

# **C J Mieny, U Mennen: Principles of Surgical Patient Care - Volume II**

## **Chapter 14: Gynaecological Emergencies**

### **Chapter 14.1: Vaginal Bleeding**

**T Maseela**

#### **Introduction**

From the average age of about 12 years to more or less 52 years - menarche to menopause - cyclic changes in the endometrium take place under the influence of ovarian hormones. This ends up with the shedding of the thickened layer - the functionalis - with the associated loss of blood. This phenomenon is termed **menstruation**. The bleeding lasts 3-5 days, with an average blood loss of 80 mL.

#### **Definition**

Menstruation is a hyperfibrinolytic uterine bleeding that accompanies the shedding of the endometrium at the end of a menstrual cycle.

Any changes in this pattern are referred to as:

- Abnormal menstruation
  - Altered menstruation interval
  - Altered bleeding intensity.
- Vaginal bleeding

This condition refers to any non-physiological haemorrhage of exogenous or endogenous causes, with blood flow through the vaginal orifice.

#### **Anatomy and Physiology**

The vulva, with its labia majora and minora, encases a vestibulum, which has anatomically the hymenal seam as its boundary to the interior.

Behind the hymen the introitus opens in an S-shaped canal, the vagina, that extends from the hymenal seam to the fornices. It has a mean length of 10 cm and has uneven walls, due to the circular ridges called ruggae, which allow for elasticity.

At the distal end projects the portio vaginalis uteri, which is the vaginal tip of the uterus. The uterus has two anatomical and physiological entities:

- the cervix
- the corpus.

The lower part of the cervix, portio, is covered by squamous epithelium and constitutes the ectocervix. The cervical canal on the other hand, is covered by columnar

epithelium, lining the mucus producing glands, and extends from the squamo-columnar junction to the internal os.

The portio exhibits various forms in the sexual development of a woman. In a prepuberty the squamo-columnar junction extends outside the os, as a pseudoerosion. In the nulliparous woman, it is commonly retracted behind the cervical os, exhibiting a dent in the middle of the portio.

This margin is pushed externally in most parous women and can be visualised, on speculum examination, as an ectopy (Ektropion), which is an extension of the glandular tissue onto the surface of the portio.

This surface is prone to the following abnormal developments on account of changed milieu:

- Erythroplakia
- Leukoplakia
- Polyposis cervicis
- Metaplasia
- Ulceration
- Dysplasia
- Neoplasia.

The walls of the vagina and the ectocervix are covered with layers of stratified squamous epithelium, the cells of which are exfoliated from time to time and, under enzymatic action and bacterial cytolysis, converted into a physiological transudate with an acidity of pH 4.

The growth of the epithelial layers is dependent on the levels of oestrogen and gestagen. The acidic vaginal milieu produced by the desquamated cells under cytolysis, forms the defence mechanism against any ascending infection of the genital tract.

The corpus uteri, a pear-shaped muscular body of the uterus, begins at the isthmus and juts into the abdominal cavity as the fundus, supported only bilaterally by ligaments. Normally it is freely mobile ventrally and dorsally, but physiologically it lies in anteflexio. It carries on its two horns the adnexa, consisting of the ovary and the oviduct (tube). Inside the muscular wall, the myometrium, the fundus has a cavity lined by the endometrium. This lining is shed at the end of every menstrual cycle and regenerates during the inter-menstruum.

In early childhood, the girl is under the influence of maternal hormones and may encounter unphysiological changes, which soon vanish. Except in precocious development, the HPO axis (hypothalamus - pituitary - ovary) is first established at puberty, with the resulting activation of gonadotropins and the ovarian response. Follicular development and oestrogen secretion commence.

Under hormonal influence the endometrium proliferates and the functionalis undergoes the secretion phase. An inhibitory feedback of ovarian hormones to the

hypothalamus causes a drop in the FSH, which results in follicle regression. The oestrogen level falls and the endometrium is shed, causing the phenomenon of *menstruation*.

### **Proliferation**

Ten days after menstruation started, a new functionalis has built up on the retained basalis. New blood vessels form and begin to spiral into the endometrium.

### **Secretion**

With the production of progesterone in the corpus luteum of the ovary, tissue changes take place in the endometrium. The glands increase in volume and the spirals of the vessels advance. The endometrial glands secrete glycogen, lipids and enzymes. The stroma loosens up and vascularisation is intensified. At this stage, the endometrium is prepared for the nidation of a fertilised ovum.

In the case of conception, implantation of the blastocyst is facilitated and decidualisation of the endometrium follows.

When no fertilisation has taken place, the developed corpus luteum in the ovary atrophies and drops its progesterone production. The hormone withdrawal causes a tissue regression in the endometrium with a disturbance of the microcirculation. An enzymatic action breaks the functionalis and menstruation begins.

FSH --> Oestrogen production  
LH --> Progesterone production

Hormone withdrawal

- Histiolysis
- Release of active peptides and amines
- Change of clotting mechanism
- Fibrinolysis

Menstruation = 3 to 4 days

Regeneration.

The most important aspect of menstruation is that menstrual blood does not clot, and the appearance of coagula in vaginal (uterine) bleeding should be considered pathological.

## **Classification**

When establishing a history it is important to distinguish:

### **Change in Rhythm**

- Polymenorrhoea (frequent)
- Oligomenorrhoea (seldom)
- Irregular menstruation.

### **Change in Intensity**

- Hypermenorrhoea
- Hypomenorrhoea
- Spotting.

### **Change in Duration**

- Menorrhagia (lengthy)
- Brachymenorrhoea (shortened).

### **Dysfunctional Bleeding**

- Anovulatory
- Ovulatory.

### **Organic Bleeding**

- Metrorrhagia
- Haemorrhagia.

### **Aetiology and Pathophysiology**

Any loss of blood through the vagina outside the regular menstrual cycle is reason for concern, and needs clarification. Organic causes of vaginal bleeding are:

### **Complications of Pregnancy**

- Abortion (imminent, inevitable, incomplete)
- Extrauterine gestation
- Placenta (praevia, abruption, retention)
- Gestational trophoblastic disease (GTD)
- Intravaginal tear

### **Endocrinopathy**

- Ovarian disease
- Diabetes mellitus
- Hormone treatment (progesterone, oestrogen)
- Abnormal prostacyclin production

## **Hyperaemia in the genital region**

- Adnexitis
- Endometritis
- Endometriosis

## **Uterine Disorders**

- Retroflexio uteri
- Maldevelopment
- Leiomyomata
- Endometriosis interna (adenomyosis)
- Foreign body, ie, IUCD (increased prostaglandin production)
- Cervical laceration

## **Medical Diseases**

- Cardiovascular
  - congestive cardiopathy
  - hypertension
- Chronic hepatopathia
- Haemorrhagic diathesis
  - thrombocytopaenia
  - thrombocytopathy
  - deficiency of factors V, VII, X
  - von Willebrand syndrome
- Hypothyroidism

## **Neoplasia**

- Polyposis, adenoma
- Carcinoma, sarcoma

### Table 14.1.1. Diagnostic Methods

Vagina: Inspection, Smear, Biopsy.

Ectocervix: Inspection, Cytology, Colposcopy, Cone biopsy.

Endocervix: Cytology, Curettage, Cone biopsy.

Cavum uteri: Cytology, Curettage, Hysteroscopy.

Tube/Ovaries: Palpation, Laparoscopy, Hormone - cytology.

## Bleeding of Organic Origin

1. Granulation - post-hysterectomy.
2. Vagina ca.
3. Colpitis (acute vaginitis).
4. Laceration (foreign body, coitus).
5. Ulceration.
6. Cervical polyp.
7. Ectopsy.
8. Cervix-carcinoma (squamous).
9. Adenoca.
10. Endometritis.
11. Endometrial ca.
12. Polyposis uteri.
13. Submucous myoma.
14. Tubal ca.
15. Ovarian tumour.
16. Adnexitis.
17. Extra-uterine pregnancy.

### **Diagnosis**

History: Bleeding and associated diseases.

Examination: General and pelvic.

Laboratory tests: FBC, LFT, BP, clotting profile, endocrinology.

Special investigations: Fractional curettage, hysteroscopy.

### **Special Investigations and Treatment**

#### **Dilatation and Curettage**

The scraping of the uterus is the commonest gynaecological intervention anywhere (Ga-Ranmkuwa = 50%). It is performed both for diagnostic and therapeutic purposes, the major indications being:

- Abnormal uterine bleeding
- Menstruation disturbance
- Abortion
- Polyps
- Intrauterine pathology and removal of IUCD
- Cervical and endometrial neoplasia.

The procedure is conducted preferably under general anaesthesia. In some cases, however, it can be safely performed under local anaesthesia, ie, PCB or PDA (Paracervical block or peridural anaesthesia).

## Complications of D & C

- Even in the hands of an experienced surgeon, pathological conditions of the uterine cavity can be missed, ie, polyps, submucous myoma and small carcinoma.

- In the case of endometrial carcinoma, curettage can enhance the risk of dissemination by opening the blood and lymphatic vessels.

- The most important complications are:

- Laceration of the cervix
- Perforation of the uterus
- Aschermann's syndrome

In the place of a metal curette, a plastic suction curette can be utilized with greater safety, is recommended for vacuum-aspiration in gestational trophoblastic disease (molar pregnancy).

It must be emphasised that curettage should not be over enthusiastic and so vigorous as to scrape the entire endometrium, including the basalis.

Abortions and puerperal uteri to be scaped with **blunt curettes**.

## Hysteroscopy

Attempts to diagnose pathological changes in the uterine cavity endoscopically are not new, but modern developments have enabled accurate diagnosis by:

- CO<sub>2</sub>-gas insufflation
- Fluid insufflation
- Contact hysteroscopy.

Direct biopsies can now be made with ease.

## Hysterectomy

In an increasing number of cases an indication for hysterectomy is made for therapy-resistant uterine bleeding, especially if permanent sterilisation is desired, or in the case of early malignant lesions.

## Medical Therapy

### Hormones

- Oral progesterones are used extensively
- 10 mg norethisterone, 2 hourly x 4 doses then 5 mg, 8 hourly for 14 days
- Medrogeston 5 mg 12 hourly x 5 days

High doses of oestrogens with progesterone

- Estradiol 17 n valerianat
- Ethinyl - estradiol
- Mestranol

### **Uterotonics**

- Ergometrine
- Oxytocin

### **Antiprostaglandins**

- Mefenic acid - 300 mg 8 hourly x 5 days indicated often in IUCD haemorrhage.

### **Antifibrinolytics**

- Tranexaemic acid 500 mg, 6 hourly x 3 days
- Trans-AMCHA

### **Clomiphene**

- From 5th to 9th day of menstrual cycle 50 mg - 100 mg per day for 3 cycles (cave polycystic ovaries).

### **Local Therapy**

#### **Wound Suture**

- Cervical and vaginal lacerations

#### **Coagulation**

- Electrocoagulation or metacresylsulphonic acid (benign cervical and vaginal lacerations).

#### **Vaginal Plug**

- 12-24 hours immersed in metacresylsulphonic acid (not post-partum or abortum).

### **Prognosis and Complications**

The complications of prolonged vaginal bleeding are:

- anaemia
- ensuing hypovolaemic hypotension
- shock syndrome.



These extreme conditions will necessitate resuscitation treatment with:

- fluid balance
- plasma and blood transfusion
- iron-substitution therapy.

Irrespective of the aetiology of the haemorrhage, a quick restitution of the haemodynamic balance will enable better diagnostic measures and optimal treatment. The prognosis is always fair to good.

### **Comment**

## **Vaginal Bleeding**

### **H S Cronje**

The current tendency is to speak of abnormal uterine bleeding instead of vaginal bleeding, except when abnormal bleeding from vaginal sources is included. Abnormal uterine bleeding can occur before the menarche, during the reproductive years and in the postmenopausal period. This chapter concentrates only on abnormal bleeding during the reproductive years.

Abnormal uterine bleeding during the reproductive years (not during pregnancy) is classified as:

- bleeding of organic origin
- dysfunctional uterine bleeding (DUB)
  - anovulatory
  - ovulatory.

Bleeding of organic origin is well-covered in this chapter. DUB means abnormal uterine bleeding in a woman who appears normal at clinical consultation. If she is anovulatory the defect is a lack in progesterone on a cyclic basis. Correction of this deficiency will result in a normal cyclic menstrual pattern. Ovulatory DUB is associated with an intact ovulatory mechanism. The abnormal bleeding may result from two causes: "hidden" organic disease (ie, endometrial polyp) and abnormal corpus luteum function. The corpus luteum has a remarkably consistent lifespan of 14 days. Under abnormal conditions its lifespan may be shortened (corpus luteum defect) or prolonged (Halban's disease).

The treatment of anovulatory DUB has two aims: to stop bleeding if it exists at consultation, and correction of the cyclicity of the menstrual pattern. Bleeding can be stopped in a number of ways:

- Norethisterone 5-10 mg 2-4 hourly per os until the bleeding stops.
- A progesterone-dominant combined contraceptive tablet, 1 tablet 3 times per day for 1 week.
- Unconjugated oestrogen 25 mg intravenously 6-8 hourly until the bleeding stops.
- A dilatation and curettage (D & C).

D & C is rarely necessary under the age of 40 years. After 40 years, it should form the basis of management in order to exclude an endometrial carcinoma.

Cyclicity can also be corrected in a number of ways. Norethisterone can be administered 5-10 mg 3 times a day for 7-10 days per os. It should be repeated for 2 additional cycles from day 14-24. In young females treatment may be stopped after 3 cycles of treatment since spontaneous ovulation may follow. Women in the premenopausal years will not become ovulatory spontaneously. When cyclic norethisterone therapy is stopped, persistent anovulation will lead to a repetition of anovulatory DUB. Therefore, medical therapy can either be maintained, or a hysterectomy can be performed as an elective procedure.

Instead of adding progesterone, the patient can be stimulated to produce her own progesterone with colomiphene citrate (50 mg per day for 5 days; ideally from day 5-9). Another method of treatment is the combined contraceptive tablet, 1 tablet per day for 21 days each 28 days.

Ovulatory DUB with a normal cycle length should be investigated by hysterectomy or D & C. Patients with an abnormal cycle length may be treated with progestogens similar to the treatment of anovulatory DUB, or antiprostaglandins may be used. If the patient wants to become pregnant or complains of reproductive failure, specialised treatment is necessary.

Abnormal uterine bleeding in the postmenopausal period should always be investigated with cervical cytology and a fractional D & C. The endocervical canal is curetted first, followed by a curettage of the fundal cavity. Hysteroscopy is a valuable additional investigative procedure since D & C is a blind procedure where pathology in the uterus can pass unnoticed.

Premenarcheal abnormal bleeding should be investigated under anaesthesia. A vaginal examination is best performed with an otoscope in girls under 2 years of age. In older girls, a nose speculum is adequate. Following the speculum examination, the vagina should be rinsed with a sterile saline solution to remove foreign material.

Finally, abnormal uterine bleeding may be caused by complications of pregnancy. This is a subject on its own and will not be discussed here.

## **Chapter 14.2: Vaginal Discharge**

### **T Maseela**

The commonest symptom with which gynaecological cases seek medical treatment is vaginal discharge. The incidence among the sexually active women can be rated at 45%. It is, however, often left undiagnosed, either for lack of concern by the patient - due to misconception about its significance - or for lack of facilities for the doctor to reach a diagnosis.

Although the incidence of classical venereal diseases in the UK may have remained static, penicillin resistant new cases have been reported. Reports on incidences of STDs on

the African continent are too sporadic to be reliable, but in general there has been a recorded high rate as a whole. This has led to the misdirected broad-spectrum "shotgun approach" practised by many doctors in the treatment of vaginal discharge.

In the first instance it would be best that:

- Investigation be made for specific causes.
- Malignancy be excluded.

### **Definition**

Vaginal discharge (*fluor vaginalis*) refers to an increased transudation of the squamous epithelium in the vagina, or increased secretion of the glandular tissues in the genital tract, with a descending flow through the vaginal orifice.

### **Classification**

For practical purposes and better clinical management it is best to distinguish according to:

Origin: vestibular, vaginal, cervical, corporal, tubal.

Consistency: thick, mucoid, viscous, fluid, foamy, cheesy.

Colour: colourless, straw coloured, milky, purulent, haemorrhagic, brown.

### **Anatomy and Physiology**

Between the two lateral halves of the vulva lies the vestibulum, with the perineum forming its base and the clitoris as the apex. On both sides of the vestibulum open the Bartholin's glands caudally and the Skene's glands cranially.

Below the clitoris opens the urethral meatus, and in the middle of the vestibule lies the introitus vaginae with the hymen - in a virgo - or the hymenal seam in a sexually active or parous woman, as the border.

The keratinising squamous epithelium of the vulva transforms into the non-keratinising, stratified squamous epithelium of the vagina immediately behind the hymenal seam. The vaginal canal extends to the fornices, and the posterior cove builds a reservoir for seminal fluid and vaginal discharge.

The vaginal transudate, with its constant acidity of pH=4 is the physiological defence mechanism against ascending infections of the lower genital tract (*S. Tab*), whereas the vulva and perineum form the anatomical protection.

### **Aetiology**

Sexually transmitted diseases (STDs) have always been incriminated whenever a patient presented with vaginal discharge. There is no doubt that the prevalence of ascending pelvic infections can be attributed to the untreated infections of the vagina.

There is, however, no justification for the common administration of penicillin or broad-spectrum antibiotics as therapy for vaginal discharge.

An inflammation of the vagina, colpitis (vaginitis), is caused by an infection by any of the following:

- Trichomonas vaginalis
- Candida albicans
- Gardnerella vaginalis
- Viral diseases (HSV, HPV, CMV)
- PLT (psittacosis, lymphogranuloma, trachoma)
- Treponema, gonococci, chlamydia
- Bilharzia (schistosoma).

The causative factors for disturbing vaginal discharge are manifold, and should be listed with conditions that effect a disturbance of the equilibrium that maintain the acidic vaginal milieu. These causative factors can be:

#### **Endogenous**

- Oestrogen deficiency and pregnancy
- Progesterone deficiency
- Diabetes mellitus
- Other diseases of internal organs.

#### **Iatrogenic**

- Antibiotic therapy
- Mechanical prolapse therapy (pessaries)
- Radiation therapy.

#### **Exogenous**

- Mechanical alteration (vita sexualis, tampons)
- Chemical alteration (vaginal douche)
- Contraceptive
- Massive infection.

#### **Descension (Organic)**

- Menstrual blood
- Cervical mucous (alkalising effect)
- Polyposis cervicis
- Cervical glandular ectopy and cervicitis
- Diseases of internal genitalia (including malignancy).

## **Pathophysiology**

Any disturbance in the equilibrium formed by the transudate of exfoliated cells and the bacterial cytolysis through lactobacilli causes a change of milieu. Resistance to infection is reduced.

This is often the case when a predisposing endocrinopathy or an anatomical abnormality adversely affects the proliferation of the squamous epithelium. The glycogen production is disrupted and cytolysis cannot be effected.

On the other hand, primary infection can promote a secondary milieu change. In general the pathophysiology can be considered thus:

### **Vestibular Discharge (Fluor Vestibularis)**

- Vulvo-vaginitis
- Bartholinitis
- Condylomata

### **Vaginal Discharge (Fluor Vaginalis)**

- Functional disorders
  - Defect perineum
  - Hormone deficiency (atrophy, hypoplasia)
  - Diabetes mellitus
  - Systemic antibiotic therapy
- Inflammatory changes
  - Colpitis
  - Trichomoniasis
  - Candidiasis
  - Amin-colpitis, "Gardnerella vaginalis", STD, including viral infections
- Local agents
  - Vita sexualis (seminal fluid)
  - Mechanical and chemical alterations
- Vaginal neoplasms
  - Carcinoma
  - Condylomata.

### **Cervical Discharge**

- Ectopy (ectropion)
- Cervix - polyps
- Hypersecretion
  - Functional (psychovegetative)
  - Infection (Gonorrhoea, Chlamydia trachomatis)
- Cervix carcinoma

## **Corporal Discharge**

- Pyometra, ie, postirradiationem
- Degenerating leiomyoma
- Endometrial carcinoma
- Other uterine neoplasma

## **Tubal Discharge**

- Pyosalpinx
- Hydrops tubae profluens
- Tubal carcinoma.

## **Clinical**

### **Symptoms**

There is no uniformity in the symptomatology of vaginal discharge. Depending on the causative factors, the symptoms can be:

- Pruritus and pain
- Offensive smell
- Secondary vulvo-vaginitis
- Dysuria and pollakisuria (frequency).

### **Signs**

According to how acute or chronic the condition is, there will be an erythema, due to hyperaemia of the vagina, which in older women exhibits a punctation - Colpitis granularis - as a granular diathesis.

Very often a speculum examination reveals characteristic plaques and a diagnostic consistence and colour.

### **Diagnosis**

With good illumination of the vagina and the cervix, after inspection of the vestibulum, it is not difficult to diagnose colpitis or cervicitis, but causative factors can best be determined after:

- accurate history taking (anamnesis)
- clinical examination
- microscopic examination (wet smear, PAP smear)
- eventual virology.

## **Special Investigations**

### **Vaginal pH**

Specimen from vaginal pool taken with special care not to contaminate with alkaline cervical mucus. Acidometry can be done with pH indicator or by means of chemical titration.

### **Wet Smear**

Specimen from the posterior fornix is examined on a slide with a cover slip under light microscope or phase-contrast microscope. KOH solution can be added.

### **Gram Staining**

Cervical and urethral swabs are utilised for smears on clean, dry slides for gram staining. Methylene blue could suffice, but strains of gram positive Diplococci easily lead to false diagnosis.

### **Immunofluorescence Staining**

Specimens taken with sponge swab from the endocervix are treated with 2-SP medium containing fetal-calf serum and vancomycin, then frozen in liquid nitrogen. With the aid of McCoy cells a diagnosis of Chlamydia trachomatis can be made.

### **Papanicolau (PAP) Smear**

Specimen taken from either the vaginal pool, ectocervix or endocervix or lateral vaginal wall, preferably with cotton wool swabs, are fixed in fresh condition on a clean, dry slide and later stained with Papanicolau reagents for:

- cytological examination (dysplasia, malignancy)
- hormonal analysis (maturation)
  - Candida diagnosis: possible to sure
  - Trichomonas: identification possible
  - Gardnerella: suggestive (clue-cells)
  - Viral infections: suggestive (koilocytosis).

### **Further Investigations**

Culture:

- bacteria
- Candida
- Mycoplasma

#### Virology:

- HSV (Herpes simplex virus)
- HPV (Human papilloma virus)
- CMG (Cytomegalo virus)

#### Serology:

- Treponema

#### Colposcopy:

- epithelial lesions
- warts and papillomata
- malignancy

#### Currettage:

- endocervix
- uterine cavity.

### **Therapy**

- Reduction of local agents
- Oestrogen-substitution, ie, topical creams
- Resuscitation of vaginal milieu
- Specific therapy:
- Trichomonas, ie, Metronidazole +
- Candida:
  - Imidazole derivatives +
  - Nystatin +
  - Amphotericin B +
- Chlamydia:
  - Erythromycin
- HSV:
  - Acyclovir or other virustatics
- HPV:
  - Podophyllin or Interferon
- VD:
  - Penicillin or Cephalosporine.
- Partner-treatment
- Local therapy
  - Adstringent agents
  - Cryotherapy
  - Laser-vaporisation
  - 5-fluoro uracil cream
- Vaginal lavage - Povidone iodide 10%
- Curettage (histology obligatory).



## **Prognosis and Complications**

Among the most dreaded risks are:

- ascension (endometritis/salpingitis/pyometra/pyosalpinx)
- PID (pelvic inflammatory disease, sepsis)
- infertility (tubal occlusion, pelvic adhesions, chronic cervicitis, etc).

In the case of a pregnancy, there is always a high risk of maternal morbidity and pregnancy loss, due to inflammatory vaginal discharge. The morbidity and mortality of neonates is significantly high.

The emergence of the present-day STDs, including AIDS and cervical neoplasms, necessitates greater care in the management of patients presenting with vaginal discharge.

### **Comment**

#### **Vaginal Discharge**

**H S Cronje**

The complexity of the nature of vaginal discharge and diagnosis of vaginal infections were excellently outlined by the author. Facultative organisms can be recovered from the vaginas of virtually all women. Due to the wide variety of organisms found, it is virtually never useful to obtain a general vaginal culture in hopes of identifying the cause of vaginal discharge. For instance, group B streptococci, *Escherichia coli* and anaerobic organisms are isolated in 20-40% of normal women without symptoms of vaginal discharge.

When this complex balance of organisms becomes altered, a situation arises in which potential pathogenic organisms (which are usually part of the normal vaginal flora) are able to proliferate to a concentration sufficiently high enough to produce symptoms. The mechanisms causing an alteration of this complex micro-organism milieu have not been elucidated. Examples of organisms that overgrow to produce symptoms include *Candida albicans* and *Gardnerella vaginalis*. *Trichomonas vaginalis*, in contrast, is a sexually transmitted disease and not part of the normal vaginal flora.

Lactobacilli are present in more than 90% of women with normal vaginal discharge. They produce hydrogen peroxide which inhibits overgrowth of organisms, notably anaerobes that are observed in patients with nonspecific vaginitis (NSV).

Most cases of infectious vaginitis can be placed in three categories: NSV (50% of cases), candidiasis (20%), and trichomoniasis (20-30%). History taking, physical examination, pH determination, the amine test and two wet smears (one in saline and the other in KOH) will allow the physician to properly diagnose vaginitis with at least 80% accuracy. The pH is raised in NSV and trichomoniasis. When 10% KOH is placed on a glass slide and mixed with vaginal discharge, the presence of a fishy odour indicates a positive amine test. The amine odour is caused by diamines that become volatilized by alkalization. Diamines result from anaerobic metabolism, usually associated with

*Gardnerella vaginalis* and *Trichomonas vaginalis* infections. *Trichomonas vaginalis* is easily recognised in a saline wet smear. It can, however, be immobilised by white blood cells, low temperature and drying up. Under these conditions diagnosis may be difficult. *Gardnerella vaginalis* is usually associated with NSV and is recognised by the typical "clue cell" in a saline wet smear. *Candida albicans* is best diagnosed in a KOH wet smear.

As outlined by the author of this chapter, treatment is specific for the causative organism. In a case of sexually transmitted disease, the male consort should also be treated. Controversy exists whether the male partner should be treated in cases with candidiasis and NSV. Personally, I treat them only in cases of recurrent or overwhelming disease.

Atrophic "vaginitis" is not really an infection, but may be associated with symptoms similar to a pathologic vaginitis. The cause is an oestrogen deficiency as seen in lactating women and in the postmenopausal state. A saline wet smear will show a predominance of parabasal epithelial cells and the treatment is a vaginal oestrogen cream, 2-3 times per week.

Finally, is vaginitis dangerous to the patient? *Trichomonas vaginalis* vaginitis may lead to an overgrowth of anaerobic organisms with lower abdominal pain, probably due to a salpingitis. In patients with candidiasis, especially in repeated form, diabetes mellitus should be ruled out. NSV is probably not dangerous, only troublesome due to a foul odour after coitus.

### **Chapter 14.3: Ectopic Pregnancy**

**T Maseela**

#### **Introduction**

A significant contributor to obstetrical and gynaecological emergencies still remains the extrauterine gestation, commonly referred to as ectopic pregnancy.

According to medical history the condition was first described in 863 AD by Albucasis. Statistics from various centres in Southern Africa indicate incidence of 2-3%. It is however not possible to estimate the actuarial incidence, since the total number of pregnancies in the different population groups is indeterminable.

Although world literature on all manifestations of extrauterine gestation abounds, the issue of incidence will still remain debatable as long as comparable denominators have not been fixed. Often, reported pregnancies do not include abortions and still births. Nonetheless, the overall view is that the incidence is on the rise.

The contribution of "ectopic pregnancy" to maternal deaths can only be based on hospital records, which are unfortunately often lacking in conclusive detail. In the United States, it has become a major cause of maternal mortality.

## **Definition**

The nidation of a fertilised zygote on any tissue outside the endometrial cavity results in an extrauterine pregnancy - *Graviditas extrauterina*. The common expression ectopic derives from the Greek term *ektoios*, meaning "out of place". Therefore, it embrace various manifestations of gestation outside the uterine cavity.

## **Anatomy and Physiology**

After ascension of motile spermatozoa through the cervical mucus, the uterine cavity and the isthmus tubae, conjugation of one of them with the ovum occurs. The fertilised ovum (zygote) is then transported within the salpinx by a complex action involving the ciliae, the endosalpingeal secretion and the tubal motility; which action is under hormonal and humoral influences. On the 6th day "post ovulationem" the original zygote has changed beyond the morula into a blastocyst and reaches the place of nidation in the endometrium. During implantation the trophoblast buries itself in the transformed endometrium, the decidua.

## **Aetiology**

The causes of extrauterine pregnancy can be divided into two categories:

- Disturbances in the transport mechanism.
- Factors inherent in the embryo.

## **Transport Mechanism (Maternal)**

### **Infection**

Previous salpingitis appears to be the principal factor, as many authors agree. This condition impairs ciliary activity and tubal motility. In this context, one recalls the sequelae of unrecognised tubal tuberculosis and the various STDs.

## **Intrauterine Contraceptive Devices**

Apart from the fact that the risk of pelvic infection is increased fivefold in IUCD users, the effect of progesterone in some makes of IUCDs has been the cause for much concern.

### **Tubal Sterilization**

There is a significant evidence of ectopic pregnancies following all modes of tubal sterilisation, ie, ligation, electrocoagulation and thermocoagulation.

## **Congenital Abnormalities**

This may include diverticula inside the lumen of the tube and salpingitis isthmica nodosa, and these can predispose to ectopic pregnancy.

## Transmigration

Transmigration of the fertilised ovum has been implicated as another factor in extrauterine gestation. There can be *internal* transmigration, whereby the morula migrates across the uterine cavity from one cornu to the opposite tube, as can be demonstrated by a contralateral corpus luteum. With *external* transmigration, the conjugation will have occurred in the pouch of Douglas and the morula or blastocyst then transported and implanted in the contralateral tube.

## IVF - ET

It is worthwhile to mention the incidence of ectopic gestation reported in the in vitro fertilisation and embryo transfer programs, albeit the etiology lies in the disturbed transport mechanism of the tubes in women considered for these programs.

### Factors Inherent in the Embryo

- Embryologic malformations
- Abnormal chromosomal patterns should interest the investigator, as this type of patient may subsequently have normal intrauterine gestation.

### Classification

Extrauterine gestation can be classified according to localisation.

Table 14.3.1.

	SA estimates	Breen
Tubal pregnancy		
Ampullary	65.5%	
Isthmic	25.0%	97.7%
Intestinal	4.2%	
Entire tube	2.3%	
Abdominal pregnancy		
Primary		
Secondary	1.6%	1.3%
Broad ligament	0.5%	
Ovarian pregnancy	0.4%	0.15%
Varia		
Uterine horn	0.3%	
Rudimentary horn	0.05%	0.75%
Cervical	0.05%	
Rareties	0.10%	0.1%
Concomitant ectopic + IU gestation		
Bilateral tube pregnancy		
Tubal multiple pregnancy		
Pregnancy without uterus (post hysterectomy)		

Table 14.3.2.

### Causative Factors in EUG

#### Maternal

Chronic salpingitis  
Tubal diverticula  
Salpingitis isthmica nodosa  
Tubal sterilisation  
Intrauterine contraceptive devices  
Transmigration of the ovum  
Menstrual regurgitation

#### Embryonic

Malformed embryo  
Chromosomal anomaly

### Pathophysiology

Early hurdles to life.

Ovulation: Early fertilisation is possible, before the ovum enters the tube.

Fimbria: Disturbance of admission due to adhesions is possible.

Conjugation: Early implantation in the ampulla is possible, with the likelihood of ectopic regurgitation or reverse transportation by reflex-menstruation.

Transport: Disturbance of motility and inner wall texture or secretion can lead to nidation in the tube.

Passage: The isthmus can have a narrow canal that does not permit the morula through.

Endometrium: The blastocyst can now under the blastokinin effect prepare its bed for nidation. Delay in the transformation leads to early abortion.

### Pathology

In case the source of normal transportation is impaired, the zygote can implant outside the uterine cavity. There follows rapid invasion of the substrate by the trophoblast with penetration into the connective tissue between endosalpinx and serosa. Should the serosa be stretched beyond its limit by the ensuing hematoma, tubal rupture occurs.

The endometrium concurrently undergoes a decidual transformation. The uterus gets softened and enlarged, but size does not correspond to duration of amenorrhoea. In case the conceptus dies, regressive changes occur leading to vaginal bleeding. Curettage

then reveal pregnancy, associated changes without villi, and very often Arias-Stella reaction.

### **Ampullary Implantation**

This is the commonest site of implantation in tubal pregnancy. The course of development can be:

- tubal abortion
- tubal rupture
- tubal mole
- continuation of pregnancy.

### **Tubal Abortion**

The conceptus is partly or wholly expelled through the fimbrial end into the abdominal cavity. The haemorrhage may cease, in complete abortion, or persist, if abortion is incomplete, gradually accumulating to form a pelvic haematocele. Pain and possible shock are common, but not severe.

### **Tubal Rupture**

This often occurs between the 6th and 12th week. The accompanying haemorrhage flows into the peritoneal cavity (intraperitoneal) or between the layers of the broad ligament (extraperitoneal). Rupture may be sudden, with large quantities of blood flowing out, causing profound collapse in a short time. Gradual perforation causes recurring attacks of severe abdominal pain and syncopes. This may be complicated or mask a tuboabdominal ectopic. Here pregnancy can advance towards term without rupture of the sac.

### **Tubal Mole**

The conceptus is surrounded by thick clotted blood and the tube is distended. The embryo dies early and gets absorbed or becomes indistinguishable. Intraperitoneal haemorrhage is minimal. The diagnosis is often missed due to the lack of symptoms.

### **Isthmic Implantation**

Perforation and rupture of the tube occurs very early, even in the 3rd week, before the menstruation can be missed. There is usually a sudden pain with possible syncope, which subsides within a short time, only to recur some days later with the accumulation of a pelvic haematocele. On laparotomy a tube detached from the uterine horn is very often seen.

### **Interstitial Implantation**

In this rare condition, the uterus get distorted and is found much enlarged in one corner. This may give rise to *angular pregnancy* or "*graviditas in substantia uteri*". The diagnosis may be very difficult.

## **Abdominal Pregnancy**

In the majority of cases, the site of primary implantation is not recorded. Among the documented ones, the sites include the retrouterine cavity, the bladder peritoneum, the omentum, small bowel, broad ligament and the serosa of the fallopian tube. We recently missed confirmation of a subdiaphragmatic implantation with fetal transverse-lie and scanty amniotic fluid in a pregnancy of 32 weeks gestational age, as demonstrated clearly by ultrasound. The patient refused surgical intervention and preferred to consult another hospital. We lost trace of her. Most cases are secondary to ruptured tubal gestation. Primary abdominal implantation is very rare.

## **Ovarian Pregnancy**

Several well authenticated cases have been recorded. In recent years it involved women with an IUCD, and it can be postulated that an endometriosis of the ovary promotes the occurrence subsequent to an *in situ* fertilisation.

## **Varia**

The other sites of implantation are extremely rare and are often diagnosed only at laparotomy.

## **Clinical Presentation**

Depending on the site of implantation, the symptomatology of an extrauterine gestation can be manifold. However, one can distinguish between:

- the masked phase
- the dramatic phase.

Case history plays a major role, when there is suspicion of an ectopic - the duration of amenorrhoea, a previous lower abdominal operation, history of PID, treatment and any infertility investigations. Finally, the use of intrauterine contraceptive devices should always be questioned.

### **Masked Phase (Premonitory)**

The organism first reacts the same as in normal pregnancy, with lividity, softening of the uterus and with endocrine changes. The pregnancy test is positive and the adnexal findings reveal no abnormalities.

Beyond the 5th week "post menstruationem" there may commence uncharacteristic pain, often localised. The assumption is that the uneasiness is caused by the distending tube and by accumulating pelvic haemorrhage. Sometimes it combines with dysuria and frequency of micturition. The tenesmen can also be referred to the bowel.

On account of nutritional insufficiency in the ectopic conceptus, with an ensuing deficit in the hormone production, uterine bleeding (decidual haemorrhage) commences.

This can be misinterpreted as delayed menstruation, functional imbalance or threatened abortion.

The other signs like nausea, orthostatic dysregulation and breast tenderness have no characteristic intensity and can only indicate the probability of a pregnancy.

### **Dramatic Phase (Acute)**

Once again, depending on the localisation of the ectopic pregnancy, the symptoms will be protean.

### **Tubal Pregnancy**

This will commonly cause:

- early, unilateral abdominal pain
- sudden, fulminating pain with syncope
- development of pelvic haematocele.

In case of tubal abortion, the symptoms change to another pattern involving:

- uterine bleeding after a period of amenorrhoea
- unilateral abdominal pain
- shoulder tips pain
- progredient hypovolaemia and shock.

### **Intra-Abdominal Pregnancy**

This very often shows signs of:

- "spurious labour"
- severe abdominal pain
- expulsion of decidua.

If the extrauterine pregnancy is still intact, there will be more energetic fetal movements, which decrease with progressive gestation, due to faulty position and lack of room.

Should the fetus die, and the gestation remain undiagnosed, the fluid gets *mummified*. The resulting intraabdominal tumour can undergo *calcification* and cause abdominal symptoms. Occasionally, the fetus turns into a *lithopaedion*, and can remain asymptomatic for many years.

The symptomatology mentioned for tubal pregnancy is valid in most instances for ovarian pregnancy and ruptured cornual pregnancy as well.



## **Differential Diagnosis**

- Fulminating appendicitis
- Torsion or rupture of ovarian cyst
- Rupture of gastric or duodenal ulcer
- Acute haemoperitoneum.

## **Diagnosis and Special Investigations**

Considering the protean symptomatology of extrauterine gestation, the classical triad of:

- lower abdominal pain
- abnormal uterine bleeding, and
- adnexal mass

is found in far less than 50% of the cases.

The most valuable tools in modern day practice, if preoperative diagnosis is imperative and time is available and case history warrants are:

- Beta - hCG
- Ultrasonography.

## **Routine Investigations**

Full blood count (FBC) to determine the Hb level and degree of anaemia. Leucocytosis may be of little significance and specificity, but will pick up a concurrent infection.

Blood typing (ABO/Rh system): In most cases blood transfusion will be needed for resuscitation and for the operation, even though autotransfusion "in tabula" should be performed whenever possible.

Beta - hCG: If negative, this may not exclude a dead conceptus and if positive, then viable trophoblastic tissue is present.

## **Special Investigation**

Culdocentesis is still very common as a diagnostic measure. False positive rates are high, however, in many cases caused by haemorrhagic corpus luteum, puncture of a retroflected uterus, or even retrograde menstruation. Negative results do not rule out the existence of an ectopic pregnancy. The method is dangerous in patients who, in addition also have chronic PID.

Ultrasonography: A noninvasive ultrasound examination will demonstrate a gestation-sac, either in utero or extrauterine, already in the 6th week postmenstruationem. Rare cases of heterotopic pregnancy can be picked up.

If the conceptus is intrauterine, then appropriate therapy for abortus imminens or incipiens should be planned. A negative ultrasound finding does not, however, exclude the possibility of a ruptured, early tubal pregnancy. Vary often an IUCD is seen and cystic masses in either adnexa can demonstrate an ectopic pregnancy. Hyperdensity of internal echoes help distinguish from ovarian cyst and hydrosalpinx or haematosalpinx.

In the case of "acute abdomen", a laparoscopy should be done immediately. Above 20% of the cases are admitted in shock or with shock symptoms, and history-taking is inaccurate. Repeated vaginal examinations to assess size of uterus and affection of the adnexal organs should be avoided, as the procedure can result in the rupture of an extrauterine gestation. A quick, preoperative, bimanual examination under anaesthesia can, however, be useful.

Laparoscopy: Any suspicion of ectopic pregnancy is enough indication for laparoscopy. Earlier reports gave a false negative rate of up to 5% (Samuelson, 1972), but pathoanatomical alterations in PID, organised coagula in pelvic haematocele and early gestations are complicating factors. An operating laparoscopy enables the aspiration of the haematocele and very often the diagnosis of ruptured corpora lutea and retrograde menstruation (Frangenheim, 1980). A plan for laparotomy can then be dropped. Today, the laparoscope is a valuable diagnostic tool that helps pick up more than 50% of ectopic pregnancies before rupture and enables early operation.

### **Treatment**

At the end of all diagnostic measures and eventual resuscitation with transfusion, correction of haemodynamic and treatment of shock symptoms, a laparotomy should not be delayed. In the absence of an operating laparoscope, laparotomy should be performed under the same anaesthesia.

### **Digital Expression**

This method, often referred to as "milking" of the conceptus was performed in ampullary implantations and when conservative treatment of the tube was considered advisable. Other authorities advise strongly against the method because of haemorrhage and recurrent ectopics. Other recommended, however, a vacuum aspiration of the tube to reduce the trauma and haemorrhage.

### **Salpingoscopy**

On the basis of the obstetric history, one should consider a longitudinal incision of the tube to extract the contents. This can be performed equally well with an operating laparoscope. Even if rupture has occurred, the wound can be refreshed and the lumen cleansed. After accurate haemostasis, preferably with diathermy, adaptation of edges with 000 dexon is recommended.

### **Partial Salpingectomy**

One of the conservative methods of treatment is the resectioning of the isthmic site and keeping the distal end for microsurgical anastomosis at a later date.

## **Salpingectomy**

In an attempt to retain the ipsilateral ovary, resection of the affected tube is commonly performed. It has the risk of transmigration of fertilised ova and predisposition to recurrent ectopics

## **Salpingo-Oophorectomy**

This is the easiest surgical treatment, especially in damaged tissues and acute haemorrhage. It can, however, not be recommended in young patients and cases where future fertility is at stake.

## **Hysterectomy**

In a few cases an indication for hysterectomy will be unavoidable, when haemostasis cannot be secured, or in women above the age of 45, with concurrent pelvic pathology - including repeated ectopic pregnancies.

An abdominal pregnancy of any localisation is treated by simply delivering the fetus and clamping the afferent vessels to the placenta before its removal can be contemplated. Should the afferent vessels could not be identified, the placenta can be left in situ. Most authorities believe that resorption of placenta material takes place in the course of time. If later intraabdominal complications develop, a relaparotomy can be performed, when tissues are better reorganised.

## **Colpocoeliotomy**

In cases, where a chronic tubal abortion with pelvic abscess is diagnosed, a colpopuncture should be followed by posterior colpotomy with vaginal drainage. Laparotomy should then be planned for a later date, after infection control.

## **Prognosis**

Depending on the community under study, the mortality rates vary from 5% to 6% for extrauterine gestation. The predisposing factors include the availability of blood for transfusion, as well as the time required before surgical intervention.

## **Fertility**

The chances for further pregnancies is not adversely influenced by surgery. High rates of subsequent intrauterine pregnancy (85%) have been reported after salpingostomy or salpingectomy. On the other hand recurrent ectopic pregnancies have been reported. A prognostic factor is the condition of the contralateral tube at operation. The conservation of unaffected ovaries will, in future, give patients wishing an in vitro fertilisation (IVF) a greater chance of conception.

## **Comment**

### **Ectopic Pregnancy**

#### **H S Cronje**

The topic of ectopic pregnancy is excellently covered in this chapter. One minor point of note is the following: if a patient is Rh negative she should receive at least 100 mg anti-D globulin immediately after the operative procedure.

Although the use of beta human chorionic gonadotrophine (beta hCG) is mentioned in the chapter, its actual role in the diagnosis of tubal ectopic pregnancy is not adequately described. Diagnosis is commonly confirmed by culdocentesis or paracentesis, ultrasonography and laparoscopy. Since laparoscopy involves an anaesthetic and an operative procedure, a need remains for a less invasive procedure. Recently, the utilisation of a rapid radioreceptor assay for beta hCG provided such a method.

When an ectopic pregnancy is suspected, a beta hCG pregnancy test (sensitive from 5 mIU/mL beta hCG and more) is performed on urine or preferably serum. If negative, another cause for the lower abdominal pain should be considered. A positive test is an indication for pelvic ultrasound examination. Ultrasound is not as sensitive in diagnosing a tubal pregnancy as in an intrauterine pregnancy. If an intrauterine gestational sac is seen, the patient is followed expectantly. Without an intrauterine gestational sac, an ectopic pregnancy is considered. The next step is to consider the duration of amenorrhoea. If it is 6 weeks or more, a culdocentesis with or without laparoscopy under anaesthesia, is indicated. Under 6 weeks amenorrhoea the hCG is quantified if the patient is clinically stable (if not, she is taken to the operative room for laparoscopy or laparotomy). A hCG value of more than 6500 mIU/mL the essay is repeated in 2 days time. If it has increased by at least 66%, an intrauterine pregnancy is considered and a pelvic ultrasound is repeated. If the hCG has risen, but less than 66%, a culdocentesis/laparoscopy is indicated. A decrease in hCG values is followed expectantly.

The different methods of treating ectopic pregnancy is well-covered in the chapter, but what is best? Careful histopathologic examination of tubal ectopic pregnancies suggested that, in the majority of cases, the growth of the developing trophoblast involved a large extraluminal site (tubal muscularis and surface). The mass associated with the tubal pregnancy is not dilated tube, but infiltration of trophoblastic tissue within the tubal wall. Therefore, extremely conservative methods of treatment ("milking out" and salpingostomy) will not remove all this trophoblastic tissue. The fate of this tissue if left in situ is unclear. In view of these findings, a logical approach is resectioning of the involved section of the Fallopian tube. Microsurgical reanastomosis can be performed at a later stage if necessary.

Ectopic pregnancy is one cause of the acute lower abdomen pain syndrome, a very common syndrome in gynaecology. Common gynaecologic causes of acute pelvic or lower abdominal pain apart from ectopic pregnancy are salpingitis, torsion of adnexus or ovarian cyst, rupture of ovarian cyst, red degeneration of a myoma, haemorrhagic corpus luteum, and endometriosis. Non-gynaecologic causes include appendicitis, pyelonephritis, renal stone, intestinal obstruction, diverticulitis, other bowel disease, and mesenteric adenitis. Furthermore, other pregnancy-associated conditions (ie, abortions) can also cause pelvic

pain. The important point to remember is that clinical examination is 60-70% sensitive in diagnosing the condition correctly. Laparoscopy is a valuable diagnostic method in the management of lower abdominal or pelvic pain of unknown origin.