscess at K.N.H. M.Med Thesis U.O.N. 1990.
6. Hankins, Clark, Cunningham. Surgical treatment of lower genital tract infections; in Operative obstetrics 1995 17:292-299.

7. Joo Yein Cho, Mycoceng D.A, Kyung S.C. Window operation; an alternative treatment method for Bartholin's gland cyst and abscess. Obstet Gynaecol 1990; 76:886.

8. Oliphat M, Anderson G.V. Management of Bartholin's duct cyst and abscess. Obstet Gynaecol. 1960 16:476.

9. Davis G.D. Management of Bartholin's duct cysts with CO_2 laser. Obstet. Gynaecol. 1985; 65:279.

10. Horowtz I.R, Buscemma J. surgical conditions of the vulva. In Te Linde's operative gynaecology 8th edition pp 891:1997.

11. Markusen T.E. and Barclay D.L. In current obstetric & gynecologic diagnosis & treatment. Ninth edition. McGraw Hill. 2003. pg 672.

GNAECOLOGY CASE 6

VESICOVAGINAL FISTULA: SUCCESSFUL VAGINAL REPAIR

NAME: N.W. IP NO: 0905162

AGE: 26 YEARS D.O.A: 4/11/04

PARITY: 3+0 D.O.D: 10/11/04

PRESENTING COMPLAINTS

She was admitted to the cold gynaecological ward through the vesico-vaginal fistula (VVF) clinic with complaints of effortless leakage of urine since her last delivery five months earlier.

HISTORY OF PRESENTING COMPLAINTS

The patient was admitted through the special VVF repair program for examination under anaesthesia (EUA) and repair. She had been delivered by emergency caesarian section in 1998 after prolonged labour at Muranga Hospital to a fresh stillbirth, whose birth weight she did not know. She had been in labour for two days at home under the care of her grandmother who was a TBA and came to hospital only after realizing that she couldn't give birth. Ten days later she developed urinary but not foecal incontinence.

PAST MEDICAL HISTORY

This was not significant.

OBSTETRIC AND GYNAECOLOGIC HISTORY

She was para 3 + 0 and her last delivery was in 2004. Her first delivery was through spontaneous vaginal delivery in 1996 to a live male infant who was doing well. The second delivery was also by spontaneous vaginal delivery in 1998 to a live female infant who was also doing well. Both deliveries were at home with the help of a traditional birth attendant and the birth weights were not documented. The third was by caesarian section as

outlined above. She attained menarche at 14 years. The menses were regular with a flow of 4 days in a 28day cycle. She did not experience dysmenorrhoea. Her LMP was on 20/11/04 and had never used any contraceptives.

FAMILY AND SOCIAL HISTORY

She was a housewife with two living children. Her husband was a casual labourer and they lived together in Kangema. The couple had dropped out of school at standard five and got married soon after. There was no family history of any chronic illness. She did not drink alcohol or smoke cigarettes.

PHYSICAL EXAMINATION

She was a young lady in good general condition. She had no pallor, oedema, jaundice or lymphadenopathy. She was afebrile. Her blood pressure was 120/80mmHg; Pulse rate was 79/minute and regular. Respiratory rate was 20/minutes and the temperature was 36.8°C.

The Cardiovascular, Respiratory and Central Nervous Systems were essentially normal.

ABDOMINAL EXAMINATION

The abdomen had normal fullness and was moving with respiration. She had a midline sub-umbilical incision scar. There was no tenderness and no masses were palpable.

PELVIC EXAMINATION

The external genitalia was normal. The perineum was excoriated, wet and had an offensive ammoniac oduor. On speculum examination the anterior wall of the vagina wall had a defect about 5cm from the urethral ring, through which urine drained into the vagina. The posterior vaginal wall was

normal. The cervix was long, posterior and the os was closed. There was no abnormal vaginal discharge.

DIAGNOSIS

A diagnosis of vesico-vaginal fistula was made.

INVESTIGATIONS

1. Haemogram: Haemoglobin was 13.4g/dl, WBC 7.2 x 10^9/L and platelets 280x 10^9/L

2. Urea and Electrolytes: Bun 5mmol/L, Na^+ 134mmol/L, K^+ 4.1mmol/L

MANAGEMENT

The nature of illness and planned management was explained to the patient and informed consent obtained.

She was advised on light diet and she had an enema at 6.00p.m, the day before the operation and at 6.00am in the morning of the operation. She was starved overnight and on the day before surgery. On the operation day her pubic hair was shaved, premedication with atropine sulfate 0.6mg and pethidine 50mg intramuscularly half an hour before theatre was give.

In theatre she was given spinal anaesthesia and placed in exaggerated lithotomy position. Vulvo-vaginal toilet was done and she was draped. Excoriations were noted over the perineum, and there was no obvious leakage of urine. A dilute solution of Adrenaline and normal saline (jungle juice) was infiltrated on the right mediolateral region of the perineum and an episiotomy given to improve exposure. Auvard's speculum was introduced

into the vaginal cavity and urine noted to be leaking from a 1.5cm defect over the mid anterior vaginal wall. The defect was oval in shape about 3cm from the urethral orifice and 3cm from the cervix. The later and the rest of the vaginal were normal.

Jungle juice was also infiltrated around the fistula. Two transverse incisions were made around the fistula and the vaginal mucosa dissented away from the bladder mucosa. The inner edges of the incision were approximated with Vicryl No.2/0 interrupted mattress sutures, starting the repair at the two lateral ends and proceeding medially. Methylene blue dye was introduced into bladder through a urethral catheter and there was no leakage. The outer edges were then approximated with the same suture. A gauze pack soaked in Iodine solution was left in the vagina. A self-retaining Foleys catheter was left in situ for continuous drainage of the bladder.

POST-OPERATIVE CARE

The patient was taken to the recovery where she was observed quarter hourly till sensations were back in the lower limbs. She was then transferred to the general ward where observations continued twice daily. She was allowed to drink fluids freely and supplemented with intravenous fluid on the 1st postoperative day. Continuous catheter drainage was maintained and an input output chart instituted. She was also given pethidine 100mg intramuscular 6 hourly for 24 hours.

After 24 hours the vaginal pack was removed, intravenous fluids stopped and patient advised to drink at least 6 litres of fluids per day. She was put on oral analgesic (Brufen 400mg 8 hourly). The urine remained clear and the input output chart was satisfactory. On the third day, she was carefully ambulated with the catheter. She remained dry.

On the 7th day she was discharged home, to come again for review in the VVF clinic in a week. She was advised to take plenty of fluids and to avoid sexual intercourse.

FOLLOW UP

On the fourteenth postoperative day, she came to the VVF clinic as booked. She was reviewed and found not to be leaking. In lithotomy position, the repair site was visualized by use of a Sim's speculum and found intact. Dye test was done and no leakage found. The catheter was removed.

She was instructed to avoid sexual intercourse for at least 6 months, take plenty of oral fluids and void frequently even at night. She was subsequently seen at 2 months, 3months and 6 months. She remained well without leakage of urine and was discharged from the clinic with permission to resume sexual activity. She was counselled on contraception but declined to have any. She was instructed to come for antenatal care on first missed periods and advised on elective caesarian delivery in subsequent pregnancies.

DISCUSSION

The patient presented was a 24-year-old para 3+0 who developed vesico-vaginal fistula (VVF) following obstructed labour. She underwent successful repair through the vaginal route.

Vesicovaginal fistula (VVF) is an abnormal fistulous tract extending between the bladder and the vaginal epithelia that allows the continuous involuntary discharge of urine into the vaginal vault. The fistula leads to constant seepage of urine through the vagina down the patient's legs, wetting and soiling clothes. It is a severely demoralizing and disabling injury among women and due to the accompanying smell, most communities consider these women as outcasts (1, 2).

Fistula is most common in poor communities in sub-Saharan Africa and South Asia where access to or use of obstetric care is limited. Good data on fistula are scarce. In 1989, the World Health Organization estimated that more than two million women remain untreated in developing countries and that at least 50,000 to 100,000 new cases occur each year. But the secrecy and shame that surround the condition make it difficult to get a reliable estimate of its prevalence. United Nations Fund for Population (UNFPA) funded campaign to eliminate VVF, suggest the above figures are an under-estimation and the true ones are much higher. In fact, WHO experts have also estimated that in areas of high maternal mortality, two to three women per 1,000 pregnancies develop fistula, which would mean that the prevalence is likely much higher than the 1989 estimates (3). Our patient was a housewife and the husband was a casual labourer. Though their annual income was not known, it is expected to be low like most farm labourers. She had not attended any antenatal care and had delivered at home previously though there was a health centre nearby. The reason for failure to go to hospital was not certain.

Causes of vaginal fistulae vary between developing and developed world. In the developed world, VVF is mostly due to gynaecological operations, neoplasm, radiation therapy and rarely operative vaginal deliveries. In the developing countries obstructed labour is the leading cause, contributing 87-92% of all VVF in Kenyatta National Hospital. A study in 1983 (4) found that surgical trauma accounts for about 53% of non-obstetrical VVF. Passage of instruments into the vagina as in criminal abortion, accidental trauma and rape also contributed. The estimated prevalence in Africa is 2-5 per 1000 deliveries (5,6). Obstetric fistulae are mainly due to devitalization of tissue through pressure of obstructed labour. However some arise from

operative delivery both vaginal (vacuum and forceps delivery) and abdominal (5). The patient presented had obstructed labour.

Diagnosis of vesicovaginal fistulae is not difficult. There is history of incontinence of urine following difficult labour, pelvic surgery or radiotherapy. Physical examination is a very important component of VVF management. The aims of examination are to; confirm presence and number of fistulae, determine the location, size and associated fibrosis. Presence of other problems that may hinder surgery such as anaemia, malnutrition and renal complication should also be noted. Examination is in two stages with the first stage being done in the clinic. During this first encounter in the clinic, general and vaginal examinations are done while the second examination is done under anaesthesia on the day of surgery (1,5). The patient presented had the first examination in the clinic before admission and the second in theatre during repair.

During examination in the clinic, speculum examination may reveal all large fistulae. Injection of methylene blue dye into the bladder may assist to visualize the smaller fistulae. The three-swab test is performed when dye injection under direct visualization is not diagnostic. During this test, three swabs (or a tampon) are put in the vagina; one next to the cervix, the second in the middle and the last in the lower third. Dye is then injected into the bladder with a catheter and patient asked to walk around for 15-20minutes before the swabs are removed and inspected. If the lower swab is stained, then the fistula is likely to be urethro-vaginal. If only the middle one is stained, then the injury is either high urethro-vaginal or vesico-vaginal. If the highest swab is wet with dye, the injury is vesico-vaginal or vesico-uterine and if wet with clear urine, the uretero-vaginal fistula is confirmed. Intravenous urogram may be performed when ureteric fistulae are suspected and cystoscopy can show bladder fistulae. These are however only

recommended for cases that evade diagnosis on examination (5). In our patient diagnosis was made by speculum examination alone

There are two types of classification that are commonly used for vesico-vaginal Fistulae. These are:

The first type is anatomic/physiologic classification: This is the most common and has bearing on surgical technique and outcome of surgery. In this classification, fistulae fall into I-III with II being further divided into capital A and B each of which is further divided into small a and b (1).

I. Fistula not involving the bladder closing mechanism. Usually those above 5cm from the urethral opening.

II. Fistula involving the bladder closing mechanism. Usually fistulae within 5cm from the urethral opening.

A. No urethral involvement;

a) Without circumferential defect,

b) With circumferential defect

B. With (sub) total involvement of the urethra;

a) Without a circumferential defect

b) With a circumferential defect

III. Refers to miscellaneous fistulae such as uretero-vaginal fistulae.

The second and less utilised classification is based on size of the fistulae as follows

Small: < 2cm

Medium: 2-3 cm

Large: 4-5cm

Extensive: 6 cm

The patient presented had class IIAa fistula.

Surgery is the main stay of repair of obstetric fistulas. Successful repair of pressure necrosis fistulas require meticulous surgery since scarring and tissue loss, which is usually present requires wide mobilization of the bladder defect to facilitate closure in layers without tension. Previously, a 3-month wait is recommended to allow for tissue reaction to subside before surgery. A catheter inserted immediately obstructed labour is detected promotes spontaneous healing of small fistulas (up to 2cm). This may occur in up to 60% of patients (7)

Currently, repair is undertaken as soon as the fistula edges are clean and the patient's condition is good enough. Its high success rate is comparable to, indeed slightly better than that of normal VVF repair at first intention. This is because the time the slough disappears is within the physiological time of wound healing process before fibrosis and scarring develops. It also prevents the psychological trauma these women undergo as they wait (7). Our patient underwent late repair not because of medical recommendation but because of late presentation. This is not unusual as a study done by UNFPA showed that many fistula sufferers are either unaware that treatment is available or cannot access or afford it (8).

During repair, the site of the fistula determines the approach. Most of the fistulas due to obstetrical conditions are easily repaired by a trans-vaginal approach. Type of anaesthesia depends of the facilities. Spinal anaesthesia is preferable for trans-vaginal approach. It is encouraged in the third world where supply and maintenance of general anesthesia equipment is poor (1,2). The patient presented had a successful trans-vaginal repair under general anaesthesia.

The aims of VVF repair are; fistula closure and attainment of urinary continence and preservation and restoration of sexual function. Some of the methods used in repair include; flap sliding, grafting of tissue e.g. omental tissues or skin flap and use of gracilis muscle from the thigh. The aim is to have a closure without tension on the suture line (5,6).

Postoperative care includes high volumes of un-interrupted urine flow to prevent ascending infection and blockage of the catheter. The bladder stays empty for at least 10 days. Laxatives help to prevent straining when passing stool. Most importantly maintain perineal hygiene. Routine antibiotics are not encouraged but should be given where there is evidence of infection (1). Study by Tomlinson found no difference between those people given prophylactic antibiotics and those not given (9).

Follow up for 6 months when patient should avoid sexual intercourse is important. Any future deliveries should be by elective caesarian section. Complications of VVF repair include blocked catheters, urinary infection, vaginal and bladder haemorrhage, ureteric obstruction and breakdown of the repair. If repair fails, another attempt should be done at least 2 months later so as the tissue are in suitable state (1). S.W. has not come again since the last visit at the end of six months followup. It is not known whether she has failed to conceive or did so but went to her primary hospital for ANC.

Prevention, rather than treatment, is the key to ending fistula menace. Making family planning available to all who want to use it would reduce maternal disability and death by at least 20 per cent. Addressing social issues that contribute to poor girl health hence obstructed labour such as early pregnancy, girls' education, poverty and women empowerment are important areas of intervention. Complementing that with skilled attendance at all births and emergency obstetric care for those women who develop

complications during delivery would make fistula as rare in the developing as it is in the developed world (3).

REFERENCES

1. Waaldijk K. Step-by-Step Surgery of VVF. A full colour Atlas 1994. Companion Pres, Edinburgh

2. Riley V.J., Spurlock J, Garris J.B., Taravera F. Vesicovaginal Fistula. eMedicine.com, Inc 2005

3. UNFPA Vesicovaginal Fistula campaign progress update. August 2002

4. Orwenyo E. A. A retrospective Study of 166 cases of acquired urinary genital and / or rectovaginal fistula treated at Kenyatta National Hospital during 1978-1983: M.med Thesis, University of Nairobi, 1983

5. Meeks G.R., Roth T.M. Vesicovaginal and urethrovaginal fistulas. Te Lindes Operative Gynecology 9th Ed.2004. Lippincott William and Wilkins Philadelphia. **36**:507-533

6. Ojanuga O, Ekwempu C. An investigation of Socio-Medical risk factors associated with vaginal fistula in Northern Nigeria. Women and Health **28(3)** 103-16 1999

7. Waaldijk K. Intermediate surgical management of fresh obstetric fistula with catheter and /or early closure. Internal Journal Obstet Gynaec. 1994; **45** (1):11.

8. Assessment of extent of VVF and need for intervention. UNFPA. Needs assessments <http://www.unfpa.org/fistula/docs/index.htm>

9. Tomlinson A.J., Thornton J.G., A randomized controlled trial of antibiotic prophylaxis for vesico-vaginal fistula repair. Br J Obstet Gynaecol 1998 Apr;**105(4):** 397-9 [Medline]

<http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Abstract&list_uids=98270427>. e

GYNAECOLOGY CASE 7

OVARIAN CANCER - LAPARATOMY AND CHEMOTHERAPY

NAME: E.W IP NO. 0999218

AGE: 56 YEARS D.O.A 31/1/05

PARITY: 7+0 D.O.D 12/6/05

PRESENTING COMPLAINT

She was admitted with six months history of abdominal pain and swelling.

HISTORY OF PRESENTING COMPLAINTS

She had been fairly well until 6 months before when she started having low abdominal discomfort that was later followed by swelling. The abdominal swelling was progressive and was associated with abdominal pain that was radiating to her back. She did not have any bleeding per vagina but she progressively lost weight, despite having normal appetite.

PAST MEDICAL HISTORY

She had been hospitalised for obstetric reasons only, had never undergone any major surgery and was not know to suffer from any chronic illness.

OBSTETRIC AND GYNAECOLOGIC HISTORY

She was para 7+0. All were spontaneous vertex deliveries at term. Her menopause occurred four years prior to admission. Her menarche was at 15 years. Her periods had been regular occurring every 30 days and lasting four days. She had never used any contraceptives.

FAMILY AND SOCIAL HISTORY

She was a housewife married to small scale farmer. Her aunty had died after surgery for what she was told was breast cancer. There was no family history of abdominal cancer. She did not smoke cigarettes or drink alcohol.

PHYSICAL EXAMINATION

She was in fair general condition with no pallor, jaundice or fever. She had no lymphadenopathy but was mildly wasted. Her blood pressure was 130/80mmHg, pulse rate 82/minute and her temperature was 36.2°C.

Central nervous, cardiovascular and respiratory systems were essentially normal.

ABODOMINAL EXAMINATION

The abdomen was markedly and uniformly distended. There were no surgical scars and no visible blood vessels. The umbilicus was flat and had no nodule. There was a mass in the left lumbar region that was firm, mobile and non tender. There was no tenderness, rigidity or guarding but fluid thrill was elicited.

VAGINAL EXAMINATION

She had normal external genitalia. The cervix was firm, posterior and os was closed. The uterus was of normal size and freely mobile. Adnexia and the pouch of Douglas felt full. There was no blood or discharge on examining finger.

DIAGNOSIS

A tentative diagnosis of ovarian tumour was made.

INVESTIGATIONS

- Haemogram: Haemoglobin was12.4g/dl, WBC 8.4X10/L and Platetlets 559X10/L
- Urea and Electrolytes: Na^+ was143mmo1/l, K^+4.5mm01/l, BUN 8.55mmo1/l and Creatinine 117umo1.
- Liver Function Tests: Aspartate transaminase was 32mmol/l, Alanine transaminase 26mmol/l, total protein 73g/l and albumin 35g/l.

PHYSICAL EXAMINATION

She was in fair general condition with no pallor, jaundice or fever. She had no lymphadenopathy but was mildly wasted. Her blood pressure was 130/80mmHg, pulse rate 82/minute and her temperature was 36.2°C.

Central nervous, cardiovascular and respiratory systems were essentially normal.

ABODOMINAL EXAMINATION

The abdomen was markedly and uniformly distended. There were no surgical scars and no visible blood vessels. The umbilicus was flat and had no nodule. There was a mass in the left lumbar region that was firm, mobile and non tender. There was no tenderness, rigidity or guarding but fluid thrill was elicited.

VAGINAL EXAMINATION

She had normal external genitalia. The cervix was firm, posterior and os was closed. The uterus was of normal size and freely mobile. Adnexia and the pouch of Douglas felt full. There was no blood or discharge on examining finger.

DIAGNOSIS

A tentative diagnosis of ovarian tumour was made.

INVESTIGATIONS

- Haemogram: Haemoglobin was12.4g/dl, WBC 8.4X10/L and Platetlets 559X10/L
- Urea and Electrolytes: Na^+ was143mmol/l, K^+4.5mm01/l, BUN 8.55mmol/l and Creatinine 117umol.
- Liver Function Tests: Aspartate transaminase was 32mmol/l, Alanine transaminase 26mmol/l, total protein 73g/l and albumin 35g/l.

- Ultrasound of the abdomen and pelvis showed a tubo ovarian complex mass measuring about 14cmx14cm, that was irregular. Uterus, liver, kidneys and spleen normal but there was moderate amount of ascitis.
- Chest x-ray was normal.

MANAGEMENT

The diagnosis and planned mode of management were explained to the patient and informed consent obtained. Two units of compatible blood were obtained and reserved for the operation. Three days before the operation, she was started on dulcolax 2 tablets every evening and fluid diet. On the eve of surgery she was fasted from midnight and given 2g of metronidazole and an enema at 6pm. At 6 am on the day of the operation enema was repeated and half an hour before theatre she was premedicated with 0.6mg of atropine sulfate and 50mg of pethidine intramuscularly before being wheeled to theatre.

In theatre, the patient was put under general anaesthesia. In semi-lithotomy position, vulvo vaginal toilet was done and bladder aseptically catheterized. Patient was then repositioned in the supine position and abdomen cleaned and draped. The abdomen was opened in layers through a midline incision.

An about one and half litre of ascitic fluid was drained and a sample was sent for cytology. The fluid was straw-coloured and not blood stained. There was a large right ovarian mass that measured about 13cm by 14cm with the right fallopian tube buried in the tumour. The omentum was adherent to the mass and the right fallopian tube. The left tube and ovary appeared normal. The uterus was normal and there were no adhesions or obvious seedlings to the gut. There were multiple seedlings on the surface of the liver, and peritoneum but the diaphragm appeared free of seedlings.

The largest seedling was less than 2cm and biopsy was taken from them. Total abdominal hysterectomy, bilateral salpingoophoroctomy and omentectomy were done. All the specimens were submitted for histology. Intra-operation diagnosis of cancer of ovary stage IIIB was made. The abdomen was closed in layers and patient reversed from anaesthesia.

POST OPERATIVE CARE

The vital signs were observed half hourly until she was fully awake and then transferred to the ward. She was put on intravenous fluids of normal saline alternating with 5% dextrose about 3 litres in 24 hours. She was also started on intravenous crystalline penicillin 2mu 6 hourly and gentamycin 80mg 8 hourly for 48 hours. For pain, she was given intramuscular pethidine 100mg 8 hourly for 24 hours.

On the first post-operative day bowel sounds were present and she was started on oral sips. On third post-operative day, she was on full ward diet, and was started on oral medication. On the seventh post-operative day, the wound was nearly healed and the stitches were removed.

The histology report showed a papillary serous cystadenocarcinoma of the ovary with involvement of the omentum. The report was explained to the patient and need for chemotherapy was discussed. Blood was taken for pre-chemothrapy baseline investigations which included haemogram, urea and electrolytes, and liver funtion tests and these were found to be within normal limits. Her weight was 60kg and her height 1.5m. Her body surface area was 1.55m sq. She was started on cisplatin, adriamycin and cyclophosphamide. She received chemotherapy in courses. Each course consisted of cisplatin at 75mg and andriamycin 50mg stat and cyclophosphamide at 500mg once a day for 5 consecutive days.

Before the drugs were administered, she was first rehydrated with 1000mls of normal saline and she was given 10mg metoclopromide intravenous to combat drug-induced nausea. She was given 6 courses of chemotherapy at 3 weeks interval. She was paroled in between courses of chemotherapy to come back a day before the next course with results of baseline investigations ready for the next course.

She was still being followed up in GOPC and was doing well.

DISCUSSION

The patient presented was a 56year old para 7+0 who was admitted with cancer of the ovary. Total hysterectomy with bilateral salpingoophoroctomy was done. Histology revealed moderately well differentiated papillary cystadenocarcinoma with metastasis to the omentum and peritoneum. She did well on chemotherapy

Cancer of the ovary constitutes 10-15% of genital tract malignancies. It is more common in the US but less common in the Asia and Latin America (1,2). In Kenya it is the third commonest female genital tract malignancy after cancer of the cervix and choriocarcinoma. It constitutes 8% of all female genital tract malignancies seen at KNH (3). Karanja reported an incidence of 9.4% in 2003 and reported change of position to second after cancer of the cervix in KNH (4). The incidence of ovarian cancer increases with age. Indeed, the average patient is 50-59 years old with a peak at 60-65 years (5). The patient presented was at 56years.

Other factors that are associated with a high risk of cancer of the ovary are; diet rich in saturated fat, family history of cancer of the ovary, breast, or endometrium or personal history of cancer of the breast or endometrium, living in the industrialized countries, nulliparity or late onset of fertility, use of clomiphene citrate to induce ovulation, use of talc for vulval hygiene and

Caucasian race. Recent studies have however disputed the role of clomiphene. Endometriosis and viral infections like mumps have also been implicated. Factors associated with reduced risk of the cancer include; early onset of fertility, multiparity, breastfeeding, lower socio-economic status, blood group O, Japanese, Hispanic, Chinese and black race. Use of oral contraceptive, tubal ligation and hysterectomy also have a protective role. 95% are sporadic with only 5% showing familial tendency (1, 2, 5).

E. W. was 56years at diagnosis, had given birth to her first baby at the age of 19years and was a mother of seven. She had breastfed all her babies for more than two years. She had not used any kind of contraception and her Aunt had died of breast cancer.

Ovarian cancer can be divided into three main classes; namely; epithelial, germ cell and sex cord tumours. Epithelial tumours make up 90% of ovarian cancer. Epithelial malignant tumours can be divided into serous, mucinous, endometroid, clear cell, transitional and undifferentiated carcinomas based on cell type. Serous tumours are the commonest (35-55%) and are bilateral in 60% at diagnosis. Mucinous tumours are second causing 10-20% of ovarian tumours but only 10% of malignant cases. They are bilateral in 10% at diagnosis. Endometroid tumours account for about 5% of ovarian neoplasm but malignant disease accounts for 20% of ovarian cancers. They are bilateral in 30-50% at presentation. Clear cell, transitional and undifferentiated tumours account for the remaining 15-20% (1, 5). Germ cell malignancies consist of dysgerminomas, endodermal sinus tumours, embryonal carcinoma, polyembryoma, choriocarcinoma and immature teratomas. Dysgerminomas are the commonest in this class accounting for 45% of malignant germ cell tumours (5). In Kenyatta National Hospital, epithelial tumours account for 71% of all ovarian malignancies (4, 6)

E.W. suffered from moderately well differentiated papillary cystadenocarcinoma: an epithelial malignant tumour.

The symptoms of ovarian cancer are nonspecific and often suggest the presence of upper abdominal disease and usually experienced in advanced disease. Patients may report abdominal fullness, dyspepsia, early satiety, or bloating as the result of increased abdominal pressure from ascites or involvement of the omentum. Occasionally, patients with early-stage disease present with pelvic pain due to ovarian torsion, although most patients with early-stage disease are asymptomatic. Physical findings are diverse and typically include a palpable ovarian mass. In this regard, ovarian cancer should be considered in any premenopausal woman with an unexplained enlargement of the ovary or any postmenopausal woman with a palpable ovary. Other findings on physical examination may include, pleural effusions, and an umbilical mass referred to as a Sister Mary Joseph's nodule. Such umbilical masses are rare and nonspecific; they can be associated with gastric, pancreatic, gallbladder, colon, and appendiceal cancers.The most common extraabdominal site of disease is the pleural space, although lung parenchymal involvement may be observed on occasion (1, 2, 5).

Paraneoplastic phenomena include humorally mediated hypercalcemia with clear-cell histologic findings (7) as well as subacute cerebellar degeneration associated with anti-Purkinje-cell antibodies (8). The Leser-Trélat sign is characterized by the sudden appearance of seborrheic keratoses and, on rare occasions, has been reported to herald the development of ovarian cancer. Trousseau's syndrome: migratory superficial thrombophlebitis, palmar fasciitis, dermatomyositis, and polyarthritis have also been observed (9)

If ovarian cancer is suspected on the basis of symptoms and physical examination, abdominal and or transvaginal ultrasonography is performed

for further evaluation of the pelvis. Transvaginal ultrasonography appears to be more sensitive than computed tomographic (CT) scanning for the detection of pelvic masses, and it provides qualitative information about the mass that is useful for further management decisions, Specifically, the finding of a complex ovarian cyst, defined by the presence of both solid and cystic components, sometimes with septations and internal echoes, is highly suggestive of cancer (10). Such cysts typically require surgery for definitive diagnosis. Percutaneous biopsy of complex cysts is to be avoided, given the risk of tumor spillage into the pelvic cavity. Simple ovarian cysts have smooth walls, are filled with fluid, and do not contain a solid component. These cysts are often benign and generally do not require immediate surgical intervention, although careful follow-up is recommended (1).

Although the serum CA-125 level is elevated in more than 80 percent of patients with advanced epithelial ovarian cancer, this measurement alone is neither sufficiently sensitive nor specific enough to be diagnostic. Elevated serum CA-125 levels may be associated with various conditions, such as pregnancy, endometriosis, adenomyosis, uterine fibroids, pelvic inflammatory disease, menstruation, and benign cysts. The serum CA-125 level may also be elevated in other malignant conditions, such as pancreatic, breast, lung, gastric, and colon cancers. Thus, measurement of the CA-125 level is not usually helpful in the preoperative evaluation of a complex ovarian cyst, and surgery is generally necessary for definitive diagnosis. However, a serum CA-125 level of more than 65 U per milliliter in a postmenopausal woman with an abdominal or pelvic mass should raise the possibility of ovarian cancer. CA-125 level however is useful in assessing the patient's response to postoperative chemotherapy and in detecting early relapse in patients who have already received a diagnosis of ovarian cancer (11)

E. W. had presented with 6months of low abdominal pain and abdominal swelling of the same duration. She had a clinically palpable mass and ascitis and a pelvic ultrasound which showed a complex pelvic mass and ascitis but CA-125 was not done either for diagnosis or followup because of financial constraints.

Ovarian cancer spreads through various routes which include: Seeding of cancer cells to the peritoneum, diapharagm, tubes and uterus, lymphatic to the paraortic nodes and umbilicus, blood stream to the lower vagina and elsewhere and direct spread to any neighbouring organ or tissue (5). E.W. had disease that at diagnosis involved both ovaries and the right fallopian tube with metastasis to the omentum and the peritoneum.

All patients suspected to have ovarian malignancy are managed by laparotomy for histologic confirmation, staging and debulking. Chemotherapy and or radiotherapy can be considered based on stage of disease and histological type therafter (1,2).

Staging is based on clinical, surgical, histologic and pathologic finding including cytologic testing of effusion and peritoneal washings. Staging is based on FIGO staging system shown below (1, 2, 5):

Stage I: Growth limited to the ovaries.

A; cancer limited to one ovary no ascitis no extension to the external surface with

an intact capsule.

B; Growth limited to both ovaries, no ascitis, no tumour on the external surface

and intact capsule.

C; Tumour either stage IA or IB, but involves the external surface of the ovary,

or capsule is rupture, or ascitis is present containing malignant cells or has

positive peritoneal washings.

Stage II:Growth involving one or both ovaries with pelvic extensions.

A; Extension/metastasis to the uterus or fallopian tubes

B; Extension to other pelvic tissue.

C; Tumour either stage IIA or IIB, but involves the external surface of the ovary,

or capsule is rupture, or ascitis is present containing malignant cells or has

positive peritoneal washings.

Stage III: Tumour involving one or both ovaries with peritoneal implants outside the

pelvis and/or positive retroperitoneal or inguinal nodes. Superficial liver

involvement is also stage III disease. Tumour limited to the true pelvis but

with histologically proven involvement of the small bowel or omentum

A; Tumour grossly limited to the true pelvis with negative nodes but

Histologically confirmed microscopic involvement of abdominal peritoneal

surface.

B; Tumour involving one or both ovaries with Histologically confirmed

involvement of abdominal peritoneal surfaces with none exceeding 2cm in

diameter. Retroperitoneal and inguinal nodes are negative.

C; Tumour greater than 2cm in diameter and or positive retroperitoneal or

inguinal lymph nodes.

Stage IV: Tumour involving one or both ovaries with distant metastasis. If pleural

effusion is present, there must be positive cytology to alot it stage IV.

Parenchymal liver involvement is allotted stage IV

Histologic confirmation is necessary to rule out other causes of a complex ovarian cyst, including metastatic disease to the ovary from another primary site (e.g., Krukenberg's tumors), or benign conditions, such as an endometriotic cyst (12). Surgical staging, performed during exploratory laparotomy, provides important information that can guide postoperative decision making, especially for early-stage disease. Finally, tumor debulking (primary cytoreduction) is a valuable component of initial surgery, since patients with residual tumor 1 cm or less in diameter have higher survival rates than those with more extensive residual disease (13).

The standard surgical approach involves a vertical midline incision to permit adequate exposure of the upper abdomen and pelvis. A total abdominal hysterectomy and bilateral salpingo-oophorectomy are typically performed, along with careful examination of all peritoneal surfaces, omentectomy,

biopsy of para-aortic lymph nodes when appropriate, random biopsies of clinically uninvolved areas, and peritoneal washings. Biopsy of the para-aortic nodes is especially important in patients with disease that otherwise appears to be limited to the ovary, since such patients may have more advanced disease (1).

Rates of long-term survival among patients with early-stage disease (stage I or II) can be as high as 80 to 95 percent, whereas patients with advanced disease (stage III or IV) have lower survival rates of between 10 to 30% (14).

Although initial surgery is almost always necessary in the management of suspected ovarian cancer, it is important to recognize at least two groups of patients for whom alternative approaches might be considered. Clinical suspicion of a Krukenberg's metastasis from a gastric or other gastrointestinal primary site should prompt an initial endoscopic evaluation before it is determined whether a surgical procedure might be necessary (5).

Patients with suspected ovarian cancer who are poor candidates for surgery because of a coexisting disease that precludes the performance of a safe cytoreductive procedure may benefit from neoadjuvant chemotherapy. In this case, 3 cycles of chemotherapy (platinum based and paclitaxal) are administered after confirming diagnosis either through open biopsy of accessible metastasis or cytology of effusions or ascitis before debulking is attempted (15).

Most patients with cancer of the ovary present at advanced stage namely stage III and IV and are candidates for radical surgery (hysterectomy and bilateral salpingo-ophorectomy, omentectomy and debulking of bulky metastasis) followed by chemotherapy. Controversy exists on the need for and the duration of chemotherapy for patients with early disease. Patients with low risk of recurrence, Stage I A and B have 90-95 5year survival on surgery alone. Patients with high risk of recurrence namely the stage IC-IIC

disease are candidates for chemotherapy. The question is how many courses they should recieve 3 or 6. A randomized trial conducted recorded similar survival but more disease recurrence with three cycles compared to six of carboplatin and paclitaxal. Patients with stage IA disease who wish to retain their fertility can be treated by unilateral salpingo-ophorectomy and followup (16).

E.W. was in the advanced disease category and needed and received six courses of cisplatin and cyclophasphomide, which are the ones available in our institution due to high cost of the taxols and carboplatin.

Irradiation is limited to stage I and II due to difficulties of enough tumoricidal doses to the whole abdomen without destroying the other sensitive intraperitoneal organs. However small local recurrence tumors can also be irradiated upto 60gy. Other adjuvant therapy include radioactive gold and phosphorous (1,2).

E.W. fell in the category that wouldn't have benefited from irradiation but is still on followup and if develops local recurrence will be considered for radiotherapy.

REFERENCES

1. Doligo O., Vicki V.B. Premalignant and malignant disorders of the ovaries and the oviduct: Current Obstestric and Gynaecologic Diagnosis and Treatment 9th Ed. 2003; McGraw-Hill New York **49**:933-958

2. Creasman W.T. Ovarian cancer: etiology, screening and surgery: Te Linde's Operative Gyanecology, 9th Ed 2001; Lippincott-Raven publishers **28**: 675-704

3. Njuki M. Primary Ovarian Malignancy. Presentation at KNH: M.med Thesis, 1979; UoN

4. Karanja D.K. characteristics of patients with cancer of the ovary as seen at KNH: M.med Thesis, 2003;UoN.

5. Stenchever M.A., Droegenmueller W., etal. Comprehensive gynecology 4th Ed 2001: Mosby Philadelphia **23**:707-739

6. Ojwang S. B. O. Makokha A.E. Sinei S.K. Ovarian cancer in Kenya: East Afri. Medical journal, 1980; **57**:2.

7. Tsunematsu R., Saito T., Iguchi H., etal. Hypercalcemia due to parathyroid hormone-related protein produced by primary ovarian clear cell adenocarcinoma: case report: Gynecol Oncol 2000;**76**:218-222.

8. Furneaux H.M., Rosenblum M.K., Dalmau J. Selective expression of Purkinje-cell antigens in tumor tissue from patients with paraneoplastic cerebellar degeneration: N.Engl J Med 1990;**322**:1844-1851.

9. Raffel G.D., Gravallese E.M., Schwab P. etal. Diagnostic dilemmas in oncology: case 2: dermatomyositis and ovarian cancer: J Clin Oncol 2001;**19**:4341-4343.

10. VanNagell J.R. Jr, Ueland F.R.. Ultrasound evaluation of pelvic masses: predictors of malignancy for the general gynecologist. Curr Opin Obstet Gynecol 1999;**11**:45-49.

11. Meyer T., Rustin G.J. Role of tumour markers in monitoring epithelial ovarian cancer. Br J Cancer 2000;**82:**1535-1538.

12. Petru E., Pickel H., Heydarfadai M. Nongenital cancers metastatic to the ovary. Gynecol Oncol 1992;**44**:83-86.

13. Bristow R.E., Tomacruz R.S., Armstrong D.K.,etal. Survival effect of maximal cytoreductive surgery for advanced ovarian carcinoma during the platinum era: a meta-analysis: J Clin Oncol 2002;**20**:1248-1259.

14. McGuire W.P., Hoskins W.J., Brady M.F. Cyclophosphamide and cisplatin compared with paclitaxel and cisplatin in patients with stage III and stage IV ovarian cancer. N Engl J Med 1996;**334**:1-6.

15. Van der Burg M.E., van Lent M., Buyse M. The effect of debulking surgery after induction chemotherapy on the prognosis in advanced epithelial ovarian cancer: N Engl J Med 1995;**332**:629-634.

GYNAECOLOGY CASE 8

POLYCYSTIC OVARY DISEASE: LAPAROSCOPIC OVARIAN DLILLING: FERTILITY RESTORATION

NAME: A.W. IP NO. 1023860

AGE: 32 YEARS DOA: 17/05/06

PARITY: 0+0 DOD: 20/05/06

PRESENTING COMPLAINT

Inability to conceive for four years

HISTORY OF PRESENTING COMPLAINT

She was admitted through the infertility clinic where she had been followed up for six months with four year history of inability to conceive. She had been married for the last four years and lived together with her husband. She gave a history of having unprotected coitus three or four times a week. Coitus was not painful and the husband's erection and penetration was satisfactory. She had used clomiphene citrate for three consecutive months without success.

PAST MEDICAL HISTORY

The couple had no history of chronic drug ingestion, never suffered from sexually transmitted diseases, endocrine or any other chronic illness. Neither of them had other sexual relationships before or after marriage. She had never suffered from pelvic inflammatory disease or tuberculosis and had not had any pelvic surgery. The husband had no history of mumps and had not had any genital tract operations.

She had no history of headache, blurring of vision or galactorrhoea.

OBSTETRIC AND GYNAECOLOGIC HISTORY

She was nulliparous. She attained menarche at 15 years and her periods had been irregular and painless lasting 4-7 days with a cycle of 30-60days. The flow was occasionally very heavy accompanied by clots. Her last menstrual period was 8/05/06. She had never used any contraceptive method before.

PHYSICAL EXAMINATION

She was in good general condition. She was not pale or jaundiced and had no peripheral oedema or lymphadenopathy. The thyroid gland was not enlarged and the breasts were normal in size and shape. She had no galactorrhoea but had hirsutism with a beard, coarse hair on the arms and legs and a masculine eustecheon. She was 161cm tall and weighed 76kg. Her body mass index was 29.3kg/m^2. Her Blood Pressure was 110/70mmHg, Pulse rate of 78/minute, regular. The respiratory rate was 18/minute and her temperature was 36.6^{o}C.

The Respiratory, Cardiovascular and Nervous Systems were essentially normal.

ABDOMINAL EXAMINATION

The abdomen was scaphoid, moved with respiration and had no obvious scars or masses.

There were no areas of tenderness and the spleen and liver were not palpable.

PELVIC EXAMINATION

She had clitoromegally in otherwise normal external genitalia. The cervix was firm, long and regular and the os was closed. The uterus was anteverted, mobile and of normal size. Both ovaries felt enlarged but were mobile and non-tender. There was normal discharge on examining finger.

INVESTIGATIONS

1. Pelvic ultrasound scan: Normal uterine size and shape. Endometrial strip thickened to 8mm.There are multiple bilateral ovarian cysts with a volume of 8ml. No fluid in the Pouch of Douglas.

2. Hysterosalpingogram: Normal Uterine Cavity, both tubes well outlined with dye. Bilateral free peritoneal spill demonstrated.

3. Semenalysis was normal with a count of 60million per millilitres and a progressive count of 60% both rapid and slowly progressive.

4. Prolactin level was normal.

5. Thyroid function tests were normal

6. Lutenizing hormone/follicle stimulating hormone ratio was more than 3 (26 and 8miU/ml respectively).

7. HIV Test-Negative.

8. Haemogram: Haemoglobin-13.7gm/dl, WBC-6.7 x 10^9/l, Platelet 297x 10^9/L.

9. Urea and Electrolytes: BUN-5.1, Potassium-4.4 mmol/l, Creatinine-84mmol/l.

DIAGNOSIS

A diagnosis of primary infertility due to polycystic ovary disease was made.

MANAGEMENT

She was planned for Laparoscopy and was admitted into the cold gynaecology ward.

Consent was obtained and the patient was starved from midnight. She was given 5mg of diazepam once on the night before surgery.

Premedication with 0.6mg Atropine was given half an hour before theatre. In theatre she was put under general anaesthesia and was put on lithotomy position. Vulvovaginal toilet was done and bladder aseptically catheterized. A Sims speculum was inserted and anterior lip of cervix was identified and held with tenaculum. The uterus was sounded and its direction and depth were noted. A metallic cervical canula was inserted and was then locked together with the tenaculum to hold them together in place. The abdomen was then cleaned and draped with sterile towels.

A port was made just below the umbilicus about 1cm long. A 10mm needle was inserted into the peritoneal cavity, which was insufflated with about 2 litres of gas (carbon dioxide). The trochar was then removed and the Laparoscope was inserted. A camera was also affixed to the laparoscope and connected to the TV monitors.

The whole of the pelvis was viewed, with the assistant manipulating the uterus to expose the different parts and to stretch the fallopian tubes. Methylene Blue Dye was instilled through the Zubia's canula.

The findings were:

The uterus was of normal size, both the fallopian tubes were normal with no pelvic or perihepatic adhesions but the ovaries were enlarged with smooth pale looking surface. There were multiple cysts with clear fluid. There was no fluid in the Pouch of Douglas. There was prompt bilateral spill of dye into the peritoneal cavity through the tubes.

Two other ports were made on both lower lumbar regions about 1-2 cm at the level of the anterior superior iliac spine. Trochar sleeves were inserted. Through these ports, a forceps and a drill were introduced. The forceps stabilized the ovaries while the drill with the help of unipolar diathermy was

used to drill 7 holes in each ovary. The ovaries shrank markedly following this procedure. Haemostasis was achieved with unipolar diathermy.

All blood was removed by gentle suction, and the pelvis irrigated with warm normal saline. The laparoscope was then removed and the gas allowed to escape. The abdominal puncture sites were stitched with No. 2/0 vicryl in two layers. The patient was successfully reversed from General Anaesthesia and taken to recovery room.

POST-OPERATIVE MANAGEMENT

She was started on parenteral medication, crystalline penicillin, gentamycin and pethidine. She was up and about and on normal diet within 24 hour of surgery and was discharged home on oral antibiotics and analgesics to be followed up in the fertility clinic.

FOLLOW UP

When seen at the fertility clinic four weeks later, she had no complaints. The small wounds had healed well and she had no abdominal pains. Her menstrual periods had ended the previous day. The periods had lasted for 3days, were moderate in amount but were painful. She was put on clomiphene citrate 50mg once a day from the 3rd day of the cycle and advised on fertile days 13th cycle day. She had three episodes of menstrual periods after surgery and while on clomiphene. She was on monthly followup and clomiphene had been increased in a stepwise manner at an interval of 50mg every month. The cycles were regular at an interval between 28-31days with a flow of 3-4days. After the third dose of clomiphine citrate, which was 150mg, she missed her periods and pregnancy test done was positive. Her LMP was on 19/8/06.

A scan done at 6weeks confirmed a single intrauterine pregnancy. She was started on oral dydrogesterone 10mg three times a day for the first 12 weeks.

She was subsequently lost to follow up.

DISCUSSION

The patient presented was a 32 year old lady admitted with primary infertility due to anovulation and polycystic ovary disease. She underwent Laparascopic ovarian drilling with good results.

Infertility is an absolute state of inability to conceive, and can affect either the male or the female partner. It is defined as failure to conceive after one year of regular coitus without contraception. It is classified as primary where the couple has never had a pregnancy or secondary where at least one conception has taken place for one or both partners (1,2). The patient presented had primary infertility and had been married for the past 4years. The cause was traced to anovulation due to polycystic ovary disease (PCOD).

Worldwide the prevalence of infertility is estimated to be between 15 and 40% (1). In Kenya infertility is a reproductive health problem of considerable importance despite the fact that the population growth rate in Kenya is considered to be one of the highest in the world. Although there are no figures available at the national lever about the extent of the problem in this country, a study in Nairobi reported that a practicing gynaecologist spends a third of his time attending to infertile patients. One in six couples need a specialists help at some stage because of infertility. (1, 2,3)

Polycystic ovary disease (PCOD) was first described by Stein and Leventhal in 1935 as consisting of amenorrhoea, hirsutism, and obesity with polycystic ovaries. This has since been challenged as being limiting and representing just part of the problem. The real definition should include the various anovulatory disorders with clinical evidence of hyperandrogenism and/or

hyperandrogenaemia. There is evidence that anovulation for whatever reason is responsible for multiple ovarian cysts the hormonal steady state which becomes self-propagating (2,4). Our patient had polycystic ovaries, dysfunctional uterine bleeding and hirsutism and was overweight but not obese (BMI-29.3).

The incidence of PCOD ranges from 3-5% (2) worldwide but the incidence in Kenya is not known.

Anovulation leads to a steady hormone state as opposed to the fluctuation that occurs in ovulatory women. The average level of oestrogen and androgen are increased and maintained by leuteinizing hormone (LH) in anovulatory patients (5). Testosterone, androstenedione, dehydroepiandrosterone (DHEA), dehydroepiandrosterone sulphate (DHEAS), 17hydroxyprogesterone and oestrone levels are high in anovulatory patients in keeping with increased ovarian secretion (testosterone, androstenedione and DHEA), adrenal gland production (DHEAS) and increased peripheral conversion especially in adipose tissue (oestrone). The high and persistent level of oestrogen and androgens maintain a high LH tone, which in turn continues to stimulate the theca zone of the ovaries to secrete more androgen. High circulating levels of androgen suppress liver production of sex hormone binding globulins (SHBG) which results in further increase in free metabolically active oestrogens and androgens. There is suppression of follicle stimulating hormone (FSH) release with reversal of LH: FSH ratio (6,7).

Androgen and oestrogen levels were not tested for our patient but LH: FSH ratio was greater than 3:1 and had clinical evidence of hyperandogenism with acne, hirsutism and clitoromegally.

Obesity can further aggravate the picture through hyperinsulinaemia. Excess insulin stimulates the theca zone of the ovaries to produce androgens

by binding the insulin-like growth factor one (IGF-1) but inhibit hepatic synthesis of SHBG and insulin-like growth factor 1 binding protein (IGFBP-1). These further increase the level of unbound and metabolically active oestrogens, androgens and IGF1. Non-insulin dependent diabetes mellitus (NIDDM) poses the same risk as obesity (8). Our patient was overweight, bordering on obesity but had no family history of diabetes. She did not have any symptoms of diabetes even in pregnancy. She was however not tested for glucose intolerance. Whether she will develop intolerance or frank diabetes as the pregnancy advances or after the age of forty like Non-insulin dependent diabetes mellitus (NIDDM) patients will remain to be seen. Oral glucose tolerance test at 28weeks is advised.

Clinical features of persistent anovulation include ammenorrhoea, which occurs in 50%, irregular heavy bleeding in 30% and oligomenorrhoea. Virilization is rare but hirsutism occurs in 70%. Acne may also be present. Overweight or frank obesity may occur but is not a necessary feature of PCOD anymore. Its presence is however associated with more incidence and severity of hirsutism (1,2,4). Most of these features were present in our patient as documented earlier.

The ovaries are usually enlarged in volume with multiple cysts arranged peripherally. Volume of greater than 10ml compared to 4.7-5.2ml in normal persons is not unusual (2). Many women with evidence of anovulation and hyperandrogenism do not have polycystic ovaries. They are said to have chronic hyperandrogenic anovulation instead of PCOD (1,2). The patient presented had ovaries with a volume of 8ml and multiple cysts and feature of hyperandrogenism with LH:FSH reversal. She qualified to be diagnosed as having PCOD.

Endometrial biopsy should be done on every woman with PCOD whatever the age due to the risk of endometrial cancer after prolonged eostrogen and

IGF-1 stimulation (1,2). Endometrial biopsy was done for our patient, which showed cystic endometrial hyperplasia. HSG and semenalysis was also done for our patient. HSG was not necessary with such overwhelming evidence of PCOD since laparoscopy was going to be done and dye injection would achieve the same purpose of checking tubal patency. However semenalysis was appropriate in determining male factors.

Treatment of patients with PCOD aims at correcting menstrual disorders, restoring fertility where desired, reducing the risk of cancer of the endometrium and that of cardiovascular disease. In patients who do not desire fertility immediately, progesterone supplementation for 10days in every cycle from day 16 is effective in restoring normal menstrual cycles and reducing risk of endometrial cancer but does not lower the risk of cardiovascular diseases. Many gynaecologists therefore recommend the use of combined oral contraceptive (9).

In patients who want to conceive, all the aims of treatment can be achieved by induction of ovulation. This can be done with clomiphine citrate, metformin and gonadotropic hormones (1,10,11). Patients treated with ovulation induction following PCOD have more risk of abortion. This is thought to be caused by high levels of pre-induction LH. This is treated by pre-induction suppression of LH by gonadotrophin releasing hormone analogues (12).

Where all these fail, ovarian wedge resection, drilling or laser vaporization can be done (13,14,15). Our patient had tried clomiphine without success and gonatrophins were too expensive for her. She was offered ovarian drilling with good outcome.

REFERENCES

1. Eskandari N., Cadieux M. Infertility. Current Obstetrics and Gynaecologic Diagnosis and Treatment. 9th Ed. 2003; McGraw-Hills New York. **53**: 979-990.

2. Stenchever M.A., Droegemueller W., Herbst A.L., Mishell D.R. Hyperandrogenism. Comprehensive Gynecology 4th Edition. 2001. Mosby Philadelphia. **40:** 1143-1167.

3. Wanjala S. Is micro-surgery useful in the management of infertility in this country? J. Obstet. Gynecol East. Cent. Afric.1992.

4. Sperrof L., Glass R.H., Kasse N.G. Anovulation and the polycystic ovary. clinical Gynecologic Endocrinology and Infertility. 5th Ed. 1994; William and Wilkins, Philadelphia. **13**: 457-482.

5. Calogero A.E., Macchi M., Montanini V. Dynamics of plasma gonadotrophins and sex steroids release in polycystic ovary disease after pituitary-ovarian inhibition with an analogue of gonadotrophin releasing hormone. J. Clin Endocrinol Metab 1987; **64**: 980.

6. Laatikainen T.J., Apter D.L., Paavonen J.A., Wahlstrom T.R. Steroids in ovarian and peripheral venous blood in polycystic ovary disease. Clin Endocrinol 1980; **13**:125.

7. Waldstreicher J., Santoro N.F. Hall J.E., Fillicori M. Hyperfunction of the hypothalamic-pituitary axis in women with polycystic ovarian disease:indirect evidence for partial gonadotrophin desensitization. J. Clin Endocrinol Metab.1988; **66**:165.

8. Poretsky L. On the paradox of insulin induced hyperandrogenism in insulin resistant state. Endocrin Rev. 1991; **12**:3.

9. Wild R.A., Painter P.C., Coulson P.B., Ranny G.B. Lipoprotein lipid concentration and cardiovascular risk in women with polycystic ovary disease. J. Clin. Endocrinol Metab. 1985; **61**: 946.

10. Johnson J.E. Jr., Outcome of pregnancies following clomiphine citrate therapy.Excerpta Medica, 1967; 101.

11. McFaul P.B., Traub A.I., Thompson W. treatment of clomiphine citrate-resistant polycystic ovary disease with pure follicle stimulating hormone or human menopausal gonadotropin. Fertil Steril. 1990; **53**: 792.

12. Hormburg R., Levy T., Berkovitz D. Gonadotropin releasing hormone agonist reduces miscarriage rate for pregnancies achieved in women with polycystic ovarian syndrome. Fertil Steril 1993; **59**: 527.

13. Katz M., Carr P.J., Cohen B.M.. Hormonal effects of wedge resection of polycystic ovaries. Obstet Gynecol. 1978; **51**: 437.

14. Kovacs G., Buckler H., Gangah M. Treatment of anovulation due to polycystic ovarian syndrome by laparoscopic ovarian electrocautery. Obstet Gynecol 1991; **98**: 30.

14.Daniell J.F., Miller W. Polycystic ovarian syndrome treated by laparoscopic laser vaporization. Fertil Steril 1989; **51**: 232.

GYNAECOLOGY CASE 9

STAGE II SQUAMOUS CELL CARCINOMA OF THE VULVA-RADICAL VULVECTOMY

NAME G.M. IP NO. 0899952

AGE 58 YEARS D.O.A 6/10/06

PARITY 5+0 D.O.D 21/11/06

PRESENTING COMPLAINTS

She was admitted through casualty with history of vulval swelling and itching for about one year.

HISTORY OF PRESENTING COMPLAINTS

The patient presented to KNH casualty as a referral from Kitui District Hospital with 1year history of vulval itchiness and 9months history of vulval pain and swelling. The swelling was progressive but had not ulcerated, bled or had a discharge. She had received antibiotic, antifungal and steroid treatment in the private clinics without improvement. She had also been seen in Kitui District Hospital where biopsy done had showed well differentiated squamous cell carcinoma.

PAST MEDICAL HISTORY

She had suffered genital ulcers when she was young which recovered within a week of treatment without recurrence. She had never been diagnosed to suffer from any chronic illness like hypertension or diabetes.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She was para 5+0. Her last delivery was in 1978. All deliveries were by spontaneous vertex delivery. She could not tell her age at menarche but was over 20 years postmenopausal. She never used any contraceptives and declined to comment on her premarital sex life.

FAMILY AND SOCIAL HISTORY

She was a second wife married to a small-scale businessman who sold Kamba traditional items at Mwala shopping centre. She used to drink alcohol, smoke cigarettes and chew tobacco when young but stopped after getting married. The first wife died 5years earlier from longstanding vaginal bleeding suspected to be from cancer of the cervix.

PHYSICAL EXAMINATION

She was an elderly lady in fair general condition with no fever, pallor, jaundice or peripheral oedema. She had no significant lymphadenopathy. Her blood pressure was 140/90mmHg; pulse rate of 86 per minute, respiratory rate 18 per minute and her temperature was 37^0C.

The Cardiovascular, respiratory and the Central Nervous Systems were normal.

ABDOMINAL EXAMINATION

The abdomen was flat and moved with respiration. There were no surgical scars. She had mild suprapubic tenderness but no masses were palpable.

PELVIC EXAMINATION

She had a fleshy cauliflower growth on the anterior part of the right labia majora and encroaching on the midline, measuring 5cm in the greatest diameter. The right labia minora, the left labia majora and minora as well as the urethra and anus felt free of disease. The vaginal and rectal mucosa, were freely mobile. Inguinal lymph nodes were not palpable. The uterus and adnexa felt normal and the cervical os was closed and not abnormalities were felt on it.

DIAGNOSIS

A diagnosis of cancer of the vulva stage II was made.

INVESTIGATIONS

- Histology showed well differentiated invasive squamous cell carcinoma.
- Haemogram: Haemoglobin-13.3gm/dl, white cell count-$4.3X10^9$/L and platelet count-$208X10^9$/L
- Urea and Electrolytes: Na^+-139mmol/L, K^+-3.4mmmol/L, Cl^--105mmm0l/L, and urea-4.8umol/l.
- Liver function tests - Normal.
- Chest Xray - Normal
- Random blood sugar - 5.2mmol/l.
- ECG - Normal.

MANAGEMENT

She was planned for examination under anaesthesia and radical vulvectomy and was counselled accordingly and informed consent was obtained. Blood was taken for grouping and cross matching and two units of blood reserved for the operation.

She was given rectal enema at 6.00pm on the eve of the operation, and repeated again at 6.00am on the day of operation. She was also given 5mg of diazepam on the eve of surgery and starved from midnight. On the morning of the operation, she was premedicated with 0.6mg of atropine and 50mg of pethidine intramuscularly before being wheeled to theatre.

In theatre she was put under general anaesthesia and placed in semi-lithotomy position. Examination under anaesthesia was done which confirmed the above findings. The uterus and cervix were normal and fully mobile and there were no masses in the pelvis. The vulva and perineum were cleaned using hibitane solution and then painted with betadine. Foleys

catheter was inserted into the bladder and retained by ballooning. The patient was draped with sterile towels.

A semi-linear transverse incision was made about 2centimetres above the upper margins of the pubis symphysis. It was extended to the labiocrural folds. Another incision extending from 2cm medial to the anterior superior iliac spine along the inguinal ligament was made and extended to join the transverse incision at the labiocrural fold. A third incision was made at the intersection of above incisions and extended downwards along the labiocrural folds to the perineum, about 1cm anterior to the anus. Similar incisions were made on the opposite side to meet in the midline. The final incision was made starting anterior to the urethral orifice and extending round the vestibule. All the skin and subcutaneous tissue enclosed in the incisions were removed. Superficial inguinal lymph nodes were removed bilaterally and put in separate specimen bottles and labeled accordingly before being submitted for histology together with the excised vulval tissue. The lymph nodes did not look suspicious though. A suction drain was inserted before suturing the incision edges with interrupted nylon sutures.

POST OPERATIVE MANAGEMENT

She was taken to the recovery ward where vital signs were taken continuously until she was fully awake. She was then transferred back to the gynaecology ward where 4 hourly observations were continued four 24 hours then twice daily. She was also started on intravenous crystalline penicillin 2 mega units 6 hourly, gentamycin 80mg 8 hourly and Metronidazole 500mg 8 hourly. She received intramuscular Pethidine 100mg 8 hourly for 48hours then diclofenac 75mg 8hourly.

Perineal toilet was also done twice daily using saline and on the 12th post-operation day, the sutures and catheter were removed. The wound had apposed well except for a raw area, which was discharging pus in the perineal area. A pus swab taken, which isolated *Escherichia coli* and *Proteus species,* sensitive to augmentin (amoxycillin and clavulanic acid combination). She was put on oral augmentin 625mg twice daily and continued on daily saline baths. The wound finally healed completely and patient was discharged home on the 22nd postoperative day for review in the gynaecology clinic.

FOLLOW UP

She was seen in the gynaecology clinic 2weeks later where she had no complaints and the incision site was well healed. Histology confirmed well-differentiated squamous cell carcinoma with no involvement of the incision margins or the sampled lymph nodes. She is being followed up in the gynaecology clinic with six monthly clinical examination and annual papsmear.

DISCUSSION

The patient presented was a 58year old para 5+0 with stage II cancer of the Vulva for which she was treated successfully by radical vulvectomy and bilateral inguinal lymphadenectomy.

Cancer of the vulva refers to malignant conditions arising from the various components of the vulva namely; the skin, subcutaneous tissues, urethra, glandular elements or from the mucosa of the lower third of the vagina. Approximately 85-90% of these tumours are of squamous cell origin. Other tumours include extramammary Paget's disease, carcinoma of Bartholin's gland, basal cell carcinoma, sarcoma and metastatic cancers form other sites

(1,2). In Kenyatta National Hospital, squamous cell carcinoma accounted for 97.4% with 57.9% being well differentiated (3).

Cancer of the vulva accounts for approximately 3-4% of all gynaecological malignancies worldwide, making it the 4th most common cancer of the female genital tract (4). At Kenyatta National Hospital it's also the fourth commonest gynaccological cancer accounting for 3.3% (3). Our patient had well differentiated squamous cell carcinoma.

It has been suggested that vulvar cancer exists as two separate diseases. The first type involves human papillomavirus (HPV) infection, which leads to development of a premalignant condition called vulvar intra-epithelial neoplasia (VIN). This may eventually develop into vulvar cancer that occurs commonest below the age of 45years. The second type involves vulvar non-neoplastic epithelial disorders and advanced age, leading to cellular atypia and cancer. The peak age of occurrence of this kind of malignancy is between 60 and 79 years (5). Risk factor for women found with vulvar cancer at age below 45years are human papilloma virus, cigarette smoking, having more than one sexual partner, sexual initiation before age 19 years and low economic status. In patients older than 45 years, risk factors include; chronic vulval irritation, granulomatous venereal diseases, vulval dystrophies, diabetes mellitus, hypertension, residence in a rural area, low economic status, menopause before age 45, poor hygiene and low serum vitamin A levels (6).

The patient presented was 58years old, smoked cigarettes, was in a polygamous family, had suffered genital ulcers, was of low social economic status, was from rural area and had menopause at around age 38. This patient therefore had risk factor from both categories of vulval cancer patients. Her age at onset of sexual activity and her hygiene were however

not established. She also never suffered from diabetes or hypertension, and her age at disease onset was not classical for either.

The anterior aspect of the vulva is the commonest site, involving the labia majora and labia minora. Clitoris is commonly involved. The spread is predictably to the vagina, urethra or anus by local spread or to the inguinal nodes via lymphatics. Extensive lesions involving the anus or rectovaginal septum can drain directly into the pelvic nodes (1,3,7). Our patient had cancer involving the anterior part of the labia majora and encroaching on the midline.

Over half of the patients usually present with pruritis, pain or a vulvar mass. Other patients complain of bleeding, discharge or dysuria, whereas approximately 20% of patients have no complaints and the tumour is found incidentally during routine pelvic examination. Examination may reveal vulvar lesion usually raised and may be fleshy, ulcerated, leukoplakic, or warty in appearance (1,2,8). Diagnosis is confirmed by biopsy. This patient presented with vulvar pruritis, pain and swelling and raised fleshy lesion on the labia majora. Biopsy done confirmed the disease at histopathology.

Carcinoma of the vulva is staged according to the revised FIGO staging system (1,2,7)

- Stage o -Carcinoma in situ
- Stage I -Tumour confined to the vulva and less than 2cm greatest diameter.

  IA-Stromal invasion not greater than 1mm

  IB-Stromal invasion greater than 1mm
- Stage II-Tumours confined to the vulva but greater than 2cm in its greatest diameter.

- Stage III-Tumour of any size with one or both of the following; adjacent spread to the lower urethra, vagina or anus and/or unilateral inguinal lymph nodes involvement.

- Stage IV -Tumour involving any of the following; upper urethra, bladder mucosa, rectal mucosa, pelvic bone and/or bilateral regional nodes or pelvic lymph node involvement.

Squamous cell carcinoma is also graded depending on the level of differentiation of the tumour cells into; well differentiated, moderately well differentiated and poorly differentiated or anaplastic carcinoma (2). The patient presented had well differentiated Stage II disease .

Surgical resection is the gold standard of treatment in patients with vulvar cancer. Surgery aims at completely removing the cancer and identifying the extent of disease to determine the stage and the need for additional therapy. The extent of disease determines the amount of surgery needed. Initially, radical vulvectomy with bilateral dissection of the groin and pelvic nodes was recommended as the standard treatment for most patients. Presently, a more individualized and conservative approach to treatment is recommended.

Most surgeons perform a radical enbloc vulvectomy or a radical local excision to treat vulvar cancer. The aim is to remove the primary lesion with a 1-cm margin and to remove the involved lymph nodes. The difference between a radical vulvectomy and a radical local excision is the bridge of skin between the lesion and the groin nodes. Metastases to the skin bridge were found to be rare without clinically suspicious inguinal nodes; when radical vulvectomy was compared with radical excision, there was no difference in local recurrence (8). Our patient underwent enbloc type of radical vulvectomy. Deep femoral lymph nodes excision was omitted

because superficial nodes were not suspicious. They were later confirmed to be disease free on histology.

Groin node dissection is associated with wound infection and breakdown, and chronic leg edema. Research has found that stromal invasion of 1 mm or less (stage IA) is not associated with groin node metastases, but any patient who develops recurrent disease in an unresected groin has a high risk of mortality (8). The Gynecology Oncology Group recommends that any patient with more than 1 mm of dermal invasion have an inguino-femoral lymphadenectomy; if more than two groin nodes are positive or there are clinically suspicious groin nodes, postoperative groin and pelvic radiation therapy is recommended (9). Our patient needed inguinal node excision because her disease was more extensive than stage IA but deep femoral nodes were spared since the superficial nodes were not suspicious. She did not experience serious wound breakdown but had mild leg oedema. She did not need radiotherapy because her groin nodes were all disease free at histology.

Recurrence in the groin appears to represent persistent disease, occurring early and near the treated site among patients treated conservatively (10). The risk of recurrent disease increases with the number of positive groin nodes. Patients with three or more positive nodes have a high incidence of local, regional, and systemic recurrence (8). Local recurrences are best treated with repeat surgical excision. Radiation therapy with surgery has been used to treat groin recurrence, while chemotherapy is used for systemic metastases. Chemotherapy has poor response rates and has been ineffective in the treatment of recurrent disease (8,11). The patient presented is still on followup with annual pap smears and clinical examination and is so far disease free.

The prognosis of patients with vulvar cancer is generally good when appropriate treatment is initiated in a timely fashion. The overall five-year survival is 70 percent and correlates with the stage of disease and lymph node status. The rate for stage I is 98%, II 85%, III 74% and IV 31% (8,12). The number of positive groin nodes is the most important prognostic factor. Positive inguino-femoral lymph node is associated with 5year survival rate of 52.4% down from 91.3% in those with negative nodes. Positive pelvic nodes reduce the rate even further to 11% (8). Other factors that influence prognosis are tumor size and tumor ploidy (i.e., number of pairs of chromosomes). Patients with aneuploid tumors (i.e., an abnormal number of chromosomes) have a poorer five-year survival rate of 23% compared to those diploid tumors whose rate is 62% (13). Good prognosis is expected in our patient due to early disease and tumour grade though ploidy was not analysed.

REFERENCES

1. David L., Barlay. Premalignant and Malignant Disorder of the Vulva and Vagina: Current Obstetric and Gynaecologic Diagnosis and Treatment, 9th Ed.2003; McGraw-Hills, New York, **46**:879-893.

2. Stenchever M.A., Droegenmueller W., Herbst A.L. Neoplastic diseases of the vulva. Comprehensive gynecology 4th Ed 2001;Mosby Philadelphia **32**:999-1021.

3. Kaguta J.K. 10 years review of Cancer of the Vulva seen at the K.N/H. M.med Thesis, University of Nairobi 1984

4. Greenlee R.T., Murray T., Bolden S. Cancer statistics, 2000. CA Cancer J Clin 2000 Jan-Feb; **50(1):** 7-33<http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Abstract&list_uids=20199188>

5. Crum C.P. Carcinoma of the vulva: epidemiology and pathogenesis. Obstet Gynecol 1992;**79**:448-54.

6. Basta A., Adamek K., Pitynski K. Intraepithelial neoplasia and early stage vulvar cancer. Epidemiological, clinical and virological observation. Eur J Gynaecol Oncol 1999;**20**:111-4.

7. Rock J.A., Horowitz I.R., Dominguez C.E. Malignancies of the vulva. Te Lindes gynaecologic surgery,9th Ed. 2001; Lippincot-Raven Publishers, Philadelphia **44**:1293-1350.

8. Hacker N.F. Vulvar cancer. In: Berek JS, Hacker NF, Eds. Practical gynecologic oncology. 3d ed. Philadelphia: Williams & Wilkins, 2000:553-96.

9. Homesley H.D., Bundy B.N., Sedlis A. Radiation therapy versus pelvic node dissection for carcinoma of the vulva with positive groin nodes. Obstet Gynecol 1986;**68**:733-40.

10. Stehman F.B., Bundy B.N., Ball H. Sites of failure and times to failure in carcinoma of the vulva treated conservatively: a gynecologic oncology group study. Am J Obstet Gynecol 1996; **174**:1128-33.

11. DiSaia P.J., Creasman W.T. Clinical gynecologic oncology. 5th ed. 1997. St. Louis: Mosby.

12. Homesley H.D., Bundy B.N., Sedlis A.,etal. Assessment of current International Federation of Gynecology and Obstetrics staging of vulvar carcinoma relative to prognosis.

13. Kaern J., Iversen T., Trope C., etal. Flow cytometric DNA measurements in squamous cell carcinoma of the vulva: an important prognostic method. Int J Gynecol Cancer 1992;**2**:169-74.

GYNAECOLOGY CASE 10

PELVIC ABCESS - LAPAROTOMY AND DRAINAGE

NAME: J. A. IP NO: 1065916

AGE: 26 YEARS DOA: 12/05/06

PARITY: 2+0 DOD: 17/05/06

PRESENTING COMPLAINTS

She was admitted to the acute gynaecological ward with history of abdominal pain for 6 days.

HISTORY OF PRESENTING COMPLAINTS

Abdominal pain developed insidiously, starting in the lower abdomen and spreading to the sides and later to the back. The pain had increased gradually to a point of hindering normal activity and was not relieved by analgesics. She had history of purulent vaginal discharge lasting between 2 and 3 weeks before onset of current illness but had not taken it seriously. Before admission, she had vomited twice but had no history of diarrhoea. Her urinary habits had not changed. She had started experiencing hotness of body, chills and headache.

PAST MEDICAL HISTORY

She was admitted with pulmonary tuberculosis in 1995 and was put on 6months course of tuberculosis treatment, which she completed. She had been treated once at a health centre for vaginal discharge, which she was told was sexually transmitted infection. Her husband was however asymptomatic and refused to be treated with her. She had no other history of chronic illness.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She was para 2+0. Her menarche was at 15 years. She had regular menstrual cycles of 28 days, and her flow lasted 3 days. Her flow was light

with associated pain. She had not used any contraceptives. Her last menstrual period was 4/5/06.

FAMILY AND SOCIAL HISTORY

She was a housewife and her husband was a long distance truck driver. There was no family history of chronic illness. She neither drunk alcohol nor smoked cigarettes.

PHYSICAL EXAMINATION

She was sick looking, in pain, clinically febrile, with no pallor or jaundice. She had no lymphadenopathy. Her vital signs were as follows; blood pressure of 105/70 mm/Hg, pulse rate of 96 per minute, respiratory rate of 24 per minute and temperature of 38.1°C.

ABDOMINAL EXAMINATION

The abdomen was slightly distended from the suprapubic region and moved with respiration. There was mild generalized tenderness over the abdomen but marked at the suprapubic region and both iliac fossae. There was rebound tenderness but bowel sounds were present and normal.

PELVIC EXAMINATION

The external genitalia was normal. The cervix was firm, posterior, long and the os was closed. There was marked cervical motion tenderness. Pouch of Douglas felt full and both adnexeal regions were full and tender. There was foul smelling purulent discharge on examining fingers.

INVESTIGATIONS DONE

- Pelvic Ultrasound: Uterus was normal in size and non gravid. There was an ill-defined complex mass extending from the right ovary to the Pouch of Douglas and had fluid with echogenic contents. Both right and left ovaries were visualized and were normal.
- HIV I and II were negative

- Haemogram: Hb - 11gm/dl, WBC - 17 x 10^9/l, N - 80%, L - 9%, platelets - 290 x 10^9/l.
- Urea and Electrolyte: Na + - 110 mmol/l, K+ - 5.22Ummol/l, Urea - 4.3mmol/l, Creatinine - 102 Ummol/ll

DIAGNOSIS

A diagnosis of pelvic abscess was made.

MANAGEMENT

She was started on Intravenous metronidazole 500mg 8 hourly, cefuroxime axetil 750mg 8 hourly. The patient was informed of the diagnosis and the intended laparotomy for drainage of the abscess. An informed signed consent was obtained. Blood was taken for grouping and cross-matching. Her pubic hair was shaven and she was premedicated with atropine 0.6mg and 50mg of pethidine intramuscularly before being wheeled to the operating theatre.

LAPARATOMY AND DRAINAGE

In theatre she was put under general anaesthesia and in semilithotomy position, bladder was catheterized under aseptic technique and clear urine obtained. Examination under anaesthesia was done which confirmed presence of fluctuant mass occupying both adnexeal regions and the Pouch of Douglas. The Cervix was long, firm, posterior and the os was closed. The uterus was however of normal size but deviating to left side.

She was then positioned in supine position and the abdomen was scrubbed and draped as outline in the introductory section. The abdomen was opened through a subumbilical midline incision. The Peritoneum was thickened. There were adhesions of the bowels to the omentum and the tubes. There was an encapsulated mass filling the Pouch of Douglas and involving both ovaries and tubes. Pus was aspirated and submitted to the laboratory for culture and sensitivity. Adhesions were released by blunt dissection and about 1200mls of foul smelling pus drained. All the pus pockets were

broken and drained. The uterus was about 8 week's size and its posterior wall was inflammed. The appendix was buried in adhesions but appeared grossly normal.

Peritoneal lavage was done with warm saline and rifocin. A corrugated drain was placed in the pus cavity and brought out through the right iliac fossa. There was minimal blood loss. Instruments and swabs were counted and found correct before the abdomen was closed in layers. The patient recovered from general anaesthesia uneventfully.

POST OPERATIVE CARE

She was observed continuosly in the recovery room until she was fully awake, then transferred back to the ward. She was continued on intravenous fluids, normal saline alternating with 10% dextrose, 3 litres in 24 hours until bowel sounds returned. She also continued on Intravenous antibiotics, cefuroxime, and metronidazole and was put on intramuscular pethidine 100mg 8hourly and diclofecac 75mg 8hourly also intramuscularly for pain relief. Culture results were obtained on the third postoperative day and had isolated *Escherichia coli* sensitive to cefuroxime, which was continued.

On the first postoperative day, bowel sounds were present and she was started on oral sips. The drain ceased to drain on the second day and was removed. On the third day, the abdominal wound was exposed and was found to be clean and dry. Haemoglobin level was 10.3g/dl. She had uneventful recovery and was discharged on the 4th post-operative day due to pressure of beds for removal if stitches in the nearest health facility. She was booked for review in GOPC after 2 weeks.

FOLLOW UP

She came to the Gynaecological out patient clinic as per appointment. She had no complaints and she had recovered fully. There was no tenderness in the abdomen. .

DISCUSSION

The patient presented was a 26year old para 2+0 who had pelvic abscess. She had laparotomy and drainage done with good outcome.

Pelvic abscess is the collection of pus in the pelvic cavity. Most cases are secondary to pelvic inflammatory diseases, while others may follow post-abortal or puerperal sepsis (1).

Perforation of the appendix as a sequela of acute inflammation may also lead to a pelvic abscess. Gynaecological procedures that breach the cervical mucus barrier such as Intrauterine Device (IUD) insertion, endometrial biopsy, uterine curretage, hysterosalpingography and hysterescopy also increase the risk (2, 3). Of the cases admitted to the emergency gynaecological wards at Kenyatta National Hospital 40% were found to be related to P.I.D (4). The patient presented had pelvic abscess most likely arising from pelvic inflammatory disease. The appendix was buried in adhesions but appeared grossly normal.

Enterobacteria and anaerobic bacteria especially E. coli and bacteroides species are isolated most commonly in pelvic abscesses. Gonococus and Chlamydia, though common causes of pelvic infection are rarely isolated from pelvic abscesses (4, 5). Carley found that 75% of all causes of PID had gonococcus while Fomulu found aerobes and anaerobes with E.coli occurring in 50% of all cases at K.N.H (6). Broad antimicrobial coverage is advised awaiting culture and sensitivity results. Cover for gram positive, gram negative and anaerobes is most appropriate (3, 5). The patient presented was put on cefuroxime and metronidazole and responded well. Culture and sensitivity justified their use which was continued.

Pelvic abscess presents with abdominal pains, fever, nausea and vomiting, rigors, chills and backache. These are the same symptoms for acute PID and therefore should be suspected if they persist after treatment for acute pelvic inflammatory disease, or develop following an abortion or delivery or after gynaecologic surgical procedure (2, 3, 5). The presented patient had low abdominal pain preceded by vaginal discharge.

The patients are usually sick looking with fever, tachycardia and tender lower abdomen. Fluctuant tender mass filling the cul-de-sac and dissecting into the recto-vaginal septum may be felt on pelvic examination. Pelvic organs are usually difficult to feel due to tenderness. Culdocentesis may reveal pus but may rupture the abscess and must be performed with extreme caution if at all (2, 3, 5). The patient presented had pelvic inflammatory disease whose treatment was delayed. She had fluctuant tender mass and aspiration was not done.

Ultrasound can be used to confirm the diagnosis. MRI and Pelvic CT scan are also sensitive but the price is prohibitive. They may also not give as much detail as a well done ultrasound scan. Other tests in case of doubt are urinalysis and laparoscopy. Haemogram shows leucocytosis both relative and absolute (2, 3, 7). The patient presented had a pelvic ultrasound which was highly suggestive of the diagnosis and an exploratory laparotomy confirmed it.

Surgery for patients with pelvic abscess is usually reserved for cases of rupture, non response to medical therapy and abscess inaccessible to extraperitoneal drainage. The management of unruptured abscess on the other hand is controversial with recommendations varying from prompt radical surgery to conserve treatment with parenteral antibiotics (8).

Conservative treatment with intravenous antibiotics and prophylactic drainage, or aspiration of abscess through laparascope is preferred (5, 8). Patients with a midline, fluctuant pelvic abscess can safely have a vaginal

drainage by dissection through the posterir fornix. Transvaginal ultra-sonography guided drainage of selected patients with pelvic abscess has been shown to be effective (8). The presented patient had primary laparotomy and drainage due to size of the abscess.

Surgical drains ensure completed evacuation of pus following laparotomy. The ideal exit for a drain is through the cul-sac except when this is completely obliterated with adhesions. In this case, drainage is done through a small stab wound in the lower quadrant above the area to be drained. Drainage through the primary incision is not good due to risks of incisional hernia, infection and fistula formation. When pus is spilled and gross contamination of the operative field is present, a closed suction drainage system is installed through a separate stab wound and abdomen closed up to the deep fascia. Closure of the superficial fascia and the skin can deferred for a few days and closed secondarily to avoid incisional breakdown from infection (2, 3). A corrugated drain was fixed for the presented patient through a separate stub wound while the incision was closed primarily with no complications.

Complications of pelvic abscess include intra-abdominal rupture, chronic pelvic pain, bowel obstruction, infertility, chronic dyspareunia and predesposition to ectopic pregnancy. Intrabdominal rupture is most life threatening complication of all. Death may occur due to septic shock and generalized peritonitis. A pelvic abscess can rupture spontaneously into the rectum, sigmoid colon and bladder or into the free peritoneal space. During surgery, there is risk of injury to the ureters, colon, rectum and small bowel (2). Our patient did not experience any of the early complications but late complications like dyspereunia, infertility or ectopic pregnancy are likely.

Prevention of pelvic abscess formation includes health education, prevention of sexually transmitted disease, pelvic inflammatory disease, puerperal and post abortal sepsis. Provision of safe abortions would reduce incidence of

self induced abortions using crude methods. Education on condom use, being faithful to one partner and abstinence may reduce the risk of sexually transmitted infections. Poverty eradication and women empowerment may reduce transactional sex hence the spread of sexually transmitted infection including HIV/AIDS.

REFERENCES

1. Whitefield C.R. Pelvic Infections. Dewhursts Textbook of Obstetric and Gynaecology for Post Graduates, 5th Ed. 1995; 43: 596
2. Martens M.G. Pelvic Inflammatory disease. Te Linde's Operative Gyanecology 9th Ed 2001;Lippincott-Raven publishers 28: 675-704.
3. Stenchever M.A., Droegenmueller W., Herbst A.L. Comprehensive gynecology 4th Ed 2001; Mosby Philadelphia 23:707-739
4. Carly M.J., Nzoik M.J., Verhage A.R. The role of Gonococcus in Acute PID in Nairobi. Africa Med. J, 1991; 49(9).
5. Hemsell D.L., Wendel G.D., Mickal A. Sexually Transmitted Diseases and Pelvic Infections. Current Obstestric and Gynaecologic Diagnosis and Treatment 9th Ed, 2003; McGraw-Hill New York 38:716-750
6. Formulu J.N. Bacterial Sensitivity Pattern in Septic Abortions. Post Abortal Sepis and Pelvic abscess at K N H. Mmed. Thesis, University of Nairobi 1981
7. Hager D.W., Eschenbach D.A., Spence M.R. Criteria for diagnosis and grading of Salpingitis. Obstetric Gynaecology, 1983; 61:113.
8. Rivilin M.E. Clinical outcome following vaginal drainage of pelvic abscess. Obstetric Gynaecology, 1983; 61:169.

SEXUAL ASSAULT: EMERGENCY CONTRACEPTION AND STI/HIV PROPHYLAXIS

NAME: G.M. OP NO: 45892/06

AGE: 20 YEARS DOA: 12/9/06

PARITY: 0+0 DOD: 12/9/06

PRESENTING COMPLAINTS

GM presented with complaints of forceful penetrative vaginal sexual assault by people unknown to her twelve hours before she came to hospital.

HISTORY AND PRESENTING COMPLAINTS

She was coming from school at about 8pm when she was accosted by two men unknown to her. They gagged her before dragging her to a bushy area where they raped her in turn. She lost consciousness during the ordeal and when she regained consciousness, she found herself alone in the bush feeling sore in her private parts and her clothes felt wet. She sought her way home where she realized that she had been bleeding from the vagina though at that time there was no active bleeding. She reported the ordeal to her mother who gave her analgesics and a hot bath and advised her to rest till the next morning when she was brought to hospital.

OBSTETRIC AND GYNAECOLOGIC HISTORY

She was a 18 year old school girl nulligravida who was in form 2. She had menarche at 17 years and her last normal mestrual period had been on 2/9/06. Her menstrual flow lasted 4-7 days and cycles between 21 days and 3months and were irregular The flow was heavy requiring change of 4-5 pads a day in the second and third days and were associated with passage of

clots. She had no history of dysmenorrhoea. She had never used any conventional contraceptives.

PAST MEDICAL AND SURGICAL HISTORY

She was known to suffer from any chronic illness, had not had any major operation and was not known to be allergic to any drugs or foods.

FAMILY, SOCIAL AND OCCUPATIONAL HISTORY

She was the 1st born in a family of 2 siblings. She was a form two student at Sunshine Secondary School where she was a day scholar. She lived with her mother and brother at Kayole. Her mother was a business-woman who owned a butchery in the area.

She was a born again Christian, had never used illicit drugs did not smoke or drink alcohol.

GENERAL EXAMINATION

She presented her torn under clothes and blood stained dress. She was in good general condition, but anxious. She was not pale, not jaundiced not dehydrated

She had bruises on both elbows knees and thighs. Her vital signs were as follows: pulse rate 81 beats per minute, BP 120/80mmhg, respiratory rate 18/minute and temperature 36.4^{O}C

The central nervous, respiratory, cardiovascular systems and the abdomen were normal and the only abnormal findings were in the genital tract.

VAGINAL EXAMIANATION

She had bruises on the anterior medial aspect of the thighs. The external genitalia were bruised and the hymenal ring was freshly broken. Speculum examination revealed that the vaginal wall was also bruised but no tears were noted. There was scanty discharge in the posterior fornix but the cervix was intact and closed. High vaginal swab was taken for microscopy, culture and sensitivity. Digital examination found the uterus to be of normal size with free Adnexae and Pouch of Douglas (POD)

INVESTIGATIONS

1. High vaginal swab microscopy found scanty immotile spermatozoa and a mixture of gram positive and gram negative cocci and bacilli. There were no gram negative intracellular diplococci, no *tricomonas vaginalis,* no *candida ulbicans* and not 'clue, cells. Culture results were availed after 48hours where staphylococcus aureus sensitive to augmentin, cefuroxime and gentamycin was grown. The specimen had however been transported in a plain sterile dry HVS specimen bottle and cultured in blood agar media due to lack of special transport and culture media especially for *Neisseria gonorrhoea.*

2. VDRL was negative.

3. HIV I &II was negative.

4. Hepatitis B and C were negative.

5. Haemogram: Haemoglobin 11.3d/dl, white cell count $5.2x10^9$/l and platelet count was $392x10^9$/l

6. Urea and electrolytes: Na+ 141mmol/l, K+4.7mmol/l, urea 5.3mmol/l and creatinine 67mmol/l

7. Liver function tests: Total serum protein was 73g/l, albumin 36g/l, total bilirubin 6.5mmol/l, direct bilirubin 3.3mmol/l, Aspartate transaminase 28mmol/l and Alanine transaminase 32mmol/l.

DIAGNOSIS

A diagnosis of penetrative vaginal sexual assault was made.

MANAGEMENT

She was sent to patient support centre where counselling was done. The counselling involved explanation of what had happened to her and the possible consequences and preventive measures. The need for HIV test was explained where she accepted. The opportunity was also used to assess her reaction to the ordeal and her willingness to use and adhere to antiretroviral therapy. A medical report (P3) form was filled and she recorded the incidence at Buruburu Police Station though the suspects were never arrested.

She was given norfloxacin 800mg once, doxycycline 100mg twice daily and metronidazole 400mg three times daily for a week. She was started on zidovudine-lamivudine combination (combivir) and efavirence for a period two weeks and was given a prescription for hepatitis vaccine since it was not available at that time in our institution. She got it from St Mary Mission Hospital. She was given a prescription for postinor II; an emergency contraceptive method containing 75mg of levonogesterol per tablet. She was advised to take one tablet as soon as she bought but within 72 hour and the second tablet 12 hours later. She was advised to come back for followup and more antiretroviral drugs in the patient support centre and high risk clinic in two weeks.

FOLLOW UP

She kept her appointment and at the time of the visit she had finished her menstrual periods four days earlier. She was more composed, had recovered from her genital tract injuries and had not experienced any reactions to antiretroviral therapy. She was still very worried about catching HIV. She

was encouraged to continue taking antiretroviral drugs for two more weeks, reassured that the risk of getting infected was small and asked to come back after another four week.

She kept this appointment too. During this visit she had no complains, had experienced her menstruation as expected a week before and had taken all drugs as advised to completion. She was counselled for a repeat HIV test, which was done. The results were once again negative.

She did not come for her third and last visit at 3 months as appointed. During that visit, a last HIV test would have been done and I can only pray with her that it would have been negative.

DISCUSSION

Sexual assault also called rape is any sexual act performed by one person on another without the persons consent. It includes genital, anal or oral penetration by a part of the accused body or by an object. It may result from force, the threat of force on the victim or somebody else or the victim's inability to give legally binding consent. The term sexual assault is preferred because of its gender neutrality as opposed to rape which traditionally referred to forced vaginal penetration of a female victim by a male assailant (1,2).

The incidence in Kenya is not known but the annual incidence in the UK is 200 per 100,000 persons accounting for 6% of all violent crime. Approximately 1 in 5 women are sexually assaulted by the time she is 21 years of age in the UK (1).

Sexual assault is a violent crime predominantly against women. Rapists can broadly be categorized into: Power Rapist, hunger Rapist and sadistic Rapist. Power rapist view rape as a form of conquest and achieve more gratification from subduing their victims and feeling that their victims are at

their mercy. Hunger rapists are people who have been sexually deprived and rape their victims just to satisfy their sexual 'hunger'. The third group usually tortures their victims. It is not clear which category of rapists attacked our patient but they are either hunger or power rapists (3).

Other forms of rape include: marital rape, acquintance rape, incest, date rape and statutory rape. Marital rape refer to engangement in any form of sexual activity in marriage without consent of one partner. Acquintance rape is committed on the victim by a person known to her. Incest is the sexual assault on a person by a close relative or a parental figure living in the same home as the victim while statutory rape refers to sexual intercourse with a minor (legally incapable of giving consent). In our country, people below 18 years are considered minors. Child sexual abuse refers to sexual contact between the child and an adult where the child is being used to sexually stimulate the adult (2). Our patient did not fall in any of these other forms of rape.

Legal perspective categorizes sexual assault as forcible, statutory, attempted, canal knowledge of juvenile or crime against nature. Rape is also categorized according to site of anatomic assault (Oral, anal, vaginal), and according to the extent of penetration [non, slight or full] (2). Our patient fell in the category of forced penetrative vaginal sexual assault.

Informed consent followed by accurate detailed history and proper examination are essential for the diagnosis and accurate documentation is important for legal proposes and treatment. The crime should be reported to the police for investigation and prosecution. Acute injuries must however be stabilized as early as possible. About 1% of the injures require admission while 0.1% are fatal in the UK (2,3). History, examination and investigation were done for our patient. Documentation and reporting were also done in

special police forms and statement made to the police. Our patient was however not admitted because she was stable.

Previous obstetric and gynaecologic conditions should be sought particularly infections, obstetric and gynaecologic conditions should be sought particularly infections, pregnancies, use of contraceptives, date of the last menstrual flow. Pre-existing pregnancy and infections should be rule out as far as possible (2). All this was considered in our client.

Physical examination should document bruising and tears and samples for microbiologic examination should be taken as were taken. Wood light should be used to find semen on patient's body. This was not available in our set up. Serum sample for HIV syphilis and hepatitis A and C should be obtained. High vaginal swab should be taken for microscopy to look for spermatozoa as evidence of penetrative vaginal intercourse. This is best done as soon as possible after the crime is committed to prevent loss of evidence. The victim should preferably be examined before taking a bath and should preserve their clothes as further evidence (2). The advice by the mother to take a shower and sleep overnight before coming to hospital though humane and posed no immediate danger to our patient, can lead to loss of evidence. Despite this, spermatozoa were still retrieve from the victim's vaginal canal. All the efforts were however still frustrated by the inability of the police to apprehend the culprits.

Swabs should also be taken for culture of bacteria for possible sexually transmitted infection. Swabs for culture of *Neisseria gonorrhoea* should be transported in Stuart media and cultured in Thayer Martin or transgrow media. Tissue culture is needed for culture of *Chlamydia trachomatis*. Polymerase chain reaction can be used to isolate HIV and hepatitis virus (2,4). These were not available for our patient.

Patients at risk for pregnancy as a result of assault should be offered emergency contraception. 2-4% or sexual assault victims not already using contraception conceive from the act. Any of the following methods if properly used are highly effective in preventing pregnancy after sexual assault (5,6,7):

1. Combined oestrogen and progesterone 100-120μg of ethinyloestrogen and and 0.5mg of levonogesterol is given and repeated after 12 hours. These reduce risk of pregnancy by 75%.

2. Progesterone only pill containing 0.75mg of levonogesterol taken also within 72 hours of assault and repeated after 12 hours.

3. Copper-based intrauterine device can be inserted up to 5 days after assault with high efficacy but poses the risk of exacerbating sexually transmitted infection that may be transmitted during the assault.

4. Mifepristone 600mg has higher efficacy in preventing pregnancy than combined oestrogen and progesterone regime.

Our patient received progesterone only prophylaxis and pregnancy test was negative 4 weeks later.

A VDRL test should be obtained at the time of initial visit and repeated after 3 months. Serology for HIV should be done initially with repeats at 3,6, and 12 weeks. Our client was counselled for the above and consented. The initial test and repeat at six weeks were negative and the last at 12 weeks was pending. Hepatitis B and C antibodies should also be checked as a base line and Hepatitis B Immune globulin given at 0.06 ml per kg body weight at 0, 1 and 6 months later. This was not available at our centre but was given at St. Mary Mission Hospital.

HIV prophylaxis with zidovudine, lamivudine, and efavirence for 28 days is recommended by Centre for disease Prevention and Control (CDC), for

high-risk occupational and non-occupational exposure. Data is however not available on its effectiveness but has been widely adopted for sexual assault victim (8). This was done for our client. HIV test was negative at one month but is yet to be tested at 3months.

Prophylaxis against other sexually transmitted infection should be provided with antimicrobials effective against chlamidial, gonococcal and trichomonal infections (2). Our patient received norfloxacin, doxycycline and metrodidazole and did not show symptoms of infection.

Thorough counseling for posttraumatic stress disorder is essential for victims of rape (2). Our patient was counseled and is on follow up at patient support centre.

REFERENCES

1. Chan P.D, Winkle C.R. Sexual assault in current clinical strategies 2002; **7**: 52-54

2. Halfin P.V., Lu J.S. Lu M.C. Domestic Violence and Sexual Assault. Current Obstetric and Gynecologic diagnosis and treatment 9th Ed. 2003 McGRAW-HILL; **60**:1087-1093

3. Petter L.M., White H. Management of Sexual Assault. American Family Physician 1998; **9**: 19-22.

4. Dutta D.C. Genital tract injuries. Textbook Of Gynaecology, 3rd Ed.2001. New Central Book Agency, Culcutta; **25:** 395-402.

5. Trussell J., Ellertson C., Stewart F. Emergency contraception . A costeffective approach to preventing pregnancy. Women Health Primary Care 1998a: **1**:52.

6. Glassier A., Thong K.J., Dewar M., etal. Mifepristone compared with high-dose oestrogen and progesterone for emergency postcoital contraception. N Engl J Med 1992;**327**:1041.

7. American College of Obstetricians and Gynecologists. FDA approves new emergency contraception drug. ACOG Today 1999a;**43**:7.

8. HIV non-occupational post-exposure prophylaxis. New York State department of Health AIDS institute. NYSDOH AI

GYNAECOLOGY CASE 12

BURST ABDOMEN: SUCCESSFUL REPAIR

NAME: J.W. I.P NO: 0890905

AGE: 26YEARS DOA: 31/12/04

PARITY: 2+0 DOD: 12/01/05

PRESENTING COMPLAINTS

J.W. complained of copious watery discharge at the incision site for one day on the fourth postoperative day.

HISTORY OF PRESENTING COMPLAINTS

J.W. was doing well following ceasarian section for heavy active antepartum haemorrhage due to abruptio placenta up to the 3rd postoperative day. On that day around midnight she felt pain at the operation site and had a feeling of something giving way during after a coughing episode. She had developed cough since she left theatre and had earlier in the day been put on a cough suppressant. She reported this to the nurse in the ward but was thought to be all right. She was reassured and went back to sleep. Later that night, she felt wet and woke up to find herself with watery discharge draining from the incision site. It is at this point that a doctor was called to review her and diagnosed burst abdomen.

OBSTETRIC AND GYNAECOLOGICAL HISTORY

She was a Para 2 + 0. Her first delivery was in the year 2000 through caesarian section due to foetal distress. The outcome was a live male infant who weighed 3000g and is alive and well. Her last delivery was four days earlier through an emergency caesarian section due to active antepartum haemorrhage at 37weeks. The outcome was a life female infant who had an apgar score of 3/1, 5/5 and 8/10. The baby was admitted to nursery for two

days and had shown great progress. He had been reunited with the mother whereby breastfeeding was successfully initiated.

Her last menstrual period was on 02/03/04 and was at a gestation age of 37weeks and 1day at the time of caesarian section. The antenatal period had been uneventful until the onset of the bleeding and abdominal pain on the day she was operated on. She reported a fall while cleaning her house before the onset of bleeding. She had attended antenatal clinic at Pumwani Maternity Hospital and her antenatal profile was as follows: Haemoglobin was 11.2g/dl, blood group O rhesus positive, VDRL and HIV were negative.

Her menarche was at 15years of age and experienced periods regularly every 23-26days with a flow of 3-4day. She used intrauterine contraceptive device after her first delivery.

PAST MEDICAL HISTORY

She had no other admissions except during her previous delivery. She did not suffer from chronic illnesses and had no drug or food allergies.

FAMILY AND SOCIAL HISTORY

She was married and lived with her family in Nairobi. There was no family history of chronic illnesses. She was a housewife while the husband was a casual labourer. She did not smoke or drink alcohol.

PHYSICAL EXAMINATION

Review in the ward revealed that she was in good general condition had mild pallor but no jaundice or oedema. Her vital signs were; blood pressure of 100/60mmHg, pulse 92/minute, respiratory rate 22/minute and a temperature of 36.8^{0}C.

Her respiratory, cardiovascular and nervous systems were found to be normal.

ABDOMINAL EXAMINATION

The abdomen was heavily dressed with gauze, which was wet. Removal of the dressing revealed a sub-umbilical midline incision from which was draining serosanguenous fluid. The incision looked intact except for a small point below the umbilicus through which the omentum herniated.

DIAGNOSIS

A diagnosis of complete wound dehiscence with impending burst abdomen was made.

MANAGEMENT

She was informed of the diagnosis and consented for emergency repair under general anaesthesia. Investigations done showed a haematocrit of 29%, urea, creatinine and electrolytes were normal. A blood sample was taken for cross matching. She was then premedicated with atropine 0.6mg and pethidine 50mg and taken to the operating theatre.

In theatre and under anaesthesia she was reexamined and earlier finding confirmed. The uterus was found to be at 14weeks pregnancy equivalent and was discharging non-foul-smelling lochia. The incision was cleaned with hibitane and painted with iodine solution before draping with sterile towels. Skin stitches were removed where it was found that all the other layers had given way. The edges were clean, there was no pus and the uterus was well contracted. The uterine incision was intact and was healing well. The abdomen was cleaned with rifocin, and freshening of the incision edges done. Mass closure of the abdominal wall was then done using looped

number 2 nylon suture. The skin was closed with number 2/0 nylon suture through interrupted mattress method then cleaned and dressed.

POST-OPERATIVE MANAGEMENT

She was put on intravenous augmentin, metronidazole and pethidine for analgesia. These were changed to oral augmentin, metronidazole, diclofenac and ferrotone when she was able to tolerate oral intake. She did well post-operatively and the wound was well apposed when it was opened on the fourth day after repair. 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, was in good general condition and the incision site had healed well.

DISCUSSION

The patient presented was admitted with antepartum haemorrhage and emergency caesarian section. She developed a cough after the operation leading to a burst abdomen on the fourth postoperative day. Repair was done subsequent to which she healed well and was allowed home in good condition.

Burst abdomen or abdominal wound dehiscence refers to separation of layers of abdominal incision. It is divided into superficial and complete. Superficial dehiscence refers to separation of the skin and superficial layers and may include up to the fascia but not the peritoneum. When the peritoneum also separates, the complication is called complete wound dehiscence. When intestines protrude through the incision, the term evisceration or burst abdomen is used (1,2). J.W. had complete wound dehiscence with impending burst abdomen.

The frequency of wound dehiscence varies from centre to centre with a range between 0.3-3% of all pelvic surgery. It is more common in general surgery patients than gynaecological patients (3). Mortality of as high as 35% was reported in older literature after evisceration but more recent study found only 2.9% (4,5). Our patient had wound dehiscence associated with less contamination of viscera compared to burst abdomen. Prompt action prevented development of burst abdomen.

Factors predisposing to wound dehiscence include (6,7):

1. Metabolic like malnutrition, uncontrolled diabetes mellitus and use of corticosteroids.

2. Mechanical factors such as obesity, retching, ascitis and coughing.

3. Processes that impair wound healing like radiotherapy and cytotoxic therapy.

Types of incision and sutures have been disputed as significant factors in wound complications (8,9). The presented patient had cough after surgery probably due to intubation during general anaesthesia. She had moderate obesity judged from general examination but not confirmed by calculation of body mass index .

Sepsis and qualities of closure are important factors in wound dehiscence. Infection causes accelerated dissolution of catgut suture material, causes formation of granulation tissue and hinders healing. Tight sutures cause areas of ischaemia with necrosis leading to wound disruption while inadequate haemostasis cause haematoma formation and incision edge separation (10,11,12). There was no haematoma or evidence of wound infection.

One of the most common signs of complete wound dehiscence is the presence of a serosanguineous discharge from the incision. When this is discovered, a sterile towel should be placed over the incision and firmly held with adhesive tape to prevent burst abdomen, minimize fluid loss and risk of infection. In burst abdomen, the bowel should be carefully replaced into the abdominal cavity and packed with abdominal pack soaked in povidone-iodine solution. These actions are however not replacement for prompt repair of the wound and this must be done as soon as patient's condition allows (1).

Burst abdomen is a surgical emergency and requires early recognition and prompt treatment. Immediate closure of the disrupted incision is indicated. However proper preoperative preparation must be done for good outcome. Intravenous access and resuscitation must be done. Analgesia and anxiolysis are important. The role of prophylactic antibiotics is unproven (1).

Secondary repair is undertaken with a general anaesthesia. Debridement and freshening of incision edges should be done before wound closure. Samples of discharge from the wound should be taken for microbiologic analysis and culture. Peritoneal lavage should be done with a lot of warm normal saline (1). Fascia closure can be done separately with interrupted Snead-Jones far-far and near-near technique if it can be well identified and is not rugged. When rugged and poorly identified however, better results are obtained with mass closure. In both cases, non-absorbable or slowly absorbable high-tension sutures like number 2 nylon or polypropylene should be used. (1,13). The skin can be closed primarily if there is no evidence of infection or otherwise left open for delayed closure (1). J.W. had a clean incision site but fascial edges were not easily identifiable hence mass closure was done. The skin was closed during the repair since there was no evidence of infection with good outcome.

REFERENCES

1. Rock J.A., Horowitz I.R., Dominguez C.E. Incisions for gynecologic surgery. Te Lindes gynaecologic surgery, 9th Ed. 2001; Lippincot-Raven Publishers, Philadelphia **12**: 255-289.

2. Dutta D.C. Practical gynaecology. Text Book of Gynaecology 3rd Ed.2001; New Central Book Agency, Culcatta. **35**: 565-602.

3. Keill R.H., Keitzer W.F., Henzel J. Abdominal wound dehiscence. Arch Surg 1973; **106**:573.

4. Rollins R.A, Corcoran J.J., Gibbs C. E. Treatment of gynecologic wound complications. Obstet Gynecol 1966; **28**:268.

5. Helmkamp B.F. Abdominal wound dehiscence. Am J Obstet Gynecol1977; **128**: 803.

6. Greenburg G., Salk R.P., Peskin G.W. Wound dehiscence: Pathology and prevention. Arch Surg 1979; **114**: 143.

7. Jurkiewicz M.J., Morale L. Would healing, operative incisions and skin graft. Hardy's Textbook of Surgery. J.B. Lippincot Co Philadelphia, 1983;108.

8. Hendrix S.L., Shimp V., Martin J. Legendary superior strength or Pfannestiel incision: A myth. Am J Ostet Gynecol 2000; **182**: 1446.

9. Stone H.H., Hoffling S.J., Strom P.R. Abdominal incisions, transversevs.vertical placement and interrupted vs. continuous closure. South Med J 1983; **76**: 1106.

10. Dineen P. A critical study of 100 conservative would infections. Surg Gynecol Obstet 1961; **113**: 91.

11. Fletcher H.S., Joseph W.L. Bleeding into the rectus abdominis muscle. Int Surg 1973; **58**:97.

12. Sinei S.K.A. Post caesarean section febrile morbidity at Kenyatta N. Hospital. M.Med Thesis University of Nairobi 1981.

13. Baggish M.S., Lee W.K. Abdominal wound disruption. Obstet.Gynaecol 1975; **46**:530

GYNAECOLOGY CASE 13

SUSPECTED GENITAL TUBERCULOSIS: SUCCESSFUL THERAPY

NAME: M. M. IPNO: 0870451

AGE: 32YEARS DOA: 17/02/04

PARITY: 4+1 DOD: 28/02/04

PRESENTING COMPLAINTS

She was admitted to the Ward as a referral from Dandora health with suspected pelvic tumour. She had presented with 8 months history of ammenorrhoea and 6 months of pelvic pain and 5 months of low abdominal swelling.

HISTORY OF PRESENTING COMPLAINT

She had generally been unwell with general malaise but no specific symptoms. Eight months prior to admission in our hospital, Her menstruation had ceased following a spontaneous abortion at 2 months. This was followed two months later by abdominal pain and in another month by low abdominal swelling. The pain was dull ache in nature, which was progressively increasing in intensity but was non-radiating. The swelling was also progressively increasing in size.

She had been seen at Mwingi hospital where a pregnancy test had been repeatedly negative and had been put on antibiotics severally without improvement. She had moved to Nairobi to seek help from her brother. She had been taken to Dandora Health Centre from where a pelvic scan had been done. The scan had showed complex bilateral ovarian masses measuring 6x4cm on the right and 5x5cm on the left leading to referral.

OBSTETRICS AND GYNAECOLOGY HISTORY

She was Para 4+1. All her deliveries were vaginal at home with the help of a traditional birth attendant. All the children were alive and well. The last delivery was in the year 2002.

Her menarche was at 16years. Her periods were regular with a flow of 3-4 days, and a cycle of 28-32day. The periods were moderate in amount and were painless. She could not remember her last menstrual period but had not seen any since an abortion 8moths earlier. She had never used any contraceptives and had not suffered any sexually transmitted infection.

PAST MEDICAL HISTORY

She had never been admitted before. She had been feeling unwell on and off for a long time but had not been diagnosed to suffer from any chronic illness. She did not suffer from any drug or food allergies.

FAMILY AND SOCIAL HISTORY

She was a housewife married to tyre-shoes dealer at Seikulu shopping centre of Mwingi District. She was the fourth born in a family of 8 sibblings. She had nursed her brother for 3 months who was suffering from pulmonary tuberculosis 4years earlier. The brother had later died from the PTB despite medication. Her first born son had also been coughing for over a month but had not been tested for PTB. The other member of her family had no complaints and none was known to suffer from PTB or any other chronic illness.

PHYSICAL EXAMINATION

M.M. was sick looking, wasted and pale but had no jaundice, oral thrush, cyanosis, oedema or lymphadenopathy. Her vital signs were as follows: Blood pressure was 130/80 mmHg, pulse rate 82/minute, respiratory rate 18/minute and temperature 37.2^{0}C

CENTRAL NERVOUS SYSTEM

She was fully conscious. The neck was soft and Kernings sign was negative. There was no lateralising sign.

CARDIOVASCULAR SYSTEM

Her pulse was regular, of good volume and non - collapsing. The apex beat was not displaced and both first and second heart sounds were heard and normal. No murmurs were heard.

RESPIRATORY SYSTEM

She had no respiratory distress. Percussion note was normal and breath sounds were equally distributed in both lung fields. No crepitations or rhonchi were heard.

ABDOMINAL EXAMINATION

The abdomen was slightly distended more to the flanks but was moving with respiration. There was mild generalized tenderness over the abdomen more severe in the suprapubic and iliac fossae. There was guarding on deep palpation but no rigidity. Fluid thrill and shifting dullness were positive. There were vague masses in both iliac fossae but no splenomegaly and hepatomegaly though guarding hindered examination

VAGINAL EXAMINATION

She had normal external genitalia. The uterine size and mobility was difficult to establish due to tenderness, however the cervical os was closed and smooth. Both adnexal regions were tender and pouch of Douglas felt boggy. Vague adnexal masses were felt though difficult to appreciate clearly due to tenderness. There was bilateral cervical motion tenderness. Speculum examination revealed normal vagina and cervix.

INVESTIGATION

1. Full haemogram: Haemoglobin-7.6g/dl, WBC-$14x10^9$/L. N-30%, L-60%, M-6%, E-4%. RBCs were normocytic hypochromic.
2. ESR: 48 using the Westergren method
3. HIV test was negative
4. Pregnancy test was negative on repeat. (Repeat on 10/02/04)-Negative

5. Repeat abdominal pelvic ultrasound (10/02/2004). The uterus was normal in shape and size. The endometrial strip was not sonographically appreciable and there was no intrinsic uterine lesion. Bilateral multiple complex adnexal masses were seen the largest of which was 4x4cm. There was moderate amount of intra-peritoneal fluid.

6. Chest x-ray showed exaggerated lung markings but no cavities or pleural effusion. Heart shadow was normal.

7. Sputum analysis for alkali and acid fast bacillus was done three times and was negative.

Mantoux test kits were out of stock in the country at the time and she could not afford CA 125. Diagnostic laparotomy was considered but therapeutic trial was done first due to her general condition the high likelihood of TB.

DIAGNOSIS

A differential diagnoses of pelvic tuberculosis or ovarian neoplasm was made.

MANAGEMENT

A decision was made to put her on TB trial therapy. Chest physicians reviewed her and concurred with the management. She was 48kg and was started on rifater 4 tablets once daily, pyridoxine and ranferon. High energy and high protein diet was also advised. She was discharged home on the medication for review in GOPC and chest clinic in 2weeks but because of financial constraints, she was not able to leave the hospital. Towards the end of the two weeks when she was ready to go home, it was agreed that she should not go home but wait for review at the postulated time.

During the two weeks when she was in the hospital, she continued receiving rifater and high-energy high-protein diet. She made marked improvement both subjectively and objectively. She reported feeling better, cough

reduced, abdominal pain reduced and gained half a kilogram of weight. She was reviewed again by the chest team, which was impressed by her changes and recommended her to be discharged through their clinic for followup. The public health team was to work in liaison with the Mwingi District public health team for screening of the close family member especially those under 5 years. She was booked for review in GOPC also in four weeks.

FOLLOW UP

She came for followup as booked. She had made remarkable improvement. She did not experience night-sweats or abdominal pain and swelling she had earlier experienced had subsided. She felt well and had resumed her normal chores. She had gained 2kg weight from the weight she had at onset of therapy. Her general condition was good and vital signs were normal. She did not have any abdominal tenderness or palpable masses. She had not resumed her menses but had also not resumed sexual activity. However she still had mild to moderate adnexal tenderness and cervical motion tenderness. No adnexal masses were felt. Haemogram, ESR and pelvic ultrasound done showed the following:

1. Haemogram: Haemoglobin was 9.8g/dl, WBC were 7.2 x 10^9/l, platelets 192 x 10^9/l, N-47%, L-49%, M-3% and E-1%

2. ESR was 34 with Westergren method.

3. Pelvic ultrasound scan: The uterus was retroverted but of normal size. There was no myometrial lesion but the endometrial strip was not appreciated. The adnexa appeared normal and masses earlier noted had regressed but showed calcification in the adnexa.

Other members of the family including the husband had been screened for tuberculosis at Mwingi District Hospital but none had evidence of the disease. She was transferred back to Mwingi Distict Hospital for further

treatment and followup after completion of the intensive phase, to ease cost and reduce chances of abscording.

DISCUSSION

The patient presented is a 32year-old Para 4 + 1 who was wrongly diagnosed as having ovarian malignancy, but turned out to have upper genital tract tuberculosis.

Pelvic tuberculosis is part of a wider topic; genital tuberculosis. It is a chronic granulomatous inflammatory disease caused by mycobacteria involving the genital tract; vulva, vagina cervix, endometrium, fallopian tube and the ovaries and the neighbouring structures. Tuberculosis of the genital tract is rare in the developed world just as is tuberculosis in general. This is however not the case in the developing countries of Asia, Middle East, Latin America and where other forms of tuberculosis are also common. Migration and HIV/AIDS are causing resurgence of the disease in these countries (1,2,3).

Various studies give different prevalence's of genital tuberculosis with an incidence in the range of 0.69 - 19% reported. With the advent of HIV/AIDS, the incidence of genital tuberculosis has gone up, as tuberculosis is one of the opportunistic infections (1,4,5). The patient presented was screened for HIV antibodies and was found negative.

Tuberculosis of the genital tract is caused primarily by either *M. tuberculosis* or *M. bovis*. Infection by *M. tuberculosis* usually has a primary involvement in the lungs and other sites with the exception of hair, teeth and nails. Usually it is the human bacillus -*M. tuberculosis,* which is responsible for pelvic tuberculosis. If the pelvic disease is secondary to tuberculous peritonitis, lymph nodes or bowel disease, then the bovine bacillus is likely

to be the cause. The disease may spread during the primary disease, secondary reactivation and rarely during the latent phase (1,3,6). In our patient, it was not established which mycobacterium was responsible for her disease since it was not isolated but it was suspected to be *M. tuberculosis* because of suspected but not confirmed previous chest disease.

The routes of spread to the reproductive tract are haematogenous, lymphatic system and by direct extension from abdominal lymph nodes and adjacent abdominal organ. During miliary tuberculosis haematogenous spread occurs to the whole body. Miliary tuberculosis is a feature either of primary disease or secondary reactivation usually due to an immunosuppressive event. The haematogenous route is responsible for spread of the latent phase disease during the period of rapid growth of puberty (1,6). Lymphatic spread can occur from a primary pulmonary site to intestinal lymph nodes and then to the pelvis by direct extension to the pelvis (1,6,7). The patient presented did not have an active pulmonary lesion but it is probable that she had latent disease in the lungs. Haematogenous spread may have occurred from here either during the primary infection or the considered latent phase. In absence of evidence however, this remains speculative and the possibility of primary intestinal lymph node infection with subsequent direct extension to the pelvis cannot be ruled out.

Sexual transmission of genital tuberculosis has been reported with primary genital infection in the woman occurring after coitus with a sexual partner who had penile tuberculosis (8). This route of spread was unlikely for our patient because the spouse was not found to have tuberculosis at screening. The screening was however for pulmonary tuberculosis with a chest x-ray and could not therefore eliminate the possibility of genital disease absolutely

The fallopian tubes are affected in 90-100% of cases, with the cervix in 5-15% and the vulva and vagina in 1%. The endometrium is affected in 50%

of patients with tuberculous salphingitis while only 30% of such patients have ovarian involvement (1,3,6). The patient presented had 'fallopian tube, ovarian and endometrial involvement with destruction of the endometrium as suspected from ultrasound scan.

Infertility is the commonest symptom of pelvic tuberculosis and is found in 70% of patients. Menstrual disorders are found in 50%, of which 54% have oligomenorrhoea, 19% menorrhaghia, 14.3% ammenorrhoea and 1.6% menopausal and postmenopausal bleeding. Pelvic pain is present in 20-30% of patients with pelvic tuberculosis. Painful and painless ulcers are found in vulval and vaginal tuberculosis respectively. Systemic features include general malaise, weight loss, night sweats and fever. Some patients are however completely asymptomatic and are discovered during laparoscopy/laparotomy for other indications like tubal ligation. Bilateral adnexae tenderness is usually the rule though less marked than with acute gonococcal infection and a fifth of the cases present with clinical ascitis (2,3,9,10). Our patients had systemic features of malaise, weight loss and night sweats. She also had features more specific to the pelvis like amenorrhoea and pelvic pain. Adnexal tenderness and ascitis were also present in our patient. Amenorrhoea could have resulted from destruction of the endometrium with development of uterine synechiae or from chronic illness. Features of vulval, vaginal and cervical disease were not present.

Definitive diagnosis can only be made by positive culture in Lowenstein-Jensen media or guinea pig inoculation. Demonstration of the alcohol and acid-fast bacillus (AAFBs) in specimen is limited because of other non-tuberculous organisms that are alcohol and acid fast. Specimen that can be obtained with least-invasive method should be obtained for analysis. Sputum, biopsy of vulval, vaginal and cervical lesions should be done where pulmonary, vulval, vaginal or cervical disease respectively are suspected.

Lymph biopsy should also be done when suspicious nodes are encountered. Endometrial curettage for histology, menstrual blood for culture or guinea pig inoculation can also be tried for endometrial tuberculosis. Laparoscopy can also be used for direct visualization and biopsy. Other investigation, which provide less direct evidence include full haemogram with differential white cell count and ESR, pelvic ultrasound scan, hysterosalpingogram, chest x-ray and mantuox test. The latest and most sensitive test providing indirect evidence of tuberculosis is using polymerase chain reaction and radiolabelled DNA or RNA probe identification (1,3,6,9).

Our patient had a pelvic ultrasound scan, which showed mixed echo-pattern tubo-ovarian masses. These reduced in size markedly in six weeks of anti-tuberculosis therapy. Haemogram showed marked relative lymphocytosis and higher normal white cell count. ESR was markedly elevated. She had normocytic hypochromic anaemia usually seen in patients with chronic illness. These tests though non-specific are highly suggestive chronic inflammatory process likely to be tuberculosis in our enviroment. Chest x-ray was suggestive of chronic lung inflammation but sputum analysis for AAFBs was negative. Mantoux test was not done due to unavailability purified protein derivative. It was likely to be positive in her case. Chest x-ray may appear normal in Latent or early active disease. Latent disease was more likely for our patient. Vulval and vaginal biopsies were not done since there were no lesions. Endometrial biopsy could probably have found evidence of chronic granulomatous inflammation, which includes; numerous tubercles composed of caseation material, multinucleated giant cells of langhans and regions of central necrosis or even the tubercle bacilli (7). Laparoscopy or laparotomy for biopsy would probably also have provided similar evidence besides direct visualization of pelvic lesions. However the risk of anaesthesia cannot be justified in a very sick patient where other tests

are highly suggestive of tuberculosis. In such a situation a therapeutic trial is the better option. This was the basis of management and the response justified it.

Culture for tubercle bacillus, guinea pig inoculation and polymerase chain reaction are expensive and not routinely used for diagnosis. They are used in research and in situations where resistance to treatment is suspected (9).

The primary treatment of genital tuberculosis is chemotherapy. Standard drug treatment includes rifampicin, isoniazid and pyrazinamide for 2 months followed by administration of 2 drugs i.e., isoniazid and rifampicin or isoniazid and ethambutol for six more months. Patients whose histology is positive for tubercle bacilli a third drug; ethambutol is added during the first 2months of intensive therapy. If the patient is HIV positive - drug treatment should be continued for 9 months. The drugs are in fixed dose combinations to reduce pill burden. The combinations are of rifampicin, isoniazid and pyrazinamide in one tablet called rifater and another containing isoniazid and pyrazinamide called rifina. Pyridoxine supplementation should also be given to prevent peripheral neuropathy in patients with slow metabolism (slow acetylators) of isoniazid (11). Another fixed dose combination containing rifampicin, isoniazid, pyrazinamide and ethambutol called riva-4 was introduced in mid-2004 and is now in use. Our patient was started on rifater for 2 months and continued with rifina for the next 6 months. She was also given pyridoxine and did very well. Iron and folate (ranferon) was given to provide for the expected increase in marrow activity following treatment of tuberculosis. This was expected to raise the haemoglobin back to normal.

Surgical treatment is reserved for (1,2,6,12):

1. Persistent or increase in size of adnexal mass after 4-6months of consistent anti-tuberculosis treatment.

2. Persistence or recurrence of pelvic pain while on anti-tuberculosis treatment.

3. Anti-tuberculosis treatment resistance as shown by persistent fever, leukocytosis, persistently high ESR or positive tissue biopsy.

4. A patient who is likely to abscond from treatment.

5. Some author recommend routine surgery for all patients after six weeks of full anti-tuberculosis drug treatment.

The preferred surgical treatment includes total abdominal hysterectomy and bilateral salpingo-oophorectomy, though conservation adnexectomy should be carried out in young patients who are eager to attempt future childbearing. Such patient should be forewarned that conservation surgery will be performed only if the disease is minimal and ovarian conservation possible only if it involves the surface (1). Our patient was spared from surgery by good response to chemotherapy.

About 5% of patients with genital tuberculosis are capable of becoming pregnant and only 2% carry a pregnancy to term. Pregnancy is extremely rare in the presence of pelvic tuberculosis or tubo-ovarian abscess and conservation surgery for the purpose of preserving fertility is unwarranted (10). The high incidence of infertility has been disputed by Prof. B.J. Wadia of Grant Medical college and J.J. Hospital in Mumbai India, who found evidence of pelvic tuberculosis during laparoscopic tubal ligation. These patients had enjoyed undisturbed fertility up to the time of tubal ligation (2). It is possible that those only patients with fairly advanced disease experience the high incidence of infertility. Our patient had not conceived for eight months despite unprotected coitus.

REFERENCES

1. Rock J.A., Damario M.A. Pelvic inflammatory disease. Te Lindes Operative Gynecology 9th ed.2001;Lippincott William and Wilkins Philadelphia.28:675-708.

2. Wadia B.J., Pelvic tuberculosis. Oration; Golden Jubilee, National conference on tuberculosis and chest diseases. Ind. J. Tub. 1996; 43: 125.

3. Rajamaheswari N. Pelvic tuberculosis. C:\TITLE OF THE PAGE Pelvic TB.html, E.Mail: dr_nrm@sify.com <mailto:dr_nrm@sify.com>.

4. Emembolm J. O., Anyauwa D. O., Ewa B. Genital tuberculosis in infertile women in Northern Nigeria. West Afr. J. Med. 1993; 12(4): 211-2.

5. John M., Kullkady T. A. Genital tuberculosis and infertility. Int. J. Obstet. Gynecol. 1999 164(2); Page 193.

6. Dutta D.C. Pelvic infections. Textbook of gynaecology, 3rd Ed. 2001; New Central Book Agency, Culcatta. 9: 116-136.

7. Anderson J.R. Chronic bacterial infections (infective granulomas), Muir's Tectbook of Pathology. 12th Ed. 1985;Butler and Tanner, London. 9: 15-22

8. Sutherland A.M., MacFarlane J.R. Transmission of genital urinary tuberculosis. Health Bull (Edinb), 1982; 40: 87.

9. Kumar P., Clark M. Respiratory diseases. Kumar and Clark Clinical Medicine, 5th Ed 2003. W. B. Saunders Edinburgh, 14: 833-920.

10. White Field C. R., Pelvic tuberculosis. Dewhurst Textbook of Obstetrics and Gynaecology for postgraduates 4th Ed. 1986; Blackwell. **19**: 604-6.

11. Guidelines for Tuberculosis and Leprosy Control for Health Workers in Kenya. 2000 Ed. Ministry of Health. Nairobi, Kenya.

12. Sutherland A.M., Surgical treatment of tuberculosis of the female genital tract. Br. J. Obstet. Gynecol. 1980; 87: 670.

GYNAECOLOGY SHORT CASE 14

INCOMPETE ABORTION – MANUAL VACUUM ASPIRATION

NAME	J.M.	L.M.P.	10-12-06
AGE	21YRS	PARITY	1+0
OP NO.	4279		
DOA	1-03-2007		
DOA	1-03-2007		

PRESENTING COMPLAINTS

She presented with per vaginal bleeding, backache and lower abdominal pain for eight hours prior to arrival in hospital.

HISTORY OF PRESENTING ILLNESS

J.M. was well until the morning of the consultation when she started experiencing lower abdominal pains. This was accompanied by backache and soon followed by vaginal bleeding that was of moderate amount. She had changed pads four times by the time she arrived. She also reported to be dizzy although she could walk unaided. She had been seen at a clinic near where she lived where she was given some analgesics and referred to KNH. She had missed her periods for three months. She did not have fever and the blood was not foul smelling. She denied any interference with this pregnancy.

OBSTETRICS AND GYNAECOLOGY HISTORY

She attained her menarche at 13 years. Her last menstrual period was 10-12-2006 giving her a 12 week period of amenorrhea at the time of presentation. Her menstrual cycles were regular occurring every 28 days and duration of bleeding of 4 days. She gave no history of dysmenorrhea.

She was a para 1+0. She delivered in 2004 in Kericho district hospital vaginally after labor that lasted 11 hours. She used depo provera in the year 2005 but stopped due to her desire to get pregnant again.

PAST MEDICAL HISTORY

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

FAMILY SOCIAL HISTORY

She was the third born in a family of seven siblings. There is no history of chronic illnesses in her family. She has been married since 2003 and is a housewife. Her husband is a janitor and they live in south C.

SYSTEMIC REVIEW

This was nil contributory.

PHYSICAL EXAMINATION

She was in fair general condition, was mildly pale, was not cyanosed and had no edema. 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 although 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 to bimanual palpation. Both adnexa 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 was 84 per minute, regular and of normal volume. Her blood pressure was 100/60. The apex beat was normal and she had no murmurs.

RESPIRATORY SYSTEM

The respiratory rate was 24 breaths per minute. She was not in respiratory distress. Air entry was equal bilaterally and no added breath sounds were heard.

NERVOUS SYSTEM

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

IMPRESSION

An impression of incomplete abortion was made.

INVESTIGATION

None

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 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:

She 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. Her bladder was emptied and a bimanual examination done which confirmed the above findings. A cuscos speculum was inserted into the vagina and the cervix exposed. The cervix was cleaned and clots expelled. The cervix was then inspected for any lacerations or injuries and none were found. The anterior cervical lip was then grasped with a tenaculum forceps. Using no touch technique, canula size 12 was inserted into the uterine cavity up to the fundus while all the time applying traction to the tenaculum. The aspirator was then attached to the canula and the vacuum released. The uterine contents were evacuated by gently and slowly rotating the canula 180° with concurrent in and out movements. The procedure was judged complete when we noted that reddish/pinkish foam was being suctioned, a gritty sensation was encountered, the uterus became well contracted and was gripping the canula and that the patient complained of increased pain and cramping. Approximately 80 mls of products of conception were evacuated which were not foul smelling. The 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 cleaned and the patient was provided with a perineal pad to monitor any bleeding.

POST MVA CARE

The patient was taken to the recovery place and was put on metronidazole 400mg eight hourly and doxycycline 100 mg twice daily to cover for secondary infection. She was also put on ranferron 10ml daily and ibuprofen 400mg three times daily for pain relief. Her condition stabilized and she was discharged home on the same day. She was advised on contraception and booked to the family planning clinic for follow up.

FOLLOW UP

She did not honor her appointment.

DISCUSSION

A case is presented of a 21 year old para 1+0 with spontaneous incomplete abortion at a gestation of 12 weeks that was managed by manual vacuum aspiration and discharged home on antibiotics.

Abortion is defined as termination of a pregnancy that is less than 20 weeks (139 days) gestation or a fetus that is less than 500g in weight. The weight of 500g according to world health organization is the bare minimum at which a fetus can survive[1]. Abortion can be classified as spontaneous or induced. Our patient had spontaneous abortion as there is no evidence that the pregnancy was interfered with. Abortion can be further classified as complete, incomplete, inevitable, missed and threatened abortion. Our patient had incomplete abortion. An abortion can also be septic or non-septic. Our patient did not have any signs of sepsis. 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; hemorrhage, sepsis, perforation of the uterus and 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].

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 fetus and placenta are unlikely 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. Sometimes patients may not volunteer information on having procured abortion due to the legal implications. It is estimated that worldwide, 50 million pregnancies are terminated each year.

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 it is done on medical grounds and thus most patients presenting with induced abortion do not volunteer information on interference. Legal abortion was introduced in South Africa in 1997 making it one of the few countries in Africa with such legislation[3-7].

Although histological examination was not carried out on the evacuated material due to the high costs that had to be met by the patient, no identifiable cause was found in the patient presented. Causes of spontaneous abortion are varied. Chromosomal anomalies cause 50-60% of first trimester abortion. Autosomal trisomy is the most frequently identified chromosomal anomaly associated with abortion followed by monosomy (45XO). Abnormal zygote development,, polysomy and triploidy are the other abnormalities associated with abortion. Other causes include infection, anatomical defects, endocrine factors, immunological factors, maternal systemic disease, emotional factors and trauma. Hyperthyroidism, hypothyroidism, diabetes mellitus and infections such as the TORCHES (Toxoplasmosis, Rubella, Cytomegalovirus, Herpes simplex and Syphilis), brucellosis, mycoplasmosis and malaria are all known to cause abortion. Insufficient progesterone from the corpus luteum of pregnancy or the placenta also causes abortion. Environmental factors or habits such as excessive alcohol consumption, cigarette smoking also increase the risk of abortion. Antiphospholipid antibody and connective tissue disorders such as

rheumatoid arthritis, systemic lupus erythromatosis have also been associated with increased risk for abortion and recurrent fetal death. Uterine defects like fibroids, synaechiae, mullerian tube defects and cervical incompetence are associated with recurrent pregnancy losses[1,4,5,7,8,10].

History and physical examination are sufficient to make a diagnosis of abortion as was the case in our patient. History of vaginal bleeding and lower abdominal pain after a period of amenorrhea is highly suspicious of abortion. The range of presentation is varied in that some patients are stable while others present with severe hemorrhage and severe hemodynamic derangement and shock. Our patient had vaginal bleeding but was not in shock. In early abortion, hemorrhage, 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 karyotypic index of less than 10 if there are chromosomal abnormalities. Ultrasonography demonstrates retained products of conception in the uterine cavity. Differential diagnosis of incomplete abortion include membranous dysmenorrhea, hyperoestrogenism, hydatidiform mole, pedunculated myoma and cervical neoplasia[1,2].

Successful management of abortion depends on early diagnosis. Stabilization of the patient is achieved by use of plasma expanders or blood transfusion where necessary. Our patient was started on intravenous normal saline. Uterine evacuation to stop the bleeding is the ultimate procedure in management of incomplete abortion. In our patient this was achieved by use of the manual vacuum aspiration (MVA) technique. MVA is safe and easier

to perform than the use of sharp curettes in management of incomplete abortion. Additionally increased awareness of the risks of general anesthesia led the procedure to be moved from the operating theatre to outpatient facility with the patient under sedation or regional anaesthesia[8,9]. The technique of vacuum aspiration is simple and provides developing countries such as ours 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. The canulae are in sizes 4-12mm. a vacuum of 60mHg is created when the syringe valves are locked and the plunger pulled.

Evacuation is judged complete when four or more of the following are noted during the procedure;

- Reddish/pinkish foam was being suctioned.
- A gritty sensation encountered of the canula against the uterine wall.
- The uterus became well contracted with only minimal blood flow.
- Uterus is tightly gripping the canula.
- The patient complains of increased pain and cramping.
- No further flow of tissue when syringe is not full or canula not blocked.

During the procedure our patient was continuously talked to by the doctor and the assisting nurse.the canula was inserted once using the no touch technique to minimize infection. A paracervical block can be used especially if cervical dilatation is desired. Kizza[9] at Kenyatta National hospital found the MVA procedure as described to be safe especially in early gestation. Complications of MVA are rare and mainly include sepsis and hemorrhage. Thus prophylactic antibiotics are instituted to prevent infection. Makokha

observed that 22.2% of maternal deaths were due to post-abortal sepsis, 85% having evidence of interference and hemorrhage was second to sepsis as a cause of maternal death.

This patient was adviced on contraception but unfoortnately did not turn up for provision of a method.this was a missed opportunity to provide comprehensive post abortion care. The concept of post-abortion care was formulated by governments at the international conference for population and development in Cairo in 1994 and reaffirmed at the Cairo+5 meeting. It has gained wide acceptance as one of the ways of improving services provided to women with complications from spontaneous or induced abortions. This would help break the cycle of repeat abortions and reduce maternal mortality and morbidity. 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. The provider ought to provide adequate counseling and screen the women for the standard precautions for use of the chosen method[12].

This patient did not develop any complications during or after the MVA procedure. These complications include, severe hemorrhage, sepsis, uterine perforation, injury to the bowel and bladder and fistula formation. Other complications include uterine synaechiae after excessive curettage, infertility and death from shock[8,10,11,12].

REFERENCES

1. Garmel S.H. Early pregnancy risks; In current obs. & gynaecologic diagnosis & treatment 9th edition 2003 pg 272-78

2. Cunningham F.G. MacDonald P.C, Leveno K.J et al. Abortions; In Williams Obstetrics 2st edition 2001 Appleton and Lange publications, Stanford

3. Aggarwal V.P, Mati J.K.G Epidemiology of induced abortion in Nairobi. J Obst. Gynaecol East afric 1982;1:54

4. Richard F.M Thompson J.D. Telindes operative gynaecology 6th edition pg 524-525

5. Macintyre J.A, Mange A.L Beer A.E et al Clinical immunologic and genetic definitions of primary and secondary recurrent spontaneous abortion. Fertil. Steril 1994 42:549

6. Valentino Lema A.M, Macharia J.K; A review of abortion in Kenya, March 1992. Center for adolescent studies.

7. W.H.O.; Unsafe abortion, Global and regional estimates of incidence and mortality due to unsafe abortion with listing of available country dat 3rd edition. Document WHO/RHT/MSM/97.16

8. WHO 1994; Clincal management of abortion complications WHO/FHE/MSM/94.1

9. Kizza A.P.N, Rogo K.O, assessment of the manual vacuum aspiration equipment in the management of incomplete abortion. EAMJ 1990 II:67

10. Howard W, Weutz A.C. Burnett L.S Novaks textbook of gynaecology 11th edition Churchill Livingstone pg. 328

11. Derek, Llewlyn, Jones. Abortion; in fundamentals of Obstet. Gynaecol Vol. 2, 5th edition 1990 pg 127.

12. Berer M; Making abortions safe a matter of good public health policy and practice. Bulletin of WHO. The Int. J. of public health Vol 78, 5;2000: 580-592

GYNAECOLOGY CASE 15

PERSISTENT GESTATIONAL TROPHOBLASTIC DISEASE : REMISSION AFTER CHEMOTHERAPY

Name A. N. IPNO 1024960

Age 25YEARS DOA 12/5/06

Parity 3+1 DOD 20/8/06

PRESENTING COMPLAINTS

A. N. was admitted with 8months history of per vaginal bleeding and 1 day history of vomiting headache and abnormal behaviour.

HISTORY OF PRESENTING ILLNESS

Bleeding had been moderate with occasional clots for the eight months. She had had manual vacuum aspiration done for incomplete abortion in a private clinic before the onset of illness. She had subsequently been put on combined oral contraceptive pills (COCs) but took them irregularly. One day before admission, she had developed vomiting, headache and abnormal behaviour. This made her go to a different private clinic where examination had revealed an ulcerative vaginal mass leading to referral to our hospital for workup.

At casualty she was found to be sick looking, confused, pale and wasted. An ulcerative and easily bruisable mass in the upper lateral wall of the vagina was found. The mass did not involve the vulva, urethra, fornices or the cervix. The uterus was bulky and masses were felt in the adnexae. Urgent ultrasound scan was done which revealed solid endometrial masses and cystic bilateral ovarian masses. A pregnancy test was found to be positive. A diagnosis of gestational trophoblastic tumour was made and she was admitted for further evaluation and chemotherapy.

OBSTETRIC AND GYNAECOLOGIC HISTORY

She was para 3+0. Her last delivery was in 2004 (2years earlier) and had an abortion 8 months before the admission. She had been on COCs even before the onset of the illness but was taking them irregularly leading to failure. She had continued the COCs after evacuation. Menarche was at 14years. Flow lasted 3-4 days and cycle was 28-30 days and was regular before she fell sick. She had history of intermenstrual spotting but not postcoital bleeding before illness.

FAMILY AND SOCIAL HISTORY

She was a housewife married to a guard with a local security company. She lived with her husband in Kayole. She and her husband neither smoked nor drunk alcohol

PAST MEDICAL HISTORY

She was not known to suffer from any chronic illness, had not undergone any major surgical operation and was not known to be allergic to any medications. She had been hospitalized for obstetric indications only.

PHYSICAL EXAMINATION

At admission, she was found to be sick looking pale and emaciated but not jaundiced and had no lymphadenopathy.

No abnormalities were found in the nervous, respiratory, cardiac and musculoskeletal systems.

ABDOMINAL EXAMINATIN

The abdomen was flat and moving with respiration. There were poorly defined masses in both iliac fossae with mild tenderness. The liver and spleen were not palpable and there were no abnormal masses.

VAGINAL EXAMINATION

The external genitalia was normal. There was an approximately 3 by 4 cm ulceration palpated on the anterior wall of the vaginal wall overlying the urethra. The urethra was however not perforated. The uterus was bulky and

there were bilateral ovarian masses. There was mild adnexal tenderness but cervical motion tenderness was negative. There was blood stained discharge on examining finger. Speculum examination confirmed the ulcer but found the rest of the vagina and the cervix to be normal.

INVESTIGATIONS

1. Ultrasound scan; Showed a bulky uterus with endometrial hypoechoic masses. There were bilateral cystic ovarian masses but no fluid in the Pouch of Douglas. The cervix was reported to be normal.
2. βHCG were 317,570IU/l.

DIAGNOSIS

A diagnosis of high-risk persistent gestational trophoblastic disease most likely choriocarcima was made.

MANAGEMENT

She was admitted to ward ID then transferred to ward IB for chemotherapy. In IB the following investigations were done:

1. Liver function tests - Total bilirubin was 11mmol/l and direct was 5. Total protein was 75g/l and albumin was 36. Aspartate transaminase was 34 while Alanine transaminase 28 mIU/ml
2. Renal function tests - Sodium was 142mmol/l, potassium 4.6mmol/l and urea was 5.5mmol/l, and creatinine 102mmol/l.
3. FBC Haemoglobin-7.6g/dl, MCV-72µl, White cell count-$5.2x10^9$cell/l, Platelet count-$167,000x10^9$/l.
4. Blood group-B rhesus D positive

She was transfused with three units of blood before being transferred ward IB. Check haemoglobin was found to be 34%. She was started on methotrexate, actinomycin D and cyclophosphamide. She received a total of five courses of the above chemotherapy as follows:

- 18/5/06
- 9/6/06
- 1/7/06
- 29/7/06
- 20/8/06

βHCG, FBC, liver and renal function tests were as summarized in the table below:

βHCG LEVEL

DATE	βHCG LEVEL (mIU/ml)
18/5/06	317, 570
9/6/06	16,212
1/7/06	7
29/7/06	3
20/8/06	3

The normal reference range for laboratory used was 3-10

FULL BLOOD COUNT

DATE	HB (g/dl)	WBC (x109/l)	Platelets (x109/l)
18/5/06	11.3	5.2	167
9/6/06	10.8	6.3	213
1/7/06	10.2	5.6	199
29/7/06	11.6	5.9	242
20/8/06	10.9	4.5	227

She was transfused with one unit of blood before the fourth course of chemotherapy because haemoglobin was below 10g/dl actually 9.4g/l

LIVER FUNCTION TEST

DATE	Total protein(g/l)	Albumin (g/l)	Alanine transaminase(iu/l)	Aspartate transamine(iu/l)
18/5/06	75	36	28	34
9/6/06	78	35	35	76
1/7/06	73	36	29	62
29/7/06	76	37	36	66
20/8/06	78	37	62	62

RENAL FUNCTION TEST(U/Es)

DATE	Na (mmol/l)	K (mmol/l)	Urea (mmol/l)	Creatinine (mmol/l)
18/5/06	144	4.3	5.3	62
9/6/06	137	4.9	3.9	76
1/7/06	139	3.8	4.8	59
29/7/06	142	3.6	4.3	67
20/8/06	136	4.2	5.6	71

DISCUSSION

A.N. was a 25years old patient, who had high-risk persistent gestational trophoblasric disease following an abortion. She was put on methotrexate, actinomycin-D and cyclophosphamide with good results.

Gestational trophoblastic disease (GTD) is a spectrum of pregnancy-related trophoblastic proliferation abnormalities, which include hydatidiform mole, invasive mole, placental site trophoblastic tumour and choriacarcinoma. (1, 2, 3). GTD have properties similar to those of the placenta like invasion and liberation of human chorionic gonadtrophin (HCG) (2). GTD is among the rare human tumours that can be cured even in the presence of widespread dissemination. Hydatidiform mole represents the benign end, while choriocarcinoma is on the other extreme end of anaplasia (2, 4).

Hydatidiform mole occurs 1:125 pregnancies in the Mexico and Taiwan but 1:1500 in the United States. Chroriocarcinoma is rare, reported in 2-5% of all cases of gestational trophoblastic neoplasia. The incidence in the USA is 1 in 40,000 pregnancies, but is higher in the orient (2). An incidence of 1 in 1, 118 pregnancies has been reported at KNH. About half of all cases of chriocarcinoma follow molar pregnancy, 25% an abortion and the remainder occurs following other forms of gestations (ectopic pregnancy and term pregnancy (1, 2, 5, 6). The patient presented here developed GTD after an abortion.

Persistent GTD also called gestational trophoblastic tumour (GTT) is diagnosed if βHCG rise on two consecutive visits, stagnate in three, remain high 15weeks after evacuation, rise after reaching normal level or resumption of bleeding after evacuation of molar pregnancy. High level of HCG and evidence of extrauterine metastasis or growth in the uterus are diagnostic for patients who present after termination of non-molar pregnancies (2, 3, 4).

Gestational trophoblastic tumour (GTT) following a molar pregnancy may exhibit the histological features of either hydatidiform mole or choriocarcinoma but that following a nonmolar pregnancy, usually exhibit histological pattern of choriocarcinoma. Histologically choriocarcinoma is characterized by sheet of anaplastic syncytiotrophoblast and

cytoptrophoblast without chorionic villi with invasion into the nyometrium causing haemorrhage and necrosis (4). Histology was not done for A. N. but it would most likely have shown a histological pattern of choriocarcinoma having arisen from a non-molar pregnancy.

The commonest features of GTT is persistent vaginal bleeding following termination of pregnancy and which occurs in 90% of patients. Other symptoms are nausea, vomiting, vaginal discharge, sub-involution of the uterus, pre-eclampsia and thyrotoxicosis. Cachexia and anaemia may occur if treatment is delayed. Some patients present with features concomitant with the site of metastasis, like haemoptysis for chest metastasis. The commonest sites of metastases are the lungs (over 75%) and the vagina (about 50%). Liver is involved in 10%, the vulva, kidneys and ovaries may also be involved. Ovarian theca-lutein cysts are identified in over one third of the cases (1, 2, 3). The patient presented had prolonged bleeding and anaemia due to vaginal metastasis and theca lutein cysts were found on ultrasound scanning. The confusion suggested CNS metastasis, but this was not supported by any CNS localizing. CT scan of the skull also did not show any metastasis. The confusion could have been caused by other systemic complications of chronic illness or microscopic multifocal metastasis.

Investigations aim at establishing a diagnosis, establishing the health status of the patient and finding metastasis if any. High level of ßHCGs and a pelvic ultrasound are needed for diagnosis of GTT. A chest xray and abdominal ultrasound are used to screen for chest, liver, renal and splenic metastasis while CT scan or MRI are used to confirm suspicion of lung, liver renal or GIT metastasis if suspected on screening. MRI and CT scan are also used to investigate patients with CNS symptoms. Full haemogram, liver and renal function tests are routine especially for patient needing chemotherapy.

(2, 3, 4). The patient presented had haemogram, LFTs, and U/Es, which were within normal save for anaemia of 7.6g/dl. This was however corrected by transfusion. CXR was normal and abdomino-pelvic ultrasound showed solid intrauterine masses and theca lutein cysts but no pelvic or abdominal metastasis. ßHCGs were elevated above 10^5 but CT scan was essentially normal.

There are three ways of classification used for GTD. These are:

A. The classification recommended by WHO scientific group on GTD in 1983 and adopted by the National Cancer Institute of America. It classifies gestational trophoblastic disease as shown below (7),

- Hydatidiform mole, a) Complete
  b) Partial
- Gestational trophoblastic tumour; which is further divided into, non-metastatic and metastatic disease.

Metastatic disease is divided into good and poor prognosis disease as follows;

a) Good prognosis-no risk factors
b) Poor prognosis-any risk factor

These risk factors that are considered are;

- Pretherapy HCG level >40,000mIU/ml
- Duration of >4month between termination of mole and start of treatment
- Presence of liver or brain metastasis
- Previous failed chemotherapy
- Disease onset after term pregnancy

Using this classification A. N. was in the poor prognosis metastatic gestational trophoblastic tumour category due to high HCG levels (317,570) and long duration between onset and starting treatment (>8months).

B. A more recent classification recommended by WHO is called The WHO scoring system. This classification assigns point for various risk indicators and the sum total is used to assign patient their classes. It is superior in predicting resistance to chemotherapy than the other two (8, 9). The scoring is as shown below:

	SCORE			
Parameter	0	1	2	4
Age (years)	<39	>39		
Antecedent pregnancy	mole	Abortion	Term preg	
Interval (months)	<4	4-6	6-12	.12
Pretreatment levels	10^3	10^{3-4}	10^{4-5}	$>10^5$
ABO blood group Male-female		A-O, O-A	B, AB	
Largest tumour (cm)		3-5	>5	
Site of metastasis		Spleen, Kid.	GIT, liver	Brain
Number of metastasis		1-4	4-8	>8
Prior chemotherapy failed			Single	>2

Those who score less than 5 are put in low risk class, 5-7 medium risk and >7 are in high risk.

The patient presented was of blood group B positive, the antecedent pregnancy was an abortion, had stayed for 8months from antecedent gestational event and treatment and βHCG was 317,570 ($>10^5$) mIU/ml. This put her in the high-risk group with a score of 9.

C. The third type of classification was recommended by the International Federation of Gynaecology and Obstetrics (FIGO). This classification uses anatomic staging as shown below (11).

Stage I: Disease confined to the uterus.

Stage II: GTT extending outside uterus but limited to genital organs. (adnexa, vaginal and broad ligament)

Stage III: GTT extending to the lungs with or without known genital tract involvement.

Stage IV: All other metastatic sites.

All the stages are further divided into A, B and C on the basis of presence of defined risk factors as follows; A: No risk factors

B: One risk factor.

C: Two risk factors

The risk factors affecting the staging include the following:

- Serum HCG> 40,000miu/ml
- Duration of the disease longer than 4 months from termination of antecedent

gestational event.

In our patient, the HCG level was 317,570iu/ml and presented 8months after evacuation for presumed abortion and disease was limited to the genital structures.. She was therefore in stage IIC.

Uniformity is lacking worldwide in application of either the WHO scoring or the FIGO anatomic staging. The National Cancer Institute classification has been largely discarded except in the US. Most patients in class I of the FIGO system however almost always have a low-risk disease, while stage IV patients have a high-risk disease. Distinction however needs to be made in stages II and III for the purpose of management. Hence most centres use the two systems simultaneously (10).

Stage I disease is generally management by single agent chemotherapy using methotrexate or actinomycin D with a remission rate of between 84-96%. Resistant cases respond well to triple therapy using methotrexate, actinomycin D, and cyclophosphamide or chrolabucil (12).

Hysterectomy is rarely used these days but is still a good option for patients who don't need further fertility, have resistance to chemotherapy or are likely to abscond from treatment (13)

Stage II and III disease on FIGO staging are managed depending on whether they are low, medium or high-risk diseases according to WHO scoring system. Low and medium risk diseases are managed by single agent chemotherapy with combination therapy being reserved for resistant cases. In high-risk disease, triple therapy is the starting therapy with etoposide, methotrexate, actinomycinD, cyclophasphomide and vincristine (EMA/CO) being employed for resistant disease. Hysterectomy, thoracotomy and local vaginal resection are often employed to control acute bleeding or resistant tumours. Using this regimen, Berkowitz and colleagues have achieved 100% remission rate in stage II and 92.2% in stage III (14).

In stage IV, triple therapy is used from the beginning with EMA/CO being used as second line drugs. Further resistance is treated by replacing cyclophosphamide and vincristine with etoposide and cisplatin (EMA/EP). Whole brain irradiation is used for brain metastasis. It reduces incidence of acute haemorhagic episodes. Surgery is used to control bleeding episodes in the brain, resent brain and hepatic metastasis or resistant masses elsewhere. Intrathecal methotrexate has also been used successfully for brain metastasis (15).

In our centre the 3 classification systems are acceptable though FIGO staging and WHO scoring are mostly used. In the patient presented, both were used simultaneously.

During followup, effective contraception is mandatory. Combined oral contraceptive (COC) is preferred not just because of its effectiveness if well used but also because of its predictable menstruation pattern. Deviation from this pattern and especially bleeding during follow-up period may be a sign of recurrence and need for evaluation. Earlier studies suggested that use of COCs was associated with more disease persistence but more recent work proves otherwise. Progesterone only contraceptives, have the drawback of progesterone breakthrough bleeding but can be used if serious

contraindications to COCs like history of thrombo-embolic disease exist. Intrauterine devices can also be used if the above methods cannot be used. However they are only started after the HCG levels normalize due to the higher risk of uterine perforation if inserted earlier (4, 15,16, 17). Our patient was put on COCs.

Molar pregnancies are followed up first on weekly basis until three consecutive negative βHCGs are obtained then monthly for twelve months. In some centres, follow-up is on two weekly basis until three consecutive negative results, then one monthly for six months, followed by 2monthly for a total of 12months. (4, 18).

Low risk GTT is followed up for 12 months after completion of chemotherapy. Some centres reduce followup visits to 2monthly if the first 6 months are disease free. High-risk disease however must be followed up for 24months to avoid late disease recurrence (4, 18, 19).

REFERENCES

1. Cunningham F.G., McDonald P.C., Leveno K.J. Disease and abnormalities of the placenta: Williams obstetrics, 21st Ed. 2001; McGraw Hill New York. 32: 835-847
2. Tricia B.E., O'Quinn G. Gestational Trophoblastic Disease: Current Obstetric and Gynecologic Diagnosis and Treatment 9th ed. 2002; McGraw Hill, New York. 50: 947-958.
3. Senchever M.A., Droegemueller W., Herbster A.L., Mishell D.R. Gestational Trophoblastic Disease in Comprehensive Gynecology 4th ed. 2001; Mosby, Misouri.35:1047-1062.
4. Berkowitz R.S., Goldstein D.P. Gestational trophoblastic disease in; Novaks gynaecology 13th Ed 2002:1353-1373. Lippincot, Williams and Wilkins,Philadelphia
5. Fongoh F.B. clinical and laboratory evaluation of choriocarcinoma at Kenyatta National Hospital 1980-1983. Mmed thesis, 1984 UoN

6. Makokha A.E., Mati J. K. G. Choriocarcinoma at Kenyatta National Hospital 1973-1979. J. Obstet Gynaecol E. and Cent. Afri. 1982;1:27

7. Gestational trophoblastic diseases: report of a WHO scientific group. WHO Tech Rep Ser 1983;692:51.

8. Ayhan A., Yapar E.G., Deren O., Kisnisci H. Remission rates and significance of prognostic factors in gestational trophoblastic tumors. J Reprod Med 1992;37:461-465.

9. Dubuc-Lissoir J., Sweizig S., Schlaerth J.B., Morrow C.P. Metastatic gestational trophoblastic disease: a comparison of prognostic classification systems. Gynecol Oncol 1992;45:40-45.

10. Berkowitz R.S., Glodstein D.P. Chorionic tumours. New England Medical Journal, 1996; 335: 1740-1748

11. Goldstein D.P. Revised FIGO staging system for gestational trophoblastic tumour. J. Reprod. Med. 1998; 43:37.

12. Kohorn E.I. Single-agent chemotherapy for nonmetastatic gestational trophoblastic neoplasia: perspectives for the 21st century after three decades of use. J Reprod Med 1991;36:49-55.

13. Soper J.T. Surgical therapy for gestational trophoblastic disease. J Reprod Med 1994;39:168-174.

14. Newlands E.S., Bagshawe K.D., Begent R.H.J., Rustin G.J., Holden L. Results with the EMA/CO (etoposide, methotrexate, actinomycin D, cyclophosphamide, vincristine) regimen in high risk gestational trophoblastic tumours, 1979 to 1989. Br J Obstet Gynaecol 1991;98:550-557.

15. DuBeshter B., Berkowitz R.S., Goldstein D.P., Bernstein M.R. Vinblastine, cisplatin and bleomycin as salvage therapy for refractory

high-risk metastatic gestational trophoblastic disease. J Reprod Med 1989;34:189-192.

16.Stone M., Dent J., Kardana A., Bagshawe K.D. Relationship of oral contraception to development of trophoblastic tumour after evacuation of a hydatidiform mole. Br J Obstet Gynaecol 1976;83:913-916.

17.Berkowitz R.S., Goldstein D.P., Marean A.R., Bernstein M.R. Oral contraceptives and postmolar trophoblastic disease. Obstet Gynecol 1981;58:474-477.

18.Curry S.L., Schlaerth J.B., Kohorn E.I. Hormonal contraception and trophoblastic sequelae after hydatidiform mole (a Gynecologic Oncology Group Study). Am J Obstet Gynecol 1989; 160:805-811.

19.Lisa E.M., Debra W., Pritzker J.G. emedicine

GYNAECOLOGY LONG COMMENTARY

TITLE: REASONS FOR EARLY DISCONTINUATION OF SUBDERMAL LEVONORGESTREL CONTRACEPTIVE IMPLANTS IN FAMILY HEALTH OPTIONS OF KENYA CLINICS IN NAIROBI.

SUMMARY

Background

Premature removal and discontinuation of contraceptive implants is a cause of a sizeable expenditure in Kenya.
Evaluation of the factors that influence the decision to remove the implants and being able to predict which users are likely to discontinue prematurely could provide useful information for counseling patients as they are considering contraceptive implants and enormous savings in terms of manpower and finance, not only for the health facilities but the country at large.

Objective

The objective of the study was to determine the reasons for removal and discontinuation of implants among users as seen in Family Health Option of Kenya clinics in Nairobi.

Study design

This was a retrospective cross-section descriptive study.

Study population

The family planning clients who had implants removed in the Family Health Option of Kenya clinics situtated in Nairobi, within the first three years of insertion.

Study Hypothesis

Can early discontinuation of implants be reduced in our set up?

Study Period

The data was collected from March 2008 to July2008 and involved the collection of data from records of clients, dating from January 2003 to July 2008, this being the period when the implants wer removed.

Method

Scrutiny of the records of clients who had implants removed in these clinics prior to and during the study, until the required sample size of 138 in total was achieved. Earlier, the principal investigstor, had done test study with a questionnaire and ten records of the clients to ascertain the suitability of the data.

Main Outcome Measures

These include the demographic characteristics such as marital status, age, parity, number of living children, education level and reasons for removal and whether self initiated or health provider initiated.

Results

The majority of the clients were married (63%) and (26%) were divorced while 3% were widows. Most of them were of the age group 20-30 years (53.8%). Those of parity of 3-5 constituted 83.3% of the users while those of parity greater than 5 constituted only 3.7%.Most of them had atleast primary education (98.7 %).

The commonest reason for removal within the first twelve months was menstrual changes (38.4 %) followed by plan to conceive (28.3%).Acne and complexion changes was a reason in 13.8% of those who discontinued during the first one year.

Conclusion

The most common reason for discontinuation at 12 months was menstrual disturbances but by 24 months and later on it was intent to conceive. At low parity thus 3 and below, the main reason was intent to conceive. Majority of those who discontinued had family sizes of 2 – 3 and had attained at least primary education.

Recommendations

- Good counseling prior to insertion, during use and on removal.
- Good medical support during use to alleviate the side effects should be mandatory.

- Frequent screenings to rule out other illnesses which mimick their side effects, e.g. cancer of the cervix would create more credibility to this valuable service.

INTRODUCTION

Implants sub-dermal have been in use for the last 3 1/2 decades having been initially tried in Finland. To date, an estimated 6 million women world wide use implants and as at 2000, it was in use in more than110 countries including Kenya

In Kenya, it was introduced through Machakos project in 1986 prior to being fully incorporated into the family planning services throughout the country, by 1991 [2]

Types [14]

- NORPLANT- which has six 3.4cm long flexible silastic capsules of 2.4mm diameter filled with 3.6milligrams of levonorgestrel- (thus 216mg in total)/ .
- JADELLE- has two silastic rods of 4.4cm length and 2.4mm diameter containing 70mg of levonogestrel (140mg in total)/.

Implants are long-term reversible contraceptives which last between 3 to 5years, and anytime during this period it can be removed. [4, 6, 8]

- IMPLARNON- the single silastic rod containing etenorgestrel which services for three years once inserted.

Mode of Action

The devices are designed to prevent conception by releasing the progesterone hormone levonogestrel or its analogue into the blood circulation at a slow steady rate of 30-35microgram/day for three or 5 years once inserted, similar to minipill-30mg progestin taken orally [12.13].

Sites of Insertion

The flexible silastics are inserted on the medial aspect of the non-dominant upper arm as an out patient or in surgery office as a procedure under local anaesthesia but other sites have been used including the thighs and even abdomen [3.8].

Mechanisms of Action

The hormone levonogestrel once in the body prevents pregnancy via the following mechanisms:

- Suppression of ovulation from the ovarian level and pituitary level by suppressing LH production.
- By enhancing cervical hostility mainly through the thickening of cervical mucus and hence spermatozoal unfavorability.
- Inhibiting nidation through interference with endometrial mucosa by causing pseudo-distrophy.

The above result in 99% effective prevention of pregnancy and the effects are easily reversed on the removal of the capsule. Conception has been

reported as soon as 14 days after removal and the contraceptive effect is realized only 48 hrs soon after insertion [1, 3,4 , 5].

LITERATURE REVIEW

Since implants incorporation into family planning services of different countries and its wide acceptance all over the world, it has been confirmed that it is highly effective in contraception and continues to register high continuation rates [1].

However, despite the high success rate, the discontinuation rates are also unfavorable. For example; a study done in Mexico on 816 clients between 1990-1995 by Servin & Co.'s found continuation rates of [5, 7];

77.1% at 12 months

53.7% at 24 months

44.6% at 36 months

Several factors have been attributed to this with;

- Irregular mensturation being the commonest complaint and reason for discontinuation, with individuals varying in the number and pattern of bleeding days. But despite the bleeding, there was an increase in haemoglobin levels during implant use and there was no noticeable change in systolic and diastolic blood pressure [2,10,14,15,]

Else where, studies in other parts of the world, e.g. Malaysia, Shanghai 1990 – 1995 reported continuation rates of 76% - 96% at the end of the year and 33% - 78% at 5 years[9]

Despite the side effects the advantages are appreciable especially [6, 7, 11, 12];

- Being very effective in preventing pregnancy over long period (three or 5years).
- Easy reversibility at any time by capsule removal.
- It is free of oestrogen side effects hence can be used even where the later is contraindicated;
- Convenience and easy to use with increased compliance [1,2,9,11]
- A three year study of implants in China Shanghai, 1991-1994 confirmed high continuation rates of;

  -96.8% at 12 months

  -86.2% at 24 months

  -77.2% at 36 months

Sharp rise in termination was mainly due to menstrual disturbances which included heavy bleeding, spotting, irregular cycles amenorrhea with only but a few complaints of medical problems such as headache and weight gain. Most of the complaints were reported during the third month post insertion [1,2,4,8]

The commonest side effects can categorically be divided into; [8 9 12]

- Menstrual problems -Amenorrhoea
  -Spotting
  -Menorrhagia
  -Dismenorrhoea
  -Excessive discharge
- Medical problems
  - Headache
  - Weight gain
  - Rise in blood pressure
  - Breast and nipple discomfort
  - Acne
  - Fatigability
- Implant site complications ;
  - Itchiness
  - Hyper pigmentation
  - Scar formation
  - Keloid formation

Several side effects have been associated with this contraceptive for example

- Decreased libido
- Decreased milk output

- Increased appetite
- Obesity
- Increased incidence of coagulopathy.

A study in UK reported that being a surgical procedure, to fit and remove was a big disadvantage to many women, the insertion is uncomfortable and the local anaesthetic injection painful and anaesthetic fanning sub-dermally irritating and painful too. However majority were willing to recommend the method to others[11].

In Kenya in 1997, a study to determine the social- demographic characteristic of implant clients and reason for early removal of the implant, found continuation rate of 91% after 12 months and 80% after 24 months. But since then perceptions have changed and more and more women tend to prefer implants.

RATIONALE

Misconception about implants and their side effects discourage the utilization of implants which is an affordable and highly effective contraceptive. It is not only a long term contraceptive but also easily reversible. They require minimal maintenance once inserted and hence convenient for the client. It is not costly than other existing methods taken into consideration its duration of use once inserted. Therefore it is important to carry out a study to establish the socio-demographic characteristics of levonogestrel implant users as well as their complaints during follow up visits and reasons for early discontinuation. This will reveal the actual reasons for the removal of implants. No similar study has been done elsewhere in the recent past. The out come of the study would be of value in improving these services and can be used in counseling of clients prior to insertion.

Increase in demand has necessitated its provision and **Family health option of Kenya** being one of the service providers has three established clinics in the busy capital city of Kenya providing these services; Two in the busy highly congested central business district and one in the over populated mid-class habited Nairobi west area.

It is worth noting that ***family health option Kenya***, formally ***family Planning Association of Kenya*** changed names in 2002 but still maintained its status in the provision of contraceptives

This study established the main reason for the discontinuation of these vital service.

The study generalized the use of two implants – Norplant and Jadelle due to similarities in the mode, duration of action and insertion method. Though Norplant, is not available for insertion currently, there are several clients still using it now or discontuining from its use, having had it inserted earlier.

BROAD OBJECTIVE

To determine reasons for early discontinuation of Subdermal Levonorgestrel Contraceptive Implant in Family Health Options of Kenya clinics in Nairobi:-

Specific Objectives

- To determine reasons for early removal of the implant.
- To determine the common side effects of implants in the general population.
- To determine wether premature removal of the plant is clinician initiated or client initiated.
- To determine association between age, parity and premature implants removal.

METHODOLOGY

Study Design -This was a retrospective cross-sectional descriptive study in which the records for the clients who had voluntarily chosen to use implant for contraception were scrutinized and complaints listed during follow up visits and reason for discontinuation analyzed. The data so collected, which also included their socio- demographic characteristics and parity, was analyzed using SPSS-PC Programme.

Study Areas: -

The multicentre, Family Health Options of Kenya Clinics in Nairobi. The clinics are located in Nairobi West, Central Business District and Industrial area of Nairobi town. The clinics are manned by nurses and medical officers and have gynecologists on call for consultations. The implants are removed by nurses routinely, but by the doctors if complicated. The removal is done on patient's request or clinician's advice.

Study population:-Women attended to in these clinics and had Implant removed in these clinics. The follow up records were scrutinized retrospectively until the sample size was achieved. Any incomplete records were foregone and the next client recruited.

Study Sample:-The sample size was calculated using EPI-INFO version 5 software of epidemiology and disease surveillance 1999.The continuation

rate for levonogestrel implants at 12 months was found to be 76 – 96%[9].

Taking P to be 90 the sample was calculated as such.

5%=d

$N=\frac{Z^2PQ}{D^2}$

Z=1.96 when confidence level is

Q=100-P

$N=\frac{1.96x90x10}{5x5}$

=138.3

=138

SAMPLING PROCEDURE

This involved retrieval of the family planning records cards of implant users who had implants removed at the Family Planning options. The sampling method was randomized to involve every other alternate client or rather all even numbered clients in the register until the required sample was obtained. With the help of the registers which had the details and numbers of those who had implants removed, the correct cards were identified and returived from records. In cases where the card had no adequate information the case was skipped and the next enlisted. Thus a total of 144 clients were considered but only 138 were recruited due to adequacy of information.

Relevant information from these records was retrieved and entered into questionnaires. The information required include; clients' family planning number, age, parity number of living children, education level, complaints during follow up visits and main reason for method discontinuation and date of removal.

Study stickers were applied on the card after being retrieved to ensure no repeat file retrieval takes place.

The principal researcher was involved in the retrival of all the cards and filling of the questionnaire, having been convinced of the suitability of the questionare from the test study done earlier. The data collection tool was predominatly the questionnaire and the retrieved clients cards.

Early implant removal in this study was considered as removal of implant within three years, from the day of insertion. From the data base of family health options of Kenya, it was possible to know the number of clients who had levonogestrel implants removed and after what duration of use.

CONSTRAINTS

- The forms or records were assumed to have information as stated by the client and that they were facts but this could not be verified.
- The contraceptive method requires minimal follow up and most patients did not come for follow up as required.
- This being an urban town client's migration is very high and follow ups were not consistent
- Some clients records had minimal information on the cards and hence inadequate for completion of questionnaire.

Ethical Consideration: -

Permission for the study was sought from: -

Kenyatta National Hospital Ethical Committee and the director and management of Family Health Options of Kenya before the study commenced. The name of the client was not recorded but only the Family Planning number. But the FP number quoted in the study.

The information obtained was used strictly for the study purposes and not otherwise as to incriminate or harm anybody

The findings will be availed to the Family Planning Options of Kenya so as to improve service delivery of Implant Method

DATA MANAGEMENT

The data collected was edited before being entered in the computer for analysis. Data was entered in the computer using statistics packages for social sciences (SPSS- PC) data entry program. The analysis involved descriptive statistics like means, frequency, and distribution and tests of significance.

Outcome measures

The main outcome measures were the total number of clients with levonogestrel implants contraceptive requesting for removal, reasons for removal, duration of use and the're-call' features and complaints like change in weight as recorded in the card.

RESULTS

A total of 138 clients were enrolled, being those who discontinued implants within 36 months after insertion, out of the totals; 288 clients who had implants removed from Jan 2003 to July 2008.

TABLE 1: Socio demographic characteristics (n = 138)

CHARACTERISTICS	NUMBER	PERCENTAGE (%)
Marital Status		
Single	11	8
Married	87	63
Divorced	36	26
Widowed	4	3
Age (years)		
< 20	18	13
20≤ 30	64	46.4
30≥ 40	56	40.6
> 40	-	-
Parity		
0-2	18	13
3-5	115	83.3
> 5	5	3.7
Level of education		
Nil education	3	2.2
Primary	66	47.8
Secondary	43	31.2
Post secondary	26	18.8

Table 1 shows that most of the clients were married (63 %) and of modal age group 20 – 30 years. Majority were para 3 -5 (83.3%) and had at least primary education (97.8%)

Table 2: Period of use prior to discontinuation of implant. n=138

Period of use	Number	Frequency (%)
< 12 months	14	10.1
12 – 24 months	56	40.6
24 – 36 months	68	49.3
TOTAL	**138**	**100**

Table 2 shows that most clients (49.3) removed the implants after 24 – 36 months of use and only 10% removed them during the first year of use.

TABLE 3: Premature removal of implant by decision maker. (n= 138)

Decision Maker	Number	Percentage (%)
Health provider	11	8
Client	127	92
TOTAL	**138**	**100**

Table 3 indicates that early removal was mainly requested by clients at 92% and only 8% was instigated by the health provider.

TABLE 4: Reasons for premature removal (n= 138)

(Premature removal being removal of implant within the first 36 months)

Reasons for removal	Number	Percentage (%)
Weight gain	11	8.1
Menstrual changes	53	38.3
Headache	9	6.5
Palpitation	5	3.6
Acne and complexion changes	19	13.8
Desire to conceive	39	28.3
Plan to use another method	2	1.4
TOTAL	**138**	**100**

Table 4 shows that the major reason for premature removal was menstrual changes (38.4%) followed by desire to conceive (28.3%). Removal with intent to change to another family planning method was least (1.4%.).

TABLE 5: Changes in diastolic blood pressure within the first twelve months

Diastolic BP increase (mmhg)	Change in BP after 3 months (n =138)		Change in BP after 6 months (n = 134)		Change in BP after 12 months (n= 130)	
	no	%	no	%	no	%
<10	133	96.4	132 98.5		129	99.2
10 ≥ 20	4	2.9	1 0.75		-	0
>20	1	0.7	1 0.75		1	0.8
TOTAL	**138**	**100**	**134**	**100**	**130**	**100**

Table 5. Shows that only changes in blood diastolic pressure of ≤ 10mmhq were predominant (97.1%) in the first 3 months, (98.5%)after 6 months and after 12 months (99.2%). The **n** value subsequently decreased since those who had increases in their diastolic pressures, opted to discontinue the implant use.

TABLE 6: Menstrual problems (n = 53)

Reason	Number	Percentage (%)
Amenorrhea	11	20.8
Spotting	22	41.5
Menorrhagia	20	37.7
TOTAL	**53**	**100**

Table 6 shows that of those who had premature removal due to menstrual disorders spotting was the commonest (41.5%) followed by menorrhagia.

TABLE 7: Age by reason for early implant removal.

n for removal	Age in Years					
	Age < 20(n=18)		Age 20≤30(n=64)		Age 30≥40(n=56)	
	No.	%	No.	%	No.	%
gain	4	22.2	6	9.4	1	1.8
ual changes	14	77.8	26	40. 6	13	23.2
he	3	16.7	2	3.1	4	7.1
tion	1	5.5	2	3.1	2	3.6
nd complexion s	12	66.7	5	7.8	2	3.6
to conceive	3	16.7	28	43.8	8	14.3
change to r method	0	0	1	1.6	1	1.8

Table 7 shows that weight gain was a very important reason for early discontinuation in those aged below 20 years (22.2%) as compared to other age groups. Menstrual changes was also a leading cause in this age group (77.8%) compared to other groups, while desire to conceive was a leading reason for those aged between 20 to 30 years (43.8%) and plans to change to another method was a leading cause for those in the age group 30 -40 years. In overview, weight gain (22.2%), menstrual changes (77.8%), acne and complexion changes were the predominant reasons for removal in age group less than 20 years. For those aged between 20 -30 years, menstrual changes (40.6%) and desire to conceive (43.8%) were the leading reasons for early discontinuation, while for thos aged over 30 years, menstrual changes (23.2%) and desire to conceive (14.3%) were the leading reasons for early discontinuation.

TABLE 8: Parity and reasons for early implant removal

n for removal	Parity					
	Para 0-2 (n=18)		Para 3-5 (n=115)		Para>5 (n=5)	
	No.	%	No.	%	No.	%
t gain	4	22.2	6	5.2	1	20
ual changes	15	83.3	34	29.6	4	80
che	5	27.8	2	1.7	2	40
tion	3	6.7	1	0.9	1	20
nd complexion s	9	50	6	5.2	4	80
to conceive	16	88.9	20	17.4	3	60
change to method	0	0	1	0.9	1	20

Table 8 shows that for para 0 -2 the predominant reason for premature removal of implants was desire to conceive (88.9%), followed by menstrual changes (83.3%). For para 3 – 5, the leading reason was menstrual changes (29.6%), followed by desire to conceive (17.4%). For para >5, the main reasons were menstrual changes, acne and complexion changes (80%). Weight gain (22.2%) was a major reason amongst para 0-2 as compared to other parities.

TABLE 9: Association between age 30years and reasons for early implant removal (n=138)

Reasons	Age		OR (95%CI)	P-values
	≤ 30, n (%)	> 30, n (%)		
Weight gains	6(7.3)	3(5.4)	1.4(0.3-5.8)	0.647
Menstrual change	38(46.3)	35(65.5)	0.5(0.3-1.0)	0.062
Headache	5(6.1)	4(7.1)	0.8(0.2-3.3)	0.807
Acne complexion	6(7.3)	15(26.8)	0.2(0.1-0.6)	0.002
Palpations	5(6.1)	2(3.4)	0.3 (0.2-0.5)	0.060
Desire to conceive	15(18.3)	4(7.7)	2.9(0.8-11.1)	**<0.001**

Table 9 shows at age 30 and the ODDS ratio tabulated, desire to conceive had clear association as reason for removal with a P value of <0.001. The other reasons for removal show no significance (P values >0.001).

DISCUSSION.

This was a retrospective cross sectional descriptive study which was undertaken at Family Health Options of Kenya during the period March to July 2008. A total of 138 clients were recruited, having had their implants removed within the first three years of insertion. A total of 288 clients had implants removed and so the percentage of those who had their implants removed prematurely was 48%. Of these 63% were married, 26% divorced, 8% single and 3% widowed. The mean age of the study participants was 24.6 years with a median of 28 years.

The commonest parity was 3-5(83.3%), with parity of 5 children and above constituting the least at 4.3%. Majority (97.8%) of the Study participants had attained at least primary education, indicating strong association between implant users and education.

The study found that the commonest reason for discontinuation of implants was menstrual changes at 38.4% followed by plans to conceive at 28.3%. Only 33.3% discontinued implant for all the other medical reasons. Early removal with intent to use another method was minimal at 1.4%. The study shows that most of the early discontinuations were initiated by the clients themselves (92.0%) and only 8% were initiated by the health provider. This could be due to the fact that the side effects, if and when they occur are not of any serious health hazard.

The above concurrs with other studies, Mc Cauley AP Geller J.S. in Zambia [14] found that the leading cause of Norplant removal was bleeding disorders at 67%, followed by intent to conceive at 21% hence similarity in causes

though the percentages differ. Omenge Thesis[9] , study revealed that of the menstrual disturbances, spotting was the commonest at 53.2% with amenorrhoea being the least common at 12.8%[10]. The bleeding disorders are manageable conditions and study by Nilson CG, Hoffman P [4] proper medical intervention would reduce the implant removal to less than 18% at 12 months as seen in Malaysia. In our study, of the menstrual disorders, spotting was the commonest at 41.5%, followed by menorrhagia at 37.7%, amenorrhoea was leat at 20.8%

There was no significant change in diastolic blood pressure in the first three months and the one or so recorded was of no scientific significance.

Implants are long lasting contraceptives and where desire for child spacing is required as is the case for most para 1 – 3 mothers. Chances are high that by 24 month, removal is likely due to desire to conceive hence the rise in percentage after 2 years of use.

The effectiveness of implants as contraceptives is evident and could explain the tendency that people with at least primary educational background would prefer to use implants. Ruminjo and Achwal [2] found 40% of users to have secondary education. In our study, 31.2% had secondary education and about 50% had above primary education.

Early removal of implant as requested by client was at 92% with only 8% being health providers initiated. This shows that the reasons for removal were actually not life threatening and with counseling and medical intervention could be controlled.

---

[9] 16
[10] 16

Most clients who had bleeding disorders could have opted for implants removal due to fear of malignancy, especially Cancer of the Cervix which is very common in our set up and also presents with spotting in early stages and bleeding as its advances. Frequent screening for the same would thus cause assurance and hence reduce removal of the complaints.

In this study, a number of reasons were advanced for requesting implants contraceptives removal. It was noted that the leading reason advanced by most clients by the end of the 1st year post insertion was menstrual disturbances thus constituted 38.4% comparatively, Omenge [16] found 41.5%, Ruminjo and Achwal [2] 40.6% and Levine AS et al [8] found 39.2%.

Despite menstrual alteration being the leading cause of early Norplant removal, there are no standard drug regimes for managing menstrual disruption, to compliment counseling. Ethinyl oestradiol, combined oral contraceptive pills, anti-prostaglinds and other non steroidal anti inflammatory agents and even uterine curettage have been tried under different conditions, with varying success. A special diagnostic concern is that irregular bleeding is a common presentation of cervical carcinoma, the leading cause of death from gynaecological cancer in Kenyan women which should therefore be sought and ruled out. Amenorrhea is uncommon, as is menorrhagia [5].

Implants are popular as contraceptive method in clients of age 30 years and below, being 59.4% of the users and 40.6% being over 30years.The method seems to be more appealing to married women with 63% and least preferred by widows, at 3.0%.

Of the users, the parity was significant with those Para 3-5 preferring this long term contraceptive which also has short term for return to normal fertility. These stood at 83.3% clients.

Women with menstrual disorders were likely to opt for early removal of implant as the study revealed-38.4% of the discontinued users and only followed by intent to conceive at 28.3%. The two groups constituted 66.7% leaving the rest of factors to only 33.3%.

Thus women of high parity with education, married, aged over 20yrs, and with no menstrual disorders are not likely to discontinue the implants.

In this study, there were several reasons advanced for implant removal but the leading reasons was menstrual alterations thus similar to study by Omengo-2004, 41.5% and Ruminjo et al in 39.2%. Similar study in Shangai China correlates at 30.1 % [28. 13].

At approximate removal cost of £29, the control of bleeding disorders and the psychological impact that goes with it would go along way to save costs incurred due to removal. Thus good counseling and availability of Health Management Services is essential. The resultant amenorrhea has for long

been managed in combined oral contraceptives or on Ethinyl oestradiol with good results and better still on assurance and counseling. Menorrhagia has been managed on oestradiol, minipill but to disappointments as it soon recurs, use of primolut. N has shown good results (4, 6, and 15)

Table 7 shows that weight gain was a very important reason for early discontinuation in those aged below 20 years (22.2%) as compared to other age groups. Menstrual changes was also a leading cause in this age group (77.8%) compared to other groups, while desire to conceive was a leading reason for those aged between 20 to 30 years (43.8%) and plans to change to another method was a leading cause for those in the age group 30 -40 years. In overview, weight gain (22.2%), menstrual changes (77.8%), acne and complexion changes were the predominant reasons for removal in age group less than 20 years. For those aged between 20 -30 years, menstrual changes (40.6%) and desire to conceive (43.8%) were the leading reasons for early discontinuation, while for those aged over 30 years, menstrual changes (23.2%) and desire to conceive (14.3%) were the leading reasons for early discontinuation.

CONCLUSIONS

1. The major reason for removal of implants was menstral changes followed by desire to conceive .

2. There was no significant change in the clients diastolic blood pressure during the first one year.
3. Premature removal of implants is mainly client intiated and constitutes 90% of cases.
4. There is significant association between age, parity, reason for removal (side effects) and premature implant removal.
5. Weight gain, menstrual changes and complexion changes are important reasons for early discontinuation in clients aged less than 20 years while desire to conceive and menrual changes are the leading reasons in those over 30 years.
6. Desire to conceive is the leading reason for premature removal of implants in clients of para 0-2 while menstrual changes is the leading reason in those of para 3-5.

RECOMMENDATIONS

1. To enhance the popularity of implants good counseling is required not only prior to insertion but also during use and before discontinuation.

2. The adverse effects arising from implant use are manageable medical conditions, and with good health support can be treated promptly.Hence the need for valuable medical personnel.

3. Choice of implants for the right clients taking in consideration age, parity and possible side effects would enhance its acceptability and prolonged use.

4. The reasons for implant early removal are manageable side effects and with good health support systems can be controlled.

REFERENCES

1. **Hatcher RA/ Kowal D**
Implants, Injection and Other Progestin Only Contraceptive:
Contraceptive Technology, Atlanta: 166:281 year 1997.

2. **Ruminjo and Achwal**
Norplant Removal and Discontinuation Among Users in Kenya:
Journal of Obstetrics and Gynaecology for East and Central Africa: 13(1); 32; 1997.

3. **Fouler P.**
Norplant, a General Practitoner's Perspective One Year On.
British Journal of Family Planning 21 (1);5;2000

4. **Nilson C.G , Hotman P.**
Menstrual Blood Loss With Contraceptive Subdermal Levonorgestrel Implants.
Fertil.Steril: 35(3); 304; 1999.

5. Croxatto, Diaz S.
Clearance of Levonorgesterel From Circulation Following Removal of Subdermal Norplant Implants.
Contraception: 18(5): 506; 1998

6. Ololo E. J and Bronham C.O
Treatment of Menstrual Side Effects in Norplant Users (Leeds UK):
British Journal of Family Planning: 21(1); 3: 1998.

7 Smallwood G H ; Meador ML; Lenhar J.P shangold GA; Fisher AC Greasy GW
Efficancy and safety of a transdermal contraceptive system
Obstetric and gynecology 2002:5 pf 1

8.*Subdremal contraceptive implant continuation rates among adolescents and adults in family planning clinic.*
Levine AS; Holmes MM; Haseldon C; Buther W
Gynecology g (2) 67-70, 2001 April

9.**Mayeait LY Yr Maddox SA Baudoin M**
Early Removal of Norplant systems

Journal of American Board of Family Practice (3): 215-9, 2004 May –June

10. Diaz J: Founder A; Olmas P
Bleeding complaints during the first years of Norplant implant use and their impact on removal rate
Contraception 57(2):103-111 2003 feb.
Hormonal Contraceptive Manual for clinical family planning practice
KMA and MOH 12; 123:1995

11. **Barnett B;finge R.W and Kim B.**
Norplant first in a new generation of Long acting Hormonal methods
Family planning International: 11(4); 2001

12. **Konje J.C, Ofolorin E.O**
Return of ovulation after removal of Norplant Sub dermal Implants.
British journal of Family Planning: 18(2); 44; 2002

13. **Selvin J, Sanchez FA Holma C etal**
Experience with Norplant Subdermal
Contraceptive Ferlil, Steril: 39(6):7992002

14. **Mc Cauley AP, Geller J.S**
Decision for Norplant Programs
Population reports series K:(4);1;1996

15. **Shoupe D.M, Bopp. B.L, Fielding M.**
The significance of bleeding patterns in norplant users
J.Obs. Gynae 77; 256:199

16.**Dr. Omenge thesis obs\gyn. 2004**

Appendix

Appendix 1-Client consent form

Induction of Labour at Kenyatta National Hospital

Dear client

My name is **Dr. Fredrick O. Obago**; I am a post graduate student at the **University of Nairobi**, doing a prospective study, **Induction of labour at Kenyatta National Hospital** among women presenting for delivery at the labour unit, KNH. I am requesting you to consider participating in the study. I am assuring you of strict confidentiality to the information you will give to me. No names will be written on the questionnaire. If you prefer not to be part of the study; please feel free to say so. If you have accepted to participate, please

sign below

Client's signature_____

Date_____

Uchunguzi kuhusu uwezeshaji wa uchungu wa kuzaa katika hosipitali kuu ya kitaifa ya kenyatta

ITHINI

Kwa Mteja,

Kwa majina naitwa **Dkt. Fredrick O. Obago**. Mimi ni mwanafunzi wa

Shahada la pili katika chuo kikuu cha Nairobi.

Niko nafanya uchunguzi kuhusu **Uchunguzi kuhusu uwezeshaji wa Uchungu wa kuzaa katika hosipitali kuu ya kitaifa ya Kenyatta.**

Ninakusihi wewe ufikirie kujumuhishwa kwa uchunguzi huu.

Ninakuhakikishia kwamba kila kitu kitakuwa siri na hutaandika jina

Lako popote. Kama hutaweza kujuhishwa, basi usiwe na shaka kusema la!

Ukikubali kushiriki, tafadhali weka sahihi.

Sahihi yako_____

Tarehe_____

Appendix 2

Questionnaire: - Induction of labour

Date interviewed.....
Study number.....Hospital number.....

Section A - Client Record (Social demographic profile)

I. Age.....
II. Parity.....
III. LMP.....
IV. Marital status:
    1) Single.....
    2) Married.....
    3) Separated/divorced.....
    4) Widowed.....
V. Age of spouse.....
VI. Level of education:
    1) None.....
    2) Primary.....
    3) Secondary.....
    4) College/Secondary.....

VII. Religion of the client
    1) Catholic.....
    2) Protestant.....
    3) Muslim.....
    4) Other.....
VIII. Occupation
    1) Employed.....
    2) Not employed.....
    3) Self-employed.....

Section B: - Indication for induction of labour

I. At what stage was the induction started (state in weeks).....
II. What was the indication for induction of labour
    1. BOH
    2. Postdates
    3. PROM
    4. IUFD

5. Others (Tick)
    - Multiple pregnancy
    - Macrosomia
    - Fetal growth restriction
    - Maternal request

III. What method was used?
1. Amniotomy
2. Amniotomy + Oxytocin
3. Oxytocin
4. Prostaglandin
5. Prostaglandin and later Oxtocin
6. Others (tick)
    - Membrane sweeping
    - Hygroscopic dilators
    - Enema and warm bath
    - Breast Stimulation

IV. How long did labour last?
1. 12 hours or less
2. 12- 24 hours
3. More than 24 hours

V. Did any complications occur during labour
1. Yes
2. No

VI. IF YES in (V) above specify its nature

VII. _____

_____

_____

Section C: - Bishops score

I. Done.....

II. Not done.....

If done, score

1. >7

4. <7

Section D: Outcome of induction of labour

I. What was the mode of delivery?
   1. SVD
   2. Cesarean section
   3. Vacuum Extraction

   If cesarean section, state indication_____
   _____
   _____

II. What was the fetal outcome
   1. FSB
   2. Live baby
   3. MSB
   4. Early neonatal death

III. Apgar Score after 1,5,10 minutes
   1. 1-.....
   2. 5-.....
   3. 10-.....

IV. What was the fetal birth weight
   1. <2000g.....
   2. 2000 – 2999g.....
   3. 3000 – 3999g.....
   4. _> 4000g.....

V. State amount of blood loss during labour & delivery
   1. <100m/s.....
   2. 100 – 200m/s .....
   3. 200 – 500m/s.....
   4. >500m/s.....

(a) Did any complications occur during the 1st 24hours? (Both Maternal & fetal
   1. YES.....
   2. No.....

(b) If yes in V (a), what was the nature of the complication(s)?

_____

_____

_____

VI. Maternal Outcome: was there a maternal mortality?

1. YES……..
2. NO……..

(a) If yes in VI, what was the cause of the death?

_____

_____

_____

VII was there a maternal morbidity?

Yes_____

No _____

If yes specify_____

IX (a) would you consent to induction of labour in Future?

1. YES……
2. NO……..

(b) State reason for willingness or unwillingness in 7 (a) Above? _____

_____

_____

X Would you recommend the procedure to another person?

Yes……..

No……

If no state reason_____

_____

_____

End of questionnaire

ESTIONNAIRE

TA COLLECTION AND ENTY FORM

DENTIFY FACTORS

lient family planning number: - | | | | | | | |

Case study No. | | | |

Date client 1st attended to | | | | | | | |

Social –demography Characteristics married
1. Single
2. Married | |
3. Divorced
4. Widow

Age in completed years | | | | | | | |

Parity | | | | |

Number of living children
a. 0-1
b. 1-2 | |
c. 4-5
d. More than 5-4

Education Level

1) Nil-1
2) Primary-2
3) Secondary-3
4) Above secondary-4

Occupation:

a) Non employed-1
b) Self –employed-2
c) Employed-3

IMPLANT USE: -

Date of Implant insertion

Date of first follow up visit

Complaints during the follow up visits

Medical: - -Headache 1
-Breast/nipple discomfort 2
-Weight gain 3
-Fatigability 4
-Others 5

Menstrual problems - Amenorrhea 6
-Spotting 7
-Mennorhagia 8

Implant site complaints 9

- Date of second follow up visits

- Complaints / remarks during Second visit

as C above]

- After 12 months
  24 ''
  36 ''

BLOOD PRESSURE READING

- BP on insertion
- BP on 1st visit
- BP on 2nd visit
- BP at 12 months

DISCOUNTINUATION

- Early Implant removal Yes---1

  No---2

If yes go to 5b

- Early removal if client requested 1
- Early removal if recommended by health provider
- Main reason for early removal
  -Menstrual disturbance - 1
  -Medical problems -2
  -Implant site complaints -3

-Desire for pregnancy -4
-Desire for another method -5
-Others specify -6

Date of removal | | | | | | | |

Completed years with Norplant |

3-3 5-5
4-4