

GYNAECOLOGY CASE 5

PRIMARY INFERTILITY, TUBAL FACTOR-LAPAROSCOPIC TUBOPLASTY

NAME	A. W.	DOD	4/08/04
AGE	33 YEARS	PARITY	0+0
IP. NO.	0954877	WARD	1B
DOA	2/08/04		

PRESENTING COMPLAINT

A.W. presented with a 6-year history of inability to conceive.

HISTORY OF PRESENTING COMPLAINTS

She was admitted via the infertility clinic with a 6-year history of inability to conceive. She was married for 6 years and had regular unprotected vaginal intercourse about 3-4 times a week. She gave no history of contraceptive used in her married life, though she had used oral contraceptive for two years before marriage. She had never become pregnant in her life. To the best of their knowledge her husband had never fathered a child with any woman. She had never been treated for sexually transmitted infections (STI) or per vaginal discharge. Investigations done in the infertility clinic had shown that both her fallopian tubes were blocked.

OBSTETRICS AND GYNAECOLOGY HISTORY

The lady was a nullipara. Her menarche was at the age of 16 years and her menses were regular lasting 4 days and with an interval of 28 days. There was no associated dysmenorrhoea or dyspareunia. Her last menstrual period was on 18/07/04. She had never been operated before. A pap smear done a month before admission was normal.

PAST MEDICAL HISTORY

The past medical history and surgical history was unrevealing.

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PAST MEDICAL HISTORY

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FAMILY AND SOCIAL HISTORY

She was a teacher by profession. Her husband was also a teacher. They worked and lived in Kigumo, Muranga. She had never drunk alcohol or smoked cigarettes. She was not using any prescription or non-prescription drugs. She had never been exposed to radiation or chemicals. Her husband drank alcohol occasionally. There was no family history of chronic ailments e.g. diabetes, hypertension or tuberculosis.

SYSTEMIC INQUIRY

This was non-revealing.

PHYSICAL EXAMINATION

She was a young lady in good general condition. She was well nourished, with a weight of 64 kilograms. She was not pale, did not have jaundice, edema or lymphadenopathy. There were no masses in the neck anteriorly. The breasts were well developed, tanner class IV, and there was no galactorrhoea.

RESPIRATORY AND CARDIOVASCULAR SYSTEMS

These were examined and found to be essentially normal.

CENTRAL NERVOUS SYSTEM

This was non-revealing.

PELVIC EXAMINATION

She had a normal adult female genitalia, and normal pubic hair. The cervix was posterior, long and grossly normal. The uterus was normal sized and anteverted. The adnexae and pouch of Douglas were free. Cervical excitation was negative.

INVESTIGATIONS

- A semi analysis done on 22/4/04 was normal
 - Volume - 4.0ml (range > 2.0ml)
 - Consistency- droplets (normal – droplets)

Motility-	85% (normal $\geq 50\%$)
Concentration-	100 million/ml. (20 million/ml)
Morphology-	18% (normal strict criteria $> 14\%$)
WBC's-	0.6×10^6 /ml (normal upto 1.0×10^6 /ml)
Conclusion-	normozoospermia

- Elisa for HIV – negative
- Pap smear - Normal squamous and endocervical cells.
- HSG (Hysterosalpingogram) 17/5/04 – showed a normal uterine cavity. The left tube was not visualized. The right tube showed terminal blockage. No dye spill was obtained.

Conclusion: Left tubal blockage at the cornua and right hydrosalpinx.

- Pelvic ultrasound – normal uterus. Right adnexal mass and fluid in the POD, suggestive of pelvic inflammatory disease. Following this report the patient received a course of antibiotics for one week.

DIAGNOSIS

An impression of primary infertility due to tubal blockage was made. She was planned for laparoscopic tuboplasty and hysteroscopy.

MANAGEMENT

She was counseled on Laparoscopic tubal surgery and admitted for preparation for theatre.

Pre-operative investigations included:

- Hemogram - (22/7/04) - WBC 5.8×10^3 /mm³
 - RBC 4.89×10^6 /mm³
 - Hb 13.7g/dl
 - HCT 43.4%
 - Platelets 416×10^6 /mm³

- Blood Chemistry
 - Na⁺ 142 mmol/l
 - K⁺ 4.4 mmol/l
 - Urea 3.6 mmol/l (normal 2.5-8.3 mmol/l)
 - Creatinine 91 μmol/l (normal 53-97 μmol/l)

PROCEDURE AND FINDINGS

Informed consent was obtained and patient pre-medicated with Atropine 0.6mg intramuscularly 30 minutes before theatre. In theatre the patient was placed on the operation table, and repositioned to the lithotomy position. Vulvovaginal toilet was done and bladder catheterized. A cuscus speculum was introduced and opened. The cervix was held in a tenaculum and cervix dilated with Hegars dilators. A hysteroscope was then introduced and normal saline instilled. On hysteroscopy, the uterine cavity was grossly normal. The hysteroscope was then removed, and an endocervical cannula introduced. The abdomen was cleaned and draped. A periumbilical stab was made and the Veress needle pushed into the abdominal cavity. Carbon dioxide gas was introduced up-to 2.5 liters to cause a pneumoperitoneum. The trocar was then introduced and the fibro optic camera was fixed in place. The uterus was visualized and found to be grossly normal. The left tube was buried in mass of peri-tubal and periadnexal adhesions around the ovary. The right tube had an ampullary hydrosalpinx with peritubal adhesions. Two incisions were made in both flanks for the introduction of surgical instruments. Adhesions were released to free both tubes. Cuff salpingoneostomy was done on the left side. The distal cystic end of the right tube was excised and adhesions released. On instillation of dye, both tubes were partially filled with spill only on the right side. Irrigation and suction was done and heparin and hydrocortisone introduced to minimize formation of adhesions. The wounds were closed and general anesthesia reversed successfully. In conclusion, there were very limited chances of pregnancy following the operation and even open tubal surgery was thought unwise.

The patient was allowed home 2 days after the surgery on Doxycycline, Flagyl and Ibuprofen tablets to be seen in the gynecology out-patient clinic for follow up and repeat hysterosalpingogram in 3 months.

DISCUSSION

The patient presented was a 33-year-old nullipara with primary infertility secondary to tubal factor. She underwent laparoscopic surgery where adhesionolysis and neosalpingostomy were done and patency only achieved in the right tube. The extent of pelvic adhesions made the surgeons conclude she had a poor prognosis of achieving a pregnancy.

Infertility can be defined as the failure to achieve a conception or to bring pregnancy to term after one year or more of unprotected intercourse (1). Infertility can be divided into primary and secondary depending on whether an individual has ever had children or not. Secondary infertility is more common than primary especially in the developing countries where sexually transmitted diseases play a central role (2). A normal fertile couple having regular unprotected intercourse achieves pregnancy at a rate of 25% during each menstrual cycle. About 10% of these fertile couples fail to conceive within the first year of attempt and another 5% after 2 years. According to the WHO estimates, infertility affects about 8-10% of all couples attempting to conceive with slight geographical variations (3). On a worldwide scale, this means that about 50-80 million people suffer from infertility. In Kenya, the incidence of infertility is unknown, though Mati reported that 60% of all patients seeking outpatient gynecology services at KNH complained of infertility (4). Infertility is therefore a widespread problem that requires investigations and management.

The aetiology of infertility is multi factorial. About 25-40% of the cases are exclusively due to the male factor; a further 40-55% is due to the female factor alone, while about 10% are due to combined factors. The remaining 10% are idiopathic (5). A World health organization's (WHO) study between 1979 and 1984 in developing countries found that up-to 64% of females in Africa and 28-35% in other areas had infertility that

could be traced to pelvic infection (6). This extrapolated to 49% bilateral tubal occlusion among African women and 11-15% among others.

At the Kenyatta National Hospital, pelvic inflammatory disease has been blamed for tubal occlusion in over 70% of the cases (7, 8), with gonococcal infection accounting for about 55%. *Chlamydia trachomatis* has also been blamed for a significant proportion of tubal blockage, while tuberculosis accounts for a small proportion (7, 8, and 9).

Although gonococcus and *Chlamydia* may be responsible for acute salpingitis, residual chronic salpingitis and subsequent tubal damage is mainly due to secondary invaders, both aerobic and anaerobic (10). The presence of an intra-uterine device has been known to predispose to pelvic infection. Oral contraceptives and barrier methods protect against pelvic infection. Other etiological factors of infertility include male factor, disorders of ovulation and mullerian system defects among others (11,12). In significant proportion of cases, no demonstrable cause is found. In the case presented, the probable cause of infertility was tubal occlusion due to previous tubal (pelvic) infection.

- The causes of infertility may be broadly classified as male or female. The male causes are generally due to abnormalities with sperm count, motility and morphology. Female causes of infertility may be divided into;
 - Ovulatory disorders
 - Tubal\ Peritoneal factors
 - Cervical factors
 - Uterine factors

Tubal and peritoneal factors account for between 30-40% of all causes of female infertility (14). These factors include;

- Damage or obstruction of the fallopian tubes usually associated with previous PID, surgery of the tube etc
- Peritubal and peri-adnexal adhesions, which generally result, from PID or surgery or endometriosis.

Factors that affect the reproductive performance of a couple including their ages, frequency of coitus and duration of coitus thus together with the history. Five primary

tests have been adapted and are useful in the initial evaluation: documentation of ovulation; semen analysis; post-coital test; hysterosalpingogram and diagnostic laparoscopy. For the male partner, physical examination and semen analyses that are non-invasive usually suffice. Feminine tests should provide information on ovulation, tubal patency and surgical prognosis.

Hysterosalpingogram (HSG), has been known to have fertility enhancing effects hence laparoscopy should be done after about 6 months following normal HSG report. Conception rates have been known to increase by 30% after the examination (14). In this patient HSG was done pre-operatively while laparoscopy was done as a diagnostic and therapeutic measure.

The goal of all infertility treatment is to reduce the time a couple takes to conceive without intervention. Such therapy is often undertaken in an effort to achieve pregnancy before or during the natural age-related decline in female fertility (13).

Various methods of tubal surgery have been described. These include; salpingolysis, salpingostomy or fimbrioplasty, end-to-end anastomosis and tubal re-implantation, which have been perfected by microsurgery. Success rates for tubal surgeries have been particularly promising in-patients with distal tubal blockage with live births in 40-60% of cases reported (15). Success rates generally depend on patient selection, and are inversely proportional to the severity of tubal damage and the extent and nature of adhesions. Since female fecundability decreases markedly after 35 years, infertility treatment after this age has lowered success rates. The role of repeat tubal surgery following a failed attempt remains controversial. It has been recommended only in selected patients with limited damage and has been found to have a better prognosis for overall term pregnancy rates than that achieved by in-vitro fertilization programs for patients with limited tubal damage (16).

The patient presented had adhesiolysis and salpingostomy done by microsurgical techniques. However, the extent of adhesions meant that her prognosis for pregnancy was poor. Poor prognostic factors for distal tube occlusion include; hydrosalpinx > 30mm

in diameter, absence of visible fimbriae, dense pelvic or adnexal adhesion and appearance of the tubal mucosa. An Important complication of tubal surgery is an increased risk of tubal pregnancy.

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GYNAECOLOGY CASE 6

PERSISTENT GESTATIONAL TROPHOBLASTIC TUMOR-COMBINATION CHEMOTHERAPY

NAME	E.N	DOA	3/3/04
AGE	23 YEARS	DOD	17/04/04
IP NO.	0933633	PARITY	0+1
WARD	1B		

PRESENTING COMPLAINTS

She was admitted with a 2-month history of vaginal bleeding and lower abdominal pain.

HISTORY OF PRESENTING COMPLAINTS

E.N. last normal menstrual period was on 19/09/03. On 10/12/03 at a gestational age of 13 weeks, she presented to a private clinic in Nairobi with complaints of vaginal bleeding, nausea and vomiting. A diagnosis of incomplete abortion was made and manual vacuum aspiration (MVA) done. However, after discharge home the bleeding persisted for 2 weeks after the MVA. She went back to the clinic and a repeat MVA was done. Bleeding stopped but not for long. 3 weeks later, in February 2004, there was recurrence of vaginal bleeding associated with swelling and pain in the lower abdomen. She now decided to seek medical attention at a different private clinic. An ultrasound scan done there showed features consistent with hydatidiform mole and bilateral ovarian masses. Dilatation and curettage was done and tissue taken for histology. The Beta HCG level was 206 mIU/ml. She was discharged on antibiotics and given an appointment in 2 weeks. The bleeding and abdominal swelling persisted. She went back to the clinic, and the histology of uterine curetting revealed she had an invasive mole. She was referred to the KNH for further management. She was admitted on 3/3/04.

OBSTETRICS AND GYNAECOLOGY HISTORY

She was para 0+1, the antecedent pregnancy having been a hydatidiform mole. Her menarche was at the age of 15 years, and before the current illness was regular, lasted 3-4 days and occurred every 28 days. There was no associated dysmenorrhoea. She had used oral contraceptive for 3 years before marriage. She had never had a pap smear done.

SYSTEMIC ENQUIRY

She also complained of dizziness, headache as well as awareness of her heartbeat. She got easily tired on walking for some distance.

PAST MEDICAL HISTORY

She had never been admitted previously.

FAMILY AND SOCIAL HISTORY

She was now married for 1 year. They lived together with her husband in Umoja. She did not smoke or drink alcohol. There was no history of chronic ailments in the family.

PHYSICAL EXAMINATION

She was a young lady in fair general condition that looked sick. She had moderate pallor, slight pedal pitting edema, but no jaundice, cyanosis or lymphadenopathy. Her blood pressure was 100/60mmhg, pulse 108/min, respiratory rate 16/min and temperature of 36.5°C.

RESPIRATORY AND CENTRAL NERVOUS SYSTEMS

These were essentially normal.

CARDIOVASCULAR SYSTEM

There was Tachycardia. The first and second heart sounds were heard. There were no murmurs.

ABDOMINAL EXAMINATION

The abdomen was distended in the suprapubic region. There were no surgical or therapeutic scars. A tender suprapubic mass corresponding to 20 weeks was palpated. It was mobile from side to side but not up and down. You could not go below it. Its surface felt smooth.

PELVIC EXAMINATION

There were normal external genitalia. There were blood clots in the vagina. The uterus was bulky corresponding to about 20 weeks. The cervix was smooth, posterior, long and closed. There was blood on the examining finger.

TENTATIVE DIAGNOSIS

A tentative diagnosis of persistent gestational Trophoblast disease (Invasive Mole) was made.

INVESTIGATIONS

1. Beta HCG levels – 893 940 mIU/ml.
2. Hemogram – Hb 7.2g/dl;
WBC $6.2 \times 10^3/\text{mm}^3$
Platelet $354 \times 10^3/\text{mm}^3$,
RBC count $3.2 \times 10^6/\text{mm}^3$
3. Urea and electrolytes.
Urea 8.2 mmol/litre
Creatinine 154 $\mu\text{mol/litre}$
Na⁺ 138 mmol/l
K⁺ 3.7 mmol/l
4. Chest X-ray – Normal chest radiograph
5. Blood group – A positive
6. Pelvic ultrasound – showed a markedly enlarged uterus. Within the uterus there was an amorphous mass with mixed echogenicity having solid and tiny cystic areas. There were small bilateral ovarian cysts. Uterine mass approximately 5cm by 8 cm.

7. Liver Function Test – normal

DEFINITIVE DIAGNOSIS

Persistent gestational trophoblastic disease (invasive mole), with high-risk prognostic score. Her prognostic score was calculated according to the World Health Organization (WHO), criteria as follows;

1. Age – less than 35 years. Score –0
2. Antecedent pregnancy – Hydatidiform mole score – 0
3. Interval between end of antecedent pregnancy and start of chemotherapy – 4 months – score –1
4. Beta HCG levels 893 940 miu/litre – score 4.
5. Blood group – A – score 1
6. Largest tumor size (5*8cm) - score 2.

TOTAL SCORE = 8 = HIGH RISK SCORE

MANAGEMENT

The patient was worked up for triple therapy, consisting of Methotrexate, Actinomycin – D and Cyclophosphamide. Before chemotherapy she received 3 units of blood. Her pre-chemotherapy Hb was 10.6g/dl. On the 2nd of April 2004 she was started on:

- Intravenous Methotrexate 50mg daily for 5 days.
- Intravenous Actinomycin D 0.5mg daily for 5 days
- Intravenous Cyclophosphamide 500mg daily for 5 days.

She did well after the first course of chemotherapy with cessation of vaginal bleeding. During the resting period she received folic acid 15mg once daily.

After the first course of chemotherapy, Beta HCG levels fell to 75040m.IU/l. She received another unit of blood. Baseline investigations were normal. She received the second course two weeks after the first course. After this course the Beta HCG levels fell to 9500miu/l and were allowed home on hematinics to come with baseline

investigations in 2 weeks. She was commenced on oral contraceptive pills. She was informed of the great need to continue with follow up before discharge.

DISCUSSION

The patient presented was a 23-year-old Para 0+1, who was admitted with high risk persistent gestational trophoblastic disease following a molar pregnancy. She was started on triple therapy, which was continued till remission was achieved.

Gestational trophoblastic disease is a spectrum of pregnancy related intraepithelial tumors among them hydatidiform mole, invasive mole, placental site trophoblastic tumor and choriocarcinoma which have varying propensities for local invasion and metastasis (1). These tumors arise from fetal tissue within the maternal host and are composed of syncytiotrophoblast and cytotrophoblastic cells except placental site tumor, derived from intermediate trophoblastic cell (2). The incidence of trophoblastic disease is reported to be highest in S. East Asia, with Taiwan reporting an incidence of 1:82 pregnancies. In Europe and North America, the reported incidence is between 1:1500 and 1:2500 pregnancies (3,4). In a study at the KNH, 65 cases of choriocarcinoma were diagnosed in a period of 7 years and a repeat study in the same hospital in 1984 reported an incidence of 1:1118 deliveries (5,6). The actual incidence of choriocarcinoma in Kenya is however unknown.

Sometimes, something goes wrong very early in pregnancy. The fetus fails to develop but the placental elements continue to grow. There is swelling of the villi and overgrowth of the cyto- and syncytiotrophoblast cells. The villi then become so swollen that they become visible and look like drops of water. The Latin name for this mass of water drops is hydatidiform mole, and is often referred to as molar pregnancy. These trophoblastic cells make the pregnancy hormone Human Chorionic Gonadotropin (HCG), which is the basis of all pregnancy tests. There is overproduction of HCG as well as exaggerated symptoms of pregnancy. Why molar pregnancies occur is unknown, but there are some remarkable features about them.

- They have the ability to invade the wall of the uterus.
- They can metastasize to other organs.
- They have 23 pairs of chromosomes, all of which are paternal in origin.
- They are XX genotype, and both of the X-chromosomes are paternal in origin.
- They can develop into gestational trophoblastic cancer (2).

Our patient presented with a molar pregnancy about 4 months prior to the diagnosis of persistent gestational trophoblastic disease. Evacuation was done

After molar pregnancy is evacuated, there must be rigorous surveillance for any sequelae. The consequences of a mole can be persistent mole, invasion mole, metastatic mole or choriocarcinoma. The follow up is done by weekly Beta HCG levels. If the Beta HCG decreases but then levels off and starts to rise again, then the diagnosis of gestational trophoblastic disease is made. This may be an invasive mole, metastatic mole or choriocarcinoma. Our patient developed invasive mole. When a diagnosis of gestational trophoblastic disease has been made, chemotherapy is needed.

Non-metastatic persistent gestational trophoblastic disease presents clinically with the following symptoms:

- Irregular vaginal bleeding
- Theca lutein cysts
- Uterine sub-involution or asymmetric enlargement.
- Persistently elevated serum Beta HCG levels. (1).

All these were present in the patient presented.

The diagnostic evaluation for the optimal management of persistent gestational trophoblastic tumors includes;

- i. Complete history and physical examination
- ii. Measurement of serum Beta HCG levels
- iii. Hepatic, thyroid and renal function tests
- iv. Baseline peripheral white blood cell counts and platelet counts.

The metastatic work up should include:

- i. Chest radiograph or computed tomography (CT) scans.
- ii. Ultrasound or CT scans of the abdomen and pelvis.
- iii. CT or magnetic resonance imaging (MRI) of the head.

In our patient, since no metastasis were found in the chest and pelvic, imaging of the head was thought unnecessary. Indeed literature indicates that when pelvic and chest radiographic findings are negative, metastatic involvement of other sites is uncommon (1).

The international federation of gynecology and obstetrics (FIGO) has adopted an anatomic staging system for gestational trophoblastic tumors. In this system;

Stage I: Patients with persistently elevated Beta HCG and tumors confined to the uterus corpus.

Stage II: Patients with metastasis to the vagina and or pelvic or both.

Stage III: Patients with pulmonary metastasis with or without uterine, vaginal or pelvic involvement.

Stage IV: Patients with advanced disease and involvement of the brain, liver, kidneys, or gastrointestinal tract (1).

Our patient was placed in stage II. In addition to anatomic staging, its important to consider other variables to predict the likelihood of drug resistance and assist in selecting appropriate therapy (7). A prognostic scoring system proposed by the World Health Organization (WHO) reliably predicts the potential for resistance to chemotherapy. When the prognostic score is higher than 7, the patient is categorized as high risk and requires intensive combination chemotherapy to achieve remission. Those with a score less than 4 are low risk, while those between these values are moderate risk.

WORLD HEALTH ORGANIZATION PROGNOSTIC SCORE SYSTEM

SCORES

Prognostic Factors	0	1	2	4
Age	<39	>39	-	-
Prior pregnancy	Mole	Abortion	Term	-
Interval	< 4 months	4-6 months	7-12 months	> 12 months
B-HCG	<1000	<10000	<100000	>100000
ABO blood group	-	OxA or AxO	B or AB	-
Size of largest tumor	-	3-5 cm	75cm	-
Site or metastasis	-	Spleen, Kidney	GIT, Liver	Brain
Number of metastasis	-	1-4	4-8	78
Prior chemotherapy	-	-	Single agent	2 or more

Total score: 0-4 low risk, 5-7 intermediate risk, >8 high-risk (1).

Our patient was placed on the high-risk category.

A protocol of treatment for Gestational Trophoblastic disease is presented below ;(1).

- Stage I Single agent chemotherapy or hysterectomy with adjunctive chemotherapy. If resistant combination chemotherapy is used.
- Stage II and III
 - Low risk – Single agent chemotherapy
 - High risk - Combination chemotherapy
- Stage IV Combination chemotherapy
 - Whole-wheat irradiation (3000CGY)
 - Craniotomy to manage complications
 - Resection to manage complication

Our patient was placed in stage II, high risk and started on the MAC protocol (Methotrexate, Actinomycin D and cyclophosphamide), which was given for 5 days. Methotrexate is given at a dose of 0.4mg/kg daily, Actinomycin D at 0.5mg daily, and Cyclophosphamide at 3mg/kg daily. The course is repeated with a minimum rest period of 14 days and ensuring that the Hb level is above 10g/dl, white cell count above 2000/ul and platelet count above 150/ul before commencing on the next course (8).

Remission is attained after a patient achieves three consecutive weekly Beta HCG titers of less than 10mili/ul/ml, and sustained remission if repeated Beta HCG titers at monthly intervals remain negative for one year (1, 2). On our unit two to three more courses of chemotherapy are given once the B. HCG titers are negative.

All patients with stage I through III disease should receive follow up with:

- Weekly measurement of B. HCG, till they are normal for 3 consecutive weeks.
- Monthly measurement of B.HCG, till normal for 12 consecutive months.
- Effective contraception during the entire interval of hormonal follow-up.

The prognosis depends on the extent of disease and aggressiveness of treatment. For a molar pregnancy well managed, the cure rate is 100%. For non-metastatic trophoblastic disease vigorously treated, the cure rate is also about 100%. Widely metastatic disease if recognized promptly and treated aggressively with multi-agent chemotherapy, surgery and radiation if necessary, is curable in 80% of the cases.

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GYNAECOLOGY CASE 7

OVARIAN CARINOMA STAGE I C – LAPAROTOMY – TOTAL ABDOMINAL HYSTERECTOMY AND BILATERAL SALPINGOOPHORECTOMY DONE; SUBSEQUENT CHEMOTHERAPY

NAME	-	J.N	DOA	-	1/7/04
AGE	-	53 YEARS	DOD	-	15/7/04
IP. NO.	-	0969519	PARITY	-	2 + 0
WARD	-	1B			

PRESENTING COMPLAINTS

J.N Presented with a 3 months history of a progressively growing lower abdominal mass and dull abdominal pain.

HISTORY OF PRESENTING COMPLAINTS

She said she was well till 3 months prior to admission when she noticed a mass in the lower abdomen. The mass was progressively increasing in size. Subsequently she developed lower abdominal pain. The pain was intermittent and dull in character. It was radiating to the back. There were no associated changes in bowel or urinary habits. She did not give a history of weight loss. Concerned she sought assistance in a private clinic, where an ultrasound scan revealed an adnexal mass. She was referred to KNH for further investigations and treatment.

OBSTETRICS AND GYNAECOLOGY HISTORY

The patient was a 53-year-old Para 2+0. Her last delivery was in 1969. Both her deliveries were spontaneous vertex deliveries (SVD), and both children were a live and well. She could not recall her age at menarche, but was now 3 years post-menopausal. Her periods had otherwise being regular at an interval of 26 days and lasting 3-4 days. She had used an intra-uterine contraceptive device for about 10 years after her last delivery. It was removed in 1981. Since then she had not used any other methods of

contraception. A Pap smear done one month prior to admission was reported as normal.

FAMILY AND SOCIAL HISTORY

She was married for close to 30 years. They lived together with her husband in Muranga. They were small-scale farmers. She did not drink alcohol or smoke cigarettes. There were no known chronic ailments.

PAST MEDICAL HISTORY

Other than for her deliveries, she had never being admitted to hospital or had any form of surgery.

SYSTEMIC INQUIRY

This was non- revealing

PHYSICAL EXAMINATION

She was a middle-aged lady in good general condition and nutritional status. She was not pale, did not have Jaundice, edema or lymphadenopathy. Her vital signs were;

Blood pressure	110/65mmhg
Temperature	36.5°C
Pulse rate	84/minute
Respiratory rate	16/minute

RESPIRATORY, CARDIOVASCULAR AND CENTRAL NERVOUS SYSTEMS

These were found to be essentially normal.

ABDNOMAL EXAMINATION

The abdomen was moderately distended in the suprapubic region up to the level of the umbilicus. Otherwise it was moving with respiration, and there were no therapeutic or surgical scars.

On light palpation there was an abdominal mass extending to the level of the umbilicus, which was slightly tender. On deep palpation, a firm tender mass was palpated in the right lumbar region but extending toward the hypo gastric region. The mass had a smooth surface and was mobile from side to side. The mass was emanating from the pelvis. There was no splenomegaly or hepatomegaly.

PELVIC EXAMINATION

The external genitalia were atrophic but no bleeding or mass were noted at the vulva. On speculum examination, the vaginal walls were smooth and the cervix had a smooth surface and was tightly closed. No cervix lesions or bleeding were noted. On Bimanual examination, there was a mass in the right adnexae, which was separate from the uterus. The uterus was felt to be of normal size.

RECTAL EXAMINATION

Rectal examination revealed fullness in the pouch of Douglas with normal rectal mucosa. The uterus felt normal size.

DIAGNOSIS

A working diagnosis of a right ovarian tumor was entertained.

MANAGEMENT

To collaborate earlier findings a repeat ultrasound scan was requested. Also requested were a full hemogram, urea, creatinine and electrolytes.

RESULTS

- Pelvic scan – on 28/6/04; showed a normal sized uterus. The endometriun was also normal. However, the uterus was displaced to the left by a large mixed echogenic mass. The mass had cystic and solid components and measured 7.4cm by 16.6cm. It extended to the pouch of Douglas. The right ovary was not visualized. A conclusion of a right adnexal mass; probably ovarian in origin was made.

- Hemogram;

WBC count	-	$4.8 \times 10^3 / \text{mm}^3$
RBC count	-	$4.72 \times 10^6 / \text{mm}^3$
Hb level	-	13.3g/dl.
HCT	-	41.2%
MCV	-	$87 \mu\text{m}^3$
MCH	-	28.3pg
MCHC	-	32.4g/dl
Platelets	-	$399 \times 10^3 / \text{mm}^3$

- Urea / electrolyte

Na ⁺	-	137 mmol/l
K ⁺	-	4.0 mmol/l
Urea	-	4.2 mmol/l
Creatinine	-	62 $\mu\text{mol/l}$ (micromoles/l).

- She was planned for total abdominal hysterectomy and Bilateral Salpingoophorectomy. The operation was scheduled for 10th of July 2004.

TREATMENT

After admission the probable diagnosis was explained to her and the operation necessary. She gave informed consent. Blood was grouped and cross-matched and kept ready.

On 10th July 2004, she was premedicated with atropine 0.6mg and started on an intravenous drip of normal saline. In theatre, the patient was placed on the operating table, repositioned in to semi-lithotomy position, vulvalvaginal toilet done, and catheterized. Examination under anesthesia confirmed previous findings. She was repositioned to the supine position after the vagina was painted with methylene blue. The anterior abdominal wall was cleaned with antiseptic, and then draped. The abdomen was opened using a lower midline incision.

Upon entering the abdominal cavity, no free fluid was found in the pelvic cul-de-sac. Peritoneal washings were done and submitted for cytology. A systematic exploration of the abdominal cavity revealed smooth and normal visceral surfaces. The peritoneum and omentum were also grossly normal. A right multi-loculated well-encapsulated ovarian mass with capsular rupture was found extending to the midline. The tube felt grossly normal, as was the uterus. The mass measured about 10 x 15 cm.

Total abdominal hysterectomy and bilateral salpingoophorectomy were done with partial omentectomy. The specimens were submitted for histology. The abdomen was closed in anatomical layers. The patient received one unit of blood intra-operatively. General anesthesia was successfully reversed and patient taken back to ward 1B.

HISTOLOGY REPORT

The specimen received consisted of the uterus, tubes and ovary with a mixed mass measuring 10 x 15 cm. There was also some omental tissue. Histology revealed normal endometrium, myometrium, and fallopian tubes. The omental tissue was normal. The ovarian tumor showed highly malignant epithelial tumor whose feature was in keeping with malignant serous cystadenocarcinoma of the ovary. Malignant cell extended beyond the capsule.

SUBSEQUENT MANAGEMENT

Based on these findings the patient was classified as stage 1, High grade, and High-risk ovarian cystadenocarcinoma. Additional treatment with chemotherapy was decided. She was to receive 6 courses of chemotherapy. She was prepared for the first course. The liver function tests, urea and electrolytes were normal. The hemogram showed a Hb –level of 11.2 g/dl, platelets of $389 \times 10^9/L$ and a WBC count of $5.2 \times 10^9/L$. All the organ function tests being normal, she was started on;

- Isolation – 50mg intravenous start dose. Prior to the drug administration she was rehydrated with about 2 litres of normal saline.
- Cyclophosphamide- at a dose of 500mg intravenously daily for 5 days.

She tolerated the drugs well, with the occasional vomiting responding to intramuscular Metoclopramide. After completion of her course, she was allowed home to return for the second course after three weeks. She was advised to have all the necessary investigations ready at the date of admission.

DISCUSSION

Presented is a 53-year-old Para 2+0, post-menopausal woman who presented with a slowly growing abdominal mass and pain. The mass was histologically proven to be serous cystadenocarcinoma of the ovary, which was highly malignant. She underwent total abdominal hysterectomy and bilateral salpingoophorectomy. She was subsequently commenced on chemotherapy.

Of all the gynecologic cancers, ovarian malignancies represent the greatest clinical challenge. They also represent a major surgical challenge, because they require intensive and often complex therapies, and are extremely demanding of the patients' psychological and physical energy. It has the highest fatality-to-case ratio of all the gynecologic malignancies. Epithelial cancers are the most common ovarian malignancies and because they are usually asymptomatic until they have metastasized, patients present late with advanced disease in more than two thirds of the cases (1,2). Our patient had serous cystadenocarcinoma of the right ovary, which fortunately at the time of diagnosis had not spread extensively.

Approximately one in 70 newborn girls will develop ovarian cancer during their lifetime. In general ovarian cancer is a disease of the postmenopausal woman and prepubescent girl, although it is documented to occur in females of all ages (1,2). Our patient was 3 years post-menopausal.

The cause of ovarian cancer is unknown, though a number of risk factors have been identified. Repeated ovulation, increased dietary fat consumption, infertility, use of fertility drugs, exposure to talc and asbestos are some of these risk factors. Genetic factors also appear to influence the development and progression of ovarian cancer.

Turners' syndromes (45, XO), the presence of the Y-chromosome as found in turners syndrome with mosaicism (45 XO, 46 XY) and Klinefelters syndrome (XXY), Further increases the risk. Other genetic disorders associated with increased risk include the familial occurrence of teratomas and the increased risk of sex cord tumors in patients with peutz-jeghers syndrome (2,3). Molecular biologic studies now suggest the presence of one or more tumor suppressor genes on chromosome 17, which may play a role in the etiology of this disease (2).

Some factors have been found to be protective against ovarian cancer. These include chronic anovulation, multiparity and prolonged breastfeeding. Pregnancy decreases the risk of ovarian cancer by 30-60% while oral contraceptives use decreases the risk by 30-60%, depending on the duration of use (1,2). Tubal ligation also decreases the risk (3).

Ovarian cancer may be divided into 3 major categories based on the cell type of origin. The ovary may also be a secondary site of metastatic disease from other sites. Unlike cervical and endometrial cancer, no precursor lesions have been identified to date. The major histopathological categories of ovarian cancer are: -

- Epithelial – these may be serous, mucinous, endometrioid, clear cell, transitional cell and undifferentiated.
- Germ cell – these include Dysgerminoma, endometrial sinus tumor, immature teratoma, embryonal carcinoma, choriocarcinoma, gonadoblastoma and mixed germ cell tumors.
- Sex cord and stromal tumours – These include granulosa cell tumor, fibroma, thecoma and sertoli-leydig cell tumors.
- Metastasis – These may arise from the cervix, endometrium, breast, stomach or the lymph nodes.

Epithelial tumors accounts for over 60% of all ovarian neoplasms, and more than 90% of malignant ovarian cancer. Serous cystadenocarcinoma is the most common malignant tumor of the ovary. Our patient presented with this tumor.

Ovarian tumor typically develops as an insidious disease, with few signs and symptoms. Our patient presented with a slowly growing abdominal mass and dull abdominal pains. There were no other symptoms and signs. A history of non-specific gastrointestinal complaints, including nausea, dyspepsia and altered bowel habits may occasionally be elicited. Other symptoms and signs like abdominal distension secondary to ascites generally indicate advanced diseases. Menstrual abnormalities may be noted in as many as 15% of reproductive age patients with ovarian cancer. Abnormal vaginal bleeding may also be a presenting complaint. Androgen producing tumors may cause virilization, or hirsutism. Granulosa and thecal cell tumors are estrogen producing tumors that usually present with abnormal uterine bleeding or precocious puberty in young girls.

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The prognosis of ovarian cancer is markedly improved with early diagnosis. Unfortunately, no clearly useful screening methods have been developed. The value of tumor makers and ultrasonography in the screening for ovarian cancer has not being clearly established in prospective studies (2, 3). In the evaluation of a patient with suspected ovarian tumor, several factors must be considered. The age of the patient, characteristics of the mass on both pelvic and sonographic examination as well as presenting complaints. The work up should probably include a thorough physical examination, Radiographic and ultrasonographic examination, as well as laboratory studies. Screening for the various tumor markers for gynecologic malignancies should also be undertaken. Some of these tumor markers include (4): -

- Ovarian epithelial tumor – CA – 125, NB 70K, LASAP.
- Gestational trophoblastic disease – beta – HCT
- Dysgerminomas – LDH and HCG
- Endodermal Sinus tumor – AFP and LDH
- Sertoli – Leydig tumour – Testosterone
- Granulosa cell tumors – Estradiol
- Endometrial carcinoma – CA – 125

However, the risk of malignancy using an index incorporating CA 125, menopausal status and ultrasound is superior to any tumor markers alone in the differential diagnosis of a pelvic mass (5).

Surgery is the mainstay of therapy for ovarian cancer, regardless of the cell type or stage of the disease. A gynecologic oncologist should be consulted. Surgical procedures for ovarian cancer aim for: Surgical staging; debulking of advanced disease; secondary debulking of recurrent or progressive disease and palliation for ovarian cancer induced intestinal obstruction. Accepted surgical procedures may include; hysterectomy and bilateral salpingoophorectomy, resection of fixed ovarian tumor, pelvic lymphadenectomy, paraaortic lymphadenectomy, omentectomy, small bowel resection and bypass, large bowel resection; partial gastrectomy, splenectomy, ureteral resection and debulking of liver, diaphragm and intestinal metastasis (4). Our patient underwent abdominal hysterectomy and bilateralsalpingoophorectomy. No tumour seedlings were noted on the partial omentectomy and lymph nodes. She was placed at stage IC.

Ovarian malignancies are stage according to the international federation of gynecologists and obstetricians (FIGO). The FIGO staging is based on findings at surgical exploration:

FIGO STAGING OF PRIMARY CARCINOMA OF THE OVARY

STAGE		DESCRIPTION
STAGE I		Growth limited to the ovaries
	1a	Growth limited to one ovary; No ascites containing malignant cells. No tumors on external surface; capsule intact
	1b	Growth limited to both ovaries. No ascites with malignant cells. No tumors on external surface; capsule intact.
	1c	Tumor either stage 1a or 1b but with tumor on the surface of one or both ovaries; or with capsule ruptured; or ascites with malignant cells or positive peritoneal washings.
STAGE II		Growth involving one or both ovaries with pelvic extensions
	IIa	Extension and /or metastasis to the uterus and /or fallopian tubes.
	IIb	Extension to other pelvic tissues
	IIc	Tumor either stage IIa or IIb but with tumour on the surface of one or both ovaries; or with capsule(s) ruptured; or ascites containing malignant cells or positive peritoneal washings.
STAGE III		Tumor involving one or both ovaries with peritoneal implants outside the pelvis and / or positive retroperitoneal or inguinal nodes. Superficial liver metastasis. Tumor limited to the true pelvis with histologically proven malignant extension to small bowel or omentum.
	IIIa	Tumor grossly limited to the true pelvis with negative nodes but histologically proven microscopic seedlings of abdominal peritoneal surfaces.
	IIIb	Tumor one or both ovaries, with histologically proven implants on abdominal peritoneal surfaces, none exceeding 2cm in diameter. Negative nodes
	IIIc	Abdominal implants >2cm in diameter or positive retroperitoneal or inguinal nodes or both
STAGE IV		Growth involving one or both ovaries with distant metastasis. Positive cytology of pleural effusion. Parenchymal liver metastasis.

Our patient was allotted to stage 1c, with high risk because of the histologic type-clear cell, surface excrescences, high grade, and tumor growth through the capsule. She was therefore started on chemotherapy.

The type of chemotherapy given depends on the patients overall health and status. One common regimen utilizes Cisplatin 50 – 100mg/m² and Cyclophosphamide 750-1000mg/m² given every 3 weeks for 6-8 cycles. Potential toxicities of this regimen include alopecia, Nephrotoxicity, Ototoxicity and Myelosuppression. This was the regimen used in our patient. Another regimen uses Carboplatin (an analogue of Cisplatin) and Paclitaxel for three to six cycles. Assessment of response to chemotherapy is based on physical examination, changes in the size of palpable or Radiologically measurable lesions as well as changes in CA-125 levels. Although the pre-operative CA-125 levels do not correlate with the tumor burden, changes in response to chemotherapy appear to be of prognostic benefit.

The prognosis for patients with ovarian cancer is primarily related to the stage of the disease at diagnosis. The five year survival rate for patients with stage one epithelial ovarian tumor is approximately 80% stage II, 40-50%, stage III, 30% while stage IV is less than 10%. In general, germ cell tumors have a better 5 years survival compared to epithelial ovarian tumors.

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GYNAECOLOGY CASE 8

CARCINOMA OF THE CERVIX STAGE 2B – TREATED BY RADIOTHERAPY

Name	L.N.	D o A	2/8/04
Age	40	D o D	25/8/04
Parity	2+1	1P. No.	0959080
Ward	1 D		

PRESENTING COMPLAINTS.

L.N Presented with a recurrent vaginal bleeding, general weakness and easy Fatigability.

HISTORY OF PRESENTING COMPLAINTS

L.N was a re-admission, having being admitted in the same ward on 19/5/04 and discharged on 6/6/04. At her initial admission L.N presented with complaints of abnormal vaginal bleeding associated with a foul smelling vaginal discharge for about 3 months. The bleeding was often heavy and came in clots. She also complained of dizziness, headache, weakness and easy Fatigability. A diagnosis of cancer of the cervix with severe anaemia was made. She was transfused 4 units of blood. Subsequently an examination under Anaesthesia was done on 4/6/04. She was discharged on haematinics, and antibiotics awaiting histology results for treatment. She was readmitted on 2/8/04 with heavy vaginal bleeding and easy fatigability.

OBSTETRICS AND GYNAECOLOGY

She was a Para 2 + 1, whose first delivery was in 1986 and had an abortion in 2000. The first and second deliveries were spontaneous vertex deliveries (SVD), and both children, girls were alive and well. She had an induced abortion at 3 months in 2000 at a private clinic. Her menarche was at 16 years, cycles were regular and menses occurred every 21-24 days and lasted 3-4 days. There was no associated dysmenorrhoea. She had used oral contraceptives from 1982 to 1990. From 1990 she had used Depo-provera injection for contraception. Her last normal menstrual period was on 25/4/04. She had about 10 sexual partners in her lifetime. She had never had a Pap smear examination.

FAMILY AND SOCIAL HISTORY

She was a single lady, who was a hawker in the city of Nairobi. She did not smoke cigarettes but took alcohol occasionally. She was the 4th born in a family of 8 siblings. *There were no chronic ailments in the family.*

PAST MEDICAL HISTORY

Other than during her deliveries, she had never been admitted to hospital. She had never had any surgical operation.

PHYSICAL EXAMINATION

She was a middle-aged lady in fair general condition who looked weak. She was mildly pale, did not have Jaundice, edema or lymphadenopathy. Her vital signs were a blood pressure of 100/60 mmhg; pulse rate of 98/min; respiratory rate 14/min, and a temperature of 36.5° C.

CENTRAL NERVOUS, CARDIOVASCULAR, AND RESPIRATORY SYSTEMS.

These were essentially normal.

ABNORMAL EXAMINATION

The Abdomen was scaphoid, non-tender, and had no therapeutic or surgical scars. There were no masses or organomegaly.

PELVIC EXAMINATION

She had normal external genitalia, with some blood on the Introitus. On speculum examination there was a friable mass in the cervix, involving the vaginal vault. Digital examination was not done.

EXAMINATION UNDER ANAESTHESIA - FINDINGS

On speculum the above findings were confirmed. On digital examination there was a friable mass involving the cervix and upper vagina. There was hardening of the left parametrium, but no involvement of the pelvic sidewall. On rectal examination, the rectal mucosa was free. A Biopsy was taken.

DIAGNOSIS

A diagnosis of cancer of the cervix stage 11 b was made. A hemogram, urea and electrolytes, liver function tests and a chest x-ray were ordered to facilitate management. Blood group and cross match was also requested.

RESULTS OF INVESTIGATIONS

Urea and electrolytes – urea 4.1 mmol/l; Creatinine 95 micromoles/L; Na⁺ 134 mmol/l; K⁺ 3.5 mmol/L; CL⁻ 96 mmol/l

- Hemogram
 - Hb level - 9.1 g/dL
 - WBC count - $8.4 \times 10^3/\text{mm}^3$
 - RBC count - $3.44 \times 10^6/\text{mm}^3$
 - Platelets - $378 \times 10^3/\text{mm}^3$
- Liver Function Tests
 - Total protein 13.4 g/dL
 - Albumin 3.5 g/dL
 - Total bilirubin 4.1 μ mol/l
 - Direct bilirubin 2.2 μ mol/l
 - AST 13 i.u/l
 - ALT 8 i.u/l
 - ALP 71 i.u/l
- Chest X-ray - Normal chest radiograph
- Biopsy results - infiltrating moderately well differentiated squamous cell carcinoma

DEFINITIVE MANAGEMENT

After the histology report confirmed cancer of the cervix, she was counseled on the extent of the disease and the treatment modality available. She was transfused 2 units of blood and planned for radiotherapy. She went for radiotherapy on the 23.8.04. Subsequently the vaginal bleeding stopped and she was allowed home on hematinics, for more radiotherapy sessions as an outpatient.

DISCUSSION

Presented is a 40-year-old Para 2 + 1, who was admitted with anemia secondary to cancer of the cervix. She was transfused and received radiotherapy, which helped control the vaginal bleeding. She was booked for more radiotherapy sessions as an outpatient.

Cervical carcinoma is the most common gynecologic malignancy in the developing world. In the developed world, in the United States for instance, the incidence and mortality rates for cervical carcinoma have declined by as much as 70-75% (1). This has been attributed to the effective screening programme using the papanicolaou's (pap) smear test in detecting pre-malignant precursors (2). Despite this, however, cervical carcinoma remains a significant health care problem worldwide. Even in the USA, 13,000 new cases of invasive cancer resulting in about 4,100 deaths were anticipated in 2002(2). The True incidence in Kenya is unknown although it is the most common gynecological cancer encountered in clinical practice (3). Studies in Kenya show that the mean age of onset is 42 years, with peak incidence at 25, 30 and 35 years. This is 10 years earlier than in the developed world, where two peaks of incidence at 35 years and 50-55 years have been found (2,4).

Past studies have always indicated that cervical cancer is a disease with multifactorial causes and long latency. Recently however, unlike most other cancers, in which multiple environmental, biological, and lifestyle determinants contribute independently or jointly to carcinogenesis, cervical cancer has now been shown to have a central causal agent, Human Papillomavirus (HPV), infection (5,6,7), whose contribution to the risk of

the disease is much greater than that of any other recognized determinant (8). Indeed some authors have questioned the existence of HPV-negative cervical carcinoma. They have proposed that the occurrence of cervical cancer "without involvement of specific HPV's is exceptional or impossible". HPV types have been assigned to high and low risk categories on the basis of their propensity to malignant transformation. HPV types 16, 18,31,33,35 and 45 are considered high risk whereas types 6,11,42,43 and 44 are considered low risk (9). Indeed other factors previously associated with increased risk of cervical intraepithelial neoplasia such as increased lifetime number of sexual partners, early age at first intercourse, lower level of education and lower socio-economic factors are now thought to confer this risk by co-existent HPV exposure and infection (10).

Additional risk factors for cervical cancer include cigarette smoking, long-term use of oral contraceptives, certain nutritional deficiencies and immuno-suppression. Women infected with the human immunodeficiency virus (HIV) are nearly five times as likely to develop cervical neoplasia compared to those who are not infected (11). It has also been shown that if cervical cancer does develop in an HIV-positive woman, it may be more aggressive and less responsive to treatment compared to HIV-negative women. Additionally, HIV infection and more advanced disease as determined by lower CD4 levels and higher viral load have been found to be a major risk factor for HPV infection.

Carcinoma of the cervix is a progressive disease; it begins with intra-epithelial, preneoplastic changes, which may develop over a period of the time into invasive cancer. Cervical cytology by means of Papanicolaou's smear offers great hope for early diagnosis and treatment of these pre-malignant conditions of the cervix. This is possible because:

- (i) Cells from precancerous lesions exfoliate and can therefore be detected.
- (ii) The cervix is a highly accessible organ for examination
- (iii) There exists a spectrum of changes from cellular atypia to carcinoma
- (iv) The Natural history of cancer of the cervix takes several years.

Most Kenyan studies have indicated that patients with carcinoma of the cervix present late, when survival rates are low (3). In the case presented, the patient had been treated for abnormal vaginal bleeding for over one year, with cancer of the cervix not being considered a possible diagnosis. This led to a delay in diagnosis and possible progression of the cancer. At KNH, only 11% of patients have been found to present with stage 1 disease while stage II accounts for 27% (12).

The symptoms of cancer of the cervix include abnormal vaginal bleeding (most common), postcoital bleeding, vaginal discharge, pelvic and referred pain and swelling of the lower limbs. In early stages, carcinoma of the cervix is asymptomatic.

Histologically, cancer of the cervix is usually squamous cell type (90%), while Adenocarcinoma accounts for about 10% of cervical carcinoma (2). These could be well, moderately or poorly differentiated. Our patient had moderately well differentiated squamous cell carcinoma.

The prognosis of cancer of the cervix depends on the stage at presentation, Histologic type and grade as well as the size of the tumor. It can be treated surgically or by radiotherapy. Each mode of treatment carries its own indications and complications. Wertheim's hysterectomy is the preferred surgical mode of treatment for stage 1B-2A disease. The operation may be complicated by Fistula Formation, stress incontinence, and venous thromboembolism (2). Radiation therapy may be used in all stages of the disease. It may be complicated by bowel perforation and Fistula formation among other common complications. There have recently been great strides in the treatment of cervical carcinoma, including adjuvant radiation and chemo radiation in patients discovered to have high-risk cervical carcinoma after radical hysterectomy and in patients with locally advanced cervical carcinoma.

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FAMILY AND SOCIAL HISTORY

She was the 6th born in a family of 8 siblings. She was single and worked as a house help in Hurlingham. She stayed with an aunt in Kangemi. She drunk alcohol occasionally and smoked cigarettes infrequently. There was no family history of chronic family ailments.

PAST MEDICAL AND SURGICAL HISTORY

This was non - revealing

PHYSICAL EXAMINATION

She was a young lady in good general condition but in pain. She was not pale, did not have edema, jaundice or lymphadenopathy. There was no oral thrush. Her blood pressure was 110/70 MMHg pulse rate of 81/minute, temperature of 37.6 degrees Celsius and a respiratory rate of 14/ minute.

ABDOMINAL EXAMINATION

It was soft, non tender and moving with respiration. There were no palpable masses or organomegaly.

PELVIC EXAMINATION

This revealed a marked indurated and tender swelling on the right labia majora extending to about 4 by 6 cm. there was a yellowish per vagina discharge at the introitus. There was no axillary adenopathy. There were no ulcers.

OTHER SYSTEMS

There were essentially normal.

DIAGNOSIS

A diagnosis of a right-sided Bartholins abscess was made. The patient was admitted for marsupialisation.

MANAGEMENT

On admission, she was commenced on antibiotics and analgesics. She was put on

I.M Tramal 100mg start

I.V Crystapen 2 Mu q6h

I.V Flagyl 500mg q8h

Intravenous augmentin 1.2g q8h was ordered to substitute Crystapen when available

A haemogram, urea/creatinine were ordered to prepare the patient for theatre. These were found to be normal.

PROCEDURE

Informed consent was obtained and patient premedicated with I.M atropine 0.6mg half hour before theatre. In theatre, a vulvovaginal toilet was done. A wedge shaped vertical incision was made in the vaginal mucosa over the center of the abscess. The abscess cavity was opened and drained of pus. The pus was taken for culture and sensitivity. The lining of the abscess cavity was then everted and sutured to the vaginal mucosa with interrupted sutures of No. 2/0 Vicryl. General Anaesthesia was successfully reversed.

Post operatively; she was started on intravenous Augumentin 1.2g q8h for 24 hours. On the third post-operative day she was commenced on oral Augumentin 625 mg twice daily Ponstan 500mg three times a day and allowed home. Four weeks later in the gynecology clinic she was doing well.

DISCUSSION

Presented is a 25 year old Para I+O who presented with Bartholins abscess underwent marsupialisation under general anaesthesia with good recovery.

Obstruction of the main duct of the Bartholins gland results in retention of secretions and cystic dilatation. Infection is the major cause of obstruction. Insipissated mucus and congenital narrowing of the duct may also lead to obstruction. Little is known about the

incidence and complications of Bartholins gland infection in women. It is most commonly associated with low socioeconomic status, multiparity and prior history of sexually transmitted infections (1). It's most prevalent in the 20 – 29 years age group. Our patient was a househelp and had being treated previously for sexually transmitted infection. Before the current illness she had unprotected sexual intercourse with a new acquaintance. Indeed many authors have suggested that Bartholins infection is a sexually transmitted infection. However, recurrent infections or abscess may be the consequence of scarring of the gland duct damaged by infection (1, 2).

The microbiology of Bartholins gland infection is similar to that of pelvic inflammatory disease (2, 3). Most abscesses have mixed aerobic and anaerobic pathogens. Some will be single microorganisms while in upto one third of the cases, no pathogen is isolated. The predominant pathogens are those of the vaginal flora including gram - negative anaerobic rods, gram – positive anaerobic rods, Escherichia coli, staphylococci and streptococcus. Some investigators have isolated Neisseriae gonorrhoea and even Chlamydia trachomatis (3, 4). Mycoplasma hominis and ureaplasma urealyticum do not appear to be important pathogens in Bartholins gland infection.

The clinical presentation of Bartholins gland infection is usually at the abscess stage. Abscesses are generally unilocular and several centimeters in diameter. The surrounding erythema, induration and tenderness may obscure actual abscess size.

Several treatment modalities have been proposed for management of Bartholins gland abscess. These include incision and drainage marsupialisation and word catheter drainage. None of these therapeutic options have undergone randomized prospective evaluation in sufficiently large trials to exclude differences in outcome(s). The goals of surgical treatment of Bartholins abscess include: adequate drainage of infected gland; preservation of the gland to continue with its secretory function; creation of new gland

ostium or fistula to replace the function of the damaged duct to prevent recurrences and prevent the complications of Bartholins abscess such as necrotizing fasciitis and sepsis.

There is need to carry out more research on Bartholins infection at the KNH, the last such study having being undertaken by Mumia (6) over 20 years ago. At that time Bartholins abscess accounted for 1.7% of emergency gynaecological admissions and 55% of these patients were pregnant.

Our patient possibly developed Bartholins gland infection as a consequence of sexually transmitted infection most likely gonorrhoea.

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GYNAECOLOGY CASE 10

BLIGHTED OVUM – DILATATION AND CURRETAGE.

NAME T. W	DOA	18/5/04
AGE 30 YEARS	DOD	18/5/04
PARITY 1 + 0	I P. No.	26332

Ward 1D

PRESENTING COMPLAINT

T.W presented to WD ID casualty as a referral from a private clinic with a diagnosis of missed abortion.

HISTORY OF PRESENTING COMPLAINTS

T. W was otherwise well and had her last menstrual period on 31/1/04. She had not done any pregnancy test however. One week prior to admission she visited a private clinic in Nairobi to start her antenatal clinic. She was attended to, and a pelvic scan requested to confirm the pregnancy. To her dismay, the pelvic scan revealed that she had a blighted ovum and was referred to KNH for evacuation.

OBSTETRIC AND GYNECOLOGY HISTORY

T.W was now Para 1 + 0, her last delivery having being 10 years earlier while still in school. The delivery was a spontaneous vertex delivery (SVD), and her son was alive and well.

Her menarche was at 15 years, and her menses were regular, lasting 4 –5 days, and occurring every 28 – 30 days. There was no associated dysmenorrhoea. She had used oral contraceptive pill for over 10 years, having stopped using them about 1 year earlier in order to get a child. She had never had a pap smear done.

PAST MEDICAL HISTORY

She had been admitted about 2 year earlier with malaria and discharged after 3 days. She had never had any surgical operation.

FAMILY AND SOCIAL HISTORY

She was now married for about one and a half years and lived with her husband in Kayole. Both were business people. There were no known chronic ailments in their family.

PHISIAL EXAMINATION

She was a young lady in good condition and well nourished. She did not have pallor, Jaundice, edema or lymphadenopathy. Her vital signs were normal with a blood pressure of 110/70 mmhg; pulse of 74/min; respiratory rate of 15/min and a temperature of 36.7⁰c.

CENTRAL NERVOUS SYSTEM; RESPIRATORY AND CARDIOVASCULAR SYSTEMS.

These were essentially normal.

ABDOMINAL EXAMINATION

The abdomen was scaphoid, non-tender and moving with respiration. There were no masses palpable or any organomegaly.

PELVIC EXAMINATION

There were normal external genitalia. The cervix was posterior, long, firm consistency and closed. The uterus was about 8 weeks. The adnexae and pouch of Douglas were free. There was no blood or abnormal discharge on the examining finger.

PELVIC SCAN

A pelvic scan done at Nairobi hospital showed an empty gestational sac with no fetal pole. The radiologist had concluded that it was a blighted ovum.

MANAGEMENT

The patient was told of the management of the condition. Informed consent was obtained and blood drawn for hemogram, urea and electrolytes.

RESULTS OF INVESTIGATIONS

- ◆ Hemogram - Hb 12.8 g/dl
- Platelets $356 \times 10^9/L$
- ◆ Urea 4.8 mmol/L; creatinine 68 micromoles/ L. Na^+ 138 mmol/L; K^+ 4.3 mmol/L.

PROCEDURE.

The patient was premedicated with I.M atropine 0.6 mg ½ hour before being wheeled to theatre.

In theatre she was put under general anaesthesia and repositioned to the lithotomy position. Vulvalvaginal toilet was done with chlorohexidine solution and patient draped. Bimanual examination revealed an antverted uterus about 8 weeks in size. A Sims speculum was introduced into the vagina and the anterior lip of the exposed cervix grasped with a tenaculum. The cervix was drawn and cleaned with Betadine.

The cervix was gradually dilated with metal Hegar's dilators; till no 10 Hegar's dilator could pass easily. A curette was then introduced into the uterine cavity, and the products of conception scraped away. About 100cc of POC's were evacuated. Once a grating sound was heard, the instruments were removed, patient cleaned and anaesthesia successfully reversed.

POST – OPERATIVE CARE

Once awake in the ward, the patient was allowed to eat normally. She was allowed home on Doxycycline 100mg twice daily and Metronidazole 500mg three times a day for one week. Since she wanted a baby, she declined any contraceptive advice.

DISCUSSION

The patient presented was a 30-year-old Para 1 + 0, diagnosed to have a blighted ovum by ultrasound. She underwent dilatation and curettage under general anaesthesia with no complications.

The great embryologist and morphologist, Frank Mall, observed early in the 20th century that embryonic abnormalities were far more frequent than were congenital anomalies in fetuses or neonates at term gestation. Specific anomalies are not readily detected in early embryos but generally disorganized growth is recognized. The vast majority (about 85%) of conceptuses spontaneously aborted in the first 8-10 weeks of gestation and were abnormal. Among these growth-disorganized conceptuses is the one with a chorionic vesicle without an umbilical cord remnant or embryo commonly referred to as a "Blighted ovum", but which is better termed descriptively as an "empty chorionic vesicle" or "unembryonic sac". (1)

Hertig described a condition that he termed "Blighted ovum" (2), early in the 20th century. This term is probably a misnomer because the condition appears to arise after implantation as a result of the death or distortion of the embryonic disk after the trophoblast has differentiated. A high level of chromosomal abnormalities have been found in blighted ovum (2); compared with their embryonic counterparts, spontaneous abortion rates linked with blighted ova were 14% higher (67%); exhibited a much higher rate of trisomies (74% vs 35%), especially in chromosomes 16 and 22; and totally lacked 45 X karyotype (2). It was therefore speculated that arrest in early embryonic development might be correlated to genes on autosomal chromosomes, particularly 16 and 22.

Blighted ova survive for longer than biochemical pregnancies, usually 30 days or longer, or even through the majority of the first trimester. The patient may be asymptomatic or present with regression of the symptoms and signs of pregnancy. More often a patient has missed her menses and expects to be pregnant only for the

pregnancy test to be negative. Falling or abnormally low plasma levels of, B HCG are predictive of an abnormal pregnancy, among them a blighted ovum.

On ultrasound, an abnormal gestational sac, without a yolk sac or embryo, is consistent with a blighted ovum (3). At abortion, no obvious fetal parts are found; and when examined microscopically, the tissue is entirely trophoblastic (2,3).

A blighted ovum may miscarry (85%) or require Dilatation and curettage since it cannot continue normally. Our patient had dilatation and curettage done and was discharged home in good condition

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GYNAECOLOGY CASE 11

HYDATIDIFORM MOLE – SUCTION AND SHARP CURETTAGE

NAME: Z. A
IP. NO: 0977963
D.O.A: 24/7/04
D.O.D: 3/7/04
AGE: 34 YEARS
PARITY: 2 + 2; 1 LIVING CHILD
WARD: 1D

PRESENTING COMPLAINTS

Z. A. presented with vomiting, anorexia and vaginal bleeding after manual vacuum aspiration for incomplete abortion at Kisii District Hospital.

HISTORY OF PRESENTING COMPLAINTS

Z.A. last menstrual period was on 24/4/04 and was pleased to be pregnant again. However in Mid June 2004, she developed persistent vaginal bleeding, and lower abdominal pain, and was seen at Kisii District Hospital where a diagnosis of incomplete abortion was made and manual vacuum aspiration done. At that time the vomiting and nausea was said to be due to pregnancy. However about 2 weeks after MVA, the vaginal bleeding recurred, associated with nausea and vomiting. Soon thereafter she developed lower abdominal pain and swelling. She sought help at the KNH.

OBSTETRIC AND GYNECOLOGY HISTORY

Z.A. was now Para 2+2. Her first delivery was in the year 1994, by cesarean section because of cephalopelvic disproportion (CPD). The outcome was a life male infant who was her only living child. In 1997, she had a molar pregnancy diagnosed at the KNH, where evacuation was done. She was however lost to follow up. In 1999 she had her 3rd pregnancy, which was complicated by preterm premature rupture of membranes (PPROM). A cesarean section was done but the baby succumbed to the complication

of prematurity – respiratory distress syndrome. Her last pregnancy was the abortion preceding her current admission which was evacuated at Kisii District Hospital.

Her menarche was at the age of 14 years, menses were regular, occurring at an interval of 26 days and lasting 3-5 days. She had never used any form of contraception. She had never had a pap smear done.

PAST MEDICAL HISTORY

This was non-contributory.

FAMILY AND SOCIAL HISTORY

She was married and lived mainly in Kisii. However she often visited her husband who was a hotel chef and lived in Dandora. She did not drink alcohol NOR smoke cigarettes. There was no family history of chronic ailments.

SYSTEMIC ENQUIRY

Non – revealing.

PHYSICAL EXAMINATION

She was a middle-aged lady who was in fair general condition. She was however pale, but did not have jaundice, cyanosis, edema or lymphadenopathy. Her vital signs were a blood pressure of 110/70 mmHg, pulse of 80/minute, respiratory rate of 16/min and a temperature of 36.5°C.

RESPIRATORY, CARDIOVASCULAR AND CENTRAL NERVOUS SYSTEMS WERE ESSENTIALLY NORMAL

ABDOMINAL EXAMINATION

The abdomen was distended in the supra pubic region. There was a midline sub-umbilical scar. There was tenderness in the lower abdomen. There was a pelvic mass, thought to be the uterus extending to the level of the umbilicus. It felt firm, was mobile from side to side, and was slightly tender. There were normal bowel sounds.

PELVIC EXAMINATION

There were normal external genitalia. The vaginal walls were also normal. The cervix was posterior, closed and long. The uterus was bulky to approximately 20 weeks equivalent and slightly tender. The Adnexae were free and the pouch of Douglas empty. Cervical excitation test was negative. There was blood on the examining finger.

TENTATIVE DIAGNOSIS

A diagnosis of recurrent hydatidiform mole was made. A pelvic scan, serum B HCG Levels, full hemogram, urea, creatinine and electrolytes were ordered. A chest radiograph was also requested.

RESULTS OF INVESTIGATIONS

Pelvic scan – showed a non-gravid uterus, which was enlarged measuring 182 by 141 by 92 mm. It was seen as a huge almost homogenous mass giving the impression of a ‘Snowstorm’. No Adnexal masses were seen.

Serum B, HCG levels. - 200 000 miu/ml – Normal below 5MIU/ML.

Hemogram - WBC $4.7 \times 10^6/L$
- RBC $3.54 \times 10^6/L$
- Hb 9.5 g/dl
- HCT 30.1%
- Platelets $200 \times 10^9/L$

Urea 12.1 mmol/l; creatinine 53 μ/L

Na⁺ 134 mmol/L, K⁺ 4.3 mmol/L.

Chest x-ray – Normal

A diagnosis of recurrent H. mole was confirmed and patient informed of the diagnosis. The management was explained to her and informed consent obtained. Blood was drawn for blood group and cross-match. She was prepared for suction curettage in theatre. She received I.M Atropine 0.6mg start.

PROCEDURE

In theatre, she was placed in lithotomy position and general anaesthesia induced. An oxytocin infusion of 40 I.U was begun before the induction of anaesthesia. Vulvo-vaginal toilet was done, and the bladder aseptically catheterized. A speculum was introduced to expose the cervix, which was cleaned with Betadine. It was held with a tenaculum. A uterine sound was introduced but the cavity was too long. Progressive cervical dilatation was done using Hank's dilators. When the 8mm dilator was inserted there was a gush of blood from the uterus. The cannula was introduced into the uterine cavity and connected to the vacuum apparatus. Within about 10 minutes the uterus had decreased dramatically in size. About 2.7 litres of blood and vesicles were evacuated. Bimanual palpation of the uterus was done to stimulate uterine contraction. The bleeding soon subsided and a vaginal pack was left. One unit of blood was transfused. Anaesthesia was reversed.

A day after the suction curettage, the patient was discharged home on antibiotics and hematinic Ranferon to come back for sharp curettage after 10 days.

On 10/8/04, she was taken back to theatre and gentle sharp curettage done. There was minimal residual tissue, which was sent for histology. The uterine size had regressed to about 8 weeks equivalent. She was discharged home on oral contraceptive pills and weekly review in the GOPC, with B HCG levels.

FOLLOW UP

On follow up, the serum, B HCG levels fell to undetectable levels by the 4th week. She was then scheduled for monthly follow up, with B HCG levels.

DISCUSSION

The patient discussed here was a 34-year-old Para 2+2, who had hydatidiform mole in 1997, and presented, yet again, with another mole after an abortion at about 9 weeks. She underwent suction and sharp curettage.

Hydatidiform mole belongs to a spectrum of pregnancy related trophoblastic tumors. It comprises of abnormal proliferation of the syncytiotrophoblast and abnormal replacement of normal placental trophoblastic villi by hydropic placental villi. The grapevine vesicles fill and distend the uterus (1). The incidences of molar pregnancies vary in different parts of the world. The incidence in Europe and North America has been reported to be between 0.6 and 1.1 per 1000 pregnancies (2), compared to 2 per 1000 pregnancies in Japan.

Case control studies have identified how dietary intake of carotene and thus vitamin A to be associated with complete molar pregnancy (3). Dietary factors may therefore explain regional variations in the incidence of molar pregnancy. This may also be related to socioeconomic factors. Maternal age older than 35 years has consistently been shown to be a risk factor for complete moles. Ova from older women are thought to be more susceptible to abnormal fertilization. In one study, the risk of a complete mole was found to be increased 2.0 fold for women older than 35 years, and 7 – 5 fold for women older than 40 years (4). Limited information is available concerning the risk factors for partial molar pregnancy. However, these have been shown to differ. For instance, there is no association between maternal age and risk of partial mole (4). The risk of partial mole has been reported to be associated with the use of oral contraceptives and a history of irregular menstruation, but not with dietary factors (5). Hydatidiform mole may be categorized as either complete or partial moles on the basis of gross morphology, histopathology, and karyotype (1). In histopathology, complete moles lack identifiable embryonic or fetal tissues and the chorionic villi exhibits generalized hydatidiform swelling and diffuse trophoblastic hyperplasia. Cytogenetic studies have demonstrated that complete hydatidiform moles usually have a 46, XX karyotype, and the molar chromosomes are entirely paternal in origin (6). It appears that complete moles usually arise from an ovum that has been fertilized by a haploid sperm, which then duplicates its own chromosomes. The ovum nucleus may either be absent or inactivated (7). Although most complete moles have a 46, XX chromosomal pattern, about 10% have a 46 XY karyotype (8). Chromosomes in a 46, XY complete

mole also appear to be entirely of paternal origin, although mitochondrial DNA is of maternal origin (9).

Partial hydatidiform moles are characterized by the following pathologic features (10): Chorionic villi of varying size with focal hydatidiform swelling, cavitations and trophoblastic hyperplasia; marked villous scalloping; prominent stromal trophoblastic inclusions, and identifiable embryonic or fetal tissues. Partial moles are generally triploid in karyotype (69 chromosomes); the extra haploid set of chromosomes usually being derived from the father (11). The patient discussed had a complete hydatidiform mole for the 2nd time in her life, having had a similar condition 6 years earlier (1997).

The classical and current clinical features of a complete mole include:

- Vaginal bleeding – most common symptom. Currently reported to occur in 84% of patients (12). Because vaginal bleeding may be considered and prolonged, about one half of these patients have anemia (Hb <10.0g/dl).
- Excessive uterine size – excessive uterine enlargement relative to gestational age is one of the classic signs of a complete mole, although its present in only half of the patients.
- Preeclampsia – preeclampsia almost exclusively develops in patients with excessive uterine size and markedly elevated HCG levels. Hydatidiform mole should always be considered whenever preeclampsia develops early in pregnancy.
- Hyperemesis gravidarum – This may lead to electrolyte disturbances which require treatment with parenteral fluids.
- Hyperthyroidism – This develops almost exclusively in patients with very high HCG levels. Some investigators have suggested that HCG is the thyroid stimulator in women with GTD; because positive correlations between serum HCG and total T3 or T4 concentrations have been observed.
- Other rarer presentations include trophoblastic embolization and Theca Lutein ovarian cysts. The former may lead to respiratory distress characterized by chest pain, dyspnoea, tachynea, and tachycardia. The patient discussed presented with vaginal bleeding, excessive uterine enlargement and hyperemesis gravidarum.

Patients with partial Hydatidiform mole usually do not have the dramatic clinical features characteristic of complete molar pregnancy. In general, these patients have the signs and symptoms of incomplete or missed abortion, and partial mole can be diagnosed after histologic review of the tissue obtained by curettage (13).

Ultrasonography is a reliable and sensitive technique for the diagnosis of complete molar pregnancy. Because the chorionic villi exhibit diffuse hydropic swelling, complete moles produce a characteristic vesicular, ultrasonographic pattern, the so called "snow storm". This was found in the ultrasound done in the patient discussed. Ultrasound may also contribute to the diagnosis of partial molar pregnancy, by demonstrating focal cystic spaces in the placental tissues and an increase in the transverse diameter of the gestational sac (14). When both of these criteria are present, the positive predictive value for partial mole is 90%.

After molar pregnancy is diagnosed, the patient should be evaluated carefully for the presence of associated medical complications, including pre eclampsia, hyperthyroidism, electrolyte imbalance, and anemia. After the patients' condition has been stabilized, a decision must be made concerning the most appropriate method of evacuation.

If the patient desires surgical sterilization, a hysterectomy may be performed with the mole in situ. The ovaries may be preserved at the time of surgery, even though prominent theca lutein cysts are present. They may be decompressed by aspiration. Hysterectomy does not prevent metastasis. Therefore, patients still require follow – up with assessment of HCG levels.

Suction curettage is the preferred method of evacuation, regardless of uterine size, for the patient who desires to preserve fertility. It involves the following steps:

1. Oxytocin infusion – This procedure is begun in the operating room before induction of anesthesia
2. Cervical dilatation – As the cervix is being dilated, the Surgeon frequently encounters increased uterine bleeding. However, active bleeding should not deter prompt completion of cervical dilation.
3. Suction curettage – The use of a 12 – mm cannula is strongly advised to facilitate evacuation. If the uterus is larger than 14 weeks of gestation, one hand should be placed on top of the fundus, and uterus massaged to stimulate uterine contraction and reduce risk of perforation.
4. Sharp curettage – when suction is believed to be complete, gentle sharp curettage is done to remove any residual molar tissue. At the KNH, this is done after 10 days.

Our patient underwent suction and sharp curettage.

The use of prophylactic chemotherapy at the time of molar evacuation is controversial. However in a prospective study in the United States, It was shown to prevent metastasis and reduce the incidence and morbidity of local uterine invasion (15). Indeed, the author recommends that prophylaxis may be especially useful in the management of high-risk complete molar pregnancy, especially where hormonal follow-up is unavailable or unreliable.

After molar evacuation, patients should be monitored with weekly determinations of the β , sub unit of HCG levels, until they are normal for three consecutive weeks, followed by monthly levels until normal for six consecutive months. At the completion of follow up, pregnancy may be undertaken.

Patients are encouraged to use effective contraception during the entire interval of HCG follow – up. If the patient does not desire sterilization, the choice is to use oral contraceptives or barrier methods.

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GYNAECOLOGY CASE 12.

IMPERFORATE HYMEN - HEMATOCOLPOS; HEMATOMETRA: CRUCIATE INCISION.

NAME: A.A. DoA: 1.4.04
AGE: 14 DoD: 3.4.04
PARITY: 0+0 IP NO. 16884
WARD: 1D

PRESENTING COMPLAINTS

She presented with cyclical lower abdominal pain and back pain for 6 months.

HISTORY OF PRESENTING COMPLAINTS

She was well till about 6 months prior, when she developed some discomfort in the lower abdomen. Every month, the discomfort increased to become cramping lower abdominal pain and back pain. Subsequently she noted a swelling in her lower abdomen. She had never had any menses. She did not have any urinary complaints. There was no change in the bowel habits.

OBSTETRICS AND GYNECOLOGY HISTORY

She was a nulliparous lady who had never had menses.

PAST MEDICAL HISTORY

She had never been admitted to hospital for any illness or surgical operation. She had no known chronic illness.

FAMILY AND SOCIAL HISTORY

She was the 2nd born in a family of 3 siblings. She was currently in class 7 in a local primary school. Her father was a primary school teacher while her mother was a businesswoman. There were no known chronic ailments.

PHYSICAL EXAMINATION

She was a young lady in good general condition and well nourished. She was not pale, did not have edema, cyanosis or lymphadenopathy. Her vital signs were normal with a blood pressure of 100/60 mmhg, pulse rate of 76/min, respiratory rate of 14/min and a temperature of 36.6°C.

CENTRAL NERVOUS SYSTEM: CARDIOVASCULAR AND RESPIRATORY SYSTEMS

These were essentially normal.

ABDOMINAL EXAMINATION

The abdomen was distended in the suprapubic region. There was a mass, which you could not go under approximately 18 weeks equivalent. It was tender and mobile side to side. Bowel sounds were present and normal.

PELVIC EXAMINATION

There were normal external genitalia. There was protrusion of the hymen as a mass that was dark in color. The mass was tender and further pelvic examination was abandoned.

RECTAL EXAMINATION

The rectum was full. The uterus could be felt, below which was a cystic mass thought to be the vagina.

DIAGNOSIS

Imperforate hymen with hematocolpos and hematometra. A pelvic ultrasound was requested.

MANAGEMENT

The pelvic ultrasound confirmed examination findings. The diagnosis was explained to the patient and her mother and they accepted to go to theatre for incision of the hymen.

A hemogram, urea, creatinine and electrolytes were requested. The mother signed a consent form.

RESULTS OF INVESTIGATIONS

- Hemogram - Hb 12.6 g/dl
- - RBC $4.2 \times 10^6/\text{mm}^3$
- - WBC $4.8 \times 10^3/\text{mm}^3$
- - Platelets $370 \times 10^6/\text{mm}^3$
- Urea 6.2 mmol/l
- Creatinine 50 micromoles/L
- NA+ 140 mmol/L. k+ 4.1 mmol/L

PROCEDURE

Half-hour before being wheeled to theatre, she was premedicated with 0.6mg I.M atropine. On the theatre table, she was put under general anaesthesia. She was re-positioned to the lithotomy position, Vulvo vaginal toilet and aseptic catheterisation done. Vaginal examination confirmed previous findings. The hymen membrane was incised, at 2, 4, 8 and 10 O'clock. The quadrants of the membrane were excised after drainage of about 2 litres of chocolate brown altered blood. The patient was cleaned and anaesthesia successfully reversed.

Post-operative care

She was put on Tramadal 50mg 8 hourly for 24 hours. 6 hours after coming from theatre she was started on oral sips. On the 2nd post-operative day she was discharged home on Augumentin 375 mg TID x 5/7, Brufen 400mg TID x 5/7 and follow-up in the Gynecology clinic in two weeks.

FOLLOW UP

In the gynecology clinic 2 weeks later, the patient was doing well and the abdominal swelling had disappeared. She was discharged from the clinic unless in need.

DISCUSSION

Presented is a 14-year-old schoolgirl with hematocolpos and hematometra due to imperforate hymen. Cruciate incision was done with good results.

The hymen is the junction between the sinovaginal bulbs and the urogenital sinus. It is a thin membrane sometimes cribriform in appearance composed of endoderm derived from the epithelium of the urogenital sinus (1). The hymen is usually perforated in embryonic life to establish a connection between the lumen of the vaginal canal and the vestibule. It is usually torn in pre-pubertal years. When there is no perforation through this membrane it is called imperforate hymen (1, 2, 3).

Imperforate hymen is perhaps the most common obstructive anomaly of the female genital tract, with an incidence of between 0.01 and 0.1% among female newborns. Most of the cases are sporadic with no evidence to suggest any genetic factor (4). It is almost always an isolated finding. Associated anomalies, including urinary tract anomalies, are rare. Some of these anomalies may include: imperforate anus, bifid clitoris, hypoplastic kidney and vascular abnormalities (4).

Imperforate hymen has no symptoms if the uterus is absent or functionless (5). If the uterus is present, symptoms usually occur at puberty. Occasionally in early childhood, placental hormones may stimulate the uterus and cervix leading to the collection of mucus in the vagina, causing mucocolpos, at birth (2,5).

Symptoms in imperforate hymen are due to the accumulation of menstrual blood. The blood of the first period or two is collected in the vagina. The vagina can hold blood from one or two cycles without undue stretching and with no other symptoms. Accumulation of menstrual blood in the vagina is termed hematocolpos. The patient may feel a little fatigue and have crampy discomfort suggesting menstruation, but no blood appears at the vaginal outlet. As menstruation recurs, the vagina becomes greatly over distended, and the cervical canal also dilates. Hematometra, which is the accumulation of menstrual blood in the uterine cavity, may form. This was the presentation in our patient. When the intrauterine pressure reaches a certain point,

there is retrograde passage of blood into the tubes, forming a hematosalpinx. Adhesion formation within or at the fimbriated end of the tubes can seal them, and little or no blood may enter the peritoneal cavity. If it enters the peritoneal cavity it forms hemoperitoneum (1, 2).

Diagnosis of imperforate hymen is rare before puberty. Most patients are brought to the doctor at 13 to 15 years of age when their mothers begin to notice symptoms and the girls appear not to have begun menstruating. At puberty, primary amenorrhea and cyclic cramping lower abdominal pain are the most common complaints. Other symptoms may include discomfort in the pelvis and how back pain (1, 2, 3). Urination can also be difficult, because pressure of the distended vagina on the urethra may compress the urethra and preventing emptying of the bladder. Cramps like pains recur in the supra-public region, a long with the common urologic symptoms of dysuria, frequency, and urgency. Overflow incontinence may develop eventually (1).

When the patient is examined, a tender mass is often palpable supra-pubically, the result of uterine enlargement and upward displacement, or bladder distension, or both. If hemoperitoneum occurs, the irritation of the free blood may cause the patient to experience all the symptoms and demonstrate signs of peritonitis. Our patient did not have these symptoms and/or signs. Vulval inspection reveals the bulging pink imperforate hymen, which may or may not be bluish in color depending on its thickness. On rectal examination, the vagina is palpable as a large cystic mass (6).

The management of imperforate hymen is usually surgical. This involves excision of the membrane to allow flow of the accumulated blood. The hymenal membrane is incised preferably at 2,4,8 and 10 O'clock. The quadrants of the hymen are then excised and the mucosal margins are approximated with fine delayed absorbable suture. If hematocolpos has already developed all unnecessary intrauterine instrumentation should be avoided because of the risk of perforating the thin overstretched uterus wall. To prevent scarring and stenosis, which could result in dyspareunia, the hymenal tissue should not be excised too close to the vaginal mucosa. Should the uterine mass fail to

regress in 2 - 3 weeks, then inspection and dilatation of the cervix should be performed to make certain that drainage from the uterus is satisfactory.

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GYNAECOLOGY CASE 13

PELVIC ABSCESS – LAPAROTOMY AND DRAINAGE

NAME: P.N. PARITY: 1 + 0
AGE: 18 DoA: 7/7/04
IP. NO: 0983827 DoD: 15/7/04
WARD: 1D

PRESENTING COMPLAINTS.

She was admitted with a one-week history of lower abdominal pain, fever and general weakness with associated foul smelling per vaginal discharge.

HISTORY OF PRESENTING COMPLAINTS

She was well till about one week prior to admission when the above complaints began. She had delivered on 20/6/2004 at home, assisted by traditional birth attendant (TBA). The placenta was retained but was removed in a local private clinic. Initially she was well, but about 1 week after delivery the Lochia became yellowish and foul smelling. She then developed lower abdominal pain, fever and weakness. She also had diarrhoea and occasional vomiting.

OBSTETRIC AND GYNAECOLOGY HISTORY

She was Para 1 + 0, this having been her 1st delivery. Menarche was at 14 years. Her cycles were regular, occurring every 28 days, and lasting 3-4 days. She had never used any form of contraception. She had never had a pap smear done.

PREVIOUS MEDICAL HISTORY

This was not significant.

FAMILY AND SOCIAL HISTORY

She was a high school student who stayed with her parents. She neither smoked cigarettes nor drank alcohol. There was no family history of chronic illness.

PHYSICAL EXAMINATION

She was sick looking; pale, febrile but did not have Jaundice or Lymphadenopathy. She looked dehydrated. Vital signs were:

Temperature: 38.6°C

Blood pressure: 100/60 mm hg.

Pulse rate: 108/minute

Respiratory rate: 24/minute.

RESPIRATORY, CARDIOVASCULAR AND CENTRAL NERVOUS SYSTEM EXAMINATION WAS NORMAL.

ABDOMINAL EXAMINATION

The left iliac region was slightly distended and tender. There was guarding but no rebound. Bowel sounds were heard. No masses or organomegally was found.

PELVIC EXAMINATION

The external genitalia were normal. There was a profuse yellowish per vaginal discharge. The vaginal walls were inflamed; the cervix was long, central and closed. Cervical excitation test was positive bilaterally. The adnexae and pouch of Douglas were full and very tender. Bimanual examination was abandoned because of the tenderness.

DIAGNOSIS

An impression of puerperal sepsis with pelvic abscess was made.

MANAGEMENT

The patient was started on parenteral antibiotics using a combination of crystalline penicillin 2 mu 6 hourly, Gentamycin 240mg daily, and Metronidazole 500mg 8 hourly. She was also put on intravenous fluid, 3l/24 hours, alternating Hartman's solution with 5% dextrose. Specimens were taken for blood cultures, full hemogram, urea, creatinine

and electrolytes, high vaginal swab and urine for microscopy, culture and sensitivity. She was also booked for an abdominal pelvic ultrasound.

RESULTS:

Hemogram: Hb – 7.5 g/dl

WBC – $15.8 \times 10^6/l$

Neutrophils – 75%

Lymphocytes – 20%

Monocytes – 5%

Electrolytes: BUN 4.1 mmol/l

Creatinine 68 micromoles/l ($\mu\text{mol/l}$)

Na⁺ 136 mmol/l

K⁺ 4.1 mmol/l

Pelvic ultrasound: showed an empty endometrial cavity; with fluid collection in the pouch of Douglas. An impression of pelvic abscess was made.

The culture results were not obtained.

She was transfused with 2 units of blood. Informed consent was obtained and patient prepared for laparotomy.

OPERATION

She was pre medicated with 1.m. atropine 0.6 mg half hour before theatre. In theatre, she was put under general anaesthesia and vulvo-vaginal toilet done. The bladder was catheterised and clear urine obtained. Examination under anaesthesia revealed a bulky uterus and full pouch of Douglas. There was bilateral cervical tenderness. The abdomen was opened via a sub umbilical midline incision.

On opening the abdomen, some adhesions between the intestinal loops, the tubes, ovaries and uterus were found. About 3l of foul smelling pus was found in the pelvic and peritoneal cavity; the uterus was slightly bulky, and the tubes were grossly inflamed. The pus was drained and a sample taken for culture microscopy and

sensitivity. The abdomen was cleaned with Rifocin and mass closure done. A corrugated drain was left in situ. Anaesthesia was successfully reversed.

POST OPERATIVE PERIOD

The patient was observed quarter hourly till fully awake, when she was transferred to the ward where observations were taken four hourly till discharge. She was put on Intravenous Cefuroxime (zinazef) 750mg 8 hourly and Metrinidazole 500mg 8 hourly. She was also transfused one more unit of blood. Then was removed after 24 hours when it was not draining any more. The culture did not grow any organisms. Post operatively, she did well and was discharged home on the 10th postoperative day in good condition. She was advised to come to GOPC in 3 weeks for review. At review, she was in good general condition, the wound well healed and no complaints.

DISCUSSION

The patient presented was an 18-year-old para 1 + 0, who had a pelvic abscess following a normal delivery at home. Laparotomy and drainage was done with good results.

Pelvic abscess is said to exist when pus collects in the pelvic cavity. It is commonly found in association with Pelvic inflammatory disease (PID), which describes infection of the uterus, fallopian tubes and occasionally the peritoneal cavity (1,2). It may occur as a sequel to post abortion and puerperal sepsis (1, 2). In the case discussed, it followed normal delivery at home. Pelvic abscess complicates about 2% of all induced abortion in KNH (3).

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The common symptoms of pelvic abscess include: abdominal pain, fever, nausea, and vomiting, rigors, chills and back pain. Pelvic abscess should be suspected in patients who continue to run high fever, after abortion or delivery or after treatment for acute pelvic inflammatory disease. Examination may reveal tachycardia, abdominal tenderness though adequate abdominal and pelvic examination is often impossible because of tenderness. Culdocentesis and paracentesis may reveal, but may rupture

the abscess and must be performed with extreme caution, if at all. Investigations include urinalysis, full hemogram and ultrasound (1, 2, and 3).

Abscess formation is often associated with enteric and anaerobic bacteria especially the bacteroides (3). The choice of antimicrobial agents should be broad to cover both aerobes and anaerobes, while awaiting culture results. The patient presented developed a pelvic abscess in puerperium following a home delivery, and was therefore likely to have had a mixed infection. Supportive care, mainly intravenous fluids, pain relief as well as blood transfusion when needed are essential. Laparotomy to drain the abscess is also needed.

A lower midline incision is used to allow exploration of all abdominal and pelvic structures for presence of small pockets of pus. Thorough irrigation of the peritoneal cavity with Rifocin in saline should be done and a drain left in situ (2). This was the treatment received by our patient. Another option is colpotomy and drainage (4, 5). Complications of pelvic abscess include substantial morbidity, infertility, ectopic pregnancy, bowel obstruction, peritonitis and endotoxic shock. With early treatment, the prognosis for the woman with a well-localized abscess is good. In case of a ruptured abscess, the prognosis for fertility is very poor (6). The outlook is worse after surgery than with conservative management (2).

Prevention of pelvic abscess formation includes health education, prevention of sexually transmitted diseases and prompt diagnosis and proper treatment of sexually transmitted disease, pelvic inflammatory disease, and puerperal and post-abortal sepsis. Provision of safe and affordable antenatal and delivery services would have prevented puerperal sepsis in our patient.

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GYNAECOLOGY CASE 14

SYMPTOMATIC UTERINE FIBROID: TOTAL ABDOMINAL HYSTERECTOMY

NAME: J.W. DoA: 9/8/04
AGE: 38 YEARS DoD: 17/8/04
IP.NO: 693693 PARITY: 3 + 0
WARD: 1B

PRESENTING COMPLAINTS

J.W was on follow in the GOPC with complaints of progressive lower abdominal swelling, pain and heavy menses for about 3 years.

HISTORY OF PRESENTING COMPLAINTS

She had been well till about 3 years prior. She presented with complaints of lower abdominal swelling and heavy menses at the gynaecologic casualty, where she was put on some medications and referred to GOPC. The pains were radiating to the back and worse during menses. The menses were heavy, lasting up to 10 days, from a previous 3-4 days. The last menstrual period was on 3/8/04. The menses came every 26-30 days.

OBSTETRIC AND GYNAECOLOGY HISTORY

J. W. was Para 3 + 0. Her last delivery was 5 years ago, to a life male infant who was alive and well. All deliveries were by spontaneous vertex delivery (SVD), and children were alive and well. She was blessed with 2 sons and a daughter. Her menarche was at 15 years. Prior to the current problems, 3 years earlier her periods were regular coming every 26-30 days and lasting 3-4 days. She had used oral contraceptive pills for the last 5 years. She had previously used Depo provera irregularly. She had no history of sexually transmitted disease. She had a normal pap smear 3 months prior to admission.

PAST MEDICAL HISTORY:

She was admitted with malaria in 1998, treated and discharged. She had no prior surgical history.

FAMILY AND SOCIAL HISTORY

She was a housewife who stayed with her husband in Murang'a. There was no family history of chronic illness. She had never drunk alcohol or smoked cigarettes.

SYSTEMIC INQUIRY.

This was non-revealing.

PHYSICAL EXAMINATION

She was a middle-aged lady in good general condition. She was not pale, was afebrile, and did not have Jaundice, edema or lymphadenopathy. The vital signs were BP 130/70 mmhg, pulse rate 82/min, respiratory rate 16 per minute and temperature of 36.8°C.

RESPIRATORY, CARDIOVASCULAR AND CENTRAL NERVOUS SYSTEMS WERE NORMAL.

ABDOMINAL EXAMINATION

There was a swelling in the lower abdomen, corresponding to uterine size of 20 weeks. The mass was firm, had a smooth surface, non-tender and mobile from side to side. There was no hepatosplenomegaly.

PELVIC EXAMINATION

The external genitalia were normal. The cervix was central, smooth and with a parous os. Both adnexae and pouch of Douglas were free. The uterus was bulky, corresponding to 20 weeks gestation. There was normal, white discharge on the examining finger.

DIAGNOSIS

A diagnosis of symptomatic uterine fibroids was made.

MANAGEMENT

She was planned for total abdominal hysterectomy.

INVESTIGATIONS

1. PAP smear 21/5/04: Normal cervical smear.
2. Pelvic ultrasound: showed multiple uterine fibroids with normal adnexae.
3. Hemogram: Hb 1.29 g/dl.
 WBC 4.9×10^6 /L
 Platelets 267×10^9 /L
4. Urea and electrolytes: Na⁺ 132 mmol/L: K⁺ 3.8 mmol/L
 BUN 4.9 mmol/L
 Creatinine 84 μ mol/L
5. Two units of blood were grouped and cross-matched to accompany the patient to theatre.

Informed consent was obtained. The patient was fasted overnight. A soap enema was given the previous night and the morning before theatre, pre medication 1.m atropine 0.6 mg was given ½ hour before theatre.

PROCEDURE

In theatre the patient was put under G.A and placed in lithotomy position. Vulvo-vaginal toilet was done, bladder catheterised and about 200mls of clear urine obtained. Pelvic examination confirmed earlier findings. The vagina was painted with methylene blue. The patient was then placed in supine position, and the anterior abdominal wall cleaned and draped.

The abdomen was opened via a sub umbilical incision. The uterus was found to have multiple fibroids about 22 weeks in size. The ovaries and tubes were grossly normal. A

total abdominal hysterectomy was performed as described earlier with ovarian preservation. The abdomen was cleaned and closed. The specimens were taken for histopathology. Anaesthesia was successfully reversed.

POSTOPERATIVE CARE:

She was observed in the recovery room half hourly until she was stable and fully awake. She was transferred to the ward where observations were continued four hourly till discharge. She was put on I.v Gentamycin 240 mg daily and Crystapen 2mu q6h, each for 48 hours, then oral Amoxycillin 500mg q8h for 5 days. She did well and was discharged home on the 5th postoperative day to be seen in GOPC in 2 weeks. At review after 2 weeks, the wound was well healed and patient had no complaints.

HISTOLOGY REPORT

The histology showed uterine myoma. The cervix and endometrium were also normal.

DISCUSSION

Uterine leiomyoma (fibroids or myomas), are benign clonal tumours composed mainly of smooth muscle cells of the human uterus but containing varying amounts of fibrous connective tissue (1). They are the single most common indication for hysterectomy. They are clinically apparent in about 25% of the women (2) and with newer imaging techniques; the true clinical prevalence may be higher. Careful pathological examination of surgical specimens suggests that the prevalence is as high as 77% (3). The cause of leiomyomas is unknown. Several studies have suggested that each Leiomyoma arises from a single neoplastic cell within the smooth muscle of the myometrium (4). There appears to be an increased familial incidence (5). Hormonal responsiveness and binding has been demonstrated in vitro. Steroid hormone concentrations has a role in formation and growth of these tumours, as do growth factors important in fibrotic processes of the endometrium are important to the pathogenesis of myoma related bleeding (6).

Most myoma's are asymptomatic, with less than one-half of uterine leiomyomas are estimated to produce symptoms (2). Symptoms attributable to myonma's can generally be classified in three distinct categories. Abnormal uterine bleeding; pelvic pressure and pain; and reproductive dysfunction (1, 2 and 3). The bleeding pattern most characteristic of myomas is menorrhagia or hypermenorrhoea prolonged or excessively heavy menstruation. The heavy bleeding may cause anaemia (2,3). Our patient presented with lower abdominal swelling, pain and heavy menses. Location rather than size seems to be more important than seize in determining the bleeding symptoms. Sub mucosal myomas, those in our partially intruding into the endometrial cavity, are most likely to cause monorrhagia. The causal relationship is not clear (2,3).

Pelvic pressure arises when uterine size is increased. The size of myomatous uterus is described in menstrual weeks, as is a pregnant uterus. Unlike a pregnant uterus, a myomatous uterus is irregularly shaped and the specific symptoms can arise from myomas in a particular location. Chronic pelvic pain may also be present. Pain may be characterised as dysmnorrhoea, dyspareunia or pelvic pressure. Acute pain may result from torsion of a pedunculated leiomyoma or infarction and degeneration.

Reproductive dysfunction is not inevitable with a myomatous uterus, but the risk of placental abruption is substantially increased if a myoma is under the placental site (7). Other pregnancy complications including pain and premature cause of infertility and have been reported as a sole cause in only a small percentage of infertile patients (8). Before the role of myomas is evaluated, a complete infertility assessment is recommended.

Myoma's respond to the gonadal steroids estrogen and progesterone, and their epidemiology parallels the ontogeny and life cycle changes in reproductive hormones. Myoma's have not been described in pre-pubertal girls. Although they have been reported in adolescents, most women are in their 3rd and 4th decades of life when the myomas become symptomatic. In most women the symptoms are relieved at menopause. However, increasingly there are reports of women who develop symptoms

or have continuing symptoms while taking hormone replacement therapy in their post reproductive years (9).

Race is an important epidemiological risk factor for myomas. Among women undergoing hysterectomy, black women are significantly more likely than white women to have myomas and tend to be younger at the time of diagnosis and hysterectomy (10). They also have more severe disease in terms of higher uterine size and greater likelihood of anaemia. Prospective studies show that black women have over a three fold greater frequency of myomas and a relative risk of two to three times that in white women (5).

Reproductive factors also affect the risk of myomas. Many studies have shown that being parous (having one or more pregnancies extending beyond 20 weeks) decreases the chance of myoma formation (11, 12). Although clinical teaching for many years was that oral contraceptives were contraindicated for women with myomas, these drugs actually protect against clinically evident fibroids (11, 13, and 14). Timing of use is important. Exposure to oral contraceptives between the ages of 13 and 16 years led to an increased relative risk of myomas, whereas, use in general showed protection in direct proportion to duration of use (13). During both pregnancy and use of oral contraceptives, concentrations of oestrogen and progesterone are high, yet both factors decrease the risk of fibroids. Thus influences other than concentrations of steroid hormones are important. The common factor may be the lack of menstrual cyclicity. One hypothesis is that myoma formation may be viewed as a response to injury, potentially from hypoxia in myometrial cells during menstruation (15).

Environmental factors also influence the risk of fibroid formation. Several studies have shown that smoking decreases the risk (11, 16). Substantial consumption of red meats was associated with an increased relative risk and consumption leads to changes in the incidence or symptom patterns.

The diagnosis of myomas is often suspected on the basis of palpation of an enlarged irregular uterine contour on pelvic examination. Ultrasonography is typically used to confirm the diagnosis and exclude the possibility of ovarian neoplasm. Magnetic resonance imaging gives better visualization of individual myomas but is expensive (4). Clinical examination and ultrasound were used in making the diagnosis in our patient. Uterine myomas as benign tumours can generally be managed expectantly unless they cause symptoms. Several factors determine treatment, including the size, and location of myomas presenting symptoms the age and reproductive desires of the patient and the skill of the surgeon. There has been little evidence-based assessment of myoma therapies.

Surgery has been the main mode of therapy for myomas. Hysterectomy eliminates both the symptoms and chance of recurrence. This is the best option for women who have no desire for children or have completed family size (18). This was the case in our patient. However, for women who desire to have children or retain the uterus or other reasons, other options include myomectomy (removal of the fibroids with uterine conservation), and myolysis. Myomectomy has distinct advantages. For women who have completed childbearing and for whom bleeding is the primary problem, endometrial ablation alone or in combination with hysteroscopic myomectomy will give relief. Uterine artery embolization is a novel technique for the treatment of myomas based on the hypothesis that control of arterial blood flow will control the symptoms (18).

Medical therapy: symptomatic treatment with haematinics, analgesics and use of progestagens is widely practised. Danazol is useful and acts by inducing amenorrhoea. GnRH agonists, the mainstay of medical therapy for myomas, work by first increasing the release of gonadotropins, which is followed shortly, by desensitisation and down regulation to hypogonadotrophic hypogonadal state, clinically resembling menopause. These agents produce a significant reduction in uterine size, generally 35% to 65% as well as amenorrhoea in most women. However, on discontinuation of medication, there is rapid resumption of menses and return to pre-treatment uterine volume. In

addition to severe hypoestrogenism that accompanies this therapy can cause significant symptoms and most importantly, bone loss that can lead to osteoporosis with long-term use. Thus, GnRH agonists are primarily used to temporise or allow a woman to prepare for surgery, and this use does have the proven benefit of a documented decrease in blood loss at surgery, and an increase in preoperative packed cell volume (18). Their use in Kenya is limited by their high cost.

After pituitary down regulation, steroid add-back may lead to increased compliance with long-term therapy. The major steroid studied for this is Tibohone, which has estrogenic, androgenic, and progestational activities. Tibohone was associated with higher rates of amenorrhoea in post menopausal women with myomas than was conventional hormone replacement therapy, when used in pre menopausal women there is preservation of bone density and lipid profiles without reversal of uterine shrinkage (18).

Steroid antagonists like mifepristone are another way to manipulate steroid hormone concentrations. It acts as a progesterone antagonist producing equivalent volume reduction and induction of amenorrhoea as GnRH antagonists while maintaining follicular concentrations of oestradiol.

There are attempts to modify surgical approaches to increase effectiveness and decrease invasiveness. Use of MRI- guided percutaneous laser ablation is one such model. High intensity ultrasound technology may also be used in this system (18).

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GYNAECOLOGY CASE 15

COMPETE FAMILY – VOLUNTARY SURGICAL CONTRACEPTION

NAME:	J.W.	PARITY	5 + 0
AGE:	38 YEARS	D.O.A	6/9/04
IP. NO.	0982375	D.O.D	11/9/04
WARD	ID		

PRESENTING REQUEST

The patient presented with a completed surgical sterilization consent form in labour ward for her 5th delivery. She progressed well and had a normal vaginal delivery. She requested that the procedure be done before she could go home. She was booked via ward ID for bilateral tubal ligation in main theatre.

PRESENTING HISTORY

She was now Para 5 + 0, all children being alive and well. Her first delivery was in 1981, and the last in 2004. Of the 5 children, 3 were girls and 2 were boys. She had used an intrauterine contraceptive device (IUCD), on which she conceived the last pregnancy. Before using an IUCD, she had used Depo-medroprogesterone acetate (DMPA) injection but developed excessive vaginal bleeding. She was also noted to have a slightly elevated blood pressure of 150/90 mmhg. Disturbed by the adverse effects of other methods of contraception, and wanting no more children she decided she wanted a bilateral tubal ligation. Her husband readily accepted her suggestion and after counseling both voluntarily chose permanent sterilization by bilateral tubal ligation.

OBSTETRICS AND GYNAECOLOGY HISTORY

Her menarche was at age of 15 years. The cycles were regular with an interval of 28 days, before she started using Depo-medroxy progesterone acetate (DMPA) injection, when they became irregular. Initially they used to last 3-4 days. She had no associated dysmenorrhoea. She had never had a pap smear done.

FAMILY AND SOCIAL HISTORY

She was in a monogamous marriage and lived with her husband in Ngong. Both were small-scale farmers. She neither smoked cigarettes nor drank alcohol. There was no history of chronic illness in the family.

PAST MEDICAL HISTORY

This was non-revealing.

SYSTEMIC INQUIRY

Non-revealing.

PHYSICAL EXAMINATION

She was a middle-aged lady in good general condition. She was not pale; had no jaundice, no edema, no cyanosis and no lymphadenopathy. Vital signs were a blood pressure of 110/70 mmhg, pulse rate of 80 beats per minute, respiratory rate of 18 per minute, and a temperature of 36.2°C.

SYSTEMIC EXAMINATION

The respiratory, cardiovascular and central nervous systems were essentially normal. The abdomen was scaphoid, with no organomegally or masses. Other systems were also normal.

PELVIC EXAMINATION

Speculum examination showed normal vaginal walls, a healthy cervix with a parous OS. On digital examination, the uterus was anteverted and felt about 18 weeks in size. It was mobile. The adnexae and pouch of Douglas were free. Cervical excitation test was negative. There was normal lochia on the examining finger.

DIAGNOSIS

Desired family size and desired surgical contraception with no contraindication.

INVESTIGATION

A full hemogram, urea, creatinine and electrolytes were requested. They were normal.

MANAGEMENT

She was scheduled for postpartum tubal sterilization using mini-laparotomy under local anaesthesia. She was asked not to have any solid meals for 12 hours prior to the operation time, and take nothing orally on the morning of the operation.

PROCEDURE

The pre-operative vital signs were found to be normal with a blood pressure of 110/70 mmhg; pulse of 78/ minute; temperature of 37.2°C and respiratory rate of 18/minute. She was then wheeled to theatre. The procedure was explained to her, her cooperation sought when necessary, especially when she needed to draw in her abdomen to facilitate fallopian tube retrieval.

She was put in lithotomy position, the abdomen cleaned and draped. Using the uterine elevator, the edge of the uterine fundus was noted on the abdomen, then an area 2cm below the fundus noted and infiltrated with 20mls of 2% lignocaine. A pinprick test revealed good anesthetic effect. A transverse minilap incision measuring 3cm was made on the site, using blunt dissection, the rectus sheath was identified and a small nick made, and extended to 4cm using scissors, again by blunt dissection, the peritoneum was identified, held by long-artery forceps and divided. With the help of the uterine elevator, the uterine fundus was easily identified, table put in head-down position, and the patient asked to draw in her abdomen.

The right tube was identified and picked using a hook, and held using a Babcock in the isthmal region, and the tube 'Walked' externally and the Fimbrial end identified. The tube was ligated using NO. 2 chronic catgut using the Pomeroy's technique. Hemostasis was easily achieved. The left tube was also retrieved also the same procedure repeated. With both tubes ligated, the bed was repositioned into its normal position. The abdomen was closed in layers. There was minimal oozing of blood. The

procedure lasted 30 minutes. The patient was then taken to the recovery area. The vital signs remained normal. She was subsequently transferred to ward ID. She was allowed home on Amoxicillin, Ibuprofen and Metronidazole for review in the family welfare clinic in 10 days. 10 days later she had no complaints and the wound was healed. A follow up appointment in 3 months was scheduled.

DISCUSSION

The patient presented was a 38 year old Para 5+0, whose last delivery was about 1 week prior, who had opted to have permanent contraception by bilateral tubal ligation during or soon after delivery. She was evaluated and found to be suitable, and bilateral tubal ligation using the pomeroys technique performed via mini-laparotomy under local anaesthesia. Her recovery was excellent.

Bilateral tubal ligation (BTL) is surgical permanent method of contraception also known as tubal sterilization. Tubal sterilization was first performed in 1823 to prevent pregnancy in women would need repeat cesarean section during child birth (1). Since then, many modifications have taken place and it is offered to women who have desired family size and want a permanent method of contraception (2). Other indications include women in whom a pregnancy could represent a significant clinical and medical risk, such as patients with Diabetes and severe vascular complications or severe cardiac disease. In Kenya, it is not clear when tubal sterilization was first used. It has however gained popularity alongside other modern methods of contraception.

The contraception prevalence rate for Kenya as at 2003 was 38.3%, with most current users of contraceptives using a modern method; about 30.5%, while 7.8% use traditional methods (considered less effective for prevention of unwanted pregnancy). Injectables and pills are the most commonly used contraceptive methods; used by 13.8% and 7.2% of married women respectively. A striking feature of the 2003 survey was the fact that no significant change in the contraceptive method mix used by the respondents had occurred. However, there was a slight decline in the use of female sterilization (4.3% vs. 6%) compared to 1998. Condom use remained low at 1-2%, with

men three times (17%), more likely than women (5%) to use condoms during a sexual encounter with any partner. Contraceptive use, which had shown a steady increase from the 1980's, slowed down between 1993 and 1998, and in the 2003 survey, the rate seemed to have reached a plateau. No wonder, the total fertility rate which declined from 8.1 in the mid 1970's to 4.7 children per woman in 1998, seems to have plateaued, if not risen, with a figure of 5.0 recorded in 2003 (3).

Tubal sterilization is indicated for women who want a permanent method of contraception and are free of any gynaecologic pathology that would otherwise dictate an alternative procedure. The patient presented had her desired family size and did not have any gynecologic pathology in the immediate post-partum period (within 48 hours of vaginal delivery). In comparison to interval sterilization, BTL following delivery in the early puerperium is convenient, simple, and cost effective. BTL may be done after closure of the uterine incision during cesarean section or following completion of vaginal delivery within 48-72 hours.

As alluded to earlier, postpartum BTL is technically simple because the uterine fundus is at the level of the umbilicus, making the fallopian tubes readily accessible through a small periumbilical abdominal incision (4, 5). If the procedure is delayed for several days all if the patient has a significantly involuted uterus (as might occur after delivery of a preterm infant), then delaying to an interval procedure usually is prudent because of the increased risk of sepsis (4).

Surgical approaches to BTL include laparoscopy; minilaparoscopy; laparotomy (concomitant with cesarean section), vaginal as well as mini-laparotomy. In KNH, Minilaparotomy and at cesarean section are the commonest approaches but laparoscopy is also done. Local anaesthesia is generally used during minilaparotomy. Informed consent and preoperative counseling is vital to inform the couple that the procedure is permanent and often irreversible. The chances of failure are small. Screening for risk indicators for regret, including young age, low parity, single parent status, or marital instability should be undertaken. Counseling on the need to use barrier

methods of contraception for protection against sexually transmitted infections including HIV need be emphasized.

Many surgical techniques for accomplishing tubal ligation have been described. Pomeroy technique is the simplest and most commonly performed method of tubal sterilization. The midportion of the oviduct is grasped with a Babcock clamp, creating a loop, which is tied with 2-0 or 0 plain catgut sutures, and each limb of the tubal knuckle, is cut separately. Specimens are submitted for histology. The endosalpinx at the cut ends may be cauterized (optional). The ligation sutures are held while the tube is cut to prevent retraction of the cut tubal stumps into the peritoneal cavity before they can be adequately examined for hemostasis (4, 5, 6). Failure rates are reported to be 1 in 300-500 patients (7). In the parkland technique, there is a mid segmental resection similar to the pomeroy technique, the difference being that each leg of the loop is tied separately. Others techniques include the Uchida, Irving, Electrocoagulation, Bipolar and Unipolar current. Mechanical techniques of tubal sterilization include: Falope (Yoon) ring, the Hulka – Clemens clip and Filshie clip techniques (8).

Follow up care both in the immediate postoperative and long term is important. A follow up visit, 1-2 weeks after operation is ideal. At KNH, it is usually 10 days. The patient should be informed of the danger signs, for which medical attention should be sought. Fever, increasing or persistent abdominal pain, bleeding or purulent discharge from the wound are such danger signs.

Some complications may arise during tubal sterilization, such as mortality. The risk of death from tubal sterilization is 1-2 cases per 100, 000 procedures; most of these are complications of general anaesthesia. The most common cause of death during laparoscopic BTL appears to be hypoventilation related to anaesthesia. Sepsis as a cause of death from laparoscopic sterilization is directly related to bowel perforations or electrical bowel burns. Unintended laparotomy occurs in 1-2% of laparoscopic procedures, usually attributable to technical inability to complete the procedure rather than to complications of the procedure (4).

BTL failure (pregnancy or ectopic pregnancy) can occur. Although sterilization is highly effective and considered the definitive form of pregnancy prevention, it has a failure rate during the first year of 0.1 – 0.8%. At least one third of these are ectopic pregnancies. Sterilization failures can be grouped in to 4 categories: Luteal phase pregnancy defined as a pregnancy in which conception occurs before the BTL, but pregnancy is diagnosed after an interval tubal sterilization. Misidentification of the oviduct because of poor visualization from inadequate exposure, adhesions, adnexal pathology, or poor lighting may result in mistakenly ligating the round ligament, ovarian ligament, infundibular ligament or dilated broad ligament blood vessels instead of the oviduct. Incomplete occlusion of the oviduct occurs because of poorly placed mechanical clips or the use of mechanical devices on edematous or dilated tubes. Incomplete tubal occlusion with electrocoagulation generally is associated with too brief an application of current instead of unmodulated and cutting current. In KNH, patients are given prophylactic antibiotics and analgesics for given prophylactic antibiotics and analgesics for pain relief after the procedure.

Post-sterilization regret is a complex condition often caused by unpredictable life events. Risk factors for regret that may be useful in post-sterilization counseling include young age, low parity, and single parent status or being in an unstable relationship. As many as 6% of women who are sterilized report regret, or request information about tubal reversal within 5 years of the procedure. Follow up interviews 14 years post procedure demonstrated that regrets were expressed by 20.3% of women aged less than 30 years at the time of BTL and by 5.9% of women older than 30 years at the time of the procedure (4, 6, 9, 10, 11). The proportion of women who actually undergo microsurgical tubal re-anastomosis is only 0.2% in the first 5 years after BTL.

The most important factor in determining the success rate of reversal by tubal anastomosis is the length of normal tube remaining after sterilization. Isthmic-to-isthmic anastomoses are most successful.

Among the non-contraceptive benefits of surgical sterilization are a decreased risk of ovarian cancer and incidence of pelvic inflammatory disease. Both of these effects are

thought to result from reduced spread of organisms and / or potential environmental carcinogens to the ovaries and pelvic (12). BTL has few if any, menstrual abnormalities within several years after sterilization. It may lead to reduced number of days of bleeding, less overall bleeding, and menstrual pain increased cycle irregularly has also been documented (5 13).

At KNH, and probably among a wide range of women, the major reason given by the women to decline BTL even when it is indicated on medical grounds, is the myth that 'mwili inakua baridi', one becomes cold in bed, meaning they either do not enjoy sex or never reach orgasm.

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