

## **OBSTETRIC CASE 13**

### **SEVERE PRE-ECLAMPSIA – EMERGENCY CAESAREAN SECTION: LIVE BABY**

|                      |                        |                              |
|----------------------|------------------------|------------------------------|
| <b>Name:</b> N.N     | <b>Parity:</b> 3+0     | <b>Admission:</b> 18/04/2001 |
| <b>IPNo:</b> 0991759 | <b>LMP:</b> 10/09/2000 | <b>Delivery:</b> 15/05/2001  |
| <b>Age:</b> 31 years | <b>EDD:</b> 17/6/2001  | <b>Gestation:</b> 36 weeks   |

#### **Presenting complaints**

Patient was admitted through antenatal clinic (ANC) due to elevated blood pressure.

#### **History of presenting illness**

Patient was admitted from ANC due to high blood pressure of 160/120mmHg at 30 weeks gestation. She had been referred to Kenyatta National Hospital ANC from a private clinic with a diagnosis of PET at 21 weeks gestation. During her follow-up in the private clinic she had been started on Aldomet 250mg 8 hourly, Junior Asprin (75mg) 12 hourly and hydralazine 25mg 8 hourly, these drugs were continued in her ANC follow-up. On two occasions in her ANC follow-up she was noted to have proteinuria of +1 though the blood pressures were controlled. Prior to admission she had noted progressive swelling of legs and puffiness of the face. She had no headache, blurring of vision, dizziness or epigastric pain.

#### **Past obstetric and gynaecology history**

She was para 3+0 with no living child. Her first delivery was in 1998 by a hysterotomy to a macerated still birth at 6 months due to high blood pressure after failed induction of labour. The second delivery was in 1999 at 7 months gestation. She got a macerated still birth after she went into preterm labour and also high blood pressure in pregnancy. Her 3<sup>rd</sup> delivery was in 2000 at 34 weeks. This was by caesarean section due to high blood pressure. A live baby was delivered but succumbed on the 6<sup>th</sup> day of life. She was not hypertensive outside pregnancy. She attained her menarche at 14 years. Her

cycles were regular with flows lasting 3 days and comes after every 28 days. There was no history of contraceptive use.

### **Past medical and surgical history**

This was not significant.

### **Family and social history**

She was married, a primary school teacher staying with her husband in Kiambu. Her husband was also a teacher. She did not smoke cigarettes or take alcohol. There was no history of any chronic illness in the family.

### **Physical examination**

She was in fair general condition, not pale, not jaundiced, cyanosed or febrile. She had bipedal pitting oedema but no lymphadenopathy. The pulse rate was 82/minute good volume and regular. The blood pressure was 160/120mmHg, the respiratory rate was 20/minute.

The respiratory and central nervous systems were normal.

### **Abdominal examination**

The abdomen was noted to being moving with respiration, was uniformly distended and gravid and there was a sub-umbilical midline surgical scar noted. The liver and spleen were not palpable. The uterine size was 30 weeks, fetal lie was longitudinal with cephalic presentation. The head was five fifths above the pelvic brim. There were no contractions noted, The fetal heart rate was 136 beats per minute and regular.

### **Pelvic examination**

This was not done as there was no indication.

### **Diagnosis**



An impression of severe P.E.T, 2 previous scars and Bad Obtetric History at 30 weeks was made.

**Management**

Patient was admitted to labour ward’s acute room, for control of blood pressure. She was given a stat dose of 20mg hydrallazine as an intravenous bolus. She was started on Aldomet tablets 500mg 8 hourly and valium 1mg 12 hourly and was to be observed half hourly. She was observed for 12 hours and the blood pressure came down to 130-150mmHg systolic and 100-105mmHg diastolic. She was then transferred to the lying in wards for further investigations and management.

**Investigations**

|                       |   |              |          |                           |
|-----------------------|---|--------------|----------|---------------------------|
| Haemogram             | - | HB           | -        | 11.2gm/dl                 |
|                       |   | WBC          | -        | 6.7 x 10 <sup>9</sup> /l  |
|                       |   | Platelets    | -        | 247 x 10 <sup>9</sup> /l  |
|                       |   | RBC          | -        | 3.68 x 10 <sup>9</sup> /l |
| Urea and electrolytes | - | Na+          | -        | 126mmol/l                 |
|                       |   | K+           | -        | 4.32mmo/l                 |
|                       |   | Cl           | -        | 108mmol/l                 |
|                       |   | Creatinine   | -        | 0.6umol/l                 |
| Uric acid             | - | 6.2mg/d      |          |                           |
| VDRL                  | - | Negative     |          |                           |
| Blood group           | - | O Rhesus “D” | Positive |                           |
| Urinalysis            | - | Protein      | +++(+3)  |                           |
|                       | - | Sugar        | – Nil    |                           |
| HIV                   | - | Negative     |          |                           |

**Ultrasound**

- 23/4/2001 - a single viable intra uterine fetus in cephalic presentation at 31 weeks gestation.  
Estimated fetal weight (EFW) – 1761gm  
Biophysical profile score of 10/10
- 3/5/2001 - fetus in cephalic presentation. Normal fetal cardiac activity fetal biophysical profile score of 6/10.  
BPD - 85mm corresponding is 34 weeks  
FL - 63mm in 33 weeks + 4 days  
AC - 94mm in 33 weeks + 5 days  
Average gestation was 34 weeks.  
EFW - 2.108Kg
- 4/5/2001 - Repeat scan for umbilical artery Doppler flows  
- Doppler flow index was 0.615 which was normal for the gestation.  
- S/D ratio was 2.6, which was normal for the gestation.  
- Cerebral artery flow was checked and found to be within normal range.

#### **Stay in ward.**

Patient had been put on bed rest. She continued with anti-hypertensives. The blood pressures were not well controlled and this necessitated upward adjustments of Aldomet to 750mg six hourly, hydrallazine 50mg 8 hourly and Adalat 20mg 12 hourly. Despite the above drugs the blood pressure remained uncontrollable. It was therefore decided to monitor the patient and fetus closely and if possible to get the pregnancy to 34 weeks. On 3<sup>rd</sup> May 2001, the blood pressure was noted to be too high and therefore patient was started on I.V dexamethasone 12mg 12 hourly. An urgent ultrasound was also ordered. This scan showed a viable fetus at 34 weeks gestation with a biophysical

profile score of 6/10. Due to the poor biophysical profile score another scan was ordered to show the umbilical Doppler flows. This was shown to be within normal limits. On 4<sup>th</sup> May 2001 a decision to deliver the patient was made and patient was taken to labour ward for emergency caesarean section. This section was performed on 5<sup>th</sup> May 2001. A live male infant with Apgar score of 6 at one minute, 7 at five minutes and 8 at ten minutes, and weighing 1700gm was delivered. The placenta was noted to have areas of calcification. The baby was admitted to NBU due to prematurity and managed there until he had attained a weight of 2000gm. He was then discharged to join the mother.

### **Post operative period**

Post operatively, the general condition of the patient remained satisfactory. She was on intravenous fluids of normal saline and 5% dextrose for 24 hours, intramuscular pethidine 100mg 8 hourly for analgesia. Her input of fluids was well balanced. Post operative blood pressure ranged between 150/90 –150/100mmHg. She was maintained on Aldomet 500mg 8 hourly and hydralazine 25 mg 8 hourly. The urine proteinuria gradually reduced to nil. She was covered with intravenous antibiotics Crystalline penicillin 2 mega units 6 hourly and gentamycin 80mg 8 hourly. She did well post operatively and was discharged to the mother's hostel on 10<sup>th</sup> Post operative day after removal of stitches. The incision site was well healed. She was scheduled for review in the postnatal clinic at two weeks.

### **Follow up**

She was seen in the postnatal clinic after 2 weeks and found to be in good general condition. She had no complaints. She was not pale and the wound had healed well. Her blood pressure was 150/100mmHg. Her breasts were active and not engorged and she was still breast feeding. Lochia loss was normal. She was advised to continue with Aldomet 500mg 8 hourly and to be reviewed after 4 weeks. She was counseled on the recurrence of the problem and therefore advised to seek antenatal care early.

She did not turn up for her appointment at 6 weeks post-delivery.

## DISCUSSION

A 31 year old para 3+0 with no living child , admitted with severe P.E.T and delivered at 34 weeks gestation is presented.

Pre-eclampsia is a major cause of maternal and perinatal morbidity and mortality world wide (1). The patient presented had had 2 previous abdominal deliveries. In one occasion she had IUFD at 6 months due to P.E.T. The other she had a premature baby delivered at 7 months also due to severe P.E.T. The second pregnancy ended as a spontaneous vaginal delivery at 5 months while she still had P.E.T.

Pre-eclampsia is a triad of oedema, hypertension and proteinuria occurring primarily in nulliparas after the 20<sup>th</sup> gestational week and most frequently near term (2). Hypertension is defined as a diastolic pressure of at least 90mmHg or systolic blood pressure of 140mmHg or a rise of diastolic blood pressure by 15mmHg or rise in systolic blood pressure by 30mmHg . These readings should be taken in two occasions at least 6 hours apart. Pre-eclampsia occurs in about 8% of the general population. The incidence varies with geographical location. The predisposing factors are, multi-parity, black race, maternal age below 20 or over 35 years, low socio-economic status, multiple gestation hydatidiform mole, non immune fetal hydrops, diabetes, polyhydramnios, chronic hypertension and underlying renal disease (2). In Kenya the incidence was reported to be 1.5% to 9% of all pregnancies by Mati (3). Kibaru found a prevalence of 5.6 per 1000 deliveries at Kenyatta National Hospital (4).

There are three levels of pathology of pre-eclampsia. The primary pathology must be placental or of the placental bed (Redman 1991), (5). Because the condition is pregnancy specific, it always resolves after delivery but does not require the presence of the fetus as it can develop with a hydatidiform mole (6). The secondary pathology comprises the sub critical signs of the placental problem, both maternal and fetal. These disturbances can progress to decompensation of one system or another leading to the tertiary pathology (7).



Etiology of pre-eclampsia is unknown and only theories have been advanced. Until recently, the best explanation for the vasospasm of pre-eclampsia was the hypothesis that it resulted from an imbalance in the production of the prostanoids prostacycline and thromboxane (8). Pre-eclampsia involves a relative deficiency of vasodilating prostacycline and a relative excess vasoconstricting thromboxane. Circulating prostacycline is probable mainly derived from the vessel wall whereas the principal source of circulating thromboxane are platelets.

Pre-eclampsia can be classified into mild and severe. In severe pre-eclampsia the blood pressure is greater than 160mmHg systolic or 110mmHg diastolic recorded on 2 occasions at least 6 hours apart, proteinuria exceeding 5g in a 24 hour period or 3 – 4+ on dip stick testing, oliguria (<500ml) in 24 hours period, cerebral or visual disturbances epigastric pains and pulmonary oedema or cyanosis (2). The patient presented had protein +3, blood pressure spiking to 160/110mmHg but she never had blurring of vision, nor epigastric pain.

Up to 10% of patients with severe pre-eclampsia end up developing the HELLP syndrome which is characterized by haemolysis, elevated liver enzymes, and low platelets (2,7). The patient presented was found not have this syndrome.

The principles of management are early diagnosis, early admission to hospital, optimal timing of delivery to pre-empt complications (7). Our patient was admitted at 30 weeks gestation and delivered at 34 weeks due to the uncontrollable blood pressure and poor biophysical profile score.

Multi-parity is the best way of avoiding pre-eclampsia but this cannot be prescribed (7). Calcium and other dietary supplements e.g. zinc appear to be more effective in the prevention of pre-eclampsia. It has been postulated that calcium deficiency predisposes to pre-eclampsia, (9,10). Vitamin E, of which blood concentration is significantly reduced in pre-eclampsia may be used to reduce the onset of pre-eclampsia. As an anti-oxidant it may help prevent the formation of free radicals,

which could initiate endothelia or other forms of tissue damage (11, 12). The use of anti-platelet agents in particular low dose aspirin is important in preventing pre-eclampsia (13). The patient presented was put on junior aspirin at her early gestation.

It is very important to monitor the fetal well-being, hence serial abdominal circumference measurements should be done every 2 weeks. In severe pre-eclampsia it is important to monitor the fetus on a daily basis. The methods of monitoring includes fetal heart rate analysis, fetal tick charts, ultrasound biophysical profiles and Doppler wave form analysis of the fetal circulation. Both biophysical profiles and Doppler flow waves were done in the case presented.

Once the diagnosis of pre-eclampsia has been made, definitive therapy in the form of delivery is the desired goal, since it is the only cure for the disease (14). The decision for immediate delivery versus expectant management is usually dependent on one or more of the following: the severity of the disease process, fetal condition, fetal gestational age, maternal condition, and Bishop score (14).

Severe pre-eclampsia is associated with many maternal complications e.g. Abruption placenta, thrombocytopenia, HELLP syndrome, eclampsia, disseminated intra-vascular coagulation and acute renal failure (14). Although the patient presented had severe pre-eclampsia she never got any of these complications.

Long et al (15) reported the pregnancy outcome in 2,434 singleton, pregnancies with pre-eclampsia during a 7 year period. They found that patient with preterm pre-eclampsia had a worse perinatal outcome than those with pre-eclampsia at 37 weeks or later. Our patient had a preterm baby of weight 1700gm and was admitted to the NBU with RDS. The baby had improved well by the time of the mother was discharged.

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## **OBSTETRIC CASE 14**

### **BREECH PRESENTATION: EMERGENCY CAESAREAN SECTION – LIVE BABY**

|                       |                        |                              |
|-----------------------|------------------------|------------------------------|
| <b>Name:</b> J.K      | <b>Parity:</b> 0+0     | <b>Admission:</b> 24/04/2001 |
| <b>IP No:</b> 0920663 | <b>LMP:</b> 03/08/2000 | <b>Delivery:</b> 24/04/2001  |
| <b>Age:</b> 22 years  | <b>EDD:</b> 10/05/2001 | <b>Discharge:</b> 28/04/2001 |

#### **Presenting history**

The patient was admitted from home with a history of labour pains for 8 hours. She had not drained liquor.

#### **Past medical history**

This was insignificant

#### **Obstetric and Gynaecological history**

She was primigravida. Her last menstrual period was on 3/8/2000. Her expected date of delivery was 10/05/2001. She was at a gestation of 37 weeks 5 days. She had attended antenatal clinic at a private facility in Dagoretti. She had begun at 5 months gestation. Antenatal period had been uneventful and profiles were:-

|               |            |
|---------------|------------|
| Haemoglobin - | 12.1gl/dl, |
| VDRL -        | Negative   |
| Blood group - | O positive |

#### **Family and social history**

She was a housewife married to a businessman. They lived at Dagoretti. She neither smoked cigarettes nor drank alcohol.

#### **Physical examination**



She was a young lady in good general condition, not pale and was febrile. There was no oedema, vital signs were: Blood pressure 100/60mmHg, pulse 76/minute, and respirations 22/minute, temperature 36.70C. The respiratory, cardiovascular and central nervous systems were normal.

### **Abdominal examination**

The fundal height was term, lie longitudinal and presentation breech. She had 2,10 contractions, each lasting 20 seconds. Fetal heart rate was heard and regular, at 144 beats per minute.

### **Vaginal examination**

Her external genitalia were normal. Her cervix was 4cm dilated and membranes were intact. The breech was felt. The cord was not felt. She had an adequate pelvis.

### **Diagnosis and management**

A diagnosis of breech presentation in a primigravida in active labour was made. The plan was to deliver her by emergency caesarean section. Informed consent was obtained from her. Blood was taken for grouping and cross matching and 1 Unit of compatible blood availed. An infusion of 5% dextrose was started and she was prepared for caesarean section as described in the introduction. The outcome of delivery was a live male baby who had an Apgar score of 9 at 1 minute and 10 at 5 minutes. He weighed 3600grams. The placenta was delivered, found normal and weighed 610g.

### **Post operative recovery**

She was mobilized and started on oral sips on the 1<sup>st</sup> post operative day. On the 3<sup>rd</sup> day, the wound was inspected and found clean and dry. The check haemoglobin was 11.1g/dl. Since the stitch used on the skin was subcuticular vicryl, she was allowed to go home with advice to present to the postnatal clinic after 6 weeks.

### **Postnatal clinic review**

The mother had no complaints. The baby was breastfeeding well and had gained weight appropriately. The wound was healed and her uterus well involuted. She was counseled on contraception and chose Norplant implant. She was referred to the family welfare clinic for insertion.

## DISCUSSION

The patient presented was a 23 year old primigravida at 34 weeks gestation, admitted in labour. Further evaluation showed that her fetus was in breech presentation. She was delivered by emergency caesarean section with good outcome.

Breech presentation occurs when the fetal pelvis or lower extremities engage in the maternal pelvis (1). It is common in preterm deliveries. Most fetuses turn spontaneously to cephalic presentation before onset of labour. So that breech presentation persists in only 3-4% of term singleton deliveries (2). Njuki in 1979 found an incidence of 3.5% at Kenyatta National Hospital (KNH) (3).

3 types of breech are recognized :

- Frank or extended breech – the lower limbs are fully flexed at the hips and extended at the knees.
- Flexed or completed breech – the fetal hips and knees are flexed with feet closely applied to the posterior aspect of the thighs.
- Footling or incomplete breech - one or both feet of the fetus present (4).

Frank breech presentation is the commonest, accounting for 38% of all singleton breech infants weighing above 2500g of birth and up to 51% of all breech deliveries below 2500g. Complete breech accounts for 12% of those over 2500g (1). At a gestation of 20-25 weeks, 30-40% of singleton pregnancies present by the breech. This falls to 15% at 32 weeks and 3-4% at term (4).

Aetiology of breech presentation is varied. The commonest is prematurity, accounting for up to 25% of breech presentations. Conditions preventing spontaneous

version in patients who reach term may be incriminated these include uterine anomalies e.g. Septate, bicornuate at multiple pregnancy, /polyhydramnios (2). The cause of the breech presentation in our patient was not obvious. Other causes include pelvic tumours and fetal congenital malformations.

Diagnosis of breech presentation may be made on physical examination. Palpation reveals a round hard ballotable head occupying the uterine fundus. The narrow, softer breech may be mobile over the pelvic brim or be engaged in the pelvis. The fetal heart is best heard above the umbilicus. On vaginal examination in labour the presenting part is softer than the head and may be irregular in outline. If membranes have ruptured, finger introduced into the fetal anus may have meconium.

The breech may be differentiated from face presentation in that on vaginal examination, the ischial tuberosities and anus form a straight line whereas the malar prominences and mouth form the corners of a triangle in breech presentation (4). Before onset of labour diagnosis may be confirmed by ultrasound scan which also excludes placenta praevia multiple pregnancy and some fetal and uterine abnormalities. X-rays will also exclude multiple pregnancy and major skeletal abnormalities. Both methods will show hydrocephalus and usually rule out major degrees of spine bifida (4). Palpations and vaginal examination made the diagnosis in our patient.

The decision on mode of delivery is individualized. Vaginal delivery is allowed when there is the following:-

- Frank breech presentation
- Gestation of 34 weeks or more
- Estimated fetal weight of 2500-3500gm
- Adequate maternal pelvis
- No maternal indication for caesarean section
- Pre-viable fetus
- Documented lethal fetal congenital abnormalities



- Presentation of mother is advanced labour with no fetal or material distress.

Caesarean section is performed if:-

- Fetal weight exceeds 3500gm
- The pelvis is borderline or contracted
- The fetal head is deflexed
- The presenting part is unengaged
- The fetus is at gestation of 25-34 weeks
- The breech is footling
- The mother is primigravida, had infertility or bad obstetric history
- There fetal distress or any other contraindication to vaginal delivery (4).

The practice at KNH is to deliver most breeches by caesarean section. Babies delivered by caesarean section in breech presentation had no perinatal mortality against an overall perinatal mortality of 76.9 per 1000 (3). Besides, the number of skilled operators with ability to safely deliver fetuses presenting by the breech continue to dwindle as training of new ones is hampered by medicolegal considerations (5).

External cephalic version (ECV) can reduce the expected 3-4% of term breech deliveries by half. Risks of version include maternal death, placental abruption, uterine rupture, fetomaternal hemorrhage with rhesus iso- immunisation preterm labour, fetal distress and fetal death (6). Subcutaneous administration of 0.25mg terbutaline increased the success rate of ECV from 27% 52% (7).

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## **OBSTETRIC CASE 15**

### **CARDIAC DISEASE IN PREGNANCY**

|                       |                       |                             |
|-----------------------|-----------------------|-----------------------------|
| <b>Name:</b> A.A.     | <b>Parity:</b> 0+0    | <b>Admission:</b> 2/5/2001  |
| <b>IP No:</b> 0720165 | <b>LMP:</b> 7/8/2000  | <b>Delivery:</b> 6/5/2001   |
| <b>Age:</b> 23 years  | <b>EDD:</b> 14/5/2001 | <b>Discharge:</b> 16/5/2001 |

#### **Presenting history**

The patient was a known cardiac patient with mitral regurgitation who had been followed up in the cardiac clinic since 1992. She was admitted via the antenatal clinic where she presented with dyspnoea on mild exertion, orthopnea and paroxysmal nocturnal dyspnea. There was no cough or leg swelling.

#### **Obstetrics and gynaecology history**

Her last menstrual period had been on 7/08/2000. Expected date of delivery was 14/05/2001. Gestation by dates on admission was 38 weeks and 2 days. She was para 0+0. She had not attended antenatal clinic. Antenatal profile was done. Her blood group was A positive, haemoglobin 10g/dl, VDRL was negative. She was classified as grade II at the cardiac clinic where she had been attending at Nairobi Hospital, at the heart to heart foundation.

#### **Past medical history**

She had been on follow-up in the cardiac clinic since 1992. She absconded in 1995 and was on traditional medication up to the current conception.

#### **Social and family history**

She was single and living with her parents in Muranga. There was no family history of chronic illness.

#### **Physical examination.**



The patient was not pale, there was no jaundice, no leg oedema and was afebrile. On the respiratory system, the chest was clear. She had a loud systolic murmur at the apex, which was radiating to the axilla. She had regular pulse of 80 per minute. The central nervous system was normal.

**Abdominal examination.**

The liver and spleen were not palpable. The fundal height was term, longitudinal lie and cephalic presentation. The foetal heart rate was 140 per minute and was regular.

**Diagnosis and management**

A diagnosis of mitral regurgitation and functional grade III cardiac disease at 38 weeks and 2 days gestation was made.

She was admitted for bed rest in a propped up position. Treatment started included digoxin 0.25mg once daily and Frusemide tablets 40mg once daily which was commenced after cardiology review.

**Investigations**

Full Haemogram:

|                        |                     |
|------------------------|---------------------|
| Haemoglobin            | 10.5g/dl            |
| White blood cell count | $6.5 \times 10^9/l$ |

Renal function tests

|            |          |
|------------|----------|
| Potassium  | 3.6mol/l |
| Sodium     | 112mol/l |
| Urea       | 2.6mol/l |
| Creatinine | 70umol/l |

|            |                                                                                                                                |
|------------|--------------------------------------------------------------------------------------------------------------------------------|
| Urinalysis | showed nitrites in urine. Urine culture after nitrofurantoin revealed growth of Staph. aureus that was sensitive to Augmentin. |
|------------|--------------------------------------------------------------------------------------------------------------------------------|

ECG and echocardiogram:      Showed features of hypertrophic cardiomyopathy with mitral regurgitation.

Obstetric ultrasound:          Confirmed the presence of a single intrauterine pregnancy at 38 weeks gestation.

The patient continued with bed rest with an aim of allowing her to go into spontaneous labour. She was put on nitrofurantoin for urinary tract infection. Her functional status improved to Grade II. However, on 6/5/2001 she went into spontaneous labour. At a cervical dilatation of 4cm, she was noted to have developed an irregular fetal heart pattern. A decision was made to deliver the patient by emergency caesarean section. She had not given consent for bilateral tubal ligation.

The patient was prepared for theatre as described in the introduction. Anaesthetic review was requested and the paediatrician informed. Three units of packed cells were cross-matched for the patient. Hyoscine was given for premedication.

At operation, the outcome was delivery of a live female infant whose weight was 2650g with an Apgar score of 9 in 1 minute and 10 in 5 minutes. The baby was admitted to the Newborn Unit due to the maternal condition. Zinacef 1.5mg stat was given intraoperatively. Oxytocin was given after delivery of the placenta.

### **Postoperative recovery**

The patient was observed in the high dependency unit for 48 hours. She was stable and was transferred to the maternity ward. Meanwhile, she was started on parenteral Augmentin. The rest of the postoperative course was uneventful. The patient was discharged home on the tenth postoperative day with her baby. She was scheduled for postnatal review after six weeks. Meanwhile she was scheduled for review by the cardiologist in the clinic.

When she was reviewed in the postnatal clinic, her cardiac function had remarkably improved because she was symptomless. The wound had completely healed and the baby was gaining weight appropriately. On contraception she opted to use barrier methods. She was reluctant to undergo bilateral tubal ligation. She was referred to the family welfare clinic and advised to continue with the cardiac clinic.

## DISCUSSION

The patient presented was a known cardiac patient who had been followed up in the cardiac clinic since 1992. She was classified as having grade II cardiovascular disease on booking at the antenatal clinic. She was admitted at 38 weeks and 2 days with dyspnoea on mild exertion, orthopnoea and paroxysmal nocturnal dyspnoea (grade III). She was started on digoxin and frusemide, which improved her functional status to grade II.

Cardiovascular disease is the most important non-obstetric cause of disability and death in pregnant women, occurring in 0.4 – 4% of pregnancies. The reported maternal mortality rate ranges from 0.4 – 6.8%. Maternal deaths due to cardiac disease in the period between 1968-1992 in Turkey was 8.5%<sup>(11)</sup>. The added haemodynamic burden of pregnancy, labour and delivery can aggravate symptoms and precipitate complications in a woman with pre-existing cardiac disease<sup>(1)</sup>. It accounts for 86% of all the cases of cardiac disease in pregnancy at Kenyatta National Hospital<sup>(3)</sup>.

In developing countries, the incidence of rheumatic heart disease has been declining because of the overall trend in the decline of rheumatic fever. This has been noticed to decrease in the last half of the twentieth Century mainly because of the improvement in socio-economic status and the discovery and increasing use of penicillin<sup>(4)</sup>.

Evaluation of the pregnant woman in whom cardiac disease is suspected includes a careful history, complete physical examination and non-invasive laboratory tests in order to establish a diagnosis and anticipate the prognosis. The degree of functional disability is graded according to the New York Heart Association classification i.e.



- Grade I: No symptoms limiting ordinary physical activity.
- Grade II: Slight limitation to mild to moderate activity but no symptoms at rest.
- Grade III: Marked limitation on minimal activity.
- Grade IV: Symptoms at rest or of frank congestive heart failure.

Sudden and unpredictable changes in classification can occur during pregnancy

(1)

The aim of management during pregnancy is to restrict physical activity and thus reduce the strain on the cardiovascular system. Patients in failure should be treated. Risk factors for failure include exertion, anaemia, infections, (especially urinary tract infections in pregnancy), hypertension, multiple pregnancy, arrhythmias and rarely hyperthyroidism <sup>(5)</sup>. The patient presented was a 23-year-old para 0+0 who had been followed up in the antenatal clinic as a known cardiac patient since 1992. Her haemoglobin level was 10g/dl. She did not have signs of any infection. She was initially classified as grade II cardiac patient. However on review at 38weeks and 2 days, she complained of dyspnea on mild exertion, orthopnea and paroxysmal nocturnal dyspnea. She was admitted, put on bed rest in a propped up position and started on digoxin tablets 0.25mg once daily and frusemide tablets 40mg once daily. A urinalysis showed nitrites in urine. She was treated with nitrofurantoin. Her status improved to grade II while in the ward on treatment.

In general, patients in grade III are best admitted to hospital for the duration of pregnancy from 28 weeks. Digitalis is generally required. Patients in grade IV are at grave risk and must be in hospital throughout pregnancy. Should any patient develop heart failure, she must be admitted to hospital and remain there until delivery <sup>(6)</sup>. Admission remote from delivery has become a common place in women with Grade II cardiac disease <sup>(7)</sup>. Our patient was admitted when her functional status shifted from grade II to III. Urinary tract infection was the most likely cause.

Delivery in cardiac disease should be accomplished vaginally unless there are obstetrical indications for caesarean section. Relief from pain and apprehension without undue depression of the infant of the mother is important during labour. During labour,



the mother should be placed in a semi recumbent position. Measurements of the pulse and respiratory rate should be made at least four times every hour during the first stage of labour and every 10 minutes during the second stage. An increase in pulse rate much above 100 per minute or in the respiratory rate above 24 particularly when associated with dyspnea suggest cardiac decompensation. Intensive medical management should be instituted. This includes morphine, oxygen and frusemide in the presence of pulmonary oedema. Oxygen should be given by intermittent positive pressure ventilation. In the presence of cardiac failure, delivery by any known method carries a high maternal mortality rate. Accordingly treatment of heart failure in pregnancy is primarily medical. The prime objective is to correct the decompensation, for only then will delivery be safe<sup>(7)</sup>. Vacuum delivery may be used to shorten the duration of second stage. The use of ergometrine is contraindicated in patients with heart disease. Oxytocin may be safely used in management of the third stage of labour in patients with heart disease. Oxytocin may be safely used in management of the third stage of labour in patients with heart disease<sup>(8)</sup>. The most dangerous time for the development of congestive cardiac failure or pulmonary oedema is immediate after delivery. Careful attention must be paid to patients at risk at this time<sup>(9)</sup>.

For caesarean section, the combination of thiopental, succinyl choline, nitrous oxide and at least 30% oxygen may be used<sup>(7)</sup>. The patient presented had been planned for vaginal delivery. She was however delivered by emergency caesarean section due to fetal distress.

Postpartum haemorrhage, infection and thromboembolism are much more serious complications of pregnancy in the woman with cardiac disease. Breastfeeding is usually not contraindicated<sup>(7)</sup>.

Sterilization is the method of choice for contraception if the patient has completed her family size. Optimal spacing of births will ensure that no more than one child at a time is nurse, lifted and carried. Barrier methods are preferable but the failure rate is high. Oral contraceptives are preferable to the insertion of an intrauterine device but the

former has been associated with thromboembolism (9). Our patient declined to have tubal ligation and opted to use barrier methods instead.

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## OBSTETRIC LONG COMMENTARY

A REVIEW OF PREGNANCY OUTCOME IN  
PATIENTS WITH HYPERTENSIVE DISEASE AS  
SEEN AT PUMWANI MATERNITY HOSPITAL,  
NAIROBI.



## **ABSTRACT**

**OBJECTIVE:** To determine pregnancy outcome in patients with hypertension in pregnancy.

**DESIGN:** This was a cross-sectional study based on the retrospective analysis of records of patients with hypertension in pregnancy.

**SETTING:** The study was conducted at the Pumwani Maternity Hospital, Nairobi.

**SUBJECTS:** All case notes of patients with blood pressures higher than 140 systolic or 90 diastolic who were admitted between January 1<sup>st</sup> and December 31<sup>st</sup> 2001 were studied

**MAIN OUTCOME MEASURES:** Maternal and fetal morbidity and mortality.

**RESULTS:** There were 21560 deliveries at Pumwani Maternity Hospital during the study period. Hypertensive disease in pregnancy occurred in 2.9% of deliveries, with eclampsia occurring in 0.39% of deliveries. The perinatal mortality rate in patients with hypertensive disease was 303/1000 live births. This was higher in teenagers (71.66%), women of parity 4 and above (44.4%), those with severe disease (42.9%), those who had no antenatal care (34.2%), those in whom diagnosis of hypertension was made before 28 weeks gestation (60.1%) and those delivered by vaginal breech (53.8%). There were no maternal deaths attributable to hypertension.

**CONCLUSION:** This study shows that hypertensive disease is a significant cause of maternal morbidity and perinatal morbidity and mortality at Pumwani Maternity Hospital. Timely and aggressive management may help to reduce this.

## INTRODUCTION

Hypertensive disease in pregnancy is one of the leading causes of maternal morbidity and mortality (4-6,33). Up to 500,000 women die each year of pregnancy and childbirth related problems. In Kenya, the average maternal mortality is 660 – 1000/100000 live births (23,32). The presence of hypertension in pregnancy substantially increases the rate of perinatal and maternal morbidity and mortality(5-7,23,32,34). Obore, working at Kenyatta National Hospital found hypertension in pregnancy to account for 9.4% of maternal deaths (23).

Mothers with hypertensive disease are usually hospitalized for long periods, which is costly. Much can be done to provide quality obstetric care to patients with hypertensive disease in pregnancy even where resources are scarce.

Hypertension is the most common medical disorder in pregnancy in the developed world (1,3). Mati (1975) found that hypertension complicates pregnancy in 1.5 to 9 per cent of Kenyan women in his series (4). Wanjohi (1984) found 58 eclamptic patients over two years at the Kenyatta National Hospital (5), while Bansal (1985) at the Pumwani Maternity Hospital found a prevalence of pre-eclampsia of 3.7% (6). Kibaru found a prevalence of hypertension in pregnancy of 5.4% at Kenyatta National Hospital (33). The incidence of pre-eclampsia varies widely worldwide. The incidence in tropical Africa is much lower than in Europe, although reliable data is not available especially for the former. These differences are not easily explained but speculation centres on dietary and climatic factors (7). In a study in America, hypertension was found to complicate approximately 15% of pregnancies depending on different populations and diagnostic criteria used(8).

## LITERATURE REVIEW

Hypertension in pregnancy is defined as a blood pressure reading of 140/90mmHg after 20 weeks of gestation, at least two readings are necessary six hours apart. Where previous blood pressure is known an increase of at least 30mmHg systolic or 15mmHg diastolic is also indicative of hypertensive disease. The mean arterial pressure can also be used to denote the presence of hypertensive disease. If prior blood pressure is not known, a mean arterial pressure of 105mmHg is indicative. For those with a known previous blood pressure, an increase in arterial pressure of 20mmHg is also indicative of hypertensive disease (1,2). In our unit we use the reading of 140/90mmHg six hours apart or an increase of 15mmHg and 30mmHg diastolic and systolic respectively as indicative of hypertensive disease in pregnancy. Mean arterial pressure (MAP) is measured using the formula:

$$\text{MAP} = \frac{\text{Systolic pressure} + (2 \times \text{diastolic pressure})}{3}$$

3

Hypertensive disease in pregnancy is classified in several ways (1-3,8). Basically there is the pregnancy-induced hypertension (PIH), which is synonymous with pre-eclampsia/eclampsia and pregnancy-aggravated hypertension (PAH), which refers to chronic hypertension, worsened by pregnancy. PIH accounts for over 70% of hypertensive disease in pregnancy (1,2). This condition occurs mainly in young patients, usually primigravidae of low socio-economic status with a protein intake of less than 55g/day(3). The onset is usually after 20 weeks gestation and there is vascular hyperactivity as evidenced by increased sensitivity to angiotensin II and a positive roll over test (1,3). In this test, blood pressure is measured in the left lateral position. The patient is then asked to turn to the supine position and blood pressure is retaken immediately, then 5 minutes later. The test is positive when the diastolic pressure rises by 20mmHg or more (1,3). The hypertensive disease within these patients usually clears within six weeks postpartum with no residual damage. On the other hand PAH occurs in much older patients (over 30 years), usually multiparous, of any socio-economic class. PAH is classified further as (1) Superimposed preeclampsia and (2) Superimposed eclampsia. The onset of the hypertension is usually before 20 weeks. There is no



evidence of vascular hypersensitivity and the hypertension may persist beyond six weeks postpartum. There may be evidence of deterioration of the condition after delivery (1,3). Differentiation between pregnancy induced hypertension and chronic hypertension in pregnancy can be very difficult especially when the patient is first seen after the second trimester, as usually happens in developing countries where most of the patients start antenatal care late (5,7). For clinical management, however, a woman with an acute rise in blood pressure in the latter half of pregnancy must be regarded as having pre-eclampsia with the possibility of progression to eclampsia and should be managed accordingly (9).

According to the committee on terminology of the American college of obstetrics and Gynaecology, pre-eclampsia is defined as a rise in blood pressure during the latter half of pregnancy, labour or in the early puerperium with a return of blood pressure to normal within 10 days of delivery. There should also be proteinuria or facial, digital or generalized oedema or both (1). Dependent oedema is common in pregnancy due to impaired venous return and lymphatic drainage occasioned by the gravid uterus (13). The diagnosis is questionable in the absence of proteinuria for Sheehan et al (1973) rarely found the characteristic renal lesion of glomeruloendotheliosis unless the patient had proteinuria (10). Proteinuria is defined as 300mg or more of urinary protein in a 24hour period, or at least 100mg/100ml urine in a minimum of 2 random samples collected 6 or more hours apart (1). Eclampsia is the occurrence of convulsions not caused by any coincidental disease such as epilepsy in a patient whose condition also satisfies the criteria for pre-eclampsia (1,10).

A marked rise in blood pressure is an ominous sign even in the absence of proteinuria. 10% of eclamptic fits occur before overt proteinuria (9). Proteinuria is a sign of worsening hypertensive disease, with higher maternal and fetal risks. Browne (1958) said any explanation on the etiology of pre-eclampsia should explain:

- 1 Why it occurs only in association with pregnancy and only in humans.
- 2 Why it occurs in pregnancy in a hydatidiform mole in the absence of a fetus.



- 3 Why it is commoner in multiple pregnancy and diabetes
- 4 The presence of placental infarcts.
- 5 Its preference for first pregnancy.
- 6 Why it resolves soon after pregnancy (11).

Other authors have suggested that the placenta rather than the fetus is responsible since the disease occurs in the absence of the fetus as in hydatidiform mole. It can occur or continue after fetal death if there is living placental tissue or postpartum in the presence of retained placental tissues (12). Several aetiological factors have been postulated in the genesis of pre-eclampsia/eclampsia. Increased vascular sensitivity coupled with a rise in corticosteroids and other placental hormones have been implicated. The placenta is thought to have a protective role. Its enzymatic activities require the presence of certain enzymes and minerals, hence the association of pre-eclampsia with certain dietary deficiencies such as pellagra and beriberi (11,13). Other studies have suggested that there may be familial and immunological basis for pre-eclampsia/eclampsia (14-17). It was found in these studies that pre-eclampsia/eclampsia had occurred in 14% of mothers of patients with eclampsia as compared to 3% of controls (15). A study in Nigeria (17) found that 74% of multiparous eclamptics had a new partner for the current pregnancy, hence a likelihood of an immunological factor being implicated. It has been suggested therefore, that pre-eclampsia/eclampsia may be genetically determined, susceptibility based on a single gene with incomplete penetrance. The pathogenesis may be multifactorial involving coagulation, vascular reactivity or an abnormal immunological reaction between the mother and the fetus (15-17).

Pre-eclampsia/eclampsia has been found to vary significantly with the weather. The incidence of eclampsia has been found to increase with wet, humid weather and low temperatures (18,19). Hence meteorological factors have been implicated in its etiology through as yet unestablished mechanisms. Fever has been postulated to be a trigger factor for eclampsia (20). Machoki (1989) found that 25.6% of the eclamptics in his series had fever (21) while Ekwempu (22) found that 77% of his eclamptics had fever. It appears that infection through fever with or without some other factors may modify the

nervous transmission in the central nervous system to trigger off convulsions in those women who are prone to develop eclampsia.

Preeclampsia is a multisystem disorder of unknown etiology. The pathology is basically endothelial dysfunction with generalized vasospasm arising from an imbalance in the production of vasodilating prostacyclin and vasoconstricting thromboxane prostaglandins (10,26) leading to arterial hypertension (1). These vascular changes, together with the local hypoxia of the surrounding tissues, lead to haemorrhage and necrosis. At the utero-placental bed, the ageing process of the placenta is accelerated. The vessels become non-responsive and tortuous with an apparent constriction between the radial and decidual arteries. The functional result is reduced blood flow and hence reduced placental perfusion (1,8). Perinatal death results from large placental infarcts and placental abruption. The renal lesion characteristic of pregnancy-induced hypertension is "glomerular endotheliosis". There is swelling of the endothelial cells leading to occlusion of the capillary lumen and apparent glomerular avascularity. Impaired tubal function leads to inability for the kidneys to concentrate urine and a raised level of serum urea, intravascular creatinine and other urates (1). There is an increase in the total body water especially if oedema is present. The intravascular volume is, however, decreased irrespective of the total body water, hence these patients tolerate blood loss very poorly. The plasma electrolytes show little change(1,8). The cardiac vasculature is also involved in the generalized vascular changes and cardiac failure can result. Pulmonary vascular thrombosis and multiple small haemorrhages occur. Secondary bronchopneumonia is usually present in severe cases (1).

In the central nervous system there are usually haemorrhagic lesions. These may range from petechiae to massive haemorrhages in the subcortex, basal ganglia, pons and subarachnoid space. There is also ischaemic softening due to terminal asphyxia. The etiology of eclamptic fits is controversial but it is thought to be due to the combination of brain oedema and hypoxia (8). In the liver obvious changes occur in 10% of the patients with pre-eclampsia/eclampsia and in about 50% of the fatal cases (1,8,10). There are widespread petechial haemorrhages and larger subcapsular haemorrhages may become

confluent and rupture, causing intraperitoneal haemorrhage. Liver damage resolves in a matter of weeks in survivors (8).

The severity of PIH is assessed by frequency and intensity of abnormalities as shown in the table below:-

| ABNORMALITY              | MILD ECLAMPSIA | SEVERE ECLAMPSIA |
|--------------------------|----------------|------------------|
| Diastolic Blood Pressure | < 100 mmHg     | > 110 mmHg       |
| Systolic Blood Pressure  |                | > 160 mmHg       |
| Headache                 | Absent         | Present          |
| Visual disturbances      | Absent         | Present          |
| Upper abdominal pain     | Absent         | Present          |
| Oliguria                 | Absent         | Present          |
| Serum creatinine         | Normal         | Present          |
| Thrombocytopenia         | Absent         | Present          |
| Hyperbilirubinemia       | Absent         | Present          |
| Liver enzyme elevation   | Absent/Minimal | Marked           |
| Fetal growth retardation | Absent         | Obvious          |
| Pulmonary oedema         | Absent         | Present          |

(1)

Blood pressure per se is not always a dependable indicator of severity. An adolescent patient may have convulsions with a blood pressure of 140/90 and a proteinuria of 3+ whereas other patients may not convulse with much higher blood pressures.

The syndrome of haemolysis, elevated liver enzymes and low platelet count (HELLP) characterized by microangiopathic haemolytic anaemia, liver dysfunction and thrombocytopaenia occurs in severe preeclampsia. Sibai and others (26,27) diagnosed HELLP syndrome by the presence of the following laboratory findings.



- (a) Haemolysis defined by an abnormal peripheral blood smear, which includes Burr cells, and/or schistocytes with raised bilirubin levels greater than or equal to 1.2mg/dl.
- (b) Elevated liver enzymes:  
SGOT greater than or equal to 70u/l  
Lactate dehydrogenase > 600u/l
- (c) Platelet count <100,000/mm<sup>3</sup>  
The HELLP syndrome is a poor prognostic sign. It may occur remote from term with an apparently normal blood pressure (25,28,29).

Ngayu (1994) at Kenyatta National Hospital found the HELLP syndrome to occur in 2.8% of patients with PIH (25). Severe preeclampsia can also manifest with epigastric pain, dizziness, blurring of vision and vomiting.

All the pathological changes in the various systems, increase the perinatal and maternal morbidity and mortality in patients with hypertensive disease in pregnancy. Perinatal complications are worse for patients with severe pre-eclampsia and in pre-eclampsia superimposed on chronic hypertension. This is due to the reduction in utero-placental perfusion hence placental insufficiency, which leads to chronic hypoxia. This can result in asymmetrical growth retarded fetuses. These are associated with high chances of death in-utero or intrapartum (1,8,10). Deteriorating maternal condition on the other hand leads to early delivery of premature fetuses with the attendant dangers of prematurity including respiratory distress syndrome, hypothermia, hypoglycaemia and electrolyte imbalance. Hypertensive disease in pregnancy therefore affects the perinatal mortality significantly. Some studies, have shown it to increase to 2-3 times that of normal obstetric population (1,8). However, it has also been shown that with good antenatal care, perinatal loss should not exceed 20 in 1000 (8).

Maternal complications of hypertension include eclamptic fits. These mostly occur antepartum but may occur intrapartum and up to 10 days post partum. Other complications include acute renal failure, haemorrhage due to disseminated intravascular

coagulation, sepsis, intravascular haemorrhage and acute pulmonary failure. Blindness may arise from retinal detachment or occipital lobe oedema or haemorrhage. Vision usually returns with time (1,2,8)

Preeclampsia/eclampsia are the commonest causes of maternal death in the developed world. This mortality rate has ranged from 1-20% since 1945 (1). Maternal deaths have become rare especially in the developed countries unlike in the developing world. However, hypertension remains one of the commonest causes of maternal deaths (7,10). Makokha (1980) found pre-eclampsia/eclampsia to contribute to 3% of maternal mortality at the Kenyatta National Hospital (30), whereas Machoki (21) in 1989 found it to account for 465/1000 deliveries at Kenyatta National Hospital. Lawson (1988), in Nigeria, found it to account for about 10% of the maternal mortality (7). More recently, Obore (23) found it to account for 9.4% of maternal deaths at Kenyatta National Hospital. In England and Wales hypertension and related complications are directly responsible for about 1 in 6 maternal deaths (8). The commonest causes of death are cerebral vascular complications, aspiration pneumonia, hypoxic encephalopathy, thromboembolism, hepatic rupture, renal failure and anaesthetic accidents (1,8).

The risk of pre-eclampsia recurring in subsequent pregnancies is 33% and when this was superimposed on chronic hypertension, the recurrence rate was about 70% (1). The effect of pre-eclampsia/eclampsia on subsequent development of chronic hypertension is unclear. Some authors have implicated it as a possible sequel, while others maintain that pre-eclampsia does not cause permanent organ damage and does not lead to chronic hypertension or affect long-range health of the mother (1,2,7-9).

Several studies in our unit have implicated hypertensive disease as a cause of perinatal morbidity and mortality (4,5,31). However, the study last done to examine the socio-demographic characteristics of these patients and their pregnancy outcome at Pumwani Maternity Hospital is over 15 years old (6). It is presumed that a lot of socio-economic and medical changes have taken place since then and hence some of the previously observed findings may have altered. This study was therefore designed with

the aim of determining the socio-demographic profile of women with hypertensive disease in pregnancy at the Pumwani Maternity Hospital, pregnancy outcome and associated factors. The findings of this study will assist us in determining the sociodemographic features of the mothers with hypertensive disease in pregnancy as well as improving their management and the outcome of pregnancy. This is through identification of problems with a view to appropriate resource allocation.

## **RATIONALE FOR THE STUDY**

Studies done at Kenyatta National Hospital and Pumwani Maternity Hospital have implicated hypertension in pregnancy as a cause of maternal and perinatal morbidity and mortality. The last such study conducted at Pumwani is more than 15 years old. In this time, changes in medical technology, socioeconomic status and population density have occurred, thus the prior observations may have changed. This study was designed to evaluate retrospectively, the maternal and perinatal outcomes of women with hypertensive disease in pregnancy at Pumwani Maternity Hospital. It is recognized that:-

- 1 Certain categories of expectant mothers are more prone to developing hypertensive disease in pregnancy.
- 2 Detection of the above may help in improving the maternal and fetal outcome in these patients.
- 3 High medical costs due to prolonged hospitalization make this an important disease.
- 4 Aggressive management and prompt, timely interventions reduce maternal and perinatal morbidity and mortality.



## **NULL HYPOTHESIS**

Hypertensive disease in pregnancy has a negative effect on the fetal outcome. Improved obstetric care for patients with hypertension in pregnancy leads to favorable maternal and fetal outcome.

## **OBJECTIVES**

### **Broad objective**

To determine the magnitude of the problem of hypertensive disease in pregnancy, the pregnancy outcome among the patients with hypertensive disease in pregnancy, and factors which may influence the fetal outcome among these patients at the Pumwani Maternity Hospital.

### **Specific objectives**

- 1 To determine the prevalence of hypertensive disease in pregnancy.
- 2 To determine the sociodemographic characteristics of women with hypertensive disease in pregnancy.
- 3 To determine the utilization of antenatal care services by hypertensive women.
- 4 To determine the Apgar score of babies born to hypertensive women.
- 5 To determine the birthweight of babies born by hypertensive women.
- 6 To relate severity of disease at presentation to fetal outcome.
- 7 To relate mode of delivery to foetal outcome.

## **METHODOLOGY**

### **Study Design**

This was a retrospective cross-sectional study.

### **Study Area**

This study was carried out at the Pumwani Maternity Hospital (PMH). This is the largest and busiest maternity hospital in Kenya catering for an average of 60 deliveries daily. It is run by the Nairobi City Council and caters for the residents of the city of Nairobi and its environs. It also serves as a referral center for the city council clinics and health centers.

### **Study Period**

The study period extends between 1<sup>st</sup> January to 31<sup>st</sup> December 2001, both dates inclusive.

### **Study Population**

This included all the patients admitted with hypertensive disease in pregnancy and delivered during the study period.

### **Sample Size and Selection Criteria**

All the patients with hypertension in pregnancy who were admitted and delivered within the study period were included in the study. Hypertension is defined as the recording of a blood pressure of 140/90mmHg on two occasions at least six hours apart, or a rise of 15mmHg or 30mmHg diastolic and systolic blood pressures respectively where previous blood pressures were known. No attempt was made to differentiate between pregnancy induced hypertension (PIH) and pregnancy aggravated hypertension (PAH). This is

because most patients usually present late in the second and third trimesters, in labour, or after convulsing. The hypertension was graded from mild to severe on the basis of the Goecke-score. This takes into account systolic blood pressure, diastolic blood pressure, proteinuria and oedema, and awards points according to the table below (6)

**The Goecke-score**

| Measuremen<br>t          | Score | Measuremen<br>t | Score | Measuremen<br>t | Score |
|--------------------------|-------|-----------------|-------|-----------------|-------|
| Systolic BP<br>140 – 160 | 1     | 161 – 180       | 2     | >180            | 3     |
| Diastolic BP<br>90 – 95  | 1     | 96 – 100        | 2     | >100            | 3     |
| Proteinuria<br>+         | 1     | ++              | 2     | 3+ or more      | 3     |
| Oedema legs              | 1     | Generalised     | 2     |                 |       |

Goeke –score has a total of 11 points and classified hypertension in pregnancy as follows:

- 1. Mild hypertension = 1 – 3 points
- 2. Moderate hypertension = 4 – 7 points
- 3. Severe hypertension = 8 – 11 points

**Data Collection**

Records of all patients admitted with hypertensive disease in pregnancy were retrieved from the records department. Case notes of women delivering at PMH during the study period were studied. Case notes of all patients with hypertension were selected and studied to assess maternal and perinatal outcome. All mothers were divided into mild, moderate or severe disease using the Goecke score. Detailed information



concerning sociodemographic characteristics, parity gestation at booking and delivery as well as outcome of delivery and hospital stay was entered into questionnaires.

### **Inclusion Criteria**

Women with hypertension in pregnancy in whom:-

- No other disease was identified from the case records.
- The pregnancy was singleton.

### **Exclusion Criteria**

Women with hypertension in pregnancy in whom:-

- Any other medical condition(s) could be identified.
- The pregnancy was multiple

### **Data Management**

All the information was recorded in a pre-prepared questionnaire. The information was coded and entered into a computer using SPSS Epi-info system. The information was analysed using the chi square test to ascertain the level of statistical significance, and the data described using terms such as mean and median (35). At analysis, fresh stillbirths, macerated stillbirths and early neonatal deaths were considered together as babies who died.

### **Ethical Considerations**

Authority to carry out the study was obtained from the Kenyatta National Hospital ethical and research committee, as well as the Medical Officer of Health of the Nairobi City Council. Names of the patients were not entered into questionnaires.

## RESULTS

During the study period, there were 21560 deliveries. There were 623 (2.9%) cases of hypertensive disease in pregnancy. Eclampsia occurred in 83 (0.38%) of these.

Table 1. Socio-demographic characteristics n = 623

|                       | FREQUENCY | PERCENTAGE |
|-----------------------|-----------|------------|
| <b>AGE GROUP</b>      |           |            |
| <15                   | 0         | 0          |
| 15 – 19               | 102       | 16.4       |
| 20 – 24               | 176       | 28.3       |
| 25 – 29               | 199       | 31.9       |
| 30 – 34               | 108       | 17.3       |
| 35 +                  | 38        | 6.1        |
| <b>Parity</b>         |           |            |
| 0                     | 326       | 52.3       |
| 1                     | 72        | 11.6       |
| 2                     | 87        | 14.0       |
| 3                     | 66        | 10.6       |
| 4                     | 58        | 9.3        |
| 5 or more             | 14        | 2.2        |
| <b>Marital Status</b> |           |            |
| Married               | 412       | 66.2       |
| Single                | 175       | 28.1       |
| Divorced              | 36        | 5.7        |
| <b>Education</b>      |           |            |
| None                  | 26        | 4.2        |
| Primary               | 240       | 38.5       |
| Secondary             | 275       | 44.1       |
| College               | 82        | 13.2       |

16.4% of the patients were teenagers, with 77.5% being in the optimum reproductive age group of 20-34 years . Most of the patients were of low parity. Only 2.2% were para 5 or higher.

Most of the patients (66.2%) were married. Most of the patients(95.4%) had some education with only 4.2% having had no formal education.

Table 2. Utilisation of antenatal services      n=623

|                                                | FREQUENCY | PERCENTAGE |
|------------------------------------------------|-----------|------------|
| <b>Clinic attended</b>                         |           |            |
| City Council                                   | 278       | 44.6       |
| Pumwani Hospital                               | 125       | 20.1       |
| None                                           | 85        | 13.6       |
| Other                                          | 135       | 21.7       |
| <b>Gestation at antenatal booking in weeks</b> |           |            |
| < 12                                           | 32        | 5.1        |
| 13 – 20                                        | 93        | 14.9       |
| 21 – 28                                        | 325       | 52.2       |
| 29 – 36                                        | 135       | 21.6       |
| > 36                                           | 38        | 6.2        |

76.7% of the patients had attended somewhere for antenatal care, with 20.1% having attended the Pumwani Hospital antenatal clinic. The mean gestation at antenatal booking was 28 weeks, with a standard deviation of 3.8 weeks and a range from 10 to 42 weeks. 72.2% of the patients had booked antenatal care by the 28<sup>th</sup> week of gestation.



Table 3. Delivery                    n = 623

| GESTATION               | FREQUENCY | PERCENTAGE |
|-------------------------|-----------|------------|
| < 20                    | 0         | 0          |
| 21 – 28                 | 15        | 2.5        |
| 29 – 32                 | 114       | 18.3       |
| 33 – 36                 | 138       | 22.1       |
| 37 +                    | 366       | 57.1       |
| <b>Mode of delivery</b> |           |            |
| SVD                     | 320       | 51.4       |
| Vacuum                  | 32        | 5.2        |
| Caesarean               | 232       | 37.2       |
| Vaginal breech          | 39        | 6.2        |

The mean gestation at delivery was 37 \_+ 2.8 weeks with a range of 24 – 43 weeks. The majority (57.1%) delivered at 37 weeks of gestation and above. Only 2.5% delivered before 28 weeks gestation, with the earliest delivery occurring at 26 weeks. Most of the patients (51.4%) had spontaneous vertex deliveries. Assisted vacuum delivery was carried out in only 5.2%.

Table 4. Fetal Outcome      n = 623

|                        | FREQUENCY  |      | PERCENTAGE   |      |
|------------------------|------------|------|--------------|------|
| Birth Weight           |            |      |              |      |
| < 1000                 | 13         |      | 2.1          |      |
| 1001 – 1500            | 60         |      | 9.7          |      |
| 1501 – 2000            | 125        |      | 20.1         |      |
| 2001 – 2500            | 72         |      | 11.2         |      |
| 2501 – 3000            | 154        |      | 24.8         |      |
| > 3000                 | 199        |      | 32.1         |      |
| Apgar Score            |            |      |              |      |
|                        | ONE MINUTE |      | FIVE MINUTES |      |
|                        | Number     | %    | Number       | %    |
| 0                      | 57         | 9.2  | 57           | 9.2  |
| 1 – 4                  | 56         | 9.0  | 36           | 5.8  |
| 5 – 7                  | 133        | 21.3 | 103          | 16.6 |
| 8 – 10                 | 377        | 60.5 | 427          | 68.4 |
| fetal deaths           |            |      |              |      |
|                        | FREQUENCY  |      | PERCENTAGE   |      |
| Fresh still births     | 26         |      | 18.0         |      |
| Macerated still births | 52         |      | 35.9         |      |
| Early neonatal deaths  | 67         |      | 46.1         |      |

The mean birth weight was 2555\_+478g with a range from 650 to 4700grams. 43.1% of the babies were of birth weight below 2500 grams. Most of the babies (60.5% and 68.4%) had good Apgar scores at 1 and 5 minutes respectively. 9.0% and 5.8% of the babies had poor Apgar scores at 1 and 5 minutes respectively. There were 145 perinatal deaths, giving a perinatal mortality rate of 232 per 1000 live births. 67 (46.1%) were early neonatal deaths.

Table 5. Relationship between maternal age and perinatal outcome

| MATERNAL AGE IN YEARS | ALIVE  |      | DEAD   |      | TOTAL  |      |
|-----------------------|--------|------|--------|------|--------|------|
|                       | number | %    | number | %    | number | %    |
| 15 – 19               | 68     | 66.7 | 34     | 33.3 | 102    | 16.4 |
| 20 – 34               | 402    | 83.2 | 81     | 16.8 | 483    | 77.5 |
| 35 +                  | 20     | 52.6 | 18     | 47.4 | 38     | 16.1 |
| Total                 | 478    | 76.7 | 145    | 23.3 | 623    | 100  |

Patients 35 years or older had a higher perinatal mortality rate (47.4%) than the other age groups. The teenagers had a worse perinatal outcome than mothers of age 20- 34. These differences were statistically significant ( $X^2 = 30.08$ ,  $p < 0.001$ )

Table 6. Relationship between maternal age and 5 minute Apgar score

| APGAR SCORE<br>maternal age | 0   |      | 1 – 5 |      | 6 – 10 |      | TOTAL |      |
|-----------------------------|-----|------|-------|------|--------|------|-------|------|
|                             | NO  | %    | NO    | %    | NO     | %    | NO    | %    |
| 15 – 19                     | 73  | 71.6 | 9     | 8.8  | 20     | 19.6 | 102   | 16.4 |
| 20 – 34                     | 32  | 6.6  | 89    | 18.4 | 362    | 75.0 | 483   | 77.5 |
| 35 +                        | 9   | 23.7 | 4     | 10.5 | 25     | 65.8 | 38    | 6.1  |
| Total                       | 114 | 18.3 | 102   | 16.4 | 407    | 65.3 | 623   | 100  |

Most still births (71.6%) occurred in teenagers. The 20-34 years age group had the highest rate (18.4%) of babies with poor Apgar score at the same time the highest rate (75%) of babies with good 5 minute Apgar scores. These differences were statistically significant ( $X^2 = 239.8$ ,  $p < 0.001$ ).



Table 7. Relationship between maternal age and birth weight

| Birth Weight          | < 1500 |      | 1500 – 2500 |      | 2501 – 3500 |      | > 3500 |      | TOTAL |      |
|-----------------------|--------|------|-------------|------|-------------|------|--------|------|-------|------|
| Maternal Age in Years | No     | %    | No          | %    | No          | %    | No     | %    | No    | %    |
| 15 – 19               | 13     | 17.8 | 35          | 15.1 | 48          | 17.0 | 4      | 11.4 | 102   | 16.4 |
| 20 – 34               | 38     | 52.1 | 187         | 80.6 | 229         | 80.9 | 29     | 82.9 | 483   | 77.5 |
| 35 +                  | 22     | 46.1 | 10          | 4.3  | 6           | 2.1  | 2      | 5.7  | 38    | 6.1  |
| Total                 | 73     | 11.7 | 232         | 37.2 | 283         | 45.4 | 35     | 5.6  | 623   | 100  |

Patients aged 20 – 34 years had the highest rate of babies weighing below 1500g and between 1500 – 2500grams. This may be because this age group comprised 77.5% of the patients. The differences were not significant.

Table 8: Relationship between parity and perinatal outcome

| PARITY | DEAD |      | ALIVE |      | TOTAL |      |
|--------|------|------|-------|------|-------|------|
|        | No   | %    | No    | %    | No    | %    |
| 0      | 48   | 14.7 | 278   | 85.3 | 326   | 52.3 |
| 1 – 3  | 65   | 28.8 | 160   | 71.1 | 225   | 36.1 |
| 4+     | 32   | 44.4 | 40    | 55.6 | 72    | 11.6 |
| TOTAL  | 145  | 23.3 | 478   | 76.7 | 623   | 100  |

The para 4 and higher had more perinatal deaths (44.4%) than the rest of the categories. The differences were statistically significant ( $X^2 = 35.39$ ,  $p < 0.001$ )

Table 9: Relationship between parity and 5 minute Apgar score

| PARITY | APGAR SCORE |      |       |      |        |      |       |      |
|--------|-------------|------|-------|------|--------|------|-------|------|
|        | 0           |      | 1 – 5 |      | 6 – 10 |      | TOTAL |      |
|        | No          | %    | No    | %    | No     | %    | No    | %    |
| 0      | 38          | 12.0 | 53    | 17.0 | 222    | 71.0 | 313   | 50.2 |
| 1 – 3  | 39          | 16.2 | 27    | 11.3 | 171    | 72.5 | 237   | 38.1 |
| 4 +    | 37          | 50.7 | 22    | 30.1 | 14     | 19.2 | 73    | 11.7 |
| TOTAL  | 114         | 18.3 | 102   | 16.4 | 407    | 65.3 | 623   | 100  |

Those para 4 and above had a higher rate (30.1%) of babies with poor 5 minute Apgar scores. Primigravidas and para 1 – 3 had similar rates of babies with good 5 minute Apgar scores. These differences were significant (  $X^2 = 87.86$  ,  $p < 0.001$ )

Table 10. Relationship between level of education and perinatal outcome

| EDUCATION | ALIVE |      | DEAD |      | TOTAL |      |
|-----------|-------|------|------|------|-------|------|
|           | No    | %    | No   | %    | No    | %    |
| None      | 22    | 78.6 | 6    | 21.4 | 28    | 4.5  |
| Primary   | 181   | 75.4 | 59   | 24.6 | 240   | 38.5 |
| Secondary | 201   | 73.6 | 72   | 26.4 | 273   | 43.8 |
| College   | 74    | 90.2 | 8    | 9.8  | 82    | 13.2 |
| TOTAL     | 478   | 76.7 | 145  | 23.3 | 623   | 100  |

The highest rate of perinatal deaths (26.4%) occurred in those with secondary education. This may be because most of the patients with hypertension disease were from this group. These differences were significant. ( $X^2 = 10.14$  ,  $p < 0.035$ ).

Table 11. Effect of severity of disease at presentation on the 5 minute Apgar score

| GOECKE<br>SCORE   | 0   |      | 1 – 5 |      | 6 – 10 |      | TOTAL |      |
|-------------------|-----|------|-------|------|--------|------|-------|------|
|                   | No  | %    | No    | %    | No     | %    | No    | %    |
| Mild 1 – 4        | 32  | 10.1 | 34    | 10.3 | 253    | 79.6 | 319   | 51.2 |
| Moderate 5 –<br>7 | 37  | 14.4 | 19    | 7.2  | 143    | 78.4 | 199   | 41.7 |
| Severe 8 – 11     | 45  | 42.9 | 49    | 46.7 | 11     | 10.4 | 105   | 7.1  |
| TOTAL             | 114 | 13.1 | 102   | 10.9 | 407    | 76.0 | 623   | 100  |

Most still births (42.9%) and poor 5 minute Apgar scores occurred in those with severe disease. These differences were significant ( $X^2 = 176.6$  ,  $p < 0.001$ ).

Table 12. The effect of the severity of disease on fetal weight

| GOECKE<br>SCORE   | < 1500 |      | 1501 – 2500 |      | 2501 – 3500 |      | >3500 |     | TOTAL |      |
|-------------------|--------|------|-------------|------|-------------|------|-------|-----|-------|------|
|                   | No     | %    | No          | %    | No          | %    | No    | %   | No    | %    |
| Mild 1 – 4        | 8      | 2.2  | 141         | 38.8 | 190         | 52.3 | 24    | 6.6 | 363   | 58.3 |
| Moderate<br>5 – 7 | 17     | 11.3 | 67          | 44.4 | 60          | 39.7 | 7     | 4.6 | 151   | 24.2 |
| Severe 8 –<br>11  | 48     | 44.0 | 24          | 22.0 | 33          | 30.3 | 4     | 3.7 | 109   | 17.5 |
| TOTAL             | 73     | 11.7 | 232         | 37.2 | 283         | 45.4 | 35    | 5.6 | 623   | 100  |

Patients with severe disease had the highest number of babies (44%) of birth weight below 1500g and the lowest rate (3.7%) of babies above 3500g. Patients with mild disease had the best fetal weights. These differences were significant ( $X^2 = 146.4$  ,  $p < 0.001$ ).

Table 13. Effect of gestation at diagnosis of hypertension on fetal weight

| FETAL WEIGHT     | < 1500 |      | 1501 – 2500 |      | 2501 – 3500 |      | >3500 |     | TOTAL |      |
|------------------|--------|------|-------------|------|-------------|------|-------|-----|-------|------|
| MATURITY (WEEKS) | No     | %    | No          | %    | No          | %    | No    | %   | No    | %    |
| < 28             | 42     | 44.2 | 38          | 40.0 | 15          | 15.8 | 0     | 0   | 95    | 15.2 |
| 29 – 33          | 28     | 15.6 | 67          | 37.4 | 71          | 39.7 | 13    | 7.3 | 179   | 28.7 |
| 34 +             | 3      | 0.9  | 127         | 36.4 | 197         | 56.4 | 22    | 6.3 | 349   | 56.1 |
| TOTAL            | 73     | 11.7 | 232         | 37.2 | 283         | 45.4 | 35    | 5.6 | 623   | 100  |

A higher fraction of babies weighing less than 2500grams (84.2%) were delivered in mothers in whom diagnosis was made before 28 weeks gestation relative to those in whom diagnosis was made after 34 weeks gestation (37.3%). These differences were significant ( $X^2 = 158.83$  ,  $p < 0.001$ ).

Table 14. Relationship between place of antenatal care and fetal outcome

| PLACE        | ALIVE |      | DEAD |      | TOTAL |      |
|--------------|-------|------|------|------|-------|------|
|              | No    | %    | No   | %    | No    | %    |
| City Council | 203   | 73.0 | 75   | 27.0 | 278   | 44.6 |
| None         | 56    | 65.8 | 29   | 34.2 | 85    | 13.6 |
| Pumwani      | 104   | 83.2 | 21   | 16.8 | 125   | 20.1 |
| Other        | 115   | 85.2 | 20   | 14.8 | 135   | 21.7 |
| TOTAL        | 478   | 76.7 | 145  | 23.3 | 623   | 100  |

Most perinatal deaths (34.2%) occurred in those patients who had not attended antenatal clinic. The majority of babies whose mothers attended antenatal clinic at Pumwani maternity hospital survived. The differences were significant. ( $X^2 = 16.08$  ,  $p < 0.002$ ).



Table 15. Relationship between gestation at delivery and perinatal outcome

| MATURITY IN WEEKS | ALIVE |      | DEAD |      | TOTAL |      |
|-------------------|-------|------|------|------|-------|------|
|                   | No    | %    | No   | %    | No    | %    |
| < 28              | 27    | 39.1 | 42   | 60.9 | 69    | 11.1 |
| 29 – 33           | 91    | 73.3 | 33   | 26.7 | 124   | 19.9 |
| 34 +              | 360   | 83.7 | 70   | 16.3 | 430   | 69.0 |
| TOTAL             | 478   | 76.7 | 145  | 23.3 | 623   | 100  |

Babies did best when delivered after 34 weeks gestation. Most still births occurred when delivery occurred at gestation below 28 weeks. The differences were significant. ( $X^2 = 16.08$  ,  $p < 0.002$ ).

Table 16. Effect of mode of delivery on perinatal outcome

| MODE OF DELIVERY | ALIVE |      | DEAD |      | TOTAL |      |
|------------------|-------|------|------|------|-------|------|
|                  | No    | %    | No   | %    | No    | %    |
| SVD              | 239   | 74.7 | 81   | 25.3 | 320   | 51.4 |
| Vacuum           | 21    | 65.6 | 11   | 34.4 | 32    | 5.2  |
| Caesarean        | 200   | 86.2 | 32   | 13.8 | 232   | 37.2 |
| Vaginal breech   | 18    | 46.2 | 21   | 53.8 | 39    | 6.2  |
| TOTAL            | 478   | 76.7 | 145  | 23.3 | 623   | 100  |

The highest proportion of perinatal deaths occurred in those who had vaginal breech deliveries. Caesarean section had the best outcome. The differences were significant ( $X^2 = 35.04$  ,  $p < 0.001$ ).

Table 17. Relationship between mode of delivery and 5 minute Apgar score

| 5 minute<br>Apgar score | 0   |      | 1 – 5 |      | 6 – 10 |      | TOTAL |       |
|-------------------------|-----|------|-------|------|--------|------|-------|-------|
|                         | No  | %    | No    | %    | No     | %    | No    | %     |
| SVD                     | 56  | 17.5 | 73    | 22.8 | 191    | 59.7 | 320   | 51.4  |
| Vacuum                  | 10  | 31.2 | 5     | 15.6 | 17     | 53.2 | 32    | 0.05  |
| Caesarean               | 33  | 14.2 | 6     | 2.6  | 193    | 83.2 | 232   | 37.2  |
| Vaginal<br>Breech       | 15  | 38.5 | 18    | 46.2 | 6      | 15.3 | 39    | 11.35 |
| TOTAL                   | 114 | 18.3 | 102   | 16.4 | 407    | 65.3 | 623   | 100   |

Babies born by vaginal breech had a higher rate (46.2%) of poor Apgar score 1 – 5. The highest proportion of still births were also delivered by vaginal breech. The best outcome resulted after caesarean section. These differences were statistically significant. ( $X^2 = 98.50$  ,  $p < 0.001$ ).

Table 18. Relationship between severity of disease and length of hospital stay

| Duration of<br>hospitalization<br>(days) | < 3 |      | 4 – 7 |      | > 8 |      | Total |      |
|------------------------------------------|-----|------|-------|------|-----|------|-------|------|
|                                          | No  | %    | No    | %    | No  | %    | No    | %    |
| Mild 1 – 4                               | 199 | 62.3 | 89    | 27.9 | 31  | 9.8  | 319   | 51.2 |
| Moderate 5 – 7                           | 61  | 30.7 | 102   | 51.3 | 36  | 18.0 | 199   | 41.7 |
| Severe 8 – 11                            | 0   | 0    | 43    | 41.0 | 62  | 59.0 | 105   | 7.1  |
| Total                                    | 260 | 41.7 | 234   | 37.6 | 129 | 20.7 | 623   | 100  |

Patients with severe disease had the highest fraction of patients staying in hospital for more than 8 days (59.0%). These differences were statistically significant ( $\chi^2$  152.3,  $p < 0.002$ )

## DISCUSSION

Hypertensive disease in pregnancy still remains a major cause of maternal and perinatal mortality and morbidity worldwide (10). The prevalence of hypertensive disease among the study group was found to be 2.9%. There were 84 cases of eclampsia, giving a prevalence of 0.39%. Mati (1975) found an incidence of between 1.5 – 9% among Kenyan women in his study among various hospitals and health centers. He found a higher incidence among patients in the hospitals as compared to health centers (5). The figure for Kenyatta National Hospital then was 9.06%. This is much higher than in our study. Bansal found a prevalence of 3.6% at Pumwani (7). The lower prevalence in our study may partly be explained by the fact that more of the women with hypertension present directly at the Kenyatta National Hospital, a tertiary care institution. There are no recent studies to show the current prevalence of the disease in our community as the Mati study was done over 25 years ago. During this time many changes in the socio-demographic indices have taken place. For instance there are more specialists at Pumwani Maternity Hospital and in the provincial and district hospitals and hence fewer patients are referred to Kenyatta as the care can be given elsewhere. There are also more private hospitals and more patients who can afford may opt for private care.

Elsewhere in the world hypertension has been reported to complicate 2 – 15% of all pregnancies, with eclampsia occurring between 0.05 – 0.9% of all deliveries (1,2,10,13). This compares favourably with our study, where we found it to occur in 0.39% of deliveries. The lower prevalence of eclampsia may be due to more aggressive management of pre-eclampsia.

The prevalence of hypertensive disease in pregnancy is different between hospitals, regions, countries and ethnic groups. It has been noted that while the incidence



of pre-eclampsia does not differ very much between developed and developing countries, that of eclampsia is still higher in the latter (1,8). As living standards improve and better health care is provided, the incidence of eclampsia declines. This is attributed to the improvement in the antenatal care, which enables pre-eclampsia to be detected early and managed accordingly (8).

As in other studies (1,2,10,13), this study has shown that hypertensive disease in pregnancy is commoner among primigravidae. They accounted for 52.3% of all the patients with hypertensive disease in pregnancy. It is also commoner in young patients, 44.7% of all the patients were aged between 15-25 years with a mean age of 23 years. This may be due to the fact that these young primigravidae seek antenatal care late hence problems are not detected early.

86.4% of the patients received antenatal care. 44.6% had attended City Council clinics, which are the main catchment area for those who deliver at Pumwani. 72.2% of the patients had booked antenatal before the 28<sup>th</sup> week of gestation. This would tend to imply that these patients had good antenatal care. Only 13.6% of the patients had not had any antenatal care (25). Kapesa (1988) in a review of eclampsia in Dar-es-Salaam, suggested that prevention of eclampsia might be related to the quality of antenatal care rather than mere antenatal attendance (28). Our study indicated an advantage among the patients who had antenatal care compared to those who had none. The perinatal death rate was higher in those who did not attend antenatal care (34.2%), compared to those that attended other clinics (14.8%). Complications can be detected early and the patient admitted or treated accordingly during the antenatal period.

Most of the patients (51.4%) had spontaneous vaginal deliveries. 37.2% of all our patients were delivered by caesarean section. This caesarean section rate is very high compared to a figure of 17.3% reported by Karanja (1980) among the general obstetric patients at Kenyatta National Hospital (36). This high rate may be explained by the high rate of induction of labour in these patients. Induction of labour was done in 36.8% of the cases. The commonest reason for induction was "pre-eclampsia at term". The



commonest reason for caesarean section among these patients was failed induction of labour in 53.1% and fetal distress in 34.5% of the cases. This shows that induction of labour contributed significantly to the high caesarean section rate.

It may be that in an attempt to deliver patients with hypertension who had reached term, induction of labour is done with an unfavourable Bishop's score, hence the high rate of failure. In patients with severe hypertensive disease, delivery has to be effected as soon as control of blood pressure and convulsions are achieved. This may lead to a higher chance of caesarean section rate due to the need for urgent termination of pregnancy. However, in patients with mild or moderate disease who are at term and have an unfavourable cervical score, close monitoring can be done to allow the cervix to ripen. The hazards of a hurried caesarean section may be greater than when the pregnancy is allowed to continue and induction of labour is done appropriately (1,2,3). In our study only 17.5% of the patients had disease severe enough to warrant early intervention. Thus the high caesarean section rate is not justified. However, delivery by caesarean section was associated with good perinatal outcome in terms of fewer babies with poor Apgar Score as compared to other modes of delivery.

Assisted vacuum delivery was done in only 5.2% of the patients. This is despite the fact that it was indicated in the 56.6% of patients who delivered vaginally. Further enquiry from labour ward staff on why this was so revealed that most are not well versed with use of the venthouse. Others were of the opinion that the vacuum extractor is associated with poor fetal outcome.

The mean gestational age at delivery was 37 weeks with a range of 24 to 43 weeks. Those who were delivered very early were done so in the maternal interest due to the severity of the disease. The mean birth weight was 2555 grams, 43.1% of the babies weighed less than 2500grams. This is similar to the other studies where hypertensive disease in pregnancy has been shown to contribute significantly to the rate of low birth weight babies and prematurity (5-7).

The apparent lack of maternal deaths may be attributed to referral of severely ill patients to Kenyatta National Hospital. No record of a maternal death resulting from hypertension in pregnancy was found.

Lekha (1989) found pre-eclampsia/eclampsia contributed to 12.5% of all babies born weighing less than 2000 grams at the Kenyatta National Hospital (25). In this study, the fetal weight was found to be related to the severity of the hypertensive disease at presentation, with fetal weight reducing with increasing severity of the disease. The difference was statistically significant, ( $p < 0.001$ ). Those with severe disease may have been left too late before intervention and hence the higher chance of small for gestational age fetuses. The management of this group of patients needs to be assessed. This low birth weight has been said to be due to large placental infarcts and thrombi leading to poor placental perfusion and intra-uterine growth retardation (1,2,9). Tafari (1978) found evidence of fetal under-nutrition in patients with severe pre-eclampsia. In those babies who died there was relative undergrowth of the adrenals, spleen and liver (13). In this study, birth weight was found to be affected by maternal age, being higher in those aged 20-34 years. Normally teenagers are reported to have a higher incidence of low birth weights babies.

The mean Apgar scores were 7 and 9 at one and ten minutes respectively. The five minute Apgar scores were found to be affected by the severity of disease at presentation, a greater percentage (46.7%) having poor scores of 1-5 in those with severe disease as compared to 10.3% in those with mild disease. These differences were statistically significant ( $p < 0.001$ ).

These poor Apgar scores are due to poor placental perfusion, intrauterine growth retardation and hence increased chances of birth asphyxia. The Apgar scores were also found to be affected by maternal age, with poor scores in those patients more than 35 years as compared to other age groups. Kibaru's and Bansal's studies showed poorer scores in teenagers.

The perinatal mortality rate related to hypertensive disease in pregnancy among our patients was 222/1000. This is slightly lower than Kibaru's 236/1000. Others have shown a better outcome in their units. Tafari in Addis Ababa found a perinatal death rate due to pre-eclampsia to be only 1.3/1000 (13). Hibbard (1988) said that with good antenatal care, perinatal loss related to pre-eclampsia/eclampsia should not exceed 20/1000 (9).

Our fatality rate is obviously unacceptable. This high rate may be explained partially by the fact that our patients attend antenatal clinic late, hence late diagnosis and management. However, management of the patients may not be optimal hence there is need to reevaluate management standards.

Most (67%) of the neonatal deaths were due to respiratory distress syndrome. This may be related to the high rate of low birth weight babies with prematurity among our patients. The high prematurity rate is related to the need for early delivery due to compromised fetuses as a result of placental insufficiency. This is due to the vascular changes, which result in multiple infarcts (1,2,9). Deteriorating maternal condition also leads to the need for early delivery.

## CONCLUSIONS

1. The prevalence of hypertensive disease in pregnancy at the Pumwani Maternity Hospital is 2.9%.
2. Hypertensive disease in pregnancy has a negative fetal outcome. This was shown by a very high perinatal mortality rate of 222/1000. The mean birth weight was also low – 2555 grams. The mean Apgar scores were also low – 7 and 9 at one and 10 minutes respectively.
3. Several factors were found to influence the fetal outcome negatively. These included maternal age, where the older patients were found to have babies with poorer Apgar scores. Severity of disease at presentation had a poor prognosis



since the severer the disease at presentation, the greater the risk for perinatal deaths and also the higher rate of babies with poor Apgar score. Gestation at diagnosis of pregnancy was also found to have a negative effect. Where it was made early the fetal weights and perinatal deaths were worse. This indicates that these patients had a longer exposure to the disease process and hence the related placental problems. Some of these patients may also have had chronic hypertension as they were seen in the first and second trimester. Perinatal mortality is higher in patients with chronic hypertension than in pure pre-eclampsia due to the long term vascular changes. Patients who were delivered by caesarean section had a better outcome. Maternal level of education did not affect the perinatal outcome.

4. The management of patients with hypertensive disease in pregnancy at Pumwani is not optimal and at times the poor outcome could be avoided and minimized.

## **RECOMMENDATIONS**

1. Increase health education to the community in order to increase awareness of hypertensive disease and motivate early antenatal attendance in order for early diagnosis and management of this disorder to minimize complications and poor outcome.
2. Improve the quality of antenatal care provided in the hospitals and health centers by encouraging and making available, basic facilities for blood pressure reading and urine protein testing in order to facilitate early diagnosis and timely intervention in patients with hypertensive disease in pregnancy.
3. Aggressive management of hypertensive patients in this unit with proper monitoring antenatally and during labour, with prompt and timely interventions as required. There is need also to encourage the use of assisted vacuum delivery in

these patients, and train staff attending to these patients in the use of the venthouse.

4. Equip the laboratory at Pumwani Maternity Hospital to be able to carry out renal and liver function tests. This will add to the quality of care.

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**GYNAECOLOGY      SHORT      CASES**

## **CASE NO.1**

### **SEXUAL ASSAULT.**

**Name** :J. M. **DOA**:25/4/1999

**IP No.** :0652303 **DOD**:27/4/1999

**Age** :20 Years **Parity**:0 + 0

#### **Presenting complaints**

The patient was admitted through casualty department to the acute gynaecological ward on 25/4/1999 with a history of having been sexually assaulted by two strangers 10 hours prior to admission.

#### **History of Presenting complaints**

The patient was walking home from work at 7.00 p.m. of 24/4/1999 when she met with two strangers at Muthurwa Estate who sexually assaulted her repeatedly. She was hit with a blunt object on her head and strangled by one of the assailant before being assaulted. The assailants did not use condoms. After the attack she noted per vaginal bleeding. She reported the matter to Makongeni Police Station, and was brought to hospital by the officers.

#### **Past Obstetric and Gynaecological History**

She attained her menarche at the age of 14 years. Her periods were regular, lasting 3 to 4 days every 28 days. She had no dysmenorrhoea and the flow was not heavy. She was para 0 + 0. Her last menstrual period was on 10/4/1999. She had never used contraceptives. She was sexually active.

#### **Past Medical History**

This was not significant.

#### **Family and Social History**

She was a single lady, staying with her cousin at Buruburu. She had dropped out of school at standard 8 and she was working in a Hotel at Land-Mawe. She was the 5<sup>th</sup> born



in a family of 6 siblings. She neither drunk alcohol nor smoked cigarettes. There was no history of chronic illness in the family.

### **Physical Examination**

The patient was a young lady in good general condition, not pale, not jaundiced, not cyanosed and had no lymphadenopathy. Her temperature was 36.8<sup>0</sup>C, pulse was 76 per minute regular and of good volume. The respiratory rate was 16 per minute and blood pressure was 100/70 mmHg. Her moods were low and she was sobbing occasionally. Cardiovascular, respiratory and central nervous systems were normal.

### **Musculoskeletal system**

She had bruises on the face, forearms and legs

### **Abdominal Examination**

The abdomen was soft, not distended and there were no areas of tenderness. The liver, spleen and kidney were not palpable.

### **Speculum Examination**

In the procedure room, the patient was put in lithotomy position. On inspection, the labia majora were bruised. The vaginal walls were bruised. A sterile speculum was inserted and white creamy thick smelly discharge in the posterior vaginal fornix found. The cervix was bruised on the posterior lip. There were bruises at the posterior and anterior vaginal fornices. A high vaginal swab was taken for microscopy and culture. The vagina and cervix were then cleaned with betadine solution. Her underwear, which was blood stained was also taken for examination. Digital examination was done and the cervix was 3cm long, closed and of normal consistency. The uterus was anteverted and normal in size. The adnexa were free and the pouch of Douglas was empty. The patient was returned back to the ward to await results.

### **Impression**

A diagnosis of sexual assault was made.

**Management**

Specimens of her blood were taken to the laboratory for baseline investigations: VDRL test, ELISA for HIV antibodies and urine for microscopy and culture. Antibiotics were given- oral Norfloxacin 800mg stat, Doxycycline 100mg 12 hourly and Tabs Metronidazole 400mg 8 hourly for 5 days. In the ward there were no anti retroviral drugs and therefore she was requested to buy. She was also given postinor-2, 1 tablet stat, then another 12hours later for emergency contraception. While in the ward, the patient was counselled and emotionally encouraged. She was informed of the risks associated with the rape and the importance of further follow up. She was discharged through the patient support centre and to be reviewed at the GOPC.

**Laboratory Results**

|                      |   |                                                  |
|----------------------|---|--------------------------------------------------|
| VDRL                 | - | Negative                                         |
| ELISA for HIV        | - | Negative                                         |
| HVS: Wet Preparation | - | Moderate pus cells, and Spermatozoa were seen.   |
|                      |   | No yeast, no T. vaginalis seen.                  |
| Gram Staining -      |   | Gram negative and gram-positive rods seen.       |
| Culture              | - | Light mixed growth of coagulase negative         |
|                      |   | Staph. aureas and Beta haemolytic Streptococcus. |

**Follow-up**

She failed to turn up for the appointment as scheduled. She was lost to follow – up.

**DISCUSSION**

This was a 20 year old para 0 + 0 who was sexually assaulted. She was put on antibiotic prophylaxis ,and emergency contraceptive pills .

Sexual assault is any forced sex against one’s will including anal or oral sex, spouse or date rape and rape that did not cause obvious trauma (1). The definition of rape varies widely from state to state, legal statutes may categorize sexual assault as forcible

statutory attempted, carnal knowledge of a juvenile or a crime against nature (2). It involves the penetration of the vulva or beyond by the male genitalia without consent of the woman or when the woman lacks the ability to consent owing to physical or mental incapacity (3). Coitus with a female below the age of consent (16 years) is statutory rape. It is an offence (3).

Rape is the most under reported crime in the U.S.A.(2). The incidence of rape in hospital records is underestimated, because most of the victims do not report the offence at all. In the U.S.A. it is estimated that 9 – 24% of the women will be raped at least once in their lifetime(1). Sexual abuse is the most frequent form of child abuse, 25% of girls and 10% of boys are estimated to have been subjected to such abuse by the time they reach 18 years age (3, 4). Rapists most often choose victims, or individuals who seem vulnerable e.g. women who live nearby, are small in size or elderly or are unaccompanied, intoxicated or disabled (2). Our patient was walking home alone unaccompanied. Almost 50% of the rapists are under 25 years of age, most are repeat offenders and average more than 10 rapes before being apprehended (2).

The victim of rape is exposed to a great risk of medical, physical and psychological consequences. Few of the victims have serious physical injuries but they all suffer psychological trauma which will affect their lives and the lives of those around them. The recent epidemics of HIV and syphilis have raised grave concerns among rape survivors and professionals who care for them. 3% of rape victims develop sexually transmitted infections (4). Several factors suggest that HIV transmission during rape is possible. Condoms are rarely used during non-consented intercourse. Survivors and assailants may suffer from genital and perineal trauma that may facilitate HIV transmissions. Women often experience anal penetration during rape. Assailants often engage in behaviour associated with HIV infection e.g. sex with multiple partners and substance abuse, and up to 25% of female survivors are assaulted by multiple assailants during a single rape episode thereby increasing their risk of HIV infection (1). Our patient was raped by two assailants who did not use condoms during the rape. She also sustained genital bruises.



Rape victims may be too emotionally traumatised to wish to report the crime at the time they present to the hospital. Provision of a quiet private area equipped for pelvic examination is necessary. General physical examination is necessary since 75% of sexually abused children show evidence of other abuse (4). Rape is a legal term and the diagnosis can not be made by the physician treating the patient, he can only give evidence of recent sexual activity.

Underwear should be removed carefully and kept in a plastic bag for examination for semen, blood or pubic hair of possible assailant (3). Pelvic examination is done with a Pederson speculum. Evidence of trauma, dilatation or disruption of the tissues is sought. Accurate measurements of the genital injuries and genital anatomy of victims of rape is necessary. This can be best achieved by use of a colposcope (5). The extent of injuries in our patient was assessed by visual inspection.

Sterile swabs are used to collect materials from the vulva, vaginal walls and the cervical portio (4-6). The swabs are examined for the presence of semen (spermatozoa) and infection such as gonorrhea. Blood is drawn for VDRL test for syphilis and for ELISA for HIV at the time of presentation to provide a baseline. These were negative in our patient. The test should be repeated after 3 months of assault to rule out infection (4, 6). Unfortunately, our patient was lost to follow-up and these tests were not repeated.

After complete examination, methods of prevention of venereal disease and pregnancy should be offered to the patient including emergency (post coital) contraception (4,6) and post-exposure prophylaxis for HIV infection(7). Our patient was covered with broad spectrum antibiotics, Postinor for emergency conception and advised to buy AZT for HIV prophylaxis since it was not available in the Hospital.

Rape is a stressful situation and can lead to traumatic neurosis (3,6). The rape trauma syndrome has 2 phases: the acute stage which is the immediate reaction, and the long term phase which results in changes in lifestyle, dreams, nightmares, phobias, anxiety



states and psycho-sexual violence. Survivors need ongoing psychological and practical support and arrangement should be made for future counselling sessions. Young children usually do relatively well if the family understands and can cope with the situation (6). Our patient was discharged through the patient support centre for further counselling and follow-up.

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## **CASE NO. 2**

### **INCOMPLETE ABORTION - MANUAL VACUUM ASPIRATION**

**Name** :C. K.                      **LMP** :6/2/2000  
**Age** :19 Years                      **DOA** :15/5/2001  
**IPNO.** :0736754                      **DOD** :16/5/2001

#### **Presenting complaints**

The patient presented with complaints of vaginal bleeding for 4 days, and an amenorrhea of 13 weeks.

#### **History of Presenting Illness**

The patient was admitted through casualty department with complaints of lower abdominal pains and per vaginal bleeding for a duration of 4 days. The symptoms were of sudden and spontaneous onset. There was no history of interference with the pregnancy.

#### **Obstetric and Gynaecological History**

The patient was para 0 + 0. She had her last menstrual period on 6/2/2001. At the time of admission she had an amenorrhea of 13 weeks. She had her menarche at 16 years and the cycles were regular, with 3 days of flow and periods coming after every 28 days. She had no history of dysmenorrhea and she had not used any contraceptives before.

#### **Past Medical History**

This was insignificant.

#### **Family and Social History**

She had dropped out of school in standard 6. She was worked as a house girl in an estate in Nairobi. She neither smoked cigarettes nor drunk alcohol. There was no chronic illness in the family.

#### **Physical Examination**

She was a young lady in good general condition. She was not pale not jaundiced, not cyanosed and had no lymphadenopathy.

Her temperature was 36.8<sup>0</sup>C, pulse rate of 74 beats/min. Regular and of good volume.

The respiratory rate was 20/minute and blood pressure was 110/70mmHg.

The respiratory cardiovascular and central nervous systems were normal.

### **Abdominal Examination**

The abdomen was not distended, and was moving with respiration. There was mild hypogastric tenderness. There were no masses palpable. Bowel sounds were normal. The liver, spleen and kidneys were not palpable.

### **Pelvic Examination**

She had normal external genitalia. A speculum examination was done and showed a healthy vaginal canal, and there was *slight bleeding from the cervix*. On digital examination, the vaginal wall was healthy and moist. The cervix was soft and the cervical os was admitting a finger. Products of conception were felt in the cervical canal. The uterus was approximately 12 weeks in size. The and adnexa were free and pouch of Douglas was empty. There was fresh blood on the examining fingers.

### **Diagnosis**

Incomplete abortion in a teenager.

### **Management**

The patient was informed of the problem and the mode of management and hence she gave consent for evacuation of the uterus.

### **Uterine evacuation**

The patient was taken to the procedure room in Ward 1D. On the couch, in the lithotomy position, vulvo-vaginal toilet was done and the patient was draped with sterile towels. Pelvic examination was repeated and confirmed earlier findings.



A sterile Cusco's speculum was gently introduced into the vagina exposing the cervix, which was then held with a tenaculum forceps. The uterine cavity was evacuated using a Karman syringe with cannula size 10, to aspirate the products of conception, which was done systematically in a clockwise direction until a gritty feeling of the uterine wall was noted. About 100mls of products of conception were aspirated and were not foul smelling. The instruments were removed and vulval toilet was repeated. The patient was repositioned to the supine position and taken back to the ward.

### **Post Operative Care**

The patient's vital signs were monitored 1/2 hourly, until when she was stable. She was put on capsules of doxycycline 100mg, 12 hourly, metronidazole 400mg, and paracetamol 1gm 8 hourly. She was counselled on use of contraception and the available methods. She opted for oral contraceptives. She was also counselled on risks of contracting STI in unprotected sex. She was discharged home to continue with oral medication for 5 days.

### **DISCUSSION**

The patient presented was a teenager who was admitted with incomplete abortion and manual vacuum aspiration of the products of conception was done.

Teenage pregnancy is a growing worldwide problem both in developing and developed countries alike. The majority of teenage pregnancies are unwanted and are associated with medical, psychological and social repercussions. The main immediate consequences of an unwanted pregnancy are induced abortion, lack of parental care, personal and family disruption, adoption and abandonment (1).

The true incidence of abortion is not known because only those abortions that develop complications end up in hospitals. In Kenyatta National Hospital, Mati and Aggarwal (2) reported up to 60% of the total gynaecological emergency admissions comprised of abortions, with 16% of them suspected to have been induced.

Studies in Africa and America show that girls are becoming sexually active at increasingly younger ages and teenage sexual activity is on the increase (3,4,5,6). In Kenya, Lema found that 23.8% of adolescents aged 12 - 19 years studied were sexually experienced and 62.3% of sexually experienced girls had started coitus at 14 - 17 years of age (7).

Abortion in Kenya is illegal unless done for medical reasons. This leads to many patients refusal to admit that there was interference of the pregnancy and majority of them may present to hospital only when there are complications such as sepsis and severe haemorrhage. The patient presented gave no history of interference.

Although no age group or social class is exempt from abortion, majority of patients are usually young, single women. Aggarwal and Mati (8) found that 28% of them were below 19 years of age. 62.3% had features of interference and 79% were single. The teenagers engage in sexual activities at a stage in their life when the consequences of premarital sex are not well known to them, thus they are not psychologically or socially ready for parenthood hence the tendency to terminate the pregnancy. There is a tendency for a repeat in teenage pregnancies, which suffer a similar fate to previous ones. Studies have shown that once an adolescent has been pregnant, recurrence before age 20 years is higher.

Teenage pregnancy and abortion are indications of unprotected sex and can be used as indicators of high risk behavior. The adolescent is at high risk of contracting Sexually Transmitted Diseases (STDs). In the last decade most countries have reported a rise in STDs with rates for teenagers rising disproportionately (9). In rural Kenya 58% of teenagers examined were found to suffer from at least one type of STD (10). They do not relate their sexual behaviour to the risk of contracting STDs including HIV. Their knowledge of HIV/AIDS as a STD does not appear to have deterred them from becoming sexually active, having multiple partners and indulging in unprotected sex (9).



The diagnosis of incomplete abortion is based on history provided by the patient and clinical examination. Patients may present with lower abdominal pains, per vaginal bleeding, with a history of amenorrhea. The cervix is found open with products of conception in the cervical canal. Ultrasound may be done to confirm presence of retained products of conception. The patient presented was diagnosed clinically from history of amenorrhea, abdominal pain and vaginal bleeding. Pelvic examination revealed a bulky uterus with a dilated cervix and products of conception were felt in the cervical canal.

Management of incomplete abortion is by surgical evacuation of uterus. Dilatation and sharp curettage can be done but this has the risk of uterine perforation and if curettage is vigorous may lead to Asherman's syndrome. Suction curettage is preferred to sharp curettage since it is faster and safer. A plastic flexible Karman cannula with a syringe as a source of vacuum suction is portable, inexpensive and convenient for out patient use. Kizza (11) at Kenyatta National Hospital found that the technique was effective and safe especially in early gestation and anaesthesia was not required. The patient presented had MVA done using Karman's cannula and syringe successfully. Kizza also noted that with use of MVA the hospital stay was reduced to a minimum with an average of 6 hours (9). The patient was admitted at night and treated and discharged the following morning.

The complications that follow MVA for incomplete abortion are mainly sepsis and haemorrhage. Sepsis is usually due to prolonged duration from time of onset of abortion to the time of the procedure, as well as to the circumstances leading to the abortion. Makokha (12), reviewing maternal mortality at Kenyatta National Hospital noted that of 22.2% deaths were due to post abortal sepsis, 85% of them had evidence of interference. Severe or persistent haemorrhage is not common after the procedure and is normally associated with advanced gestation where the amount of retained products of conception may be high. Mutungi found that 66.6% of teenagers induced the abortion after 12 weeks gestation, which was thought to be due to illegality of the procedure and lack of financial ability to do it early. The patient presented had none of these complications after MVA.



There are no effective methods available to reduce sexual activity among teenagers with sexual experience. Contraceptive use among the teenagers is limited on one hand while on the other sexual activity is increasing. Most Family Planning programs in Kenya and in other African countries do not have guidelines on contraception for teenagers. Contraceptive services are discouraged for them in the belief that providing them would be encouraging immorality. Instead easy access to modern, effective contraceptive methods should be made available to every woman who is capable of achieving pregnancy. In Kenya, contraceptive use among teenagers is quite low and was found to be between 2 - 11% in various studies (5, 14).

The concept of post abortion care (PAC) has gained wide acceptance as one way to improve services provided to women with complications from spontaneous or unsafely induced abortions, to break the cycle of repeat abortions. Most contraceptive methods can be used in the 1st 7 days after 1st and 2nd trimester abortions. Safe post-abortion contraceptives for teenagers include barrier methods, oral pills, injectables and Norplant. The patient presented was counselled and offered these options of family planning.

In Kenya there is a need to put aside more resources to educate the teenager on risks of unprotected sex and unwanted pregnancies and if possible encourage them to use family planning methods. Family life education should be encouraged in our schools, so as to enlighten majority of the teenagers on the risk and dangers of unprotected sex in this era of HIV/AIDS.

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### **CASE 3**

## **ENDOMETRIAL CARCINOMA: TOTAL ABDOMINAL HYSTERECTOMY AND BILATERAL SALPINGO OOPHORECTOMY**

**Name:** N.N.K                      **Date of admission:** 23/04/99

**Age:** 63 years                      **Date of discharge:** 2/08/99

**IP No:** 0387878

### **Presenting complaint**

She came to Kenyatta National Hospital as a referral from Nazareth Missionary Hospital with a history of postmenopausal bleeding for 3 months, backache and lower abdominal pain with foul smelling vaginal discharge. She had no prior history of any major medical ailment and no previous surgery.

### **Obstetric/Gynaecological history**

Her last menstrual period was 15 years before admission. She had never used any family planning method. She was para 6+2 all her children are alive and well.

### **Social and family history**

She lived in Ruai with her children divorced her spouse after her 1<sup>st</sup> child was born and bore other children thereafter. She was a peasant farmer, sniffed tobacco and drank alcohol.

### **Physical examination**

She was an elderly lady of cheerful disposition, in good general condition, no wasting, no pallor, fever, lymphadenopathy or oedema.

**Vital signs** were BP: 120/70mmHg, Pulse rate of 70 beats per minute regular and of good volume.

### **Abdominal examination**



The abdomen had no scars, nor therapeutic marks and no distension. On palpation, there was no hepatosplenomegaly, a suprapubic mass corresponding to a 12-week gestation was found in the suprapubic region. It was mildly tender.

### **Vaginal examination**

She was circumcised, had foul smelling brownish discharge. The cervical os was open and expelling foul smelling tissues. The adnexa was free and the pouch of Douglas was empty. The uterus was freely mobile and bulky.

Her respiratory, cardiovascular and central nervous system were normal.

### **Investigations**

- Haemogram: Hb 13.1gm/dl,
- White cell count  $6.8 \times 10^9$  g/l
- Platelet count  $440 \times 10^9$  g/l
- Sodium 133mmol/l
- Potassium 4.8mmol/l
- Urea 2.1 mmol/l
- Cervical punch biopsy, histology showed chronic and acute cervicitis
- Blood group, A rhesus positive.
- Endometrial curetting biopsy: Histology report showed infiltrating poorly differentiated carcinoma exhibiting both adenoid and squamous differentiation.
- Intravenous pyelogram: Bilateral dye excretion with no abnormalities of the renal calyces or ureters or bladder. Normal I.V.P.

**Impression:** endometrial carcinoma.

### **Management**

On admission she was prepared for examination under anaesthesia and staging of disease. In theatre a cervical polyp was found and excised, again the adnexa was free and the uterus bulky. Endometrial curetting was done and the tissues taken for histology. The

histology showed infiltrating poorly differentiated carcinoma exhibiting both adenoid and squamous differentiation. She was thereafter prepared for total abdominal hysterectomy and bilateral salpingo-oophorectomy. This was done on 28<sup>th</sup> April, 1999.

Intra-operatively we found a uterus of size corresponding to a 12 week gravid uterus, which was soft. The left fallopian tube was distended and the fimbrial end closed with what later turned out to be pus. Both ovaries were grossly normal and the right fallopian tube was also grossly normal. Total hysterectomy was done, inclusive of 1/3 of the vaginal cuff, as well as bilateral salpingo-oophorectomy.

**Histology specimen:** Uterus, fallopian tubes and ovaries.

**Histology report.**

There was a tumour mass on the uterine fundus, with a depth up to the myometrium, and breadth of 5cm. Another tumour mass was located just above the cervical os, the cervix was free of tumour. The histological type was poorly differentiated adenosquamous carcinoma.

**Post operative course**

She had good wound healing immediately post operatively, but on her 7<sup>th</sup> postoperative day she had foul smelling vaginal discharge. Vaginal swab for microscopy and culture yielded no bacteria growth with moderate pus cells. She was treated with Clindamycin intravenously, and did well. Adjuvant radiotherapy treatment was started.

**DISCUSSION**

The patient was 63 years old, 15 years post menopausal who presented with post menopausal bleeding. Diagnostic fractional curettage was done and histology showed poorly differentiated adenosquamous carcinoma of the endometrial tissues. She was operated, bilateral salpingo-oophorectomy and total abdominal hysterectomy was done. Post-operatively she was started on external beam radiotherapy.

Endometrial carcinoma is one of the commonest malignant tumors of the genital tract, in USA it is the most frequent genital tract malignancy (1) and is said to occur in 1 to 2 cases per 1000 post-menopausal women (2). The peak incidence of onset in the 6<sup>th</sup> and 7<sup>th</sup> decade of life but 5% occur before age 40 (1). Increase use of oestrogen replacement in post-menopausal woman in developed countries led to an increase in endometrial carcinoma in the 1970s (2).

The aetiology of endometrial carcinoma remains unknown but oestrogen is strongly implicated. In younger women it occurs predominantly in presence of estrogen excess such as ovarian tumor secreting estrogen, theca cell and granulosa cell tumours, hormone replacement for gonadal agenesis and ovulation induction. Patients with primary ovarian failure have been singularly devoid of endometrial carcinoma (2). Obesity is the risk factor commonly associated and again estrogen is implicated, one theory is that circulating androgens, principally in fatty tissues are converted to estrogen. (2), creating a milieu favorable to formation of endometrial carcinoma. This malignancy is also associated with diabetes mellitus, hypertension and late menopause. Diabetes and hypertension also occur more frequently in the same age group.

Use of progestin with estrogen has been observed to protect against endometrial carcinoma in hormone replacement therapy (3). The risk of endometrial carcinoma is reduced if the lowest dosage of estrogen is used in an interrupted pattern (2). Due to the dangers of osteoporosis, routine estrogen replacement is more commonly used, an endometrial biopsy is prudent management before therapy to rule out occult atypia or adenocarcinoma. It is suggested that prolonged estrogen can have a neoplastic effect on DNA metabolism in sensitive endometrial tissue.

Abnormal uterine bleeding occurs in 80% of patients with endometrial carcinoma is therefore the most important warning sign. In post-menopausal women, this may be intermittent spotting, premenopausal years it may present with excessive menstrual flow. About 20% of patients with post menopausal bleeding have underlying cancer (1) and this increases with age, at age 80 cancer is responsible in 50-60% of cases. Lower



abdominal pain and cramps may occur secondary to uterine contractions caused by entrapped detritus and blood behind a stenotic cervical os, these uterine contents may become infected forming an abscess as in this patient. Physical examination is usually unremarkable except to confirm uterine bleeding by speculum examination unless the disease is extensive.

Dilatation and fractional curettage is the definitive procedure for diagnosis of endometrial carcinoma. This should be under general anaesthesia to afford an opportunity for thorough pelvic examination. An assessment of uterine depth is done for staging purposes. Once obvious cancer is present the procedure is terminated, a tissue taken for histology to avoid uterine perforation and intraperitoneal contamination with malignant cells, blood and bacteria.

Other modalities for diagnosis are less sensitive and include:-

- Out patient endometrial biopsy, this is a cheaper procedure but it is inaccurate for diagnosis polyps.. and endometrial hyperplasia as well.
- *Aspiration biopsy with an aspirating or non aspirating curetting false negatives are also common, it has an accuracy of 70 to 80% (1,2).*
- Aspiration curetting is the most accurate modality of outpatient diagnostic procedures.
- Endometrial cytology, its accuracy is dependent on the skills of the pathologist. Papanicolaou smear generally detects only 50 to 60 of endometrial carcinoma (2).

Use of hysteroscopy has not been found useful in routine practice.

Differential diagnosis include leiomyoma, endometrial hyperplasia, ovarian tumours, atrophic vaginitis, cervical cancer. Though abnormal uterus bleeding is common during perimenopausal years, any patient presenting with the symptom requires a thorough endometrial evaluation so as not to overlook cancer. Disease staging for malignancy is important for purposes of management and prognosis. Factors influencing staging include:-

- Uterine cavity length
- Extension of disease extension beyond the uterus and pelvis.

The degree of tumour differentiation laces it in:-

- Grade I – highly differentiated
- Grade II – differentiated with solid areas
- Grade III – highly undifferentiated

This is by the FIGO classification. Stage I includes tumours confined to the uterine cavity, in stage Ia the uterine cavity is < 8cm length and stage Ib, the uterine cavity is > 8cm. In stage II the tumour involves the corpus and cervix. Stage III disease tumour extends outside the uterus but within the true pelvis stage iv extends beyond the true pelvis (2).

The vast majority of endometrial neoplasms are adenocarcinoma's comprising of 80% to 90% of all endometrial carcinoma (2,50 which often have foci of squamous metaplasia. Other histological types occur but are less frequent. Adenosquamous carcinoma as observed in this patient is the next most common and accounts for 10% of endometrial carcinoma. It comprises of a mixed glandular and squamous carcinoma.

Endometrial carcinoma has one of the best prognosis amongst malignancies of the genital tract. This is mainly because up to 75% are diagnosed in an early stage I and are well differentiated or moderately differentiated lesions (2). The five years survival rate of women with endometrial carcinoma is good with about 75 to 80% in most reported series that include all stages (4), but it is even better in stage I and II. The important factors related to the prognosis of carcinoma of the uterine corpus include, age at the time of diagnosis, histologic grade of tumour, tumour volume, including uterine size and depth of myometrial penetration and extent of disease at the time of diagnosis, including cervix, metastasis to pelvis and para-aortic lymph nodes.

The age group diagnosed before menopause have a 5 year survival of 88.8% for more than 5 years and post menopausal women have a lower survival rate which declines up to

after 15 years at 58.3% (2). This may correlate to early diagnosis of well differentiated tumours without myometrial invasion in younger women.

Adenosquamous carcinoma occurs in an older age group than adenocarcinoma and is more frequently associated with a poorer prognosis (2). The degree of histologic differentiation is a reliable indicator of the prognosis of the disease. This rating system was introduced by Broden in 1941 (2). The current classification includes 3 gradations, and correlates with patient survival. There is a decrease in survival with a decrease in tumour differentiation. This differentiation has also been shown to relate to lymph node metastases (2), Lewis found that only 5% of patients with well-differentiated tumours had positive pelvic nodes, 26% of patients with poorly differentiated tumours had lymph node metastasis. They also found that no pelvic nodes were positive if the tumour was confined to the endometrium i.e. only to the superficial myometrial layer.

In the Gynaecologic oncology group study (6) it was found that 80% of stage I tumours were grade 1 and 2 lesions, 40% were confined to the endometrium and 20% showed deep myometrial involvement. In presence of deep myometrial involvement, this may be occult or clinical.

Other recent studies, DNA indices and population in tumour cells have been found to be more accurate indicators of disease prognosis (4). This is done by processing curettage material for flow cytometric DNA analysis. This was found to identify a smaller high risk group and reduce unnecessarily aggressive therapy, which may be detrimental especially for older patients. The finding of this study demonstrated a high risk group with both deep myometrial invasion in presence of two or more DNA population. This technique might however not be applicable locally where funds may not be available for such technology.

Three basic methods of treatment, surgery irradiation and a combination of the two are used for patients with endometrial carcinomas, based on the extent of the tumour at the time of diagnosis.



Hysterectomy is considered the definitive method of therapy for endometrial carcinoma. Bleeding is an early sign of the disease and most patients present in stage 1 and 2 disease and can be adequately treated with simple hysterectomy (1). Patients with endometrial carcinoma are often elderly, weak, may be anaemic, diabetic or hypertensive. Surgery thus needs preparation to avoid mortality. Abdominal hysterectomy is the preferred mode of surgery to allow for examination and sampling of para-aortic and pelvic nodes.

If medical condition does not allow, then transvaginal hysterectomy though not optimal may be done for early disease with a small uterus and well differentiated tumour.

This patient had a Stage IB grade 3 malignancy and thus had a likelihood of spread to pelvic and para-aortic nodes of 25 to 43.7% (2). She required adjuvant radiotherapy treatment. This may be administered pre-operatively, so as to sterilize the tumour and reduce spread during surgery. Prior to surgery blood supply is always intact and the oxygenated tumour bed can receive maximum effects of the irradiation. This is usually intracavitary pre-operative irradiation has been observed to reduce the incidence of vaginal recurrence whether it is given intracavitary or by external irradiation (7). Jones showed that without prior irradiation vaginal recurrence rate was 10.3%, with post-operative vaginal radium or caesium this reduced to 5.2% pre-operative irradiation reduced it even further to 4.6%, when both pre-operative and post-operative irradiation is given recurrence reduces to 4.4 to 1.5%. Prior to the use of pre-operative irradiation, the cervix was sutured prior to hysterectomy to reduce dissemination of tumour at cellular level.

The ideal management of Stage 1b malignancy should include preoperation. This can be through a vaginal colpostat for 48-72 hours with bladder and rectal displacement. Alternatively, the patient may be treated with external irradiation. Surgery is performed 8-72 hours following intracavitary irradiation or four weeks following completion of external and intracavitary irradiation (2). Surgery is inclusive of total abdominal

hysterectomy, bilateral salpingo-oophorectomy, evaluation of the pelvic and para-aortic lymph nodes with any suspicious nodes. Positive lymph nodes or deep myometrial involvement is an indication for post-operatively. Delay of emergency to four weeks after full extend boom irradiation to allow pelvic tissue to recover.

Radical surgery for endometrial carcinoma such as Wertheim's hysterectomy and pelvic lymphadenectomy causes excessive morbidity without significant therapeutic benefit especially since these patients are elderly and have multiple co-existing medical ailments.

The technique of abdominal hysterectomy for endometrial carcinoma defers from surgery for benign disease. Closure of the cervix with a figure of 8 stitch is used in the absence of pre-operative radiation. The fallopian tubes are clamped at the cervical position of the uterus to prevent retrograde passage of the tumour cells during the operative procedure. Since ovarian metastases occur in 5% to 10% of all cases the ovaries and tubes should be removed, regardless of patient's age or stage of tumour. The fascia of the cervix and lower uterine segment is removed as it is rich in lymphatics. A 1cm to 2cm cuff of vaginal fornix should be removed with the cervix to ensure that adjacent lymphatics have been removed (2,8). Endometrial carcinoma spreads through the regional lymphatics and blood stream to recur at the lateral pelvic wall or outside the pelvis. Thus parametrial dissection is not useful in controlling the disease.

For patients with medical contraindications to surgery or unresectable lesions, irradiation in primary method of treatment, though survival rates are lowered by 10% to 20% (2).

Recurrence rates were found to be 7% when there was no definable extrauterine disease at the time of original surgery (2) and 43% in presence of disease outside the uterus. The most frequent occurrence sites were the upper vaginal, uterus pelvic lymph nodes, para-aortic nodes and lumps. Recurrences are also higher (5%) in those treated with primary surgery alone compared to those treated with radium (3%). Pre-operative radiation has been shown to lower occurrence in high-risk cases (2,6,7). Recurrence has survival rates of 15% to 20% inspite of treatment, thus they have a poor prognosis (2).

Other modalities of therapy include progestins, which are the main systemic agent in treatment of advanced endometrial carcinoma. Objective remission occurs in one third of the patients, but they are only effective in presence of progesterone acceptors. The anti oestrogen tamoxifen has also been used.

Other chemotherapeutic agents have been used with limited response rates of approximately 36.8% (12). Those drugs include Melphalan, 5-fluorouracil, cyclophosphamide, Cisplatin and Adriamycin. There is no increase in response to multidrug or single drug use. These drugs may be used in patients with unfavourable outcome, those with poorly differentiated tumours, and absent progesterone receptors (2).

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#### **CASE 4**

#### **REVERSIBLE CONTRACEPTION: NORPLANT INSERTION**

**Name:** J.N.                      **F.P. NO**                      : 50/01

**Age:** 26 years                      **Date of Insertion:** 07/06/01

#### **Presenting History**

This lady was 26 years old, para 1+0 who presented to the family planning clinic requesting of Norplant as a method of contraception. She knew about the method through talks given at the antenatal clinic in her last confinement. She had been on the combined oral contraceptive pills from six month postpartum and came for Norplant insertion during her menses on day 3. She was informed that the methods provided contraceptive for a duration of 5 years. The method of insertion and removal was outlined and side effects outlined. She noted that she had occasional headaches but had not been hypertensive or suffered any known chronic illness. She wished for a prolonged period of contraceptive use so as to bring up the first child. This had been discussed with her husband.

#### **Obstetric/Gynaecological history**

She was para 1+0, last delivery was in February 1995. She had been on microgynon from September 1995 and had regular menses. Her last menstrual period was on 04/06/2001.

#### **Past medical and surgical history**

Nothing significant.

#### **Family & Social history**

She was married and worked as a receptionist. Her husband worked as a businessman. She did not smoke cigarettes.

#### **Physical examination**

She was in good general condition, she had no pallor, fever, lymphadenopathy nor oedema. Weight: 60kg

Central nervous system - Normal

Pulse, 80/min, regular pattern

Blood pressure: 110/70mmHg

No abnormal heart sounds were auscultated.

Breasts: Normal there were no lumps

Abdominal examination – normal

Pelvic examination: not done, she was on her menses and had been on contraceptives.

A Pap smear was planned on her next visit.

### **Insertion procedure**

She was asked to change into a hospital gown, wash her left hand and lie on the examination couch. The left arm (she was right handed) was extended on an arm rest placed on the examination table. A distance of 6 cm above the elbow on the medial side of the arm was selected. This area was scrubbed with savlon and draped with sterile cloths. An aperture was left in the selected area of insertion 5cc of Lignocaine 1% was injected subdermally in a spiral manner in an area of about 75 degrees.

The capsules were counted and confirmed to be six capsules in total. A small shallow incision approximately 2mm wide was made with the scalpel. This incision just penetrated the dermis. The trochar was introduced into this incision at a shallow angle tilted upwards towards the surface of the skin. The trochar was moved forwards stopping at the second mark on the trochar. The stylet was removed and the first capsule loaded in down to the top of the hub and pushed in with the plunger towards the tip of the trochar until resistance was felt. Then the trochar was withdrawn steadily holding the stylet upto the level of the lower arm and the stylet withdrawn. The first capsule was now in place and free of trochar. This was confirmed digitally. The trochar was kept subdermally and moved by about 15 degrees and the above motions were repeated, taking care not to place them over each other but in a fan like manner.

The procedure took ten minutes. The trochar was completely withdrawn after the last and sixth capsule was inserted. There was no notable bleeding. A band aid was placed over the incision and dressing draped over it. The client was given paracetamol tablets to take for two days, two three times daily. She was advised not to carry heavy loads with that arm for a week. To remove the band aid on the 7<sup>th</sup> day. During that week she was advised to keep water from the dressing site. She was kept for five minutes at the clinic and asked to come for follow-up after one week or before if she had any problems.

## DISCUSSION

The Norplant system is highly effective long acting hormonal contraceptive method for women. Norplant was developed and researched by the population council from 1966, it became available in Kenya in 1986 in the Machakos project area in collaboration with population council New York (1). Norplant now has several delivery units including the family planning clinic of Kenyatta National Hospital. The United States Food and Drug Administration (F.D.A) approved marketing of Norplant in 1990 (2).

Norplant is an effective, long lasting, reversible contraceptive that provides protection against pregnancy for five years. Six thin silastic capsules made of a soft silicone and filled with a synthetic hormone are inserted just under the skin of a woman's upper arm or elbow in a minor surgical procedure (1). Silastic has been used in surgical applications since 1950. Contraceptive protection begins within hours of insertion. Each capsule is 34mm long and 2.4mm wide and contains 36mg Levonorgestrel, the synthetic progestin used in oral contraceptives. Blood levels of Levonorgestrel are slowly released through the polydimethylsiloxane tubing at a rate of 85 micrograms per day at first. Within one year, the concentration of the progesterone in plasma stabilizes at an average level of 30mcg per day giving a plasma concentration of 0.3 monograms per millilitre. This corresponds to the level reached 12 hours after the intake of a Levonorgestrel containing minipill (4). The plasma levels drop off rapidly after implants removal and return to normal and full fertility follows. The user may attempt a pregnancy



immediately during the next menstrual cycle. In clinical study with good follow-up of 95 women who had implant removal in order to conceive, 50% became pregnant within three months, 86% at one year and 93% at two years (4).

In a pooled monitored study by population council (40 the annual pregnancy rate was below 0.5 per 100 during the first two years of Norplant use (2,40. After the second year of use differences in pregnancy rates have been noted to be weight dependent. Women who weighed 70kg or more at insertion experienced pregnancy rates twice as high as those who weighed 60-69kg. Cumulative pregnancy rate for 3 years was 3.9 per 100 users (4,8). Ectopic pregnancies occurred among Norplant users at an average of 0.13 per 100 women years (3,40. The risk of ectopic pregnancy may increase with duration of use and possibly with weight.

Norplant works through Levonorgestrel a progestin widely used in the combined oral contraceptive and the minipill. The mechanisms of action are several (1) suppression of ovulation (2) thickening of the cervical mucus, (3) inhibiting sperm penetration into the uterus, (4) it is also thought to cause endometrial atrophy thus inhibiting implantation. In addition, Levonorgestrel may also depress progesterone in the luteal phase.

Norplant does not contain estrogen and can therefore be considered safer than the combined pill for many women for whom estrogen is not recommended due to cardiovascular effects. No increase in blood pressure had been observed on the average. Minor increases and decreases in total cholesterol and HDL cholesterol have been observed (4,7). These implant systems require no effort of compliance by the users and achievement of effectiveness is uniform (5,6). Norplant system can be used as a method of contraceptive by almost all women, who are seeking continuous contraception, long term birth spacing, a method not related to intercourse, does not require to be remembered daily, have desired family size but does not want to be sterilized, or not recommended to use estrogen containing contraceptive (8).

Contraindications include, acute liver disease, jaundice unexplained vaginal bleeding, history of thrombosis, history of cardiovascular disease (2,3,8), or if she is pregnant or has sickle cell anaemia. Proponents against Norplant fear that it can be used to abuse a woman right to control her fertility. This was observed in a court judgement U.S.A ruling that a Norplant insertion be part of the conditions of releasing a mother charged with child abuse. In China health workers have been noted to be reluctant to remove he implants on patient request.

Norplant can be given to the mother who is partially breast feeding from 6 weeks postpartum. If she is fully breastfeeding the risk of pregnancy during lactational amenorrhoea is very low, less than 25 in the first 6 months, less than 7% in the first 12 months but a method should be started from the first menses (not in first 56 days) {3}. Based on the fluctuation as human sex hormones in the first 6 weeks of live and the *immaturity of the neonatal liver for the metabolism of exogenous steroids it is considered prudent to avoid progestin only contraceptive until a breast feeding woman is at least 6* weeks postpartum or postabortal. There is no known clinical thrombogenic effect of progestin-only contraceptive (3,8). Postabortion insertion should be within 7 days as ovulation starts immediately after postabortion.

Norplant may be used at any age at which the woman is at risk of pregnancy, but there is a low risk of osteoporosis for women within 2 years post menarche in later life (3). Norplant has the added advantage of reduced menses and often results in an increase in haemoglobin levels (4). Insertion and removal of Norplant are simple minor surgical procedures that require local anaesthesia, a small incision 2mm for insertion, 4mm for removal. The only other instruments required are sterile dressing, syringe, needle 4-4.5cm long, trochars, plunger, scalpel, band aid, sterile gloves, set of 6 capsules in a sterile pouch and an examining table with an arm support (9).

Good counselling is essential to the provision of all contraceptive methods and Norplant is no exception. The client should have accurate information on the product, discuss all her fears and concerns, knowledge of alternative methods, provision of



continous care. This client was counselled well and she wished to have prolonged child spacing. Knowledge of side effects and failure rates should be communicated to the client.

The best time for insertion is any time one is reasonably sure that the client is not pregnant. This can be within 7 days of menstrual cycle, for a client on an alternative method at your clinic such as this client. Removal of the Norplant should be easily available to the client. This could be upon demand, wish to conceive or due to side effects. Irregular menstrual bleeding is the most common side effect of Norplant. Intermenstrual spotting or amenorrhoea may occur. The altered bleeding pattern usually becomes more regular towards the end of the first year of use. If amenorrhoea follows a period of regular menstruation pregnancy should be ruled out bearing in mind the chances of ectopic pregnancy. If she is pregnant the implants should be removed if not she is reassured. If counselling and reassurance are not sufficient for a woman with continous spotting or bleeding short term combined contraceptives may be used for 7-21 days of ibuprofen. Heavy bleeding is uncommon but may be controlled by administration of combined oral contraceptives or estrogens (3). This assists in rebuilding the endometrium and ibuprofen blocks prostaglandin synthesis and decreases uterine contraction.

Transient ovarian cysts, have been reported in about 105 of users (4). Headaches, nervousness, nausea, change in appetite, depression and other mood changes, dermatitis, acne, hirsutism may occur or worsen. Infection at the implant insertion site may occur and is an indication for removal and treatment. Other indications for removal include exceptional strong headaches or migraines occurring for the first time, thrombophlebitis, increased blood pressure, pregnancy, liver disease and pre-operatively in case of long term immobilization. Drugs that may reduce Norplant efficacy include barbiturates, phenytoin and phenylbutazone, isoniazid, carbamazepine and rifampicin (2,4,8,9). The progress of Norplant has set the stage for the development of other implants. Norplant II is already in use, it consists of two rods slightly longer than Norplant capsules, they contain Levonorgestrel embedded homogenously within a silastic rod, covered by a thin



sheath of plain silastic. This makes for easier insertion and removal (9). Also under development is capronor, which will be a biodegradable implant and may be removed or the capsule biodegrade gradually and is absorbed by the body.

After insertion of Norplant it is routine at our family planning clinic in Kenyatta National Hospital for the client to be asked to revisit after one week for examination of the insertion site. Thereafter she is asked to come for annual check-ups and any time during clinic hours that she had a problem concerning her contraceptive method.

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## CASE NO. 5

### VESICOVAGINAL FISTULA – SUCCESSFUL REPAIR

|                |                 |
|----------------|-----------------|
| Name :R. M.    | Parity :1 + 0   |
| Age :20 Years  | DOA :18/07/2001 |
| IPNO. :0735642 | DOD :29/07/2001 |

#### Presenting Complaints

Patient presented with complaints of leakage of urine for 8 months duration.

#### History of Presenting Complaints

The patient was referred to our gynaecology out patient clinic from Makindu Hospital with a history of leakage of urine since her last delivery on 20<sup>th</sup> December 2000. The patient had obstructed labour for three days and was delivered by emergency caesarean section of a macerated stillbirth, with a birth weight of 4.7kg. An indwelling catheter was left in situ for one week and on removal she started leaking urine.

#### Past Obstetric and Gynaecological History

She attained her menarche at the age of 13 years. Her periods were regular, with flow for 3 days in a cycle of 28 to 30 days. Since her last delivery she had not resumed her menses. She was para 1 + 0 and her last delivery was in December 2000 when she had a still birth. She had not used any method of contraception.

#### Past Medical History

This was not significant.

#### Family and Social History

She was a housewife, and stayed with her husband at Kibwezi. Her husband was a casual labourer. She neither smoked cigarette nor drunk alcohol.

**Physical Examination**

She was in good general condition and had no pallor, oedema, jaundice or lymphadenopathy. She was afebrile and her blood pressure was 110/70mmHg, pulse rate was 72/min. regular and of good volume and had a respiratory rate of 20 per minute. She was 1.65m tall, and weighed 67 Kilograms.

**Central nervous system**

This was essentially normal. She had no foot drop or sensory or motor deficits. Respiratory and cardiovascular systems were essentially normal.

**Abdominal Examination**

Abdomen had normal fullness and was moving with respiration. She had a midline sub-umbilical surgical scar. There was no area of tenderness or any masses palpable.

**Speculum Examination**

The external genitalia were normal. The perineum had excoriation, was wet and had an offensive ammoniacal odour. The anterior aspect of the vaginal wall had a defect at mid vagina through which urine was leaking. The defect measured. 2cm x 3 cm. The cervix was healthy. There was no vaginal discharge or bleeding. Digital examination was done and found that the lower third of the vagina was stenotic with defect in the mid vaginal region. The cervix was firm with a closed os and the uterus was anteverted and of normal size.

**Impression**

A diagnosis of vesicovaginal fistula was made.

**Investigations**

|    |            |             |   |                          |
|----|------------|-------------|---|--------------------------|
| 1. | Haemogram. | Haemoglobin | - | 11.2g/dl                 |
|    |            | WBC         | - | 4.2 x 10 <sup>9</sup> /l |
|    |            | Platelets   | - | 241 x 10 <sup>9</sup> /l |



|    |                       |   |                          |
|----|-----------------------|---|--------------------------|
|    | RBC                   | - | 3.2 x 10 <sup>9</sup> /l |
| 2. | Urea and electrolytes |   |                          |
|    | Urea                  | - | 4.2mmo/l                 |
|    | K <sup>+</sup>        | - | 3.5mmo/l                 |
|    | Na <sup>+</sup>       | - | 135mmo/l                 |
| 3. | Urinalysis            | - | No Abnormality detected. |

### Management

The nature of the illness and planned mode of management were explained to the patient. She was to be examined under anaesthesia and repair of the fistula was to be done. Consequently the patient gave an informed consent for the planned management. She was put on light diet and had soap enema the night before surgery as well as morning of the operation. She was starved overnight.

### Pre-operative Examination Under anaesthesia

On the day of the surgery, the patient was premedicated with intra-muscular atropine sulphate 0.6mg and pethidine 50mg. The patient was wheeled to theatre and put under spinal anaesthesia. She was placed in exaggerated lithotomy position, vulvo-vaginal toilet was done and she was draped with sterile clean towels. Examination was done and it was noted that the perineum was wet with urine. An Auvard's speculum was introduced into the vaginal cavity and urine was noted to be leaking from a defect over the mid anterior vaginal wall measuring 2x 3cm. The bladder wall was prolapsing through the defect. The fistula was about 5cm from the urethra and 2cm from the cervix. The urethra and the cervix were normal. A decision was made to repair the fistulae in lithotomy position.

### Repair of the Fistula

An episiotomy was performed after both labia major were stitched to the thighs to expose the field of operation. Two transverse incisions were made around the fistulae and the vaginal mucosa dissected away from the bladder mucosa. The fistula was closed in 2 layers, with interrupted vicryl 4/0 and 3/0 starting the repair at the two lateral ends and

proceeding medially from both sides. Then a urinary catheter was introduced to the bladder and retained. After this methylene blue dye was instilled into the bladder. The dye test was negative as there was no leakage. The vaginal wall was then closed using vicryl 3/0 and the episiotomy closed with catgut 2/0. Two pieces of sofratulle gauze were left in the vagina for 24 hours.

### **Post Operative Care**

The patient was kept in the recovery room, where her vital signs were observed half hourly for the first 2 hours, then 2 hourly. She was then taken to the ward on 4 hourly observations of blood pressure, pulse rate, respiratory rate and temperature. She was continued with intravenous fluids 500mls 4 hourly for 24 hours then oral fluid 4000mls in 24 hours. She was given pethidine 50mg intramuscularly 8 hourly for 24 hours and then oral paracetamol 1gm 8 hourly for three days to control pain. No antibiotics were prescribed. The urethral catheter was to drain continuously and if blocked to be flushed. The urinary bag was emptied 2 hourly and kept lower than the bed. She remained dry of urine and was discharged home on the 7<sup>th</sup> post operative day to come again to GOPC on her 14<sup>th</sup> post operative day for a repeat dye test and removal of the catheter.

### **Follow Up**

She was seen in the GOPC on her 14<sup>th</sup> post operative day. The incision site had healed well and a dye test was negative. The catheter was then removed. She was instructed to abstain from sexual intercourse for four months and that in her future pregnancies, delivery would be by elective caesarean section.

### **DISCUSSION**

Presented is a 20 year old para 1 + 0 who presented with VVF following a prolonged obstructed labour. Successful repair of the VVF was performed.

The actual incidence of fistulae is impossible to calculate but Harrison has suggested an incidence of 95 per 100,000 (1). By far the leading cause of vesicovaginal fistula in Africa is obstetric trauma. Mati (1982) reported that 87.8% of the cases of urinary fistula were labour related in his series (2), while Orwenyo (1984) reported a rate of 90.7% (3). This figure is in contrast to the situation in developed countries in which obstetric trauma is rarely responsible for fistulae formation (4). In developed countries the commonest causes of V.V.fistula is from pelvic surgery, cancer of the cervix and radiotherapy (4). The patient presented developed vesicovaginal fistula (VVF) after an obstructed labour which ended in a caesarean section.

Obstructed labour is primarily due to cephalo-pelvic disproportion leading to compression of the anterior vaginal wall and the bladder base between the fetal skull and the pubic bones or the posterior vaginal wall and the rectum being compressed between fetal skull and the sacrum leading to vesicovaginal fistula and rectovaginal fistula respectively (5). In this patient, only VVF occurred.

It has been pointed out that cephalo-pelvic disproportion is a common and very important obstetric complication in Africa (2,3). It has been suggested that the small pelvis may be a result of protein malnutrition in childhood. Improvement in childhood diet has been recognised as one major contribution to the reduction of cephalo-pelvic disproportion in developed countries. In Kenya the peak incidence was observed in the 20 to 24 year age group (2). Our patient was 20 years old.

The majority of women suffer the injury while giving birth to their first child and in most cases the baby is a stillborn or dies shortly after birth. The still birth rate has been reported to range between 64% and 79% (2). The patient presented sustained a fistula while giving birth to her first child and the outcome was a macerated stillbirth.

Most patients are totally or almost totally incontinent of urine. The vulva usually becomes reddened, tender and excoriated over time. The odour of urea may be so offensive as to be disgusting and embarrassing to the patient, frank depression may result



from prolonged incontinence, if repair is delayed (4). Our patient had the above symptom though she had no depression.

The fistula should be confirmed by a careful speculum examination. If it is difficult to demonstrate it, then the bladder can be filled with dilute solution of methylene blue and then the anterior vaginal wall and vaginal vault inspected. A more accurate way of demonstrating the fistula may be by use of 3 cotton tampons placed in the vagina and the methylene blue instilled into the bladder and patient request to walk about for 10 – 15 minutes, then the tampons removed for inspection (4). The patient presented had a large VVF, which was easily demonstrated on speculum examination.

Vesicovaginal fistulae are still a major cause of concern in many developing countries. Measures for prevention must include universal education, an improved status of women and improved and accessible medical services, including contraceptive services.

Obstetric fistula is associated with other complications including rectovaginal fistula. In 10% of cases (2), obstetric palsy, severe vaginal stenosis, and secondary amenorrhea may occur. The patient presented had not resumed her menses since her last delivery and hence had secondary amenorrhoea. Once a fistula is formed, continuous drainage of the bladder and control of infection with antibiotics have been shown to reduce the size of the fistula and may even lead to closure of some fistulae (5). Our patient had a catheter inserted and left in situ for 7 days post delivery. On removal, she had leakage of urine.

The repair of fistulae should not be attempted until about three months after the causative labour. This can result in reduction in the size of the fistula by spontaneous healing and allows tissue destruction to subside and revascularisation to occur (5,6). Currently, early (6 – 8 weeks) repair is advocated as soon as the slough has disappeared (7). Therefore the recommendation currently is: any woman who develops an obstetric fistula should have a catheter inserted as soon as possible. As soon as the slough has disappeared and the fistula is clean early repair (6 – 8 weeks) should be performed unless the fistula is already healed.

Post operative care is very important. The bladder should be maintained as empty as possible and must not be allowed to be distended. A catheter can be left in situ to drain the bladder continuously though some authors feel that a catheter can cause bladder trauma and hence injury to the site of repair. Suprapubic drainage of the bladder can be done (4). The patient present had urethra catheter retained for 14 days and removed after the dye test. Oral intake of at least 4 litres of water per day should be encouraged to assist in flushing the urinary tract. When this is done, antibiotics are usually unnecessary because urinary stasis is eliminated.

A successful repair is gauged by whether the woman is continent of urine. The site and extent of the fistula may affect operative success, but Lawson states that an experienced surgeon supported by competent nursing staff should be able to achieve 75% success at the 1<sup>st</sup> attempt, and a further 15% at a second attempt. Since each successive repair produces more scar tissue, successful repair at the 1<sup>st</sup> attempt is the goal. Our patient had 1<sup>st</sup> repair, which was successful.

Genital fistula is associated with many social problems such as unemployment, divorce, failure to observe religious activities and change in sexual relationship with spouse. About 89.3% of the patients have been reported to be unemployed and 10% of them attributed their unemployment to their illness. A divorce rate of 2.7% has also been observed in patients with fistula (8).

Delivery after VVF repair should be by elective caesarean section particularly if the fistula has resulted from obstructed labour. Our patient was advised to have caesarean delivery in her future pregnancies.

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CASE NO. 6

DISPLACED INTRAUTERINE DEVICE –REMOVAL BY DILATION AND CURETTAGE

|       |           |        |             |
|-------|-----------|--------|-------------|
| Name  | :M. W.    | Parity | :1 + 0      |
| Age   | :39 Years | DOA    | :18/08/1999 |
| IPNO. | :0600374  | DOD    | :20/08/1999 |

Presenting Complaint

She presented at the family planning clinic with complaints of inability to feel the threads of her intrauterine device.

History of Presenting Complaint

She presented to the clinic for her routine check up and complained that she could not feel the threads of her intrauterine device. She had had regular checks during her previous visits and confirmed the presence of the threads. This made her to be anxious and therefore an Ultrasound was done which confirmed that the device was still in situ, though the threads had cut off. Attempts to remove the device with use of alligator forceps were not successful. This necessitated removal in theatre under general anaesthesia.

Obstetric and Gynaecological History

She was para 1 + 0 with her only delivery having been in 1989. Her delivery was by spontaneous vertex. She had her menarche at the age of 12 years, her menstrual cycle occurred regularly every 28 days and the duration for flow lasted 3 – 4 days. They were painless. Her last period was on 12/08/99. She had a copper T device, which was inserted in 1995 after she had used oral contraceptive pills since 1989.

Past Medical History

This was not significant

**Family and Social History**

She was single. She was a subordinate staffer with the Department of Defence. She had no history of alcohol consumption or cigarette smoking. There was no family history of any major illness.

**Physical Examination**

She was a young lady in fair general condition. She was not pale or jaundiced and had no oedema. Her blood pressure was 110/70mHg pulse rate was 80 per minute regular and good volume. Her respiratory rate was 22 per minute with a temperature of 36.5<sup>0</sup>C.

Central Nervous, Respiratory and Cardiovascular Systems were essentially normal.

**Abdominal Examination**

The abdomen was not distended and moved with respiration. There were no scars noted. The abdomen was soft and had no palpable masses. The liver, and the spleen were not palpable.

**Pelvic Examination**

She had normal, external genitalia and no vaginal discharge was noted. Speculum examination revealed normal vaginal walls. The cervix was visible and appeared grossly healthy with a parous os. No IUCD threads were visible from the cervical os and no blood or abnormal discharge was seen. Digital examination revealed a normal vaginal cavity, cervix and adnexa. The uterus was anteverted and slightly bulky. No blood or discharge was seen on the gloved examining fingers.

**Diagnosis**

An impression of displaced intrauterine contraceptive device was made.

**Investigations**

|           |   |             |   |                          |
|-----------|---|-------------|---|--------------------------|
| Haemogram | - | Haemoglobin | - | 13.5gm/dl                |
|           |   | WBC         | - | 9.2 x 10 <sup>9</sup> /l |

|                       |   |                                                                                       |   |                     |
|-----------------------|---|---------------------------------------------------------------------------------------|---|---------------------|
|                       |   | RBC                                                                                   | - | $4.8 \times 10^9/l$ |
|                       |   | Platelets                                                                             | - | $346 \times 10^9/l$ |
| Urea and Electrolytes | - | Na <sup>+</sup>                                                                       | - | 134mmol/l           |
|                       |   | K <sup>+</sup>                                                                        | - | 3.5mmol/l           |
|                       |   | Urea                                                                                  | - | 6.2mmol/l           |
|                       |   | Creatine                                                                              | - | 107μmol/l           |
| Pelvic Ultrasound     | - | Bulky uterus with small fibroids with intrauterine device seen in the uterine cavity. |   |                     |

### Management

She was planned for retrieval of the IUCD under general anaesthesia. . Diagnosis and plan of management was explained to her. She gave an informed consent for dilatation and curettage of the uterus under general anaesthesia. She was advised to present to the ward on the morning of surgery, after starving from midnight of the day before. She was admitted into the acute Gynaecology ward as a day case. Intramuscular 0.6mg atropine sulphate and 50mg pethidine were given for pre-medication 30 minutes before theatre.

In theatre the patient was put under anaesthesia. She was placed in lithotomy position. Vulvo-vaginal toilet was done and the patient catheterised. Clear urine was drained. She was then draped and examination under anaesthesia confirmed earlier findings. An Auvard's speculum was inserted into the vagina and cervix visualized. The anterior cervical lip was grasped with a tenaculum forceps. The uterine cavity was sounded, found to be 8cm deep and the IUCD felt in the uterine cavity. The cervical canal was then gradually dilated up to Hegar size 8. A small curette was introduced into the uterine cavity up to the fundus, curettage of the uterine cavity was done and a copper T IUCD was removed. The IUCD was preserved for the patient to see. There was no significant bleeding and patient was reversed from anaesthesia.



It has been shown that about 10 – 15% of IUCDs users report missing threads (3). Failure to locate the threads usually means one of the following: the IUCD threads are too short, retraction in a displaced intrauterine device, detachment of the threads at an attempted removal, device expulsion during menstruation, in a translocated position as in a uterine perforation by the IUCD at insertion or when the patient conceives and the pregnancy pulls the device higher as the uterus enlarges (4).

Diagnosis of a displaced IUCD is usually made by the patient complaining of inability to feel the threads during her routine check-ups, speculum examination confirms these complaints. No threads are visible at the external os.

Once complaints of missing tails are reported, the IUCD should be localised. Extra uterine location or malposition of IUCD predisposes to the risk of pregnancy. Extra uterine location carries an added risk of gut perforation and for copper containing devices an intense inflammatory reaction with subsequent adhesion formation and intestinal obstruction (5). The incidence of uterine perforation is 0.05 – 13 per 1000 insertions(10). Confirmation is by plain abdomino – pelvic X-ray or ultrasonography (3). Our patient had missing tails, which was confirmed by speculum examination and the IUCD was located in the uterine cavity by use of an ultrasound scan.

Our patient had removal of IUCD under general anaesthesia. Translocated IUCDs in the peritoneal cavity are retrieved by laparoscopy or laparotomy under anaesthesia, the former method for inert devices and the latter for active devices since they are likely to be embedded in the omentum and gut (11). All displaced devices should be removed. Those within the uterine cavity should be removed by first exploring the endocervical canal for threads which may be just retracted or coiled (7). If no threads are seen, they can be retrieved by various methods eg by use of a Mimarta spiral retriever, a Lamicel cervical dilator or a Karman's cannula. An alligator forceps can also be used to grasp and retrieve an IUCD located within the uterine cavity but whose threads can not be felt

(6, 7). If these methods fail, dilatation and curettage should be done under general anaesthesia. This is what happened with our patient.

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## **CASE NO. 7**

### **OVARIAN CARCINOMA – LAPAROTOMY, DEBULKING, AND CHEMOTHERAPY**

**Name** :R. W.                      **Parity** :8 + 0  
**IPNO** :0753131                **DOA** :21/08/2001  
**Age** :63 Years                **DOD** :9/10/2001

#### **Presenting complaints.**

The patient presented with complaints of lower abdominal pains and abdominal swelling for a duration of 6months.

#### **Presenting History**

Patient was admitted to the cold gynaecological ward via GOPC where she had presented with lower abdominal pains, and abdominal swelling for 6 months. She also had painful swollen lower limbs and loss of appetite for 3 weeks. She had body weakness, and loss of weight over this duration.

#### **Obstetric and Gynaecological History**

She could not remember her age at menarche. She had regular periods every 28 days, lasting 3 to 4 days until 5 years ago when she attained her menopause. She was para 8 + 0, her last delivery was 26 years ago. She had used I.U.C.D. for 21 years and removed on reaching her menopause.

#### **Past Medical History**

This was not significant.

#### **Family and Social History**

She was a married housewife. Her husband had died of asthma fives years ago. There was no family history of major illness or similar illness. She neither drunk alcohol nor smoked cigarettes.



### **Examination**

She was an elderly lady who was sick looking. She was not pale, and was afebrile. She had pedal oedema but no lymphadenopathy. The breasts were normal. Her pulse was 78 per minute, regular and of good volume. Her blood pressure was 110/70mmHg and her respiratory rate was 22 per minute.

The central nervous, cardiovascular and respiratory systems were normal.

### **Abdominal Examination**

The abdomen was uniformly distended and moving with respiration. It was soft and tender in both iliac fossae. There was a fluid thrill and shifting dullness. The liver was enlarged about 3cm below the costal margin, but the spleen was not palpable. There were 2 palpable masses arising from the pelvis, which were firm, and with irregular surface and they were of size corresponding to a 14 weeks gravid uterus. Deep palpation was not possible due to tenderness.

### **Vaginal Examination**

She had normal external genitalia. The vaginal canal was short with the cervix pushed inferiorly. The cervix was smooth, of normal size and the cervical os was closed. The uterus was of normal size but its mobility was reduced. The adnexa were both full and tender. The pouch of Douglas was full.

### **Per – rectal Examination**

The anal orifice and anal sphincter tone were normal. The uterus was felt and was of normal size. Bilateral adnexal tenderness was elicited, with bilateral irregular fixed solid masses and could not go above them. The rectal mucosa was free.

### **Diagnosis**

A diagnosis of ovarian malignancy was made.

## Investigations

1.     **Ultrasound**     -     Liver, spleen, kidney and pancreas were normal.  
The uterus was of normal size and echo pattern. There were multicystic masses in the region of the ovaries measuring about 10.2 x 11.4 cm right and 8.4 x 10.7cm on the left. These features were highly suggestive of cystadenocarcinoma of ovaries.
2.     **Haemogram**

|             |   |                      |
|-------------|---|----------------------|
| Haemoglobin | - | 14.0gm/dl            |
| WBC         | - | $3.8 \times 10^9/l$  |
| Platelets   | - | $213 \times 10^9/l$  |
| RBC         | - | $4.09 \times 10^9/l$ |
3.     Urea and electrolytes

|                 |   |           |
|-----------------|---|-----------|
| Na <sup>+</sup> | - | 143mmol/l |
| K <sup>+</sup>  | - | 3.5mmo/l  |
| Creatine        | - | 81µmo/l   |
4.     PAP smear     -     No abnormal cells noted (CIN 0).
- 5     Blood group-     O-Positive

## Management

She was prepared for laparotomy for staging, biopsy and if possible TAH and BSO. She was starved from midnight of the day before surgery. Soap enema was also given the night before surgery. She was premedicated with 0.6mg atropine sulphate and 50mg pethidine half an hour before theatre. Two units of compatible blood were reserved.

### Laparotomy (13/09/2001)

General anaesthesia was given as in the introduction. In semi-lithotomy position, vulvo vaginal toilet was performed. The bladder was catheterised aseptically and 100mls of clear urine obtained. The catheter was left in situ. Examination under anaesthesia confirmed earlier pelvic findings.

The patient was repositioned to supine position. The abdomen was cleaned, draped and opened through a sub-umbilical midline incision. The rectus sheath was noted to be thinned and the peritoneum thickened, with midline adherence to the rectus sheath. The visceral aspect of the peritoneum was erythematous and hemorrhagic with discrete nodules. 3 litres of ascitic fluid was drained, which was straw coloured initially and gradually became bloody.

There were pelvic masses arising from both ovaries. It was not possible to differentiate the ovaries from the masses. The masses were friable, hemorrhagic and adherent to the uterus. The bladder was also adherent to the lower uterine segment. The omentum and bowel were attached to the tumour at multiple sites. There were multiple tumour seedlings on the omentum, small intestine and transverse colon. There were massive seedlings over the liver surface.

The disease was staged IV and a decision was made to debulk the tumours. Both tumours on both ovaries were resected out, and partial omentectomy was done. Total abdominal hysterectomy was not possible due to extensive tumour involvement and adherent bladder to the lower segment of the uterus. Haemostasis was achieved. The abdomen was closed in layers and blood loss was estimated at 300mls. Anaesthesia was successfully reversed.

### **Post Operative care**

Vital signs were observed half hourly till she was fully awake then 4 hourly. She was maintained on intravenous fluids of normal saline alternating with 5% dextrose. Pethidine 100mg 8 hourly was given for analgesia and crystalline penicillin 2MU 6 hourly, and gentamycin 80mg 8 hourly as antibiotic prophylaxis. Check hemoglobin level on the third post-operative day was 11.2gm/dl. On the seventh post operative day the wound was well healed and all stitches were removed. She was retained in the ward to await the biopsy report and to be given chemotherapy.



**Histology Report**

The histology report showed cellular sheets and trabecular polyhedral tumour cells which displayed moderate pleomorphism, Call-Exner bodies were seen and the mitotic index was high. Features were consistent with a granulosa cell tumour.

**Further Management**

The patient was prepared for chemotherapy. She was informed of the extent of her disease and mode of treatment. Her general condition was poor. Baseline investigations were repeated.

|    |                             |   |                  |            |
|----|-----------------------------|---|------------------|------------|
| 1. | Blood Urea and electrolytes | - | Na <sup>+</sup>  | 123mmol/l  |
|    |                             |   | K <sup>+</sup>   | 4.7mmol/l  |
|    |                             |   | Creatinine       | 71µmol/l   |
| 2. | Liver function test         | - | Total protein    | 62.3gm/dl  |
|    |                             | - | Albumin          | 30gm/dl    |
|    |                             | - | AST              | 19 IU/L    |
|    |                             | - | ALP              | 292 IU/L   |
|    |                             | - | Total Bilirubin  | 13.7mmol/l |
|    |                             | - | Direct Bilirubin | 4.3mmol/l  |
| 3. | Full haemogram              | - | Hemoglobin level | 10.9gm/dl  |
|    |                             | - | Platelets        | 436 x10/L  |
|    |                             | - | WBC              | 5.3 x 10/L |

These investigations were found to be within normal range and the patient was started on:

- Cisplatin 50mg stat
- Adriamycin 50mg stat
- Cyclophosphamide 250mg once daily for 5days, all intravenous.

She was thereafter discharged to come again for another course of chemotherapy after 3 weeks of rest.

## DISCUSSION

Presented is a 63 years old para 8 + 0 who had carcinoma of the ovaries. Debulking of the masses was done and then chemotherapy was given in her management.

In the U.S.A. Ovarian Cancer accounts for 25% of all malignancies of the female genital tract, and over 50% of the deaths associated with gynecological cancer. It is the 5<sup>th</sup> leading cause for related morbidity among American women (1, 20). The incidence of ovarian carcinoma is higher in industrialized countries than in developing ones (1, 3). At Kenyatta National Hospital, ovarian adenocarcinoma accounts for 9.8% of ovarian tumours (4). High risk countries includes Western Europe, especially Scandinavian countries and in North America (3, 4). The age specific incidence of ovarian cancer rises through out life until the mid 70's when it peaks and begins to decline (1, 2, 5). It is common in post menopausal women. The patient presented was 63 years and was 5 years post menopausal. This late onset of menopause could have been due to prolonged ovarian activity.

The specific aetiology for ovarian carcinoma is not known but a number of associated risk factors have been postulated. Repeat ovulation which causes epithelial damage and inclusion of surface epithelium within the ovarian cortex has been suggested as a possible cause, since there is a reduction of cancer risk in multiparous women, oral contraceptive pill users, breast feeding women and those with anovulatory cycles (1, 2). Our patient was highly parous but had not used any contraceptives. Other risk factors are a high fat diet, exposure to industrial agents e.g. asbestos and talc (1, 2). A familial inheritance pattern is also postulated, but this could not be established in our patient.

The marked disparity in survival rates between patients with early versus late stage ovarian cancer as well as the identification of high risk patients has led to evaluation of various screening methods aimed at increasing early detection. Three techniques available are: history and physical examination, sonography and tumor markers (1). Our patient was diagnosed at an advanced disease stage having presented with no specific symptoms.

These tumors are commonly asymptomatic with insidious onset, until they have achieved a considerable bulk and metastasis. A high index of suspicion and regular examination are important. The commonest complaints are pain and fullness in the pelvis, increasing abdominal girth, and presence of a pelvic mass as was seen in our patient. Urinary symptoms due to pressure on the bladder are frequent and menstrual disturbance or post menopausal bleeding may elicit a high index of suspicion. Our patient presented with lower abdominal pain and lower limbs swelling.

Pelvic ultrasound and computerized tomography may be useful in diagnosis and assessing the size and consistency of adnexal masses (2), though the results of sonography in screening for ovarian cancer have been disappointing (1). Other important studies to assess the extent of disease include chest X-rays, bone and liver scans. These are rarely indicative in the initial work up of these patients. Available screening tests are expensive and lack reproducible accuracy necessary for effective screening. Alpha fetoprotein (AFP) is extremely rare in ovarian epithelial cancer, it is used for germ cell tumors (2, 6). Carcino embryonic antigen (CA-125) is elevated in 30 – 50% of patients with ovarian cancer, but it is also positive for tissues derived from coelomic epithelium and the Mullerian duct, it therefore lacks specificity (1, 2, 7). However, most ovarian carcinomas can be discriminated with high probability using a panel of 3 antibodies directed against CEA, cytokeratin 7, and vimentin (7). Immunodiagnosis using an ovarian cancer associated antigen called NB/70K is available (2).

These tumors spread by seedlings, lymphatics, blood stream and direct spread to any neighbouring organs (1,2,5). Ovarian carcinoma is a surgically staged disease. Our



patient was found to have stage IV of disease at surgery, since she had ascites, tumor involvement of the omentum, intestine and liver.  $\frac{2}{3}$  of patients with ovarian carcinoma already have stage III or IV disease at the time of diagnosis (2). Serous adenocarcinomas are usually large and in over 50% both ovaries are involved (5). Histological pattern of ovarian adenocarcinoma includes papillary, adenopapillary, or diffuse patterns (5). Epithelial ovarian malignancies are also graded as per degree of differentiation, into well differentiated (G 1), moderately well differentiated (G 2) and poorly differentiated (G 3). This also has a bearing to the prognosis.

Surgery continues to play the central role in the treatment of ovarian carcinoma (1,2,8). Removal of tumour to the extent possible may be curative in some patients and provides the remainder with a good start for post operative chemotherapy or radiotherapy. In stage I and II disease when conservation of fertility is not an issue, the recommended surgery is total abdominal hysterectomy and bilateral salphingo- oophorectomy, with omentectomy and lymphadenectomy. In advanced stage III and IV, cytoreduction (debulking) surgery is done, followed by chemotherapy or/and radiotherapy. Prophylactic oophorectomy or ovarian ablation in women with strong family history of ovarian carcinoma or undergoing hysterectomy for being uterine diseases may be important in the prevention of ovarian carcinoma (1, 2). Carbon dioxide laser, ultrasonic aspiration, argon beam coagulation and loop electrosurgical excision can be used for treatment of tumours in inaccessible areas like the mesentery, diaphragm, or liver (1). Surgery followed by chemotherapy gives better 5 year survival rates.

Second look laparotomy is advocated in patients with advanced stages of disease usually to assess effectiveness of chemotherapy and therefore make a decision whether to stop chemotherapy. It is also an opportunity to debulk any residual disease. This has however been replaced by laparoscopy. High dose intraperitoneal cisplatin and other intraperitoneal drugs, cytarabine or bleomycin have been used as second line chemotherapy in persistent tumor after standard chemotherapy and/or after second look operation, with good survival results (1). Other active agents include carboplatin, ifosfamide, and paclitaxel (9).

Prognosis depends on tumor type, clinical stage at diagnosis, and histological grade, and type of neoplasm. 5 year survival rates of 20 – 30% have been achieved when all gross residual disease is removed followed by chemotherapy and radiotherapy (1, 2, 3). Survival of patients with ovarian cancer has improved in recent years due to earlier stage at diagnosis, and possible introduction of combination chemotherapy with cisplatin (3).

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