

CASE RECORDS AND COMMENTARIES  
IN  
OBSTETRICS AND GYNECOLOGY

SUBMITTED BY DR. LYDIA OKUTOYI

AS  
PART FULFILMENT FOR THE DEGREE OF MASTERS OF  
MEDICINE IN OBSTETRICS AND GYNAECOLOGY  
OF THE  
UNIVERSITY OF NAIROBI

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## **DEDICATION**

This work is dedicated to my loving husband Dr Daniel Ojuka Kinyuru and our daughter Roselee Odira and our son Joseph Okutoyi. I also dedicated to my parents Wycliffe and Jane-rose Okutoyi of Kitale

## **DECLARATION**

I certify that the cases recorded and the 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 presented here were treated and operated by me under the supervision of the senior members of the department of Obstetrics and Gynaecology at the Kenyatta National Hospital, Nairobi, Kenya.

SIGNATURE.....*Lydia Okutoyi*

DATE .....*21/1/07*

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## CERTIFICATE OF SUPERVISION

This is to certify that the long commentaries were researched upon by Dr. Lydia Okutoyi under our guidance and supervision and that this book is submitted with our approval.

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This is to certify that Dr Okutoyi Lydia managed Obstetric case Nos 2, 3, 4, 8, 10 and 12 and Gynaecologic cases 1, 2, 6, 8, 13 and 14 under my supervision at the Kenyatta National Hospital.

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1. **AIDS – Acquired Immunodeficiency Syndrome.**
2. **ANC – Antenatal clinic**
3. **ARV – Antiretroviral.**
4. **BHITS – The Breastfeeding and HIV International Transmission Study.**
5. **CIS – Carcinoma In -Situ**
6. **CPD Cephalopelvic Disproportion.**
7. **DOA – Date of Admission.**
8. **DOD – Date of Discharge.**
9. **EDD – Expected Date of Delivery**
10. **EBRT – External Beam Radiotherapy.**
11. **GBD – Gestation by Dates**
12. **GOPC – Gynecological Out Patient Clinic.**
13. **ICC – Invasive Cancer of the Cervix.**
14. **HAART – Highly Active Antiretroviral Therapy.**
15. **HSIL – High grade Squamous Intra-epithelial lesion.**
16. **HIV – Human Immunodeficiency Virus.**
17. **KNH – Kenyatta National Hospital.**
18. **LMP – Last normal Menstrual Period.**
19. **LSIL - Low grade Squamous Intra-epithelial lesion.**
20. **MTCT – Mother to Child Transmission.**
21. **PMTCT – Prevention of Mother to Child Transmission.**
22. **TAH – Total Abdominal Hysterectomy.**
23. **TVH – Total Abdominal Hysterectomy.**
24. **VCT – Voluntary Testing and Counseling.**

## **INTRODUCTION**

The obstetric and gynaecology short cases and long commentaries were managed at Kenyatta National Hospital. It is situated in Nairobi about 3km from the city centre along Ngong Road. It was started in 1901 as the Native Civil Hospital and later became King George's Hospital. In 1964, it was renamed Kenyatta National Hospital (KNH). It serves as a teaching and referral centre as well as serving the population within the city. It provides curative, preventive and rehabilitative services in all medical disciplines. It is a training centre for undergraduate and postgraduate students from the college of Health Sciences of the University of Nairobi and for nurses, clinical officers and other paramedics from the Kenya Medical Training College.

The hospital is housed in a 10-storey building complex that houses wards, operating theatres, outpatient clinics, casualty, intensive care unit, renal unit, burns units, laboratories, and pharmacies. The hospital is currently administered as a state corporation by a Parastatal Board established in 1986 by an Act of parliament.

St Mary hospital is situated approximately 6 kilometres from central business district. It caters for the middle and low class population in Nairobi and its environs.

## **OBSTETRIC AND GYNAECOLOGY UNIT (KNH)**

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

The unit is divided into three Firms, each headed by senior consultants, with a team of senior registrars, registrars, interns, and medical students. The senior medical staffs are from both the University of Nairobi and KNH.

Laboratories services are provided by the hospital laboratories. The Department of Obstetrics and Gynaecology of the University of Nairobi also offers the following laboratory services for the hospital: semen analysis, hormonal radio-immunoassay, cytology, chromosome analysis, bilirubin spectra-photometry, surfactant test and glucose tolerance test. Radiological examinations such as

ultrasound are provided in radiology department of KNH and also at the department of Radiology University of Nairobi.

### **Casualty department**

The gynaecologic casualty is run by registrars and offers services 24 hours a day. All gynaecologic emergencies are screened here. Most patients are treated and discharged or referred to the gynaecology or obstetric clinics. Patients requiring admission are admitted to the acute gynaecology ward.

### **Antenatal care (ANC)**

Antenatal patients are booked on Monday mornings by senior registrars/registrars from one of the three firms who work in rotation, booking about 50 clients every week. The patients report to the clinic at 7.30 am and are interviewed by the nurse who record personal data, medical and obstetric history. The patients' height weight and blood pressure are taken and urinalysis is done.

High risk patients are noted. The risk factors considered include: Primigravida (especially the adolescent and those above 35 years); previous operations or complicated deliveries (e.g. vacuum extraction, caesarian section, post-partum haemorrhage, and ruptured uterus); grandmultiparity; bad obstetric history including habitual abortion, still births or neonatal deaths; medical diseases complicating pregnancy such as cardiac disease, hypertension, renal diseases, diabetes mellitus, anaemia, and thyroid disease; Rhesus negative clients; previous gynaecological problems e.g. repaired genital fistulae, myomectomy, tubal surgery for infertility or ovulation induction. Other factors include; multiple gestation, breech presentation and pre-eclampsia.

All booked patients have their names entered into the register. The patient then proceeds to the examination room where a registrar does a thorough general and systemic examination noting the gestational age, uterine size, foetal lie and presentation, foetal activity and heart atone and the condition of the mother and the findings recorded on the antenatal card. Patients requiring admission are admitted to the relevant wards.

For the rest of the patients, appointments for follow-up are given and antenatal profile done i.e. Haemogram, VDRL (screen for syphilis), blood group and Rhesus factor determination and HIV serology by ELISA.

Subsequent follow-up is usually every 4 weeks up to 28 weeks gestation and 2 weekly, from 28 weeks to 36 weeks and weekly thereafter till delivery. However for patients with obstetric or medical complications, the frequency of follow-up is individualized. At each visit health education about pregnancy, breast care, puerperium and baby care is given. For first pregnancies or previous pregnancies older than 3 years, two tetanus toxoid doses are given 4 weeks apart, otherwise only booster T.T. is given during the second trimester.

Antenatal patients who present with complaints on days other than their clinic days are seen and treated in the labour ward.

At 36 weeks of gestation, clinic pelvic assessment is done on all primigravida and those with one previous scar. Radiological pelvimetry is done on patients with borderline pelvis or one previous scar with cephalic presentation. Amniocentesis for surfactant test is done at 38 weeks in those mothers who are planned for elective delivery to assess foetal lung maturity especially when the length of gestation is in doubt. Patients for elective caesarean delivery are admitted at 38 weeks of gestation.

### **Hospital Admissions**

These fall into three categories namely:-

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

The last two categories constitute the majority of admissions.

Booked patients are seen in labour ward when they are in labour or whenever they have a problem on days other than their clinic days or during weekends and at night. Unbooked patients are first seen in casualty before being sent to labour ward admission area. In labour ward all patients are seen by House Officers and Senior House Officers. Those in labour or requiring delivery are managed accordingly in labour ward. Patients not due for delivery but require admission are sent to the admitting antenatal ward.

Patients who need close monitoring are admitted to the acute room in labour ward and managed as required. The senior registrar and/or consultant are consulted as necessary.

## COMMON OBSTETRIC PROCEDURES

### **Vaginal examination**

This is an aseptic procedure and involves the speculum and/or digital examinations depending on the indication.

### **Digital vaginal examination**

The nature of the procedure is explained to the patient and a verbal consent obtained. After washing hands, and wearing sterile surgical gloves, the patient is placed in dorsal position with knees flexed. The vulva is inspected and any abnormalities noted. It 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 then 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 right and left labia minora. The left hand now separates the labia using the index and thumb fingers and the introitus is gently swabbed anteroposteriorly using the right hand.

The right index and middle fingers are gently introduced into the vagina noting the position, direction and the status of the mucous membrane of the vagina. 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. Clinical pelvimetry may also be assessed.

### **Speculum examination**

In obstetric speculum examinations, the bivalve Cusco's speculum is commonly 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 examiner washes hands and wears sterile gloves. The vulva is swabbed as described above. The labia are then separated with the index and thumb of one hand. The Cusco's speculum is then gently introduced into the vagina with the blades transverse and away from the urethral orifice. The lateral vaginal walls are observed for any abnormality after opening the blades.

The cervix is observed for dilation, bleeding, and drainage of liquor or bulging membranes. If the indication of the speculum examination is premature rupture of membranes and does not reveal any

liquor, the patient is asked to cough or fundal pressure is applied. The speculum is withdrawn if the same way it was introduced.

## **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 labour.

### **Management of the first stage of labour**

Patients may be admitted in active or latent phase of labour. Progress of labour is recorded graphically on a partogram where uterine contractions, foetal heart rate, maternal pulse rate and blood pressure are recorded half hourly. Vaginal examination to assess the cervical dilation in cms, presence and degree of moulding or caput and colour of 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 hyocine bromide 40g is given routinely to hasten cervical dilation. In patients at cervical dilatation of 4 to 6cm an intramuscular injection of pethidine or tramadol 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, cervical dilatation is marked on the alert line for patients in active phase of labour 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 interventions are instituted accordingly. Interventions may involve augmentation of labour if contractions are poor or caesarean section delivery if there is cephalopelvic disproportion (CPD). Augmentation of labour with oxytocin is avoided in those patients with a previous uterine scar, maternal or foetal distress and those who are grand multipara.

Induction of labour routinely starts with cervical ripening where the bishop score is unfavourable usually with prostaglandins followed by artificial rupture of membranes and oxytocin drip where required.

### **Management of second stage**

When the patient is confirmed to be in second stage by vaginal examination and abdominal examination, 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 on duty. Aseptic precautions are observed. The vulva and perineum are cleaned with antiseptic solutions and draped with sterile towels. The patient is encouraged to bear down with each contraction and to take deep breaths between contractions. Foetal heart rate is monitored every 5 minutes.

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

Once the head has been delivered, the mouth and nose are wiped with gauze to prevent aspiration of blood or amniotic fluid. A finger is swept around the foetal neck to check if there is a nuchal cord. If there is a tight cord around the neck, it is divided between two clamps. If it is loose, it is slipped over the head. The anterior shoulder is delivered followed by the posterior shoulder and the trunk. At delivery of the anterior shoulder, 0.5mg of Ergometrine is given intramuscularly to the mother except where contraindicated, like in hypertensive disease.

The umbilical cord is double clamped and cut. The baby is shown to the mother for sex identification before handing over to another midwife who carries out oropharyngeal suction as needed and keeps the baby warm. A paediatrician is usually in attendance in high risk cases.

### **Repair of episiotomy**

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

### **Management of the fourth stage**

Blood pressure, pulse rate, uterine contraction and lochial loss are observed and recorded. The patient is encouraged to empty the bladder. The patient is then observed half hourly for two hours and then

transferred to the postnatal ward for subsequent observations. Patients with normal delivery are discharged home after 24 hours.

## **OPERATIVE DELIVERY**

### **Vacuum extraction**

Vacuum extraction 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 where expedite delivery is desired as in foetal distress occurring in the second stage of labour.

The procedure and its indications are explained to the patient and 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 cephalopelvic disproportion and malpresentation. A mediolateral episiotomy is given under local anaesthesia. The largest suitable vacuum cap is applied on the foetal scalp taking care not to include maternal soft tissues by running a finger round the cap.

Suction pressure is then built up slowly at a rate of about  $0.1\text{kg}/\text{cm}^2$  per minute up to a maximum of  $0.8\text{kg}/\text{cm}^2$ . This allows for formation of an artificial chignon within the ventouse cap that holds firmly and allows adequate traction.

Traction is then applied with each contraction, in a downward direction following the pelvic axis until the head descends and then upwards to allow delivery by extension. On delivery of the foetal head the pressure is released. The rest of the delivery is completed as described above.

## **CAESAREAN SECTION**

The lower uterine segment caesarean section is the commonest caesarean section whereas the classical caesarean section is rarely done.

### **Pre-operative care**

Caesarean section operations are either emergency or elective. For elective caesarean section, baseline investigations like Haemogram and urea and electrolytes are done, blood is taken for grouping and cross matching and two units of blood are reserved. An informed consent is obtained. The patient is starved for at least six hours before the operation. The abdominal wall and the perineum are shaved before theatre. Premeditation 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 obtained. The abdominal wall and the perineum are shaved prior to the operation. The patient is premedicated with atropine 0.6mg intramuscularly before being wheeled to theatre. For cardiac patients, 0.4mg of hyoscine is used instead.

### **Intra-operative procedure**

In theatre the patient is placed in dorsal position with the knees flexed, 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 wall is cleaned with antiseptic solution and draped 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. The rectus sheath is opened with curved Mayo's scissors. The divided Rectus sheath is elevated with two artery forceps and the Rectus muscle separated from their attachment to it, and the peritoneum exposed. 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 spilling into the general peritoneal cavity. A Doyen's retractor is applied to reflect the bladder away and expose the utero-vesical fold of peritoneum.

The utero-vesical peritoneum is lifted up with a pair of dissecting forceps and incised. The incision is extended transversely in an elliptical fashion. The peritoneum is stripped off the lower uterine segment with mounted swab and retracted away with the Doyen's retractor. A small incision of about 2cm below the uterine attachment of the utero-vesicle peritoneal fold is made. 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 trans-abdominal 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 and all the membranes are removed from the uterine cavity. Bleeding margins of the incision are held with Green Armitage clamps. In transverse lie, breech presentation or deeply engaged head, the baby is delivered by breech extraction.

The uterine incision is repaired in 2 layers with chronic catgut stitch No 2 on atraumatic needle ensuring haemostasis. The utero-vesicle peritoneum is then closed with a continuous chromic catgut stitch No 1/0.

The abdominal packs are removed and the pelvic viscera inspected for any abnormalities. After ascertaining correct count of instruments and swabs, the abdomen is closed in 3 layers. Peritoneum is closed with continuous No 1/0. Chromic catgut stitch, Rectus sheath is similarly closed with No 1 vicryl stitch and skin with interrupt silk or nylon or subcutaneous vicryl 3/0. The wound is cleaned and dressed. The catheter is checked for the urine drainage and removed if the urine is clear. 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. The patient is extubated and oropharyngeal suctioning done.

### **Post-caesarean care**

The vital signs; blood pressure, pulse rate, respiration and body temperature are observed continuously until the patient is fully awake then 2 hourly for 2 hours and 4 hourly thereafter. Intravenous fluids are given until she can take orally.

Intramuscular pethidine 50-100mg is given every 4 hours for the first 48 hours to relieve the pain. She is also given antibiotics crystalline penicillin 2MU 6 hourly and gentamycin 80mg 8 hourly intravenously. Metronidazole 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 are present and medications are converted to the oral route. On the third post-operative day, Haemoglobin is checked. The non absorbable stitches are removed after seven days of operation. The patient is discharged home with an appointment 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. Sick babies or those at risk of complications are transferred to New Born Unit (NBU). The premature babies are managed in NBU until their weights are about 2000gms when they are discharged. All babies are immunized with BCG (*bacilli Calmette Guerin*) and oral polio vaccine before discharge. Normal mothers who have babies in NBU are lodged in the mother's hostel.

### **Post-natal follow-up**

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

## **THE GYNECOLOGY UNIT**

The Gynaecology unit consists of the outpatient wing at clinic 18 and two gynaecological wards 1 B and 1D on the first floor of the tower block.

Ward 1 D is the acute gynaecological ward while ward 1 B caters for the cold cases. The unit is managed by the three firms in the department.

### **Gynaecology outpatient services**

There are three outpatient clinics per week. Specific firms run the clinics on different days; Firm 1 on Tuesdays, Firm II on Thursdays and Firm III on Wednesday. The clinic is run by consultants, senior registrars and registrars. Teaching of the medical students takes place in 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 are done. The investigations ordered depend on the diagnosis after history and physical examination. Some of the investigations include: pelvic ultrasound, semen analysis, hysterosalpingiogram, Pap smear and pregnancy tests among others.

### **Family planning clinic**

The clinic is situated at the Family Welfare Centre (clinic 18). Oral and injectable contraceptives, intrauterine contraceptive devices, Norplant and barrier methods are offered. Previously, this clinic was situated at clinic 66 where a theatre for laparoscopy and tubal ligation procedures was available.

### **Acute gynaecological admissions – ward 1 D**

This is the 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. An average of 15 patients is admitted daily and more than two thirds of these cases are abortion related. 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 the registrar, senior registrar and consultant.

Apart from abortions, pelvic inflammatory disease and ectopic pregnancies are the next most common cases admitted into this ward.

Uterine evacuation done is done for uncomplicated cases of incomplete abortion in the procedure room in 1 D using Karman's cannula and syringe. They are discharged after being counselled about contraception.

Patients with suspected carcinoma of the cervix are usually managed as outpatients. They are only admitted when it is absolutely necessary e.g. severe anaemia for blood transfusion, antibiotic treatment etc. Examination under anaesthesia staging and biopsy is done in the caesium theatre. After staging and biopsy the patient is taken either to ward 1 B for Wertheim's hysterectomy or taken to radiotherapy unit for radiotherapy treatment. Patients with confirmed carcinoma of the cervix but have complications such as bleeding, anaemia etc are also admitted to ward 1 D for management of the complications.

**Cold gynaecology admissions – ward 1 B**

Ward 1 B is the non-emergency gynaecology ward to which 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 due to gynaecology malignancies. Uterine fibroids, vesico-vaginal fistulae (VVF), tubal infertility and gynaecology malignancies are among the conditions necessitating patient's admission to this ward.

**GYNAECOLOGICAL OPERATIONS**

A theatre is reserved in main theatre for emergency gynaecology operations daily. Laparotomy for ectopic pregnancies, pelvic abscesses, ovarian cyst and other tubo-ovarian masses are done here.

Smaller procedures like dilatation and curettage of the uterus, removal of misplaced intra-uterine contraceptive devices, marsupialization of Bartholin's abscess and suction curettage are also performed.

Elective operations are done on a firm basis with Firm II on Mondays; and Firms I and III on Thursdays. The operations are done from 8.00 am to 5.00 p.m. The operations are 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**

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

For cold (non-emergency) operations, baseline investigations such as the full Haemogram, urea and electrolyte levels are done and the date of surgery fixed. The nature and purpose of the operation is explained to the patient and an informed written consent for the operation is obtained. Blood is ordered and reserved for the day of the operation. For most operations gut preparations is done by enema at 5.00 a.m. on the operation day. The patient starves from midnight prior to 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 50-100mg 6 hourly for 24 to 48 hours is routinely given for analgesia. Prophylactic antibiotics are given routinely. A check haemoglobin level is determined on the third post-operative day.

The patient is informed about the findings at operation before discharge and is issued with a discharge summary. Patients are reviewed in the gynaecology clinic after six weeks or earlier when there is an indication.

The most common acute gynaecological operation is laparotomy due to ruptured ectopic pregnancy while total abdominal hysterectomy is the commonest cold gynaecological operations done in this unit. Total abdominal hysterectomy is described below.

### **Total abdominal hysterectomy**

General anaesthesia, induction and maintenance are done as described above. A vulvo-vagina 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 vagina is then painted with methylene blue dye. The abdomen is thoroughly cleaned with hibitane or savlon and painted with iodine and then draped with sterile towels.

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

The next step depends on whether the tube and the ovary are to be retained or removed. If they are to be retained, the tube and the ovarian ligaments are double clamped en masse and divided 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 No1 or No 0. The same is done for the opposite side.

The reflection of the bladder peritoneum onto the uterus is then freed by extending the incision in the interior 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 Vicryl 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.

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 the broad ligament, the free margin of the uterosacral ligament, then through the infundibulo-pelvic ligament, the free margin of round ligament and the anterior bladder peritoneum. The suture is tied at the centre. The same is done for the opposite side with the suture being tied at the midline and lateral angles. Suspension of the vaginal vault is done by tying the peritonization suture to the lateral and mid sutures of the vault. 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, ischemia and cystic changes of the ovary. After ensuring homeostasis, abdominal viscera are inspected and instruments and swabs counted. The abdomen is then closed in anatomical layers.

The post-operative management is as described above.

## **COUNSELLING CLINICS**

Clinics in the hospital which offer counselling to obstetrics and gynaecology patients include the patient support centre, GOPC, teenage clinic and the Nairobi Hospice.

### **The patient support centre**

This is situated in the old hospital building where patients regularly attend from all the departments of the hospital. Sometimes the counsellors are called to the wards to counsel those inpatients. The

counsellors consist of psychiatrists, sociologists, psychologists and trained nurses. Mostly, they deal with HIV counselling, puerperal psychosis patients and other psychiatric disorders.

### **The high risk clinic (HRC)**

This clinic is situated on the ground floor next to the maternity wards. It deals with young single mothers with problems such as unwanted pregnancies, induced abortion, sexually transmitted infections etc. The counsellors are also trained nurses, sociologists and consultant obstetrician/gynaecologists.

They counsel their clients, treat them for any illness they may have 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 diseases. 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.

### **THE HOSPITAL CHAPLEL**

This provides spiritual nourishment to patients it is situated on level 2 of the tower block.

### **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.

**Gynecological case 1****CARCINOMA OF THE OVARY STAGE IV- TAH BSO AND CHEMOTHERAPY:**

NAME: E.G

IPNO: 1099473

AGE: 66 YEARS

DOA: 22/8/06

PARITY: 4+2

DOD: 25/8/06

**Presenting Complaints:**

She presented with progressive abdominal swelling for 3 months.

**History of the Presenting Complaints**

She was well until three months prior to admission when she developed abdominal distension that was slowly progressive but initially not associated with pain. However two weeks prior to admission she developed abdominal pain. She had associated nausea and lack of appetite, but no vomiting. She had no vaginal discharge or bleeding. There was no history of urinary or bowel symptoms and had not noticed any weight loss.

**Obstetric and Gynecological History**

She was Para 4+0, the last delivery was 1967. All the deliveries had been vaginally all the children were alive and well. She was about 10 years postmenopausal. She had been on DMPA and combined oral contraception to space her children. She had never had a Pap smear.

**Past Medical History**

Not significant.

**Family and Social history**

She was single, stays in Nyahururu and not in any formal employment. She did not drink alcohol or smoke. There was no family history of any chronic illness. There is no familial history of breast or ovarian cancer malignancy.

## PHYSICAL EXAMINATION

### General examination

She was an elderly obese patient who was not pale, not jaundiced and not febrile. She had a blood pressure of 120/75 mmHg, a pulse rate of 80 beats per minute, a respiratory rate of 20 breaths per minute and a temperature of 36.0°C.

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

### Abdominal Examination

The abdomen was distended but moved with respiration. The flanks were not full and the umbilicus was everted. There was no area of tenderness. There was a palpable epigastric mass arising from the right hypochondria, which was non-tender and nodular. A pelvic mass corresponding to 18 weeks of a gravid uterus which was both cystic and firm was palpated. It was mobile and had a smooth surface.

### Pelvic Examination

She had normal external genitalia. The vagina and the cervix were atrophic. The cervix was posterior smooth and closed and the uterus was not bimanually palpable. There was a mass arising from the left adnexa to the pouch of Douglas. No blood was seen on the examining finger.

### Impression

An impression of carcinoma of the ovary was made.

### Investigations

- Full Haemogram: Hb 11.3g/dl; WBC  $4.7 \times 10^9/L$ ; PLT  $260 \times 10^9/L$
- Renal function tests: Na 144mmol; K 4.1mmol/l; BUN 5.3mmol/l Cr 84umol/l
- Pap smear - normal.
- Abdominal pelvic ultra-sound scan; Showed left lobe of liver was enlarged with multiple intra-hepatic masses. Large septet cystic; left adnexal mass with mainly solid areas; and a small right adnexal mass. Some ascites.
- CA 125 was 154mmol
- CXR was normal.

## Management

She was admitted to the cold gynecological ward (1B) and prepared for exploratory laparotomy. The nature of illness was explained to her and she gave an informed written consent. Bowel preparation was started 2 days before surgery; she was put on laxatives and a light diet. Enema was given the night before surgery. On the morning of surgery another enema was given. She was premeditated with atropine 0.6mg intramuscularly 30 minutes before being wheeled to theatre.

In theatre she was put under general anaesthesia and placed in lithotomy position. Vulvo- vaginal toilet was done followed by aseptic catheterization of the urinary bladder. On examination under anaesthesia, she had atrophic external genitalia with a small uterus and a left adnexal mass.

In supine position the abdomen was opened through a lower midline incision.

Intra-operative findings were: Large pelvic masses arising from the left ovary. Tumour seedlings to the omentum, peritoneum and the liver had massive hepatomegaly. There was a small right ovary with some cysts and some mild ascites. The surface under the diaphragm appeared normal.

Done: Debulking; Total abdominal hysterectomy and bilateral salpingio-oophrectomy.

Also done was infra-colic omentectomy and all the specimens were taken for histology-pathological evaluation. Peritoneal lavage was done with normal saline. The abdomen was closed after correct swab and instrument count.

## Postoperative Care

She was observed half hourly until she was fully conscious and then hourly for 2 hours and 4 hourly thereafter. She was put on crystalline penicillin 2mega units 6 hourly and gentamycin 80 mg 8 hourly intravenously for 48 hours, then Ampiclox 500 mg 6 hourly for 5 days. She was put on Pethidine 100 mg 8 hourly for 24 hours then continued with Brufen tabs 500 mg 8 hourly for six days. On the first post op day bowel sounds were heard and she was advised to ambulate and start oral sips. These were later graduated to free fluids the light diet by the second day post operative. The wound was exposed on the third post-operative day.

Histology confirmed a well differentiated serous cystadenocarcinoma of the ovary.

She was started on cytotoxic drugs, a combination of cyclophosphamide and cisplatin given as start doses. These drugs were repeated every 3 weeks. Haemogram, liver function test and renal function tests done prior to each course were confirmed to be normal. The patient received 6 courses of the chemotherapy. She had improved clinically with no evidence of ascites or pelvic masses. The

hepatomegally was gradually shrinking. CA 125 done prior to surgery was 154 and those done after 2 courses of chemotherapy were 36. 64.

She was discharged to be followed up in the gynecology out-patient clinic.

## DISCUSSION

E.G presented with an abdominal mass, which was suspected to be an ovarian tumor. She had total abdominal hysterectomy and a bilateral salphingo-oophrectomy. Histology confirmed a well differentiated serous cystadenocarcinoma. She received 6 courses of cisplatin and cyclophosphamide.

Among the gynecological tumours, malignancies of the ovary poses a great challenge. There insidious onset that lead to late diagnosis and the intensive surgical treatment they require and complex chemotherapy are extremely demanding on the patients' psychology and physical energy (1).

Ovarian cancer is the 4<sup>th</sup> common cancer affecting women. Annual world-wide deaths are estimated to be 140,000 highest in the developed world (Scandinavia and North America) lowest in (Africa and India)

(1,2). Studies done in our set up show that ovarian cancer is the third commonest gynecological malignancy after cancer of the cervix and choriocarcinoma(3,4). In Kenya the mean age of patients with ovarian cancer was reported to be 46.7 years with the majority of the cases being between 40-60 years (3). Our patient was 66 year which is between the world peak and our local Kenyan peak age. The world peak age is between 70-75 years (1).

Categorization of ovarian cancers is done based on the cell type of origin. The ovary could also be a metastatic site of tumours from other organ sites, like metastatic disease from especially the breast and the gastrointestinal tract (krukenberg tumors.) Unlike cancer of the cervix and endometrium, precursor lesion for ovarian carcinoma has not yet been found. (1, 5)

The majority of the primary ovarian cancers are derived from epithelial cells, although they can also arise from other cell types (germ cell tumors, sex cord stromal tumors, and mixed cell types tumors.

Epithelial ovarian cancer (EOC) makes 85% of all ovarian cancer and 50% of these are serous cystadenocarcinoma while the rest are endometrioid, mucinous, clear cell and mixed cell type (1,5).

EOC occurs in older women. Any factor that reduces the life time ovulatory cycles is thought to reduce the risk of ovarian cancer. These include (high parity, combined oral contraceptive use, late age at menarche, early menopause and early age at 1<sup>st</sup> pregnancy. Other associations are with lactose intake, cholesterol consumption (\* especially in eggs) low intake of green leafy vegetables, exposure to asbestos, perineal talc. There has not been found a consistent association with smoking (1, 6). History of having had a simple hysterectomy or bilateral tubal ligation is protective. Small proportion of ovarian cancer is familial. Women who are high risk as far as familial history is concerned should be investigated. Those found with the mutation (BRCA1, BRCA2) should be managed accordingly, options available prophylactic mastectomy +/- bilateral oophorectomy, hormonal manipulation. (1, 6)

Symptoms are usually of insidious onset vague and ill defined. They may not be severe enough for the patients to seek medical attention early. Symptoms commonly presented are abdominal discomfort, bloating constipation, irregular menstrual cycles, lower back pain fatigue, urinary frequency or dysperunia. (5)

Acute symptoms due to ovarian rupture or torsion are unusual. As a result 70-85% of the cases are diagnosed at advanced stages on first presentation. 40%-60% present with a palpable mass, while another 20%-30% present with clinically detectable ascites. Other signs noted are pleural effusion, leg edema, cathexia, inguinal and supraclavicular lymphadenopathy (5). Our patient had abdominal swelling for three months and a pelvic mass arising from the right adnexa.

Factors that lead to delay in diagnosis are also the omission by the health care provider to carry out a pelvic examination, presence of multiple symptoms, younger age of patient, not ordering an ultra-sound scan, or CT scan, and a previous assignment of an inaccurate diagnosis e.g. depression, stress, irritable bowel, or gastritis)

The presence of a solid, irregular fixed pelvic mass on pelvic examination is highly suggestive of malignancy (7). The diagnosis is almost certain if there is an accompanying upper abdominal mass and ascites. Serum CA125 should be done routinely pre-operative. This is a glycol-protein tumor marker that is elevated in 80% of women with ovarian cancer. The perimenopausal women with an

adnexal mass may undergo a period of observation if the mass is not clinically suspicious on ultrasound. Adnexal masses that are mobile, cystic, unilateral, less than 8 to 10 cm in diameter and regular in internal and external contours by ultra-sonography are highly unlikely to be malignant(7). These can be followed for a period of two months; the majority will regress during this period. If there is no resolution within this 2 months period then explorative laparotomy is indicated. Oral contraceptives could be used to suppress the ovaries during these periods of observation.

However women who have fixed, irregular, solid or large masses should undergo surgery. Masses that increase in size and do not regress should be considered neoplastic and operated on (7).

The diagnosis of ovarian cancer is typically made or confirmed during explorative laparotomy for a pelvic mass. Optimal staging is key to accurate prognosis and determining appropriate adjuvant therapy. Accurate staging requires the use of a midline incision to approach the peritoneum. This facilitates removal of tumor, any peritoneal fluid, ascites or wash out (saline lavage). A thorough inspection of the liver spleen, kidney, stomach, lesser sac, omentum, all peritoneal surfaces, both leaves of the diaphragm, large and small bowel and the pelvic organs. 40% of the patients are usually under staged (8).

The aim of surgery is maximum cytoreduction. For stage IA and IB surgery maybe curative. The standard procedure is TAH and BSO and infra-colic omentectomy. Those with early disease and adequate staging undergo unilateral oophrectomy if fertility is desired. Laparoscopic surgery is a contentious issue where malignancy is suspected. Primary cytoreduction has not been seen to improve the survival of these patients. Neo-adjuvant chemotherapy (2-3 cycles) with subsequent secondary cytoreduction aiming to leave less than 2 cm tumor area. Palliative surgery could be done to relieve bowel obstruction (8).

Recent studies suggest that all women with ovarian malignancies should benefit from chemotherapy regardless of stage. Those with advanced disease chemotherapy is the major adjunct to surgery in the treatment of there disease. Combination therapy is the current treatment of choice. In early 70s single agent melphalan (alkylating agent) was used. Latter cyclophosphamide, doxorubicin and cisplatin (CAP) were added with better results. Paclitaxil was added in the 90s it added 12 months to the survival of these patients. Response rates of 50% for CAP, Carboplatin/ cisplatin 65%, Paclitaxil alone 50% and Cisplatin with Paclitaxil 75% (9). Our patient benefited from the best combination

used so far with good response. The 2 monthly pelvis assessments have been normal with the CA125 levels remaining low.

Issues to be considered for research are effective combination of chemotherapy, ideal timing for surgery, gene therapy and molecular prognostication (1).

**REFERENCES**

1. Jennifer Byrom and Quentin Davis. Cancer of the Ovary. *Currents Obstetrics and Gynecology* 2005; 13, 88-94
2. Greenlee RT, Murray T, Wingo PA. Cancer statistics 2000 *CA Cancer J Clin* 2000; 50:7
3. Njuki SK. Primary Ovarian Malignancy: The presentation at KNH. Mmed Thesis University of Nairobi 1979.
4. Ojwang SBO, Makokha AC, Sinei SKA. Ovarian cancer in Kenya *EAMJ* 1980 57:131.
5. Oslon SH, Mignone L, Capto TA. Symptoms of Ovarian Cancer. *Obstet Gynecol* 2001; 98:212.
6. Jackie cook: Family history of Ovarian Cancer. *Currents Obstetrics and Gynecology* 2005; 15. 54-59
7. FortnerR, Chen M, Hricak H. Imaging of Ovarian Cancer. *J Magn Reso Imaging*. 1995; 606-613.
8. Hunter RW, Alexander NDE, Souter WP. Meta-analysis of surgery in advanced ovarian cancer – is maximum cytoreduction an independent determinant. *Am J Obstet Gynecol* 1992 166: 504-511 prognosis
9. Thegpin JT. Chemotherapy for Advanced Ovarian Cancer. Overview of randomized trials. *Semin Oncol* 2000; 27 ( suppl 7)

**Gynecological case 2****TITLE: LEIYOMAYO-SARCOMA.**

NAME: P.N            IP NO: 1106270  
AGE: 53 YEARS      DOA: 22/08/06  
PARITY: 3+2         DOD: 12/09/06

**Presenting complaints**

Abdominal swelling for 6 months.

**History of presenting illness**

P.N. was well until 2 years prior to admission when she noticed abdominal swelling. She underwent a total abdominal hysterectomy due to symptomatic uterine fibroid. The histology results revealed a metastatic leiomyosarcoma.

The mitotic index on high power field not indicated. Six months prior to admission she noticed swellings on the epigastric area and medial side of right lumbar region. For these she was admitted into the surgical and managed as multiple abdominal wall abscesses. They were drained; fine needle aspirate done showed bloody discharge and inflammatory cells in a proteinous background no neoplastic cells seen. She was referred from the surgical wards after the histological report of a FNA taken from a pelvic mass confirmed malignant cells likely metastatic from previous uterine primary.

**Past Medical History**

She is a known hypertensive for the last 5 years controlled on Losartan. History of being hospitalized at Nairobi Hospital due to pulmonary embolism in 1991 and Nairobi Women's hospital due to anaphylaxis to radiological contrast iodine in 2006. She is also allergic to Co-Amoxiclavulin.

**Obstetric and Gynecologic history**

She was Para 3+2, the last delivery was 1979 and all were by spontaneous vertex delivery. In 1975 and 1976 she had spontaneous abortion at 4 months gestation and evacuation was done for both of them. She had used injectable hormonal contraception (DMPA), but stopped after her blood pressure started rising. She later had bilateral tubal ligation in 1985. She had done 4 pap smears in her lifetime all were normal.

### **Family and Social History**

She was a married retired secretary, now a business woman, and lives in south C with her spouse. There was no family history of chronic illness in her family and she neither drank alcohol nor smoked cigarettes.

### **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 had multiple masses in the epigastric region and lumbar area. There was multiple drain scars on the anterior abdominal wall. The abdomen moved with respiration, it was difficult to assess whether there were any intra-abdominal masses because of the huge multiple superficial masses.

### **Vaginal examination**

The external genitalia were normal. The vaginal vault felt normal. The adnexae were free.

## **MANAGEMENT**

### **Investigations**

1. Abdominal ultrasound 05/08/06.: There were multiple low abdomen both subcutaneous and intraabdominal breaking down masses. There was also a fixed right iliac fossa mass. There was associated ascites. The kidneys and liver looked normal. These were most likely metastatic deposits and biopsy was recommended.
2. CT scan abdomen 14/8/06: The liver was of normal size and shape. The surface was smooth and the liver edge was sharp. No focal liver mass or dilated bile ducts were seen. The pancreas, spleen and gall bladder were normal. Both kidneys were of normal size, shape and situation. There was no hydronephrosis ascites or para-aortic lymphadenopathy. There were multiple lobulated hypogastric masses with an aggregate size of 161x155mm. The uterus and ovaries were not seen. Urinary bladder was normal.  
Conclusion: Lobulated hypogastric masses likely due to recurrent tumour.
3. FNAC (of the pelvic mass) 17/8/06: Cellular smears consisting of malignant cells lying singly and in groups were seen. The nuclei were enlarged, hyperchromatic with prominent

nucleoli and cytoplasmic vacuolation. Pleomorphism was marked. Features were consistent with those of metastatic carcinoma. Metastases from previous uterine primary were likely.

4. CXR: Lung fields were clear, heart size and shape were normal.
5. Culture report from I & D done 5/7/06 – no organisms were seen on gram stain. No growth was obtained.
6. Grouping and crossmatching of 2 units of blood was done before starting chemotherapy and was transfused due to an Hb of 8.5g/dl.

	1 <sup>st</sup> course	2 <sup>nd</sup> course	3 <sup>rd</sup> course
RFTs	139	141	139
Na+ (mmol/L)			
K+ (mmol/L)	4.9	4.3	4.4
C/- (mmol/L)	100	101	99
Urea (mmol/L)	2.5	3.5	2.4
Cr (µmol/l)	73	96	100
LFTs	5.5	7.6	8
T. Bil (µmol/L)			
D. Bil (µmol/L)	2.1	1.2	1.2
Alk Phos (IU/L)	-	215	362
Gama GT (IU/L)	-	27	34
AST (IU/L)	-	43	35
ALT (IU/L)	-	19	19
Haemogram			
WBC x 10 <sup>9</sup> /L	10.2	6.3	3.06
Hb (g/dl)	11.2	10.2	10.1
PLT x 10 <sup>9</sup> /L	465	327	802

## Treatment

She was reviewed by the gynecological oncology team and the radio-oncologist and put on chemoradiation

The diagnosis and the intended management were explained to her. She was started on a course of intravenous cyclophosphamide 1.2g stat, Adriamycin 50mg stat and Vincristine 2mg stat on 23/8/06. Prior to that, she was given 2 litres of normal saline overnight and another 1 litre after the chemotherapy. Radiotherapy was started on 28/8/06 of 2 Grays each and she was to get 30 sessions. Since she had completed the course of chemotherapy, she was discharged for a 3 week rest period before the next course of chemotherapy. She was to continue with radiotherapy as an outpatient. She came back for the chemotherapies as scheduled. The baseline investigations were normal before each course of chemotherapy as shown above. She tolerated chemotherapy well. At each admission, abdominal pelvic examination was done and the pelvic mass was found to be reducing in size. By the 3<sup>rd</sup> course of chemotherapy she had already done 23 sessions of radiotherapy and tolerated well. The report was written after her 3<sup>rd</sup> course of chemotherapy but she was scheduled for 6 courses every 3 weeks.

## DISCUSSION

Presented is P.N.M. a 53 year old para 3+2, a known hypertensive. She had presented with a one week history of vomiting and abdominal distension 9 months after total abdominal hysterectomy for degenerating uterine fibroids. A histological diagnosis of leiomyosarcoma had been made. Further evaluation revealed recurrent leiomyosarcoma. She was started on chemotherapy and radiotherapy and by the time the report was written, she had received 23 sessions of radiotherapy out of 30 and 3 courses of chemotherapy out of 6 with some improvement.

Leiomyosarcoma (LMS) is a rare malignant neoplasm composed of cells that have smooth muscle differentiation. The typical patient with leiomyosarcoma is in her mid 50s and seeks treatment for abnormal bleeding (1). In most cases, diagnoses are determined (post operatively) after microscopic examination of a uterus removed because of suspected leiomyomas as happened in the case presented.

The median age for women with leiomyosarcoma (43 to 53 years) is somewhat lower than for other uterine sarcomas and premenopausal patients have a better chance of survival. This malignancy has no relationship with parity and the incidence of associated diseases is not as high as in endometrial adenocarcinomas or malignant mixed Mullerian tumours (2). There is a higher incidence and a poorer

prognosis in black women. A history of prior pelvic radiation therapy can be elicited in about 4% of patients with leiomyosarcoma. The incidence of sarcomatous change in benign uterine leiomyomas is reported to be between 0.13% and 0.81% (2, 3). Our patient was 53 years and black. It is not clear whether the sarcoma arose denovo or in a myoma. Only about 5-10% of leiomyosarcomas are reported to originate in a leiomyoma. The outcome is better in those that develop in a pre-existing benign leiomyoma.

Presenting symptoms that are usually of short duration (mean, 6 months) and that are not specific to the disease include vaginal bleeding, pelvic pain or pressure, and awareness of an abdominopelvic mass (3). Abnormal uterine bleeding is the most common symptom of LMS occurring in about 60% of patients; 50% describe some types of abdominal pain or discomfort; 30% complain of gastrointestinal or genitourinary symptoms and only about 10% are aware of an abdominal mass (4). Occasionally a pedunculated tumour prolapses through the cervix where it is accessible for biopsy. The deeply situated intramural position of most tumours impedes diagnosis by D & C, which is accurate in only 25% of cases. The Pap smear may be abnormal; more frequently, however, the true nature of the disease becomes evident after the fact, when pathologic analysis of hysterectomy specimen reveals cancer. The patient presented had a 1 year history of abnormal uterine bleeding, pain and uterine enlargement for which hysterectomy was done for symptomatic uterine fibroids. The pathologic analysis revealed leiomyosarcoma.

LMS spread by contiguous growth, invading the myometrium, cervix and surrounding supporting tissues (4). Lymphatic dissemination is common in the late stages. Pelvic recurrence and peritoneal dissemination following resection are also common. In the more malignant types, haematogenous metastasis to the lungs, liver, kidney, brain and bones probably occurs early but is clinically evident only in the lungs until the advanced stages.

The clinical behaviour of the tumour can usually be predicted by the number of mitotic figures identified on microscopic examination. Tumours with less than 5 MF/10 HPF behaves in a benign fashion and tumour with more than 10 MF/10 HPF is frankly malignant with a poor prognosis. Tumours with 5-10 MF/10 HPF are less predictable. Therefore, in addition to mitotic index greater than 10, other histological indicators used to classify uterine smooth muscle tumours as malignant are severe cytological atypia and coagulative tumour cells necrosis (5). Uterine smooth muscle tumours with any two of these three features are associated with a poor prognosis. Gross presentation of the

tumour at the time of surgery is also an important prognostic indicator. Tumours with infiltrating tumour margins or extensions beyond the uterus are associated with poor prognosis, whereas tumours less than 5cm in size, originating within myomas, or with pushing margins are associated with prolonged survival. Interestingly, they metastasize to regional nodes in only about 4%. The patient presented had a large necrotic endometrial mass exhibiting moderate dysplasia with a number of atypical mitotic bodies. We can therefore conclude that it was of poor prognosis despite non infiltration to the myometrium and serosa.

There is no official FIGO staging for uterine sarcoma but most clinicians tend to use the surgical staging for endometrial cancer.

Surgical treatment involves total hysterectomy with bilateral salpingo-oophorectomy with removal of the adnexae and any suspicious regional nodes or metastatic nodules (6). If the cervix is also involved, radical hysterectomy should be done. Adjunctive treatment with radiation appears to offer no benefit and may indeed impede future chemotherapy or surgical treatment. Watchful expectancy may be extended in cases of sarcoma detected accidentally from the well capsulated fibroid following myomectomy. Our patient only had TAH since the pre-operative diagnosis was uterine fibroids. Regional nodes were not sampled for metastases. Histology having come back as not infiltrating the myometrium and serosa, a watchful expectancy was extended, however, she presented with recurrence within 9 months of surgery.

Several chemotherapeutic agents have been tried in cases with metastatic disease. Doxorubicin, cisplatin and Ifosfamide are the drugs used either singly or in combination with varying response. Recurrent disease should be treated with chemotherapy. Combinations of hydroxyurea, Dacarbazine and Etoposide (6) have been used with good responses. Our patient was treated with cyclophosphamide, Adriamycin and Vincristine together with adjunctive radiotherapy due to recurrent disease.

The prognosis is unsatisfactory. The 5 year survival rate ranges from 10-30 percent.

**REFERENCES**

1. Taylor H.B., Norris H.J.: Mesenchymal tumours of the uterus. IV diagnosis and prognosis of leiomyosarcoma. *Arch Pathol* 1966; 82:40.
2. Gudgeon D.H.: Leiomyosarcoma of the uterus. *Obstet Gynecol* 1968; 32:96.
3. Silverberg S.G.: Leiomyosarcoma of the uterus: a clinicopathologic study. *Obstet Gynecol* 1971; 38:613.
4. Anne Kathryn Goodman. Premalignant and malignant disorders of the uterine corpus. In: Alan H. Martin L.P.: *Current obstetric and gynaecologic diagnosis and treatment* 8<sup>th</sup> ed. Appleton & Lange. 1994, P. 937-951.
5. Bell S.W., Kempson R.L., and Hendrickson M.R.: Problematic uterine smooth muscle neoplasms: A clinicopathologic study of 213 cases. *Am. J. Surg. Pathol.* 1994; 18:535.
6. Rock J.A., Jones III H.W.: Uterine sarcomas. In: *Telindes Operative Gynaecology* 9<sup>th</sup> edition. Lippincot Williams & Wilkins Pub. 2003, P. 1474-1480.

**Gynecological case 3****SYMPTOMATIC UTERINE FIBROIDS – HYSTERECTOMY**

NAME: L. W.

IPNO: 1046924

AGE: 42 YEARS

DOA: 04/04/2006

PARITY: 1+0

DOD: 20/04/06

**Presenting Complaints**

She presented with prolonged heavy menses for 3 year.

**History of the Presenting Complaints**

She presented to the gynecological outpatient clinic (GOPC) with a 3 year history of prolonged heavy menses associated with dysmenorrhea. Her menses were heavy during the first two days being in clots and lasted 7 to 9 days and she changed fully soaked pads about 5 times a day. Prior to these she had moderate flow changing pads 3 to 4 times a day and the periods lasted for 4 days. She had noticed a progressively enlarging painless lower abdominal mass associated with heaviness for 2 years. However no urinary complains were reported, had normal bowel habits. .While in GOPC she had received haematinics and analgesics, buying time to get funds for surgery.

**Obstetric and Gynecological History**

She was para 1+0; this was a vaginal delivery in 1982. This was followed by a period of infertility but no medical help was sought. She attained menarche at 18 her periods were initially regular of moderate flow before the onset of these problem. Her last menstrual period was on 18/03/06

**Family and Social History**

She had separated from her spouse 10 years ago due to the inability to conceive.

She neither smoked cigarettes nor took alcohol. There was no family history of any chronic illness.

**Past Medical History**

She had no significant past medical and surgical history

**Physical Examination**

She was in fair general condition not pale, afebrile, no oedema or lymphadenopathy. Her blood pressure was 120/80 mmHg, respiratory rate of 16 breaths per min, pulse rate of 80 beats per min and a temperature of 36.5<sup>0</sup>C.

**Systemic Examination**

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

**Abdominal Examination**

She had lower abdominal fullness with a firm, irregular, mobile, non-tender pelvic mass that was dull to percussion and corresponded to a 24 week gravid uterus.

**Pelvic Examination**

She had normal external genitalia, the cervix was smooth and closed, the adnexa and the pouch of Douglas were normal. Bimanual examination revealed an enlarged mobile uterus corresponding to 24 weeks gravid uterus. There was no tenderness elicited.

**Diagnosis**

A diagnosis of symptomatic uterine fibroids .

**Investigations Done**

1. Pap smear was normal – CIN 0
2. Haemoglobin level of 10.7g/dl
3. Urea and electrolytes ---- Na<sup>+</sup> 136 mmol/l, ; K<sup>+</sup> 3.7 mmol/l; Cl 100 mmol/l, Urea 4.6 mmol/l; Creat 86 mmol/l,

**Management**

She was counselled for a total abdominal hysterectomy and an informed written consent was obtained. She was reviewed by the anaesthetist and found to be fit for the operation. She was starved from the midnight prior to the day of surgery. On the morning of the operation she was given an enema and the pubic hair shaved. She was premedicated with atropine 0.6mg intramuscularly half an hour before been taken to theatre.

**Operative Procedure**

In theatre, she was put under general anaesthesia and placed in lithotomy position. Examination under anaesthesia confirmed earlier findings and aseptic catheterization done. She was placed in supine position then , abdomen was cleaned and draped. The abdomen was open through a lower midline incision.

**Findings**

The uterus was enlarged with multiple subserous and intramural fibroids. She had no adhesions. The ovaries appeared grossly normal.

**Procedure**

The round ligaments were clumped cut and ligated bilaterally opening the anterior leaf of the broad ligament. The incision on the broad ligament was extended anteriorly bilaterally to the vesico-uterine peritoneal reflection which was then opened. The fallopian tubes, and ovarian ligaments were double clumped cut and ligated bilaterally. The bladder was dissected from the anterior uterine wall by blunt and sharp dissection. The uterine vessels were skeletonized and then double clumped cut and ligated. The cardinal and utero-sacral ligaments were clumped ,cut and ligated bilaterally. The upper vagina was held using two little woods forceps and the vault opened between them. The upper vagina was circumcised to free the uterus. The vaginal vault was then closed and the suspended on the round ligament. Haemostasis was achieved and reperitonization done. The abdomen was closed in layers after correct swab and instrument count. The patient was successfully reversed from anaesthesia. The specimen was taken for histology.

**Post Operative Care**

The vital signs were monitored ½ hourly until she was fully conscious and stable and then 4 hourly thereafter. She was started on intravenous fluids normal saline alternating with 5% Dextrose, 1 litre 8 hourly. She was also put on broad spectrum antibiotics and analgesics. She was informed of the intra-operative findings and what was done.

She did well post-operatively and was discharged home on the fourth post- operative day to be seen in the gynecology outpatient clinic (GOPC) after two weeks.

### Follow-Up

She was seen in GOPC after six weeks and she had no complaints. She had no complaints and examination findings were normal. The histology of the specimen revealed uterine leiomyomata. She was given an appointment to be seen after three months.

### DISCUSSION

Uterine leiomyomas are benign tumours that arise from smooth muscle cells of the human uterus. They are composed mainly of smooth muscle cells but contain varying amounts of fibrous connective tissue. Various terms are used to describe the tumour, such as fibromyoma, myofibroma, leiomyofibroma, myoma, fibroma and fibroid. The latter designation is the most commonly used. (1, 2).

Leiomyomas are clinically apparent in 25% of women, though careful examination of surgical specimens suggests that the prevalence could be as high as 77 % (2).

Leiomyomas are 3-9 times more frequent, are also larger and occur at an earlier age in black than in white women. Indeed, by the fifth decade as many as 50% of black women will have leiomyomata (3). At the Kenyatta National Hospital, fibroids account for 1.6% of all gynaecologic admissions and 66.7% of all hysterectomies performed (4).

The etiology of this common tumour is not known (3,5). Leiomyomas are not detectable before puberty and, being hormonally responsive, normally grow only during the reproductive years. Continuous oestrogen secretion, especially when uninterrupted by pregnancy and lactation, is thought to be the most important underlying risk factor in the development of myomata. Leiomyomas generally increase in size with oestrogen therapy and during pregnancy but decrease in size and even disappear after menopause (2, 3). Data from the Nurses Health study suggests that exposure of oral contraceptives between ages 13 and 16 led to an increased relative risk of myomas, whereas use in general showed protection in direct proportion to the duration of use(11).

Several studies have shown that smoking and consumption of green vegetables decrease the risk while consumption of red meats increases the risk of myomas (12, 13).

Myomas are more common in nulliparous or relatively infertile women, but it is not known whether sterility causes fibroids or vice versa, or whether both conditions have a common cause. The general view is that the uterus which is deprived of pregnancies consoles itself with myomas(6).

Leiomyomas are usually multiple, discrete, nodular or irregularly lobulated tumours that vary in size and number. Although they do not have a true capsule the margins of the tumours are clearly demarcated, none infiltrating, and pushing, and are usually separated from the myometrium by a pseudo capsule of connective tissue, which allows easy enucleation at the time of surgery. The cut surface is characteristically whorled (3, 5).

Uterine leiomyomas are classified by anatomical location into;

- Sub-mucous Leiomyomas - lie beneath the endometrium and grow towards the lumen. Their impact on the endometrium and its blood supply most often leads to irregular uterine bleeding. They may develop pedicles and prolapse through the cervical canal and may undergo torsion or get infected.
- Intramural or interstitial - lie within the uterine wall.
- Sub-serous or sub-peritoneal - lie at the serosal surface and can become pedunculated and parasitic (1, 2, 3).

Secondary (degenerative) changes that may occur in fibroids include: atrophy, hyalinization, liquefaction (cystic degeneration), red or carneous degeneration, calcification, septic, and myxomatous (fatty) degeneration. Malignant transformation (leiomyosarcoma) is reported to develop with a frequency of 0.1-0.5 % (3).

Most myomas cause no symptoms; symptoms being present in only 35-50% of patients. Even very large ones may remain undetected, particularly in the obese patient. Symptoms of leiomyomas depend on their location, size, and state of preservation and whether or not the patient is pregnant (2, 3). Symptoms attributable to myomas can generally be classified into three distinct categories: abnormal uterine bleeding, pelvic pressure and pain, and reproductive dysfunction(1).

Abnormal uterine bleeding is the most common and most important clinical manifestation of leiomyoma being present in about one third of patients with symptomatic uterine fibroids. The

bleeding pattern most characteristic of myomas is heavy (menorrhagia), prolonged (metrorrhagia) or both heavy and prolonged (menometrorrhagia) menstruation. The heavy bleeding can result in manifestations of anaemia such as palpitations and lassitude. Location seems to be a more important than size in determining bleeding symptoms. Bleeding is more common and more severe in the presence of submucous myomas. The abnormal bleeding may be due to an increase in the endometrial surface area, increased vascularity of the uterus, associated endometrial hyperplasia or ulceration of the overlying endometrium(1, 2, 3, 6).

Pain may result from degeneration associated with vascular occlusion, infection, torsion of a pedunculated tumour, or myometrial contractions to expel a submucous myoma from the uterine cavity. Large tumours may produce a sensation of heaviness in the pelvis or discomfort. The weight of an anterior tumour may also cause bladder irritability with frequency of micturition whereas a posterior tumour may cause constipation. A cervical fibroid or a corporeal one may become impacted in the pouch of Douglas and cause urine retention. Oedema and varicosities of the legs are sometimes seen with large fibroids (1, 2, 3, 6).

Leiomyomas are an infrequent primary cause of infertility and have been reported as a sole cause in only a small percentage of infertile patients(4). A number of factors may be responsible for infertility in a patient with uterine leiomyoma. Anovulatory cycles may occur more commonly. There may be interference with sperm transport caused by a distortion or in the surface area within the uterine cavity, impingement of leiomyomata on the endocervical canal or interstitial portion of the fallopian tube or interference with prostaglandin induced uterine contractions, which are thought to enhance sperm migration. Because uterine leiomyomata occur in later reproductive years, relatively greater difficulty accomplishing conception can be expected in older couples(2, 6).

The incidence of spontaneous abortion secondary to leiomyoma is unknown but is possibly 2 times the incidence in normal pregnant women. It occurs when the myomas interfere with enlargement of the fetus, initiates abnormal uterine contractions, prevents efficient placentation, or causes impaction of the uterus in the pelvis. These can also lead to preterm labour (1, 2, 5).

The risk of abruption is substantially increased if a myoma is under the placental site. During the second and third trimesters of pregnancy, myomas may rapidly increase in size and undergo red degeneration. It may also initiate preterm labour. During labour, leiomyomas may produce uterine

inertia, foetal malpresentation, or obstruction to the birth canal. Leiomyomas may interfere with effective contractions immediately after delivery which may lead to postpartum haemorrhage (1, 2, 5).

The diagnosis of myomas is often suspected on the basis of palpation of an enlarged irregular uterine contour on pelvic examination. Ultrasonography is typically used to confirm the diagnosis, as well as excluding pregnancy as a cause of uterine enlargement and ovarian neoplasm (3). MRI gives better visualization of individual myomas, but for most clinical indications the extra cost is not justified (3). Uterine fibroids were confirmed by an ultrasound scan in the patient presented.

### **Treatment**

Most (70% to 80%) uterine leiomyomata (being benign tumors) are asymptomatic and can generally be managed expectantly unless they cause symptoms. Several factors determine the mode of management of fibroids, including the patient's age, parity, pregnancy status, desire for preservation of childbearing potential, severity of the symptoms, general health of the patient, characteristics of the fibroid and the skill of the surgeon (1, 2, 3). The treatment options available for symptomatic uterine fibroids are medical, surgical and uterine artery embolization.

#### **Medical therapy:**

Many algorithms for treating abnormal bleeding due to myomas suggest a trial of oral contraceptives or progestagen therapy before proceeding to definitive therapy.

There is no evidence that this approach is effective and for many women it is ineffective, but with concomitant oligo-ovulation it may be of some use. Non steroidal anti-inflammatory drugs or antifibrinolytic agents which are useful in the treatment of idiopathic menorrhagia have not been studied with myoma-related menorrhagia (2, 3).

Danazol, an androgenic steroid most commonly used for the medical treatment of endometriosis, can be useful by inducing amenorrhoea to control anaemia due to myoma-related menorrhagia. Another androgenic steroid, gestrinone, caused volume reduction and amenorrhoea in women with myomas (14).

GnRh agonists, the mainstay of medical therapy for myomas, work by first increasing the release of gonadotropins, which is followed shortly by desensitization and downregulation to a hypogonadotropic hypogonadal state clinically resembling the menopause. These agents produce a significant reduction in uterine size, generally 35% to 65%, as well as amenorrhoea in most women

(13). However, after discontinuation of the medication, there is rapid resumption of menses and return to pretreatment uterine volume. In addition, the severe hypo-oestrogenism that accompanies this therapy can cause significant symptoms and, most importantly, bone loss that can lead to osteoporosis with long term use. Thus GnRH agonists are primarily used to temporize or allow a woman to prepare for surgery, and this use does have the proven benefit of a documented decrease in blood loss at the time of surgery and an increase in preoperative packed-cell volume. However, because iron alone had a benefit for substantial proportion of patients, the cost and adverse effects of GnRh agonists must be weighed against efficacy (11).

### **Surgical therapy:**

Surgery has long been the main mode of therapy for myomas. There are basically 4 surgical treatments for fibroids: Myomectomy, hysterectomy, hysteroscopic resection and myolysis (10).

Myomectomy is indicated in women desirous of childbearing and wishing to retain the uterus. For women with uterine size of 16 weeks or less and a small number of subserosal or intramural fibroids, laparoscopic myomectomy may be an option.

Use of an electronic morcellator allows the myomas to be efficiently removed in pieces (15). Myomectomy, when it is extensive and significantly involves the myometrium or penetrates the endometrium may necessitate caesarean delivery of subsequent pregnancies. Recurrence of myomas following myomectomy occurs in 15-40% of patients and two-thirds of these require further surgical treatment (1).

Hysterectomy eliminates both the symptoms and the chance of recurrence. For many women who have completed child bearing, this freedom from future problems makes hysterectomy an attractive option. Uncontrolled haemorrhage and unforeseen surgical difficulties during myomectomy may also necessitate hysterectomy (2). In premenopausal women at least one ovary, if normal is preserved to prevent premature menopause. Hysterectomy can be done either abdominally or vaginally. The advantages of the vaginal route are that healing is faster, there is no visible scar and there is less time spent in the hospital (7). Vaginal hysterectomy is indicated for small uterus with fibroids. Large tumours are better removed abdominally (2, 3, 8). The laparoscope can be used to aid vaginal hysterectomy or to free the uterus in its entirety with removal via the vagina (total laparoscopic

hysterectomy). Our patient had no desire for future fertility or of retaining her uterus. She underwent laparoscopic assisted vaginal hysterectomy.

In submucous fibroids, hysteroscopic myomectomy can be done (3).

Myolysis is a variation on the technique of laparoscopic myomectomy in which the fibroid tissue is coagulated rather than removed (15).

For many women who have completed child bearing and for whom bleeding is the primary problem, endometrial ablation alone or in combination with hysteroscopic myomectomy may give relief(1).

Uterine artery embolization is a novel technique that involves blocking the uterine artery permanently and was first reported in 1995. The decreased blood supply to the uterus may prevent further growth to the fibroids and may cause them to shrink. The long term effects of this procedure are still unknown and the safety of pregnancy after this procedure is questionable (8, 9).

In patients with uterine fibroids a pap smear must be done to rule out co-existing cancer of the cervix which will otherwise modify the operation. In our patient, the Pap smear was normal.

## REFERENCES

1. Elizabeth A.S. Uterine fibroids. *The Lancet* vol 357; Jan 27, 2001: 293-8.
2. Breech LL, Rock JA. Leiomyomata Uteri and Myomectomy. In: Rock JA, Jones HW (Eds). *Textbook of Gynecology*, 9th edition, Lippincott Williams & Wilkins, Philadelphia, 2003: 753-792.
3. Memarzadeh S, Broder MS, Weeler AS, Pernol ML. Benign Disorders of the Uterine Corpus, In: DeCherney AH, Nathan L, (Eds), *Current Obstetric and Gynecologic Diagnosis and Treatment*, 9<sup>th</sup> edition, McGraw Hill Companies, 2003: 693-699.
4. Wanjala SHM Uterine Fibroids, 1974-75 at Kenyatta National Hospital, Mmed Thesis, University Of Nairobi 1980.
5. Hillard PJA. Benign Diseases of the Female Reproductive Tract: Symptoms and Signs. In Berek JS (Ed). *Novak's Gynecology*, 13th edition, Lippincott Williams & Wilkins, Philadelphia, 2002:380-382.
6. Tindall VR. Tumours of the Corpus Uteri. In: Jeffcoate's Principles of Gynaecology, 5<sup>th</sup> edition, Butterworth-Heinemann, London, 1987: 418-432.
6. Muhiu G., Kigundu C.B.S., Main F.W., Mati J.K.G. Effect of Orgametril (Lynestrenol 5mg) on Menstrual Blood Loss in Uterine Fibroids. *J. Obstet. Gynecol East. Centr. Afr.* 1986; 5 : 39
7. Muto M.G., Friedman A.J. The Uterine Corpus Leiomyomas. *Kistner's Gynaecology Principles and Practice* 6<sup>th</sup> edition, Mosby, 1995; 147-150.
8. Siskin GP. Uterine Fibroid Embolization. <http://www.emedicine.com>
9. Thomason P. Leiomyoma Uterus . <http://www.emedicine.com>
10. Marshal L.M. A prospective study of reproductive factors and oral contraceptive use in relation to the risk of uterine leiomyomata. *Fertil Steril* 1998; 72: 432-39.
11. Parazzini F. Uterine myomas and smoking: results from an Italian study. *J Reprod Med* 1996; 41: 316-20.
12. Chiaffarino F. Diet and Uterine myomas. *Obstet. Gynecol.* 1999; 94: 395-98.
13. Shaw R. W. New approaches to the management of fibroids. *Cur opin Obstet. Gynecol.* 1991; 3: 859-64.

14. Brosens I.A. Pathogenesis and medical management of uterine fibroids. London. Parthenon Publishing, 1999.
15. Dubuisson JB. Laparoscopic myomectomy and myolysis. *Cur opin Obstet. Gynecol.* 1997; 9: 233-38.

## Gynecology case 4

### AMBIGUOUS GENITALIA – CONGENITAL ADRENAL HYPERPLASIA

NAME: F. K.  
AGE: 18 YEARS DOA; 28.06.2004  
PARITY: 0 + 0 DOD: 28.07.2004

#### Presenting Complaints:

She was admitted from the gynecological outpatient clinic with history of primary amenorrhea, cyclic lower abdominal pains and ambiguous external genitalia.

#### History of the presenting illness

She was admitted from the gynecological out patient clinic with ambiguous genitalia for further investigation and possible vulvoplasty. The decision to investigate her as an inpatient was made on the basis of her coming from far (Kisii) and being an orphan somebody had offered to sponsor her for the management. She was born with abnormal genitalia and was said to have had an operation on her genitalia at the age of 3 months at Kenyatta national hospital . However the old file could not be retrieved as the card was unavailable.

She had presented to the gynecological outpatient clinic with primary amenorrhea and cyclic lower abdominal pains that occurred for 3 days each month for the past 2 years. She was the first of three girls. The other two were aged 14 and 16 years. Both were taller than her, had normal external genitalia and were already menstruating. She had no history of urinary symptoms or incontinence of urine.

#### Past Medical History

Previous surgeries on her genitalia in infancy but the details of it were not clear.

#### Family and Social History

She was an orphaned form 3 student with good aptitude for her class.

### Physical Examination

She was in good general condition. She was of short stature with a deep voice but no facial hairs. She had a height of 143cm and weight of 43kg. She had a blood pressure of 100/60mmHg, a pulse rate of 78 per minute, a respiratory rate of 18 per minute and a temperature of 37.1°C.

She had scanty fine axillary hair; the breasts were poorly developed Tanner class II. The pubic hair was well developed, of normal texture with an inverted triangle distribution.

### Abdominal Examination

The abdomen was not distended and moved with respirations. There was no area of tenderness and no masses or organs were palpable.

### Pelvic Examination

She had prominent labia majora, there were no labia minora, and the urethral opening was noted to be more distal in position. There was no vaginal opening noted.

### Impression

An impression of Ambiguous genitalia was made.

### Investigations;

1. Ultrasound scan – there was an anteverted non-gravid uterus with normal echogenicity. Both ovaries were visualized and were normal. There was marked fluid collection in the pouch of Douglas. The bladder had a normal outline

2. Urea/Electrolytes/creatinine –	Na+	136 mmol/l
	K+	4.5 mmol/l
	CL-	104 mmol/l
	Urea	3.6 mmol/l
	Creatinine	80umol/l
3. Haemogram	WBC	5.15 x 10 <sup>9</sup> /l
	RBC	5.47 x 10 <sup>12</sup> /l
	Hb	15.9 g/dl



**DISCUSSION:**

Congenital adrenal hyperplasia (CAH) is an autosomal recessive disorder leading to inability to synthesize cortisol due to enzymatic deficiency (1). The majority have deficiencies in 21 hydroxylase (>90%) others are 11 $\beta$  hydroxylase (5%), and 3 $\beta$  hydroxysteroid dehydrogenase deficiency (2%). There is inadequate conversion of progesterone to the steroids. The precursors to cortisol accumulate (17 $\alpha$  hydroxyprogesterone, progesterone, and 16 $\alpha$  hydroxyprogesterone). These have a progestine effect of salt losing that may occur, there is also excessive activity of the androgen pathway (1, 2).

Virilizing adrenal hyperplasia is as a result of low levels of cortisol leading to increase in the ACTH levels which stimulate the adrenal androgen production ( androstenedione, dehydro-androstenedione) an inherited abnormality of steroid biosynthesis which result in inability to synthesize glucocorticoids. CAH is the commonest cause of ambiguous genitalia in 46XX individuals (1, 2).

The incidence of homozygous mutation of 12-OH deficiency is one in every 60. while the incidence of heterozygous mutation is 1;14000 in Hispanics and Caucasian and 1;45000 in African Americans. The 3 $\beta$  hydroxysteroid dehydrogenase deficiency which presents with a milder( attenuated) form of disease is more common among the Ashkenazi Jews (2, 3, 5).

Because the adrenals secrete an abnormally large amount of virilizing steroid even during embryonic life, these infants are born with abnormal genitalia. In extreme cases, there is fusion of the scrotal folds and in rare instances it may be mistaken for a penile urethra. The clitoris is greatly enlarged, so that it may be mistaken for a penis (3, 4). Usually, there is a single urinary meatus at the base of the phallus and the vagina enters the persistent urogenital sinus (4). Our patient may have been born with an ambiguous genitalia but detail are missing due to lost file, and passing of both parents leaves us with no information on the status of the genitalia during infancy.

During infancy, provided there are no serious electrolyte disturbances, these children grow more rapidly than normal. Unfortunately, epiphyseal closure occurs by about age 10 with the result that as adults these people are much shorter than normal (3). Progressive masculinization continues with the development of male habitus, acne, deepened voice, primary amenorrhoea and infertility (3, 4).

Hermaphroditism due to CAH must be suspected in any infant born with ambiguous or abnormal external genitalia. All patients with ambiguous external genitalia should have an appraisal of their chromosomal characteristics. In all instance of female pseudohermaphroditism due to CAH, the chromosomal composition is that of a normal female. A pelvic ultrasound in the newborn to determine the presence of the uterus is very helpful and, if positive strongly suggests a female infant. The critical determinations are those of the urinary 17-ketosteroid and serum 17 hydroxyprogesterone levels. With the 21-and 11-B hydroxylase deficiencies, the 17-hydroxyprogesterone levels may be 50 to 400 fold above normal (2, 3, 4).

The treatment of adrenal hyperplasia is to supply the deficient hormone, cortisol. This decrease ACTH secretion and lowers production of androgen precursors. The drug of choice is hydrocortisone or 9-fluoro hydrocortisone. The surgical treatment of the anatomical abnormalities should be carried out in the first few years of life when the patient is still too young to remember the procedures and too young to have developed psychological problems centred about the abnormal external genitalia. If sufficient vaginal reconstruction is necessary this is best accompanied after puberty which makes compliance possible (3, 5, 7)

**REFERENCES:**

1. Hershlag, A, Peterson CM. Endocrine Disorders. In Berek JS. (ED).Novak's Gynecology, 13<sup>th</sup> edition, Lippincott William and Wilkins Philadephia, 2002: 890-893.
2. Biggs JSG. The Adrenal. In: Phillips EE, Barnes J, Newton M (Eds). Scientific Foundations of Obstetrics and Gynaecology, 3<sup>rd</sup> edition; Heinemann Medical Books, London, 1987: 455-462.
3. Speroff L, Glass RH, Kase NG. (Eds) Normal and Abnomal Sexual Development. In: Clinical Gynaecologic Endocrinology and Infertility, 4<sup>th</sup> edition, Williams and Wilkins, Philadelphia, 1989: 379-408.
4. Meyer Banlbug HFL Gender and Sexuality in a classic case of congenital adrenal hyperplasia. Endocrinology and Metabolism. Clinics of North America. 2001; 30;155-171.
5. Langhlin D. Genetic Disorders and Sex Chromosome Abnormalities. In: DeCherney AH, Nathan L. Current Obstetric and Gynecologic Diagnosis and Treatment; 9<sup>th</sup> edition, McGraw – Hill, 2003: 120-125.
6. Tindall VR. Malformations and Maldevelopment of the Genital Tract. In Jeffcoate's Principles of Gynaecology, 5<sup>th</sup> edition, Butterworth-Heinemann, London, 1987:171-174.
7. Schitzer JJ and Donahoe PK. Surgical treatment of congenital adrenal hyperplasia. Endocrinology and metabolism. Clinics of North America 2001; 30: 173-192.

## Gynecological case 5

### TITLE: MULLERIAN DYSGENESIS

NAME: S.W.                      IP NO: 1076073  
 AGE: 26 YEARS                DOA: 2/03/2006  
 PARITY 0+0                    DOD: 4/04/2006

#### Presenting illness

Failure of menstrual periods to start  
 Lower abdominal pain for 6 months

#### History of presenting illness

S.W was first seen at Embu District Hospital 6 months before being seen at Kenyatta National Hospital (KNH) with failure of periods to begin in adolescence and difficulty of coitus. She had developed other secondary sexual characteristics but had never had periods. Her breasts had developed normally at 13 years of age.

When she made her sexual debut at 19 years her boyfriend found intromission difficult. This was the experience of other sexual partners.

Six months before admission to KNH she developed a lower abdominal pain that radiated to the back. The pain was not cyclic in nature. At Embu District Hospital she was noted to have a shallow vagina and excision of the membrane was unsuccessfully attempted.

#### Past Medical History

This was not significant.

#### Obstetric and Gynaecologic History

She was nulliparous and had never used any form of contraception.

#### Family and Social History

She was single and the 4<sup>th</sup> born in a family of 10 children. There was no history of any congenital abnormality in the family. A sister was married with children. There was no family history of any chronic illness. She neither smoked nor drank alcohol. She went to school up-to standard 8.

### **Physical examination**

She was in fair general condition, was not pale and was afebrile.

Vital signs: Blood pressure 120/80 mmHg, Temperature 37.3C, respiration rate 20/min, Pulse 74/min

### **Musculo-skeletal system**

She had a male hairline with reduced axillary hair. Her thyroid gland was not enlarged however her neck was webbed and rather short.

### **Vision**

Her vision fields were normal and visual acuity was normal.

### **Breasts**

The breasts were Tanner class 5, the nipple was everted. There were no breast lumps and no discharge or galactorrhoea.

### **Abdominal examination**

The pubic hair was of the normal female inverted triangle pattern. There was no distension in the suprapubic region. There was a no abdominal tenderness nor any organ enlargement noted.

### **Vaginal examination**

The external genitalia were normal with a normally placed urethra. The vagina was 4cm long and ended blindly.

### **Investigations**

*Haemoglobin:* 14.9 g/dl

*Renal function tests*

Urea: 7.9 mmol/l

Creatinine: 81 umol/l

Sodium: 134 mmol/l

Potassium: 95 mmol/l

#### *Pelvic ultrasound*

The uterus was not clearly visualized. Ovaries were seen they seemed normal.

#### *Barr bodies*

5% acceptable for female. ( Normal: Male: 0-2%, Female: 9-35%)

#### *Intravenous Urography*

Ectopic kidneys noted in the pelvis . The kidney calyces system appear normal. The ureters had an ectopic insertion. The urinary bladder had a normal size and outline.

#### *Diagnostic Laparoscopy.*

The ovaries and fallopian tubes were normal bilaterally. Two rudimentary horns of the uterus were identified. The ovaries were noted to have follicular activity no biopsy was taken.

### **Operation notes**

#### *Vaginoplasty.*

The patient was placed in a lithotomy position cleaned and draped.

The bladder was catheterized aseptically draining clear urine. Under general anaesthesia examination was done and the vagina was found to be ending blindly at 4 centimeters long. The cervix and upper part of the vagina were not developed. There was no hematocolpos.

Transverse incision was made between the rectum and the bladder. Blunt dissection was done into that fibrous tissue between the rectum and the bladder extending the vaginal length to 8 cm. This was packed with iodine gauze.

There was no probability of further canalization of the vagina due to the risk of entering the peritoneal cavity. The pack was removed after 48 hours. Saline sitz bath were recommended she was also put on analgesics (Iboprofen).

### **Discharge instructions**

She was counseled on the inability to conceive but was told that she would be able to develop normal sexual relations.

She was also booked for follow-up at the Gynaecology clinic.

## DISCUSSION

Our Patient had müllerian dysgenesis. Müllerian duct anomalies are an uncommon but often treatable cause of infertility (1, 2, 4, 6). Patients with müllerian duct anomalies are known to have a higher incidence of infertility, repeated first trimester spontaneous abortions, fetal intrauterine growth retardation, fetal malposition, preterm labor, and retained placenta. The role of imaging is to help detect, diagnose, and distinguish surgically correctable forms of müllerian duct anomalies from inoperable forms (3, 8). In some correctable lesions, the surgical approach is altered based on imaging findings (13).

### *Pathophysiology:*

#### *Embryology*

Two paired müllerian ducts ultimately develop into the structures of the female reproductive tract. The structures include the fallopian tubes, uterus, cervix, and the upper two thirds of the vagina. The ovaries and lower one third of the vagina have separate embryologic origins not derived from the müllerian system (2, 5, 6).

Complete formation and differentiation of the müllerian ducts into the segments of the female reproductive tract depend on completion of 3 phases of development as follows:

- Organogenesis: One or both müllerian ducts may not develop fully, resulting in abnormalities such as uterine agenesis or hypoplasia (bilateral) or unicornuate uterus (unilateral).
- Fusion: The process during which the lower segments of the paired müllerian ducts fuse to form the uterus, cervix, and upper vagina is termed lateral fusion. Failure of fusion results in anomalies such as bicornuate or didelphys uterus. Our patient had a bicornuate uterus. The term vertical fusion occasionally is used to refer to fusion of the ascending sinovaginal bulb with the descending müllerian system (ie, fusion of the lower one third and upper two thirds of the vagina). Complete vertical fusion forms a normal patent vagina, while incomplete vertical fusion results in an imperforate hymen.
- Septal resorption: After the lower müllerian ducts fuse, a central septum is present, which subsequently must be resorbed to form a single uterine cavity and cervix. Failure of resorption is the cause of septate uterus (5).

Ovaries and the lower vagina are not derived from the müllerian system. The ovaries are derived from germ cells that migrate from the primitive yolk sac into the mesenchyme of the peritoneal cavity and

subsequently develop into ova and supporting cells. The lower vagina arises from the sinovaginal bulb, which fuses with the müllerian-derived upper two thirds to form the complete vagina.

**Frequency:**

- In the US: Müllerian duct anomalies are estimated to occur in 0.1-0.5% of women. The true prevalence is unknown because the anomalies usually are discovered in patients presenting with infertility. Full-term pregnancies have occurred in patients with forms of bicornuate, septate, or didelphys uteri; therefore, true prevalence may be slightly higher than currently estimated. Simon et al found that in the healthy fertile population, müllerian duct anomalies have a prevalence of 3.2% (5, 6).

**Mortality/Morbidity:** The presence of a müllerian duct anomaly is not associated with significantly increased mortality compared to the general population. Certain types of the anomaly can increase morbidity, such as in patients with obstructed or partially obstructed müllerian systems who present with hematosalpinx, hematocolpos, retrograde menses, and endometriosis. In addition, a fairly high association exists between müllerian duct anomalies and renal anomalies such as unilateral agenesis. Often, the anomalies are found only when dedicated renal imaging is performed after the müllerian abnormality is discovered; however, these patients most commonly present for medical attention because of infertility and repeated pregnancy loss (15).

**Race:** No racial predilection is noted in the literature.

**Age:** Anomalies may be diagnosed in infancy, adolescence, or young adulthood. Female patients may present with a mass resulting from an obstructed müllerian system as infants (Mucocolypos); with primary amenorrhea, mass (Haematocolpos), or delayed onset of menarche as adolescents; or with problems of fertility and/or carrying pregnancy to term as adults. Our patient had primary amenorrhea and with no Haematocolpos (2, 5, 6).

**Anatomy:** Müllerian duct anomalies are categorized most commonly into 7 classes according to the American Fertility Society (AFS) Classification Scheme (1988) as follows (1):

- Class I (hypoplasia/agenesis): This class includes entities such as uterine/cervical agenesis or hypoplasia. The most common form is the Mayer-Rokitansky-Kuster-Hauser syndrome (8, 16), which is combined agenesis of the uterus, cervix, and upper portion of the vagina. Patients have no reproductive potential aside from medical intervention in the form of in vitro fertilization of harvested ova and implantation in a host uterus. Our patient had agenesis of the cervix and upper part of the vagina and so would probably fit in this group.
- Class II (unicornuate uterus): A unicornuate uterus is the result of complete, or almost complete, arrest of development of 1 müllerian duct. If the arrest is incomplete, as in 90% of patients, a rudimentary horn with or without functioning endometrium is present. If the rudimentary horn is obstructed, it may come to surgical attention when presenting as an enlarging pelvic mass. If the contralateral healthy horn is almost fully developed, a full-term pregnancy is believed to be possible.
- Class III (didelphys uterus): This anomaly results from complete nonfusion of both müllerian ducts. The individual horns are fully developed and almost normal in size. Two cervixes are inevitably present. A longitudinal or transverse vaginal septum may be noted as well. Didelphys uteri have the highest association with transverse vaginal septa but septa also may be observed in other anomalies. Consider metroplasty; however, since each horn is almost a fully developed uterus, patients have been known to carry pregnancies to full term.
- Class IV (bicornuate uterus): A bicornuate uterus results from partial nonfusion of the müllerian ducts. The central myometrium may extend to the level of the internal cervical os (bicornuate unicollis) or external cervical os (bicornuate bicollis). The latter is distinguished from didelphys uterus because it demonstrates some degree of fusion between the two horns, while in classic didelphys uterus, the two horns and cervixes are separated completely. In addition, the horns of the bicornuate uteri are not fully developed; typically, they are smaller than those of didelphys uteri. Some patients are surgical candidates for metroplasty.
- Class V (septate uterus): A septate uterus results from failure of resorption of the septum between the two uterine horns. The septum can be partial or complete, in which case it extends to the internal cervical os. Histologically, the septum may be composed of myometrium or fibrous tissue. The uterine fundus is typically convex but may be flat or slightly concave (<1-cm fundal cleft). Women with septate uterus have the highest incidence of reproductive complications. Differentiation between a septate and a bicornuate uterus is important because septate uteri are treated using transvaginal hysteroscopic resection of the septum, while if

surgery is possible and/or indicated for the bicornuate uterus, an abdominal approach is required to perform metroplasty.

- Class VI (arcuate uterus): An arcuate uterus has a single uterine cavity with a convex or flat uterine fundus, the endometrial cavity, which demonstrates a small fundal cleft or impression (>1.5 cm). The outer contour of the uterus is convex or flat. This form is often considered a normal variant since it is not significantly associated with the increased risks of pregnancy loss and the other complications found in other subtypes.
- Class VII (diethylstilbestrol-related anomaly): Several million women were treated with diethylstilbestrol (DES; an estrogen analog prescribed to prevent miscarriage) from 1945-1971. The drug was withdrawn once its teratogenic effects on the reproductive tracts of male and female fetuses were understood. The uterine anomaly is seen in the female offspring of as many as 15% of women exposed to DES during pregnancy. Female fetuses who are affected have a variety of abnormal findings that include uterine hypoplasia and a T-shaped uterine cavity. Patients also may have abnormal transverse ridges, hoods, stenoses of the cervix, and adenosis of the vagina with increased risk of vaginal clear cell carcinoma. Imaging findings are pathognomonic for this anomaly.
- Other classification systems: Other systems (eg, in the surgical literature) classify the anomalies differently. Rock and Adam modified the AFS classification to include a broader collection of anomalies.
  - Class 1 is identical to AFS class I (agenesis/hypoplasia).
  - Class 2 pertains to disorders compromising patency of the reproductive tract. This class includes all anomalies in which incomplete fusion occurs between the descending müllerian ducts and the ascending urogenital sinus (sinovaginal bulb). Milder forms may present with only a thin membrane at the junction, while more severe forms result in a thick atretic tissue involving up to one half of the vaginal length. Our patient would have been in this group according to this classification. Class 2 anomalies also include obstructive or nonobstructive transverse vaginal septa (although not believed to be a disorder of vertical fusion) and cervical agenesis and dysgenesis with or without obstruction.
  - Class 3 describes anomalies in a patent but often duplicated or partially duplicated reproductive tract and includes disorders of lateral fusion such as didelphys, unicornuate, bicornuate, and septate uteri (AFS classes II, III, IV, and V). The disorders arise from impaired fusion and/or septal resorption of fusing müllerian ducts

attempting to form the uterus, cervix, and upper vagina. The disorders may present in one of two forms, including either a symmetric unobstructed form or an asymmetric obstructed form. The distinguishing feature of the asymmetric obstructed form is observed in the obstructed side, which is always associated with ipsilateral renal

**Clinical Details:** Suggestion of müllerian duct anomaly may arise in different clinical situations. In the newborn/infant, the initial presentation may be an obstructed system discovered as a palpable abdominal, pelvic, or vaginal mass (mucocolpos).

Similarly, an adolescent girl may present to a clinician because of delayed menarche and/or an obstructed system presenting as an intra-abdominal mass (hematocolpos). Many patients also have cyclical pain. Our patient had amenorrhoea, a growing pelvic mass and pain which was however not cyclical (1).

Women of childbearing age often present with various problems of infertility, repeated spontaneous abortions, or premature delivery. As part of an infertility workup, routine imaging often detects the anomaly. Occasionally, the anomaly is discovered incidentally during imaging evaluation for another condition or during surgery such as elective sterilization.

**Preferred Examination:** Once a müllerian anomaly is suggested based on evidence from the patient history and physical examination, the clinician may opt for additional imaging workup. Imaging criteria for distinguishing forms of uterine anomalies are based on the configuration of the endometrial cavity and (primarily) on the configuration of the uterine fundus.

Typically, the first examination ordered is a pelvic ultrasound (US) with transabdominal and, if feasible, transvaginal imaging (10). Müllerian duct anomalies may be suggested on transvaginal 2-dimensional (2D) sonographic imaging but may not be excluded on the basis of negative US findings. Newer 3-dimensional (3D) sonographic techniques offer relatively higher sensitivity and specificity (9,12,17).

Hysterosalpingography (HSG), performed under fluoroscopy, allows evaluation of the uterine cavity and tubal patency. Anomalies may be suggested but positive findings often are nonspecific for precise diagnosis (7, 16).

MRI is considered the criterion standard for imaging uterine anomalies (11). MRI provides high-resolution images of the uterine body, fundus, and internal structure. In addition, it can help evaluate the urinary tract for concomitant anomalies. In the past, intravenous urography was used for this purpose. Most types of uterine anomalies can be diagnosed confidently using pelvic MRI.

**REFERENCES:**

1. AFS: The American Fertility Society classifications of adnexal adhesions, distal tubal occlusion, tubal occlusion secondary to tubal ligation, tubal pregnancies, mullerian anomalies and intrauterine adhesions. *Fertil Steril* 1988 Jun; 49(6): 944-55[Medline].
2. Carrington BM, Hricak H, Nuruddin RN, et al: Mullerian duct anomalies: MR imaging evaluation. *Radiology* 1990 Sep; 176(3): 715-20[Medline].
3. Dunnick NR, Sandler CM, Newhouse JH, Amis ES: *Textbook of Uroradiology*. 3rd ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2000: 45-8, 66-7.
4. Egan B: *Congenital Uterine Anomalies*. Hygeia Foundation for Perinatal Loss and Bereavement, Inc. 2(12). Available at: <http://www.hygeia.org/poems24.uterine%20anomalies.htm>. Accessed September 5, 2001[Full Text].
5. Giraldo JL, Habana A, Duleba AJ, Dokras A: Septate uterus associated with cervical duplication and vaginal septum. *J Am Assoc Gynecol Laparosc* 2000 May; 7(2): 277-9[Medline].
6. Golan A, Langer R, Bukovsky I, Caspi E: Congenital anomalies of the mullerian system. *Fertil Steril* 1989 May; 51(5): 747-55[Medline].
7. Haimovici JBA, Tempany CMC: MR of the female pelvis: Benign disease. *Applied Radiology* [serial online]. 1997 Sept: 7-22. Available at: <http://www.appliedradiology.com/articles/pdf/v0026i09/00800242/main.pdf>. Accessed September 5, 2001[Full Text].
8. Hricak H: *Female Pelvis: Anatomy & Benign Disease*. Paper presented at: The 3rd NICER HORIZON Urogenital Radiology Conference; 10-11 April 1999; Perth, Australia.
9. Jurkovic D, Geipel A, Gruboeck K, et al: Three-dimensional ultrasound for the assessment of uterine anatomy and detection of congenital anomalies: a comparison with hysterosalpingography and two-dimensional sonography. *Ultrasound Obstet Gynecol* 1995 Apr; 5(4): 233-7[Medline].

10. Nicolini U, Bellotti M, Bonazzi B, et al: Can ultrasound be used to screen uterine malformations? *Fertil Steril* 1987 Jan; 47(1): 89-93[Medline].
11. Pellerito JS, McCarthy SM, Doyle MB, et al: Diagnosis of uterine anomalies: relative accuracy of MR imaging, endovaginal sonography, and hysterosalpingography. *Radiology* 1992 Jun; 183(3): 795-800[Medline].
12. Raga F, Bonilla-Musoles F, Blanes J, Osborne NG: Congenital Mullerian anomalies: diagnostic accuracy of three-dimensional ultrasound. *Fertil Steril* 1996 Mar; 65(3): 523-8[Medline].
13. Rock JA, Adam RA: Surgery to repair disorders of development. In: Nichols DH, Clarke-Pearson, eds. *Gynecologic, Obstetric, and Related Surgery*. 2nd ed. St. Louis, Mo: Mosby-Year Book; 2000: 780-813.
14. Rosenberg HK, Sherman NH, Tarry WF, et al: Mayer-Rokitansky-Kuster-Hauser syndrome: US aid to diagnosis. *Radiology* 1986 Dec; 161(3): 815-9[Medline].
15. Simon C, Martinez L, Pardo F, et al: Mullerian defects in women with normal reproductive outcome. *Fertil Steril* 1991 Dec; 56(6): 1192-3[Medline].
16. Strubbe EH, Willemsen WN, Lemmens JA, et al: Mayer-Rokitansky-Kuster-Hauser syndrome: distinction between two forms based on excretory urographic, sonographic, and laparoscopic findings. *AJR Am J Roentgenol* 1993 Feb; 160(2): 331-4[Medline].
17. Wu MH, Hsu CC, Huang KE: Detection of congenital mullerian duct anomalies using three-dimensional ultrasound. *J Clin Ultrasound* 1997 Nov-Dec; 25(9): 487-92[Medline].

**GYNECOLOGICAL CASE 6****RECURRENT HIGH TRANSVERSE VAGINAL SEPTUM SURGICALLY CORRECTED**

NAME: L. N. IP NO: 1036609  
AGE: 20YEARS D.O.A 14/8/06  
PARITY: 0+0 D.O.D 29/8/06

**Presenting complains:**

One year history of lower abdominal pain and lower abdominal swelling.

**History of presenting illness:**

She had her menarche at 16 years. She received 3 cycles of menstrual flow that was scanty then ceased. This was followed by cyclical lower abdominal pains and progressive enlarging of the lower abdomen and Amenorrhoea. After 3 years of that she sought medical help and a diagnosis of Cryptomenorhea was made. In 2005; excision of a high vaginal septum was done in KNH; where old blood was drained. However she didn't receive periods after surgery; scheduled dilation were not done. Complain of lower abdominal mass and cyclical lower abdominal pains just as before surgery. Sexually active-dysperunia: no penetration.

**Past medical history**

This was non contributory.

**Family social history:**

She is Single; last born in a family of 6; stays with brother in Kikuyu. No history of chronic illness in the family. Neither smokes nor drinks alcohol. L.N schooled up to Form Four.

**Physical examination:**

She was a young girl in good general and nutritional condition with round female stature. She had a normal feminine hair distribution, breast development of thelarche 4.

She was afebrile with pulse and blood pressure of 74 beats /minute and 100/60 respectively.

She was not pale.

Respiratory; cardiovascular and central nervous system was essentially normal.

### **Abdominal examination:**

Abdominal examination revealed a round moderately cystic pelvic mass equivalent to a 16 week uterine size. There was a moderate tenderness and deep palpitation on the mass; however no obvious guarding or rebound tenderness was elicited.

### **Vaginal examination**

She had a normal external genitalia. Vagina ending high about (4 cm deep) Thick fibrous vaginal septum felt. The diaphragm of the pouch's roof was tense and not bulging. No significant tenderness elicited.

**Diagnosis:** Recurrent transverse vaginal septum

### **Investigation:**

- a) Pelvic u/s: Showed a pelvic cystic mass filled with fluid measuring  $12 \times 9$  cm suggestive of Haematocolpos and haematometria. Ovaries were normal.
- b) Haemogram
  1. WBC= $7.0 \times 10^9/C$
  2. Hb=12.0 g/dl Placel:- $216 \times 109$
- c) Blood group- O+ve
- d) d). Urea/Electrolyte
  - i. Na+ 135mol/l
  - ii. K+ 3.9mol/l
  - iii. BUN 1.9  $\mu$ mol/l

Intravenous urography was not done.

### **MANAGEMENT**

The patient was explained to the diagnosis and mode of management. Consent for operation (excursion of high transverse vaginal septum) was sought and obtained. She was starved overnight and premeditation with intramuscular atropine 0.6 mg before being wheeled to the theatre.

In theatre: She was given general anaesthesia; In lithotomy position, vulval-perineal toilet was done and the perineum draped with sterile towels. The bladder catheterization was done and clear urine drained. EUA (examination under anaesthesia was done)

**Finding:** Vagina about 5 cm deep: with a thick fibrous transverse vaginal septum.

**Done:** Excision of high transverse septum

Uterus pushed from above (supra-pubic area). Then a cruciate incision was made in the fibrous diaphragm of the septum. Successfully penetrated to the area with Haematocolpos. Drained 300 ml of very thick chocolate colored old blood. Dilation was done serially until Hegars number 14. Masipulization of the edges done. Haemostasis was archived. Vaginal pack left insitu (covered with suffra-tulle). The patient was successfully reversed from anaesthesia.

#### **Post operative care.**

She was taken to recovery ward and observed closely till fully awoken, and then transferred to the wards. Where ½ hrly observations was made for 4 hrs; and henceforth 4 hourly observations. She was put on Augmentin and Bruffen. Initial post-operative day needed strong pain relief-was on Pethidine. The vaginal pack was removed on the 3rd post OP day. Patient remained stable and she was discharged home on the 5th post-operative day. She was advised to come again to the clinic gynecology out patient clinic in two weeks time.

Serial vaginal dilation using a mould was thought not useful because the septum was fairly high.

#### **Follow-up**

She has been seen once in the GOP and had no complained. No abdominal mass was noted. The vaginal was admitting one finger easily. Expected to have menstrual bleeding at least 35 days. She was given a return date in 4 weeks time.

#### **DISCUSSION.**

Vaginal septum occurs in many different forms. They are commonly situated above the hymen and are often mistakenly described as imperforate hymens. It may be relatively thin and less than 1 cm or may be thick and involve the entire vagina reaching the cervix (1). Transverse septa are a result of developmental defect in vaginal embryogenesis that leads to an incomplete fusion between the Mullerian duct and the urogenital sinus component of the vagina.

There is faulty fusion or canalization of the urogenital sinus and the Mullerian duct (1, 2).

Septum located in the upper vagina are likely to be patent; whereas those located in the lower part of the vagina are more often complete. That's why they are confused with imperforate hymen (4). Our patient could have had a septum with some patency: cribriform perforation; but later sealed 3 cycles after menarche.

Septum consist of the fibromuscular membrane cover always on the lower surface can be covered by a glandular column epithelial; what is likely to be transformed into squamous epithelial by the metaplastic process after correction of the obstruction.

Transverse vaginal septum is associated with fewer urologic and other anomalies compared to congenital absence of the Mullerian duct (1, 2, 3).

Our patient had a high transverse vaginal septum that was partially patent initially. Ultrasound examination done did not reveal any other anomalies with the urogenital system; and IVU was not done.

The incidence congenital defects of the female reproductive tract is not known. Some 75% of the defects are asymptomatic. Incidence of transverse vaginal septum vary greatly from 1:21000 to 1:7200 and 1:30000 to 1:8000(2;5). Transverse vaginal septum is less common than congenital absence of vagina and uterus(4). It has been diagnosed in new-borns; infants and older adolescent girls(4)

Our patient was first diagnosed with vaginal septum at 17 yrs (adolescent)

Aetiopathogenesis of the utero genital examination may be the result of genetic error or by exposure to teratogens during embryogenic development. Transverse vaginal septum is thought to result from female sex linked autosomal recessive transmission. Partial transverse vaginal septa have been reported in diethyl-stillbestrol (DES) exposed females.(8)

Undiscovered imperforate transverse septa may lead to the formation of a large Mucocolypos in infancy. Mucocolypos result from glandular endocervical and upper septal surface mullerian epithelium secretion stimulated by the placental transfer of oestrogen. Symptoms may only develop in puberty; when accumulation of menstrual blood in the vagina with a complete septum results in great vaginal distress (Haematocolpos) and canal dilation.

This leads to accumulation of blood in the uterus (haematometria) and may be extended to the tubes. (Haematosalphynx or spill to peritoneal cavity) Endometriosis and vagina adenomyosis are known but not inevitable complication(2, 5, 6). Common presenting symptoms are cyclical lower abdominal pains; backache; pelvic discomfort and lack of visible menstrual discharge associated

with progressively enlarging pelvic mass in a fashion similar to that seen in imperforate hymen. (except that there is no bulging of the introitus)

Urinary complaints are occasionally encountered though difficulty in micturition; due to the distended vagina compressing on the urethra resulting into urethral compression and preventing of bladder emptying (1, 3, 4).

Our patient had cyclical pain and lower abdominal mass but had no urinary symptoms. She may have had more of haematometria than Haematocolpos due to the high vaginal septum. Vagina ended in the upper 2/3 as a blind pouch. The roof was thick and fibrous - this may have been as a result of the previous surgery (7, 8, 9).

If the diagnosis of a complete septum is established prior to menarche; it should be incised creating a channel for drainage. Excision is easy only if the upper vagina is distended (bulging). Otherwise it is quite dangerous performing intra-vaginal surgery on immature structure (when done in childhood); it is best to limit the procedure to just establish drainage (2, 4).

Surgical correction should be done when the patient is deemed to be sexually active or if there is obstruction of the flow of menstrual blood.

This was done by clamping the septum at the centre and then cutting through the septum circumferentially close to the vaginal wall. The vaginal mucosa above and below the septum end is then undermined for about 1 cm. Upper and lower mucosa are then repaired together by interrupted suture 3/0 vicryl. Vagina is then packed with an antiseptic gauze with lubricant i.e. suffra-tulle. This is due to the colonization of the vagina by protection bacteria which ferment glycogen to lactic acid hence making the pH acid (10)

Haematocolpos may be converted to Pyocolpos when needles are placed in it without completely damaging it.

This patient had cruciate incision successfully made on the septum then the edges were circumferentially excised then marsupialization of the edges done. She was then referred in the GOPC for 2 weekly digital exam and possible dilatation because the use of a mould seemed not productive for such a high vaginal septum.

## REFERENCES

1. Jennifer Blake: Paediatrics and gynaecology in essential of obstetric and gynaecology 3rd edition Chap 33; pp 381
2. Lisbeth Chang and David Mwan: Paediatrics and Adolescents gynaecology In Currents Obstetrics and Gynaecology diagnosis and treatment. 9th Edition chapter 31; pp602-603
3. Howkins and Stallworth: Bonkeys gynaecology Surgery 8th Edition chap 6 pp 91-92
4. John A Rocky. Surgeries for the anomalies of the mullerian tract. In Telindes Operative Gynaecology. 8th Edition chap 31; 704-707
5. Joan M Bengton; The vagina In Kistnens Gynaecology principals and practice 6th Edition chap 5 pp 83-84
6. Wendy J, Schezer and Howard Mc Clamrock; Amenorrhoea in. Novak's Gynaecology 12th Edition chapter 24; pp 820
7. Donna Shoupe. Management of utero-vaginal anomalies in the management of common problem in Obstetric and gynaecology 3rd Edition chap 107
8. Arther l Herbst, Daniel R, Mishel JR et al comprehensive gynaecology 2nd edition chap 9 pp 261-262
9. Mattingly RF, Thompson J D. Surgical condition of the vagina and the urethra. In Telindes Operatives Gynaecology 8th Edition Lippincot-Raven Philadelphian 1997 pp911-13
10. Tindal VR. Malformation and Maldevelopment of the genital tract in Jeff coats principles of gynaecolgy5th Edition Butterworth Heinemann, New Delhi India 146, 1987.

**Gynecological case 7****HIGH RISK CHORIOCARCINOMA-REMISSION FOLLOWING CHEMOTHERAPY**

NAME: E.G                      IPNO: 1083132

AGE: 34 YEARS                DOA: 16/03/06

PARITY: PARA 1 +1         DOD: 26/09/06

**Presenting complaints**

On and off vaginal bleeding for 5 months

**History of presenting illness**

The patient was referred from Meru District Hospital for evaluation and treatment. She was well until November 2005 when she developed PV bleeding. It was mainly spotting and was associated with right iliac fossa pain. A pregnancy test was done and was positive. Emergency laparotomy was done for? ectopic pregnancy. There were no significant findings intraoperatively. She had an episode of bleeding in December 2005. Pregnancy test was positive and pelvic ultrasound showed complete abortion. BhCG done in February 2006 was elevated hence referred for chemotherapy. There was no history of chest pain, no shortness of breath and no headache.

**Obstetric and gynaecological history**

She was Para 1+1 with 1 living child. Her past delivery was in 2001 by caesarean section due to cephalopelvic disproportion. She had a Hydatidiform mole in 1998. Suction evacuation was done at Chogoria Mission Hospital. Follow up for 1 year at the same hospital was uneventful and she was allowed to conceive.

Her last normal menstrual cycle was in October 2005. Prior to this, her periods were regular, of 4 days duration and with a 28 day cycle. She had an IUCD from 2002 to 2005. She had used combined oral contraceptives for one year during the follow up period for H. mole. She has never had a pap smear done.

**Past medical history**

She is a known asthmatic since 1990 and is on Salbutamol inhaler. She was transfused 2 pints of blood in 1998

**Family and social history**

She was a single lady living in Meru. She was a Primary school teacher. She did not smoke and did not take alcohol. There was no family history of chronic illnesses.

**Physical examination****General examination**

She was in fair general condition. She was not pale and the sclera was non icteric. She had no oedema or lymphadenopathy.

Her vital signs were: BP-130/80mmHg, Pulse rate 78 beats per minute, Temperature 39.4 and respiratory rate was 16 /minute. Her weight was 50 Kg and her body surface area was 1M<sup>2</sup>.

**Abdominal examination**

The abdomen was scaphoid. It was moving with respiration. There was a Pfannestiel scar. There were no palpable masses.

**Pelvic examination**

She had normal external genitalia. On speculum examination, the vaginal walls were normal. The cervix was long, closed and posterior. The uterus was bulky and corresponding to 10 weeks gestation. The adnexa and pouch of Douglas were normal. There was normal per vaginal discharge on the examining finger.

Respiratory, cardiovascular and central nervous systems; were essentially normal. She was not in respiratory distress and the breath sounds were vesicular in both lung fields.

**Impression**

An impression of choriocarcinoma was made.

**Investigations**

□hCG: 19,002 mIU/mL  
 Hemogram: Hb 12g/dl  
           WBC- $8.2 \times 10^9/L$   
           Platelets- $348 \times 10^9/L$   
 Blood group: O positive  
 Urea and electrolytes: Normal  
 Liver function Tests: Normal  
 Pelvic ultra sound: Enlarged right adnexa with multiple cystic areas suggestive of  
                           Gestational Trophoblastic neoplasia. The uterus was reported to be  
                           normal.

**MANAGEMENT**

She was categorized as non metastatic gestational trophoblastic disease and started on single agent therapy; methotrexate 50mg once daily on days 1, 3, 5 and 7 with leucovorin(folinic acid) 15mg once daily on alternate days. She had 5 courses of methotrexate and the B-hCG levels remained persistently high (639.89mIU/mL). She was recategorised as high risk and started on EMA-CO (etoposide, methotrexate, dactinomycin, cyclophosphamide and vincristine). Etoposide 100mg iv infusion was given on day 1 and 2, methotrexate 300mg IV infusion was given on day 1, Actinomycin D 0.5mg IV was given on day 1 and 2. Folinic acid was given on day 2-5. Vincristine 1 mg IV and cyclophosphamide 600mg IV infusion were given on day 8.

In between the courses she was being allowed home and readmitted after 7 days for subsequent course. Before every course, the liver function tests, full blood count and renal function tests were done to monitor the side effects of chemotherapy. All the parameters remained normal.

**B-hCG levels and treatment chart**

17.03.06	19002 miu/ml	Methotrexate
4.04.06	11231.43miu/ml	Methotrexate
2.05.06	463.67 miu/m	Methotrexate
21.05.06	436.15miu/ml	Methotrexate
20.06.06	639.89 miu/ml	Methotrexate
3/07/06	1111.20 miu/ml	EMA-CO

18/07/06	226.28 miu/ml	EMA-CO
02/08/06	11.62 miu/ml	EMA-CO
17/08/06	1.35 miu/ml	EMA-CO
31/08/06	0.13 miu/ml	EMA-CO
15/09/06	0.86 miu/ml	EMA-CO

### Follow up

She was advised on follow up and the need to avoid pregnancy for the next 2 years. She was put on combined oral contraceptives and was to be followed up monthly in the GOPC for 2 years. As this commentary was being compiled, the patient was to begin follow up.

### DISCUSSION

The patient presented was a 34 year old Para 1+1 with choriocarcinoma managed initially with methotrexate alone, developed resistance after 5 courses of methotrexate and then changed to EMA-CO where remission was achieved.

Gestational trophoblastic neoplasms (GTN) include the tumour spectrum of hydatidiform mole (complete and partial), invasive mole (chorioadenoma destruens), placental-site trophoblastic tumour (PSTT), and choriocarcinoma (1, 2). They arise from fetal tissue within the maternal host and are composed of both syncytiotrophoblastic and cytotrophoblastic cells. In addition to being the first and only disseminated solid tumours that have proved to be highly curable by chemotherapy, they elaborate a unique and characteristic tumour marker, human chorionic gonadotrophin (hCG) (1, 2).

Hydatidiform mole is the most common of the trophoblastic neoplasms and develops in 1:1500 pregnancies in USA, 1:125 pregnancies in Taiwan (1). Invasive mole comprises about 15% of all GTN. Choriocarcinoma is reported in 2-5% of all cases of gestational neoplasia while the incidence of placental site trophoblastic tumor is about 1%. The incidence of Hydatidiform mole at KNH 1998-2001 was 1 in 250 deliveries and that of choriocarcinoma in the same period was 1 in 544 deliveries (3). In the same study H.Mole accounted for 0.5% of admissions to the emergency gynaecology ward and choriocarcinoma accounting for 2.5% of all admissions to the cold gynaecology ward (3).

Risk factors associated with GTDs include age less than 20 and over 40 years, low socioeconomic status, diets deficient in protein, folic acid and carotenes, blood group A women with a group O

spouse increases the risk by 10 times. Blood group AB and B have poor prognosis (1). Our patient was 34 years old, her blood group was o+ve and she did not have any known risk factors.

Choriocarcinoma is a pure epithelial neoplasm, highly malignant tumor, comprising both neoplastic syncytiotrophoblastic and cytotrophoblastic elements without chorionic villi. Early systemic haematogenous metastasis tends to develop in gestational choriocarcinoma (1, 2, 4). It invades the myometrium and metastasizes locally to the broad ligament or by blood to the lungs, brain, liver, and vagina. The most common sites of metastases are lung (80%), vagina (30%), pelvis (20%), liver (10%), and brain (10%) (2, 4). There was no obvious metastasis in our patient.

Approximately 50% of choriocarcinoma occur after molar pregnancy, 25% after abortion and ectopic pregnancy and 25 % after normal pregnancy. About 3-5% of all patients with molar pregnancy develop choriocarcinoma (4). Juma in her review of GTDs at KNH 1998-2001 found that the commonest pregnancy preceding choriocarcinoma was hydatidiform mole (53%), followed by abortion (31%) while the rest were mostly undetermined (3). The antecedent pregnancy in this patient was a term pregnancy.

Locally invasive gestational trophoblastic tumour develops in about 15% of patients after molar evacuation and infrequently after other gestations<sup>2</sup>.

The usual type is the localized nodular type (often gives negative diagnosis on uterine curettage) and diffuse type involving entire endometrium (4).

Clinical features depend on the location of primary growth and on its secondary deposits. There is usually a history of molar pregnancy in the past or any other gestational event. GTN after a non molar pregnancy is always a choriocarcinoma as was the case in this patient (4). These patients are usually seen clinically with irregular vaginal bleeding at times brisk or with continued amenorrhoeae. Other symptoms due to metastatic lesions may include: cough, breathlessness, haemoptysis, vaginal bleeding, headache, convulsion, paralysis, coma, epigastric pain and jaundice (1, 4). Our patient presented with vaginal bleeding.

On examination the patient may look ill and may have pallor. Signs related to organ of metastasis may be present. On speculum examination there may be a purplish red nodule in the lower third of the anterior vaginal wall. Bimanual examination may reveal unilateral or bilateral ovarian enlargement

due to theca lutein cysts, uterine sub-involution or asymmetric enlargement (1, 4). The trophoblastic tumour may perforate through the myometrium, causing intraperitoneal bleeding, or erode into the uterine vessels causing vaginal haemorrhage; bulky necrotic tumour may involve the uterine wall and serve as a nidus for infection. Patients with uterine sepsis have a purulent vaginal discharge and acute pelvic pain (2). Examination in our patient revealed a bulky uterus.

Once the diagnosis of malignant trophoblastic disease has been established a chest X-ray and scans of the brain and liver should be obtained. CT scan is the diagnostic procedure of choice for brain, lung, liver, and renal metastasis (1, 2, 4). Pelvic ultrasound may help to localize the lesion and to differentiate gestational trophoblastic neoplasm with normal pregnancy. Metastatic brain lesion is suspected when the ratio of HCG in spinal fluid/in serum is more than 1:60. Diagnostic uterine curettage is not recommended as may trigger brisk haemorrhage and its value in diagnosis is questionable (1, 2, 4). No biopsy should be taken of a vaginal lesion unless choriocarcinoma has been ruled out by B-HCG because vaginal metastasis may bleed profusely because they are highly vascular and friable (2). Chest X-ray done in our patient was normal.

Malignant gestational trophoblastic tumour is categorized into non-metastatic or metastatic disease. Non-metastatic disease is confined to the uterus while metastatic disease has extrauterine metastases. Metastasis is usually associated with choriocarcinoma, which has a tendency towards early vascular invasion with widespread dissemination (1, 2, 4). Metastatic GTD is further categorized into good and poor prognosis disease. Good prognosis metastatic disease has a short duration from the antecedent pregnancy of less than 4 months, has initial serum  $\beta$ HGG levels less than 40,000 mIU/ml, metastasis limited to lung and vagina (no metastases to the brain or liver); and has no prior chemotherapy. In poor prognosis metastatic disease there is a long duration of disease (>4 months) from the antecedent pregnancy, has high levels of serum  $\beta$ HGG above 40,000 MIU/ml, has metastases to brain or liver, has had previous unsuccessful chemotherapy or follow a term pregnancy (1, 2, 4). The anatomic staging for gestational trophoblastic disease as described by

International Federation of Gynaecology and Obstetrics (FIGO) is as follows: (1, 2, 4).

### *Stage*

Stage I	Confined to the uterus
Stage II	Limited to the genital structures i.e. vagina and pelvis or both.
Stage III	Lung metastases
Stage IV	Other metastases such as brain, liver or gastrointestinal tract.

### *Sub stage*

A	No risk factor
B	One risk factor
C	Two risk factors

### *Risk factors*

1.  $\beta$ hCG >100,000 mIU/ml
2. Duration from termination of antecedent pregnancy to diagnosis >6 months.

The drawbacks of anatomic staging are: the lesion cannot be located precisely, it does not help in treatment and prognosis is poorly correlated (4). Our patient was stage I B.

In addition to the anatomic staging, it is important to consider other variables to predict the likelihood of drug resistance and to assist in selecting appropriate chemotherapy (2). A prognostic scoring system proposed by the World Health Organization reliably predicts the potential for resistance to chemotherapy. When the prognostic score is higher than 7, the patient is categorized as high risk and requires intensive combination chemotherapy to achieve remission. Patients with stage I disease usually have a low risk-score, and those with stage IV disease have a high-risk score. The distinction between low and high risk applies mainly to patients with stage II or III disease.<sup>1</sup>

### W.H.O. Prognostic Scoring System for GTD

Parameter	0	1	2	4
Age (years)	<39	>39		
Antecedent pregnancy	Mole	Abortion	Term	
Interval between end of antecedent pregnancy and start of chemotherapy (months)	<4	4-6	7-12	>12
$\beta$ hCG (mIU/ml)	<10 <sup>3</sup>	10 <sup>3</sup> - 10 <sup>4</sup>	10 <sup>4</sup> - 10 <sup>5</sup>	>10 <sup>5</sup>
ABO groups	-	O or A	B or AB	-
Largest tumour including uterine (cm)	<3	3-5	>5	-
Site of metastasis	-	Spleen, kidney	GIT, Liver	Brain
8 Number of metastasis	-	1-3	4-8	>8
9 Prior Chemotherapy	-	-	1 drug	□2 drugs

The total score is obtained by adding the individual scores for each prognostic factor: <4 low risk, 5-7, middle risk; □8, high risk (1).

Chemotherapy is now the mainstay in the treatment of gestational neoplasia. The need for chemotherapy following a complete mole is 15% and 0.5% after a partial mole (5). Patients with malignant, non-metastatic disease and metastatic low risk GTN are treated with single agent chemotherapy. Methotrexate is considered the drug of choice. However, Actinomycin D can be used in a patient with poor liver function. During treatment, the serum  $\beta$ -HCG titres are monitored weekly. One additional course of chemotherapy is administered after a normal serum HCG titre. After 3-4 normal HCG titres, the titres are done monthly for 1 year.

A switch from methotrexate to Actinomycin is made if the patient receiving methotrexate for non-metastatic or metastatic low-risk GTN develops rising or plateauing serum HCG titres or develops severe methotrexate induced mucositis (1, 2). Though our patient was classified as low risk, she developed resistant to chemotherapy manifested by rising □hCG necessitating use of combination chemotherapy.

Prior treatments for poor prognosis/high risk gestational trophoblastic disease have included combination therapies such as MAC (Methotrexate, Actinomycin D, and Chlorambucil or cyclophosphamide) and EMA-CO. Currently EMA/CO (Etoposide, methotrexate, Actinomycin D,

Cyclophosphamide and vincristine (oncovin)) chemotherapy provides the best response rate (approximately 80%) with the lowest side effect profile hence is the preferred primary treatment in patients with metastasis and a high risk prognostic score (1). MAC is reported to provide a response rate of about 50%. If patients develop resistance to EMA-CO, they may then be treated successfully by substituting etoposide and cisplatin on day 8(EMA-EP). Treatment of malignant trophoblastic disease must be continued with repeated courses of combination chemotherapy until HCG titres have returned to non detectable levels. It is recommended that all high risk patients receive at least two courses of combination chemotherapy after HCG titres have returned to normal to reduce the risk of relapse(3 consecutive negative weekly B HCG). After remission is achieved, follow-up is as for non-metastatic or good prognosis disease (1, 2, 4). Our patient achieved remission after 3 courses of EMA-CO and was given an additional 3 courses after the negative result.

Indications of surgery include:-lesions confined to the uterus in women aged above 35 years to improve efficacy of chemotherapy, localized uterine lesion or metastatic lesion unresponsive to chemotherapy, intractable bleeding, accidental uterine perforation during uterine curettage and placental site trophoblastic tumor which is unresponsive to chemo therapy. Total hysterectomy is enough. Lung resection in pulmonary metastasis in drug resistant cases. Hepatic resection may also be required to control acute bleeding or to excise a focus of resistant tumor. Craniotomy may be required to provide acute decompression or to control bleeding. Cerebral metastasis that are resistant to chemotherapy may be amenable to local resection.(2, 4).

Role of radiotherapy is limited to patients with brain metastasis which require whole brain radiation of 3000cGY in 10 fractions along with high dose methotrexate and in liver metastasis along with chemotherapy (2, 4).

During the period of follow-up care, patients with GTN should use combined oral contraceptives after HCG levels have reverted to normal. The serum hCG titres are critical in monitoring the status of the disease, and a normal intrauterine pregnancy interferes with this critical monitoring tool (5). .Women who have undergone chemotherapy are advised not to conceive for one year after completion of treatment while those who never required chemotherapy can conceive if B-HCG have been normal for six months (5).

Follow up for patient with placental site trophoblastic tumor after hysterectomy is by serial serum HPL (5).

The prognosis for non-metastatic GTN is excellent with cure rate with chemotherapy of close to 100%. Metastatic low risk GTN has a cure rate with chemotherapy of close to 100%. Metastatic high risk GTN has a cure rate with chemotherapy of approximately 75 % (12, 4).

There is no adverse effect on the subsequent pregnancy provided the conception occurs after 1 year after completion of chemotherapy. The damaged ova are likely to be wasted by this time (2, 4).

Prevention strategies for gestational trophoblastic neoplasm include (4):

- Prophylactic chemotherapy in at risk women following evacuation of molar pregnancy. Those at risk are those women aged more than 35 years, initial levels of HCG in urine is >100,000 IU/24 hour urine, histologically diagnosed as infiltrative mole and previous history of molar pregnancy.
- Meticulous follow up following evacuation of hydatidiform mole for at least 1 year to detect early evidence s of trophoblastic reactivation.
- Selective hysterectomy in hydatidiform mole in patients over 35 years reduces the risk of choriocarcinoma by 4 fold.

REFERENCES

1. Markusen TE, O'Quinn A.G. Gestational Trophoblastic Diseases In: DeCherney AH, Nathan L. (Eds). Current Obstetric and Gynecologic Diagnosis and Treatment. 9th edition, McGraw Hill, 2003, Pg 947 - 958.
2. Berkowitz R.S. Goldstein D.P. Gestational Trophoblastic Diseases In. Berek J.S. (Ed) Novak's Gynaecology, 13th edition, Lippincott Williams and Wilkins, Philadelphia, 2002:1353-1371.
3. Juma EA, et al. Gestational trophoblastic diseases at the Kenyatta national Hospital. JOGEGA .Vol17No1:46-56-80
4. Dutta DC: Gestational Trophoblastic Neoplasia. In: Textbook of Gynecology, 6th edition, 2004, New Central Book Agency (P) Ltd, Calcutta, India Pg334 – 340.
5. Royal College of Obstetricians and Gynaecology. The management of Gestational Trophoblastic Neoplasia. guideline No.38. february 2004.

**Gynecology case 8****POLYCYSTIC OVARIAN DISEASE (PCOD) – LAPAROSCOPIC OVARIAN DRILLING:**

NAME: A.N	IPNO: 1110562
AGE: 38 YEARS	DOA: 9/10/06
PARITY: 0+0	DOD: 16/10/06

**Presenting Complaint**

She was admitted with a history of failure to conceive for 10 years and irregular menses for 3 years.

**History of Presenting Complaint**

She had been followed up in the gynaecology outpatient clinic where she presented with a history of failure to conceive for 10 years despite regular unprotected coitus. There was history of irregular heavy and prolonged menses alternating with periods of amenorrhoea. She did not have increase in her facial hair neither was there any change in weight. There was no history of a vaginal discharge or change in bowel and urinary habits. A pelvic ultrasound was done and revealed polycystic ovaries. She had been put on clomiphene citrate for ovulation induction for 3 months while on follow up in the clinic without conception.

**Obstetrics and Gynecology History**

She was a para 0+0. Her menarche was at 14 years. Prior to the onset of the illness; her cycle was 30 days long, duration of 4 days, and it was regular. She had used oral contraceptive pills in 1996 for about one year.

**Family and Social History**

She was married and lived with the husband. She did not smoke or take alcohol. There was no family history of chronic illness.

**Past Medical History**

Not significant

**PHYSICAL EXAMINATION**

She was in fair general condition, afebrile, had no palor, lymphadenopathy, jaundice, oedema or cyanosis. Her weight was 58 kg.

## MANAGEMENT

She was admitted to the cold gynecological ward for diagnostic laparoscopic ovarian drilling. The diagnosis and mode of management were explained to the patient and an informed consent obtained. Enema was given the night before surgery she and was kept nil per oral overnight. She was premedicated with Atropine 0.6mg intramuscularly half an hour before being wheeled to theatre.

### Operative management

In theatre, she was put under general anaesthesia and placed in lithotomy position. Vulvovaginal toilet and aseptic catheterization was done. Examination under anaesthesia confirmed earlier findings. A uterine elevator was inserted.

The abdomen was cleaned and draped. A stab incision was made at the umbilical region with a blade and carbon-dioxide introduced via a veress needle to create a pneumoperitoneum. After introduction of the telescope at the primary port, two more secondary ports were created to allow clamps, dissecting forceps and diathermy drills.

### Findings

The uterus and both tubes appeared normal. The right ovaries was enlarged and polycystic. The left ovary was however atrophic. There were no adhesions.

### Procedure

Drilling of each ovary was done using unipolar diathermy with about 10 – 15 punctures on each. Chromopertubation was done and demonstrated free spill bilaterally. The port entries were closed using absorbable sutures. The patient was then reversed from general anaesthesia and the specimen sent for histology.

### Post Operative Care

The vital signs were monitored ½ hourly until she was fully conscious and stable and then 4 hourly thereafter. She was started on intravenous fluids normal saline alternating with 5% Dextrose, 1 litre 8 hourly, crystalline penicillin 2 mega units 6 hourly and Gentamicin 80mg 8 hourly intravenously for 24 hours then Ampiclox 500mg 6 hourly for 5 days.. For analgesia she was given pethidine 100mg every 6hours and diclofenac 75mg every 12hours intramuscularly for 24 hours. On the first post-

She had a pulse rate of 72 beats per minute, a respiratory rate of 18 per minute, a blood pressure of 110/70 mmHg and a temperature of 36.0°C.

**Systems:**

Respiratory, Cardiovascular, and Central Nervous systems were essentially normal.

**Breasts:**

The breasts were normal and there was no galactorrhoea.

**Abdominal Examination**

The abdomen was not distended, was soft, not tender and no masses were palpable.

**Pelvic Examination**

The external genitalia were normal; the cervix was firm, smooth and closed. The uterus was normal in size and anteverted. The adnexa felt full bilaterally but not tender and the Pouch of Douglas was free. There was no discharge on the examining finger.

**INVESTIGATIONS**

- Pelvic ultrasound** - the uterus was normal in size and echogenicity. The endometrial thickness was 12mm. Both ovaries were enlarged with multiple cysts. The right ovary measured 4.3cm x 3.0cm while the left measured 3.8 by 3.0cm. There was no fluid in the pouch of Douglas.
- Hysterosalpingiogram(2000) - the uterine cavity was normal in size and shape with no filling defect. Left tube had hydrosalpinx there was some spill on that side. The left tube had limited spill with? Some terminal blockage. .
- Semen analysis – normozoospermia
- Full Heamogram: Hb 14.4g/dl; WBC 5.4 X 10<sup>9</sup>/L; PLT 382 X 10<sup>9</sup>/L
- Renal function tests: Na 140mmol; K 3.6mmol/l; BUN 2.3mmol/l Cr 89umol/l
- RBS - normal

**IMPRESSION**

An impression of polycystic ovarian syndrome with secondary infertility was made.

operative day, bowel sounds were present; she was started on oral sips graduated to light diet as tolerated. She was counselled on the intra-operative findings and what was done.

She did well post-operatively and was discharged home on the third post-operative day to be seen in the gynaecology outpatient clinic (GOPC) after two weeks.

### **Follow-Up**

She was seen in GOPC after two weeks. She had no complaints and examination findings were normal. She was given an appointment to be seen after three months.

### **DISCUSSION**

A.N was a 38 year old para 0+0 who was admitted with primary infertility due to polycystic ovarian disease. A diagnostic laparoscopy and ovarian drilling was done.

The association of amenorrhoea with bilateral polycystic ovaries was first described in 1935 by Stein and Leventhal and was known for decades as the Stein-Leventhal syndrome (1). There is disagreement on the definition of polycystic ovary syndrome (PCOS), across the two sides of the Atlantic Ocean, with the American definition having arisen from a conference held by the National Institute of Child Health in 1990 (2), where an informal survey of participants concluded that the diagnostic symptoms in order of importance were: hyperandrogenism/hyperandrogenemia, oligo-ovulation and the exclusion of other pathology. The British/European definition of polycystic ovaries (PCO) is on the basis of ultrasonic appearance (3). Whilst PCO describes the ultrasonic diagnosis, the term PCOS is used if the ultrasound appearance is combined with clinical symptoms of hyperandrogenemia such as oligomenorrhoea, hirsutism acne or seborrhoea.

The most recent consensus conference (ESHRE ASRM Consensus Conference on PCO, Rotterdam, 2003) concluded that any two out of the three parameters (morphology, hyperandrogenism—clinical or biochemical, and oligo-amenorrhoea) was sufficient to diagnose PCOS. The morphology of polycystic ovary was redefined as the presence of an ovary with 12 or more follicles measuring 2–9 mm in diameter and/or an increased ovarian volume ( $> 10 \text{ ml}^3$ ) (5).

Polycystic ovary syndrome (PCOS) is a heterogeneous disorder of unknown etiology affecting 5–10% of women of reproductive age. It is a disorder that affects the reproductive, endocrine and metabolic systems, and it is the most common cause of anovulatory infertility.

Its exact cause is unknown; however, there are several theories about its pathophysiology. In the last decade, the insulin resistance hypothesis has gathered several converts (6). It is thought that relative insulin resistance results in chronic hyperinsulinaemia which gives rise to abnormal ovarian androgen metabolism, impaired follicle growth and altered gonadotrophin response (7).

A variety of theories/systems have been implicated in the pathophysiology of PCOS. These include aberrations of the hypothalamo–pituitary–ovarian axis, intra-ovarian growth factors, fetal programming and more recently insulin resistance and its metabolic consequences including the metabolic syndrome X. The increased prevalence of symptoms of PCOS amongst family members of affected women also suggests a genetic link. However, how these systems interact to result in the PCOS phenotype is still uncertain (8). The patient presented had a normal blood glucose level and was not obese.

Insulin resistance is the main hypothesis that appears to be fast gathering pace as the single unifying hypothesis in PCOS. However, the fact that it is thought to be present in 30–60% of women with PCOS and not all of them raises more questions than it answers. The mechanisms of insulin resistance in PCOS are also uncertain. It is thought that compensatory hyperinsulinaemia augments the stimulatory action of LH on theca cell androgen biosynthesis through up-regulation of genes encoding steroidogenic enzymes (9). It has also been postulated that hyperinsulinaemia acts on the hypothalamus and pituitary leading to increase LH concentration and on the ovarian stromal cells resulting in increase free circulating androgens and a decrease in sex hormone binding globulin (SHBG) (9). Hyperinsulinaemia also leads to imbalance in the production of insulin-like growth factor binding protein-1 resulting in abnormal local steroidogenesis. Insulin resistance may also lead to direct stimulation of an ovarian protease inhibitor (plasminogen activator inhibitor-1 (PAI-1)) resulting in limitation of follicular growth.

The principal features of PCOS include menstrual irregularities, anovulatory infertility, androgen excess (hirsutism, acne and temporal balding) and obesity. About 50% of the patients have hirsutism, 40% have infertility, and 65% are obese. Menstrual irregularity (oligo- or amenorrhoea) is the most common reason for gynaecological presentation. The endometrium of oligoamenorrhoeic women with

PCOS is under continuous exposure to oestrogen until the endometrial growth outstrips its blood supply. This results in heavy and sometimes erratic bleeding as the endometrium breaks down as was the case in our patient. The exact mechanism underlying the association between infertility and PCOS remains unknown. It was thought that the sub-fertility could be due to the presence of high LH and androgen levels. It would appear that several factors interact to form part of the vicious cycle of abnormal steroidogenesis, folliculogenesis, abnormal oocyte maturation, decrease endometrial receptivity and early pregnancy loss (10). Androgen excess presents as hirsutism. The degree of hirsutism can be evaluated by using the modified Ferriman and Galway score before commencing therapy and for monitoring response. Our patient presented with menstrual irregularities and anovulatory infertility but no features of androgen excess.

The critical finding to diagnose PCO is ultrasound examination of the ovaries with the multiple peripheral small cysts. There is still debate about whether there has to be more than 10 or 12, and whether both ovaries have to be effected. There is also usually an increase in ovarian volume and change in ovarian dimensions with the ovary being more spherical (3).

Ovaries in women with PCOS are 2 to 5 times the normal size and have been called oyster ovaries because they are enlarged and sclerocystic with smooth pearl-white surfaces without indentations. Many small fluid filled follicle cysts lie beneath the thickened fibrous surface cortex. Luteinization of the theca interna is usually observed, and occasionally there is focal stromal luteinization (4).

Laboratory testing in PCOS often reveals:

- Mildly elevated serum androgen levels
- Increased LH:FSH ratio
- decrease in sex hormone binding globulin (SHBG)
- Lipid abnormalities
- Insulin resistance

Treatment depends on a patient's goals. Some require hormonal contraception whereas others require ovulation induction. Ovulation induction strategies in general include: weight reduction, medical treatment (anti-oestrogens-mainly clomifene citrate, tamoxifen and gonadotrophin therapy) and surgical treatment (11).

Clomifene citrate (50–100 mg) taken on days 2–6 of a natural or induced menstrual bleed can be used to induce ovulation. Overall, clomifene citrate is successful in about 80% of anovulatory women; however, pregnancy occurs in about half of them (11). Its effectiveness is reduced in obese women with PCOS. A therapeutic daily dose over 100 mg may cause thickening of cervical mucous and has not been found to be beneficial. Administration of clomifene citrate should be monitored using ultrasound scanning to assess follicular development in order to reduce the risk of multiple pregnancies (10–16%). The patient presented was put on clomiphene citrate as first line treatment for ovulation induction.

The insulin resistance hypothesis in PCOS has provided the rationale for the use of insulin-sensitising agents (such as metformin to improve the reproductive and metabolic abnormalities in non-diabetic women with PCOS (1). Metformin has been shown to improve insulin sensitivity, reduce LH, PAI-1 and testosterone levels (total and free) which are thought to impair folliculogenesis.

FSH and SHBG levels are also increased [12]. The UK's National Institute for Clinical Excellence (NICE) (13) recommendation is that metformin can be used as an adjunct to clomifene in women who failed to respond to clomiphene alone. Where clomifene and or metformin treatment do not result in ovulation, the second-line options include either gonadotrophins therapy or surgery. Treatment with metformin was not tried in our patient.

Laparoscopic ovarian drilling using electrodiathermy is currently an alternative second-line option in the treatment of women with PCOS who have failed to respond to medical treatment (1); this was done in our patient. It is associated with considerably lower multiple pregnancy rates compared to gonadotrophins and no case of ovarian hyperstimulation was reported in a trial by Farquhar et al. (14). The suggested mechanism of action of ovarian drilling is that the destruction of the androgen-producing stroma of the ovaries with electrocautery leads to a reduction in serum androgen levels, thereby decreasing the amount of substrate available for peripheral aromatisation to oestrogens. It has been postulated that this will restore the feedback mechanism to the hypothalamus–pituitary axis, allowing appropriate gonadotrophin stimulation for follicular development and ovulation (15). Laparoscopic ovarian drilling using diathermy is potentially associated with periovarian adhesions and theoretically could lead to premature ovarian failure due to over destruction of ovarian stroma; however, there is no clear evidence that this is the case.

Medical treatment with parenteral gonadotrophins (pure FSH or human menopausal gonadotrophins) can be employed in patients resistant to anti-oestrogens. However, gonadotrophins have been shown to be associated with an increased incidence of multiple pregnancy and ovarian hyperstimulation syndrome (OHSS) because of the recruitment of multiple follicles (11).

PCOS patients who are obese (BMI >30 kg/m<sup>2</sup>) should be encouraged to lose weight because obesity is associated with worse endocrine and biochemical features of PCOS. The low-dose combined oral contraceptive pills (COCP) can be administered to induce regular menstrual cycles. Regular endometrial withdrawal bleeding can also be induced by the administration of medroxyprogesterone acetate or dydrogesterone taken cyclically for 12 days every 1–3 months. Modalities of physical treatment of hirsutism include shaving, waxing and bleaching, electrolysis, and laser and photothermolysis. Progression of hirsutism can be reduced by anti-androgens, such as cyproterone-acetate, spironolactone, ketoconazole, dutasteride and finasteride (11).

**REFERENCES**

1. Avner H.C, Peterson M. Polycystic ovary syndrome. In Berek JS. Novak's Gynaecology, 13th edition, Lippincott Williams and Wilkins, Philadelphia, 2002: 876 - 885.
2. Zawadki JK, Dunaif A. Diagnostic criteria for polycystic ovary syndrome: towards a rational approach. In: Dunaif A, Givens JR, Haseltine F, Merrigan GR, editors. Polycystic ovary syndrome. Boston: Blackwell Scientific Publications; 1992. p. 377-84.
3. Robert Y, Ardens Y, Dewailly D. In: Kovacs GT, editor. Imaging polycystic ovaries. Cambridge: Cambridge University Press; 2000. p. 56-69.
4. Purcell K., Wheeler J.E. Benign disorders of the ovaries and oviducts. In DeCherney AH, Nathan L. Current Obstetric and Gynaecologic Diagnosis and Treatment 9<sup>th</sup> edition, McGraw Hill, 2003: 708 - 710.
5. Fauser B, Tarlatzis B, Chang J, Azziz R, Legro R, Dewailly D, et al., The Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Work-shop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic syndrome (PCOS). Hum Reprod 2004; 19:41-7.
6. Dunaif A. Insulin resistance and the polycystic ovary syndrome: mechanisms and implication for pathogenesis. Endocr Rev 1997; 18:774-800.
7. Lord J, Wilkin T. Metformin in polycystic ovary syndrome. Curr Opin Obstet Gynecol 2004; 16(6):481-6.
8. Legro RS. Polycystic ovary syndrome: current and future treatment paradigms. Am J Obstet Gynecol 1998; 179: 101-8. 2000; 73:266-9.
9. Amato P, Simpson JL. The genetics of polycystic ovary syndrome. In: Arulkumaran S, editor. Clin Obstet Gynaecol 2004; 18(5): 707-718.
10. Van der Spuy ZM, Dyer SJ. The pathogenesis of infertility and early pregnancy loss in polycystic ovary syndrome. In: Arulkumaran S, editor. Clin Obstet Gynaecol 2004; 18(5):755-771.
11. Legro RS. Polycystic ovary syndrome: current and future treatment paradigms. Am J Obstet Gynecol 1998; 179:S101-8. 2000; 73:266-9.
12. Sinha A, Atiomo W. The role of metformin in the treatment of infertile women with polycystic ovary syndrome. TOG 2004; 6:145-51.
13. NICE 2004. Infertility. Technology appraisal guidance no. 22. Available at <http://www.nice.org.uk/page.aspx?o=15724>, last accessed January 2004.

14. Farquhar CM, Williamson K, Gudex G, Johnson N, Garland J, Sadler L. A randomised controlled trial of laparoscopic ovarian diathermy versus gonadotrophin therapy for women for women with clomiphene resistant polycystic ovarian syndrome. *Fertil Steril* 2001; 78(2): 404–11.
15. Felemban A, Tan SL, Tulandi T. Laparoscopic treatment of polycystic ovaries with insulated needle cauterly: a reappraisal. *Fertil Steril* 2000; 73:266–9.



### Physical Examination

Her general condition was poor. She was sick looking, dehydrated, mildly pale and afebrile. She had a blood pressure of 100/50mmHg, a pulse rate of 100 per minute, a respiratory rate of 26 per minute and a temperature of 36.8° C.

### Respiratory and the Cardiovascular Systems

She had rapid shallow respirations but no crepitations or rhonchi. She had tachycardia, the first and second heart sounds were normal and there no murmurs.

### Abdominal Examination

The abdomen was uniformly distended and moved slightly with respirations. She had generalized marked abdominal tenderness with guarding and also had a full bladder. Paracentesis yielded frank pus. The bowel sounds were very sluggish.

### Pelvic Examination

She had normal external genitalia; the cervix was closed and irregular on the left from the 2' o'clock positions. There was positive cervical excitation tenderness bilaterally. The pouch of Douglas was full and the adnexa were tender bilaterally.

### Investigations:

1. Abdominal Ultrasound Scan – The uterus was bulky and non-gravid. There was gas in the endometrial cavity suggestive of endometritis. There was thick free fluid in the abdominal cavity that also surround the uterus. The adnexa and pouch of Douglas had features of fluid collection.
2. Haemogram
 

WBC	12.5 x 10 <sup>9</sup> /l
RBC	2.22 x 10 <sup>12</sup> /l
Hb	7.0g/dl
Platelets	197 x 10 <sup>9</sup> /l
3. Culture and sensitivity of pus – mixed growth of Enterococcus species and E. coli sensitive to Augmentin and cefuroxime (Zinacef).
4. Urea, electrolytes and creatinine
 

Na+	119 mmol/l
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K+	4.3 mmol/l
Urea	18.9 mmol/l
Creatinine	126 $\mu$ mol/l

## DIAGNOSIS

Post-Abortal Peritonitis to rule out perforated uterus.

## Management

BMW was admitted in the acute gynecological wards for rehydration and antibiotics prior to surgery. Zinacef 1.5g eight (8) hourly, and metronidazole 500mg 8 hourly and intramuscular diclofenac for analgesia. The patient was explained to the nature of her illness and the need for an emergency laparotomy. Blood was taken for grouping and cross matching and haemogram and urea and electrolytes. She was then prepared for laparotomy.

## Operative Management

In theatre, she was put under general anaesthesia and aseptically catheterized examination under anaesthesia found normal external genitalia; the cervix was closed and rugged on the left from 2' o'clock to 5' o'clock positions. The pouch of Douglas was full. There was no vaginal bleeding or discharge noted.

She was then positioned in supine position and the abdomen cleaned and draped with sterile towels. It was open through a midline subumbilical incision. There was copious amount of pus in the whole abdomen which was suctioned. There was faecal matter oozing from a perforation on the ileum. On further exploration a large rugged perforation on the posterior aspect of the uterus was noted. The uterus was empty.

A surgical registrar was consulted who came and explored the gut and performed repair of the perforation on the ileum. The edges of the uterine perforation were freshened and repaired. The abdomen was thoroughly rinsed with normal saline and Rifocin and two drains left insitu.

The abdomen was closed by mass closure using nylon. Interrupted stitches with nylon were used on the skin. She was reversed successfully from anaesthesia. She continued on the same antibiotics that she had been started on.

When further inquiries were made postoperatively she confirmed a back street abortion had been procured in a clinic at the Mathare slums.

On the third postoperative day, faecal matter was noted to be oozing from the drains. The surgical team was consulted who noted that she had developed a high output faecal fistula. She was transferred to the surgical ward where she developed wound sepsis and a burst abdomen and was planned for exploratory laparotomy on 10.12.04. However her condition continued to deteriorate with anaemia and septicaemia. She eventually died on 14.12.04 before she could be taken back to theatre for exploration.

## **DISCUSSION**

A case is presented of a 23 year old single woman who had an unsafe induced abortion in a back street clinic sustaining perforations to the uterus and the ileum. She presented with peritonitis and had laparotomy with repair of the perforations but eventually succumbed to the complications.

Unsafe abortions and their complications are a major cause of maternal mortality (1). In Kenya induced abortions are illegal unless performed when the life of the mother is in danger (2).

The exact incidence of abortions is unknown because only those that complicate end up in the hospital. In most developing countries the incidence has continued to rise posing a public health problem.

Illegal abortion are carried out in all countries (1). It is thought that up to 80% of patients admitted at Kenyatta National Hospital (KNH) with an abortion are induced (3). The number of patient admitted with abortion both in Kenya and other African countries is high and up to 60% of acute gynaecological emergency admissions are abortion related (2, 3, 4). With the introduction of MVA most of this patients don't need admission they are managed and observed in casualty setting and allowed home the same day , unless for any complications. The majority of illegal abortions are induced outside hospitals. In a study in rural Kenya, it was found that 32% are induced in hospitals, 65% are self induced at home, and 1% is in a n office and 1% in the bush or shamba ( 7). The patients presented had an abortion in a back street clinic.

Spontaneous cases of abortions also occur commonly. This arise as a result of various physiological, chromosomal or genital tract anomalies and other medical illnesses eg malaria, cardiac disease, anemia etc. Our patient had procured an abortion it was not spontaneous.

Induced criminal abortion has previously been associated with the young single women. In some studies, these are also the patients that were likely to have a fatal outcome (5, 6). In Kenya, the adolescent girl, the unmarried poor and unemployed woman has been found to be the main culprits (6). This has also been found to be tha case in the rest of Africa (5). In the 1970's a study done to review abortions in Kenya showed that in Nairobi, 53% of patients with septic abortions were teenagers while 79% of single women were in the induced group. Our patient was young, poor and unmarried.

Induced (illegal) but safe abortions are expensive in Nairobi (8). Safe abortion remains a preserve of mainly the elite. W. H. O. defines unsafe abortion as a procedure for terminating an unintended pregnancy carried out either by persons lacking the necessary skills or in an environment that does not conform to minimal medical standards or both (9).

Worldwide it has been estimated that 2 out of every 5 pregnancies are unplanned- the result of non-use of contraception or of ineffective contraceptive use or method failure. Defing pregnancy as either planned or unplanned is complex; contraceptive prevalence is low and women have lirttle autonomy to birth control. An unplanned pregnancy could either be unwanted or wanted and like-wise a planned pregnancy could become unwanted. Expanding and improving family planning services can help reduce unintended pregnancy and induced abortion. However, family planning services are frequently unable to meet the demand, or may be inaccessible or unaffordable, or there may be a range of social barriers that prevent women and couples from using them. Studies shows that many married women in developing countries do not have access to the contraceptive methods they would prefer to use in order to space pregnancies or limit family size. The situation is worse for unmarried women, particulary adolescents, who reraly have access to reproductive information and counseling, and are frequently excluded from contraceptive services (9). In our case the patient had all those factors unmarried, unemployed, had never used any contraceptives before.

Back street arbotions are dangerous because they are usually carried out by semi skilled individuals under conditions which, while making for secrecy, deny asepsis. Apart from introducing infection,

instruments passed blindly may perforate the uterus or the vagina and other abdominal viscera (7). This was certainly the case with our patient who had perforation of the uterus and the small intestines with generalized peritonitis.

Abortion complications include incomplete abortion, sepsis, uterine perforation, vaginal bleeding and trauma to the genital tract. Causes of death include infection heamorrhage , pulmonary embosism, shock, renal failure, peritonitis and septicemia. Air embolism is also not an unknown finding at post mortem examination. A later complication is tubal occlusion leading to infertility other psycho-social effects (8).

Physical examination of the patient rarely allows a deduction that the abortion has been deliberately induced. Occasionally, signs of recent injury to the cervix, uterus or vaginal are found, or the circumstances of the case may rise suspicions and even provide proof. Usually the patient will not admit to criminal interference (10).

Reproductive health education should be made available to the young adolescents who find themselves pregnant before they even understand their sexuality.

**REFERENCES:**

1. Tindall VR: Abortion. In: Jeffcoate's Principles of Gynaecology, 5<sup>th</sup> edition, Butterworth – Heinmann; London 1987:210-211.
2. Rogo KO. Induced Abortion in Sub – Saharan Africa. EAMJ 70, 386; 1993.
3. Aggarwal VP. Review of Abortions at Kenyatta national Hospital. EAMJ 57,138;1980.
4. Omuga BOO. Presentation of Abortion and its preventive problems at Kenyatta National Hospital. Mmed Thesis, University of Nairobi, 1989.
5. Unoigbe JA; Oronsaye AU, Orhue AAE. Abortion Related Morbidity and Mortality in Benin City, Nigeria. (1973-1985). Int J. Gynecol. Obstet 26:435:1988.
6. Murugu N. A. Ten Year Review of Mortality due to Abortion at Kenyatta National Hospital. Mmed Thesis, University of Nairobi, 1985.
7. Lema V, Rogo K, Kamau K. Epidemiology of Abortion in Kenya. The Centre for the study of Adolescents, Nairobi, 1989.
8. Strostrand M, Quist V, Jacobson A, Bergstrom S, Rogo KO. Socio-economic client characteristics and Consequences of Abortion in Nairobi. EAMJ 72:325-232.
9. WHO. Global and Regional Estimates of the Incidence of Unsafe Abortions and Associated Mortality in 2000 Fourth Edition, World Health organization 2004.

**Gynaecological case 10****NORPLANT IMPLANT REMOVAL DUE TO METRORRHAGIA**

NAME: C.M.                      OP NO. 0002267  
AGE: 32 YEARS                DATE OF REMOVAL: 2/02/2006  
PARITY: 1+0

**History of presenting complaint**

The patient had norplant implants inserted in November 2004 because she had wanted a long term reversible method of contraception. Since insertion, she started experiencing per vagina spotting in between periods on and off. The periods were prolonged in duration occurring over seven days instead of the three days as previously. The spotting had been almost daily and occurred several times a day. As of January 2006 she started having continuous per vaginal bleeding. Though not much in amount it was inconveniencing and expensive as she had to use several sanitary towels a day. She had been followed up at the Family Welfare Clinic and treated at various times with combined oral contraceptive pills without any improvement. She presented at the Family Welfare Clinic on 8/3/06 requesting removal of the implants.

**Past medical and surgical history**

This was not significant

**Obstetric and gynaecologic history**

She was para 1+1. Her last delivery was in October 2004; she had spontaneous vertex delivery. The child is alive and well. She had an incomplete abortion in 2000 evacuation was done. This was followed by a long period of sub-fertility due to tubal factor. Saphingoneostomy was done with good results leading to the pregnancy already alluded to. She attained menarche at 16 years. Prior to her last pregnancy and before Norplant insertion her menses had been regular, occurring every 28 days and lasting 3 days. She had not used any other method of contraception. She had so far done 2 pap smears within a 3 year period which were satisfactorily evaluated CINO.

**Family and social history**

She was a married business lady. Her husband was a business man. She stayed with her husband and children. She did not smoke cigarettes or take alcohol. There was no family history of chronic illness.

**Physical examination**

She was in good general condition. She was not pale and had no jaundice, cyanosis or lymphadenopathy. She was afebrile. Her blood pressure was 110/70mmHg, pulse rate 70/minute and respiratory rate was 18/minute.

**The respiratory, cardiovascular and central nervous systems**

These were essentially normal.

**Abdominal examination**

The abdomen was flat and moved with respiration. She had no scars. There were no areas of tenderness and no masses were palpable. The liver and spleen were not palpable

**Speculum examination**

She had normal external genitalia but blood stained. The vaginal mucosa and cervix were healthy. There was blood emanating from the cervical os. On digital vaginal examination, the vaginal mucosa was healthy, the cervix was smooth, firm and the os was closed. The uterus was normal in size and the adnexae were free. The pouch of Douglas was empty. There was blood on gloved examining fingers.

**Diagnosis**

An impression of metrorrhagia secondary to Norplant implant was made.

**MANAGEMENT**

The implants were to be removed. The patient was placed in supine position and her left arm, which had the implants, extended and supported on an arm rest. The upper arm was washed with savlon and a sterile drape applied with exposure of only the area of the implants. Six millilitres of 2% lignocaine was drawn. The implants were located by palpation. The needle was inserted over the insertion scar at the base of the "fan". A small amount of the anaesthesia was infiltrated and from this site a small amount of local anaesthesia agent was infiltrated subdermally under each of the six implants. Using

the scalpel, a 4mm incision was made over the previous scar. The implants closer to the incision were pushed digitally towards the incision. The visible tips were grasped with mosquito forceps. Fibrous tissue around the implant was gently scrapped away using the scalpel and the implant grasped with a second mosquito forceps and removed. For the implants that could not readily be pushed into the incision, a closed forceps was inserted into the incision. The implants were palpated and grasped using the forceps and gently guided into the incision. Once again, the fibrous tissue around the implants was carefully dissected using the scalpel thus releasing the implant, which was grasped with a second forceps and removed. The six capsules took 20 minutes to remove. After all had been removed, the edges of the incision were pressed together and an elastoplast applied. This was covered with gauze and gauze bandage was wrapped round the arm to prevent bleeding. She was instructed to keep the inner upper arm dry for the next 3 days and to remove the gauze after that. The elastoplast was to be removed after 5 days. She was given combined oral contraceptives as an alternative method which she chose after counselling. She was discharged to be reviewed after a week.

### **Follow up**

She was reviewed as planned. The bleeding had stopped 5 days after Norplant removal. The operation site had healed well. She was given COC pills for 3 months. She has so far been seen twice since removal of Norplant for non-gynaecologic conditions her menses had become regular.

### **DISCUSSION**

Presented is a 32 year old Para 1+1 who had norplant removal due to metrorrhagia. Norplant implants consist of flexible non-biodegradable tube filled with levonorgestrol, a synthetic hormone of the progestin family. The implants are placed subdermally on the inside of a woman's upper arm. The hormone is slowly released at an almost constant rate for several years.

The implants are in three forms. The first, called norplant, consists of six hollow silastic (silicon rubber) capsules. Each is 3.4cm long, with a diameter of 2.4mm and contains 36mg levonorgestrel. The sides of the capsule are sealed with silastic adhesive. Its contraceptive effect lasts for five years. It was previously widely used at the Family Welfare Clinic, Kenyatta National Hospital, and had been inserted in the client. The other two are the latest varieties called Jadelle and Implanon. Jadelle consists of two solid silastic rods, each 44mm long and each containing 75mg levonorgestrel. Its contraceptive effect lasts 5 years. Implanon consists of one silastic rod and its contraceptive effect

lasts 3 years (2). In Kenya National Hospital, currently, Jadelle is widely used and norplant has been phased out.

In Kenya, introductory trials of norplant took place in 1986 in Machakos (1). The mechanism of action of norplant is through suppression of ovulation and increase in cervical mucus hostility creating a thick mucus plug at the cervix unfavourable for sperm penetration. It also suppresses the cyclic development of the endometrium in half of the users (2).

Continuation rates after one year of norplant use range from 87 to 95% (1, 2) and after 2 years from 66 to 92% (2). Bleeding irregularities is the most frequent reason for discontinuation and accounts for 2-7% discontinuations in the first year. This is lower than the 6-30% discontinuation rates reported for long acting injectables such as depo provera (2).

Menstrual irregularities are the most common side effects of norplant. Approximately 60 percent of women notice changes in their menstrual patterns in the first year after insertion (1, 2, 3, 4). These changes vary from woman to woman and include prolonged menstruation, spotting between the period, amenorrhoea or combination of patterns. The most common are an increase in the number of days of bleeding and/or spotting per cycle and a decrease in the length of the menstrual cycle. Our client had experienced both a prolongation in the number of days of menstruation as well as intermenstrual spotting. Though irregular bleeding decreases over time for most women, this client's problem persisted and in fact appeared to be getting worse. This necessitated her request for removal of the implants.

Such menstrual changes as experienced by this client have shown to have no apparent harmful effect. Heavy bleeding is not common, although many women bleed more often than before; the volume of blood does not change. In some studies, the average haemoglobin levels increased significantly after norplant insertion. In others, the average level remained the same (2). Our client had been bleeding for about 1 year yet her haemoglobin was 10.5g/dl.

Amenorrhoea is less common than prolonged bleeding or spotting but may occur in as many as 25% of users (2). Other side effects associated with norplant use are few. Headache is the most common other complaint that may warrant removal of the implants. Minor changes all within normal range have been observed in liver function, carbohydrate metabolism, blood coagulation, blood pressure,

immunoglobulins, serum cortisol, urea nitrogen, uric acid, sodium, potassium, calcium, inorganic phosphorus and body weight (2). Implants may also be removed at the client's request for whatever reason. Also in the rare instance that pregnancy occurs.

The removal of the 6-capsule norplant is usually more difficult than the insertion. It usually takes 15-20 minutes. Usually only one incision is required. Occasionally implants cannot be removed at the first attempt. Complications that may occur after removal of the implants include haematoma formation and wound infection. In this client, all the 6 capsules were removed within 20 minutes. She required only the incision. Sterile technique was used and she did not develop wound infection neither did she develop a haematoma (4).

The contraceptive effect of the implants ends soon after they are removed. Former users of norplant conceive as rapidly as women who have not used contraception. A backup method of contraception, such as combined oral contraceptive pill used by this client, are thus necessary upon removing the implants if the client does not immediately want another child (2, 4).

**REFERENCES**

1. Mati J.K.G.: Norplant experience in Kenya. 14<sup>th</sup> Annual Scientific Conference KOGS 1989.
2. Hatcher R.A., Kowal D.: Implants, injections and other progestin only contraceptives In: Contraceptive technology; 1989; 16:281.
3. Sataypan S. et al.: Perception and acceptability of norplant implants in Thailand. Studies in family planning 14 (6-7): 179-176 June-July 1983.
4. Norplant: Subdermal implant system for long term contraception. Am. J. Obstet Gynecol 1989; 160(5):1286-92

**Gynecological case 11****VESICO –VAGINAL FISTULA SUCCESSFUL URETERO-NEOCYSTOSTOMY**

NAME: J.A.	IP NO.:	0946959
AGE: 32 YEARS	D.O.A:	29/09/2006
PARITY: 5 + 0	D.O.D.:	18/10/2006

**Presenting complaint**

The patient presented with a 3 year history of effortless leakage of urine.

**History of presenting complaint**

The patient was being followed up in our GOPC as a case of vesico vaginal fistula following a sub-total hysterectomy due to ruptured uterus in labour in 2004. She had laboured at home with a previous scar(Then she was a para 4+0 IPSC). She was brought to Kenyatta National Hospital in hemodynamic shock she was adequately managed as previously alluded to. Intra-operative period no obvious injury to the bladder or ureters was noted. However there was a generalized edema of the bladder area and the uterine tear ran from one lateral end to the other. The outcome was a fresh still birth who weighed 3400 grams. Post operatively she had an indwelling catheter for 2 weeks. She developed continuous leakage of urine a few days after removal of catheter and uretero-vaginal fistula was diagnosed after a dye test that confirmed leakage of urine however it was clear with no dye in it. She was asked to have an Intravenous Urogram then be scheduled for surgery. Due to financial reasons she opted to wait for the AMREF project for investigation and surgery.

She gave no history of faecal incontinence or weakness of the lower limbs.

**Obstetric and gynecologic history**

She was para 5+0. Her last delivery was in 2004. She had a still birth following uterine rupture. The previous pregnancy was a caesarean delivery due to breech in 1995. The other pregnancies had been vaginal deliveries at term all the four children were alive and well.

She had never had a pap smear before and had been amenorrhoeaic since surgery.

**Past medical history**

This was not contributory

**Family and social history**

She was a business woman who had separated from her spouse. Her spouse left her after she developed leaking of urine. She lives in Kawangware, her children are up-country with her parents. She did not drink alcohol or smoke cigarettes. There was no family history of chronic illness.

**Physical examination****General examination**

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

Vital signs were:

Blood pressure: 110/70mmHg

Temperature: 36°C

Pulse rate: 72 per minute

Respiratory rate: 20 per minute

**Abdominal examination**

She had a lower midline surgical scar. There were no palpable masses.

**Pelvic examination**

She had moderate excoriation of the vulva and perineum. It was obviously wet. Continuous dribbling of urine was noted. Using a Sim's speculum, no obvious fistula was seen in the vaginal vault but there was urine collection in the vagina fornixes. Dye test was done. There was leakage of clear urine in the vagina from an area above the anterior lip of the cervix .

**Central nervous system**

The lower limbs had power grade V in all muscles groups with normal reflexes.

**Respiratory and cardiovascular systems**

These were essentially normal.

## Diagnosis

A diagnosis of vesico-vaginal fistula type I was made.

## Management

The patient was again informed about the diagnosis and the intended mode of management. She raised no objection. She was planned for examination under anaesthesia and repair of the fistula at the same sitting. The following pre-operative investigations were done:-

Haemogram	-	Hb	10.6g/dl
		WBC	8.52 x 10 <sup>9</sup> /L
		N	63.1%
		L	28.8%
		PLT	337x10 <sup>9</sup> /L
Renal function test		Urea	4.2mmol/L
		Creatinine	94µmol/L
		Chloride	98mmol/L

### *IVU Contrast films*

Report: There was prompt bilateral excretion of contrast. Both kidneys were in normal position. There was bilateral semiduplex\compound calyceal system with left hydrocalycosis, dilated extra-renal pelvis and hydroureter. The right showed a normal sharp calyceal system with normal dynamic peristaltic waves. There was a left distal ureteric (pelvic cavity) tight structure with beaking. The oblique films showed leakage into the pelvic cavity (probably leading to communication with the vagina)

Conclusion: Left obstructive uropathy.

On the day of admission, informed consent was taken. the patient was given fluid diet and enema in the evening prior to the operation. Enema was given again at 6am on the day of the operation. She was starved from midnight and started on intravenous fluid. On the morning of operation, half an hour before theatre, she was premedicated with intramuscular atropine 0.6mg and intravenous gentamicin 80mg stat. Shaving was done. She was wheeled to theatre.

### *Uretero-neocystostomy*

In theatre, the patient was put under general anaesthesia and placed in lithotomy position. Aseptic catheterization of urinary bladder done. Examination under anaesthesia performed the findings concurred with what was found at the previous evaluation. Patient was now in supine position. The abdomen was cleaned and draped with sterile towels. The abdomen was opened through a repeat midline sub-umbilical incision.

Findings; no remarkable findings other than a few adhesions between the sigmoid colon and the left tube and ovaries. The ureters were not dilated.

Done: Few adhesions released. The bladder was opened; right ureteric orifice was seen to be producing urine. The left ureteric orifice was not identified. The broad ligament on the left was dissected and the left ureters identified. The left ureter was dissected and tied distally 2 times with vicryl 2/0. Stay in sutures were placed on the most proximal part. A tunnel was created in the broad ligament to the bladder then the ureter was tunnelled into the bladder. This was done with care not to kink it. It was then spatulated and sutured on to the bladder mucosa at 6 sites with vicryl 4/0. A ureteric catheter was inserted into the bladder then to the left ureter and fixed on to the bladder mucosa using vicryl 4/0. Bladder wash out was done and the bladder closed in a continuum using vicryl 3/0. Reperitonization was done on the left broad ligament area. The abdomen was cleaned and closed after correct swab and instrument count.

The ureteric catheter was sutured to anterior abdominal wall, while the urethral foleys catheter was strapped firmly on the lateral side of the anterior abdominal wall to avoid traction and mobility. It was fixed to a urine bag. Anaesthesia was reversed successfully. The patient was then wheeled to the recovery area.

### **Postoperative care**

Vital signs were observed continuously until she was fully awake then 4 hourly. An intravenous drip of normal saline alternating with 5% dextrose 500mls 4 hourly for 24 hours was started in theatre. The patient was also encouraged to take free fluid orally. She was advised to have bed rest, and take at least 6 litres of fluids per day. After 24 hours, intravenous fluid was stopped. The catheter was inspected regularly for drainage and clarity of the urine. A fluid input-output chart was kept. This was satisfactory. On the first post-operative day, she was started on light diet. . On the second day, she was on normal diet and was encouraged to get out of bed. No leakage of urine was observed the bed remained dry and her urine was clear. The stitches and the ureteric catheter were removed on the

10<sup>th</sup> day post operative. She did well and was discharged home on 14<sup>th</sup> post operative day. The catheter was removed on the 21 day post operative there was no leakage noted even then. She was advised not to have coitus for 6 months. She was to empty her bladder every 1-2 hours during the day and every 3-4 hours at night. She was asked to come for review in the outpatient clinic after 6 weeks during which she was found to have no complains and was encouraged to uphold the advice that had been given earlier.

## DISCUSSION

Presented is J.A., a 32 year old para 5+0 who developed Uretero-vaginal fistula following sub-total hysterectomy after uterine rupture while in labour with a previous scar. She underwent a successful repair.

Obstetric fistula is a common problem in countries with limited medical resources and access to antenatal care (1,2). This is compounded by many social and cultural factors which lead to delay in seeking obstetric intervention when labour is obstructed or prolonged (4). Specific cultural problems include marriage at an early age, often before skeletal maturity, illiteracy and lack of contraception all of which result in high parity and poor nutritional status (1-4). This problem is exacerbated by the rural nature of most of East Africa with lack of public transportation.

Urinary and feecal incontinence is a devastating social handicap common in countries with limited medical resources (4).

In East Africa, genitourinary fistula is a common complication of childbirth, occurring in 3-4 per 1000 deliveries (2). About 50,000 to 100,000 new cases are reported each year (1,2). The most common risk factors leading to obstetric fistula are first delivery and prolonged labour (3,4). A smaller number of obstetric fistulae being associated with emergency caesarean section and instrumental delivery for obstructed labour. In our case JA may have gotten the fistula from damage during the uterine rupture or during the subtotal hysterectomy procedure.

The pathogenesis of genital fistula is pressure necrosis and ischemic injury rather than direct mechanical or surgical trauma (1-4, 5-8).

Isolated vesico-vaginal fistula is more common than combined vesico-vaginal and recto-vaginal fistula (5-9).

In a study done in Ethiopia 45% of women with fistula had a vesico-vaginal fistula involving the bladder neck or vault (13). The high rate of bladder neck and vault fistula is most likely due to

cephalopelvic disproportion (2, 6, 7, 8). This problem may arise due to a combination of factors including early age at first delivery, poor nutrition, skeletal immaturity and an android pelvis (2,7).

Observational studies from Africa report successful closure of obstetric fistula in 75-95% of delayed women with primary repair (10,11). Despite the high rate of success, persistent urinary incontinence following fistula surgery has long been recognized, occurring in 10-12% of women (12).

Causes of post-operative urinary incontinence include failure of repair, stress and urge incontinence and detrusor instability (11). Urodynamic assessment has shown that genuine stress incontinence is usually the cause of persistent urinary incontinence after surgery. Fortunately for our patient, she had no leakage post repair.

Faecal incontinence has been reported in association with repair of vesico-vaginal fistula but the frequency of the problem is unclear (3,6,7,10). The mechanism of injury leading to faecal incontinence is unclear.

Several authors have noted footdrop in a quarter of African women who have obstetric fistula (2,4). This finding linked to the increased frequency of high obstruction in labour may suggest a neuropathic rather than mechanical aetiology (1,2,6,7). This hypothesis remains to be tested. Our patient did not have foot-drop with normal power in both lower limbs.

Goodwin and Scardino refer to urogenital fistula in a woman as one of the great romances between urologic and gynaecologic surgery .J. Marion Sims (1852) is considered the father of surgery for vesicovaginal fistula. Fistulous openings between the urinary tract and the genital tract may involve the urethra, bladder or ureter. The most common occur between the bladder and the vagina.

Fistulae may be located at any point along the anterior vaginal wall. High fistula with uterus in situ are supratrigonal in location, that is, above the interureteric ridge. Midvaginal fistula are located below the interureteric ridge and are mainly caused by obstructed labour.

Vesicovaginal fistula fall under 4 groups. Those resulting from

- 1) Obstetric injury,
- 2) Operative accidents,
- 3) Extension of carcinoma of the cervix or radiotherapy treatment,
- 4) Miscellaneous causes.

The patient discussed developed vesicovaginal fistula as a result of obstetric injury. The incidence of vesicovaginal fistula is high where childhood malnutrition is prevalent, formal education for girls is not encouraged, childhood marriages are encouraged and quality of antenatal care is poor or not

available at all. Our patient had formal education up to standard eight and did not attend antenatal clinic during her pregnancy.(2)

Lawson in Ibadan, Nigeria (1975) and Hamlin in Addis Ababa, Ethiopia reported vesicovaginal fistula to be predominantly obstetric in aetiology. At John Hopkins Hospital 44% of fistulas were due to gynaecologic surgery 20% due to obstetric injury, 32% were related to cancer of the cervix and its treatment and 4% were from miscellaneous causes. Tahzib and Orwenyo report that 52% and 47% of vesicovaginal fistulae occur with the first pregnancy respectively. Orwenyo further reported that 43% of the patients with vesicovaginal fistulae are between 19-25 years, while in Northern Nigeria, 8.6% of those with vesicovaginal fistulae occur mainly in young women whose reproductive capacity is at its peak. The patient discussed developed the fistula during her first pregnancy, she was 21 years of age and married (3, 4,5).

Vesicovaginal fistulas (VVF) are the most common genitourinary fistulas and mainly arise from obstetric causes especially in Africa and Asia. Since they occur in young women, it is a tragic social and medical circumstance, turning them into misfits. During labour the urethra and the bladder are pulled up into the abdomen. *During prolonged labour, pressure occurs on the vaginal and bladder wall between the head of the fetus maternal pubic bone.* This results in ischaemic necrosis of these soft tissues and the devitalized tissues slough off in 3 to 10 days leaving a defect thus forming the fistula. The same process occurs on the vaginal and rectal walls between the fetus and sacral promontory. In Orwenyo's series the two types of fistulas occur simultaneously in 2% of the patients (5).

Incidental causes of fistulae include injuries during sexual assault, falls on sharp objects and certain harmful traditional practices such as female genital mutilation.

The diagnosis of vesicovaginal fistula is made mainly through history taking. With a small fistula, the urinary leakage will be slight and in some instances depends on the position of the patient. Such a fistula may allow voiding of large amounts of urine. In large fistulas sufficient urine does not collect in the bladder to permit voiding (1, 2). Most fistulas are painless except those caused by radiation which are extremely painful, especially aggravated by walking or sitting.

With marked incontinence, the vulva usually becomes reddened, tender and excoriated over time. The odour of the urea is usually offensive, disgusting to the patient and repulsive to other people(11). This was the case with our patient.

If the patient had received radiotherapy the necrotic fistula edges must be biopsied to rule out malignancy recurrence.

A fistula that cannot be readily demonstrated may be found by instilling a dye in the bladder and inspecting the anterior vaginal wall and vaginal vault. When the fistula is small and can not be demonstrated when the patient is in lithotomy position or knee chest position the three- tampon test of Moir is used. In this test, three cotton tampons are placed in the vagina in tandem, after instillation of the methylene blue dye in the bladder. If the lowest tampon is wet and stained, a trans urethral urinary incontinence is suspect. If the upper tampon is wet and blue, then vesicovaginal fistula is suspect and if it is wet and not blue, a ureteral fistula is the likely diagnosis. This was not the case in our patient, there was leakage of urine but it was negative of dye. Other diagnostic aids include air or water cystoscopy and intravenous indigo carmine (1)

Before definitive repair of the fistula, concomitant medical problems such as malnutrition, anemia and infections are controlled first (3). An asymptomatic fistula is never repaired. For pinpoint fistulas 2-4 weeks of catheter drainage may suffice. With the catheter drainage technique 5% of the fistulas undergo spontaneous closure. Cauterization or steroid application on the fistula is not recommended.

Definitive surgery is planned depending on the number of the fistula found, their size and position. The amount of fibrosis, fixity and amount of tissue available for mobilization are also assessed. Vesicovaginal fistula can be repaired with an abdominal or a vaginal approach, which have similar success rates (>85%). Vaginal approach is preferred in most instances due to its advantages which include excellent exposure, minimal blood loss no compromise to subsequent abdominal attempts at repair, reduced operative time and post operative pain and shortened hospital stay (2, 16, 18). Relative contraindications for abdominal approach include radiation fistulas multiple prior abdominal surgeries, the obese, elderly and the immobile patients.

Abdominal route is best for high fixed vesicovaginal fistula which are inaccessible per vaginum. If the route of repair is vaginal, the position of repair has to be determined. The lithotomy position is best for juxtacervical and high mid-vaginal fistulae. The knee elbow position is best for juxta urethral and low mid-vaginal fistulae. If the vesicovaginal fistula is associated with a recto vaginal fistulae, a defunctioning colostomy is made at the same sitting. Regardless of the approach used, suprapubic catheter and the urethral catheter must be in place and urethral stents inserted if the fistula is close to the ureters. Our patient had a uretero-vaginal fistula which was successfully repaired abdominally.

Post operatively the bladder is drained continuously to avoid bladder distension which may disrupt the suture line. This also ensures washing away of blood clots which avoids infection (5,6).

The most common complication of fistula repair is breakdown of the repair. This usually occurs 7-10 days after operation. If it occurs, catheter drainage should be continued for several weeks to provide an adequate opportunity for the fistula to heal spontaneously. The other complications include urinary tract infection, wound infections intravesical haemorrhage, vaginal stenosis with cryptomenorrhea. Urethral obstruction which is also a complication will manifest as persistent fever, abdominal distension, pain and tenderness (7, 10, 11, 19). Our patient did not have any of these complications.

Following successful repair abstinence from sexual intercourse is advised for a period of not less than 3 months. In the event of pregnancy the patient is advised to start antenatal clinic early and plans are made well in advance for an elective caesarean section (1, 2, 7, 14) .

Vesicovaginal fistula is a preventable problem. Programmes should address provision of adequate , accessible, affordable and quality antenatal services. Professionals should be available to handle complicated cases like obstructed labour. The problem of poor formal education , early marriages and female genital mutilation once addressed will reduce the incidence of vesicovaginal fistulas.(1, 2, 6)

## REFERENCES

1. Richard F. L., Gary E. L. Prevention and management of Urovaginal fistulas *Clinical Obstetrics and Gynaecology*. Vol: 33, NO. 2 June 1990
2. Mattingly F. R. Thomson J.D. Te Linde's *Operative Gynecology*. 7<sup>th</sup> Edition Page 637
3. Lwason J. Vesicovaginal fistula-a tropical disease. *Transactions of the Royal Society of Medicine and Hygiene* 83: 454-456. 1989.
4. Tahzib F. Epidemiology determinants of vesicovaginal fistulae.  
*Br J Obs. Gynae*. 1983: 90., 387-91.
5. Orwenyo E. A. A restrospective Study of 166 cases of acquired urogenital or rectovaginal fistulae treated at Kenyatta National Hospital during 1979-1982, M. Med. Thesis 1985.
6. Howard W. J. Anne C. W. Lonnie S. B. Novak's *Textbook of Gynaecology*.  
12<sup>th</sup> Edition Page 463.
7. Goh JT. Genital tract fistula repair on 116 women. *Aust N Z J Obstet Gynaecol* 1998;38(2):158-161.
8. Margolis T, Mercer LJ. Vesicovaginal fistula. *Obstet Gynecol Surv* 1994 ;49 :840-847.
9. Danso KA, Markey JO, Wall LL, Elkins TE. The epidemiology of genitourinary fistulae in Kumasi, Ghana, 1977-1992. *Int Urogynaecol J Pelvic Floor Dysfunct* 1993;7(3):117-120.
10. Mathelaer JJ, Williams G. Extraurethral urinary incontinence after incompetent vaginal obstetrics. *Br J Urol* 1999;84(1):10-13.
11. Steiner AK. The problem of post-partum fistulas in developing countries. *Acta Trop* 1996;62:217-223.
12. Goh JT, Krause HG. 1995 Brown Craig Travelling Fellowship destination: Ethiopia. *Aust N Z J Obstet Gynaecol* 1996;36(3):335-338.
13. Gharoro EP, Abedi HO. Vesicovaginal fistula in Benin City, Nigeria. *Int J Obstet Gynecol* 1999;64(3):313-314.
14. Amr MF. Vesico-vaginal fistula in Jordan. *Eur J Obstet, Gynaecol Reprod Biol* 1998;80(2):201-203.

15. Hilton P, ward A. Epidemiological and surgical aspects of urogenital fistula: a review of 25 years experience in south east Nigeria. *Int Urogynaecol J Pelvic Floor Dysfunct* 1998;9(4):189-194.
16. Woo HH, Rosario DJ, Chappler CR. The treatment of vesico-vaginal fistulae. *Eur Urol* 1996;29 (1):1-9.
17. Hilton P. Urodynamic findings in patients with urogenital fistulae. *Br J Urol* 1998;81(4):539-542.
18. Hilton P. debate: postoperative urinary fistulae should be managed by gynaecologists in specialist centers. *Br J Urol* 1997;80(Suppl 1):35-42.
19. Murray C, Goh JT, Fynes M, Carey MP. Urinary and faecal incontinence following delayed primary repair of obstetric genital fistula. *BJOG* 2002, 109:828-832.

**Gynecology case no. 12****TITLE: ECTOPIC PREGNANCY-RIGHT SALPINGECTOMY DONE**

NAME: E.S.                    IP NO: 0998756  
AGE: 30 YEARS                DOA: 12.1.2004  
PARITY: 1+0                    DOD: 16.1.2004  
LMP: 1.12.2003

**Presenting complaint**

Severe lower abdominal pain for 2 days

Fainting for 1 day

Vomiting for 1 day

Per vaginal bleeding for 1 day

**History of presenting illness**

The patient developed sudden onset lower abdominal pain 2 days before admission. The pain continued to get worse with time and was not relieved by analgesics. She then developed dizziness and later began to faint. She also began to vomit and developed per vaginal bleeding which was scanty and brownish.

**Obstetric and Gynecologic history**

She was Para 1+0, her last delivery was in 2000. It was by spontaneous vertex delivery to a male who weighed 2900g. The puerperium was normal.

She reached menarche at 15 years. She was currently not using any contraceptive method. Her last menstrual period was 1.12.2003 giving a period of amenorrhea of 6 weeks.

**Past Medical History**

This was not significant

**Family and social history**

She was a married police officer and her husband was a teacher. There was no family history of any chronic illness. She did not drink any alcohol or smoke cigarettes.

**Physical examination**

She was ill looking and markedly pale. There was no jaundice or oedema. She was also afebrile. She was in mild respiratory distress.

*Vital signs:* blood pressure 90/50 mmHg, temperature 36.7C, pulse 100/min, respiratory rate 30/min.

**Central Nervous System**

She was in a state of agitation but was oriented in time, place and person. The cranial nerves were intact and the pupils were bilaterally and equally reacting to light. The motor and sensory systems were normal.

**Abdominal examination**

The abdomen was slightly distended in the suprapubic region. The abdomen was not moving normally with respiration. There was marked suprapubic tenderness, guarding and rebound tenderness. Paracentesis was positive for non-clotting blood.

**Pelvic examination**

The external genitalia were normal. The perineum was covered with blood clots. The cervix was 3cm long and the cervical os was closed. The cervical motion test was positive. The pouch of Douglas was full and tender. The uterus was bulky and there was tenderness in the adnexae.

**Impression:** Ruptured ectopic pregnancy

**Management**

The patient was put on intravenous fluids and blood taken for grouping and cross-match. The patient was informed of the diagnosis and the need for immediate laparotomy. Informed consent was obtained and premedication with atropine sulphate was done.

**Laparotomy**

In semi-lithotomy the perineum was cleaned and the bladder aseptically catheterized draining clear urine.

In supine position the abdomen was cleaned and draped. The abdomen was opened in layers via a lower midline incision.

*Findings:* massive haemoperitoneum of 3 litres, ruptured right tubal pregnancy in the ampullary portion of the tube and adhesions involving the tubes, ovaries and uterus. The left tube was essentially normal.

*Done:* Right salpingectomy. Haemostasis was achieved. Abdominal lavage was done. The patient was transfused 1 unit of whole blood intra-operatively.

The abdomen was closed in layers and general anaesthesia was reversed uneventfully.

### **Post-operatively**

The patient was observed continuously until fully awake then 4 hourly. She was also put of intravenous fluids 5% dextrose and normal saline alternately. The patient was put on prophylactic broad spectrum antibiotics ( gentamicin, metronidazole and crystalline penicillin.)

While in the ward oral feeding was gradually re-introduced. She was discharged fairly well on the fourth post-operative day.

### **Follow up**

The stitches were removed on the 7<sup>th</sup> post-operative day at a peripheral clinic. She was seen in the gynaecology clinic a week later and was found to have recovered fully. She was then put on routine gynaecology follow-up.

## **DISCUSSION**

Ectopic pregnancy occurs when the blastocyst implants outside the mucous membrane lining the uterine cavity and is a condition that is peculiar to humans (1). The fallopian tube is the commonest site for ectopic pregnancies to occur. Tubal ectopic pregnancies may be isthmic (25%) ampullary (55%) fimbrial (17%) interstitial (2%). Other rare ones include uterine (cornual, angular, in a rudimentary horn or intramural), cervical, intraligamentous, ovarian (0.5%) abdominal (0.1%) or combined with an intra uterine pregnancy also called compound or hetroteropic (1/ 17 000-30 000)(2). In this patient it was at the ampulla of the fallopian tube.

The incidence of ectopic pregnancy ranges from 1 in 200 to 1 in 250 and varies with country, socio economic status and age (2,3,4). Mwathe reported 2 to 5 patients per week at Kenyatta National Hospital in 1984 (5). There is a significant increase in ectopic pregnancies all over the world. This is due to increase sexually transmitted infections, increased use of intrauterine contraceptive devices, unsuccessful tubal sterilization, previous pelvic surgery, post abortal and puerperal sepsis and better

efficiency of antibiotic therapy thereby preventing total occlusion of tube following salpingitis but with residual tubal pathology amongst others (1,3,4,6,7). This patient had none of these risk factors.

Ectopic pregnancy occurs any time in the reproductive age group through 40% of them occur between 20-29 years. Both tubes are involved with equal frequency and rarely tubal pregnancy may occur in both tubes or co-exist with an intrauterine pregnancy (2,5,8) This Patient was 30 years old and had a right tubal pregnancy.

A high index of suspicion is needed to make the diagnosis. It should always be suspected in a woman of childbearing age with pelvic pain. The diagnosis usually rests on history and not physical signs and negative physical signs should not overrule the symptoms. The clinical features of tubal ectopic pregnancy may be acute, chronic or acute on chronic. The acute type though less common than the chronic type is more dramatic. It occurs when there is tubal rupture. There may or not be a period of amenorrhoea. Abdominal pains then irregular bleeding with abdominal tenderness. There is lancinating pain in the iliac fossa or hypogastrium. The patient may then collapse and there is pallor, hypotension, subnormal temperature a weak rapid pulse and death may quickly ensue(4,9,10,11)This patient had abdominal pains on and off for a week and sustained severe pain and vaginal bleeding which made her seek medical attention much later. At the Kenyatta National Hospital low abdominal pain is the commonest complaint in 84-100% of these patients of these patient with 91.2% of them having haemoperitoneum.(5)

Quantitative beta hCG measurements are the diagnostic cornerstone for ectopic pregnancy. The hCG level correlates with the gestational age (80) and an subnormal rise in hCG may indicate ectopic pregnancy. A serum progesterone level can be used as an ectopic pregnancy screening test. A serum level of less than 5ng/ml is highly suggestive of an abnormal pregnancy. Serum levels of progesterone higher than 25 ng/ml is indicative of viable intrauterine pregnancy in 70% of cases (13).

Improvements in ultrasonography have resulted in the earlier diagnosis of intrauterine and ectopic gestations (14). However, the sensitivity of the beta hCG assay usually allows the diagnosis of pregnancy before direct visualization by ultrasonography. The earliest normal gestational sac is seen at 5 weeks gestation with transabdominal ultrasonography and at 4 weeks gestation with transvaginal ultrasonography. (15). Nonvisualization of an intrauterine gestation with serum hCG levels higher than 6500 mIU/ml indicates an abnormal (failed intrauterine or ectopic gestation) pregnancy (16).

The demonstration of an adnexal gestational sac with a foetal pole and cardiac activity is the most specific but least sensitive sign of ectopic pregnancy, occurring in only 10-17% of cases (17).

17. Rottem S, Thaler I, Levron J, et al. Criteria for transvaginal sonographic diagnosis of ectopic pregnancy. *J Clin Ultrasound*. 1990; 18: 274-279.
18. Stovall TG, Ling FW, Buster JE. Outpatient chemotherapy of unruptured ectopic pregnancy. *Fertil Steril* 1989; 51: 435-438.
19. Gomel V: Laparoscopic tubal surgery in infertility. *Obstet Gynecol* 1975;46:47.

**Gynecological case 13****PELVIC ABSCESS**

NAME: C A. IP NO: 1121252  
AGE: 26 YEARS DOA: 6/10/06  
PARA 0+0 DOD: 13/10/06  
LMP: 28/9/06

**Presenting illness**

She was a 26 year old nullipara who presented these complaints;

- Abdominal pains for 1 month.
- Vomiting for 5 days.
- Dysuria and foul smelling vaginal discharge

She experienced progressively worsening abdominal pains which became generalized.

This was associated with vomiting and fever. There was associated foul smelling per vaginal discharge. She had Dysuria and frequency of micturation. She had not opened bowels for two days prior to admission.

**Past Medical History.**

The medical history was not significant.

**Obstetric and gynecological history**

She was nullipara

LMP 28/9/06. She was not sure of her menarche. She had rregular flow of 4-7 days cycle of 30 days.

No dysmenorrhea or hemorrhagic. No pap smear was done.

**Family and social history**

Married and lives in Runda. Does not smoke cigarettes or drink alcohol. No history of chronic illness in the family.

**On examination.**

She was sick-looking, pale and febrile. She was not jaundiced. BP was 130/70 mmHg. Pulse of 120/min. Respiratory rate of 20/min. Temperature 38°C.

**Abdominal examination.**

The abdomen was not distended; there was slight movement with respiration. There was marked generalized tenderness with guarding. Bowel sounds were present and normal.

**Vaginal examination.**

She had normal external genitalia. The cervix was central firm and closed. The Pouch of Douglas felt full. Adnexa were free but with tenderness especially on the right.

**Abdominal pelvic ultrasounds scan.**

There was a large collection of septed fluids in the pelvis region. The uterus was normal. There was a moderately echogenic solid mass around the bladder, consistent with sludge ball. The pelvis spleen and both kidneys had normal features.

Conclusion: pelvic abscess.

**Diagnosis** of pelvic abscess was made.

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**MANAGEMENT.**

The patient was made aware of the nature of her illness and counseled on the eventuality of an emergency laparotomy. A written consent was obtained. She was given atropine 0.6 mg before being wheeled to theatre.

She was wheeled in theatre and introduced to the theatre staff. She was put in supine on the operating table. She was given general anaesthesia. She was catheterized and clear urine drained. She was cleaned and draped. A subumbilical midline incision was made.

Findings;

- Abscess in the entire Pouch of Douglas.
- Complex tubo-ovarian abscess on the right.
- Left hydrosalpinx.

The abscesses were drained, all loculi of pus were broken and the peritoneal cavity cleaned with saline, rifocin was put in the peritoneal cavity and a drained left in situ. The abdomen was closed in

layers after correct swab and instrument count. The anaesthesia was reversed and the patient taken to the recovery ward for observation.

### **Post operative management.**

She was put on intravenous flagyl 500 mg 8 hourly, zinacef 750 mg 8 hourly and intravenous fluids(normal saline and 5% dextrose 1 litre 8 hourly),and pethidine 100mg 8 hourly for analgesia. The pus specimen was taken for culture and sensitivity. She did well postoperatively and by the third day, the drain was removed. She was discharged home on the seventh post operative day after removal of the stitches.

### **DISCUSSION**

Pelvic abscess is defined as localized collection of pus in the pouch of Douglas. It is the commonest type of intra-peritoneal abscess (1, 2).

Pelvic abscess is a major cause of morbidity and mortality amongst women in the reproductive age group. The incidence varies from country to country due to differences in management of gynecological admissions (2). A recent study in 2004 by Murima at KNH found that pelvic abscess accounted for 3.8% of all acute gynaecological admissions and was associated with a mortality rate of 9.5 % (3).

Patients who develop pelvic abscess are usually in their 20s and 30s with a large percentage (20%-59%) of these women being nulliparous (2). Our patient was 30 years old.

The aetiology of pelvic abscess can be classified into either pelvic causes or extra pelvic causes. The pelvic causes are the commonest and include post-abortal sepsis, puerperal sepsis, acute pelvic inflammatory disease, perforation of uterus such as during curettage and irritant peritonitis following contamination by meconium spilled during the caesarean section.

The extra pelvic causes include appendicitis, diverticulitis, and ruptured gallbladder and perforated peptic ulcer. In postmenopausal women, a pelvic abscess is usually secondary to pathology in the intestinal tract such as appendicitis and rupture gallbladder or immunosuppressive conditions such as diabetes mellitus (2, 4, 5, 6).

Offending organisms in Murimas study were E.Coli which accounted for 20.3% staphylococcus aureus 11.9% and enterococci 10.1% (3).

Complete resolution is unlikely .If left uncared it may produce generalized peritonitis. In others, the abscess may burst into the rectum but rarely through the vagina because of tough endopelvic fascial sheath (2).

Clinical presentation is varied. The patient may be asymptomatic or symptomatic. Symptoms include fever with chills and rigor, rectal tenesmus, lower abdominal pain and urinary symptoms such as retention. Examination may reveal a flushed anxious face, tachycardia, tenderness and rigidity in lower abdomen and tender, soft, irregular suprapubic mass resonant on percussion. Pelvic examination reveals a hot and tender vagina, uterus is pushed anteriorly and cervical motion tenderness is present. A boggy, fluctuant bulging, tender mass is felt in the pouch of Douglas. Rectal examination precisely defines the mass (2, 5). Our patient had a tender lower abdominal mass and pouch of Douglas was bulging.

The differential diagnosis for pelvic abscess include: tubo-ovarian abscess, appendicular abscess, ectopic pregnancy, ovarian neoplasm, infected uterine fibroids, pelvic endometriosis and diverticulitis with perforation (2)

Investigations include full blood counts which show leucocytosis with increased polymorphs. Bacteriological study including both aerobic and anaerobic cultures from swabs taken from high vagina, endocervical canal and from the pus should be obtained. Pelvic ultrasonography reveals accumulation of fluid in the pouch of Douglas (2, 4, 5).

Haemogram in our patient revealed leucocytosis and the abscess was evident in the ultrasound.

The initial treatment consists of supportive measures such as intravenous fluids, nasogastric tube suction, blood transfusion analgesics and broad spectrum parenteral antibiotics(2, 4, 5, 6). In Murima,s study augumentin, ceftazidime and gentamicin had good sensitivity towards common micro organisms implicated in the aetiology of pelvic abscess while resistance to tetracycline and cotrimoxazole was noted (3). Good response is judged by absence or declining fever, decrease in white blood cell count by at least 3000 per mm<sup>3</sup> and improved symptomatology (6,7)

In cases where the diagnosis is in doubt or when a ruptured abscess is suspected then surgical intervention is needed (5, 6). Posterior colpotomy is restricted to cases where diagnosis is certain and the abscess is in the midline (2). The loculi should be broken with finger. Percutaneous drainage under ultrasonographic or CT guidance can also be done. Laparotomy is indicated when the diagnosis is uncertain or incases with spreading peritonitis (2, 4, 5, 6, 7). The pus should be sent for culture and drug sensitivity. Pus swab revealed E.coli sensitive to ceftriaxone and augumentin.

Laparoscopic drainage is not advised because it may not be possible to drain the entire abscess cavity and there is risk of bacteria (pus) spilling to the rest of the pelvis (2, 6, 7).

In patients with recurrent pelvic abscess and are past the reproductive age or have desired family size or whose pelvic organs damage is too expansive for future fertility a total abdominal hysterectomy may be recommended (7)

**REFERENCES**

1. Tindall V.R.Jeffcoates Principles of Gynaecology 5 th Ed.1987; Butterworth and company, London.26:260
2. Dutta DC. Infection of the individual pelvic organs. In: Textbook of Gynecology, New central, Publications, 2001; pg 165-167.
3. Murima J. 5 Year Review of Pelvic Abscess as seen at Kenyatta National Hospital. . MMed Thesis, University of Nairobi, 2003
4. Allan HD, Martin LP. Sexually transmitted Diseases and Pelvic infections: In: Current Obstetric and Gynecologic Diagnosis and Treatment 9th edition, McGraw Hill companies, 2003: 73-78.
5. Thomas M.D. Joseph A.H. Pelvic infections. In Novak's Gynaecology, 13 th edition; William and Wilkins, Philadelphia, 2002 page 431-459.
6. Martens M.G. Pelvic Inflammatory Diseases. In: Te Lindes operative Gynaecology 8 th edition; Lippincott-Raven.Philladelphia, 1997, p657-685.
7. Maurice K. Bewes P.Cairns J .The surgery of sepsis. In: Primary Surgery: on trauma, vol 1, Oxford Medical Publication 1990; pg 161-178.



**Physical examination**

She was middle aged lady in good general condition. She was not pale and had no lymphadenopathy. Her vital signs were normal.

**Systemic examination**

Respiratory, cardiovascular, central nervous and the abdomen were examined and found to be normal.

**Pelvic examination**

She had normal external genitalia. Speculum examination showed normal vaginal walls and ecto cervix.

On digital examination the cervix was smooth with no palpable masses. The uterus was normal size and the adnexa and pouch of Douglas were normal. There was no blood in the examining finger.

**Investigations**

Pap smear- reported as High Grade Squamous intra epithelial lesion with koilocytosis (HSIL)

Colposcopy revealed mosaicism and biopsy taken from iodine negative areas. Histopathology showed severe dysplasia in keeping with CIS. HPV positive

HIV test- positive.

CD4 cell count done 3 months ago 215.

Haemoglobin-12.7g%, Urea and Electrolytes- $\text{Na}^+$ -136mmol/l, $\text{K}^+$ -4mmol/l,BUN-3.2mmol/l

**Diagnosis**

A diagnosis of Carcinoma in-situ in HIV positive was made.

**MANAGEMENT**

The modes of treatment were discussed and she opted for a total abdominal hysterectomy. She was prepared for the operation. Informed consent was obtained and two units of cross matched blood reserved. Simple hysterectomy was carried out. Her post operative period was uneventful. She was allowed home on 4th postoperative day to be followed up in the outpatient clinic.

### **Follow up**

She was reviewed one week after discharge. She didn't raise any complains and the wound was healing well. Histology of the specimen confirmed CIS the margins were tumour free. The uterine corpus was free of tumor. The patient was appraised of results .She was to continue follow up.

### **DISCUSSION**

H.A A 39 years old, HIV sero-positive, para3+0 who presented with history of having 3 consecutive HSIL pap smear report for which colposcopy and biopsy was done which showed CIS.

Cervical intraepithelial neoplasia, formerly called dysplasia refers to disordered growth of the epithelial lining of the cervix (1). It is a histopathological condition where part or whole thickness of cervical squamous cells is replaced by cells showing various degrees of atypia (2).

CIN is predominantly a disease of younger women. With the peak incidence of CIS being in women aged 25-35 years, about 15 years less than that of invasive carcinoma. The epidemiologic risk factors for cervical intraepithelial neoplasia (CIN) are similar to those for cervical cancer. Many of the risk factors are linked to sexual activity and exposure to sexually transmitted diseases. These include young age at first intercourse (<16 years), multiple sexual partners, multiparity and history of sexually transmitted diseases. Other risk factors include cigarette smoking, immunosuppression and long term oral contraceptive use (1, 2, 3). Our patient was 36 years old and was HIV sero-positive.

Human papillomavirus (HPV) is the prime aetiologic factor in development of CIN and cervical cancer. It is found in over 80 % of all CIN lesions(1, 2, 3). HPV infection is extremely common with a prevalence of about 40% in women aged 20-29years of age, with a slow decline thereafter to a plateau of 5% in women aged 50 and old.<sup>1</sup> There are about 130 subtypes of HPV. The highly oncogenic subtypes are 16, 18, 31, 39, 45, 56, 58, 59 and 68 (1, 2, 3). There is increased incidence of pap smears suggesting herpes viral infection in women with cervical dysplasia but herpes has not been proved as a causative agent.<sup>2</sup> Cigarette smoking and HPV infection have synergistic effects on the development of CIN. Cigarette smoke carcinogens have been found to accumulate locally in the cervical mucus. The incidence of CIN is increased by HIV infection .This is attributed to increasing risk of de novo HPV infection and persistent HPV infection (1). There were koilocytes reported in the Pap smear indicative of HPV infection.

The cervix is composed of columnar epithelium, which lines the endocervical canal, and squamous epithelium, which covers the ectocervix. The point at which they meet is called the squamocolumnar junction (SCJ) (3).

The squamocolumnar junction rarely remains restricted to the external os. Instead, it is a dynamic point that changes in response to puberty, pregnancy, menopause, and hormonal stimulation. In neonates, the squamocolumnar junction is located on the ectocervix. At menarche, the production of oestrogen causes the vaginal epithelium to fill with glycogen. Lactobacilli act on the glycogen to produce lactic acid that lowers the vaginal pH. This in turn stimulates the subcolumnar reserve cells to undergo metaplasia. The term metaplasia is defined as the transformation of one mature cell type to another (3).

Metaplasia advances from the original squamocolumnar junction inward, toward the external os, and over the columnar villi. This establishes an area called the transformation zone (TZ). The TZ extends from the original squamocolumnar junction to the physiologically active squamocolumnar junction. Histologically the original squamous epithelium of the ectocervix has four layers namely the basal layer, the parabasal layer, intermediate layer and superficial layer. Cervical neoplasia almost invariably originates within the transformation zone is most likely to begin either during menarche or after pregnancy, when metaplasia is most active due to high oestrogenic state which lowers vaginal PH. The metaplastic cells have the potential to undergo atypical transformation by trauma or infection (2, 3).

On cytological examination, the dysplastic cell is characterized by anaplasia, an increased nuclear: cytoplasmic ratio, hyperchromatism, multinucleation and abnormalities in differentiation. Histopathologic examination show anaplastic and hyperchromatic cells with loss of polarity and abnormal and increased mitotic figures (1).

There are usually no symptoms or signs of CIN and the diagnosis is most often based on biopsy findings following an abnormal routine cervical cytology smear (1).

Exfoliative cytology (Pap smear) has become the gold standard for screening (2). False-negative and false-positive results occur. In clinical practice the false negative rate is about 15 to 20 percent. The sensitivity of conventional cytologic testing in detecting cervical cancer precursor lesions is 51 % with a specificity of 98% (3, 4). Liquid based cytology has increased sensitivity of 60%. It is considered

better because it picks up more atypical cells and infection because results are not obscured by blood. Repeat liquid cytology increases sensitivity to 85% (5). Pap smear classification system has evolved over the years. In 1988, the National Cancer Institute (NCI) sponsored a workshop to standardize Pap smear reporting. This came up with the Bethesda system for cytologic reporting which was revised in 2001 (3). In this system, potentially premalignant squamous lesions fall into three categories: atypical squamous cells (ASC), low-grade squamous intra-epithelial lesions (LSIL), and high-grade squamous intra-epithelial lesions (HSIL). The ASC category is subdivided into two categories: those of unknown significance (ASC-US) and those in which high-grade lesions must be excluded (ASC-H). Low-grade squamous intra-epithelial lesions include CIN 1 (mild dysplasia) and the changes of HPV, termed koilocytic atypia. HSIL includes CIN 2 and CIN 3 (moderate dysplasia, severe dysplasia, and carcinoma insitu) (3).

In our patient pap smear was reported as High grade Squamous Lesion (HSIL).

According to the American college of Obstetrician s and gynaecologists, all women who have been sexually active or reached age 18 should have a pelvic and cytologic examination at least once a year. After three or more consecutive, satisfactory, normal annual examinations, the screening interval may be extended in selected low risk patients. <sup>1</sup> Women who are age 70 and older with an intact cervix and who have had three or more documented, consecutive, technically satisfactory normal cervical cytology tests, and no abnormal cytology tests within the 10-year period prior to age 70 may elect to cease cervical cancer screening (6).

Visual inspection of cervix with acetic acid (VIAA) may be used in areas where Pap smear is not available. The application of acetic acid coagulates the proteins of the nucleus and cytoplasm and makes the proteins opaque and white. It's inexpensive, widely applicable and easy to learn method with a sensitivity of 82%. Women with acetowhite lesions are considered for colposcopic evaluation (2, 3).

Due to the low sensitivity of Pap smear as a screening test (51%), there is need to apply a diagnostic test for abnormal Pap smears. Literally translated, colposcopy means to look into the vagina (i.e., colpo means vagina, scope means to look). Colposcopy was first described by Hans Hinselman of Germany in 1925. Colposcopy is the primary technique for the evaluation of an abnormal cervical cytology. The colposcopic examination is usually under magnification of 5-15 to identify those abnormal cervical, vaginal, vulva and anal areas areas that require biopsy (1, 2, 3).

After excess mucus is gently wiped away from the cervix and a Pap smear has been taken, 3% acetic acid is applied and examination performed. Satisfactory colposcopic examination includes visualization of the original squamous epithelium, the transformation zone and the columnar epithelium of the endocervical canal (1, 3).

Indications for colposcopy are:-abnormal cervical cytology smear or HPV testing, abnormal or suspicious looking cervix and postcoital bleeding even if the smear is negative.

Abnormal colposcopic findings indicative of dysplasia and carcinoma insitu are those of (1, 2, 3).

Leukoplakia-white, thickened epithelium prior to application of stain.

- Acetowhite epithelium-epithelium turning white following application of acetic acid
- Mosaicism-capillaries encircling polygonal shaped blocks of acetowhite epithelial cells
- Punctuation –dilated capillaries which appear on the surface as collection of dots.
- Atypical blood vessels- corkscrew, comma shaped spaghetti like, vessels with irregular diameter and branching .Atypical vessels are suggestive of invasive carcinoma.
- Irregular surface contour with ulceration and friability

Our patient had mosaicism and punctations on colposcopic examination.Histological evaluation of the specimen revealed severe dysplasia and Carcinoma insitu.

Only when the entire transformation zone can be seen and if biopsies are taken from the most abnormal areas is the examination considered technically satisfactory. If the transformation zone extends into the endocervical canal above the examiner's vision, the colposcopic examination is termed unsatisfactory and diagnostic conization or loop electrosurgical excision procedure (LEEP) is performed. If the colposcopic examination is technically satisfactory, biopsy specimens are taken of the most abnormal areas (3, 7).

Evaluation of the non visualized portion of the endocervical canal by endocervical curettage (ECC) should be performed in cases where colposcopy is unsatisfactory, where the lesion is extending into the endocervical canal or where the colposcopic impression does not explain the cervical cytology finding (1). Endocervical curettage is mandatory whether or not the entire transformation zone can be seen (2).

histologically evaluated lesions are characterized using the CIN nomenclature as follows: 1 CIN  
 I. Mild dysplasia-disordered growth of the lower third of the epithelial lining  
 CIN II. Moderate dysplasia-Abnormal maturation of two thirds of the lining  
 CIN III. Severe dysplasia-Disordered growth of more than two-thirds of the epithelial thickness with carcinoma in situ representing full thickness dysmaturity. Diagnosis of CIN is only established by cervical biopsy.

Colposcopic directed biopsy was done in our patient and histopathological examination showed severe dysplasia.

The management of abnormal pap smears is as follows (3, 8): -

*Atypical squamous cells (ASC):* A programme of repeat cervical cytological testing after 4-6 months, colposcopy, or DNA testing for high-risk types of HPV are all acceptable methods of testing women with ASC-US. The recommended management of women with ASC-H is referral for colposcopic evaluation.

*Low-grade squamous Intra-epithelial Lesions (LSIL):* Repeat pap smear after 6 months is recommended. Colposcopy is the recommended after more than 2 consecutive LSIL reports.

*High-grade Squamous Intra-epithelial Lesions (HSIL):* Colposcopy with endocervical assessment is the recommended management of women with HSIL.

Management of CIN depend on the age of the patient, desire for reproduction, risk factors present, degree of dysplasia and facilities available for follow up such as colposcopy and/or cytology.<sup>2</sup> CIN is mainly a disease of young women in their reproductive years; hence treatment should be by conservative procedures that preserve the reproductive function. This can be accomplished through ablative (destructive) techniques (cryotherapy, cold coagulation, electrodiathermy and carbon dioxide Laser) or excision procedures (LEEP or cone biopsy) (1, 2, 3).

Because most cases of CIN 1 spontaneously regress (57%) without therapy, many experts advocate follow-up without treatment if the colposcopic examination is satisfactory. If lesions progress during follow-up or persist at 2 years, ablative treatment should be performed. The preferred treatment for patients with biopsy-confirmed CIN 1 and an unsatisfactory colposcopic examination is a diagnostic

excisional procedure (i.e. LEEP, laser conization, or cold-knife conization) (9). She was counselled on the need of close followup after the LEEP and instead opted for a hysterectomy.

Management decisions in women with biopsy-confirmed CIN 2, 3 are determined by whether the colposcopic examination is classified as satisfactory or unsatisfactory. Both excision and ablation of the transformation zone are acceptable for women with biopsy-confirmed CIN 2, 3 and a satisfactory colposcopy. However, in patients with recurrent CIN 2, 3, excisional modalities are preferred. A diagnostic excisional procedure is recommended for women with biopsy-confirmed CIN-2, 3 and unsatisfactory colposcopy (9).

Hysterectomy is currently considered too radical for treatment of CIN. However, there are situations in which hysterectomy remains a valid and appropriate method of treatment of CIN, these include microinvasion, CIN3 at limits of conization specimen, poor compliance with follow-up and other gynaecological problems requiring hysterectomy such as fibroids, uterine prolapse, endometriosis and pelvic inflammatory disease (3). Our patient opted for hysterectomy because of her completed family size and her sero-reactivity status.

The recurrence rate is about 3-5 per cent and development of invasive carcinoma ranges between 0.1-0.4 per cent. Therefore after treatment of CIN 2, 3, follow-up using either cervical cytology or a combination of cervical cytology and colposcopy at 4- 6 month intervals until at least 3 cytologic results are "negative for squamous intraepithelial lesion or malignancy" is recommended. Annual cytology follow-up is recommended thereafter (2, 9).

HIV is thought to increase oncogenic potential of HPV hence rapid progression to severe dysplasia and cancer of the cervix. HAART reduces recurrence of dysplasia and progression to cancer of the cervix. Following treatment the risk of recurrent CIN is high, especially in the immunocompromised patients. Recurrence rates may reach 80 percent within 3 years (1). Our patient was screened and found to be sero-positive. She was on HAART.

In pregnancy colposcopy is challenging because pregnancy induced changes in the cervical epithelium mimic those of cervical dysplasia. Although the gravid cervix is more vascular, directed ectocervical biopsies can be performed safely with minimal increase in the risk of significant bleeding. Endocervical curettage is not performed because of the potential risk of abortion and infection (1).

Preventive measures for CIN include delaying sexual exposure until the cervical epithelium, especially in the transformation zone, has attained physiological maturity, adequate treatment of any vaginal infection, use of condom especially during early sexual life, maintenance of penile hygiene as it may be a reservoir for high risk HPV, reducing or quitting smoking and HPV vaccine (2).

## REFERENCES

1. Holschneider CH. Premalignant and malignant disorders of the uterine cervix. In: DeCherney A.L., Nathan L, eds. Current Obstetric and Gynaecologic Diagnosis and Treatment, 9<sup>th</sup> edition, New York; McGraw-Hill, 2003:894-915
2. Dutta DC: Premalignant lesions. In: Textbook of Gynaecology, 6th edition, 2004, New Central Book Agency (P) Ltd, Calcutta, India Pg 298-- 307.
3. Hatch KD, Berek J.S. Intraepithelial disease of the cervix, vagina, and vulva. In: Berek J.S, ed. Novak's Gynaecology, 13<sup>th</sup> edition, Philadelphia; Lippincott Williams & Wilkins, 2002: 471-505
4. Garcia AA, Bi Jia. Cervical cancer. <http://www.emedicine.com/int/2002>.
5. Virochana K. Cervical Intraepithelial Neoplasia: Management Protocols. Obs & Gynae. Today. Vol.X No.4 April 2005.
6. Ndavi PM. Cervical cytology in a rural population. MMed Thesis University of Nairobi, 1986 ;417-450
7. Chamia JM. The validity of Pap smear as a means of screening for cervical intraepithelial neoplasia (CIN) as compared to colposcopic and histologic findings. MMed Thesis, University of Nairobi, 1986;322-352
8. Kibugucchy W. Mbugua SCT, Kigundu S, Mati JK. Carcinoma of the cervix and cervical intraepithelial neoplasia (CIN): Screening for a high risk group. J Obstet Gynecol East Central Africa 1988; 4:29-35
9. Stenchever M.A, Droegemueller W., Herbst A.L, Mishell DR, (Eds). Premalignant Lesions of the Cervix. In: Comprehensive Gynaecology, 4<sup>th</sup> edition. St. Louis; Mosby; 2001:857-888



**Physical examination****General examination**

She was elderly woman in fair general condition. She was not pale, not jaundiced and she had no lymphadenopathy. Her vital signs were; temperature 37.0, pulse rate 78/min, respiratory rate 16/min and blood pressure 110/70 mmhg.

**Abdominal examination**

The abdomen was scaphoid and moving with respiration. It was soft and no masses were palpable.

**Respiratory, cardiovascular and CNS**

Were examined and found to be normal.

**Pelvic examination**

The vulva was atrophic with scanty pubic hair. There was protrusion of anterior vaginal wall extending to the hymenal ring. The cervix and uterus were protruding beyond the hymenal ring. There was no ulcer seen. There was also protrusion of posterior vaginal wall extending to the hymenal ring.

**Diagnosis**

A diagnosis of 3<sup>rd</sup> degree uterine prolapse (complete procidentia) with cystocele, rectocele diabetes, hypertension in a postmenopausal woman was made.

**Investigation**

Haemogram: Haemoglobin-12.0g/dl, WBC- $6 \times 10^9/l$ , Platelets  $315 \times 10^9/l$

Urea and electrolytes: Na-142mmol/l, K-4.5 mmol/l, Urea 4.9mmol/l, creatinine 91  $\mu$ mol/l.

Chest X-Ray: normal cardiac shadow

ECG: sinus rhythm

Random blood sugar: 5.2 mmol/l

## MANAGEMENT

She gave informed consent to undergo surgery. Blood was taken for grouping and cross matching. She was starved from midnight and premedicated with intramuscular pethidine 50 mg and atropine 0.6mg half hour before theatre.

In theatre she was positioned supine and put under general anaesthesia. She was put on lithotomy and vulvovaginal toilet done. She was then draped and catheterized. Examination under anaesthesia was done and confirmed earlier findings.

The labia majora were stitched to the medial aspect of the thighs for adequate exposure. The cervix was grasped with a tenaculum and semi lunar incision made at the junction of the vaginal mucosa just below the bladder attachment. Blunt dissection was done to push the bladder away upto the level of vesico-uterine pouch. A similar procedure was done posteriorly and rectum pushed away. The uterosacral ligaments and cardinal ligaments were identified, clamped, divided and ligated bilaterally. The uterine arteries were identified clamped, divided and ligated bilaterally. The peritoneal cavity was then entered both anteriorly at the vesicouterine pouch and posteriorly at the pouch of Douglas. Traction was applied to the cervix and the broad ligaments with fallopian tube, ovarian vessels and round ligaments identified, clamped, divided and ligated sparing the ovaries. The uterus was then delivered. Marcus stitches both internal and external were then inserted to be tied after closure of the vault. The peritoneum was then closed with a purse string.. The angles of the vaginal vault were closed with vicryl no1 and suspended with the pedicles of the cardinal and uterosacral ligament.

A midline incision was then made on the anterior vaginal wall and the flaps of the vaginal walls dissected laterally and the paraurethral and paravesical fascia was mobilized by blunt dissection. Placation on the lateral margin of the paravesical fascia and tied at the miline. Care was being taken not to injure the urethra by use of a metal catheter in the urethra. Excess vaginal mucosa was excised and margins approximate in the midline. The vault was then closed. Posterior colporhaphy was also done

The vaginal canal was cleansed with betadine and packed with gauze soaked in betadine which was removed after 24 hours. Continuous urinary drainage was maintained for 48 hours. She was put on parenteral antibiotics and analgesics. She had uneventful postoperative period. She was discharged

home on the fourth postoperative day on oral medications to be seen in gynaecology clinic after 1 month.

### FOLLOW UP

She was seen in GOPC as scheduled. She did not have any complaints. Speculum examination showed good healing of the vaginal vault and anterior vaginal wall. There was no evidence of prolapse or urinary incontinence.

### DISCUSSION

S.H was a 76 year old Para 10+0 with relaxation of her pelvic support structures. This complicated with genital prolapse, cystocele and rectocele. Vaginal hysterectomy with anterior and posterior colporrhaphy was done. She did well postoperatively.

A prolapse is a downward or a forward displacement of one of the pelvic organs from its normal location (1). A variety of terms are used to describe genital prolapse in women namely:- (1, 2)

- A cystocele-a downward displacement of the bladder
- A cystourethrocele-a cystocele that includes the urethra
- A uterine prolapse-a descent of the uterus and cervix down the vaginal canal
- A rectocele-a protrusion of the rectum into the posterior vaginal wall
- An enterocele-herniation of the small bowel into the vaginal lumen.

The patient presented had uterine prolapse, cystocele and rectocele.

Pelvic organ prolapse commonly occurs in multiparous white women. It is uncommon in black women.<sup>2</sup> In a retrospective study covering the period 1974 to 1979, Mwalali found an incidence of 0.1% at Kenyatta National Hospital (3).

Relaxation of pelvic support occurs as result of child birth injuries to the pelvic support structures. Other risk factors are: obesity, asthma, chronic bronchitis, lifting of heavy objects, ascites, large uterine and ovarian tumors, diabetic neuropathy, sacral nerve disorders and congenital weakness e.g.

enhancers Danloss syndrome. It becomes more common after menopause because of weakening of the pelvic floor due to hypoestrogenic state (2, 4).

Two classifications are used to describe and document the severity of the pelvic organ prolapse. The most commonly used system is based on descend within the vaginal vault. The most dependent position of the pelvic organs during maximum straining is used to rate the degree of prolapse. For cystocele it is the anterior vaginal wall, for uterine prolapse is the cervix and for rectocele the saccular protrusion of rectovaginal wall.<sup>2</sup> The prolapse is classified as follows (1, 2).

- First degree-the most dependent position is halfway to the hymen.
- Second degree-the most dependent position descend to the hymen.
- Third degree-the most dependent position extends or protrudes beyond the hymen.

Based on this, our patient had a 2<sup>nd</sup> degree cystocele, 3rd degree uterine prolapse and 2<sup>nd</sup> degree rectocele.

The Pelvic Organ Prolapse Quantification (POP\_Q) system is a new method adopted in 1996 which employ objective measurements from fixed anatomic points. Prolapse in each segment is evaluated and measured relative to the hymen which is a fixed anatomic landmark. The anatomic position of the 6 defined points for measurements should be in centimeters above the hymen (negative number) or centimeters below the hymen (positive number). The plane at the level of the hymen is defined as zero (2, 4).

The POP-Q staging is as follows (1).

- Stage 0: no prolapse is demonstrated.
- Stage 1: the most distal portion of the prolapse is more than 1cm above the level of the hymen.
- Stage 11: the most distal portion of the prolapse is less than 1cm proximal or distal to the plane of the hymen.
- Stage 111: the most distal portion of the prolapse is more than 1 cm below the plane of the hymen but protrudes not further than 2 cm less than the total vaginal length in centimeters.
- Stage 1V: complete to near complete eversion of the vagina.

Based on this staging, our patient had Stage 11 cystocele, Stage 111 uterine prolapse and Stage 11 rectocele.

The diagnosis of pelvic organ prolapse is based on good history taking and physical examination. It may be asymptomatic or symptomatic. The most common symptom is a feeling of pressure or that something is coming outside the vagina worsened by standing, straining and worsen as the day goes by. It is relieved by lying down and is less noticeable in the morning. Also they may feel a bearing down sensation, backache worse as day goes by, purulent or bloodstained discharge when there is decubital ulceration. In case of cystocele there may be frequency of micturition, stress incontinence and difficulty in emptying the bladder. In case of rectocele there may be difficulty in emptying the rectum (1, 2, 5). In KNH, the commonest symptom was the feeling of something coming down which was present in 88.1% of patients (3). Our patient presented with a feeling of something coming outside the vagina. She also had frequency of micturition.

Physical examination is done both in the dorsal lithotomy position and when standing. The presence, type and extent of prolapse and presence of stress incontinence can be determined on Valsalva maneuver. Rectal examination is useful to demonstrate rectocele and to distinguish it from enterocele (5).

The differential diagnosis includes; cervical elongation, cervical and endometrial tumors prolapsing through a dilated cervix (e.g. pedunculated myoma or polyp), uterine inversion, urethral caruncle or fecal impaction (5).

Investigations include; hemogram, serum urea and electrolytes, urine culture and sensitivity, intravenous urogram in cases of proctitis and urothrometry in cases of urinary incontinence. Papanicolaou smear should also be done to rule out cervical malignancy.<sup>1,2</sup> These investigations were done in our patient except intravenous urogram and urothrometry. Urothrometry was not indicated as she was continent.

Complications of prolapse include; leukorrhoea, abnormal uterine bleeding, abortion, chronic decubitus ulcer, urinary tract infection, hydronephrosis, hemorrhoids and in rare occasions small bowel obstruction. (1, 2, 5).

Treatment depends on the severity of the symptoms. Asymptomatic patients require no treatment (1). Symptomatic prolapse can be treated either conservatively or by surgery depending on the individual. Conservative treatment is indicated in the following circumstances; in pregnancy,

immediately after pregnancy, when further childbearing is intended, when surgery is unsafe due to senility and medical diseases, to promote healing of a decubital ulcer prior to surgery and refusal of operation by the patient (5). Conservative treatment modalities are kegel exercises, weight reduction for the obese and use of vaginal ring pessary. Adverse effects of the vaginal pessary include; infection, foreign body vaginitis, ulceration, chronic irritation and vesicovaginal fistula (1, 2).

Postmenopausal women should be put on hormone replacement therapy or local estrogen cream to improve the tissue tone there by correcting atrophic vaginitis and this makes the pessary comfortable to wear (1).

Surgery is the ideal treatment of symptomatic uterine prolapse (1, 5). The aim is to correct all defects during same operation. It's generally carried out through the vaginal route. Vaginal hysterectomy is the operation of choice for postmenopausal women who are sexually active. It allows other vaginal surgery e.g. anterior and posterior colporrhaphy to be performed at the same time without need for a separate incision or for reposition the patient. The uterosacral and cardinal ligaments are reattached to the vaginal cuff to provide additional support (1, 2, 4). Our patient was menopausal and in good general condition hence surgery was recommended.

For patients who are poor surgical risk or those who require fertility the Manchester-Fothergill operation may be done. It involves amputation of the cervix, sewing of the cardinal ligaments to the anterior cervical stump and antero-posterior colporrhaphy. Lefort operation which involves partial vaginal closure may be performed for the elderly who are chronically ill and are not sexually active. Transabdominal approach is not recommended because it has more morbidity and is time consuming. Other operations which can be done are colpocleisis and colpectomy (2, 4).

Our patient was done transvaginal hysterectomy, anterior colporrhaphy and posterior colpoperineorrhaphy.

Complications of surgery include urinary tract infections, hemorrhage, pelvic pain thrombosis, recurrent prolapse, dyspareunia and stress incontinence (1, 4).

Our patient did not develop any of these complications.

Prevention is by prenatal, intrapartum and postnatal kegel exercises to strengthen the pelvic floor. Early and adequate episiotomy in second stage of labour and proper repair of perineal tears may help

prevent prolapse. Obesity, chronic cough, straining must be avoided and if present corrected. Estrogen replacement therapy for the menopausal woman may help maintain the tone and vitality of pelvic musculofascial tissues (1, 2, 5). There is an emerging and ongoing debate on the use of prophylactic cesarean delivery in prevention of pelvic organ prolapse (4).

REFERENCES

1. Wall, LL. Incontinence, prolapse and disorders of pelvic floor. Novaks textbook of Gynaecology. 13<sup>th</sup> ed. 2002; William and Wilkins, Philadelphia, 20:657.
2. Tarnay, Dorr. Relaxation of Pelvic support. In: De Cherney A.H., Nathan L. (eds). Current Obstetrics and Gynaecology Diagnosis and treatment. 9<sup>th</sup> edition. McGraw Hill 2003. 41:776-797.
3. Mwalali P.N. Retrospective study of genital prolapse at Kenyatta National Hospital. M. Med thesis, UoN. 1982.
4. Rock, J.A, Thomson J.D. Te Lindes operative Gynaecology, 9<sup>th</sup> Ed. 1997; Lippincott-Raven, 26:946-62.
5. Tindall V.R. Genital prolapse. Jeffcoates Principles of Gynaecology 5<sup>th</sup> Ed. 1987; Butterworth and company, London. 26:260.