MANUAL OF GYNECOLOGY

2024 - 2025

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Table of contents

PREFACE

What urged us to make this concise '*MANUAL OF GYNECOLOGY'* is the reduction of the study weeks for our medical student in' 2+5 program' in faculty of medicine – Mansoura University.

We tried to summarize knowledge for our students to meet the basic requirements needed to know in Gynecology for the general practitioner.

We have eliminated the sophisticated in-depth knowledge about the unproved etiologies and pathogenesis of some gynecological diseases.

We tried to give up-to-date knowledge about each subject.

We hope this edition be easy to study, useful to gain knowledge for our students (daughters & sons) in our beloved medical school.

This book will be offered on its digital (soft) form ***free of charge****.*

The authors

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PART ONE

- Anatomy, Physiology, and Embryology
- Puberty, menopause
- Premenstrual tension syndrome
- Dysmenorrhea
- Amenorrhea
- Galactorrhea
- Female genital mutilation
- Poly cystic ovary syndrome

ANATOMY OF THE FEMALE GENITAL TRACT

The anatomy of the female genital organs includes external genitalia "vulva" and internal genitalia.

The External Genitalia (Vulva)

1- Mons veneris: a pad of fat on the symphysis pubis, covered by hairy skin.

2- Labia majora:

- Two large skin folds covered by hairs except from the inner aspects.
- They are homologous to male **scrotum.**
- They contain in its posterior **1/3**, **Bartholin's glands** which are modified racemose sebaceous glands secreting sebaceous material for lubrication during sexual excitement.
- Each gland has a duct of about 2 cm long that passes between the hymen and labia minora to open in the vagina. The duct openings are not seen except when inflamed or obstructed.

3- Labia minora:

- Two folds of smooth non-hairy skin lying medial to labia majora and smaller than it.
- They are very vascular organ so become turgid and congested with sexual excitement.
- Both divide anteriorly to form "prepuce" above and "frenulum" below glans of clitoris.
- They unite posteriorly to form the "fourchette".
- It is homologous to **penile urethra** in male.

4- Clitoris:

- It is a highly sensitive erectile organ that is homologous to male **penis.**
- It is formed of glans and body which in turn is formed of 2 crura cavernosa.
- The glans is covered by prepuce from above and frenulum from below.

5- External urethral meatus:

- It is a triangular slit below the clitoris and can be seen by separating the labia minora.
- The normal female urethral length is 3-4 cm, so it is totally related to anterior vaginal wall.

6-External vaginal orifice: it is the lower vaginal end, closed by the hymen in the virgin.

7-The hymen:

- It is a double layered mucous membrane that closes the lower vaginal orifice in the virgin.
- It has an opening for the menstrual blood to pass; this may be single, bi-partite, cribriform, annular, or crescentic.

9-Vestibule:

- It is the closed space between the 2 labia minora and in which the following open; external urethral meatus, external vaginal orifice, and Bartholin's gland. **Vestibular bulbs, t**wo oblong erectile tissues, lying on each side of the vaginal orifice and seen on straining.

10-Perineum:

- It is the area that lies between the vaginal orifice anteriorly and the anus posteriorly & covering the perineal body. It is formed of:

i. Perineal skin.

ii. Subcutaneous tissues.

iii. Perineal muscles, which are:

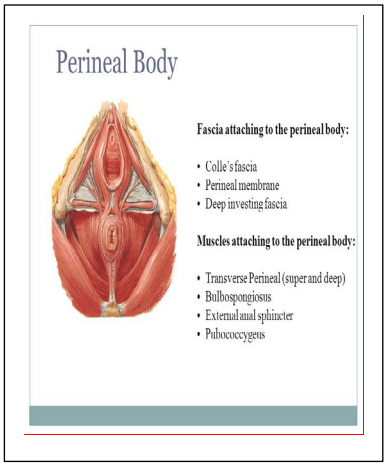
1-Superficial and deep transvers perinii ms.

2-Bulbospongiosus ms.

3-Deep fibers of levator ani ms "pupococcegeus part".

"All these muscles. meet in the midline to form a strong fibromuscular tendon sharing in the support of the genital organ which is <u>the perineal body".</u>

4-Ischiocavernosus muscle. **"Not sharing in the perineal body".**



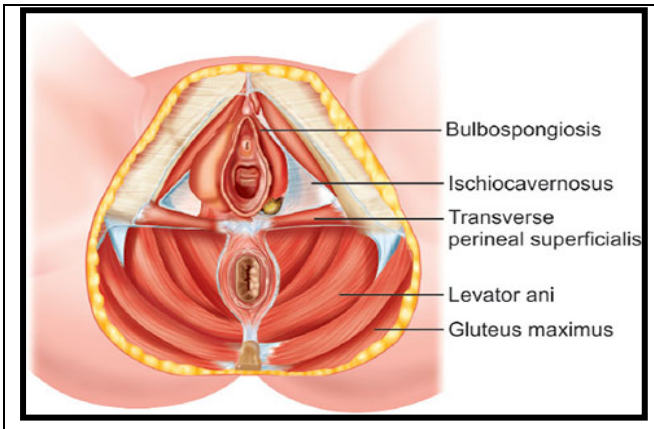
11-Blood and nerve supply of the vulva:

a- Arterial: internal and external pudendal arteries.

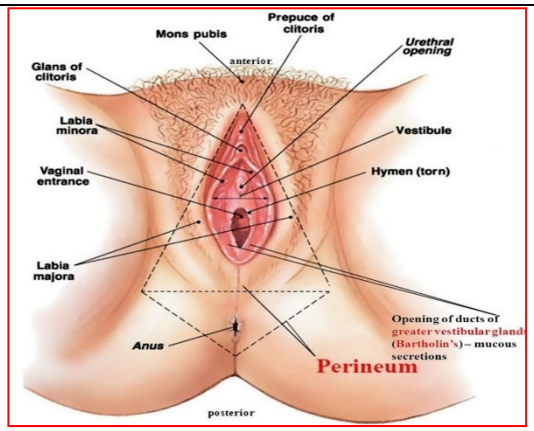
b- Venous: corresponding to the arteries.

c- Lymphatic drainage: to superficial and deep femoral and inguinal L.N

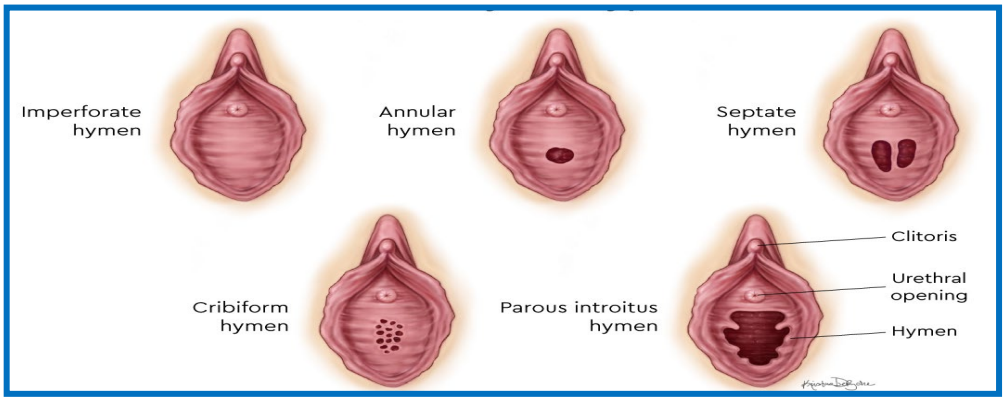
d- Nerve supply: **Pudendal nerve** "from S2, S3, S4 motor and sensory", i**lioinguinal nerve, genital branch of genitofemoral nerve and posterior cutaneous nerve of the thigh.**



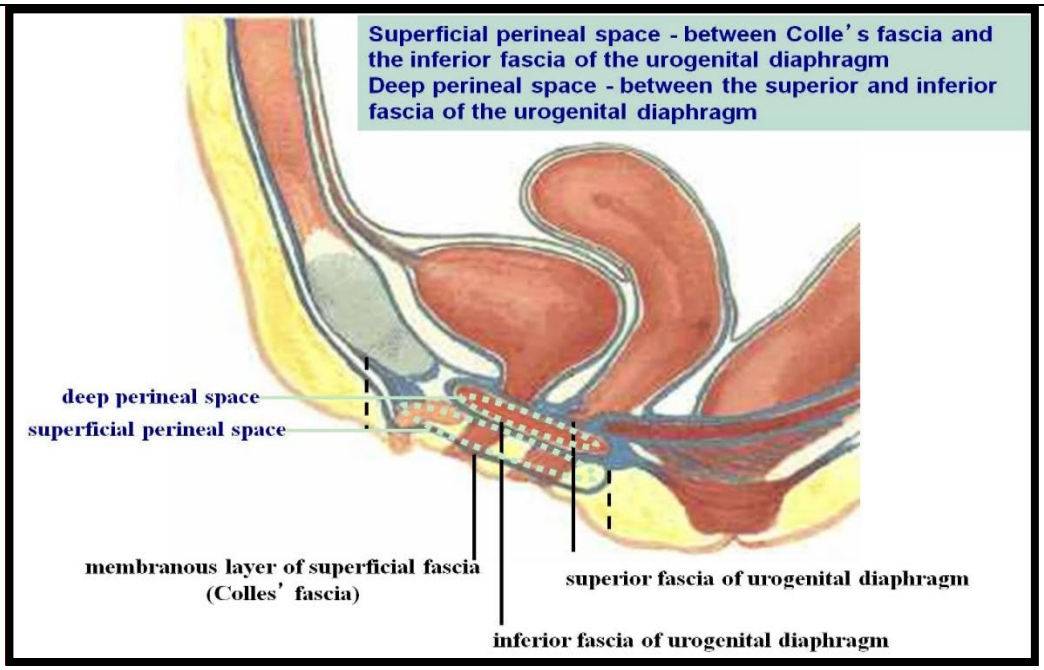
Perineal muscles



External genitalia



Types of the hymen



Superficial & deep perineal pouches

THE VAGINA

- It is a fibro-muscular organ extending from the vulva to the uterus.
- It is apparent as inverted flask as the upper end is twice capacious than the lower end.
- The lower end is closed by hymen in virgins whereas its upper end is blind "vaginal vault".
- The vaginal vault is pierced by the cervix and so there are 4 pouches or fornices, one anterior, one posterior and two lateral fornices.
- The anterior wall measures from 7-9 cm, while the posterior wall from 9-11 cm.
- **Histology:** formed of 4 layers :

1-Mucosa:

- It is stratified squamous non keratinized epithelium with no glands.
- The epithelium is thrown into folds called vaginal rugae.
- The rugae harbor certain organism known as **Doderlin's bacilli** that is responsible for vaginal acidity **[PH at 4.5]** by breaking down glycogen and releasing lactic acid.
- The action of these bacilli is stimulated by estrogen.
- The vagina is kept moist by cervical secretions and exudates from the epithelium.
- The anterior vaginal wall shows 4 depressions or sulci in the mucosa.

2-Tonica propria: a thin layer of elastic vascular connective tissues.

3-Muscle layer: a thin layer of musculo-facial envelope that surrounds the vagina and continuous above with that of the cx and uterus.

4-Adventitia: outer fibrous tissue layer, which is excessive and known as "paracolpos" and allows high dispensability of the vagina.

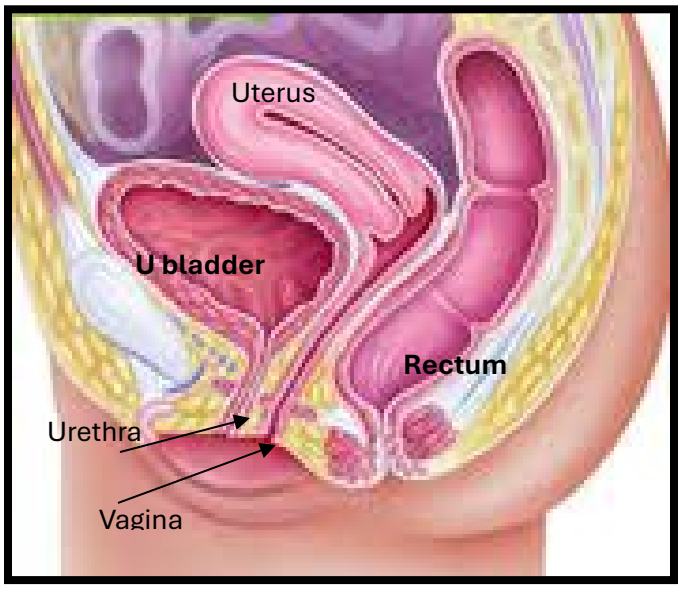
- Blood supply of the vagina:

1-Arterial:

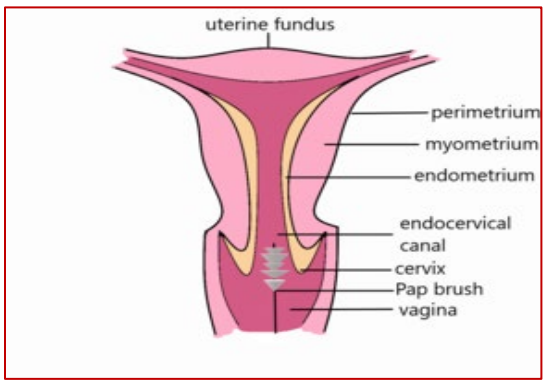
Vaginal artery: arises from internal iliac or from uterine artery and supplies mainly the upper vagina.

Branches from: middle, inferior rectal, or vesical arteries supply mainly the lower vagina.

2-Venous: accompany the corresponding arteries and drain to the internal iliac vein.



THE UTERUS (the womb)



- It is an inverted pear-shaped organ that lies central in the pelvis with the external os at the level of the ischial spine while internal os is at the level of sym. pubis in the standing position.
-It measures 1x 2 x 3 inches and its weight is about 50-80 gm.
-The uterus is almost always anteverted-anteflexed position *[AVF position]:*
●***Anteversion*** i.e., the cervix is bent on the axis of the vagina by a right angle.
●***Anteflexion*** i.e., the relation between the long axis of the cervix and that of the fundus & body makes an obtuse angle of about 150-160°.
-The uterus is divided anatomically into:

1- Uterine body = corpus uteri:

- Is the main part of the uterus and lies above the level of the internal os.
- The part above the insertion of the fallopian tubes is dom shaped and called the fundus.
- The part in which the tube inserts is called the "the cornu".
- To the cornu 3 structures are attached: Fallopian tube laterally, ovarian ligament posteroinferiorly, and round ligament antero-inferiorly.

-Histology: uterine body is formed of 3 layers

A- Endometrium*:*

-It is a tubular or columnar epithelium, which forms simple tubular glands and rests on the myometrium without basement membrane.

-This layer under the effect of ovarian hormones can be differentiated into:

1- Superficial compact layer shed with menstruation.

2- Middle spongy layer also shed with menstruation.

3- Basal compact layer from which regeneration occurs again.

B- Myometrium = muscle layer:

-Which is formed of three layers "outer longitudinal, inner circular and intermediate

interlacing fibers in crisscross fashion".

-The arrangement of the intermediate one is important in this fashion as it surrounds blood vessels and by contraction of the muscle, there is compression of blood vessels & control of blood flow and blood loss during menstruation and third stage of labor.

C- Perimetrium:

- The uterus is covered by peritoneum anteriorly and posteriorly.
- From ant. surface, it is reflected on the bladder to form "uterovesical pouch".
- From post. surface it is reflected on the rectum to form "Douglas pouch = cul de sac".
- From both sides the 2 layers meet to form the broad ligament.

2)- Isthmus uteri:

- It is the lower most and narrowest part of the uterus just above the cervix.
- It lies between the histological internal os below and anatomical internal os above.
- The anatomical os is that constriction where the body joins the cx, while the histological os is the area of transition of endometrial lining into cervical one.
- It measures 4-5 mm and seen by microscopy.
- It forms the lower uterine segment during pregnancy & labor.
- **Progesterone** causes contraction while **estrogen** causes relaxation of the isthmus.

3)- Cervix:

- It is the lower most part of the uterus and measures about one inch [2.5 cm].
- It is divided by piercing the vaginal vault into supra-vaginal part and portiovaginalis.
- **Histology:**

a- Mucous membrane:

- At the level of the internal os there is gradual change of endometrial epithelium into low columnar epithelium "histological internal os" and then continues to line the cx.
- At the external os it is gradually or abruptly changed into modified squamous epithelium "this area of change is called **transformation zone".**

b- Muscle layer: outer longitudinal and inner circular muscle layers only.

c- Adventitia: a layer of fibrous tissues.

Blood supply of the uterus:

1-Arterial:

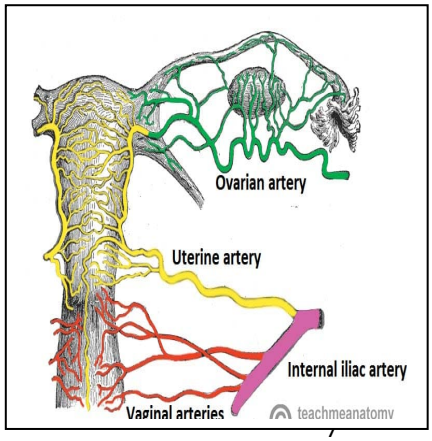
-Uterine artery: from the anterior division of the internal iliac artery.

-Ovarian artery: from the aorta.

2-Venous drainage:

-Uterine vein, pampiniform plexus of veins, internal iliac veins.

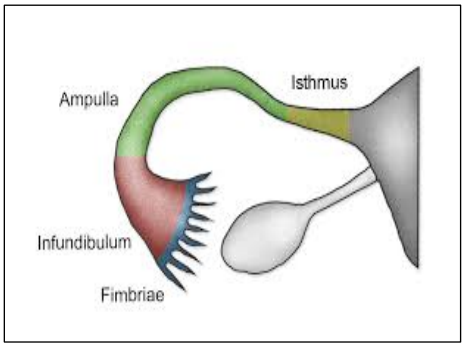
-Ovarian veins "the Rt to I.V.C while the LT-to-LT renal vein".



THE FALLOPIAN TUBES

These are two long tortuous tubes arising from the cornu of the uterus and pass in the free upper end of the broad ligament.

- Each measure about **10-12** cm.



- - It is divided **into 4 parts** "from in out":

- **Interstitial part:** it is that part inserted in the uterine wall.
- **Isthmus:** the narrowest part lying adjacent to the uterus.
- **Ampulla:** the widest and longest part (5-6 cm) lying lateral to the isthmus.
- **Infundibulum:** the fimbriated outer end that opens in the peritoneal cavity by the "ostium". There is a long one fimbria which is directed towards the ovary and is responsible for picking up the ovum and is called "fimbria ovariaca".

- Histology:

A- Endosalpinix:

- An inner layer of columnar epithelium that shows 3 cell types: ciliated type which move its cilia towards the uterine cavity, Goblet like cells [secretory cells], Peg-shaped cells which act as reserve cells.

B- Muscle layer: outer longitudinal and inner circular.

C- Outer serous layer: the peritoneal covering of the tube.

- Blood supply of the fallopian tubes:

1. Arterial: ovarian artery and uterine artery.

2. Venous: ovarian vein, uterine vein and pampiniform plexus of veins.

- Surgical importance of the Fallopian tubes:

1- It has dual blood supply, so gangrene cannot occur.

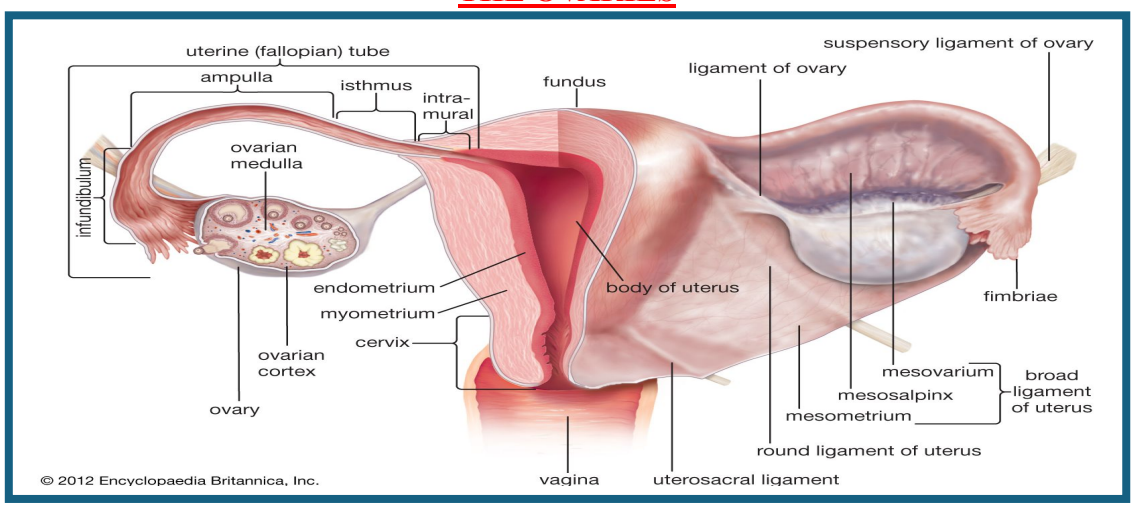
2- Ectopic pregnancy commonly occurs in Fallopian tube [site of fertilization].

3- Tubal causes are the commonest causes of female infertility.

4- It is the site of some procedures of ART like G.I.F.T (gamete intrafallopian transfer), Z.I.F.T.(zygote intrafallopian transfer).

5-Tubal ligation is considered as a method of contraception.

THE OVARIES



-*The ovary is the primary sex organ in the female*

- It is a retroperitoneal structure lying in the ovarian fossa on the lateral pelvic wall.
- It measures l x 2 x 3 cm and its weight is about 5-10 gm.
- It is dull white in color with a smooth surface in young females but becomes corrugated in adult due to cyclic ovarian trauma after each ovulation.
- **Gross anatomy:** the ovary is formed of hilum, cortex and medulla

  1. Hilum: it is the part on the ventral aspect via which the blood vessels, nerves and lymphatics pass to and from the ovary.

  2. Cortex: it is the active outer part of the ovary as it contains the ovarian follicles.

  3. Medulla: it is the central core formed mainly of fibrous tissues.
- It is kept in place by the following attachments:

  1. Mesovarium: part of the broad ligament suspending the ovary to its back.

  2. Ovarian ligament: fibrous ligament that attaches it to the back of the uterus.

  3. Infundibulo-pelvic ligament: it is the most lateral tapering part of the broad ligament and attaches the ovary to the lateral pelvic wall.
- **Histology:** from out-in -→ germinal epithelium, tunica albuginea, cortex and medulla.

- Blood supply of the ovaries:

1. Arterial: ovarian artery directly from aorta and some branches of uterine artery.

2. Venous: Rt ovarian vein pass to ICV, while Lt ovarian vein pass to Lt renal and some venous drainage pass with uterine vein to pampiniform plexus then internal iliac.

LIGAMENTARY SUPPORTS OF THE UTERUS

- Ligaments that support the female genital tract includes:

A-The cardinal ligaments: these are condensation of pelvic fascia and include:

1- Pubo-cervical ligaments:

-A pair of ligaments Passing from the front of the cervix and upper vagina to the back of the symphysis pubis.

Each is divided into pubo-vesical and vesico-cervical ligaments due to the presence of the bladder and urethra in its course.

- On its reflection on the lower end of the bladder; it joins Mackenroadt's ligament to form a strong support to the bladder known as *bladder pillars.*

2- Mackenroadt's ligaments:

-The **main cardinal ligaments**; it passes as a fan shaped from the lateral aspect of the cx. and upper vagina to be inserted into the lateral pelvic wall.

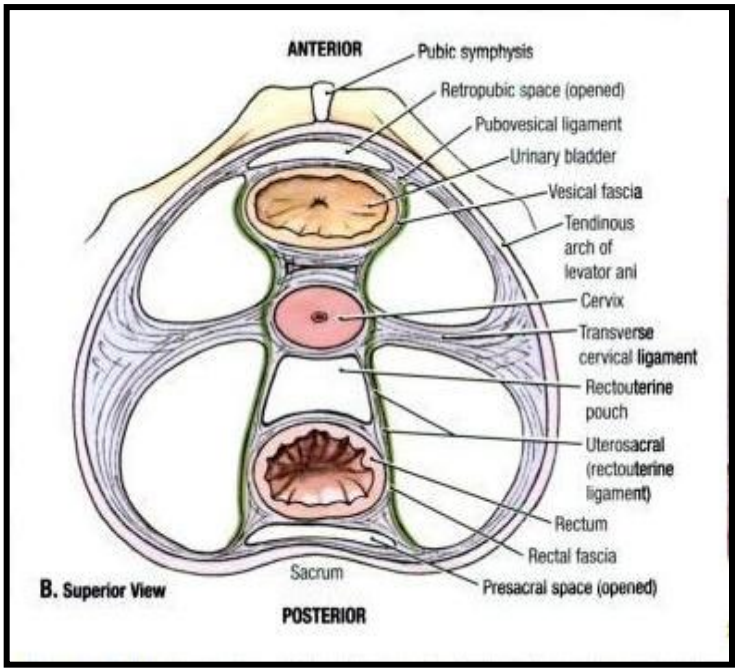
- In this course it is related laterally to uterine artery and the ureter.

3- Uterosacral ligaments:

-The most developed supportive ligament of the uterus.

- It passes from the back of the cx. and upper vagina to the 3rd piece of the sacrum.

- Its tone is responsible for the anteversion position.



B-Accessory ligaments include:

1- Broad ligament:

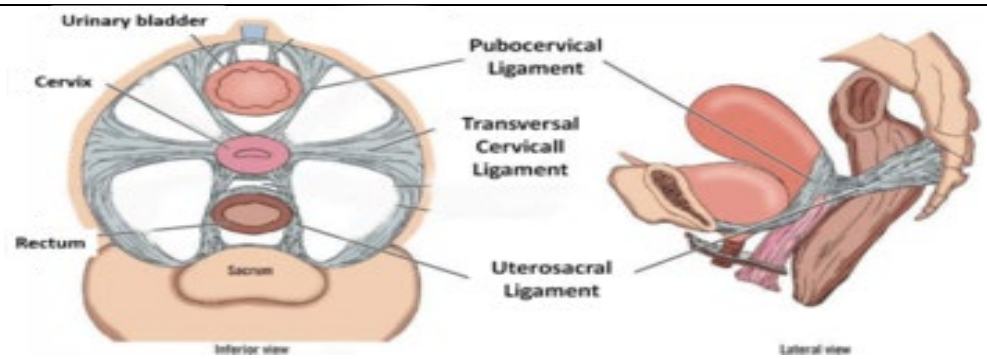
- It is a double sheeted peritoneal layer that arises from the side wall of the uterus due to meeting of the ant. and post. peritoneal covering.

- It then passes laterally to be inserted into the lateral pelvic wall by a tapered end called "infundibulo-pelvic ligament".

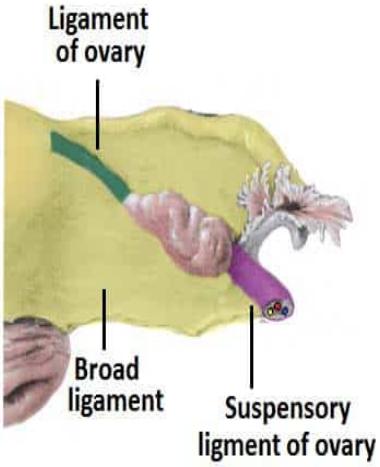
Contents:

1. **Ligaments:** round, ovarian and Mackenroadt's ligaments.

2. **Tubes:** Fallopian tubes above and terminal part of the ureter behind its lowermost end.
4. **Blood vessels:** uterine and ovarian vs and their anastomosis on lower surface of the tube.
5. **Nerves:** ovarian and paracervical nerves.
6. **Lymphatic** drainage of the uterus and along the round ligament.
7. **Vestigeal remnants:** Gartner's duct, epoophron, paroophron and hydatid cyst of Morgagni.



Supportive ligaments of the uterus & upper vagina



Ligaments of the ovary

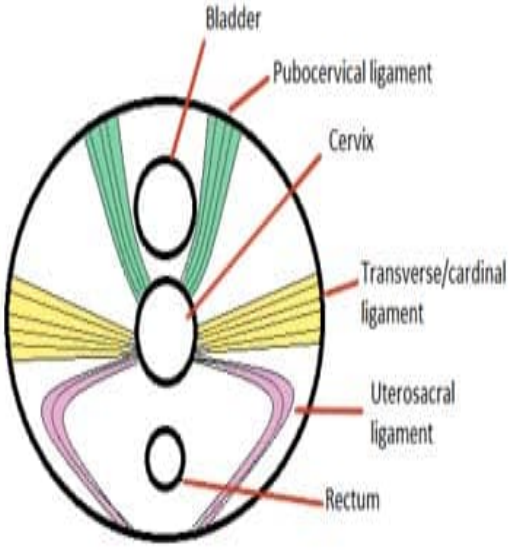


Diagram for the set of uterine supportive ligaments

Pelvic organ support & prolapse lecture via this Link
https://youtu.be/uaxykTlAQ1s?si=cqUPvtsiaIdGlv_8

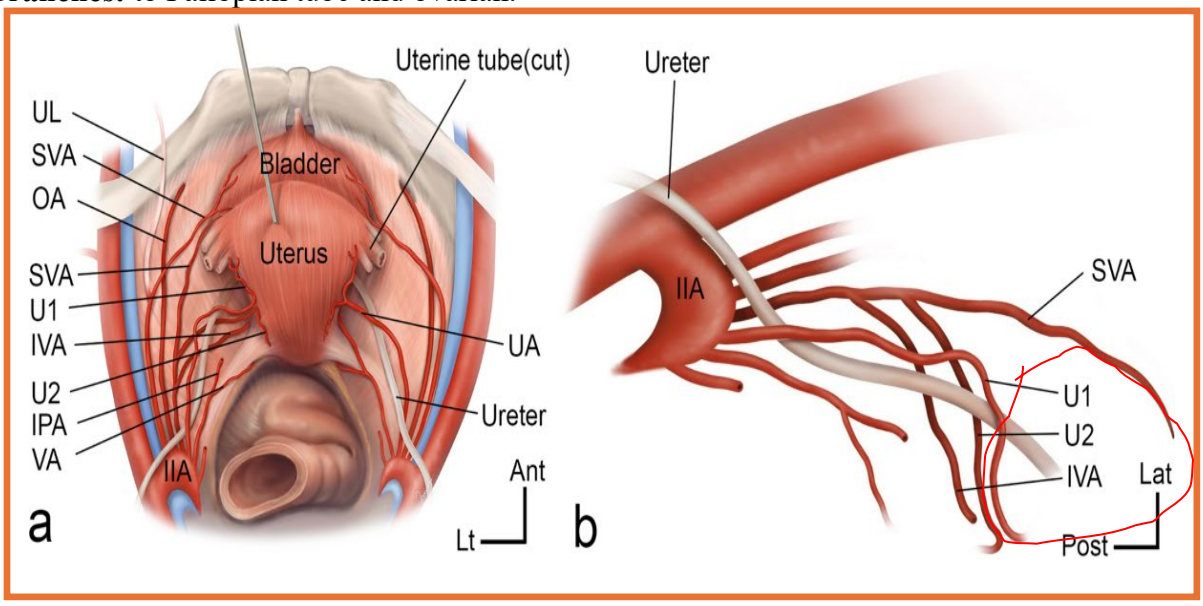
THE UTERINE ARTERY

- It arises from the anterior division of the internal iliac artery as one of its terminal branches.
- It then runs forward & inwards in the base of the broad ligament.
- It crosses above the ureter 1.5-centimeter lateral to the supra-vaginal cervix.
- It reaches the uterus at the level of the internal cervical os.
- It ascends in a tortuous way alongside the uterus between the two leaves of the broad ligament to supply the uterus at all its levels and the ends by anastomosing with the ovarian artery at the distal third of Fallopian tube to form a continuous arterial arch.

- It gives off:

- **The main uterine branch:** divides into anterior & posterior arteries which are disposed circumferentially, in the myometrium and anastomose with those from the opposite side. The uterus is therefore least vascular in the middle line.
- **A descending cervical branch: a** circular artery of the cervix which gives the anterior & posterior azygos arteries to the vagina.
- **Vaginal artery:** it may arise as a separate branch of the uterine artery.
- **Other branches:** to the ureter, bladder and upper vagina.

Branches: to Fallopian tube and ovarian.



IIA= internal iliac artery, UL= umbilical ligament, SVA= superior vesical artery, OA= obturator artery, U1=uterine (from internal iliac) , IVA= inferior vesical, U2= uterine (from superior vesical), IPA= internal pudendal a., VA= vaginal artery.

SUPPORTS OF THE UTERUS, VAGINA, AND PELVIC FLOOR

The position of the uterus and vagina in the pelvis is maintained supported by:

A- Upper supports: by AVF position, round and broad ligaments.

B- Middle supports: by the cardinal ligaments.

C- Lower supports:

- Levator ani and coccygeus muscles.
- Urogenital diaphragm.
- Perineal body.

LEVATOR ANI: which is formed of three main parts:

1. Pubococcygeus part: it is the main part.

Origin: from the back of S, pubis and from part of the white line.

Insertion: it is subdivided into

- Medial fibers that surround the urethra.
- Intermediate fibers form a loop around the vagina, its contraction close the lower vagina.
- Lateral fibers form a loop around the lower rectum and anal canal.

Lastly, the whole fibers are inserted in anococcygeal ligament and lateral margins of the coccyx.

2. Iliococcygeus:

Origin: the white line of the obturator facia.

Insertion: blends with the pubococcygeus to be inserted in the lateral margin of the coccyx.

3. Ischio-coccygeus:

Origin: from the ischial spine.

Insertion: in the lateral margin of the coccyx and last piece of the sacrum.

- The three parts are supplied from sacral 2, 3, 4 nerves.

PELVIC FLOOR:

The pelvic floor is formed of:

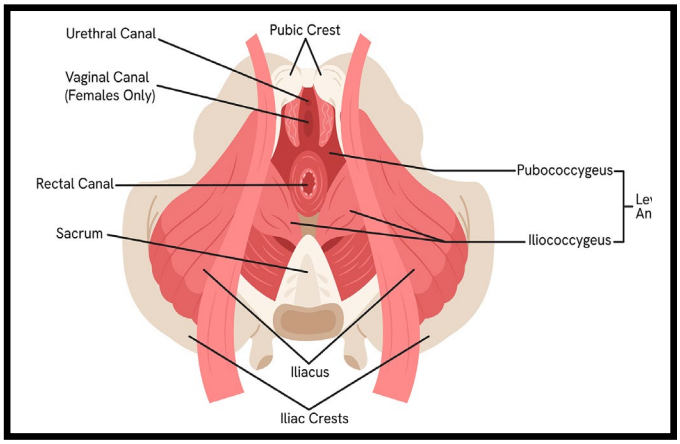
1- Pelvic peritoneum.

2- Pelvic cellular tissues= pelvic facia.

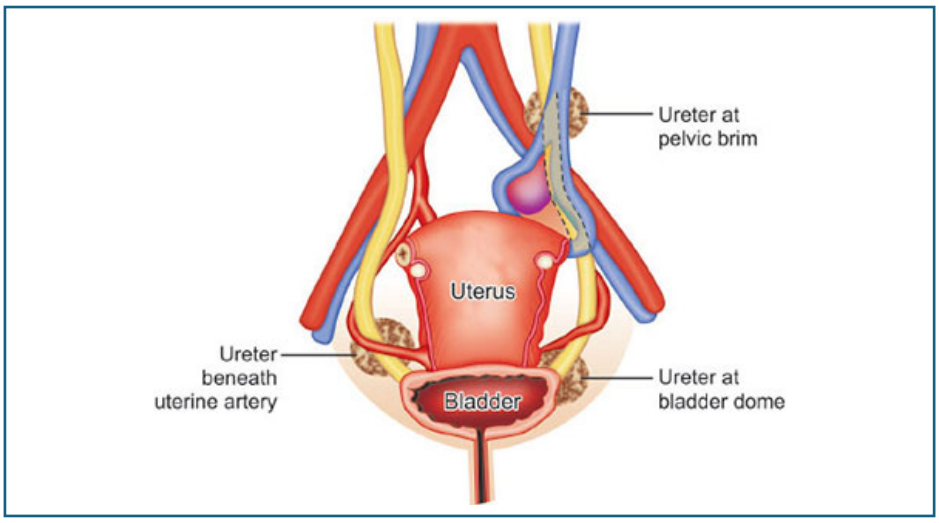
3- Levator ani muscles.

4- Perineal muscles.

5- Subcutaneous fat and skin.



THE PELVIC URETER

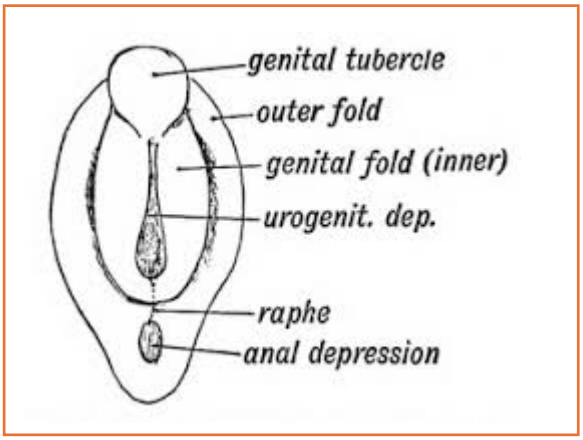


- It is a narrow muscular tube, **25** cm in length that lies retroperitoneal in its whole course.
- It is formed of muscular layer lined by transitional epithelium.
- It enters the pelvis by crossing over the common iliac vessels at the site of its bifurcation.
- It then descends along the front of the internal iliac vessels till the level of ischial spine.
- It then turns inwards and forwards in the base of the broad ligament below and at right angle with uterine artery **"water under bridge";** 1.5 cm lateral to the supra-vaginal cx and above the vaginal vault.
- It then enters the ureteric canal, which lies just above the lateral vaginal fornix and then passes medially to enter the trigone of the bladder.
- It has blood supply from uterine, vaginal and inferior vesical arteries OR a separate special branch from internal iliac called Michael's artery.

Points of ureteric injury during gynecologic surgery:

- At or below the infundibulo-pelvic ligament
- Along the course of ureter on the lateral pelvic side wall just above the uterosacral ligament.
- Where the ureter passes beneath the uterine vessels.
- Beyond the uterine vessels as the ureter passes through the tunnel in the cardinal ligament and turns anteriorly and medially to enter the bladder.
- Intramural portion of ureter when it traverses the bladder wall.
- Devascularization especially in the lower 1/3rd.

EMBRYOLOGY
(DEVELOPMENT OF FEMALE GENITALIA)



I- Development of the external genitalia:

- Median genital **tubercle** gives the clitoris.
- Medial genital **folds** give the labia minora.
- Lateral genital **swellings** give the labia majora.
- **Urogenital membrane** gives vestibule, vaginal and urethral orifices.
- Outer layer of the urogenital sinus covers the vaginal orifice as the **hymen.**

II-Development of the internal genitalia:

1. Ovaries:

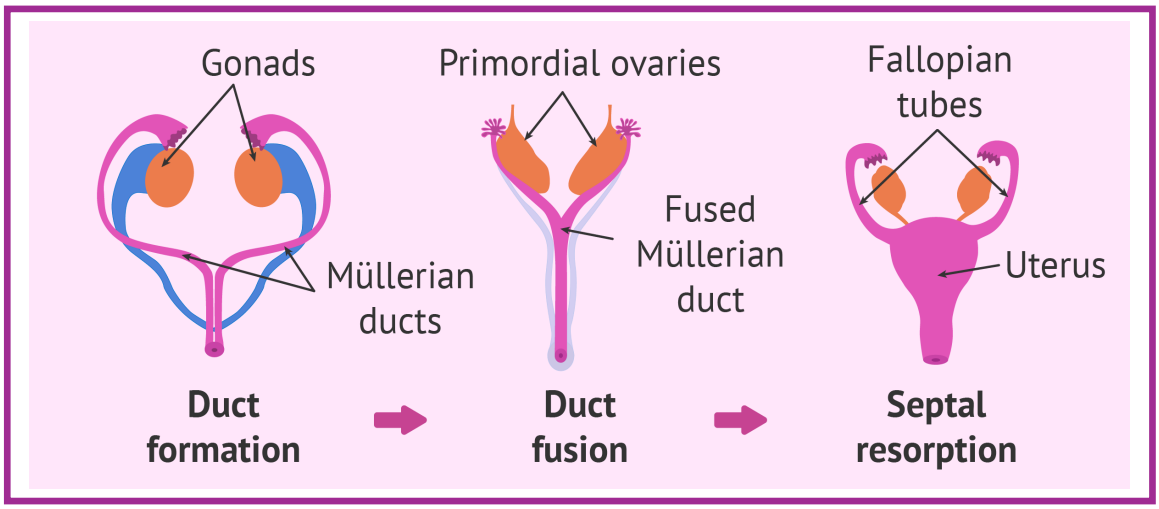
- Develop as the **genital ridge.**
- The surrounding mesoderm migrates to form the cortex and medulla.
- The future **"oogonia"** migrate as germ cell from the yolk sac.
- The ovary descends alongside the **"gubernaculum"** to become a pelvic organ.

2. Fallopian tubes & the Uterus, and upper 3/4 of the vagina:

- They develop from the **"Mullerian ducts"** that appear in the dorsal aspect of the fetus.
- They descend caudally to the pelvis and eventually meet the urogenital sinus.
- Canalization occurs with separation of the septum in between both ducts except its upper free ends. - These upper non fused ends will give fallopian tubes.
- The lower fused part will give uterus, cervix and upper 3/4 of the vagina.

3. Vagina:

- The upper 3/4 as mentioned above.
- The lower 1/4 of the vagina develops from urogenital sinus.



CONGENITAL ANOMALIES OF FEMALE GENITAL TRACT

(1) ANOMALIES OF THE OVARY:

1- Aplasia: no development → primary amenorrhea.
2- Hypoplasia: causing Olig hypomenorrhea or premature menopause.
3- Savage syndrome: i.e., receptor defect with no response to FSH and LH.
5- Pure Turner's syndrome [45 X0]: it causes primary amenorrhea.
6- Mosaic Turner's syndrome: they can present by premature ovarian failure.
7- Testicular feminization syndrome = androgen insensitivity syndrome: this is a genotypically male "46 XY and testicles are present" but phenotypically female as the body responds to androgen secreted as estrogen due to absent androgen receptors.
8- Hermaphrodite: which may be
- True i.e., containing both ovarian and testicular tissues.
- Female pseudo type i.e., containing ovaries but external genitalia are masculine.
- Male pseudo type i.e., containing testicles but external genitalia are feminine.

(2) ANOMALIES OF THE FALLOPIAN TUBES:

1-Aplasia: no development causing primary infertility.
2-Hypoplasia: in the form of long narrow tortuous tube and may cause ectopic pregnancy.
3-Accessory ostium: congenital diverticulum that may cause ectopic pregnancy.

(3) ANOMALIES OF THE UTERUS:

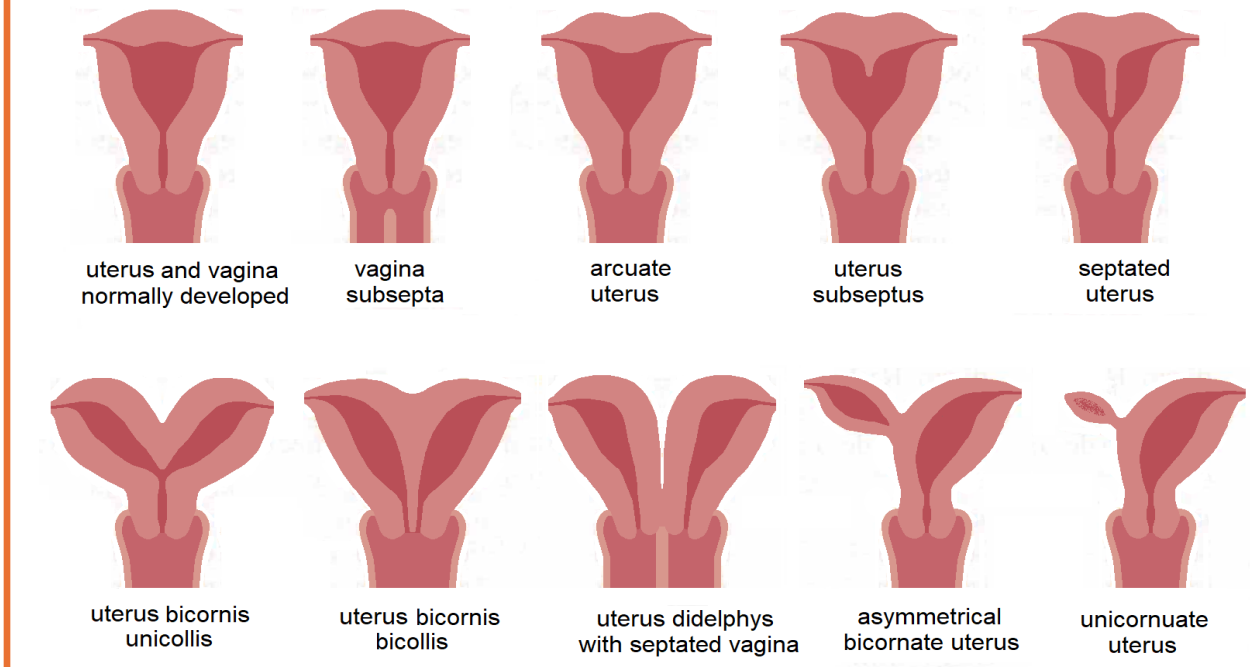
1- Aplasia: no development.
2- Hypoplasia: which may be rudimentary solid uterus, infantile OR prepubescent uterus.
3- Unicornuate uterus: absence of one Mullerian duct with one tube, and narrow vagina.
4- Uterus didelphys: true [2 uteri, 2 cervices, 2 vaginae, 2 vulvae] OR pseudo "one vulva only".
5- Uterus bicornis bicollis: 2 uteri, 2 cervices and one vagina.
6-Septate and sub-septate uterus:
- **In septate uterus:** complete septum in the uterine cavity.
- **In subseptate uterus:** incomplete septum in uterine cavity.

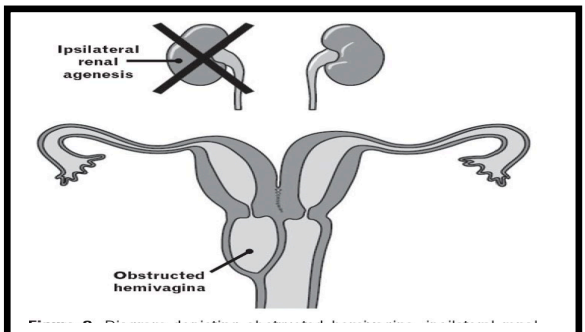
7- Arcuate uterus: slight depression at the fundus.

Patient with uterine anomaly may present by:

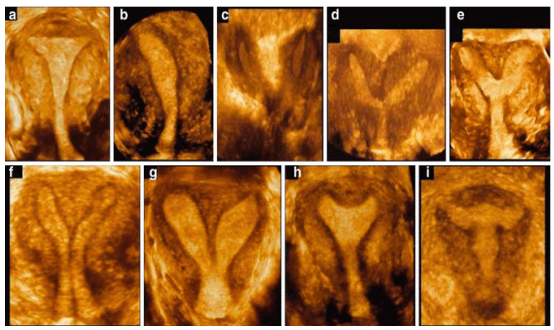
1 Primary amenorrhea **2.** Infertility. **3.** Dysmenorrhea **4.** Abnormal uterine bleeding.
5. Recurrent abortion **6.** Preterm labor
7. Ectopic pregnancy **8.** Mal presentation

Treatment of uterine anomalies: some anomalies are usually treatable by surgery.

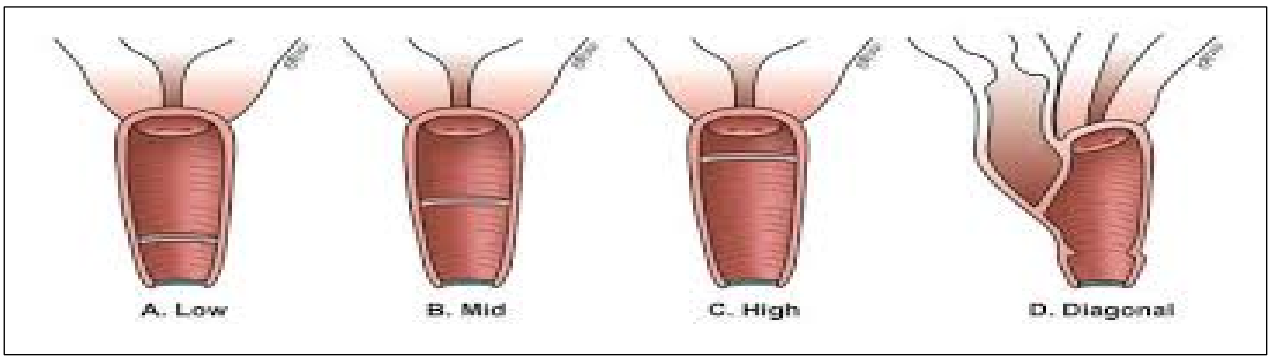




OHVIRA syndrome = Obliterated Hemi-Vagina, +Ipsilateral Renal Agenesis.



3-D transvaginal ultrasound pictures of Mullerian anomalies.

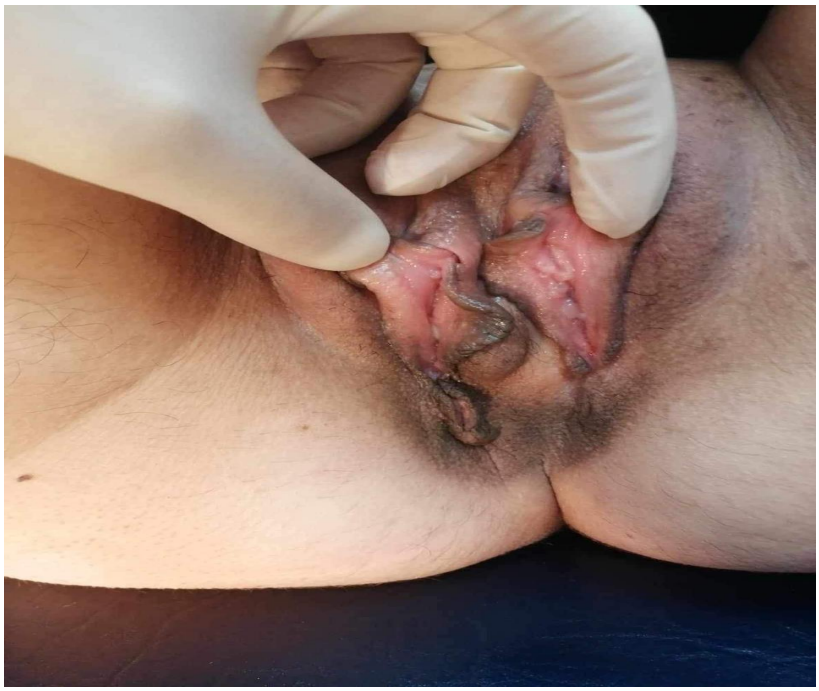


(4) ANOMALIES OF THE VAGINA:

1- **Aplasia:** no development.
2- **Vaginal septum:** which may be longitudinal causing double vagina OR transverse causing crypto-menorrhea "if complete" OR dyspareunia if incomplete.
4- **Congenital atresia.**
5- **Congenital genito-urinary** OR Genito-intestinal fistula.

(5) ANOMALIES OF THE VULVA:

1- **Aplaisa or hypoplasia.**
2- **Hypertrophy of clitoris.**
3- **Bifid clitoris.**
4- **Fusion of labia minora.**
5- **Hypertrophy or asymmetry of the labia.**
6- **Pseudohermaphrodite.**
7- **Hymen anomalies:** imperforate, tough, elastic hymen.
8- **Double vulva** (extremely rare).

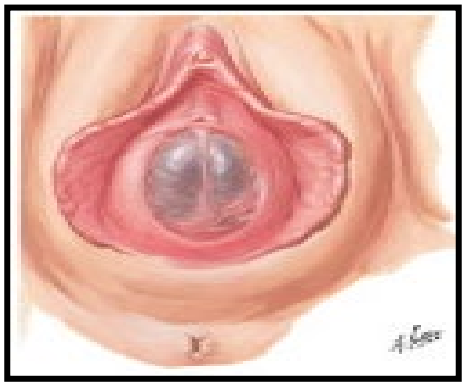


A very rare case of a woman 28-year-old with double vulva

(with permission from prof. Osama Warda)

Related links:
https://youtu.be/loVO5hQy7Js?si=EBf3m2SFXKISqyvN
https://youtu.be/NVd0ZBe-CCs?si=8H7eil6yU9pIA-lm
https://youtu.be/-1k5NRm6nGo?si=nK_Q88EiknwyfBWq

IMPERFORATE HYMEN



- It means absence of hymeneal opening.

Diagnosis:

-Symptoms:

- The patient is usually young age **1-2** years after puberty.
- There is cyclic regular monthly lower abdominal and pelvic pain [uterine contraction with menstruation].
- If the blood is **infected,** it may lead to headache, malaise, nausea and elevated temperature.
- **Amenorrhea** is the usual distressing symptom.
- Urine retention due to compression by the accumulated blood.

-Signs:

1. General: normal changes of puberty and secondary sex characters are present.

2. Abdominal:

- There may be **pelviabdominal** swelling due to accumulation of blood which lead to hemato-colpos; hemato-cevix; hematometra; hemato-salpinx or hemoperitoneum.
- There may be urine retention due to compression on the urethra by the retained blood.

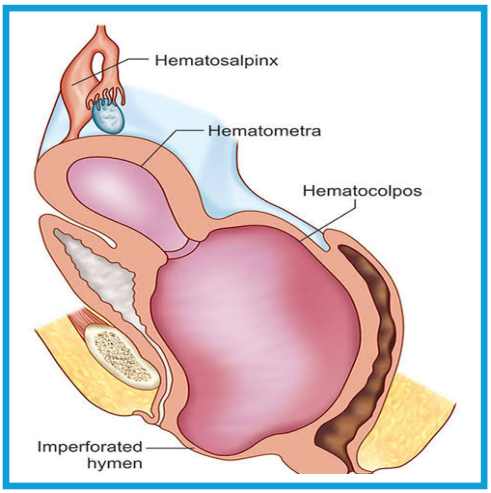
3. P.R: the uterus may be palpated as a small mass in front of the accumulated blood.

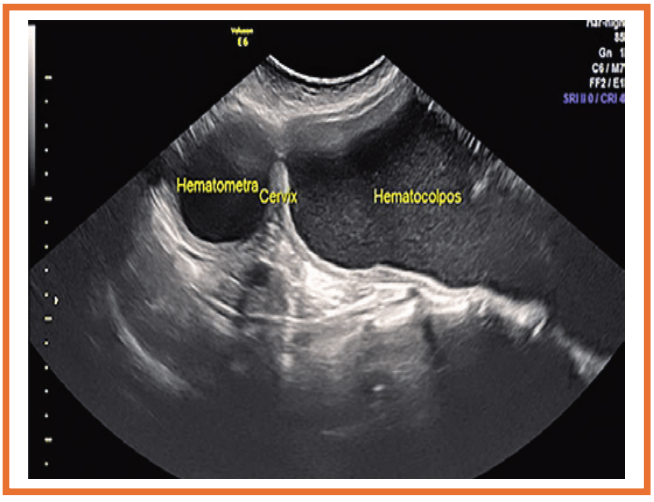
4. Local examination: by **inspection** there is bluish bulging hymen due blood behind it.

2-Investigations: Ultrasound, and IVP (intravenous pyelography to exclude associated urological anomalies.

3- Treatment:

- Should be done in **governmental** hospitals to give the patient a certificate.
- Under complete aseptic technique with the bladder empty, a cruciate or crescent shaped incision is done in the hymen followed by trimming of the edges.





PHYSIOLOGY OF FEMALE GENITALIA

- The role of CNS in female reproduction is maintained by hypothalmo-pitiutary ovarian axis.
- The hypothalamus releases gonadotrophin releasing hormone "Gn. RH or L.H.R.H".
- GnRH passes via the hypophyseal portal circulation reaching the anterior pituitary gland to release pituitary gonadotrophins "FSH and LH".
- The pituitary FSH and LH act on the ovary to produce ova and hormones and the end result either fertilization of the ova and pregnancy OR no fertilization and menstruation occurs.

Central hormones

I- Gonadotrophin releasing hormone (= Gn RH)

- It is a **decapeptide** hormone (**10** amino acids).
- Secreted from the hypothalamus in pulsatile manner every **60-90** min.
- It reaches the anterior pituitary via hypophyseal portal circulation to release FSH and LH.
- It stimulates the secretion of more L.H than FSH hormone, so commonly named LHRH.
- Its half-life is about **2-4** minutes.

II- Pituitary gonadotrophins (FSH & LH)

- Both are water-soluble glycoprotein hormones.
- Composed of **2** subunits: α-subunit which is similar in all pituitary hormones, and **β**-subunit which is **specific** for each hormone.
- They are responsible for the endocrinological background of the ovarian cycle.
- The half life is **3-4** hours in F.S.H and that of LH is **20** minutes.

a- Action of follicle stimulating hormone [F.S.H]:

1- Growth, ripening and maturation of the ovarian follicles.
2- Stimulation of the formation of L.H receptors.
3- Acts on the granulosa cells to stimulate the secretion of estrogen.
4- In combination with L.H it causes ovu1ation.

b- Action of luteinizing hormone [L.H]:

1- Stimulation of ripening and complete maturation of the follicles.
2- L.H. surge is responsible for rupture of the mature Graafian follicle i.e., ovulation.
3- Luteinization of the granulosa and theca cells of the C.L.
4- Stimulation of the C.L to release progesterone and to less extent estrogens.
5- Completion of first meiotic division in oocyte.

Ovarian Hormones

1-Estrogen

Sources: it is produced from the following sources:-

1. **Ovary:** mainly from Graffain follicles and to less extent from the corpus luteum.
2. **Placenta**.
3. **Adrenal cortex**.
4. **The peripheral conversion** of androgens into estrogen in the adipose tissue.

Types:

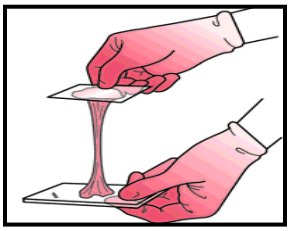
1- **El = estrone** from the adrenal cortex and peripheral conversion in adipose tissues.
2- **E2 = estradiol** from the ovary. **It is the main type of estrogen**.
3- **E3 = estriol** from the placenta.
4- **E4 = estetrole** from the fetus.

Metabolism: metabolized in the liver and the end products are secreted in urine.

Action:

I- Local action: i.e., on the genital tract.

1. Increases the vascularity causing softening of the whole genital organs.
2. **Uterus:** causes proliferation and hyperplasia of the endornetriurn and myometriurn and responsible for **proliferative phase** of the menstrual cycle.
3. **Tubes:** hyperplasia, hypertrophy, increases blood supply and motility of the tubes.
4. **Ovary:** helps in the mechanism of ovulation **"by feed back mechanism with pituitary".**
5. **Cervix:** increased amount decreased viscosity and cellularity **[+ve Spinnbarkiet and Ferning].**
6. **Vagina:** proliferation of the epithelial lining, increased vaginal acidity (by activation of the lactobacilli to convert glycogen into lactic acid).



II- Central action: i.e., on the pituitary & hypothalamus:

- Estrogen causes inhibition of F.S.H release from the pituitary but increases L.H release from the pituitary causing L.H **surge** at the time of ovulation.

III- General action:

- It is responsible for appearance of secondary sex characters at puberty.

1. **Breast:** increases proliferation of the duct system and the vascularity.
2. **Cardiovascular:** in physiological doses it protects against ischemic ht. diseases as it increases HDL and decreases LDL.
3. **On bone:** it has osteoblastic activity causing union of the epiphysis.

2- PROGESTERONE

Sources:

1.Ovary: mainly from the corpus luteum. **2. Placenta**

Types and metabolism: only one type metabolized in the liver and excreted in urine.

Action:

I- Local action:

1. **Uterus:** causes secretory endometrium (it must be prepared by estrogen). "Progesterone is the hormone of pregnancy, responsible for maintenance of the decidua during early pregnancy and also uterine relaxation".
2. **Tube:** it decreases motility.
3. **Cervix:** decreased amount, so increased cellularity and viscosity **[-ve Spinnbarkiet and Fernning].**

II- Central action: inhibition of L.H and stimulate FSH release from the pituitary".

III-General action:

1- **Thermogenic effect:** it raises the body temperature by **0.3 - 0.5** C°.
2- **Breast:** development of the alveolar system of the breast.
3- **Smooth muscle:** relaxation.

Estrogen & progesterone levels:

- **Estrogen has 2 peaks:** first peak just before ovulation and a second peak after C.L formation.
- **Progesterone has 1 peak:** mid-luteal peak (after C.L formation).

Important notes:

ANDROGENS

- The circulating androgens come mainly from adrenal gland and to less extent from the ovary.
- In female, it is responsible for maintenance of pubic and axillary hair, and control of libido.

OVARIAN PEPTIDES

1- Inhibin "A and B types": polypeptide hormone formed by granulosa cells and inhibits FSH action so determining the growth of the dominant follicle.

2- Activin "A and B types": polypeptide hormone formed by granulosa cells and stimulates FSH secretion so enhancing the action on the dominant follicle.

Two cell theory for estrogen formation: Theca cells androgen → granuolsa cells **aromatase enzyme** estradiol and Estrone .

Spinnbarkeit phenomenon: easy stretchability of cervical mucous while ***ferninng*** means appearance of salts in the mucous as palm leave.

Feedback mechanisms: ***{Feedback mechanism is either: +Ve or -Ve }.***

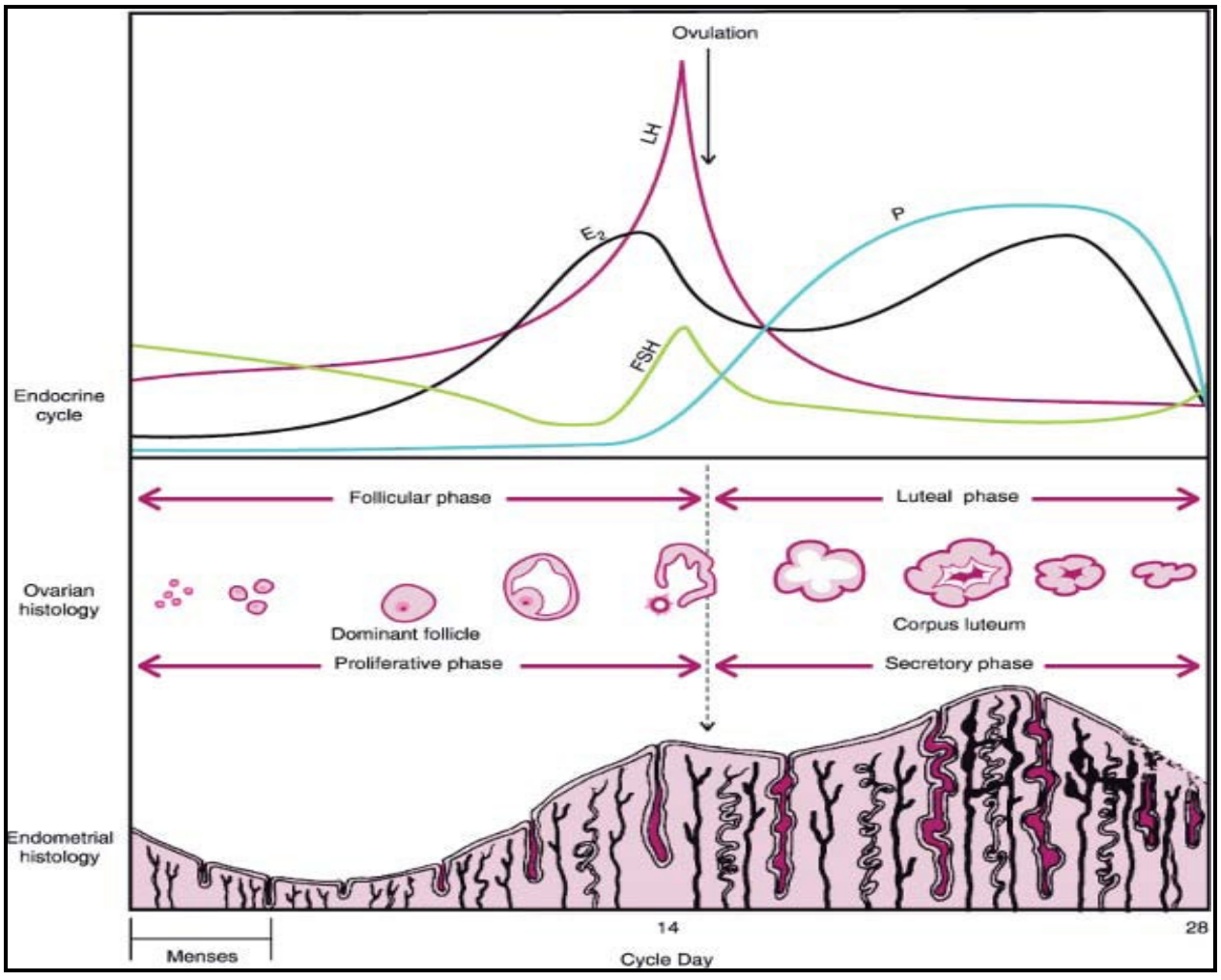
There are 3 types of feedback mechanisms:

1- Long loop feedback mechanism: between the ovaries (estrogen and progesterone) and the hypothalamus or the pituitary.

2- Short loop feedback mechanism: between the pituitary gonadotrophins and the hypothalamic releasing hormones.

3- Ultra short loop feedback mechanism: between the hypothalamus and neighboring centers.

Feed back mechanisms and its relation to ovarian and endometrial cycles



OVARIAN CYCLE

Definition: - It is cyclic monthly changes occurring in the ovary associated with ovulation.

The ovarian cycle is composed of 2 phases; the follicular phase and the luteal phase separated by "ovulation"; its duration is 21-35 days (average ± 28 day).

1-Follicuar phase:

- It is the phase of ripening of primordial follicles to form mature Graffian follicle.

Duration:

It begins on the 1st day of menstruation and occupies the 1st 14 days of the cycle "provided that the cycle is 28 days" may be longer if the cycle is prolonged OR shorter if it is short.

Steps:

- At the beginning of the cycle, there is stimulation of primordial follicles ***(50-150 follicles or more)*** to grow under the effect of pituitary F.S.H, **BUT** One only can reach maturity.
- This mature follicle is called the **dominant** follicle [Graffian follicle].
- **It** is the only one follicle that persists due to more FSH receptors on its surface and action of inhibin and activin hormones [see before].
- During development, the mature Graffin follicle **comes** directly under the **surface** of the ovary to be ready for ovulation (rupture) and releasing the ovum in the peritoneal cavity.

2- Ovulation:

-It means rupture of the mature Graffian follicle and releasing the ovum surrounded by zona pellucida, corona radiata & cumulus oophorus.

Causes of rupture:

1. **Central:** -F.S.H ++ estrogen release from the growing follicles and this in turn ++ L.H **surge** thus inducing ovulation.
2. **Peripheral:** "it is the action mainly of L.H"
   - **a-** Thinning and degeneration of the follicular wall due to proteolytic enzymes.
   - **b- PGs** in the liquor folliculi → contraction of the wall.
   - **c-** Increased intra-follicular tension (due to maximum growth).

3- Luteal phase:

- It is the phase of formation, function & early degeneration of the Corpus Luteum.

Duration:

-It occupies the 2nd 14 days (its duration **is constant** regardless of the ovarian cycle length).

Steps:

- After ovulation & rupture of the mature Graffian follicle the following changes occur:
  - **a-** Collapse in the wall of the follicle.
  - **b-** Luteinization **i.e.,** enlargement of the granulosa & theca cells to form granulosa lutein & theca lutein cells due to obtaining cholesterol, phosphohpids giving a yellow color so called C.L.

Stages (types) of C.L:

1- C.L proliferance: increased vascularization and functions of C.L after its formation with its maximum activity after *5* days and continue for 3-4 days.

2- C.L haemorrhagicum: vascular body.

3- C.L of pregnancy: if fertilization occurs the new embryo releases HCG that prevents degeneration of C.L and continues for 3 months until formation of the placenta.

4- C.L albicans: if NO fertilization; there is degeneration of C.L and turns to fibrous tissues

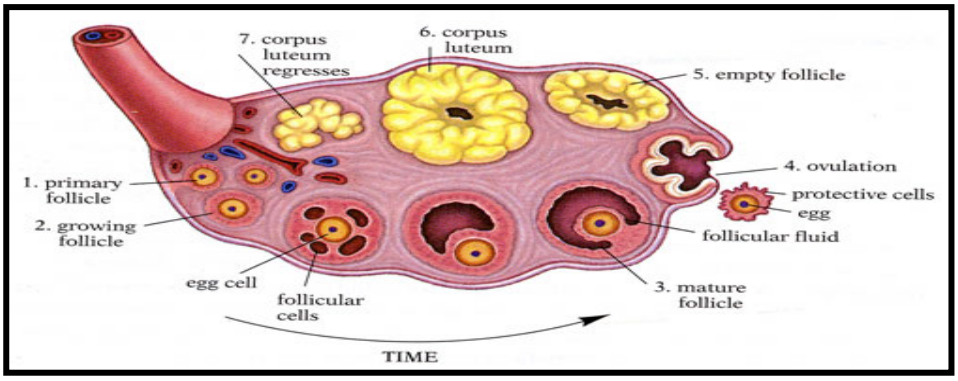
End of the cycle:

If No pregnancy occurred:

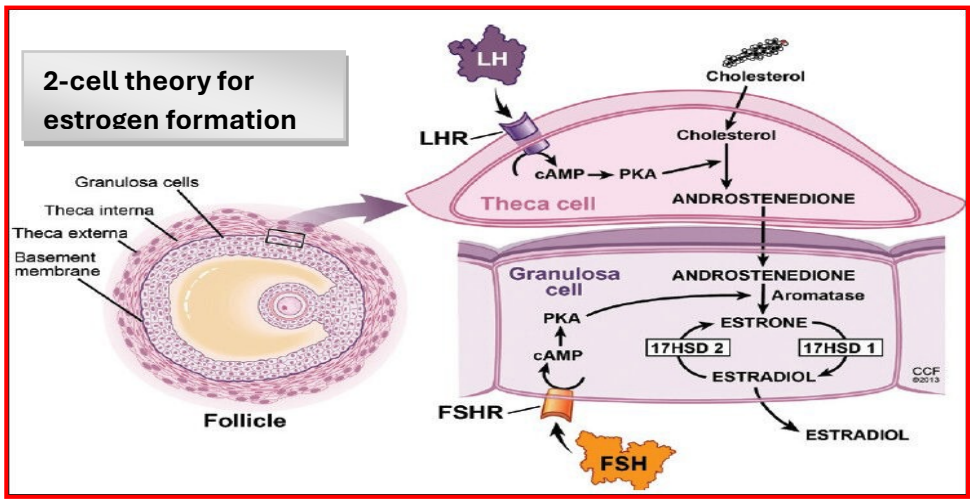
-There is decreased L.H → degeneration with decreased estrogen & progesterone so +ve feedback mechanism on the pituitary and F.S.H release & recycling again.

If pregnancy occurred:

-The C.L is maintained by H.C.G released from the trophoblast; so, estrogen & progesterone are still released.



Stages of follicles growth and development



MENSTRUAL CYCLE = ENDOMETRIAL CYCLE

Definition:- It is the cyclic changes occurring in the endometrium resulting in menstruation every month.

Menstruation: - It is periodic monthly shedding of the endometriurn accompanied by blood loss.

Mechanism: - Decreased estrogen and progesterone release from the degenerated C.L with vasospasm of the endometrial blood vessels and ischemia causing shedding & menstruation.

Contents of the menstrual flow:

1. Blood elements and endometrial debris (necrotic cells).
2. Degenerated ovum.
3. Cervical mucous, vaginal epithelial cells & vaginal discharge.

Normal amount: 30-80 ml. (80 % of this amount occurs in the 1st two days).

Duration: 2-7 days. ***Normal color*:** bricky red.

***Odor*:** Offensive due to sebaceous secretion at the vulva.

***Control of blood loss*:** By a balance between **vasoconstrictors** "$PGF_2\alpha$, endothelin-1 and platelets activating factor and fibrin deposition" and **vasodilators** "PGE_2, PGI_2 and nitric oxide".

Phases of the menstrual cycle:

1- Menstruation phase:

- Its duration is 2-7 days and occurs due to rapid drop of the level of the ovarian hormones.
- Scattered small areas of the endometrium are necrosed and shed off at alternative times.
- Then the whole endometrium is cast off except the deep compact layer from which regeneration will occur.

2- Resting phase:

- It is 1-2 days following menstrual flow with no change.
- The endometrium thickness is 1-2 mm.

3- Proliferative phase:

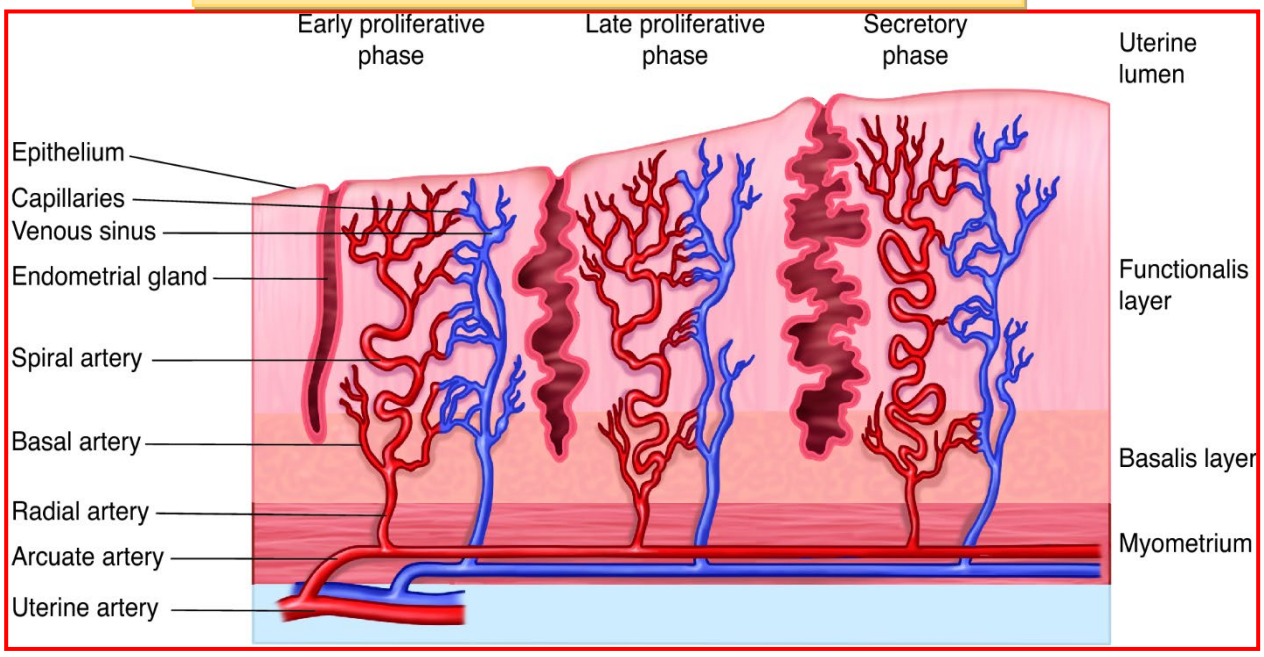
- The endoneurium is built up again under the effect of estrogen produced by the growing follicles.
- The uterine glands become more numerous, dilated, longer and tortuous with increased vascularity.
- The endometrial thickness may reach 2-4 mm.

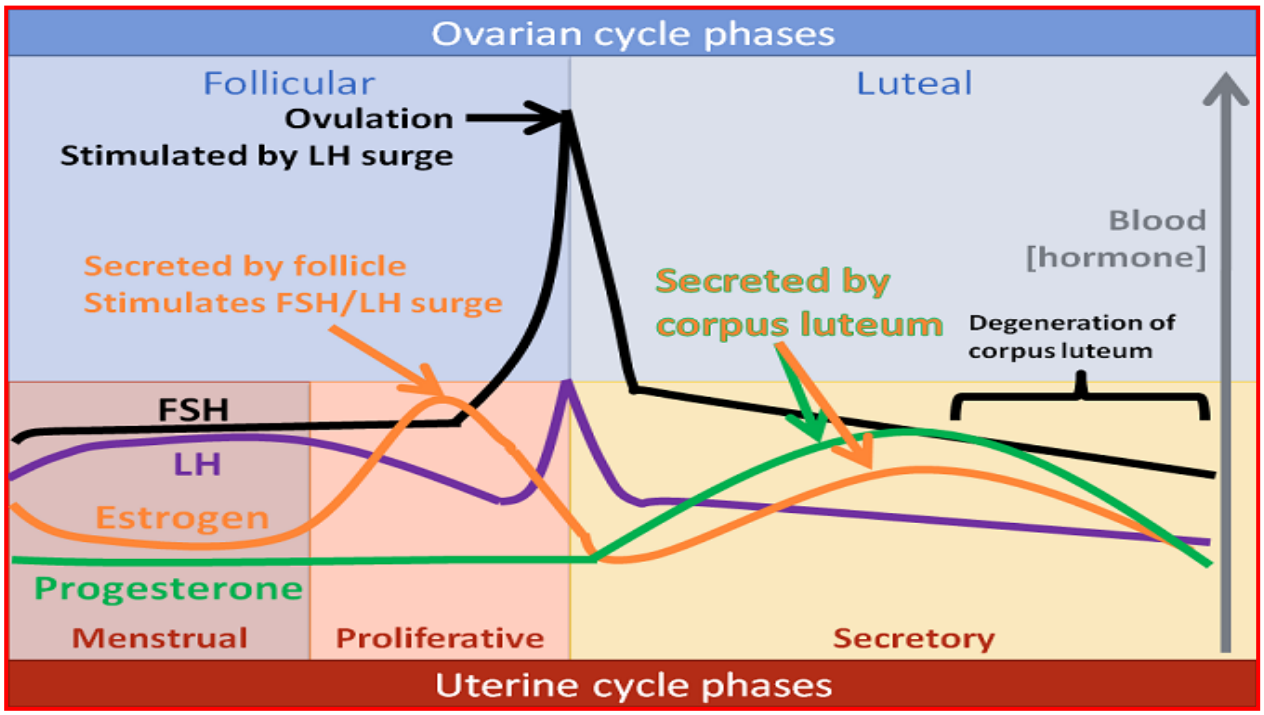
4- Secretory phase:

- It begins on the 15th day of the 28-day cycle), persists until the onset of the next menstrual flow.
- The endometrium becomes highly vascular and slightly edematous.
- The glands appear distended with secretions, tortuous with corkscrew appearance.
- The endometrial thickness may reach 4-8 mm.
- The endometrium is differentiated into 3 layers:

1- Superficial compact layer. 2- Middle spongy layer. 3- Deep compact layer.

The endometrium under different hormonal changes





PUBERTY

Definition:

-It is the period of life in which the female is changed from the childhood to the adulthood associated with endocrinological, developmental & psychological changes.

"Adolescence means complete physiological, behavioral and personal independence".

Timing: from 9-14 years old.

Changes:

I- Endocrino1ogical changes:

- Release of GnRH that acts on the pituitary to release "L.H, F.S.H" that act on the ovary to leading to menstruation and secondary sex characters.

"Also, there is release of GH, TSH, ACTH from ant. pituitary → muscular & skeletal growth".

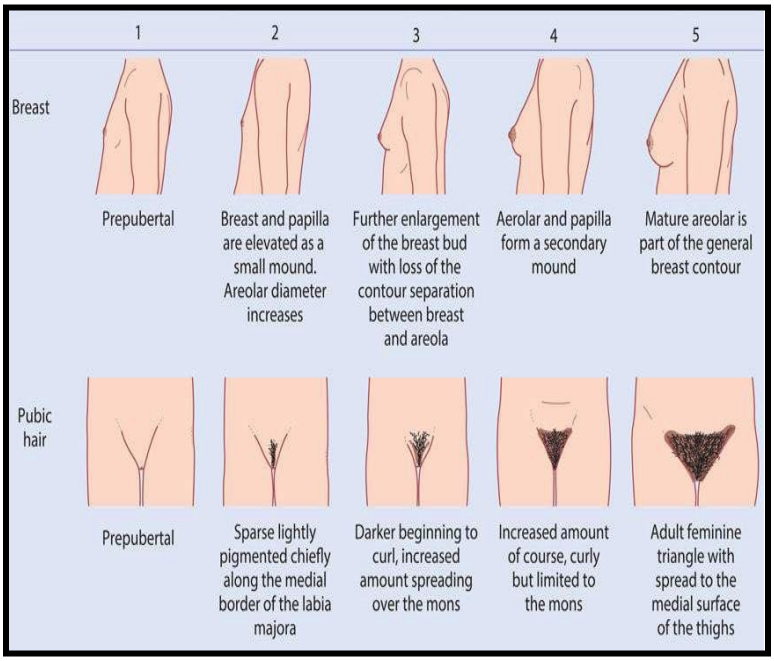
II- Developmental:

1- Growth spurt: the rate of body growth increases (the earliest sign of puberty) due to:

- Increased length of bone growth and widening of the pelvis.
- Increased body mass with deposition of fat in special sites of the body e.g., mainly in the buttocks and trunk "causing roundness and plumpness".

2- Thelarche: it means breast development in the following stages:

1- Elevated papilla only.
2- Breast bud.
3- Enlargement with roundness of lower margin.
4- Roundness of the areola.
5- Recession of the areola to the contour of the breast.



3- Adrenarche: appearance of pubic & axillary's hairs. Pubarche passes in the following stages:

1- No pubic hair.
2- Sparse hair mainly on labia majora.
3- Darkening hair and extending up and down.
4- Hair extending more to cover the mons veneris.
5- More extensions to cover the medial aspect of the thigh.

"Thelarche and adrenarche staging is called Tanner classification".

4-Menarche: it is the first spontaneous not induced cycle of menstruation.

III- Psychological:

1-The girl becomes shy. **2-**The girl refuses her parents' control.

3-Tendency to other sex

ABNORMAL PUBERTY

- Abnormal puberty may be delayed, asynchronous OR early "precocious puberty".

1- Delayed puberty:

Definition: the signs of puberty did not appear up to the age of 14 years.

Causes:

-The most common cause is gonadal dysgenesis e.g., turner syndrome, ovarian failure.

- Other causes:

i-Pan-hypopituitarism, hypothalamic failure. ii- Hypogondism or agonadism.

iii-Auto-immune diseases, iv- psychological stress, depression.

v-Constitutional.

2- Precocious puberty:

Definition: one or more of the signs of puberty appear before the age of 9 years.

Types:

1-Heterosexual: i.e., male like characters, caused by increased androgen as in:

a. Virilizing ovarian tumor **b.** Virilizing adrenal tumor

c. Congenital adrenal hyperpasia **d.** Exogenous androgen.

2-Isosexual: i.e., female like characters, which may be complete, or incomplete.

"In cases of complete precocious puberty all changes are present while in cases of incomplete type, there may be isolated single change as thelarche, adrenarche"

Causes of isosexual precocious puberty:

a- Central lesions "true precocious puberty": e.g.

1-Idiopathic or constitutional 90% (the most common).

2- Inflammatory. 3 - Traumatic. 4 - Brain tumors. 5 - Hydrocephalus.

b-Peripheral "false precocious puberty": e.g.

1-Estrogen secreting ovarian tumors. 2- Drugs e.g., Estrogen therapy.

3- Hypothyroidism.

Treatment:

i- Treatment of the cause.

ii- Anti- estrogens to avoid early metaphyseal closure: e.g. Progesterone, GnRH analogue, Androgens.

THE MENOPAUSE

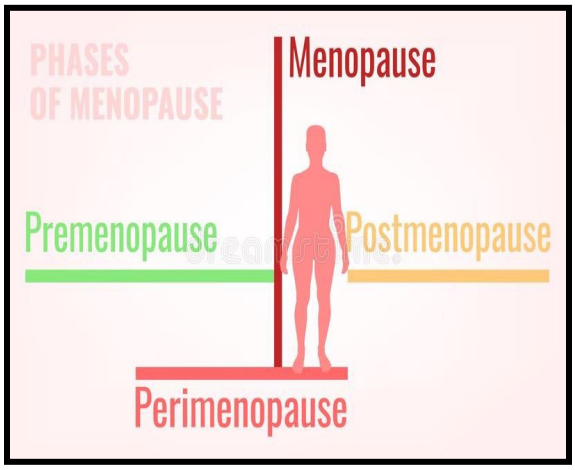
Definition:

It means permanent cessation of menstruation for more than (12 months) due to complete depletion of all primordial follicles from the ovaries.

Age of menopause: ranges from 45-55 years, the mean age is 51 years.

N.B:

Climacteric: period of life where females change from reproductive to non-reproductive states.
Pre-menopause: it is from 40-45 years.
Perimenopause: from 45-55 years (5 years before & 5 years after the average age of menopause).
Post-menopause: Period which begins one year after last period.



Types of menopause:

1- **Physiological menopause:**
   i.e., normal menopause.
2- **Pathological OR abnormal menopause:**
   1. Premature menopause.
   2. Artificial menopause "induced menopause".
   3. Menopausal syndrome.
   4. Delayed menopause

Normal changes at menopause:

I- Hormonal or endocrinal changes:

- **F.S.H** is **increased** (> 40 I.U) also; **LH increased** three folds.
  "Persistent high level of FSH more than 40 IU is diagnostic for menopause".
- **Estrogens:** decreased and the main type is E1 and **progesterone** is markedly decreased.
- **Androgen:** is relatively increased.

II- Genital changes:

1- **Vulva**: atrophy with narrowing of the introitus.
2-**Vagina**: decreased vaginal acidity, thinning, atrophy, loss of rugea & narrow vagina.
3-**Cervix:** atrophy, flushed with the vagina, increased incidence of ulcers and erosions.
4- **Uterus**: small, atrophic, and atrophied endometrium.
5- **Ovaries**: atrophic, small shrunken ovaries.
6- **Pelvic floor & ligaments:** become lax and increasing liability of genital prolapse.

III- General or systemic changes:

1- **Psychological:** anxiety, depression.

2-Breasts: atrophied so become shrunken and flat.
3- Bones: Calcium mobilization from it with subsequent osteoporosis.
4- Hair changes: weakness and grayness of hair.
5- Lipoprotein metabolism: increased LDL and decreased HDL.
6- Wrinkling of the skin and Vasomotor instability [see below].

ABNORMAL MENOPAUSE

1- PREMATURE MENOPAUSE.

- It means cessation of menstruation before the age of 40 years and may be due to:
  - Congenital cause e.g., ovarian hypoplasia and Mosaic Turner's syndrome.
  - Traumatic i.e., surgical removal of the ovary.
  - Inflammation leads to destruction of the ovary.
  - Neoplasm e.g. destructing ovarian tissues.
  - Dysfunction i.e., autoimmune disease.
  - Induced menopause.

2- ARTIFICIAL MENOPAUSE = induced menopause.

- It means destruction of the ovarian follicles before the age of the natural menopause e.g., as in bilateral oophorectomy, post irradiation, medical oophorectomy, administration of chemotherapy.

3- DELAYED MENOPAUSE.

- It means cessation of menstruation after the age of 55 years.
- Rarely, it may be constitutional cause, but commonly, it is due to causes of abnormal uterine bleeding and the patient is managed as abnormal uterine bleeding.

4- MENOPAUSAL SYNDROME.

Definition: It means exaggerated normal physiological changes at menopause to become symptomatizing.

Clinical assessment.The patient may present by one or more of the followings:

I- Psychological: anxiety, depression, irritability, insomnia, inability to concentrate, sense of end of life.

II- Vasomotor instability and hot flushes: V.D after V.C leading to hot flushes in the neck, chest resulting in sweating.

III- Osteoporosis: with more liability for fractures.

"Hot flushes and osteoporosis are considered as major symptoms".

IV-Cardio-vascular: Palpitation, chest pains, increased incidence of hypertension IHD.

V- GIT: decreased appetite, distension, dyspepsia & constipation.

VI- Genital: dyspareunia, due senile vaginitis, more genital displacements.

2. Investigations:

-Hormonal level particularly "FSH, LH and Estrogen".

-Bone densitometry. OR other investigations according to clinical presentation.

3. Treatment:

I- General lines:

1. Reassurance, tell the patient that "it is a change of life not the end of life".
2. Good diet and high tonics and regular exercise.

II-Medical:

1. Ca ++ and vitamin D (especially for the high-risk patient for osteoporosis.
2. Psychotherapy, sedatives, vitamins, minerals, B-blockers (to control flushes).

III- Hormonal: hormonal replacement therapy = H.R.T.

Indications:

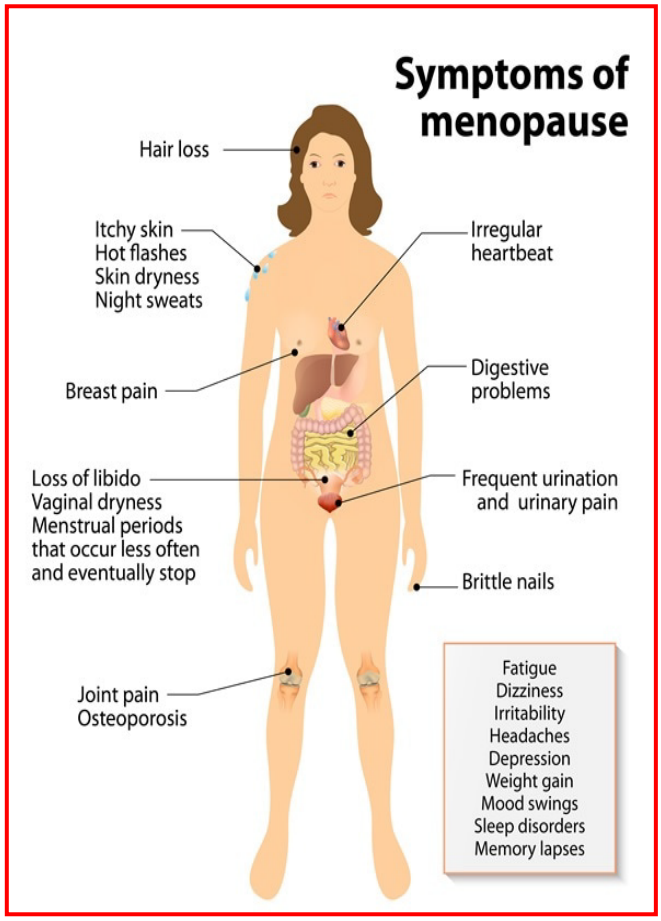
1. Proved hot flushes or osteoporosis.
2. Risky cases for IHD or osteoporosis.
3. Symptomatizing genital atrophy.
4. Proved psychological factor.
5. Premature and induced menopause.

Contraindications:

1. Undiagnosed abnormal uterine bleeding.
2. Family history of endometrial carcinoma.
3. Past or present history of cancer breast or family history of cancer breast.
4. Hyperbilirubinemia, liver diseases and gall stones.
5. Uncontrolled D.M, HTN and IHD.

Complications:

1. Carcinogenicity (it is decreased with the new regimens).
2. Defective liver metabolism.
3. Nausea & vomiting.
4. Abnormal uterine bleeding.
5. Increased risk of thromboembolic diseases.
6. Those with fibroids, endometriosis or severe migraine.



Regimen for HRT:

1. Continuous unopposed estrogen OR Progesterone.
2. Continuous combined estrogen and progesterone.
3. Cyclic unopposed estrogen OR progesterone.
4. Cyclic combined estrogen and progesterone [best to be used].
5. Cyclic androgen OR Tamoxifen "selective estrogen receptor modulators".
6. Nonbleeding agents = Tibolone.
7. Phytoestrogen = Klima Dynon.
8. GnRH analogue.

ABNORMAL MENSTRUATION

Abnormal menstruation includes:

1- Dysmenorrhea. **2-** Amenorrhea. **3-** Abnormal uterine bleeding.

DYSMENORRHEA

Definition: it is painful menstruation that interferes with patient's daily activity.

Types:

1- Primary dysmenorrhea.
2- Secondary dysmenorrhea.
3- Special types.
4- Premenstrual tension syndrome.

PRIMARY DYSMENORRHEA

- It is a painful menstruation with no obvious gross pelvic lesions.

ETIOLOGY: the actual cause is unknown, but the following theories are postulated.

1. **Prostaglandin's theories:** increase in PGs PGE2 and PGF2α → uterine ischemia and contraction → pain.
2. **Physiological theory:** with loss of uterine polarity.
3. **Hormonal theory:** with increased progesterone, E_2/P ratio, or vasopressin and oxytocin.
4. **Neurogenic theory:** with diminished pain threshold.
5. **Anatomical theory:** as with congenital hypoplastic uterus, or stenosed cx.
6. **Psychological and behavioral theory:** the patient refuses normal physiology.

DIAGNOSIS:

- The disease occurs usually in teenagers 14-24 years [nulliparous dysmenorrhea].

- **There i**s colicky sharp pain, in the supra-pubic area, started 1-2 hours before the flow, lasts for 1-2 days, radiated to the upper inner aspect of the thigh, may be associated with nausea and vomiting, headache, fatigue, dizziness and fainting attacks.

TREATMENT

1. **General lines:** health education, psychotherapy, reassurance, exercise.
2. **Medical lines:**
   - Nonspecific: as antispasmodics.
   - Specific: as **NSAIDs** [Mefenamic acid, indomethacin] OR **COCs**
3. **Surgical lines:** rarely needed e.g.
   - Acupuncture techniques.
   - Dilatation of the cervix up to no 14 Hegar's dilator.
   - Presacral neurectomy.
   - Laparoscopic uterosacral nerve ablation " LUNA".

SECONDARY "CONGESTIVE" DYSMENORRHEA

ETIOLOGY: All causes that lead to pelvic congestion e.g.:

1. Prolonged use of IUD.
2. Pelvic endometriosis and adenomyosis.
3. Chronic PID.
4. Uterine fibroids.
5. Displacements.
6. Chronic constipation.

DIAGNOSIS:

Symptoms:

- Usually occurs in multi-parous patients with history suspecting the cause.
- **The pain is** dull aching diffuse starting 1-2 days before the onset of the flow, then decreases once the flow stops and radiated to the back.
- In cases of **endometriosis**, the pain is **crescendo** type of dysmenorrhea.

Signs: pathology is detected.

TREATMENT:

1. Treatment of the cause.
2. Symptomatic treatment e.g., analgesics, antispasmodics, measures to decrease pelvic congestion.

SPECIAL TYPES OF DYSMENORRHEA.

1- Dysmenorrhea due to blood clots.
2- Dysmenorrhea with foreign body e.g., IUD treated by removal of IUD.
3- Membranous dysmenorrhea: with increased level or sensitivity to progesterone → thick secretory endometrium and shed as a cast → severe pain.
Treatment: dilatation and curettage, analgesics and antispasmodics.
4- Ovarian dysmenorrhea: it may be caused by **o**varian congestion OR ovarian apoplexy. There is sudden acute abdominal pain may be associated with fainting attacks due to massive intra-peritoneal hemorrhage.
Treatment: bed rest, analgesics, if failed OR severe hemorrhage; oophorectomy is done.

PREMENSTRUAL TENSION SYNDROME

Definition:

- It is a syndrome of physical, psychological and behavioral changes that occurs before menstruation, late in the luteal phase, and improved with the onset of the flow.

Causes: the actual cause is unknown, so different theories are postulated.

1- **Neurogenic theory:** with deficiency of B-endorphins, vitamin B complex and serotonin.
2- Hormonal theory: with **hi**gh E_2/P ratio, prolactin or aldosterone levels.
3- **Prostaglandin's theory.**
4- **Nutritional theory:** with deficiency of essential fatty acids, CHO, certain minerals.

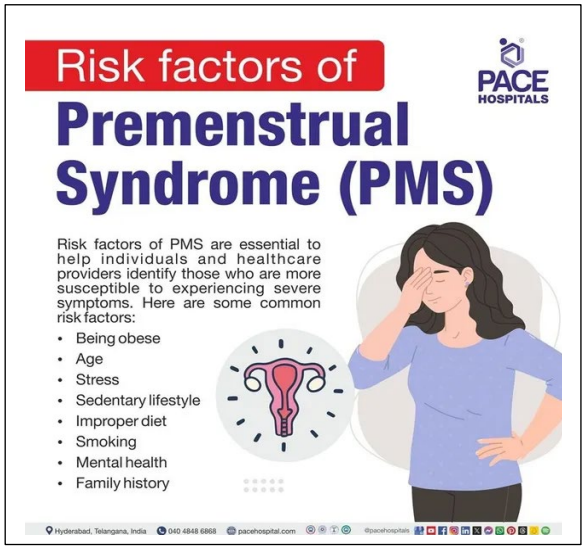
Diagnosis:

1- Physical symptoms:

- Facial and peripheral edema due to salt and water retention.
- Breast tension.
- Abdominal distension, bloating, or weight gain.
- Headache.

2- Psychological and behavioral symptoms.

- Depression, anxiety, irritability.
- Sleep disturbances, lack of concentration.
- Craving certain types of food.
- Suicidal attempts and criminal behavior.



The main symptoms are carbohydrate craving, salt and water retention, anxiety, depression".

Treatment:

Usually general and medical lines e.g.

1-Psychotherapy and reassurance to alleviate tension.

2-Diuretics. 3- COCs 4-Bromocriptine "paroldel".

5-Tranquilizers and sedatives. 6-Serotonin reuptake inhibitors.

7-Vit B complex. 8-Primrose oil.

AMENORRHEA

Definition:

Amenorrhea means absence or cessation of menstruation "it is a symptom not a disease".

Classification:

1- According to the time of onset:

Primary amenorrhea: *it is absence of menses at the age of 15 years in the presence of normal growth & secondary sexual characteristics., OR at age of 13 years, if no menses have occurred and there is complete absence of secondary sexual characteristics.*

Secondary amenorrhea: it is cessation of menstruation for 3 successive cycles (previously regular menses), or 6 months (previous irregular menses).

2- According to the cause: Physiological and pathological amenorrhea.

3- According to the actual occurrence of menstruation:

False amenorrhea = crypto menorrhea i.e., menstruation have occurred but there is outflow obstruction.

True amenorrhea: menstruation actually not happened (i.e., axis and uterine defects).

Physiological Amenoirrhea:

Amenorrhea occurs during certain times of female's life and considered normal e.g.:

1. Before puberty: as hypothalamo-pituitary ovarian axis is not yet active.

2. Adolescent period: for 1-2 years following menarche as the axis is not well established.

3. During pregnancy: due to continuous production of estrogen and progesterone. "Pregnancy is the commonest cause of secondary amenorrhea".

4. During lactation: due to continuous production of prolactin that inhibit the axis.

5. Pre-menopause: due to exhaustion of the axis and start depletion of the ovarian follicles.

6. Post-menopause: due to depletion of all ovarian follicles.

PATHOLOGICAL AMENORRHEA

- According to the level of the cause, pathological amenorrhea may be one of the following categories:

1- Hypogonadotropic amenorrhea: due to actual central causes (FSH and LH decreased).

2- Hypergonadotropic amenorrhea: occurs with ovarian causes **except PCOS**, in this case, FSH and LH are elevated, secondary to ovarian failure.

3- EU gonadotrophic amenorrhea: in which FSH and LH are within normal, occurs with uterine and outflow obstruction causes and PCOS.

I- HYPO-GONADOTROPHIC AMENORRHEA:

-The cause is either deficiency in pulsatile release of GnRh or pituitary FSH and LH.

-The causes may be:

1. General causes:

- Chronic debilitating diseases: diabetes, hypertension, anemia, T.B.
- Obesity.
- Hyperthyroidism and hypothyroidism.
- Hypercorticism "Cushing's disease" and hypocortisism "Adidsonian's disease".

2. Cerebral causes: the most important is psychological factor: e.g.

- Emotional upsets, psychosis and stress.
- Pseudocyesis [false pregnancy].
- Anorexia nervosa OR bulimia nervosa.

3. Hypothalamic causes:

1. Congenital:
   - Kalman syndrome [amenorrhea and anosmia]
   - Frohlich's syndrome [amenorrhea and obesity]
   - Laurence-Moon-Biedl syndrome [amenorrhea, obesity, mental retardation, blindness, polydactyl or syndactyl]
2. Traumatic: fracture base of the skull.
3. Inflammatory: e.g., meningitis and meningo-encephalitis.
4. Neoplastic.
5. Post-irradiation on the brain.
6. Dysfunction:
   - Exercise- related amenorrhea [= athletic amenorrhea].
   - Post-pill amenorrhea: after use of combined pills or progesterone only contraception.
   - Drugs: e.g., sedatives, and ganglion blockers.

4.Pituitary causes:

1- Congenital: Levi-Lorain syndrome "pituitary infantilism".

2- Traumatic and inflammatory as before.

3- Neoplastic: any type of pituitary adenoma or hyperplasia can cause amenorrhea.

-If the tumor from gonadotrophin secreting cells, it causes down regulation.

-If from other cells, it causes to pressure atrophy on gonadotrophin secreting cells.

-If prolactinoma, it is due to high level of prolactin.

4- Dysfunction:

i. Vascular syndromes: Sheehan's syndrome [post-partum hemorrhage] and Simmonds disease [postpartum sepsis]

ii. Empty Sella syndrome: due to pressure atrophy of the gland by accumulating CSF.

iii. Hyperprolactinemia syndrome: see later.

II- HYPER-GONADOTROPHIC AMENORRHEA [ovarian amenorrhea]:

- It is due to ovarian causes that include:

1. Congenital: aplasia, hypoplasia and all types of dysplasia [see embryology].
2. Traumatic: surgical removal.
3. Inflammatory: mumps, and tubo-ovarian infection.
4. Neoplastic: ovarian tumors or 2dries causing destruction of normal ovarian tissues.
5. Dysfunction: e.g., premature ovarian failure, ovarian enzymatic defect and perimenopause.

- *PCOS is a common cause of ovarian amenorrhea BUT eugonadotrophic type.*

III-EUGONADOTROPHIC AMENORRHEA:

- The causes include:

1. Disorders of androgen excess: as in polycystic ovarian syndrome [=PCOS].
2. Uterine causes:
   - Congenital: Mullerian duct agenesis, hypoplasia, or congenital Asherman syndrome.
   - Traumatic: Asherman's syndrome [intrauterine adhesions].
   - Inflammatory: due to destruction of the basal layer or presence of adhesion.
   - Dysfunction: end organ resistance due to endometrial receptor defect.
3. Outflow tract causes: menstruation occurred but finds NO way to pass outside e.g.
   - Congenital: cervical stenosis, complete transverse vaginal septum, and imperforate hymen.
   - Acquired: acquired vaginal atresia, cervical stenosis.

Asherman's Syndrome

Definition: it is adhesion between the anterior and posterior uterine walls.

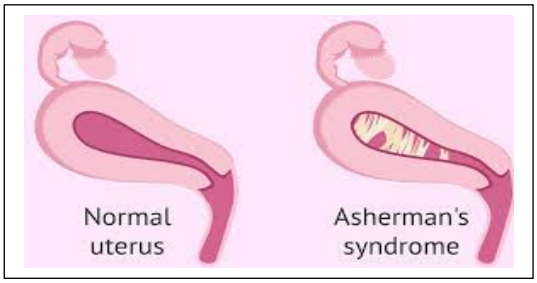
Etiology: destruction of the basal endometrium as following excessive curettage, CS or myomectomy or post-inflammatory.

Diagnosis:

- History of the cause, amenorrhea, oligo-hypo-menorrhea, infertility, or recurrent abortion.
- Local exam reveals difficult uterine sounding and irregular uterine cavities.
- Investigations mainly used for diagnosis are HSG and hysteroscope.

Treatment:

1. Prophylactic: by avoidance of the causes.
2. Active treatment:
   - *Medical:* by fibrolytic agents and antibiotics.
   - *Surgical:*
     - Cutting the adhesion, via hysteroscopy
     - Insertion of IUD for 2 cycles to prevent re-adhesion
     - Estrogen for 3-4 weeks to help epithelialization of the endometrium.



EVALUATION OF A CASE OF AMENORRHEA

1- Clinical diagnosis

A. Symptoms: history may suspect the cause e.g.:

- **Age**: physiological amenorrhea before puberty and after menopause.
- **Marital status**: pregnancy is the commonest cause of secondary amenorrhea.
- **Occupation**: as overwork and stress may cause amenorrhea.
- **Eating habits**: over feeding or under feeding.
- **Complaint**: any associated symptoms that may determine the site of the lesion
- **Menstrual history**: for determination of the duration and type of amenorrhea.
- **Obstetric history**: to exclude postpartum amenorrhea.
- **Past history** and **family history**: for medical diseases, surgical operation…etc.

B. Signs:

- General: for detection or exclusion of general causes as body weight, presence or absence of cachexia, BMI, signs of puberty, breast examination.

- Abdominal examination: to discover any masses pregnancy, ovarian tumors, abnormal distribution of hair.

- Local examination: a cause may be detected as, imperforate hymen, complete transverse vaginal septum, cervical stenosis or Asherman syndrome.

2- Investigations

1- Lab work up:

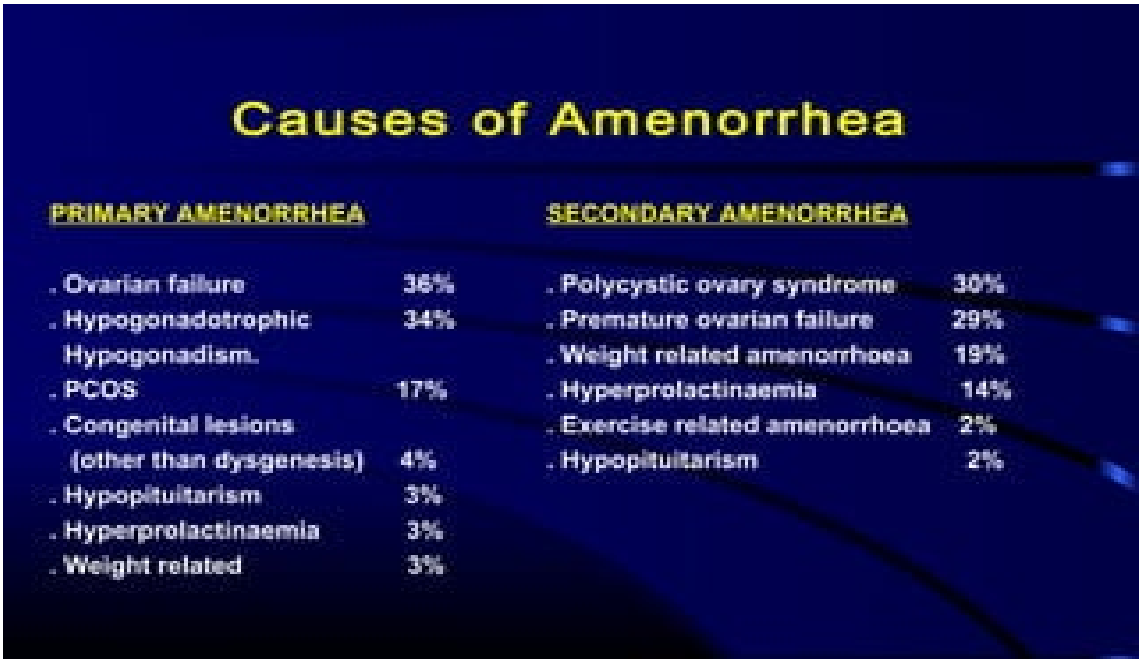
- Serum HCG, and pregnancy test.
- FSH and LH levels "to identify hypogonadotropic or hypergonadotropic type".
- Prolactin level.
- Thyroid function tests, renal and liver function tests.
- karyotype & chromosomal study to exclude gonadal failure or dysgenesis.

2- Radiological:

- X-ray on the hand and wrist to check bone age.
- C.T and MRI on the brain to exclude central cause.

3- Endoscopy:

- Ophthalmoscope: for visual field defects in CNS tumors.
- Laparoscopy for evaluati0on of the gonads and uterus.



HOW TO PROCEED IN DIAGNOSIS OF PRIMARY AMENORRHEA

Public and maxillary hair	Breast	Uterus	The result
Present	Present	Present	The case may be: 1- Cryptoemenorrhea excluded by local examination. 2- Central or hormonal defect start investigations by prolactin level as discussed below.
Absent	Absent	Absent	5-α reductase deficiency "abnormal androgen synthesis".
Present	Absent	Present	- Turner's syndrome: excluded by chromosomal analysis. - Savage syndrome: differentiated by chromosomal analysis and ovarian biopsy.
Absent	Present	Absent	Testicular feminization syndrome.
Present	Present	Absent	Mullarian duct agenesis

- First, exclude **pregnancy.** Exclude outflow obstruction ……
-Then search for presence or absence of secondary sex (**breast**) characters and secondary sex organ (**uterus**).

-If both **uterus** & **breast** are present we proceed as follows:

Do first *prolactin* level: → If high → do TSH → if high = hypothyroidism

If normal, do brain X –ray, CT, OR MRI *to exclude brain lesion and search for the cause of hyperprolactinemia.*

If normal:

Do progesterone withdrawal test:

- **If +ve** vaginal bleeding it is a case of "ovarian cause" due to "luteal phase defect"
- **If –ve bleeding** ---→ **give estrogen and progesterone** →
- **If there is NO bleeding** it is end organ defect "i.e., uterine failure"
- If this is +ve bleeding it is due to central causes, and in this case ----→

Do FSH level:

- If high, it is a case of gonadal failure.
- If low, it is pituitary or hypothalamic failure so→ Do **GnRH test:** **give external GnRH→**

- If it is followed by an elevated level of FSH it means "hypothalamic failure."
- If not followed by increased FSH level it is due to "pituitary failure."

In cases of secondary amenorrhea: do first pregnancy test "pregnancy is the commonest cause of secondary amenorrhea", if –ve proceed as primary amenorrhea starting by prolactin level.

TREATMENT OF AMENORRHEA

1. Treatment of general causes: proper control of hypothyroidism, hyperthyroidism, anemia, obesity, psychological troubles, chronic debilitating diseases.

2. Hypogonadotophic menorrhea:

- Treatment of the cause e.g., treatment of infection, surgery for tumors or radiotherapy.
- Hormone replacement therapy: e.g.
    - If the patient needs pregnancy, give GnRH or gonadotrophins.
    - If the patient is not needing pregnancy COCs may be given to maintain secondary sex characters and menstruation.

3- Treatment of hyper-gonadotrophic amenorrhea:

- Induction of ovulation in PCOS
- Hormone replacement therapy: by cyclic or continuous estrogen and progesterone.
- Removal of any dysgenetic gonads to avoid their malignant potential.

4- Treatment of euogonadotrophic amenorrhea:

1- Congenital causes:

- Hypoplastic uterus: give estrogen.
- Congenital cervical stenosis: treatment by dilatation.
- Vaginal septum: surgical removal.
- Imperforate hymen: do cruciate or crescent shaped incision.

2- Traumatic "Asherman syndrome": treated as before.

3- Inflammatory: treatment of inflammation.

HYPERPROLACTINAEMIA

Definition: it is elevation of probating level more than 20 ng/dl.

Causes: [PDEL TSH]

1- Physiological: lactation, pregnancy, stress, sleep, second half of the cycle.

2- Pathological:

- Drugs: antihypertensive, antipsychotic, H_2 blockers, opioids…etc
- Ectopic release from tumors anywhere.
- Local breast lesion: chest wall burns, wound scar, herpes zoster.
- Tumors of pituitary.
- Syndromes: Chiari fromel syndrome, Del Castello syndrome, Frobe' syndrome.
- Hypothyroidism.

Diagnosis:

1- Symptoms:

- The patient may complain of galactorrhea, amenorrhea, oligo-hypomenorrhea, or infertility.
- OR history-suspecting AE: headache, blurring of vision with pituitary tumors…etc.

2- Signs: there may be nothing characteristic, or breast lesion may be found.

3- Investigations: Serum prolactin, TSH OR brain CT and MRI to exclude adenoma.

Treatment:

1- Medical treatment: Dopamine agonist e.g., Bromocriptine [Parlodel] OR Cabergoline.

2- Surgical treatment and radiotherapy in micro and macroadenoma.

Causes of Amenorrhea + Galactorrhea (galactorrhea- amenorrhea syndromes) :

1. **Lactational amenorrhea**
2. **Chiari-Frommel syndrome**
3. **Del Castello syndrome**
4. **Pituitary tumors**
5. **Drugs (antidepressants)**

Causes of Amenorrhea + Hirsutism :

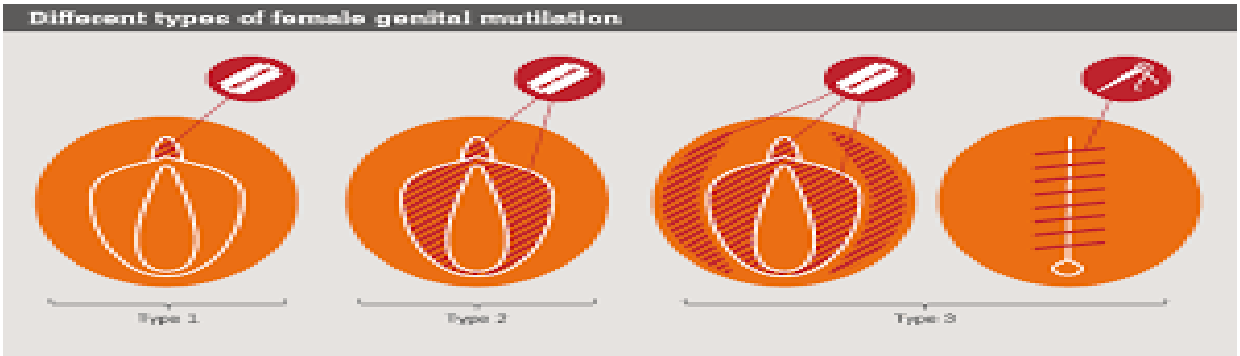
1. **PCOS**
2. **Ovarian tumors**

Female genital mutilation (FGM)

Definition: These are procedures involving partial or total removal of female external genital organs for cultural or any other non-medical reasons.

- It is primarily African phenomenon and virtually unknown in other parts of the world except emigrants from African countries.
- Some people believe that the practice has religious support BUT it was practiced in Africa before Islam & isn't requirement in Quran. Moreover, it isn't known or practiced in Saudi Arabia & many Islamic countries.
- Although Christians in Egypt practice this behavior but on non-religious basis.
- The year 2000 Egypt Demographic & Health Survey showed about 97% of ever-married women are circumcised.

WHO Classification of FGM:



Type 1:	This is the partial or total removal of the clitoral glans (the external and visible part of the clitoris, which is a sensitive part of the female genitals), and/or the prepuce/clitoral hood (the fold of skin surrounding the clitoral glans).
Type 2:	This is the partial or total removal of the clitoral glans and the labia minora (the inner folds of the vulva), with or without removal of the labia majora (the outer folds of skin of the vulva).
Type 3:	Also known as infibulation, this is the narrowing of the vaginal opening through the creation of a covering seal. The seal is formed by cutting and repositioning the labia minora, or labia majora, sometimes through stitching, with or without removal of the clitoral prepuce/clitoral hood and glans.
Type 4:	This includes all other harmful procedures to the female genitalia for non-medical purposes, e.g., pricking, piercing, incising, scraping and cauterizing the genital area.

COMPICATIONS OF FGM

Immediate	Late (remote)
Immediate complications of FGM can include: 1. severe pain 2. excessive bleeding (hemorrhage) 3. genital tissue swelling 4. fever 5. infections e.g., tetanus 6. urinary problems 7. wound healing problems 8. injury to surrounding genital tissue 9. shock 10. death.	Long-term complications can include: 1-urinary problems (painful urination, urinary tract infections); 2-vaginal problems (discharge, itching, bacterial vaginosis and other infections); 3-menstrual problems (painful menstruations, difficulty in passing menstrual blood, etc.); 4-scar tissue and keloid. 5-sexual problems (pain during intercourse, decreased satisfaction, etc.); 6-increased risk of childbirth complications (difficult delivery, excessive bleeding, caesarean section, need to resuscitate the baby, etc.) and newborn deaths. 7-need for later surgeries: women with Type 3 might require de- infibulation (opening the infibulated scar to allow for sexual intercourse and childbirth. 8-psychological problems (depression, anxiety, post-traumatic stress disorder, low self-esteem, etc.).

Polycystic Ovarian Syndrome [PCOS]

Definition: it is a syndrome characterized by presence of ≥ 2 of the following 3 criteria:

1) Oligo-ovulation or anovulation.
2) Clinical & biochemical signs of hyper-androgenism.
3) PCO morphology on ultrasound.

Etiology: Still unclear (some consider it as X-linked dominant disease).

Pathophysiology:

- There is ↑↑ production of ovarian androgens, which is due to hyperinsulinemia & insulin resistance.
- Some of ovarian androgens are peripherally converted to E1 → hyperestrogenic state → suppression of FSH secretion & stimulation of LH secretion.
- Low level of FSH is enough to allow follicular growth but not maturation & ovulation.
- High level of LH stimulates theca cells to secrete androgens which further inhibit follicular maturation & initiate a vicious cycle.

Diagnosis:

A) Clinical:

1- **Oligomenorrhea or amenorrhea.**
2- **Hyper-androgenism.**
3- **Others:** Obesity, AUB, infertility (due to chronic anovulation) & recurrent abortion.

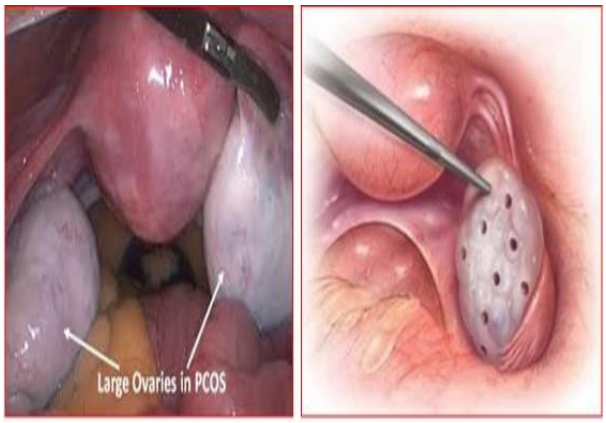
B) Ultrasound criteria: Rotterdam criteria (2003). **[Old name necklace appearance]**

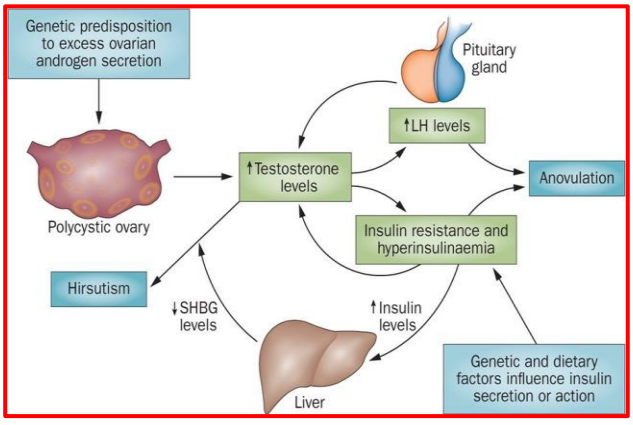
1- Presence of ≥ 12 follicles in each ovary measuring 2-9 mm in diameter.
2- ↑↑ ovarian volume (> 10 cm^3).

Only one ovary fitting the above criteria is sufficient to diagnose PCO.

C) Laboratory criteria:

1- ↑↑ **LH/FSH ratio:** 2/1 or 3/1 (normally it is 1/1).
2- ↑↑ **androgen levels:** especially testosterone & androstenedione.
3- ↑↑ **estrogen level:** mainly E1.
4- ↑↑ **serum insulin level & ↑↑ insulin resistance.**
5- ↓↓ **sex hormone binding globulin (SHBG) level.**





Treatment: it depends on the main complaint of the patient.

A) Menstrual disturbances:

1) **Cyclic gestagen therapy:** to induce withdrawal bleeding
2) **COCs:** can be used for limited time in cases seeking contraception

B) Infertility:

1) **Weight reduction** (at least 5-10%): the first line in obese or overweight patients.
2) **Clomiphene citrate** (for 6-12 months): the standard treatment for induction of ovulation.
3) **Gonadotropin therapy (HMG or FSH):** with clomiphene citrate resistance or failure.
4) **Laparoscopic ovarian drilling.**
5) **Insulin reducing agents (Metformin):** in patients with glucose intolerance.
6) **Assisted reproductive technology (ART):** if the above methods failed.

C) Hyperandrogenism: as in hirsutism.

D) Obesity: Weight reduction & insulin reducing agents.

Long-term sequels:

A) Common long-term sequels:

1) Type 2 DM (NIDDM): due to glucose intolerance & hyperinsulinemia.
2) Dyslipidemia.
3) Endometrial hyperplasia & carcinoma: Due to hyperestrogenic state.

B) Possible long-term sequels:

1) Metabolic: Obesity, metabolic syndrome, gall bladder diseases.
2) HTN & arterial diseases.
3) Depression & anxiety & loss of self-esteem.
4) Epilepsy.
5) Pregnancy complications: Gestational DM & gestational HTN, PET.
6) Recurrent abortion: Due to CL dysfunction.
7) Ovarian cancer.
8) Cancer breast.

PART TWO

- Abnormal uterine bleeding
- Leiomyoma
- Endometriosis
- Adenomyosis
- Infertility
- Contraception

ABNORMAL UTERINE BLEEDING

Definition: ***Abnormal uterine bleeding (AUB)*** is any deviation of menstruation from normal "either in amount or duration, whether by increase or decrease".

- Suggested (FIGO) normal limits of uterine bleeding in mid-reproductive years:

Category	Normal	Abnormal	☑
Frequency	Absent (no periods or bleeding) = amenorrhea		☐
	Frequent (<24 days)		☐
	Normal (24 to 38 days)		☐
	Infrequent (>38 days)		☐
Duration	Prolonged (>8 days)		☐
	Normal (up to 8 days)		☐
Regularity	Regular variation (shortest to longest ≤ 9 days)		☐
	Irregular (shortest to longest 10+ days)		☐
Flow volume	Heavy		☐
	Normal		☐
	Light		☐

Intermenstrual Bleeding (IMB) Bleeding between cyclically regular onset of menses	None		☐
	Random		☐
	Cyclic (Predictable)	Early Cycle	☐
		Mid Cycle	☐
		Late Cycle	☐

Unscheduled Bleeding on Hormone Medication (eg Birth Control Pills, Rings or Patches)	Not Applicable (not on hormone medication)	☐
	None (on hormone medication)	☐
	Present	☐

Definitions and clinical types:

A- OLD CLASSIFICATION FOR TERMINOLOGY

-Cyclic bleedings:

1- Menorrhagia: increase amount > 80mm^3 or duration > 7 days **[heavy menstrual bleeding].**
2- Polymenorrhagia: too frequent menstruation recurring in less than 21 days [too short cycles].
3- Poly menorrhagia: combination of both types mentioned above.

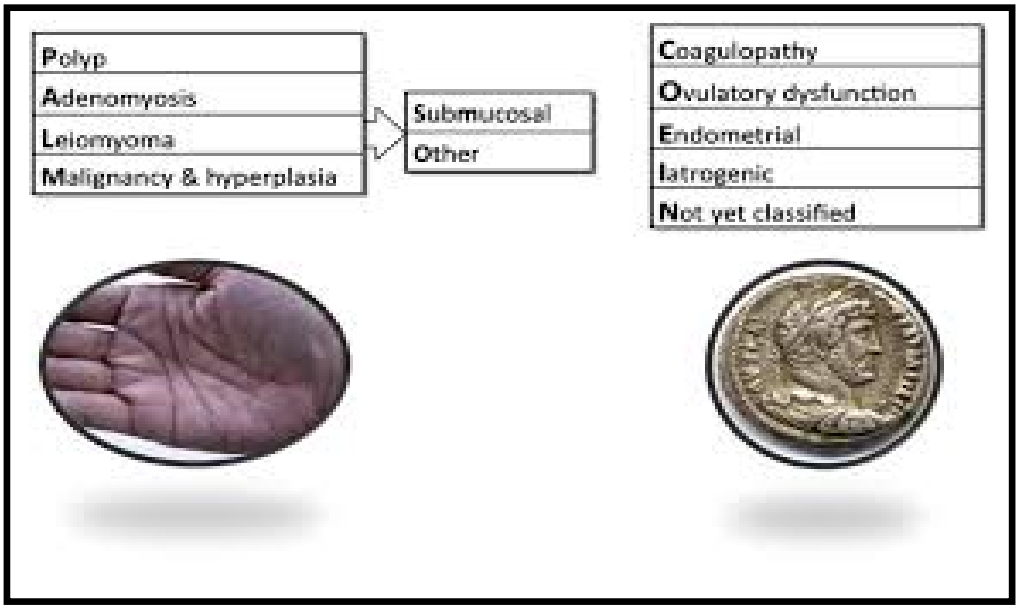
-**Acyclic bleeding:**

1- Metrorrhagia: irregular uterine bleeding not related to menstruation.
2- Meno- metrorrhagia: Menorrhagia + irregular intermenstrual bleeding.
3- Intermenstrual bleeding: contact bleeding, mid-cyclic spotting.

B-MODERN FIGO-Classification system [PALM-COEIN]

for uterine bleeding in mid reproductive years:

PALM	**COIEN**
1-Polyp **2- A**denomyosis **3 -L**eiomyoma **4-M**alignancy **(**or hyperplasia**)**	**1- C**oagulopathy **2- O**varian **3- I**atrogenic **4 - E**ndometrial **5- N**on otherwise specified



ETIOLOGY OF ABNORMAL UTERINE BLEEDING (AUB):

-It may by heavy menstrual bleeding (AUB/HMB) or intermenstrual bleeding (AUB/IMB)
-Classified into:

A- Structural causes (PALM):

1-Polyp (AUB-P) 2- Adenomyosis (AUB-A)
3-Leiomyoma (AUB-L): submucous leiomyoma (AUB-LSM), other leiomyomas (AUB-Lo)
4-Malignasncy and hyperplasia (AUB-M).

B- Nonstructural causes (COEIN):

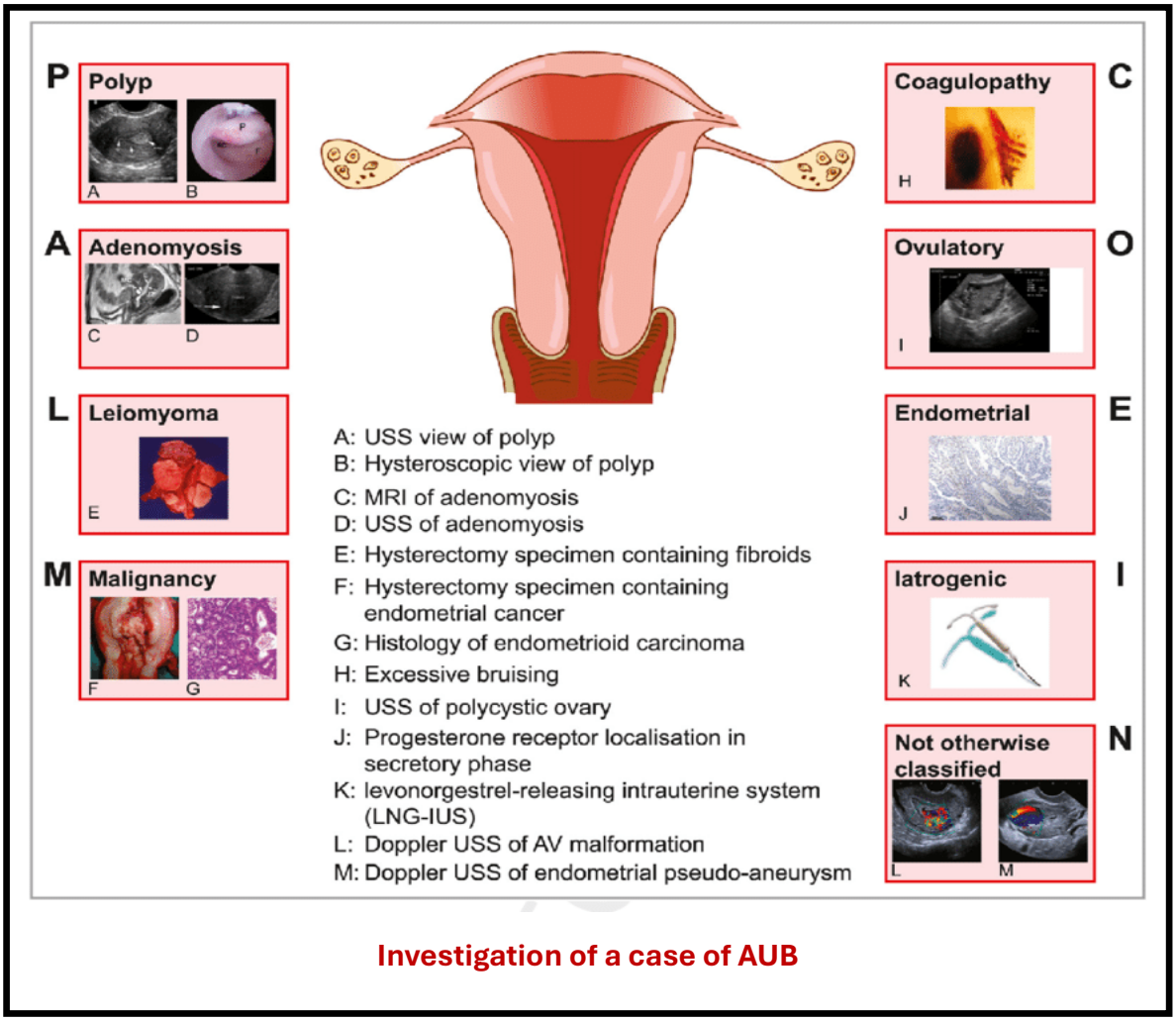
1-Coagulopathy (AUB-C) 2- Ovulatory (AUB-O) 3-Endometrial (AUB-E)
4- Iatrogenic (AUB-I 5-Not yet classified (AUB-N).

Clinical Screening for an Underlying Disorder of Hemostasis in the Patient with Excessive Menstrual Bleeding:

Initial screening for an underlying disorder of hemostasis in patients with excessive menstrual bleeding should be structured by the medical history. A positive screening result comprises the following circumstances:

1-Heavy menstrual bleeding since menarche
2-One of the following conditions:
a). Postpartum hemorrhage b). Surgery-related bleeding
c). Bleeding associated with dental work

3-Two or more of the following conditions:
a) Bruising, one to two times per month b). Epistaxis, one to two times per month
c). Frequent gum bleeding. d). Family history of bleeding symptoms



Investigation of a case of AUB

Laboratory testing for patients with AUB:

A- **Initial tests**: complete blood count, blood type & cross match, pregnancy test

B- **Initial laboratory evaluation for disorders of hemostasis:** PTT, PT, APTT, Fibrinogen.

C- **Initial testing for von Willebrand's disease:** von Willebrand factor antigen, factor VIII, Ristocetin cofactor assay.

D- **Other tests to consider:** TSH, serum iron, total iron binding capacity, ferritin, liver function, and chlamydia trachomatis.

Treatment of cases of AUB:

-Essentially treatment of the cause Especially for **chronic causes**. Treatment may be medical or surgical.

ACUTE cases of AUB treatment:

-There are 2 main goals for treatment of acute cases of AUB:

1- To control the current episode of heavy bleeding
2- To reduce menstrual blood loss in the subsequent cycles.

-Treatment is mainly medical, surgery is rarely indicated in such cases.

Medical treatment Regimens:

1- Conjugated equine estrogen: 25 mg IV every 4-6 hours for 24 hours.
2-Combined oral contraceptives: monophasic COCs that contain 35 microgram ethinyl estradiol taken 3 times per day for 7 days.
3-Medroxy progesterone acetate 20 mg orally 3 times per day for 7 days
4-Tranexamic Acid: 1.3 g (orally) or 10 mg/kg / IV (maximum 600 mg dose); 3 times daily for 5 days.

CHRONIC CASES OF AUB

A- Medical treatment:
1- Continuation of acute medical treatment
2- LARC (Long-Acting Reversible Contraception): e.g. levonorgestrel IUD, etonogestrel arm implants)
B- **Surgical treatment**:
1- Myomectomy (hysteroscopic, laparoscopic, or open surgery)
2- Hysterectomy (vaginal, laparoscopic, or open surgery, or open surgery:
3- Endometrial ablation: hysteroscopic (not further recommended by most gynecologists)

Note:
- D&C (dilation & curettage) is required in most cases of AUB for diagnosis of histopathological type of the endometrium as well as it reduces bleeding in acute cases. In chronic cases it is replaced by office endometrial biopsy.
- The details of these treatments will be mentioned under each subject later.

ABNORMAL UTERINE BLEEDING TREATMENT

THE CURB SIDERS INTERNAL MEDICINE

Acute

Treatment can be based on contraindications, but also patient preference of **DAILY** vs. **EPISODIC** medication

- **Progesterone** alone (medroxyprogesterone, norethindrone, drospirenone)
- Combined estrogen-progesterone **OCP**

- **Tranexamic Acid** (3 times daily for 5 days)

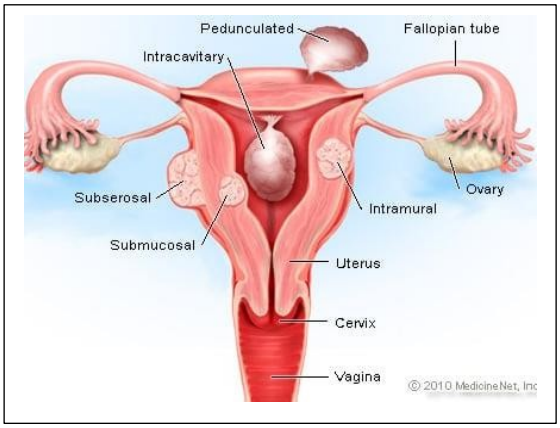
Chronic

- **Continuation** of acute treatments
- **LARC** (levonorgestrel IUD or etonogestrel arm implant)

- **Surgical** (endometrial ablation, myomectomy, hysterectomy)

UTERINE LEIOMYOMA (FIBROID)

Pathology



Definition: It is a benign tumor arising from smooth muscles of the uterus and associated fibrous tissues.

Incidence:

-It is the commonest uterine tumor as it affects 20-30% of women above 30 years.".

-It is common in nullipara or women of low parity and in black races.

Etiology and Predisposing Factors:

The cause is unknown, but the tumor is estrogen dependent because:

- It is common in nullipara & women of low parity.
- It does not appear before puberty and never arises de novo after menopause.
- It increases in size in pregnancy & regresses after labor.
- Produced experimentally in animals by estrogen.
- The hyper-estrogenic states are:
  1- Chronic anovulation as PCOS. 2- Estrogen producing ovarian tumors.
  3- Liver cell failure, obesity, exogenous estrogen administration as in HRT.
- There is genetic and familial tendency.

N/E:

Sites: **A- Corporeal (95 %)** usually multiple:

1. Interstitial fibroid: all fibroids start interstitial and then turn submucous or subserous.

2. Submucous fibroid: it projects into the uterine cavity covered with endometrium and may become pedunculated forming a submucous fibroid polyp.

3. Sub-serous fibroid: it projects on the outer surface, covered with peritoneum and may become pedunculated forming a sub-serous fibroid polyp.

B. Cervical (4%) usually single and may be

1. In the portio-vaginalis: interstitial forming barrel shaped cervix OR cervical polyp.

2. In the supra-vaginal cervix:

- Interstitial: anterior wall, posterior wall or lateral wall fibroid.
- Pseudo broad ligaments fibroid.

C. Ligaments (1%): it may

- Round ligament. - Broad ligament "true broad ligament fibroid".

- **Size:** the size varies from a small seedling up to a large tumor filling the abdominal cavity.
- **Shape:** tumor may be spherical OR polypoidal while the uterus may be symmetrically OR asymmetrically enlarged.
- **Surface:** the tumor is smooth while of the uterus may be smooth with submucous and interstitial types and is bossy irregular with subserous type.
- **Surrounding:** the surrounding muscle fibers are compressed to form a false capsule.
- **Consistency:** usually it is firm in consistency.

  -It becomes soft during pregnancy or with hyaline degeneration.
  -It becomes hard with calcification.
- **Cut section:**

  -It shows a whorl appearance & is paler than the surrounding.
  -It has a false capsule formed of the compressed surrounding myometrial muscles.
- **Capsule:** the capsule is formed of compressed surrounding muscle fibers.

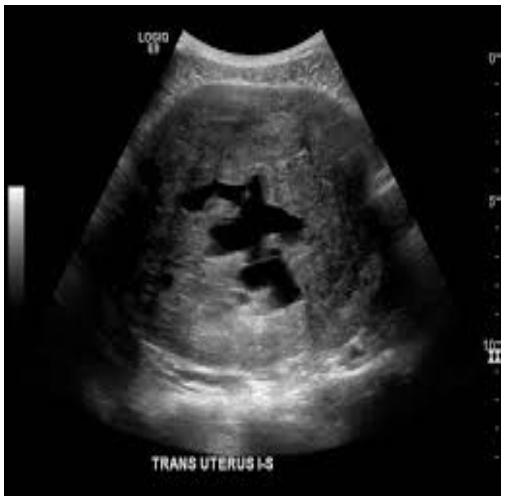
Blood supply:

-It is supplied by the vessels in the capsule that passes inwards to the tumor, so degeneration starts in the center & calcification occurs at the periphery.

-Fibroid polypi obtain their blood vessels from the pedicle, so necrosis starts at the tip.

Microscopic Picture: "Stained with Van Gieson stain"

- Smooth muscles have short thick nuclei & stains yellow.
- Fibroblasts have fusiform nuclei & stains pink.



Cystic degeneration

Complications and Pathological Changes:

A. Degenerative changes:

1. Atrophy: physiological atrophy after menopause or pregnancy.
2. Necrosis: occurs at the tip of a fibroid polyp.
3. Hyaline degeneration "the commonest":
   - It starts in the center, as it is the least vascular.
   - The whorly appearance is lost, replaced by hyaline material and consistency becomes soft.
4. Cystic degeneration:
   - It is either due to liquefaction of the hyaline material "false cyst" or telang-ectasia "true cyst".
5. Fatty degeneration:
   - The tumor becomes yellow and rubbery in consistency, and this precedes calcification.
6. Calcification:
   - White patches are seen at the periphery.
   - The tumor becomes stony hard and X- ray reveals an eggshell appearance [womb stone].
7. Red degeneration "Carneous degeneration":
   - It is common in pregnancy rapid growth, increased vascularity and increased fibrinogen.

- **C/P:** there is acute abdominal pain & tenderness, vomiting, low-grade fever & tachycardia.

Treatment: surgery should be avoided to avoid bleeding.

"Treatment is mainly conservative as bed rest and analgesics, if failed → myomectomy for the affected tumors only".

B. Malignant Change:

*Incidence***:** leiomyosarcoma is very rare (< 0.1% of myomas).
*Clinically***:** it is known by→

1-Rapid growth. 2-Rapid recurrence after removal.
3- Postmenopausal growth "it should atrophy". 4-Postmenopausal bleeding & pain.
5-At operation; there is infiltration of the capsule and loss of whorl appearance.

C. Vascular changes:

- Congestion due to torsion of a pedunculated fibroid.
- Edema due to torsion or infection.
- Lymphangiectasis or telangiectasis.

D- Infection: *Causes***:**

-In a sub-mucous fibroid after labor or abortion.
-At the necrosed tip of a sub-mucous fibroid polyp.
-In a sub-serous fibroid from a nearby infected organ e.g., appendix or intestinal flora.

*Clinical picture***:** lower abdominal pain, tenderness, FAHM & tachycardia.

Treatment: antibiotics, myomectomy or hysterectomy after control of fever.

E- Associated Changes in the Pelvic Organs:

1. The uterus:
   - Increased size may be symmetrical or asymmetrical.
   - Increased vascularity.
   - Endometrial hyperplasia.
   - Increased surface area of the endometrium and endometrial congestion.
   - Myometrial hypertrophy.
2. **The ovaries:** functional follicular cysts are commonly associated with fibroid.
3. The tubes:
   - Chronic Salpingitis.
   - Tubal block by cornual fibroid.
   - Stretch of the tube due to large broad ligament fibroid.
4. **The urinary bladder:** anterior wall sub-serous myoma causes frequency of micturition.
5. **The urethra:** stretch by large interstitial cervical fibroid that causes retention of urine.
6. **The ureter:** broad ligament or cervical myoma can cause hydroureter and hydronephrosis.

F. Torsion of pedunculated sub-serous fibroid:

Predisposing factors: moderate sized tumor with long pedicle, pregnancy or puerperium.

Clinical picture: acute abdominal pain & shock in some cases.

Treatment: anti-shock measures if the patient is shocked then myomectomy or hysterectomy.

G. Rupture of a surface vein on a sub-serous fibroid:

Clinical picture: picture of internal hemorrhage.

Treatment: anti-shock measures followed by myomectomy or hysterectomy.

H. Impaction (incarceration):

- Occurs in cervical or posterior wall sub-serous myomas.
- It is liable to occur in the premenstrual period.

I. Complications during pregnancy and labor:

A. During pregnancy:

- Abortion if sub-mucous fibroid OR ectopic pregnancy if cornual fibroid.
- Incarcerated retroverted gravid uterus if posterior wall fibroid.
- Malpresentations, non-engagement.
- Red degeneration "discussed before".
- Rarely fibroid causes accidental hemorrhage.

B. During labor & puerperium:

- Pre-term labor.
- Uterine inertia OR obstructed labor.
- Rarely, acute uterine inversion in sub-mucous fundal fibroid.
- Postpartum hemorrhage.
- Puerperal infection & sub-involution of the uterus.

Diagnosis:

[A] -Clinical:

(a)-Symptoms:

-Asymptomatic: accidentally discovered in many cases.

-Abnormal uterine bleeding (AUB-L) that may be

-Heavy menstrual bleeding (HMB) the commonest symptom BUT absent in absent in case of sub-serous or cervical fibroids.

-Irregular intermenstrual bleeding (IMB) commonly occurs with:

-Ulcerated tip of sub-mucous fibroid polyp.

-Endometrial hyperplasia, malignant change or associated endometrial cancer.

1-Pain:

-Hyaline degeneration causes dull aching pain.

-Red degeneration causes acute abdomen.

-Infection causes pain with purulent discharge & fever.

-Torsion of pedunculated subserous myoma, causes acute pain.

-Malignant transformation causes dull aching pain.

2-Infertility: due to:

@ Cornual fibroids lead to tubal block. @ Cervical fibroids cause distortion of the cervical canal.

@ Sub-mucous fibroid interfering with implantation @ Associated anovulation OR salpingitis.

3-Dys group: "dysuria, dyschazia, dysparuenia, dysmenorhea, and discharge.

4-Mass: that may be abdominal or pelvi-abdominal

(b).Signs:

1. General: pallor due to anemia, signs of hydronephrosis or renal affection.

2. Abdominal examination: a large fibroid form a pelvi-abdominal mass showing;

- The lower border cannot be felt abdominally.
- **The surface is irregular with sub-serous or smooth with sub-mucous and interstitial ones.**
- The consistency is firm, but turns soft with pregnancy & hyaline degeneration.
- Mobile from side to side but not up & down.
- Dull on percussion BUT auscultation reveals a uterine souffle due to increased vascularity.

3. Local and pelvic examination:

- A fibroid polyp can be felt.
- The uterus is symmetrically or asymmetrically enlarged and firm in consistency.
- The tumor is mobile & its movement is transmitted to the cervix.
- Associated ovarian cyst OR tumor may be felt.

[B]-Investigations:

1. Lab: blood picture, kidney function tests, liver function tests, estrogen level...etc.

2. Imaging

i. Plain X ray: may reveal calcification.

ii. Intravenous pyelography: to assess the renal functions & the course of the ureter. It is especially needed in cases of cervical or broad ligament fibroid.

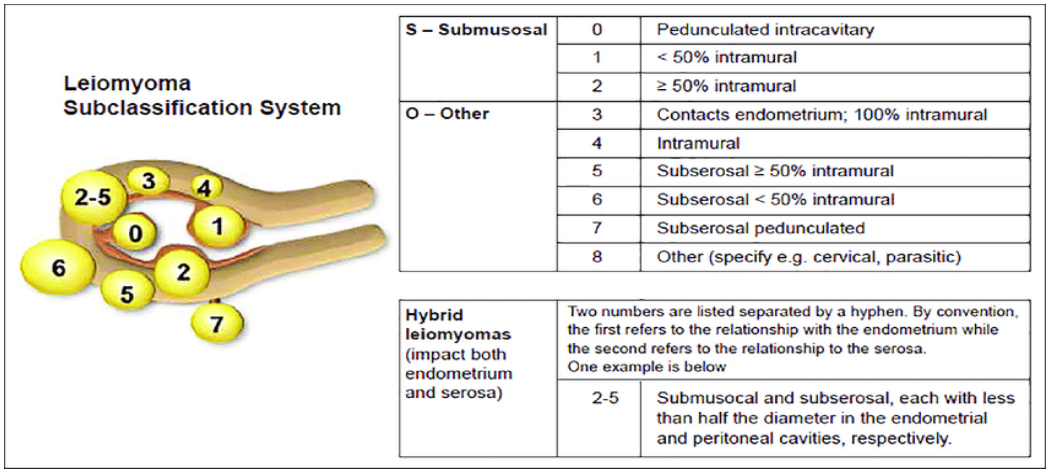
iii. Ultrasonography.

3. Endoscope:

i. Laparoscopy: to differentiate sub-serous polyp & an ovarian swelling.

ii. Hysteroscopy: to detect sub-mucous myoma.

4. Histopathology: endometrial curettage to confirm diagnosis and to exclude malignancy.



S – Submusosal	0	Pedunculated intracavitary
	1	< 50% intramural
	2	≥ 50% intramural
O – Other	3	Contacts endometrium; 100% intramural
	4	Intramural
	5	Subserosal ≥ 50% intramural
	6	Subserosal < 50% intramural
	7	Subserosal pedunculated
	8	Other (specify e.g. cervical, parasitic)

Hybrid leiomyomas (impact both endometrium and serosa)	Two numbers are listed separated by a hyphen. By convention, the first refers to the relationship with the endometrium while the second refers to the relationship to the serosa. One example is below	
	2-5	Submusocal and subserosal, each with less than half the diameter in the endometrial and peritoneal cavities, respectively.

FIGO classification of leiomyoma

TREATMENT

1- Prophylactic: by avoidance of hyper-estrogenic state and proper treatment

2. Active treatment:

I. No treatment: no symptoms no treatment except in ;

-Large myoma > 12 weeks as it is liable to degeneration.
-Rapidly growing myoma due to suspicion of malignancy.
-Pedunculated sub-serous myoma as it is liable to torsion.
-Sub-mucous myoma in a nullipara to avoid infertility or abortion.
-Large interstitial cervical fibroid to avoid ureteric compression.

II. Conservative medical treatment: it is indicated in ;

-Patients near menopause with a small myoma & slight menorrhagia.
-If the patient is unfitting for surgery.
-In the preoperative period to shrink the myoma for easy surgery.
-Preoperative until correction of anemia.

Lines: *GnRH analogues for 6-9 months OR progestogens for 6-9 months.*

III. Surgical Treatment: *that may be conservative surgery "myomectomy or polypectomy" OR radical surgery "i.e., hysterectomy".*

1- Myomectomy

Indications: young patient < 40 years desiring pregnancy and small sized uncomplicated fibroid.
Advantage: the uterus is preserved for future pregnancies.
Timing: postmenstrual to decrease bleeding.
Types and Routes: abdominal [laparotomy or laparoscopy] OR vaginal [polypectomy or hysteroscopy].

Disadvantages of myomectomy:

i-Higher mortality than hysterectomy due to higher risk of bleeding.
ii-Pelvic adhesions especially with a posterior wall uterine incision.
iii-Rupture of the uterine scar in subsequent pregnancy.
iv-Asherman's syndrome. v-Recurrent fibroid.

2- Hysterectomy:

Indications -Patients above the age of 40 or completing their family size.
-Multiple fibroids so that after myomectomy a useless uterus is left.
-Suspicion of malignancy.

Types: 1-Abdominal hysterectomy: that may be total (preferred) or subtotal (not preferred)
2-Vaginal hysterectomy (the best natural route)
3-Laparoscopic or Laparoscopic assisted vaginal hysterectomy.

NB. Salpingo-oophorectomy may be added in certain cases

Hysteroscopic myomectomy: https://youtu.be/CPP6Y7S-GFQ?si=3tJ0qxiVLHbyLUnQ
Transvaginal myomectomy: https://youtu.be/KGPhRGDrp9c?si=R48bMnolalT8fgsN
true broad ligament myoma: https://youtu.be/O84VYeZLfBg?si=tnCkwjKyewpQszhZ

ADENOMYOSIS

Definition: It is the presence of endometrial glands and stroma in the myometrium, with adjacent smooth muscle hyperplasia.

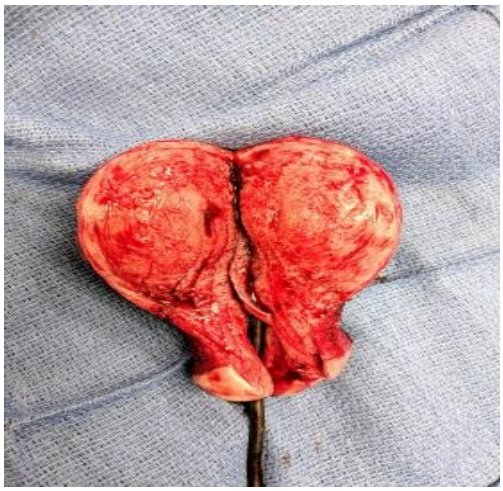
Incidence:

Total: in about 15%
Age: in the middle-aged women at the end of reproductive life.
Parity: it is more common in women with high parity.
Race: more common in high socioeconomic standard and white race.

Etiology: its causes remain unclear, however the most accepted theory is the *invagination of the endometrial tissue (glands & stroma) into the myometrium* previously known as *Cullen's diverticulum's theory.*

Naked eye:

Uterus: may appear symmetrically enlarged (diffuse adenomyosis) OR asymmetrically enlarged (localized adenomyosis) and is usually < 10 weeks.



On cut section:
1-Small areas of blood spots within the myometrium.
2-Large cystic space filled with altered blood.
3-Not capsulated with no whorl appearance.
4-Uterine cavities are enlarged and lined with hyperplastic endometrium.

M/E: - The endometrial glands are present within the stroma cells and surrounded by myometrial muscle fiber.

DIAGNOSIS:

Clinical:

a).Symptoms: the main symptoms are:
-AUB-A: (Abnormal uterine bleeding - adenomyosis).
-Secondary dysmenorrhea "crescendo dysmenorrhea" or pelvic discomfort.

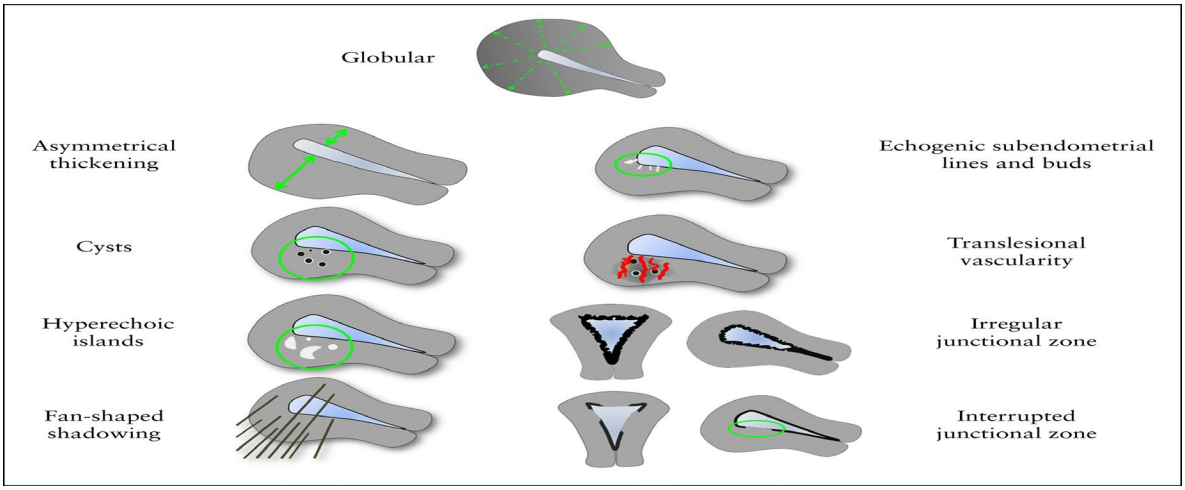
b).Signs:

-Uterus is symmetrically or asymmetrically enlarged usually BUT < 10 weeks.
-Uterus is tender on bimanual examination.

Investigation:

1- **Imaging :** *(ultrasound, MRI, hysteroscopy, laparoscopy)*

-Ultrasound:→ used as a primary screening and early diagnosis of adenomyosis.
Various sonographic appearances have been described e.g. :
i-Myometrial cysts. ii -Sub endometrial nodules.
iii-Sub endometrial linear striations. iv- Asymmetric myometrial thickening
v-Poor definition of the endometrial/myometrial border.

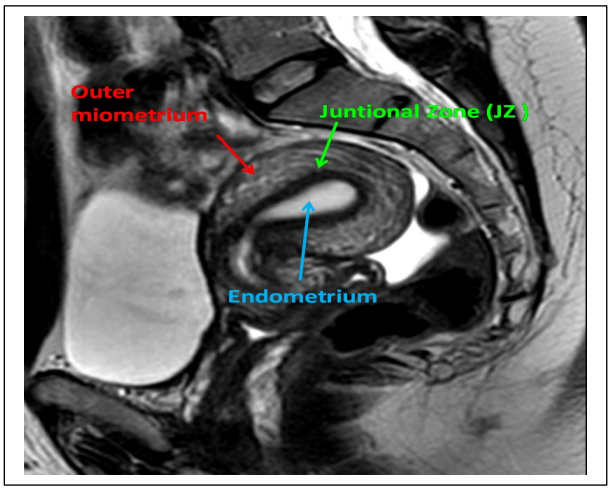


Adenomyosis - ultrasound

-**MRI.:** most accurate. ---------- >>>>>>>>>

-Junctional zone (JZ)= 8-12 mm→ suspicion,

- JZ 12 mm or more → sure adenomyosis.



Hysteroscopy: → sub endometrial cysts.

Laparoscopy and myometrial biopsy.

Histopathology: → mostly from hysterectomy specimen

TREATMENT

Prophylactic: by avoidance of predisposing f.

[I] **Active**:

A-Medical treatment: that include:

1-Non-steroidal anti-inflammatory drugs (NSAIDs).

2-Combined oral contraceptives **(COCs).**

3-High-dose **progestogens**.

4-Levonorgestrel-releasing intrauterine system **(LNG-IUS).** In **minimal** disease with little symptoms in patient near menopause.

B-Surgical treatment: that include:

1-Hysterectomy: it is the definitive treatment of adenomyosis.

- It should **not** be accompanied by oophorectomy unless there are specific indications.

2-Endometrial ablation (hysteroscopic): (not recommended) may be indicated in patients unfit for hysterectomy but usually fails in patients with deep adenomyosis.

ENDOMETRIOSIS

Definition: Presence of endometrial tissue (glands & stroma) outside normal uterine cavity.

Incidence: Accurate assessment of incidence is difficult because laparoscopy & laparotomy are needed for diagnosis (it is found in 5-30% of all laparoscopies)

Etiology: Exact cause is unknown (disease of theories).

A) **Implantation** (transplantation) theory: Endometriosis is caused by implantation of endometrial cells via trans-tubal regurgitation during menstruation. This is the most accepted theory for etiology

B) **Celomic metaplasia** theory: Endometriosis develops from metaplasia of cells lining pelvic peritoneum. No clinical or experimental support to this theory.

C) **Vascular** – lymphatic theory: Endometrium is transported via vascular & lymphatic systems. • Evidence: Occurrence of endometriosis at distant sites (as umbilicus or lungs)

D) **Genetic** factor: Risk is 7 times greater if 1st degree relative has endometriosis.

E) **Immunologic** factors: Immune system may be altered in women è endometriosis because of decreased cell mediated cytotoxicity allowing endometrial cells to survive outside endometrial cavity.

Predisposing factors:

A) Uterine manipulation during menstruation: HSG or IUCD insertion.

B) Outflow tract obstruction: Causes of cryptomenorrhea

Pathology:

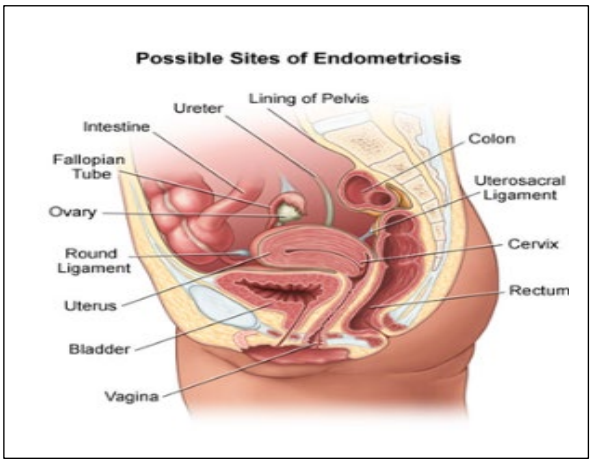
-Sites of endometriosis:

A-Pelvic: it is primarily a pelvic disease.

1-Common in: ovaries (65%), Cul-de-sac, uterine ligaments, rectovaginal septum.

2-Uncommon in bladder, bowels, pelvic peritoneum.

B-Extra pelvic: skin, lungs, umbilicus, Episiotomy scars, Nasal mucosa.

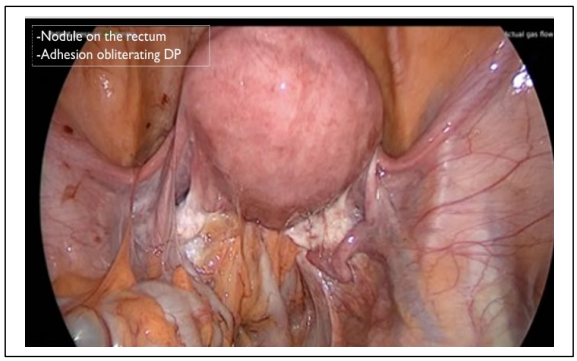


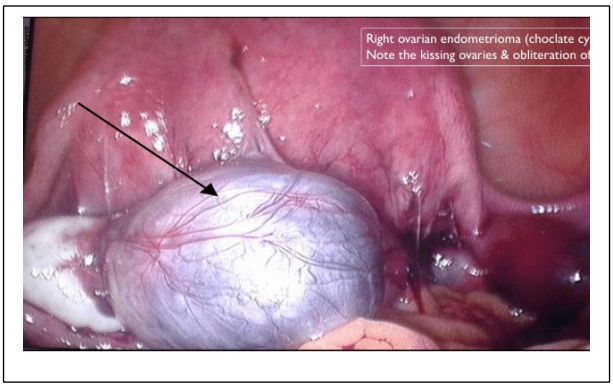
Gross appearance: Variable according to size of implants & extent of adhesions:

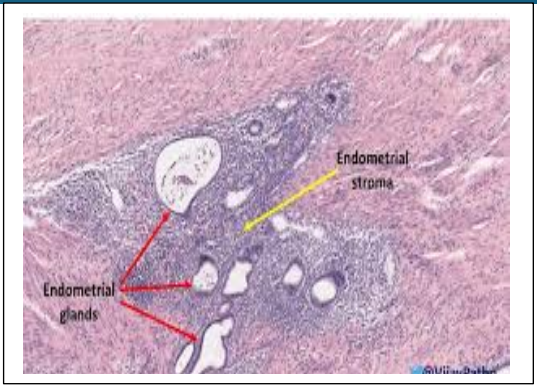
a). Multiple surface implants vary in size (pinpoint to 5 mm).

b). Chocolate cyst filled with altered blood.

c). Adhesions which vary in size from filmy to dense thick vascular adhesion







Microscopic Picture: It is essential for the diagnosis to find:

-Endometrial glands, endometrial stroma and areas of hemorrhage.

-There may be surrounding zones of inflammatory cells and phagocytes.

DIAGNOSIS:

[A] Clinical:

a) Symptoms:

1-Asymptomatic and accidentally discovered.

2-Pelvic pain: it is *a disease of pain* that may be diffuse or localized for more than 6 months the pain is caused by:

-Sequential swelling and extra-vastation of blood in the surrounding tissue.

-Scaring and retraction of peritoneum.

-Elevated levels of PGs and histamine.

3- Infertility; the most important complication of endometriosis and may be caused by:

➔***In severe endometriosis*:** due to *mechanical factors caused by tubal and ovarian adhesions*.

➔***In mild endometriosis:***

i-Ovarian factors:

- Anovulation or luteinized unruptured follicle syndrome.
- Oocyte maturation defects or luteolytic effect caused by PGs.
- Altered prolactin and gonadotrophins release.

ii-Coital factors: dyspareunia causing reduced penetration and coitus.

iii-Tubal factors: alteration of tubal motility caused by PGs.

iv-Abnormal sperm function.

v-Abnormal Endometrium: luteal phase defects or interference with implantation.

4-Dys- group:

i-Dysmenorrhea (crescendo type). ii- Dyspareunia.

iv-Dysuria, frequency, urgency and cyclic hematuria with bladder endometriosis.

v-Dyschazia, hematochezia and partial bowel obstruction with GIT affection.

5-Menstrual disturbances: as menorrhagia or polymenorrhagia.

b) Signs: refer to the gross appearance above .

(B)-Investigations:

1- Lab: serum CA 125 "increased in endometriosis.

2- Radiology: US, C.T scan or MRI.

3- Laparoscope: it is a **gold standard** for diagnosis. There is visualization of the endometriotic lesions, chocolate cysts in the ovary, pelvic adhesions.

4- Histopathology: refer to microscopic picture above

TREATMENT:

A)- Prophylactic:

1-By avoidance of hyper-estrogenic state. 2-Avoidance of coitus, vigorous P/V or HSG during menstruation.
3-Proper repair of episiotomy and C.S scars with avoidance of implantation of the tissues.

B)- Active: that include the following.

I- Expectant medical treatment: (*Indicated in young women, minimal endometriosis, or patients near menopause.* It includes):

1-Analgesics (NSAIDs) as pain killers and being anti-PGs.
2-Ovarian suppression by any of the following: .

a). Pseudo-pregnancy state:

i- Combined oral contraceptive pills: for at least 6-9 months and non-stop regimen.
ii. Gestagens only (Provera or depot medroxy progesterone acetate DMPA) for 6-9 months.

b), Pseudo-menopause:

i-Danazol: it is oral synthetic 17- alpha ethinyl testosterone (not used now due to increased side effects)

-It is the **most effective** approved drug for the treatment of endometriosis.
-It acts through suppression of pituitary gonadotrophins and inhibition of ovarian steroid- genesis (i.e., medical oophorectomy).
-Side effects: *many side effects***,** the main side effects are virilizing symptoms.

ii-GnRH analogues (agonists): [Goserelin, Triptorelin, Buserlin]

-It produces pituitary down regulation and desensitization to endogenous GnRH (i.e., medical hypophysectomy).

*Side- effects***:** menopausal symptoms e.g., hot flushes, vaginal dryness and osteoporosis.
iii- Oral GnRH antagonist: including '**elagolix**',' **relugolix**', and '**linzagolix**'**;** allow oral administration, induce dose dependent reduction of estradiol levels, do not cause initial flare-up of endometriosis symptoms, and allow the fast return of ovarian function and menstruation after discontinuation.

II- Surgical treatment: it may be conservative or radical.

A)- Conservative treatment: indicated in young age needing further pregnancy.

1-Adhesiolysis. 2-Electrocoagulation, cauterization or laser evaporation of implants.

3-Surgical excision of endometriomas. 4-Nerve ablation to relief pain (LUNA technique).

5-Reconstruction of the peritoneal surface to cover raw areas to prevent adhesion.

B)-Radical treatment: *it is indicated in* Recurrent disease, Severe endometriosis., Patient completed her family.

& failed medical therapy.

➔ ***Usually radical treatment is "total hysterectomy with bilateral salpingo- oophorectomy" that may be performed abdominally, by laparoscopy or robotic surgery.***

ENDOMETRIAL HYPERPLASIA

Definition: It is irregular proliferation of the endometrial glands with an increase in the gland to stroma ratio when compared with proliferative endometrium. Endometrial hyperplasia **is the precursor of endometrial cancer** which is the most common gynecological malignancy in the Western world.

Incidence: The incidence of endometrial hyperplasia is estimated to be at least three times higher than endometrial cancer. It more common in nulls, peri-menopausal age and white races.

Etiology: Prolonged unopposed estrogenic stimulation, genetic and familial tendency.

Risk factors include:

1-increased body mass index (BMI); with excessive peripheral conversion of androgens in adipose tissue to estrogen.
2-anovulation associated with the perimenopause or polycystic ovary syndrome (PCOS)
3-estrogen-secreting ovarian tumors, e.g. granulosa cell tumors (with up to 40% prevalence of endometrial hyperplasia)
4- drug-induced endometrial stimulation, e.g. the use of systemic ERT or long-term tamoxifen

Naked/eye:

- The uterus may be normal sized, atrophied "menopausal uterus" OR enlarged due to other associated pathology.

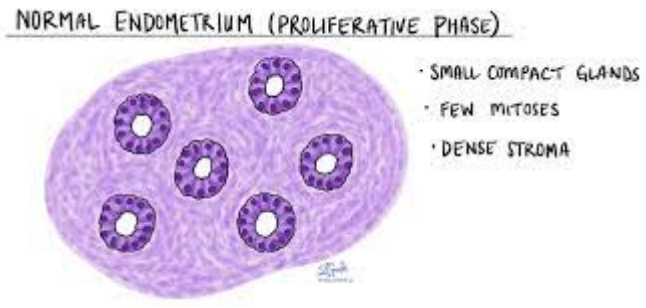
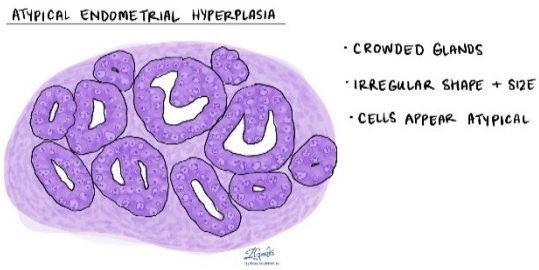
Histopathology:

The **2014 revised WHO classification**: -- Simply separates endometrial hyperplasia into 2 groups based upon the presence or absence of cytological atypia,

1-Hyperplasia without atypia and

2-Atypical hyperplasia.

The complexity of architecture is no longer part of the Classification.

EH without atypia	Atypical EH
1-simple compact glands 2-few mitosis 3- regular shape & size 4- dense stroma 5- risk for endometrial cancer < 5% over 20 years 	1-crowded glands 2- more mitosis 3- irregular shape & size 4- cells appear atypical 5- risk for endometrial cancer = 28% over 20 years 

DIAGNOSIS

A). Clinical: (Symptoms & Signs):

1-History of etiology (hyper estrogenic status)
2-Asymptomatic and accidentally discovered on screening for endometrial carcinoma.
3-**Bleeding (AUB-M)**. The most common presentation of endometrial hyperplasia is abnormal uterine bleeding; includes-

i- heavy menstrual bleeding (HMB), ii-inter-menstrual bleeding (IMB),
iii- irregular bleeding, iv - unscheduled bleeding on HRT
v - postmenopausal bleeding

4-symptoms of the etiology e.g., picture of PCOS…etc.

5-The uterus may be normal, atrophied, or enlarged and may be ovarian swelling or tumors.

B)- Investigations:

1-*Imaging***: TAS or TVS** there may be thickened endometrium.
2-*Histopathology***:** by endometrial curettage (show the types mentioned before).

TREATMENT

Depends on:
1-Type of hyperplasia (EH without atypia or EH with atypia)
2-Age of patient (childbearing age, premenopausal, or postmenopausal)
3-Desire for future fertility

[A]-Reproductive age:

1-Endometrial hyperplasia without atypia:

-Progestins: 10-20 mg oral daily for 6 continuous months. **OR**
-LNG-IUS [Mirena] for 5 years. With →*Follow -up endometrial biopsy every 6-12 mnoths*

2-Atypical hyperplasia:

-LNG-IUS or high dose progestin with→ Follow-*up endometrial biopsy every 3-6 months*

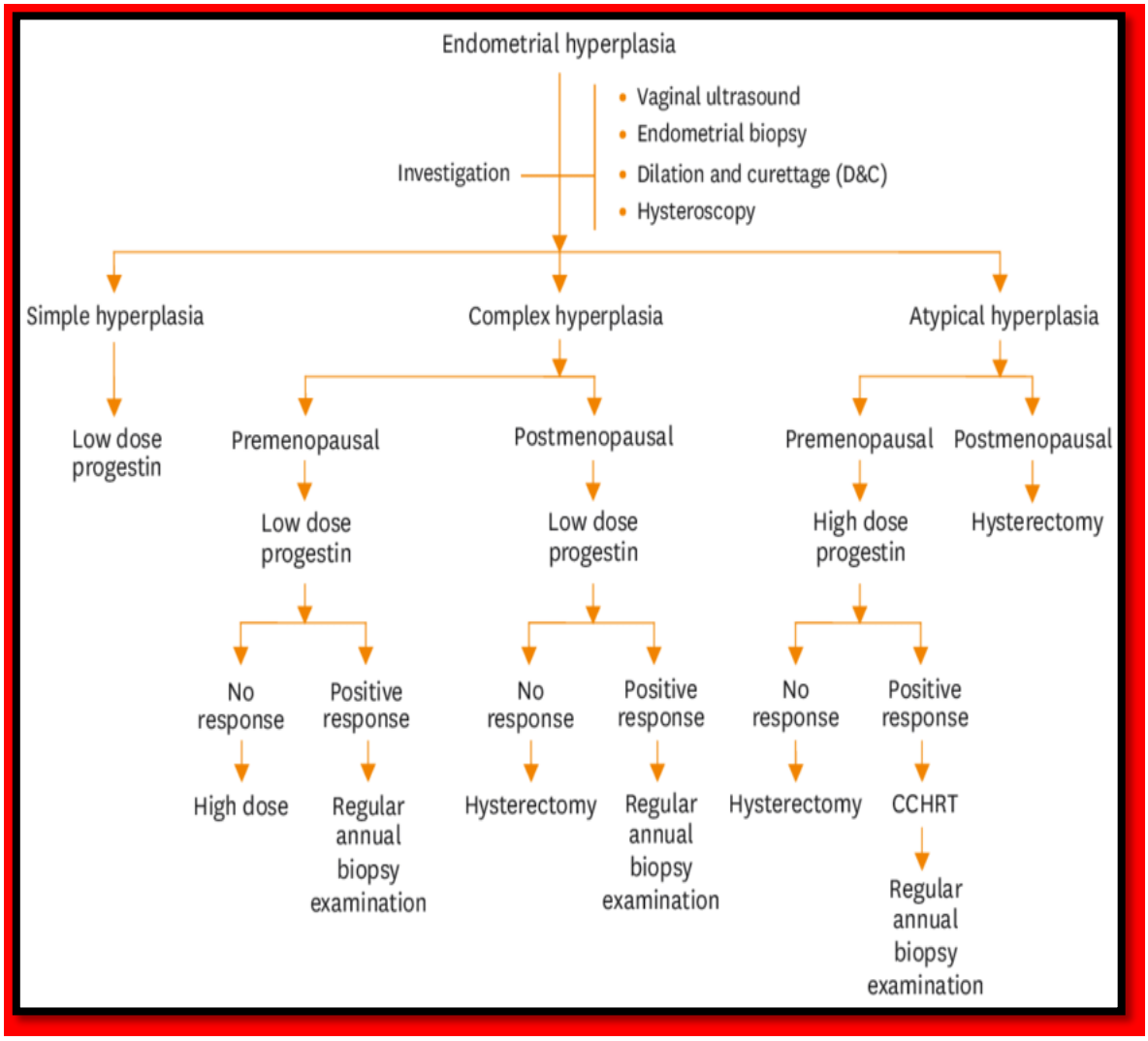
Hysterectomy, if no regression by hormonal treatment or if completed her family.

[B]-Pre-or postmenopausal age:

1-Endometrial hyperplasia without atypia:
-Progestins: as before "for 6 months".
-Hysterectomy: if no response on progestin.

2- Atypical hyperplasia: hysterectomy

- Follow up of treatment with endometrial sampling (office endometrial biopsy) not by ultrasound.
- Hysterectomy when done for EH it is best vaginal or laparoscopic (minimally invasive technique).
- Bilateral salpingectomy should be done with hysterectomy for EH to reduce incidence of ovarian cancer (when slapingo-oophorectomy is not done).



SCHEMA FOR MANAGEMENT OF EH

INFERTILITY

Definition: It is the inability of a couple to conceive despite there is a continuous regular marital relationship for at least one year without the use of any form of contraception.

Types:

- **Primary:** pregnancy never occurred before.
- **Secondary:** pregnancy occurred before even once whatever its mode of termination.
- **Relative:** ability of the patient to get pregnant but inability to maintain it till delivery.
- **Permanent "sterility":** inability of the patient to get pregnant forever due to permeant cause.

Incidence: 10-15 % during first year of marriage while 5 % from in second year later.

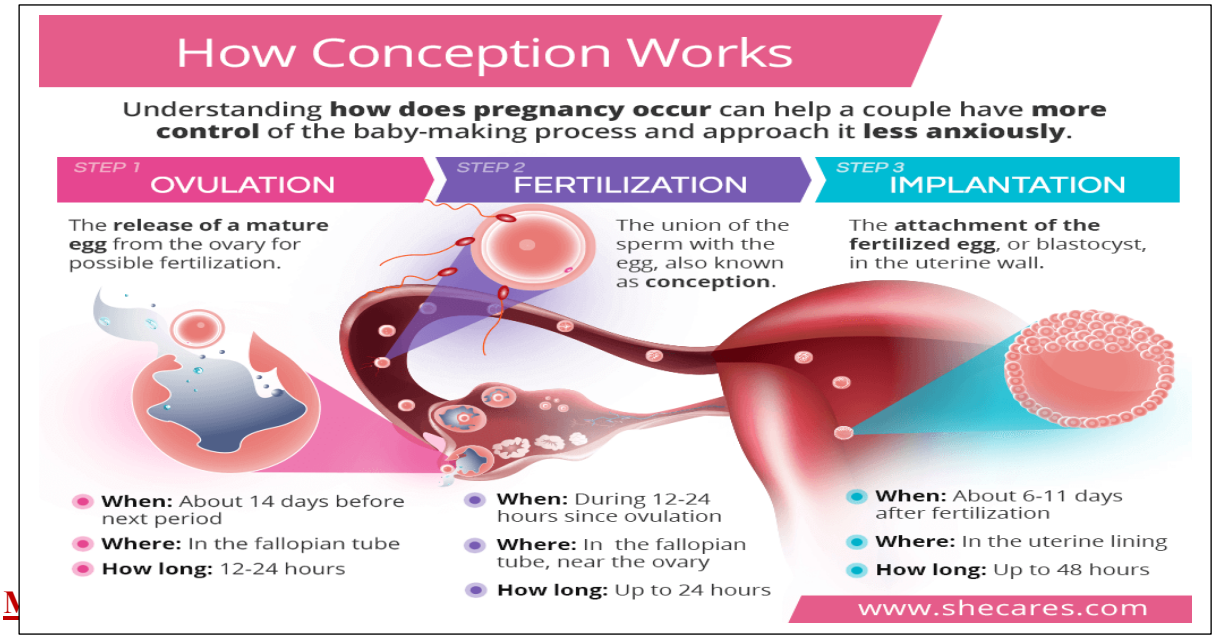
Basic requirement for fertility:

A) Male requirements:

1. Adequate spermatogenesis, then transport via →
2. Normal duct system, then →
3. Ejaculation & deposition of sperms in the posterior vaginal fornix.

B) Female requirements:

1. Production of healthy ova & good CL function (ovarian factor).
2. Patent healthy tubes suitable for fertilization & transport of the ovum (tubal factor).
3. Normal uterine cavity with secretory endometrium suitable for implantation & early development of embryo (uterine factor).
4. Patent healthy cervix with normal non hostile cervical mucus (cervical factor).
5. Vagina suitable for deposition of semen with non- hostile vaginal secretions (vaginal factor).
6. Adequate hormonal & immunological status.
"So, causes of infertility may be male, female, combined causes or unexplained infertility".



- These may be defective formation, transport, OR deposition of the sperms in the posterior fornix. The causes may affect the quality or quantity of the sperm.

1- Congenital:

- Absent vas deferens OR duct obstruction. - Hypospadias OR epispadias.
- Testicular hypoplasia OR crypt-orchidism.

2- Traumatic: e.g., injury of the vas during repair of hernia.

3-Inflammatory: orchitis, prostatitis, STDs…etc

4- Neoplastic: any neoplasm destructing testicular tissues.

5- Dysfunctions: e.g.

- Failure of sperm deposition as in impotence, premature and retrograde ejaculations.
- Abnormal spermatogenesis.
- Abnormal seminal fluid or sperm motility.
- Immunological factors e.g., antibodies against sperms.
- Varicocele "its role is controversial".

6- General causes: genetic*s,* smoking, alcoholism, liver cell failure or idiopathic.

Diagnosis

1- Clinical, history and examination of the husband:

- Age, job, and residence of the husband.
- Previous marriage, and having or no previous child
- History of diseases, trauma, surgery…etc.
- On exam: the cause may be detected e.g., loss of body hair, obesity, gynecomastia. varicocele, hypospadias, testicular hypoplasia.

2- Investigations:

1- Laboratory: - CBC., liver and renal functions and semen analysis (most important).
2- Testicular biopsy (in azoospermia). 3- Culture and sensitivity (for infection)
4- Chromosomal studies. 5- Detection of sperm antibodies.
6- Postcoital tests (not done now).

Abnormal seminogram:

- **Aspermia:** absence of semen.
- **Azospermia:** absence of sperms.
- **Oligospermia:** sperm count below normal.
- **Necrospermia:** dead sperms.
- **Pyospermia:** pus in seminal fluid.
- **Abnormal motility** e.g., sluggish OR shaking movement.
- **Heamatospermia:** blood in sperms OR RBCs.

"If any abnormality in the semen is detected no treatment is given except after repetition of analysis in another lab. 2 weeks latter".

The normal seminogram is characterized by [WHO 2021]:	
Parameter	Normal value
Volume	≥ 1.5 ml.
Color	Grayish white.
Odor	Characteristic.
PH	≥ 7.2 (alkaline).
Viscosity	< 2 cm thread forming.
Liquefaction	Within 20 minutes.
Total sperm count	≥ 39 million / ejaculate.
Sperm concentration	≥ 15 million / ml.
Sperm motility	≥ 40% totally motile (progressively & non progressively motile) within 1 hour of ejaculation.
	≥ 32% progressively motile within 1 hour of ejaculation.
Sperm morphology	≥ 4% are normal forms.
Sperm viability	**≥ 58% are viable.**
WBCs	**2-5 / HPF.**

Treatment:

- **Medical treatment:**

• Treatment of any general cause if present, general tonics good diet and vitamins.
• Hormones when needed e.g., GnRH, FSH, LH, testosterone.

- **Surgical treatment e.g.**

• Vein ligation in varicocele. • Treatment of hypospadias.

- ART "assisted reproductive techniques":

1.IUI-H (intrauterine insemination-husband).
2.IVF-ET/ICSI (in-vitro fertilization-embryo transfer)/ intracytoplasmic sperm injection. 3- Others (not commonly used now) such as GIFT (gamete intrafallopian transfer), ZIFT (zygote intrafallopian transfer).

CAUSES OF FEMALE INFERTILITY

Ovarian factor = Anovulation.

Causes:

1- Physiological: all causes of physiological amenorrhea.
2- Pathological:
A- General causes: as amenorrhea and in addition:
1- Age: fertility decreases with advanced age especially at or beyond 40 years.
2-Weight: fertility decreases with overweight and obesity.

3- Frigidity: i.e., absence of sexual desire.
4- Psychological: e.g., anxiety, depression…etc.
5-Cigarette smoking and addiction.
6-Environmental toxins: lead, toxic fumes, pesticides.
B- Hypothalamic and pituitary causes (= all causes of amenorrhea).

C- Ovarian causes: all causes of ovarian amenorrhea and in addition:

1- Luteal phase defect: with decreased progesterone level OR less sensitive endometrium; so, the secretory changes are defective.

2- Luteinized un-ruptured follicles syndrome: maturation of the follicle but fail to rupture.

Diagnosis i.e., diagnosis of an-ovulation:

I- Clinical:

A-Symptoms: *absence of normal symptoms of ovulation which are:*

- Regular cyclic menstruation.
- Ovulation pain = mid-cyclic pain "= Mittle-Shmirz pain".
- Ovulation bleeding and cascade.
- Premenstrual mastalgia.
- Elevated body temperature during the second half of the cycle.

In addition, there may be: **history OR complaint suspecting the cause.**

B-Signs:→*Absence of the following normal signs of ovulation [all depends upon progesterone]:*

1- Biphasic basal body temperature chart.
2- Changes in characters of cervical mucous.
3- Change in character of vaginal smear.
4- Signs of the cause: as signs of excess androgen, obesity, and galactorrhea…etc.

2- Investigations:

A-Laboratory:

- Estrogen level and its type in blood.
- Progesterone level in blood in mid-luteal phase is decreased “normal 10-15 ng/dl".
- Pregnanediol level in urine is decreased.
- L.H level in plasma and urine.
- Prolactin level to detect hyperprolactinemia.
- Testosterone, and its derivatives in hyperandrogenism.

B- Imanging:
- U.S For serial measurements of follicular growth and maturation, "the mature Graafian follicle is about **18-25** mm".

C- Endoscopic:- Laparoscopy for direct visualization of the stigma of ovulation.

D- Histopathological:

- Premenstrual endometrial biopsy shows no secretory changes OR luteal phase defect.

Treatment

1- Correction of general condition:

- Treatment of general causes, hypothalamic and pituitary as in amenorrhea.

2- Induction of ovulation: this may be medical OR surgical

A- Medical: by using any of the followings ---→

1. Clomiphene citrate = Clomid.

- *Used as 50-150 mg/day for 5 days starting from 2nd – 5th day of cycle for 6 months.*
- *Ovulation rate is 70% & pregnancy rate is 40%.*
- *Lower pregnancy rate may be due to:*
  - Antiestrogenic effect on cervical mucus & endometrium.
  - Luteal phase defect.
  - Improper coitus timing.
  - Other undiagnosed factors of infertility.

- *Clomiphene Citrate resistance (OR failure):* No documented ovulation after 6 months of treatment with the usual daily dose.

2. Aromatase inhibitors "letrozole"
3. H.M.G. = human menopausal gonadotrophins.
4. H.C.G = human chorionic gonadotrophins.

Direct stimulation of follicular growth

5. Pulsatile GnRH agonists OR long-acting antagonist followed by gonadotrophins.
6. Tamoxifen as selective direct estrogen receptor inhibition.
7. Bromocriptine as anti-prolactin in hyper-prolactineamia.
8. Metformin as ant-insulin in PCOS.

Indications of using ovulatory drugs :

1- Infertility due to anovulation. 2-Post-pill amenorrhea. **3-**PCOS.
4-Unexplained infertility (to ↑↑ number of available ova). 5-Luteal phase defect (LPD).
6- Controlled ovarian hyperstimulation in ART.

Pre-requisites for treatment with ovulatory drugs in infertility cases:

- Documentation of anovulation.
- Confirmation of patency of fallopian tubes.
- Investigations for other factors of infertility.
- Ensure good male factor.
- Confirmation of adequate endogenous estrogen level.
- Prepare couples psychological for prolonged therapy & possibility of failure.

B- Surgical: by using

1. Laparoscopic ovarian drilling **[unilateral or bilateral].**

2. Bilateral wedge resection **"NOT used now".**

Ovarian hyper-stimulation syndrome (OHSS):

- It is a common complication of induction of ovulation as the ovaries may enlarge up to 12 cm OR more with risk of peritoneal irritation OR ovarian rupture.
- It may be mild moderate or severe.
- **In mild form;** there is abdominal distension, pain, sickness and diarrhea.
- **In moderate form;** there may be excess fluid in the abdomen leading to more pain and discomfort.
- **In severe form;** the case may be life threatening as there may be free fluid in the abdomen that can be detected clinically without ultrasound or even there may be internal hge, hemoconcentration, hypercoagulability.
- **The main treatment is prophylactic** by adjusting the dose from the start.
- **In mild and moderate cases,** the treatment is usually conservative, stopping the drug and giving analgesics with bed rest.
- **On the other hand, in severe cases;** the treatment may even need hospitalization, correction of general condition by fluids, albumin or blood, and in rare cases, may need laparotomy, peritoneal toilet or oophorectomy.

Tubal factor of infertility

-Tubal causes are about 20-30% of causes of infertility and are as the commonest causes.

Causes:

1.Congenital: tubal aplasia, hypoplasia, diverticulum, accessory ostium.

2. Traumatic: trauma during operation followed by adhesions.

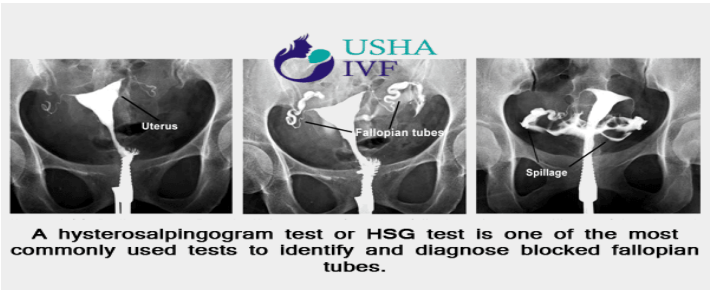
3.Inflammatory: following P.I.D "the commonest cause in the tube".

4. Neoplastic: e.g., small cornual fibroid closing the tubal ostia.

5. Endometriosis: causing pelvic adhesions and abnormal tubal motility.

6. Disturbed physiology: e.g.

-Spasm of the tubes.
-Poor ciliary movement.
-Changed tubal secretions.
-Failure of picking up mechanism.



A hysterosalpingogram test or HSG test is one of the most commonly used tests to identify and diagnose blocked fallopian tubes.

Diagnosis:

I-Clinical: history of puerperal sepsis, P.I.D., TB and on exam:
1. General: Nothing characteristic, or that of infection.
2. Abdominal: there may be inflammatory mass.
3. Local: on bimanual exam, there may be detection of adnexal swellings.
II-Investigations:

A- Radiological: the main line used for diagnosis by "tubal patency tests".

1- Hysterosalpingography = H.S.G: the commonest test used now

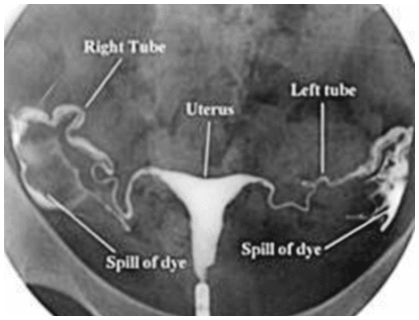
The idea:→ Inject radio opaque dye **[***Lipiodol and Urografin***]** in the uterus to pass through the tubes to the peritoneal cavity.

Timing of the test: 2-5 days postmenstrual:

-Avoid disturbance of undiagnosed pregnancy.
-Avoid iatrogenic endometriosis.
-Avoid false negative results.

Complication:

1-Neurogenic shock, hemorrhage and infection.
2-Perforation of the uterus.
3-Endometriosis.
4- Oil granuloma, Oil embolism, and Oil allergy.



Normal HSG

Values of HSG:

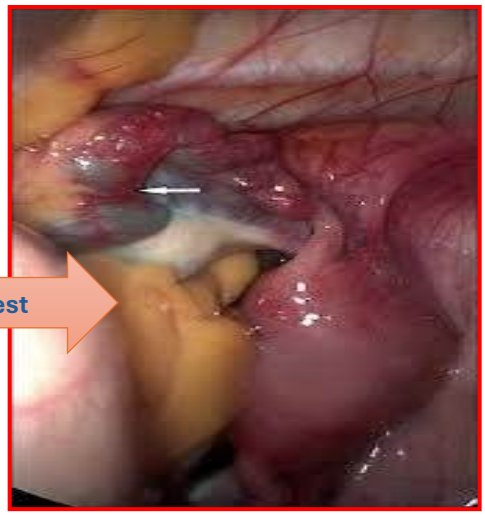
Diagnostic	Therapeutic
1-Congenital uterine anomalies. 2-Asherman's syndrome. 3-Missed I.U.D. 4-Submucous fibroid. 5-Peritoneal adhesion. 6-Cervical adhesions, incompetence, or stenosis. 7-Tubal patency, stenosis, adhesions.	1-Removal of mucous plug that may close the tube. 2-Removal or cutting of thin adhesions. 3-Straightening of kinked tube or relieve of utero-tubal spasm.

2-Saline sono-hysterography (SSHG): by injecting saline and detecting it in Dougla's pouch by TVS.

B- Laparoscopy: which has a rule in diagnosis OR treatment.

Diagnostic laparoscopy **e.g., in:**

-Tubal patency by injecting methylene blue dye through the cx, if passed from the tubal fimbriae, it means patent tubes.
-PID and peritubal adhesion.
-Pelvic endometriosis.
-Associated ovarian factor (e.g., P.C.O.S).

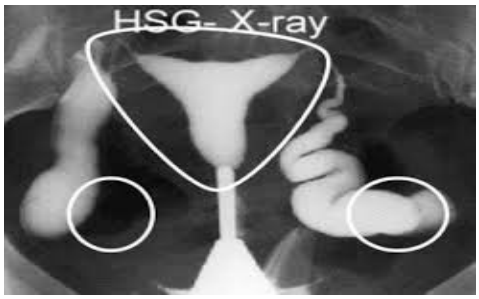


Therapeutic laparoscopy **e.g., in:**

-Salpingolysis and neosalpingostomy.
-Laparoscopic ovarian dri1ling for PCOS.
- Bipolar or Laser vaporization of endometriotic nodules.
-Help in assisted reproductive technology.

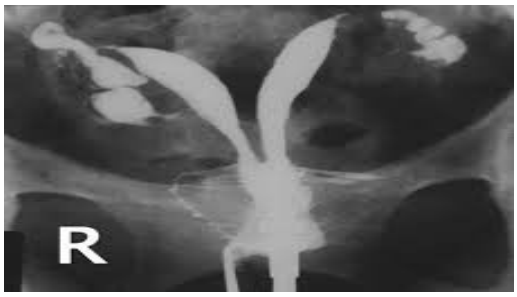
Treatment:

l- *Prophylactic***: b**y avoidance of the causes especially infection.
2- *Medical***:** by fibrinolytic drugs as colchicine, hydro-tubation OR short-wave therapy.
3- *Surgical***:** usually it is the main line of treatment, and mostly microsurgical procedures via laparoscope.
4- *Assisted reproductive technology* **(**ART) "best and commonly used now".



← Bilateral Hydrosalpinx

Bicornuate Uterus→



Cervical factor of infertility

Causes:

1. *Congenital***:** stenosis and Mullarian duct abnormalities.
2. *Traumatic***:** intracervical adhesions, incompetence or destruction the mucoa.
3. *Inflammatory***:** endocervicitis, erosion or any chronic cervicitis causing hostile mucous.
4. *Neoplastic***:** cervical fibroid and masses blocking the cx. or distorting the cervical canal.
5. *Dysfunctional***:** immunological i.e., presence of anti-sperm antibodies.
6. *Cervical incompetence***.** It is a cause of recurrent mid-trimester abortion**.**
7. *Hormonal***:** decreased estrogen causing decreased mucous and rendering it thick.

Diagnosis:

I- Clinical:
- *Symptoms***:** history of infection, pelvic pain, vaginal discharge...etc.
- *Signs***:** picture of infection, cervical mass, abnormal cervical mucous.
- *Postcoital test* and sperm penetration test.

II-Investigations:

Laboratory:

- Study of the cervical mucous character, its P.H., amount, presence or absence of inflammatory cells, content of bacteria, culture and sensitivity in cases of infection.
- Immunological tests to detect anti-sperm Abs.

Treatment

1- Treatment of the cause i.e., treatment of infection, surgical removal of fibroid, etc.
2- In immunological infertility:
-Condoms for 6 months. -Immunosuppressive drugs e.g., corticosteroids.
-ART e.g., A.I.H., IVF and embryo transfer…etc.

Vaginal factor of infertility.

1- *Congenital and anatomical disorders***:** e.g., vaginal aplasia, hypoplasia, or septum.

2- *Hostile vaginal discharge***:** e.g., in vaginitis and increased vaginal acidity.

Diagnosis: history of infection or on exam, detect the causes and post-coital tests.

Treatment: of the cause e.g., infection according to the type, surgical treatment in anatomical causes.

Pelvic and peritoneal factor:

- Any gross pathology in the pelvis may disturb the tubes ovaries OR adhesions causing limitation of movement and functions of these organs.

Diagnosis:

1-Clinical: history of cause or detection of pelvic mass on examination.
2 -*Investigations:* the best is laparoscopy.
Treatment: of the cause.

Uterine factor of infertility

Causes:

A- *Congenital anomaly*: bicornuate, unicornuate, didelphys, hypoplastic/aplastic
B- *Acquired structural abnormalities*: fibroid, endometrial polyps, Asherman's syndrome, adenomyosis,
C- *Inflammatory & infectious*: chronic endometritis, tuberculous endometritis, PID
D- *Functional abnormalities*: thin endometrium, luteal phase defect
E- *Uterine vascular & immune factors*: uterine blood flow abnormalities, immune-mediated implantation failure.

Diagnosis:

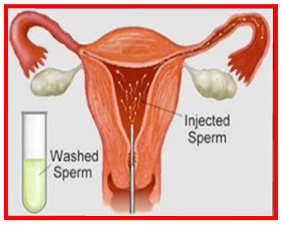
I- Clinical:
Symptoms: history of the cause e.g., vaginal discharge, amenorrhea, oligomenorrhea,
Signs: on exam, the causes may be detected e.g. hypoplastic uterus, prolapse…..etc.
II- Investigations:
1. Lab: C.B.C. and endometrial culture for infection.
2. Radiology: H.S.G and U.S.
3. Endoscopies: e.g., hysteroscopy and laparoscopy.
4. Histopathology: endometrial sampling and premenstrual endometrial biopsy.

Treatment: of the cause and ART

ASSISTED REPRODUCTIVE TECHNOLOGY (ART)

- It is any manipulation to male and female gametes to enhance the process of conception.

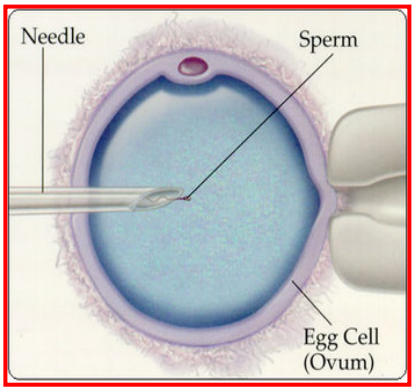
Types: there are many types e.g.
1- Gamete intra-fallopian transfer = G.I.F.T.
2- Zygote intra-fallopian transfer = Z.I.F.T.
3- In vitro fertilization and embryo transfer = I.V.F and E.T
4-Artificial insemination husband = A.I.H. i.e., non-coital
5-deposition of semen or sperms in the female genital tract.
6-Micro-insemination as S.U.Z.I = sub-zonal sperm injection, OR
I.C.S.I.= intra-cytoplasmic sperm injection followed by embryo transfer.
7- Oocyte donation and artificial insemination by donor's semen but prohibited by religious



IUI

"Of all these techniques, IVF-ET and ICSI are the most commonly used."

Steps of IVF-ET cycle:



- Initial consultation and assessment.
- Controlled ovarian hyper-stimulation.
- Trans-vaginal oocyte retrieval.
- Invitro fertilization of the ova.
- Embryo culture till 4-8 cell stages and then embryo transfer in uterine cavity

Complications:

1-Ovarian hyperstimulation syndrome
2-Complication of oocyte retrieval as trauma, hge, infection.
3-High failure rate.
4-High incidence of abortion, ectopic pregnancy,
5-More liability for twin pregnancy, pre-term labor, OR congenital anomalies.
6-High cost and highly experienced personnel need.

CONTRACEPTION

- Contraception means avoidance of pregnancy whether transient or permanent.

The ideal contraception should have the following criteria:

1. Cheap, available and easy to use.
2. Not needing medical supervision.
3. Not affecting sexual intercourse and protecting from sexually transmitted disease.
4. It has high efficacy and NO failure rate.
5. It has NO side effects OR complications.
6. Rapid regain fertility after its stoppage.

Types of contraception:

1-Physiological. 2-Hormonal. 3-Chemical "spermicidal".
4-Mechanical [barrier methods] and IUD.
5-Surgical methods [sterilization] = *permanent method.*

Measurement of contraceptive efficacy:

1-Pearl Index: it is the number of pregnancies per 100 women using the method per year (HW/Y).

2-Life Table Analysis:

- It calculates the failure rate for each month of use.
- It is more accurate and could compare different methods for a specific time.

Contraceptive Failure: it is measured by the failure rate of the contraceptive method. It may be:

1-Method failure: Failure rate with correct and consistent use of the method.
2-Use failure: Failure rate after incorrect use of the method by the client (typical use).

WHO Medical Eligibility Criteria for Starting Contraceptive Methods:

- The conditions affecting eligibility for the use of each contraceptive method were classified under one of the following **4** categories:

I. *Category 1*: there is no restriction on the use of the contraceptive method.
II. *Category 2*: the advantages of using the method generally overweighs the theoretical or proven risk so it is used but careful follow-up may be required.
III. *Category 3*: the theoretical or proven risks usually overweighs the advantages of using the method, so its use is not usually recommended.
IV. Category 4: a condition which represent an unacceptable health risk if the contraceptive method is used so the woman should not use the method under any circumstances.

For more information follow the link:

https://youtu.be/kUY3llqxSXA

PHYSIOLOGICAL CONTRACEPTION

1- Lactation Amenorrhea Method (LAM)

Mechanism of Action:

- Increased release of *prolactin* → inhibition of (GnRH) → suppresses the release of FSH and LH by pituitary gland and so suppresses ovulation.
- To use LAM ideally there should be:
1. Used in the first 6 months postpartum.
2. The infant should be exclusively breastfeeding and regularly to cause amenorrhea.

Advantages of LAM:

1. Easy, cheap and available method.
2. At least 98% effective if properly used.
3. Protection begins immediately postpartum i.e. rapid action.
4. There are proven health benefits of breastfeeding for mother and infant.
5. LAM can be used temporarily while a woman decides about other method to use.
6. Not affecting sexual activity OR needing medical supervision.
7. Have no side effects.

Disadvantages of LAM:

1. Difficult to use for some women due to social circumstances e.g. in working women.
2. If not properly used, there is high failure rate.
3. No STI or HIV protection.
4. Short duration of action "6 months when properly used".

2- Periodic abstinence

- It means abstaining from intercourse during a woman's fertile time. This is identified by:

1- The calendar or rhythm Method:

- Whereby a woman uses the length of menstrual cycles to calculate when she will be fertile.

2- The basal body temperature method:

- By noting the rise in basal body temperature that occurs just after ovulation.

3- The cervical mucus method:

- By noting changes in the appearance and texture of the cervical mucus.

4- The sympto-thermal method:

- In which several techniques are combined to predict when a woman is fertile.

5- Persona technique:

- It is urinary dipstick method, used at home depending on LH and estrone glucoronide detection.
- The change in color of the kits determines the fertile and infertile periods.

Red color= stop, green color = go on, yellow color= better stop or repeat test

Advantages of Periodic Abstinence:

- User-controlled not needing medical supervision and the patient could stop it at any time.
- Readily available, safe and free from side effects.

Disadvantages of Periodic Abstinence:

1. Require skills and motivation.
2. Signs of fertility may not be reliable.
3. Needing highly educated patient and regular cycles.
4. Requires partner's cooperation to abstain.
5. No STI/HIV protection.
6. Relatively high failure rates "10-20 HWY".

3-Withdrawal method "coitus interruptus:

- It requires removing the penis from the vagina just before ejaculation.

Advantages:

- Easy, cheap and available method.
- Not needing medical supervision.

Disadvantages of withdrawal:

- High failure rates of about 5-20% for perfect use.
- There is decreased sexual pleasure and satisfaction.
- Pelvic congestion for both partners as they cannot complete sexual intercourse.
- Provides no protection from STIs/HIV.

HORMONAL CONTRACEPTION

-It entails, combined types "estrogen and progesterone", OR progesterone only types.

1-Estrogen Component:

- Ethinyl Estradiol (EE) "the commonest used".
- Mestranol "not used as it is weaker and has many side effects".

2-Progestogen Components:

- Synthetic orally active agents and are more potent than original progesterone e.g.:
  - 19-nor-testosterone derivatives.
  - 17-alpha hydroxyprogesterone derivatives.
- The common types are:
  1. Combined oral contraceptive pills (COCs).
  2. Progesterone only pills (POPs).
  3. Injectable hormonal contraceptives.
  4. Hormonal contraceptive patches.
  5. Hormonal contraceptive vaginal silastic rings.
  6. Hormonal subdermal implants [capsules]
  7. Hormone containing devices [LNG-IUS].

Combined Oral Contraceptives (COCs)

- The most widely used type contains both estrogen and progestogen.
- This type is evaluated in *4 generations* depending on changing the dose of estrogen and progesterone aiming for simulating the normal cycle.
- These types of COCs are:

1. *Monophasic pills:* it contains the same dose of EE & progestogen each day for 21 days.
2. *Sequential pills:* progesterone is added in the third week.
3. *Biphasic pills* "not used nowadays":
   It delivers the same dose of estrogen throughout the cycle **BUT** progesterone is increased half way through the cycle.
4. *Triphasic pills:* to mimic the normal cycle

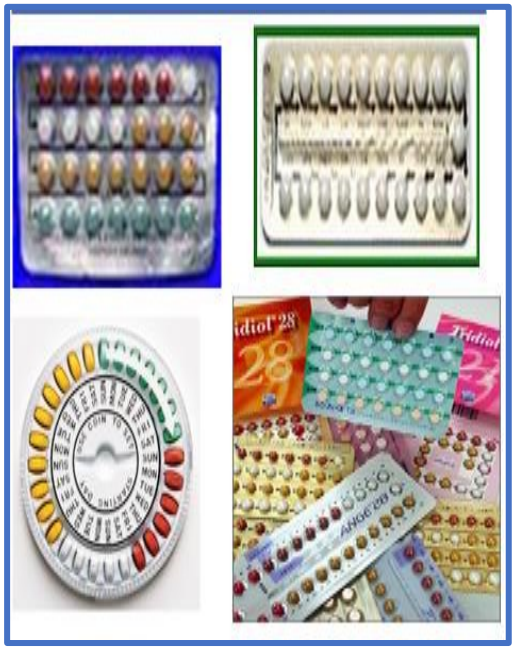


Table of days	*Dose of EE*	*Dose of progestogen*
First 6 days	30 ug	50 ug
Next 5 days	40 ug	75 ug
Next 10 days	30 ug	125 ug

Mechanisms of Action:

1. Suppression of ovulation by inhibition of the follicle-stimulating hormone (FSH) and luteinizing hormone (LH) "this is the primary mechanism".
2. Changing in tubal motility thus interfering with fertilization.

3. Changes in the endometrium rendering it improper for implantation.
4. Thickening of cervical mucus so impermeable to the sperms "action of progesterone".
5. Inhibition of sperm capacitation so interfering with fertilization "action of progesterone".

How to Take COCs:

Initiating:

- During the first 7 days of the menstrual cycle, preferably on the first day of menses.
- Given at any time provided that the pregnancy is surely excluded.
- For **NON**-breastfeeding women postpartum: delay until 6 weeks.
- For breastfeeding women postpartum it cannot be used as estrogen component can reduce breast milk [so it can be used when breastfeeding is discontinued].

Schedule:

- Take 1 pill every day until all pills in the pack are finished (21 pills).
- Seven-days break, then start another pack on day 7.
- Withdrawal bleeding usually occurs on the 2nd or 3rd day.

Missed Pill Regimen

Missed (1) pill:

- Take missed pill as soon as remembered.
- Keep taking remaining pills on schedule.
- No back-up method needed.

Missed (2) or more pills:

a. If 7 or more pills are left in the pack:

- Take 1 pill immediately and then take the remaining as usual.
- Backup method (condom or spermicidal) is used for 7 days.

b. If there are less than 7 pills left in the pack:

- Take 2 pills immediately and 2 pills at the usual time.
- Some advice to start another pack on the next day.
- Use backup method (condom or spermicidal) is used for 7 Days.

Advantages:

1. Safe (serious complications are extremely rare).
2. Highly effective when used correctly and consistently it is **99%** success rate.
3. Reversible and rapid return of fertility after its stoppage.
4. No action needed at the time of intercourse i.e. not affecting sexual activity.
5. Have beneficial health effects other than contraception.
6. Use is controlled by the woman i.e. stop it when needed.

Non-contraceptive Health Benefits of (COCs):

1. Reduced risk of ovarian and endometrial cancers.
2. Reduced risk of benign breast diseases.
3. Reduced risk of ectopic pregnancy.
4. Reduced menstrual irregularities and blood loss so reduced risk of anemia.

5. Reduced symptoms of dysmenorrhea, endometriosis, premenstrual syndrome and PID.

Disadvantages of COCs:

1. Incorrect use and pill missing is common so may be not effective as in typical use.
2. Require daily pill intake and re-supply.
3. Side effects are common.
4. Offer no protection against sexually transmitted infections including HIV.

Side Effects:

1. Nausea, vomiting, dizziness, headache, mood changes and weight gain.
2. Breast tenderness and increased risk of breast cancer.
3. Menstrual irregularities: spotting, breakthrough bleeding and amenorrhea.
4. Increased risk for cardiovascular complications such as thrombosis and stroke.
5. Small increase in the risk of cervical cancer.
6. Impaired liver function on and may predispose to begin or malignant liver diseases.

WHO Medical Eligibility Criteria for starting (COCs):

1. Women with the following conditions should NOT use COCs [absolute contraindications]:

1. Pregnancy as it may cause CFMF.
2. Heavy smokers.
3. Severe hypertension, complicated diabetes.
4. Current or past history of thromboembolic disorders or ischemic heart diseases.
5. Valvular heart diseases with complication or prolonged immobilization.
6. Continuous migraines.
7. Unexplained vaginal bleeding.
8. Family OR patient history of breast cancer.
9. Epilepsy, active liver disease, cirrhosis or liver tumors.
10. Breastfeeding mothers.

2. Women having the following conditions may use COCs only after consideration of alternative methods and with careful medical evaluation and supervision [relative contraindications]:

1. Age 35 and more.
2. Light smoker.
3. Mild and moderate hypertension.
4. Gallbladder disease.
5. Current treatment with some antibiotics (rifampin, griseofulvin) or antiepileptic drugs.
6. Non breastfeeding women during first 6 weeks after childbirth.

2. Progestin-only Pills (POPs)

Contain only a progestogen, mostly levonorgestrel, so suitable for breastfeeding women.

Mechanism of action:

- As mentioned before in COCs but the main action is peripheral by changing cervical mucus.

How to Take POPs:

Initiating:

- Postpartum breastfeeding women; start after 6 weeks.
- Postpartum non-breastfeeding women, start immediately.
- Menstruating women:
  a). Preferably in first **5** days of menstrual cycle.
  b). Can start at any time provided that the woman is not pregnant.

Schedule:

- Take 1 pill each day and better within **3 hours** of the same time each day.
- No break between packs.

Missed Pill(s) Regimen (if more than 3 hours late taking the pill)

- Take the most recent missed pill as soon as possible.
- If not breastfeeding, abstain from sexual intercourse or use back up method (condom or spermicidal) for 48 hours.
- Take the next pill at regular time.

Disadvantages:

1. Menstrual irregularities: e.g. spotting or irregular bleeding, or amenorrhea.
2. Needing daily re-supply.
3. Not protecting against STI and HIV.
4. It has some side effects e.g. weight gain, acne, headache, more liability to ectopic pregnancy.
5. Less effective than COCs "about 3% failure rate".

Contraindications:

1. Pregnancy.
2. History of ectopic pregnancy.
3. Unexplained vaginal bleeding or breast cancer.
4. Gall bladder disease, active viral hepatitis, liver cirrhosis, liver tumors.

Injectable Contraceptives

Types of Injectable Contraceptives:

1- Progestin-only Injectable:
- *Depo-provra* = Depot-Medroxy-Progesterone Acetate 150 mg (DMPA): used every 3 months.
- *Norethisterone enanthate 200 mg (NET-EN):* used every 2 months.

2- Combined injectable: not used now for their side effects.
- Mesygyna and Cyclofem.

Mechanisms of Action of progesterone only contraceptives:

- Thickening of cervical mucus making it difficult for sperms to enter the uterine cavity.
- To a less extent inhibition of ovulation.

How to Administer Progestin-only injectable:

Initiation:

- Preferably within the first **7** days of the menstrual cycle.
- May be initiated later provided that the patient is surely not pregnant.
- If given after day **7** of the cycle, a back-up method is considered for 2 days following injection.
- Postpartum:
  1. Non-breastfeeding: start immediately or at any time within the first 6 weeks after childbirth.
  2. Breastfeeding: delay to 6 weeks after labor.
  3. Post abortion: start immediately or within the first 7 days after abortion.

Advantages of Progestin-only injectable:

1. Safe and highly effective (more than 99%).
2. Long acting, but reversible.
3. Require no action at the time of intercourse.
4. Have no effect on lactation and can be used by breastfeeding women.
5. Offer non-contraceptive health benefits.

Disadvantages of Progestin-only injectable Contraceptives:

1. Side effects are common, especially menstrual changes.
2. Return to fertility after discontinuation is usually delayed "about 4 months more than COCs.
3. No STI/HIV protection.

Side Effects of Injectable Contraceptives:

1. Menstrual changes: common with progestin-only injectable e.g. spotting, irregular bleeding, prolonged or heavy bleeding or amenorrhea.
2. Weight gain, breast tenderness, headache, dizziness, and mood changes.
3. DMPA may accelerate the development of pre-existing breast cancer.
4. DMPA users have lower bone density than non-users especially women aged 21 or younger but bone loss may be reversible and remain at increased risk of developing osteoporosis later in life.

Non-contraceptive Health Benefits of Progestin-only Injectable:

1. Reduced risk of:

a) Fibroids, endometrial cancer and possibly ovarian cancer.

b) Vaginal yeast infection (moniliasis) and PlD.

2. Reduced frequency and severity of sickle-cell disease crises.

3. Reduced symptoms of endometriosis.

Absolute contraindications of progestin-only injectable:

1-Pregnancy.

2-Unexplained vaginal bleeding.

3-Breast cancer (current or previous)

Relative contraindications for progestin- only injectable:

1. Hypertension, current or past history of controlled ischemic heart disease.
2. Diabetes.
3. Past cerebrovascular accident (stroke).

4-Active liver disease, cirrhosis, liver tumors.

Sub-dermal Contraceptive Implants

- Sub-dermal contraceptive implants are capsules placed by a special applicator through a small incision under the skin of the inner side in upper arm, slowly release progestin into blood stream.

NORPLANT

- The most widely used sub-dermal implant.
- It is formed of 6 match-sized capsules OR a 2 rod system "Javelle" and implanted to deliver low doses of **levonorgestrel** to the blood stream for 5 years.

Mechanisms of Action:

1. Thickening of cervical mucus, making it difficult for sperms penetration.

2. Preventing ovulation in about half of a women by inhibition of FSH and LH secretion.

Advantages:

1. Norplant is safe at least 99% effective in preventing pregnancy.

2. Easy to use "one application", with no action needed at the time of sexual intercourse.

3. It is long-acting, begins within a few hours after insertion and lasts for 5 years.

4. Reversible at any time by removing the capsules, rapidly restoring fertility.

5. It contains no estrogen and can be used by women who are breastfeeding 6 weeks after labor.

6. Provides non-contraceptive health benefits, such as:

b) Decreased menstrual blood loss.

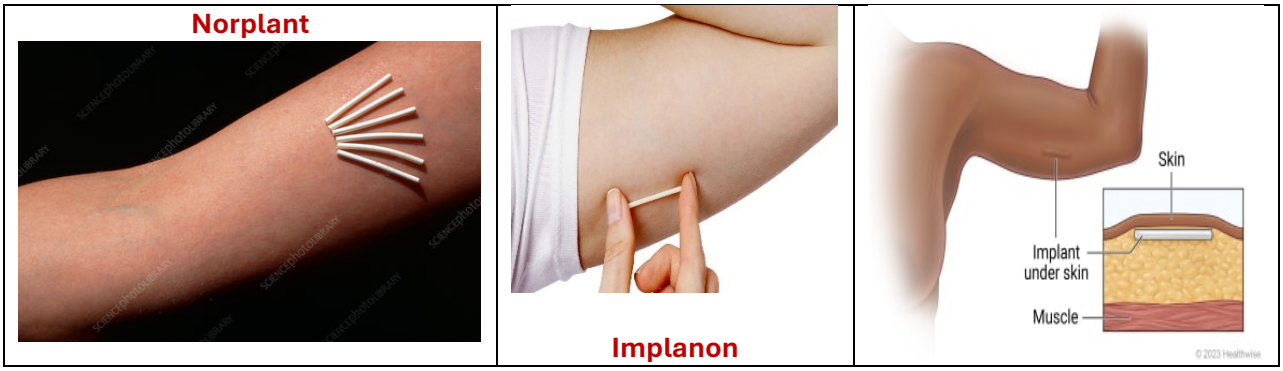
c) Possibly decreased risk of endometrial hyperplasia and cancer.

Disadvantages:

1. Side effects are common, especially menstrual changes.
2. It cannot be initiated or discontinued without a provider's help i.e. needs medical stuff.
3. A minor surgical procedure is required for both insertion and removal.
4. Provides no protection against STIs, including HIV.

Side Effects:

1. Bleeding and menstrual irregularities but decreases after the first few months.
2. Less common "headache, breast tenderness, and weight gain".



IMPLANON

A progestogen-only sub-dermal implant that consists of a single non-biodegradable rod measuring 40 mm in length and 2 mm in diameter that release 40 micrograms **etonogestrel** per day.

- It is inserted into a subdermal by a special applicator in the inner side of the upper arm.
- It provides highly reliable **protection**, nearly **100%**, for up to **3** years.
- As Norplant, the method is free of estrogen and so it is suitable for breastfeeding mothers.

Mechanism of action:

1. Mainly by inhibition of ovulation.
2. Thickening of the cervical mucus.

Side effects:

1. Amenorrhea "more common" and irregular bleeding pattern.
2. Weight gain, acne, headaches, and breast tenderness.

CONTRACEPTIVE VAGINAL RING

- It is a flexible, soft, transparent silastic ring that releases hormones.
- It can be easily inserted and removed by the women herself.
- They are placed in the vagina high up around the cervix to release hormones to be absorbed via lymphatics and blood so less liable for GIT troubles.
- The common causes for discontinuation are a foreign body sensation, coital problems and expulsion of the device. There are two types:

1- Combined vaginal ring:

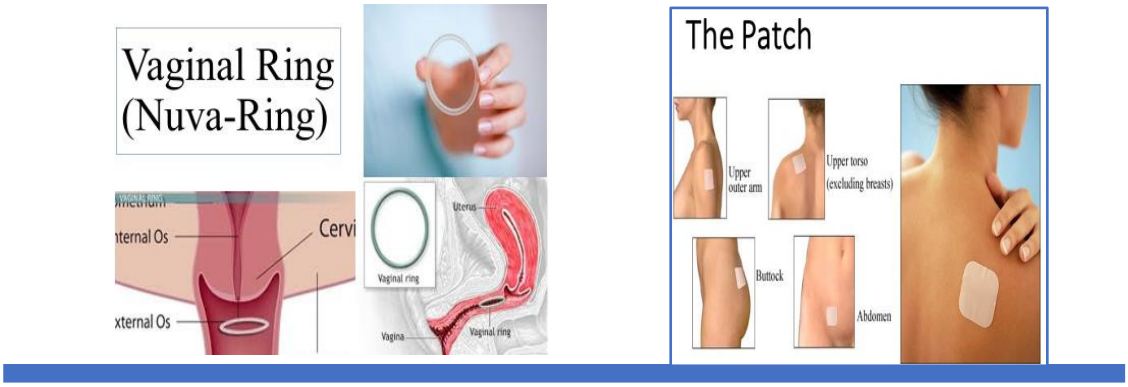
- It contains EE + levonorgestrel "the dose is sufficient for six months".
- Inserted in the vagina on the 5th day of the cycle and removed 3 weeks later to allow menses.

2-Progesterone only vaginal ring:

- It contains levonorgestrel and used every month or every 3 months according to the dose.
- The failure rate is high.

Combined transdermal Hormonal Contraception " Ortho Evra"

- A combined patch containing (progestin and EE) working in the same way as COCs.
- Applied weekly to anybody location except the breast for 3 weeks and then one week free.
- The same mechanism of action as COCs but with more side effects due to weekly use.



Intrauterine Contraceptive Devices (IUCDs)

Types of IUCDs:

1-Recent medicated types:

i. Copper containing types "copper-T and copper-7"

ii. Anti-bleeding agent containing IUCDs "to decrease blood loss during menses".

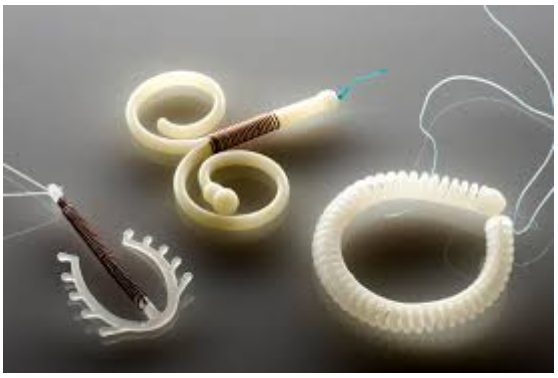
iii. Progesterone containing IUCDs:

a. Progestasert "in common use as a contraceptive".

b. Levonorgestrel containing IUCD **"Mirena" [**used in fibroid, adenomyosis and abnormal uterine bleeding**]**.

2-Multi-load containing IUDs.

- Most intrauterine devices (IUDs) being inserted today are copper T -380 A and Ag "containing copper wire and a silver core".
- Copper T 200, copper T220, Nova T "Nova Gard" and copper 7 are less used.
- The number indicates the surface area of copper in mm2.



Mechanism of Action:

1. The main action:

- Local sterile inflammatory reaction so leucocytes and macrophages engulf ascending sperms, descending ova and preventing implantation of already fertilized ovum.

2. Other actions:

- Utero-tubal spasm so preventing meeting of sperms and ova.
- Mechanical interruption for the ascent of the sperm from the cervix.
- Production of PGs which affect more and more utero-tubal motility.
- Increased level of Abs and immune globulins which prevent implantation.
- In progesterone releasing IUDs, the action of hormone is added so the efficacy is more.

Timing of IUD Insertion

1. Interval insertion:

- It is inserted 1-2 days after menses to ensure opened cervix, and absence of pregnancy.
- It is inserted by pushing technique OR pulling technique [the preferred method].

2. Postpartum insertion:

- Immediately after vaginal delivery (within 10 minutes after delivery of placenta), or during the cesarean section by manual application.
- May be used within the first 24 hours following vaginal delivery as the cervix is opened.

3. Insertions after abortion:

- If the pregnancy was 16 weeks or more, IUD insertion should be performed immediately by a specially trained provider or be delayed 4 weeks.

WHO Medical Eligibility Criteria for Starting IUD Use:

(A)-Absolute contraindications:	(B)-Relative contra-indications:
1-Pregnancy. 2-Unexplained vaginal bleeding. 3-Cervical, endometrial, or ovarian cancer. 4-Current or recent (PID), STIs, septic abortion, or pelvic TB. 5-Distorted uterine cavity "as anomaly or fibroid".	1-needs careful medical evaluation and supervision. 2-Those with risk of developing STIs, or to have ectopic pregnancy.

Advantages:

1. Safe, highly effective (more than 99%).
2. Easy to use, requiring no action at the time of intercourse or at any other time.
3. Can be removed easily by a service provider gently by pulling on the nylon threads.
4. Long acting:
   - **10** years in copper T 380.
   - **7** years in Mierena.
   - **3** years in multi-load and copper 7.
   - **1** year in progestasert.
5. Easily reversible, with return to fertility very soon after removal.
6. No systemic side effects.
7. Can be used safely by breastfeeding women.
8. Complications are relatively rare.

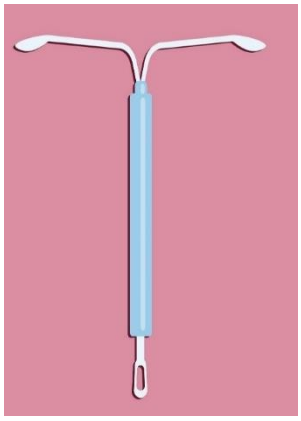
Disadvantages:

1. Can cause some side effects.
2. A trained healthcare provider is needed to insert and remove the device.
3. Offer no protection against STIs, including HIV.

Hormone-Releasing Intrauterine System (IUS)

- IUS is a newer form of hormonal containing IUDs **(e.g., progestasert and Mirena).**
- It can be used if a woman develops excessive bleeding with copper IUD and still want to use an intra-uterine device **BUT** the provider must rule out any condition contraindicating progestin use.

Mechanisms of Action: action of progesterone + IUD.



Specific advantages of IUS

-It has non-contraceptive health benefits as: reducing the duration and number of menses, its pain and it relative decrease in PID than ordinary types.

Specific disadvantages:

1. Expensive.
2. Ectopic is commoner than ordinary type of IUDs due to PID and relaxing effect of progesterone.

Complications of IUDs:

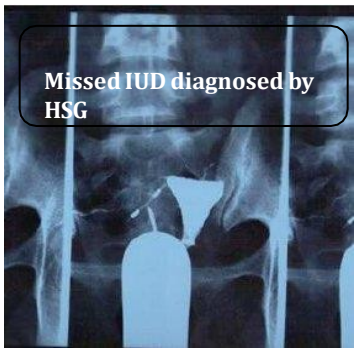
1-*Pelvic Inflammatory Disease* (PID).

2-*Perforation*: rare but a potentially serious event and its risk is increased with *decreased skill* and *experience* of the provider, and in *postpartum insertions*. "It is known by severe lower abdominal pain and neurogenic shock and should be treated immediately".

3-*Ectopic pregnancy*: more common in progesterone containing types than IUD alone due to "PID and associated relaxation of the tube".

4-Pregnancy on Top of IUD:

-Pregnancy can occur with IUD in place or after unnoticed expulsion.
-Spontaneous abortion can occur in 50% of cases with the IUD in place and usually complicated by infection (septic abortion).
-It is advised to remove IUD as soon as the pregnancy is confirmed to decrease abortion to 25%.
-No increased risk of congenital malformations.
-Increased risk of preterm labor (4 folds) if the IUD is left in place.

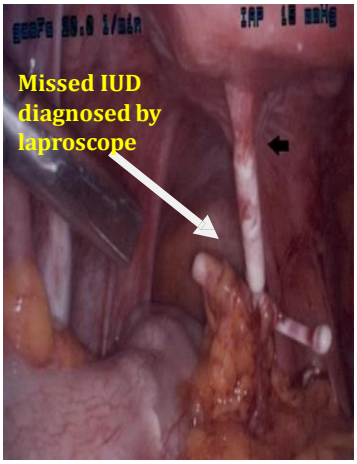


5-Inability to Feel the Threads (missed IUD): due to --

a). Unnoticed expulsion of the IUD.
b). Dislocation of the IUD in the uterine cavity.
c). Perforation of the uterus.
d). Pregnancy.
e) Threads are adherent to the cervix or vagina by thick discharge.

Management of missed IUD

-Do P/V and speculum examination to visualize the threads if adherent to the cervix or vagina.
-Explore the cervical canal by uterine sound or Bozeman forceps for retracted threads.
-Exclude pregnancy by pregnancy test or U/S.
-With -ve pregnancy test; do sounding of the uterus to feel the click of intrauterine IUD.
-Hysteroscopic examination to diagnose and remove dislocated intrauterine IUD.
-Abdominal-pelvic U/S or plain x ray to diagnose extra uterine IUD.
-Laparoscopy for the diagnosis and extraction of extra uterine IUD.



6-Expulsion of the IUD:

-Partial or unnoticed expulsion may result in: irregular bleeding, pain and pregnancy.
-Factors affecting expulsion rates include: providers skills, young age, nulli-parity and length of time since insertion.

7-Menstrual abnormalities: menorrhagia due to pelvic congestion, or metrorrhagia.

8-Discharge: with PID or due to pelvic congestion.

9-Pelvic discomfort and dyspareunia: with infection and congestion.

10-Discomfort to male: with long threads.

Barrier Methods

Barrier methods of contraception include: "all are made of polyethylene material".
1-The male condom "male sheath or French letter".
2-Female condom.
3-Vaginal diaphragm "Dutch cap".
4-Vaginal sponge [barrier and spermicidal].
5-Cervical cap.

Mechanism of Action: These methods work by physically or chemically blocking the passage of sperm.

Advantages:

1. Effective in preventing pregnancy if used consistently and correctly.
2. Condoms can be effective in preventing STIs, including HIV.
3. Safe and have no systemic side effects.
4. Easy to initiate and discontinue.
5. Immediate return to fertility after stopping method.
6. Except for the **cervical cap**; barrier methods does **NOT** require a clinic visit.

Disadvantages:

1. All have high failure rate [10-20 %]
2. Needs consistent and correct use with each sexual act "**EXCEPT** cervical cap one use for a month".
3. Some methods require partner's participation.
4. They interrupt sexual activity especially condoms.
5. Need proper storage in order to maintain the quality of the products.

Characteristics of Barrier Methods

Condoms:

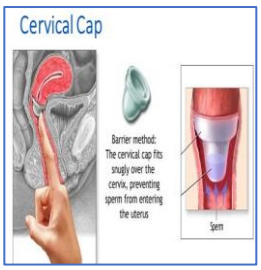
-They are widely used and more effective when spermicides are added.
-Proven *protection* against STIs, including HIV.
-Requires partners' cooperation.
-Recently non latex male condom and natural condoms are available to improve feeling and durability.

Diaphragm:

-The Diaphragm fits obliquely in the upper vagina and is more effective when used with spermicides.
-First time must be fitted by a trained provider.
-Reusable (after cleaning), inserted up to 6 hours before intercourse and should not be removed earlier than 6 hours after intercourse; no douching after intercourse or before removal.
-It may cause irritation and urinary tract infection.
-Now *Silicone diaphragm* for easy fitting is available.

Vaginal sponge:

- It is a type of birth control that prevents sperm from entering the uterus by mechanical method and contains spermicide, which kills sperm. The sponge doesn't protect against STIs.



Cervical cap:

- It is applied by a doctor on the external os after menses and removed before the next expected one.
- The main advantage is that it has longer time than others [one month].
- In addition to the advantages mentioned before, it needs a medical provider that inserts and removes it every month.

Spermicides = chemical contraception

- These are chemicals that contain the active ingredient Nonoxynol-9
- They work by inactivating sperm [direct killing] or immobilizing it [to be killed by vaginal acidity].
- They present as foaming tablets, foam, cream, vaginal suppositories or gel.
- Despite it easy, cheap and available **BUT** much less effective than other modern methods.
- Provide modest protection against bacterial STIs.
- It can increase urinary tract infection and usually associated with chemical vaginitis.

Surgical Methods for Contraception = Sterilization

A. Female Sterilization (Tubal Occlusion)

- Voluntary female sterilization is used worldwide.
- It involves surgically closing and cutting or clipping the fallopian tubes to prevent the ovum from being fertilized.

Advantages:

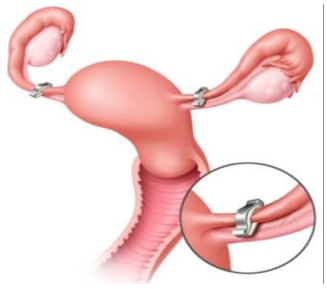
1. Highly effective (99.5% in first year of use, 98.1% overall for 10 years of use).
2. Permanent method.
3. Virtually no long-term adverse effects.
4. No effect on normal sexual function.

Disadvantages:

1. Exposes clients to a risk of surgical complications such as injury of organs, infection or bleeding.
2. Offers no protection against STIs, including HIV.
3. Cannot be reversed if the woman changes her mind.
4. It has relatively high initial cost.
5. Post-sterilization syndrome [as pelvic congestion and pelvic pain].

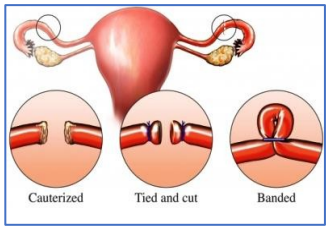
Timing of Female Sterilization:

- Immediately or within the first 7 days after a vaginal delivery (postpartum) or during C.S.
- At any other time between pregnancies.

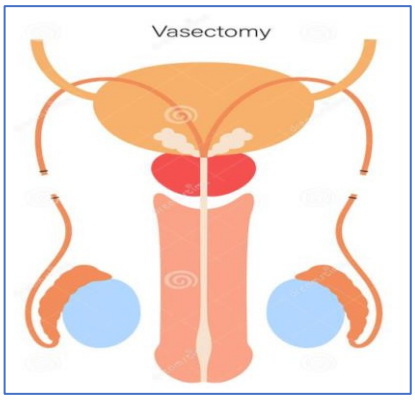


Routes of Female Sterilization:

1. Laparoscopy: via
   - **a.** Cauterization of segment of the tube.
   - **b.** Application of Fallop ring or Hulka clips.
2. Mini laparotomy:
   - **a.** Postpartum mini laparotomy by a small sub-umbilical incision.
   - **b.** Interval mini laparotomy via a small supra-pubic incision.
3. At the time of CS or other abdominal operations: via
   - **a.** Pomeroy's Technique.
   - **b.** Uchida's Technique.
   - **c.** Irving's Technique.
   - **d.** Fimbriectomy.
4. Vaginal route: as Posterior colpotomy to get the tubes by opening the Douglas pouch.
5. Hysteroscopic route: via injection of chemical agents to cause scarring and blocking of the proximal end of the tubes.



B. Male Sterilization (Vasectomy)



-Voluntary male sterilization "vasectomy or vas ligation" is a minor surgery where it is tied/cut or both to prevent sperms from mixing with the seminal fluid.
-Vasectomy is technically simpler to provide than female sterilization but not offered worldwide.
-Vasectomy is a permanent method of contraception **BUT** not immediately effective.

-A waiting period of 12 weeks or 20 ejaculations is recommended before couple can rely on vasectomy to prevent pregnancy.

Emergency Contraception

-Emergency contraception is the use of certain methods after unprotected intercourse as in:

1. No method used as during coercive sex and rape.
2. Condom tears or rupture during sexual activity.
3. Methods used incorrectly, such as missed pills or failure of withdrawal method.

- Methods used for these purposes are:

Emergency Contraceptive pills

(a)- *Progestin-only Pills Regimen:*

The first dose (0.75 mg of levonorgestrel) should be taken as soon as possible and not beyond 72 hours and repeated 12 hours after first dose.

(b)*- Combined Oral Contraceptive Pills Regimen:*

Four tablets of the standard low-dose COCs is taken as soon as possible and not beyond 72 hours and 12 hours after first dose.

- *The sooner EC pills are taken after unprotected intercourse the more effective they are.*

Possible Side Effects of ECPs: Nausea, vomiting, headache, dizziness, fatigue, breast tenderness, irregular bleeding and spotting.

Copper IUD for emergency contraception:

- Inserted within **5** days after unprotected intercourse [highly effective "0.1% pregnancy rate"].

Menstrual aspiration:

It is done using "Karman's cannula" and performed 1 week after intercourse or within 1-2 weeks of the missed period to aspirate uterine contents?! "Unreligious and not used".

Selection of a contraceptive for a special group

Contraception in elderly patients:

1-COCs containing low dose estrogen may be used in females do not risk for thrombosis, hypertension, or diabetes "but better **avoided** when other methods available".
2-POPs: considered ideal method in non-obese patients and those not liable for hypertension and thrombosis. They can protect against endometrial hyperplasia, carcinoma, and shrinks fibroid if present.
3-Injectable progesterone, sub-dermal capsules.
4-IUD and intra-uterine system "IUS" can be used.
5-Barrier method also used but with high failure rate.
6-Tubal sterilization if the patient completes her family.

Post-partum contraception:

1-Breast feeding Women "NO USE FOR COCs OR COMBINED INJECTABLES".
2-LAM: it is a method that act immediately postpartum and continue for at least 6 months.
3-IUDs and hormonal IUS.
4-Barrier methods BUT with high failure rate.
5-POPs, progestin-only injectable, sub-dermal implants (Norplant or Implanon).
6-Other physiological methods may be used but better avoided due to failure rate.
7-Female sterilization (if there is a medical indication or completing her family size).

Note that in non-breastfeeding women: →All methods could be used including (COCs) so as no contraindications.

Contraception in newly married couples:

1-Natural methods usually fail in this group due to high sexual activity.
2-COCs: is the ideal method "high efficacy, regular cycle, rapid regain to fertility after stoppage".
3-Progesterone only: better avoided due to irregular cycles and delayed regain to fertility.
4-IUD: NEVER to be used as it causes PID and affects the patient future fertility.
5-Sterilization: NOT to be discussed with the patient.

Contraception in hypertensive or cardiac ladies:

1-Hormonal are better avoided due to its complications, if the patient is controlled and hormonal methods are to be used, strict follow up is a must.
2-IUD may be used with careful follow up to avoid bleeding or infection and SBE.
3-Barrier methods could be used safely but with expect high failure rate.
4-Tubal sterilization: is **the best** if the patient completed her family.

Contraception in diabetic ladies: as cardiac and hypertensive

PART THEE

UROGYNECOLOGY

- **Genital Displacement**
- **Stress urinary incontinence**
- **Genito-urinary fistula**

GENITAL DISPLACEMENTS

Definition:

Abnormal position of the genital organ (s) away from the normal anatomical level.

Common Types of Displacements:

1. The uterus is not central in the pelvis:

1. **Dextro-position** = dextrorotation if deviated to the right.
2. **Levo-position** = levorotation if deviated to the left.
3. **Ante-position:** shifted anteriorly.
4. **Retro-position:** shifted posteriorly.

2. Prolapse: vertical downward descent of the genital organs below the anatomical level.

3. RVF: loss of ante-version flexion and shifted to the back.

4. Inversion: turning of the uterus from upside down.

PELVIC ORGAN PROLAPSE

Definition: Pelvic organ prolapse (POP) can be defined as descent of the pelvic organ(s) ; bladder, rectum, vagina, uterus; below their normal anatomical position due to distortion of their **dynamic** and **integrated** support.

Anatomical types and classifications:

-May be vaginal, uterine or combined.

A- **Vaginal**: - Anterior wall (cystocele, urethrocele or combined)
- Posterior wall (rectocele, high or low)
- Vaginal vault prolapse (in presence of the uterus →enterocele or hernia of Douglas' pouch), (after hysterectomy -→ pos-hysterectomy vaginal vault prolapse).

B- **Uterine** : - **false** → congenital elongation of portio vaginalis of the cervix
-True → actual decent due to distortion of support

.

C- **Combined**: - **-Utero-vaginal:** if the uterus descends first, this occurs in congenital weakness of support (congenital weakness of mesenchyme; virginal or nulliparous prolapse).
- **Vagino-uterine:** if the vagina descends first, this occurs in acquired weakness or distortion of support e.g obstetric trauma.

STAGES /DEGRESS OF POP:

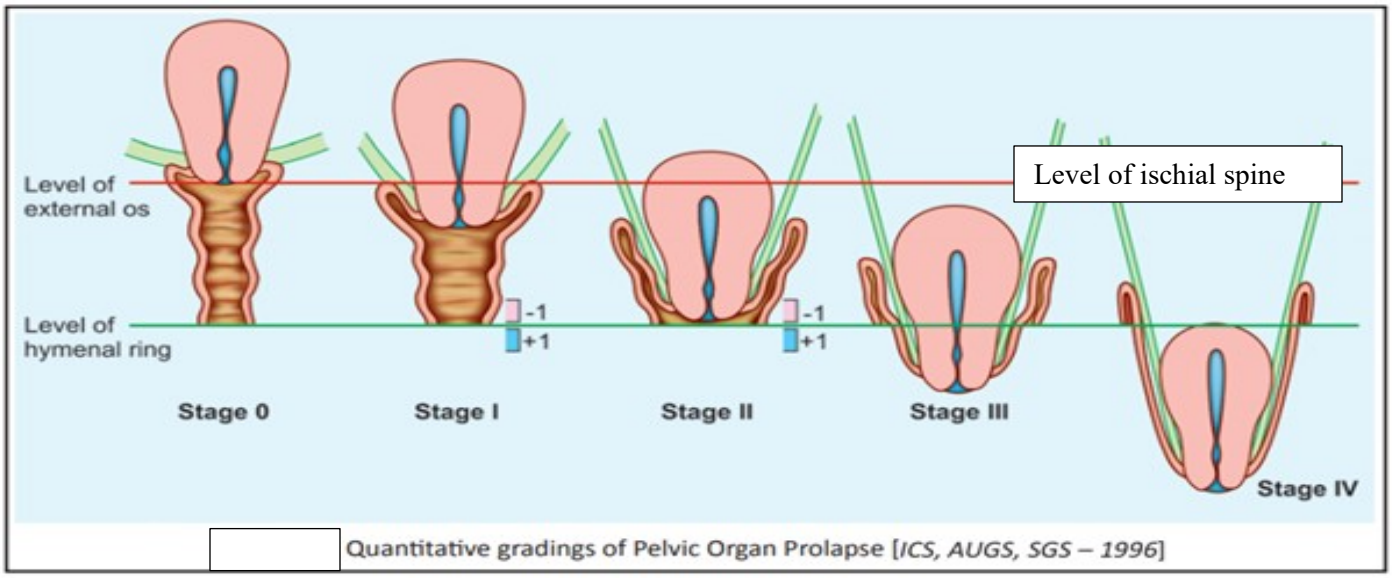
A- Old classification: considering the **uterine prolapse** mainly (on bearing down):

Stage 0 = no prolapse, the cervical os at or above level of ischial spine

Stage 1= the cervical os below the level of ischial spine but still inside the vagina

Stage 2= the cervical os is at the level of the vaginal introitus
Stage 3: the cervix is outside the vaginal introitus but the uterus still in the vagina

Stage 4: (complete procidentia) the whole uterus & the cervix are palpated outside the vaginal introitus.



Quantitative gradings of Pelvic Organ Prolapse [*ICS, AUGS, SGS – 1996*]

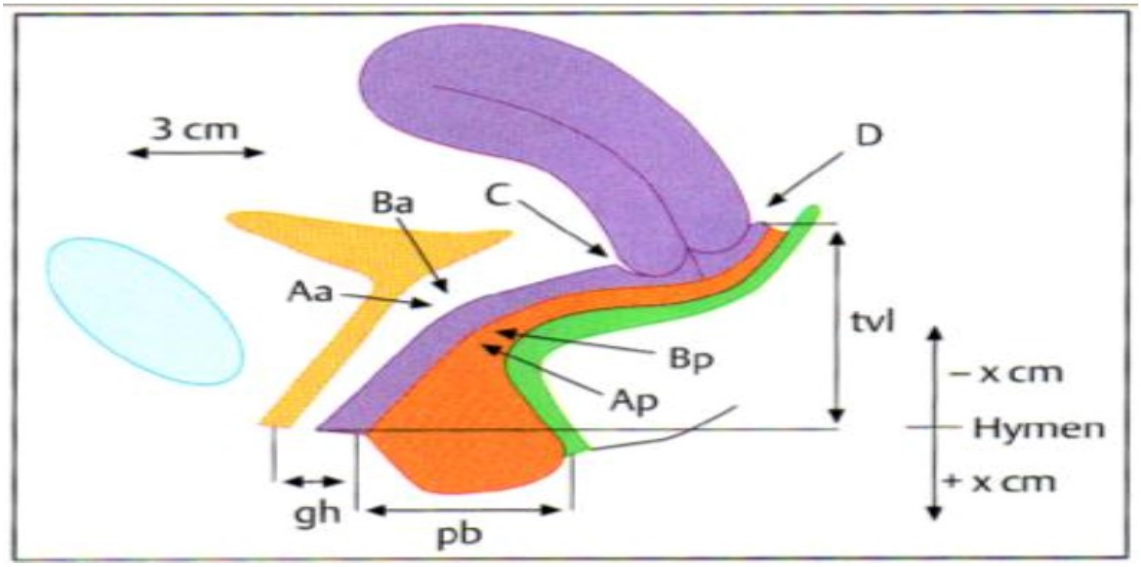
B- <u>POP-Q (Pelvic Organ Prolapse-Quantification) system:</u>

This system aims to 'quantify' POP whatever the organ, making 6 points & 3 measurements. After putting the measurements into the POP-Q grid staging is done.
<u>The points are</u>: **-Aa** = a **fixed** point on the **anterior** vaginal wall 3 cm above the external urethral meatus corresponding to urethro-vesical junction.
-Ap= a **fixed** point on the **posterior** vaginal wall 3 cm above the hymenal plane
-Ba & Bp = dynamic points; the lowest points of the prolapse between **Aa anteriorly** or **Ap posteriorly** and the **vaginal apex**.
- **C**= the cervix uteri (after hysterectomy it denote the vaginal **C**uff)
-**D**= denotes the highest point in the posterior vaginal fornix (Douglas' pouch), after hysterectomy this point no longer exists.

<u>The distances are</u>: **gh**= genital hiatus = Middle of external urethral meatus to the posterior hymen
Pb= perineal body = Posterior margin of genital hiatus to mid-anal opening.
tvl= total vaginal length = Greatest depth of the vagina in centimeters. C and D in normal position. Measurement without straining.

Aa (Anterior wall) =(-3cm)	**Ba (Anterior wall)= (-3cm)**	**C (Cervix /cuff) = (-6cm)**
gh (genital hiatus)= 2cm	**Pb (Perineal body) =3cm**	**Tvl (Total vag length) =10cm**
Ap (Posterior wall)= -3	**Bp (Posterior wall) = -3**	**D (Posterior fornix) = -10**

Note that any measures proximal to the hymenal plane (standard point) is considered minus e.g. -3 cm while measures distal to hymenal plate is considered positive e.g. + 3 cm

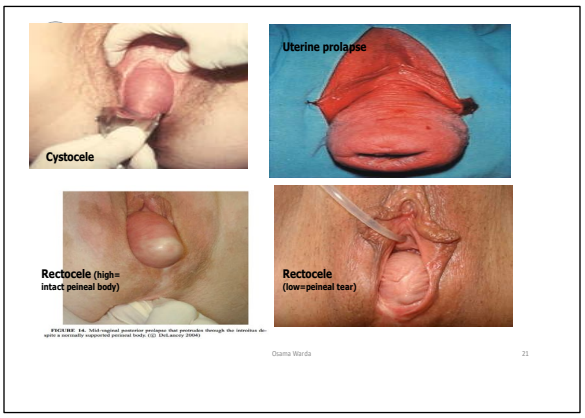


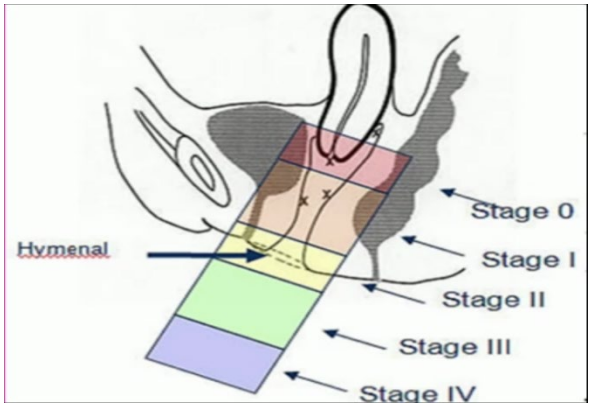
Points and landmarks for POP-Q system examination. Aa, point A anterior, **Ap**, point A posterior, **Ba,** point B anterior; **Bp**, point B posterior; **C,** cervix or vaginal cuff; **D**, posterior fornix (if cervix is present); **gh**, genital hiatus; **pb**, perineal body; **tvl**, total vaginal length.

Osama Warda 8

STAGES OF POP: Once the measurements are taken, the patients are assigned to the corresponding stage:

- **Stage 0** = no prolapse during straining.
- **stage I** = most distal portion of prolapse is > 1cm above level of hymen
- **Stage II**= the most distal part of prolapse is <1cm proximal to or distal to the plane of hymen
- **Stage III**=the most distal portion of the prolapse protrudes more than 1 cm below the hymen but not all of the vagina has prolapsed.
- **Stage IV**=complete vaginal eversion is essential.





Symptoms of POP

- POP can cause symptoms directly due to the prolapsed organ or indirectly due to organ dysfunction secondary to displacement from the anatomical position.
- **Direct POP symptoms include:**

(1) a sensation of vaginal bulge,

(2) heaviness or a visible protrusion at or beyond the introitus.

(3) lower abdominal or back pain, or a dragging discomfort relieved by lying or sitting.

- **Indirect symptoms:** will depend on which other organs are involved in the prolapse. They include:

(1) difficulty in *urination* or *defecation* (obstructive defecation)

(2) sensations of *incomplete emptying* of bladder or rectum.

(3) Patients *may have to support or reduce the prolapse* with their fingers to be able to void or evacuate stool completely (i.e. digitation)

(4) Urinary or fecal *incontinence* may also be present

(5) Sexual dysfunction due to introital laxity

(6) Bleeding from abrasion of the prolapsed part

Signs of POP:

-**General Exam**. To exclude chronic disease or causes for increase intraabdominal pressure
-**Abdominal Exam** to diagnose abdominal swellings, masses ascites, or hernia.
- Local gynecologic Exam: **inspect** for masses protruding from the vulva at coughing or straining, for escape of urine from the urethra, for any abnormality associated. **Palpate** for the mass whether reducible or not size of the mass measure the points and distances blotting in the POP-grid for staging. **Bimanual exam** to evaluate the size, and position of the uterus as well as other pelvic masses. Speculum exam to confirm diagnosis of enterocele (Malpas' test) and for detection of supra-vaginal elongation of the cervix by uterine sound in vagino-uterine prolapse.

Investigations of POP:

-Laboratory: C.B.C., renal and liver function tests, urine analysis.

-Radiology: plain X-ray chest and lumbo-sacral region, IVP, abdominal U.S.

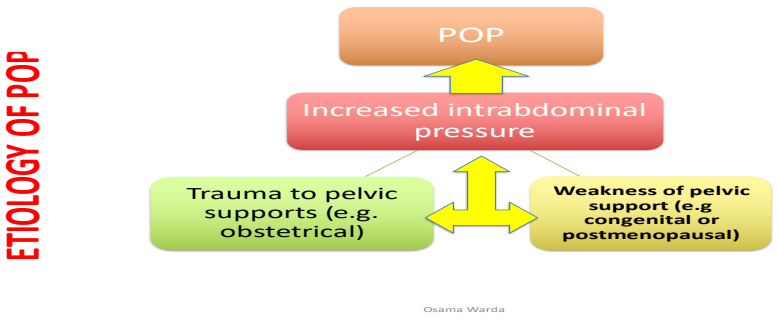
-Investigations for preoperative preparation of the patient.

-Urodynamics: if there is urinary tract symptoms and stress incontinence.

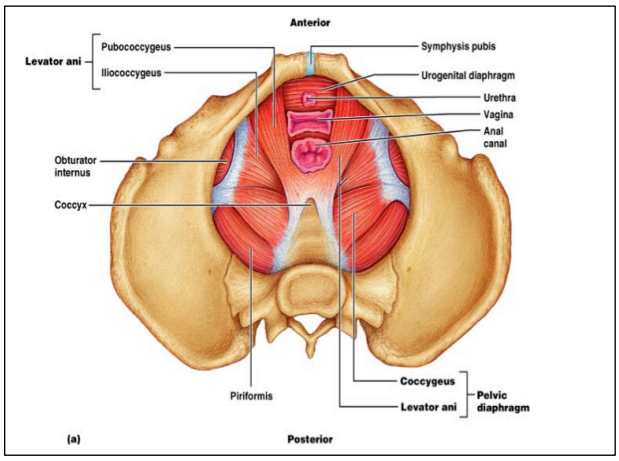
ETIOLOGY OF POP:

- Risk factors predisposing to prolapse either congenital weakness of the mesenchyme or due to obstetric trauma.
- *Obstetric trauma* causes:

1- *Pudendal nerves damaged,* with increased nerve conduction times.
2-Thinning or avulsion of the *puborectalis muscle* from its insertion on the pubic ramus on one or both sides.



RELEVANT ANATOMY OF POP:

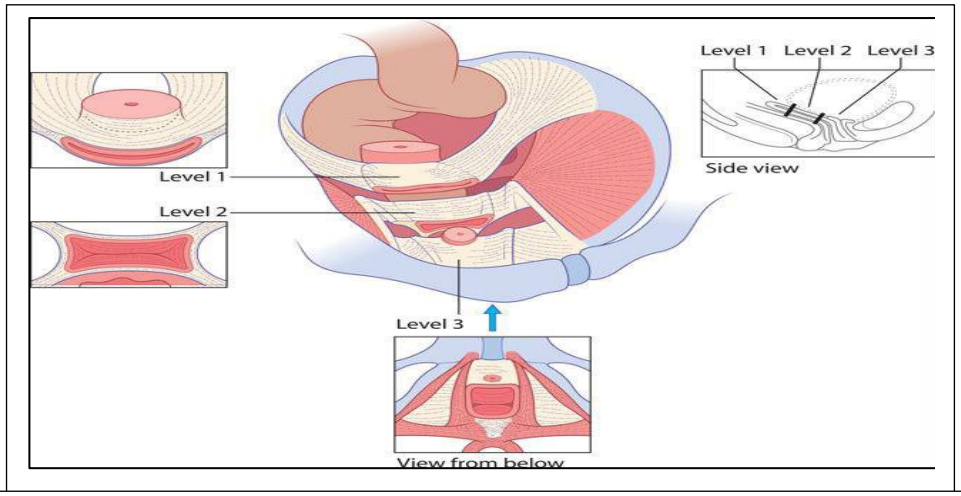


- *Uterovaginal prolapse* is caused by failure of the interaction between the levator ani muscles and the ligaments and fascia that support the pelvic organs.
- The levator ani muscles are ***puborectalis, pubococcygeus and iliococcygeus.*** They are attached on each side of the pelvic side wall from the pubic ramus anteriorly (pubococcygeus), over the obturator internus fascia to the ischial spine to form a bowl-shaped muscle filling the pelvic outlet and supporting the pelvic organs
- There is a gap between the fibers of the puborectalis on each side to allow passage of ***the urethra, vagina and rectum, called the urogenital hiatus.***
- The levator muscles support the pelvic organs and prevent excessive loading of the ligaments and fascia.
- **Fascial supports of the pelvic organs** ***(De Lancey's three levels of vaginal support)***.

-Level 1 support: is provided by the uterosacral ligaments, suspending the uterus and attached vaginal vault.

Level 2 (midvagina) support is provided by the **fascia** lying between the vagina and the bladder or rectum that fuses laterally and runs to attach on the pelvic side wall.

Level 3 support is provided by the perineal body, which has the posterior vaginal fascia fused to its upper surface.



-**Apical suspension (LEVEL 1)** →Upper para-colpium suspends apex to pelvic walls and sacrum→Damage results in prolapse of vaginal apex (vault prolapse)

-**Mid-vaginal lateral attachment (LEVEL 2)**→Vaginal attachment to arcus tendinous fascia and levator ani muscle, Pubo-cervical and recto-vaginal fasciae support bladder and anterior rectum →Avulsion results in cystocele or rectocele

-**Distal perineal fusion (LEVEL 3)** →Fusion of vaginal fascia to perineal membrane, perineal body and levators →Damage results in deficient perineal body or urethrocele

TREATMENT OF POP:

A- **Conservative treatment: includes**

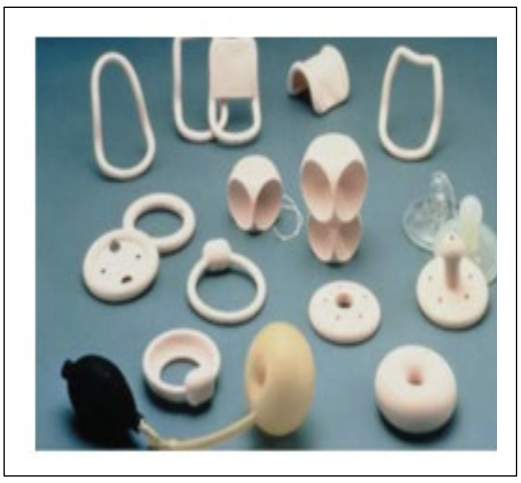
1- pelvic floor muscle exercises and 2- the use of supportive vaginal pessaries.

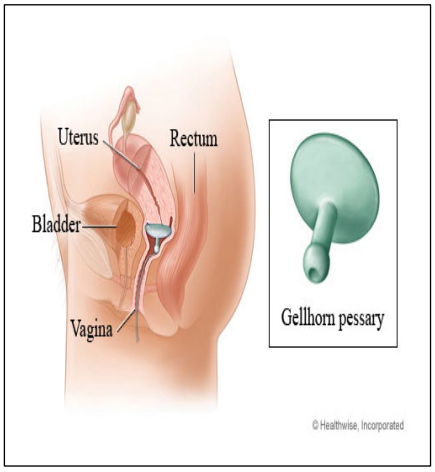
- For women with urinary or bowel symptoms as well, conservative treatment for these symptoms can be commenced at the same time as for the prolapse.

Pelvic floor exercises : - A course of supervised *pelvic floor exercises* will reduce the symptoms of prolapse and for women who are keen to avoid surgical treatment, this can be an effective first step, although there is *less evidence that pelvic floor exercise will reduce the anatomical extent of the prolapse and it is unlikely to be helpful for women whose prolapse is beyond the vaginal introitus.*

Pessary treatment:

- A vaginal support pessary is inserted to reduce the prolapse, which leads to resolution of many of the symptoms.
- Pessary use can be very effective at relieving symptoms and has the advantage of avoiding surgery and the associated risks, which can be extremely useful in the medically unfit and elderly.
- A range of shapes of pessary is available (see picture)
- Ring pessaries are usually tried first, but an intact perineal body is necessary for these to be retained.
- Shelf pessaries, **Gellhorn** pessaries and others are useful for women with deficient perineal bodies.
- It is usual practice to replace a pessary every 6 months and to examine the patient for signs of vaginal ulceration, although this frequency is traditional and not based on any evidence.
- ***Complications*** are uncommon and usually minor *(bleeding, discharge)*, although rarely the pessary can become *incarcerated*, requiring general anesthesia to remove, and rare cases of *rectovaginal or vesicovaginal fistula formation* have been reported.
- *Sexual intercourse* remains theoretically possible with a well-placed **ring pessary**, but not with the others, *so would not generally be suitable for women who are sexually active.*
- Motivated patients can be taught to insert and remove their own pessaries if they do wish to remain sexually active.





SURGICAL Treatment of POP:

-Surgical treatment for prolapse is common, and can be offered if conservative treatments have failed or if the patient chooses surgery from the outset.
-There are a wide range of specific procedures. The procedure chosen depends on
(a). which compartment is affected,
(b). whether the woman wishes to retain her uterus and
(c). whether the vaginal or abdominal route of surgery is chosen.
→The essential principles of prolapse surgery apply to all procedures.

Procedure	Key point	Short description	Complications
1-Anterior vaginal repair (anterior colporrhaphy)	-For anterior vaginal prolapse. -NOT for stress incontinence	Suture to reinforce fascia between vagina & bladder	-Bladder injury -High recurrence
2-Posterior vaginal repair (Posterior colporrhaphy)	-For posterior vaginal prolapse -Can improve obstructed defecation -Risk of recurrence is low	Suture to reinforce fascia between vagina & rectum	-Risk of rectal injury -Postoperative dyspareunia
3-Vaginal repair with polypropylene mesh	-Usually reserved for **recurrent** prolapse -Surgical repair reinforced with mesh -Very low recurrence rates -Excellent anatomical results	Mesh can be inlay (not fixed), or fixed to the pelvic ligaments to mimic the native utrosacral ligaments and fascial attachments	-mesh erosion through the vagina (5%) -Mesh erosion through bladder or rectum (<5%) -Dyspareunia -Chronic pelvic pain -Excision of mesh is difficult

4-Fothergill (Manchester) operation: it is a uterine preserving procedure, consists of D &C + anterior colporrhaphy+ shortening of the elongated cervix+ shortening of the cardinal ligaments+ posterior colpoperineorrhaphy. It is indicated when there is supra-vaginal elongation of the cervix, and the patient wants to keep her uterus.
5-Vaginal hysterectomy + Repair of the pelvic floor: indicated in cases of POP when the uterus is to be removed.
6- Partial colpocliesis (Le Forte operation): indicated in frail, non-sexually active women.

PRINCIPLES OF POP SURGERY:

- Remove/reduce the vaginal bulge.
- Restore the ligament/tissue supports to the apex, anterior and posterior vagina.
- Replace associated organs in their correct positions.
- Retain sufficient vaginal length and width to allow intercourse.
- Restore the perineal body.
- Correct or prevent urinary incontinence.
- Correct or prevent fecal incontinence.
- Correct obstructed defecation

Valuable links: https://youtu.be/bGQVZNBaAYI?si=kj8iw4JUc0H_r5tT
https://youtu.be/MpEZNWGKVPo?si=6XiRrX4T8AFHs1zG
https://youtu.be/IDkYGpCr25E?si=1ZnYQ49O3GwP5XVI

RETROVERSION FLEXION (RVF)

Definition:

- ***Retroversion:*** longitudinal axis of cervix is tilted upwards & backwards on the virginal axis.
- ***Retroflexion:*** longitudinal axis of uterine body is bent backwards on the axis of cervix.

A) Congenital: it gives no symptoms & needs no treatment. About 20% of females.

B) Acquired:

1) **Fixed RVF:** due to pelvic adhesions (as in PID or pelvic endometriosis).
2) **Mobile RVF:** due to bad management of puerperium.
3) **Pelvic tumors:** pushing or pulling the uterus backwards.

Diagnosis:

A) Symptoms:

1) **Asymptomatic: mobile RVF is nearly always symptomless.**
2) **Symptoms of pelvic congestion:** dull aching pain, low backache, congestive dysmenorrhea, dyspareunia, menorrhagia & leukorrhea.
3) **Chronic pelvic pain or dyspareunia.**
4) **Infertility or Abortion:** due to incarcerated RVF gravid uterus.

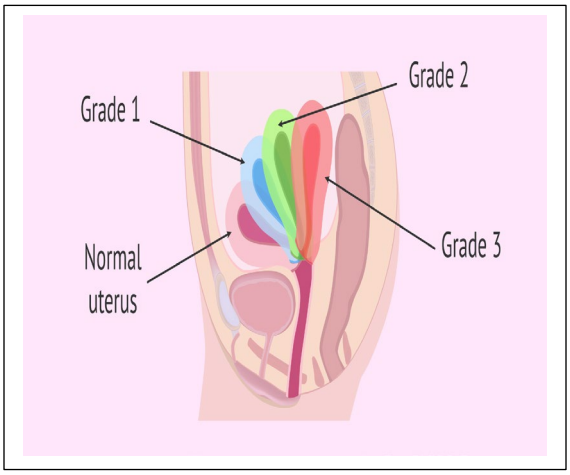
B) Signs: by vaginal examination, you can determine the degree or detect the pathology.

Degrees:

A) First degree: the fundus is directed towards sacral promontory.

B) Second degree: the fundus is directed towards sacral concavity

C) Third degree: the fundus is directed towards tip of sacrum & is at lower level than external os.



Prevention:

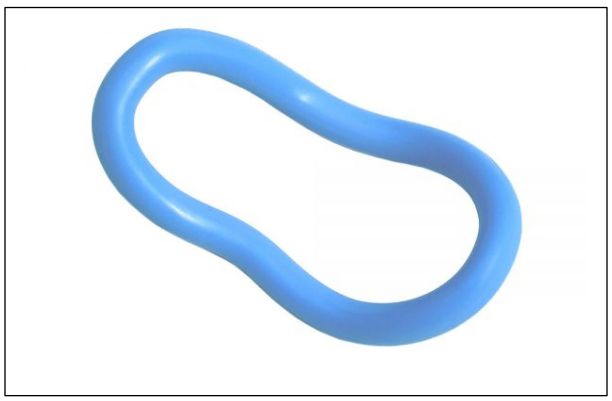
By proper management of puerperium.

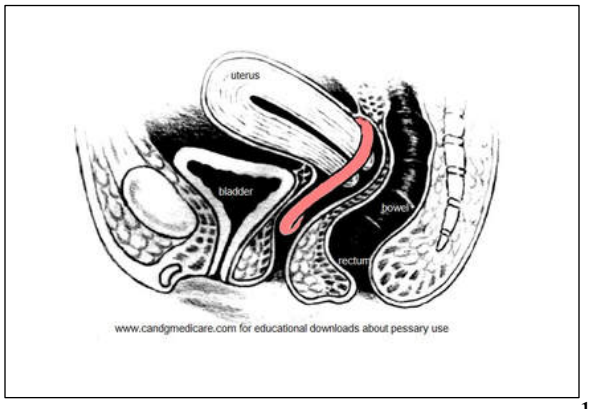
Treatment:

A) No treatment: in asymptomatic RVF.

B) Pessary: in mobile RVF by Hodge-Smith pessary

C) Surgical treatment: for the pathology present and correction of the uterine position





UTERINE INVERSION

Definition: Uterine inversion occurs when the uterine fundus collapses into the endometrial cavity, turning the uterus partially or completely inside out.

Incidence: Incidence ranges from 1 in 3500 to 20,000 deliveries.

Classification: Uterine inversions are classified by the extent of inversion and time of occurrence.

●**Extent of inversion**

•1st degree (incomplete inversion) – Fundus within the endometrial cavity
•2nd degree (complete inversion) – Fundus protrudes through the cervical os
•3rd degree (prolapsed inversion) – Fundus protrudes to or beyond the introitus
•4th degree (total inversion) – Both the uterus and vagina are inverted

●**Time of occurrence**:

•Acute – Within 24 hours of delivery
•Subacute – More than 24 hours but less than four weeks postpartum
•Chronic – ≥1 month postpartum

Risk Factors

- abnormal placentation such as retained and/or abnormally adherent placenta
- short umbilical cord
-prolonged labor,
-pedunculated fundal myoma.

Clinical Picture:

Patient presentation — Puerperal uterine inversion can follow vaginal or cesarean birth; the latter includes inversion through the hysterotomy incision. The clinical presentation depends on the **extent** and **time** of occurrence of the inversion. Signs and symptoms include one or more of the following:

●Mild to severe vaginal bleeding
●Mild to severe lower abdominal pain
●A smooth, round mass protruding from the cervix or vagina
●Urinary retention

Differential Diagnosis: - prolapsed fibroid & procidentia: clinical examination and ultrasound can differentiate the conditions.

Management:

Goals

-Replace the uterine fundus to its correct position
-Manage postpartum hemorrhage and shock, if present
-Prevent recurrent inversion

Acute cases: Interventions for the management of acute uterine inversion should begin promptly and simultaneously. A delay in diagnosis or prompt initiation of treatment increases the risk of maternal morbidity and mortality. We suggest:

1- Discontinue uterotonic drugs since uterine relaxation is needed to replace the uterine fundus.
2- Call for immediate assistance, including anesthesiology staff, operative room ready for laparotomy.

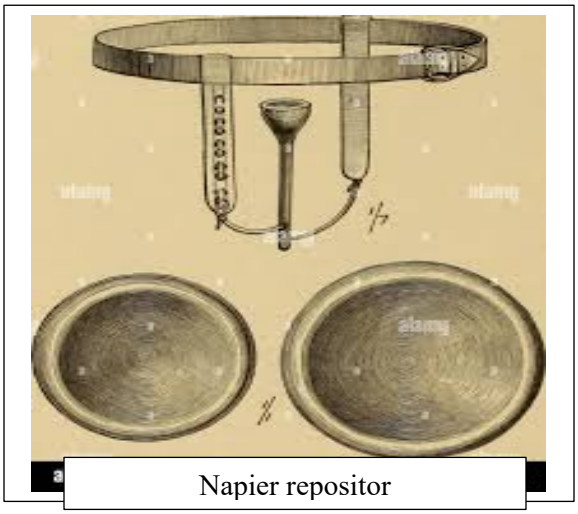
3- Establish adequate intravenous access and aggressive fluid/blood product resuscitation.
4- Do not remove the placenta.
5- Immediately attempt to manually replace the inverted uterus to its normal position.
6- **Hemodynamically unstable patients** – After an initial unsuccessful manual attempt at replacement, proceeding directly to laparotomy.
7- **Hemodynamically stable patients** – After an initial unsuccessful manual attempt at replacement in a hemodynamically stable patient, administer uterine relaxants then reattempt manual replacement.

8- **Surgical procedures at laparotomy**: If the above measures to replace the uterus fail, then the patient should be taken promptly to the operating room to attempt surgical correction of the inversion:

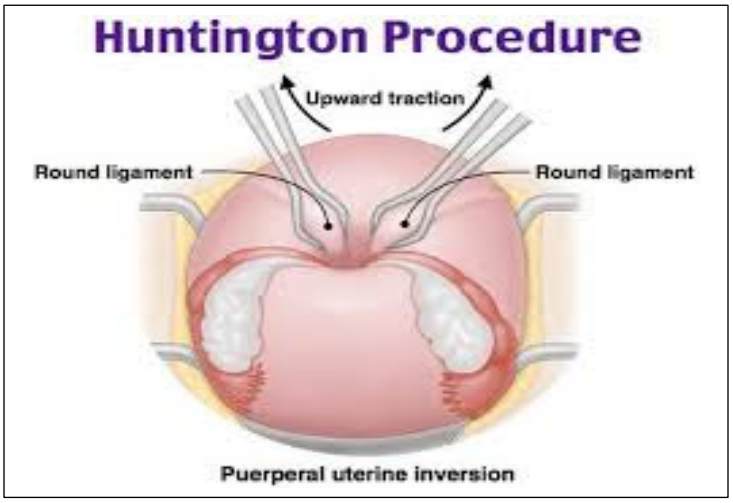
a- **Huntington procedure** **b- Haultain procedure**

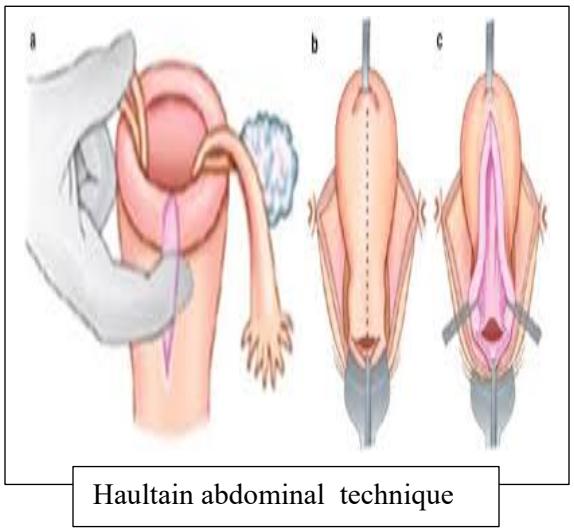
Chronic cases:

a) **Non-surgical: Napier repositor for** frail women contraindicated for surgery
b) **Surgical :** - Abdominal procedures (**Huntington** & **Haultain procedures)**
- Vaginal procedures: (**Spinelli's** and **Kustner's** techniques). : Spinelli and Kustner operations involve replacing the uterine fundus through the anterior and posterior transections respective

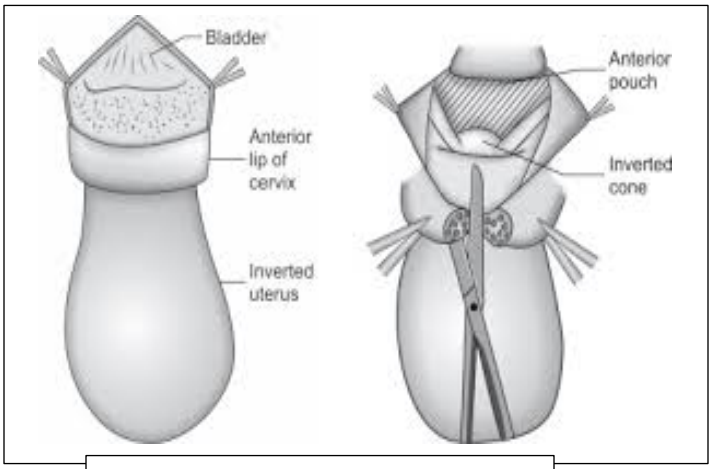


Napier repositor





Haultain abdominal technique



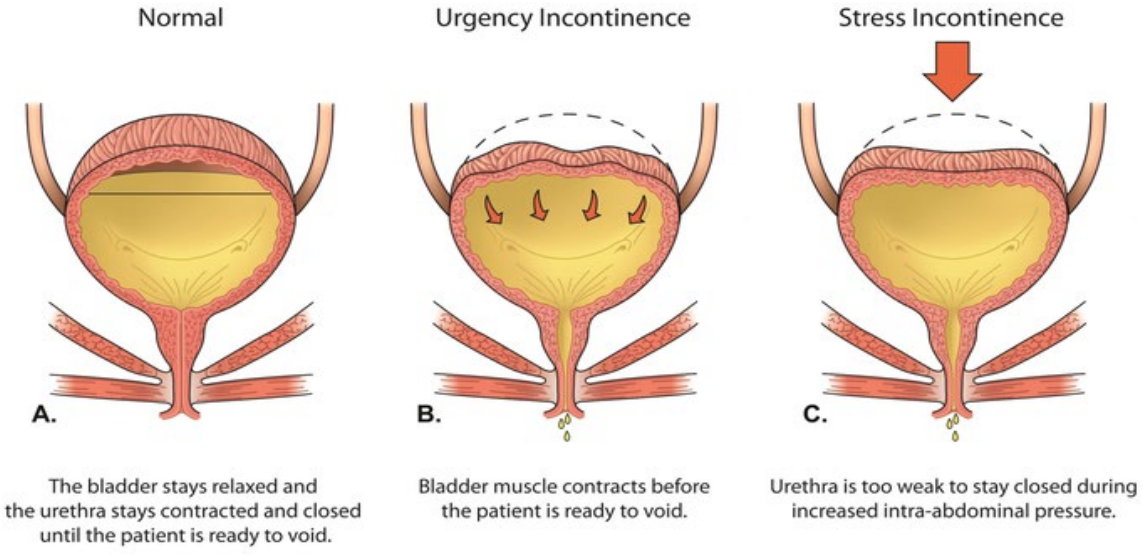
Spinelli vaginal technique

URINARY INCONTINENCE

Definition: it is a condition in which involuntary loss of urine is a social or hygienic problem and is objectively demonstrable i.e. "The complaint of any involuntary leakage of urine".

Normal continence function :

- Functional urethra is intra-abdominal
- Increased abdominal pressure transmitted equally to bladder and urethra.
- With increased stress, the urethro-vesical junction responds to stress by closing tight
- Bladder is a voluntary smooth muscle
- Inherent ability to maintain low pressure with filling-increase in volume-compliance.



Impact of urinary incontinence on health:

Urinary incontinence is not associated with increased mortality. However, incontinence can impact on *many other aspects of a patient's health:*

1- Quality of life – Urinary incontinence is associated with depression and anxiety, work impairment, and social isolation.

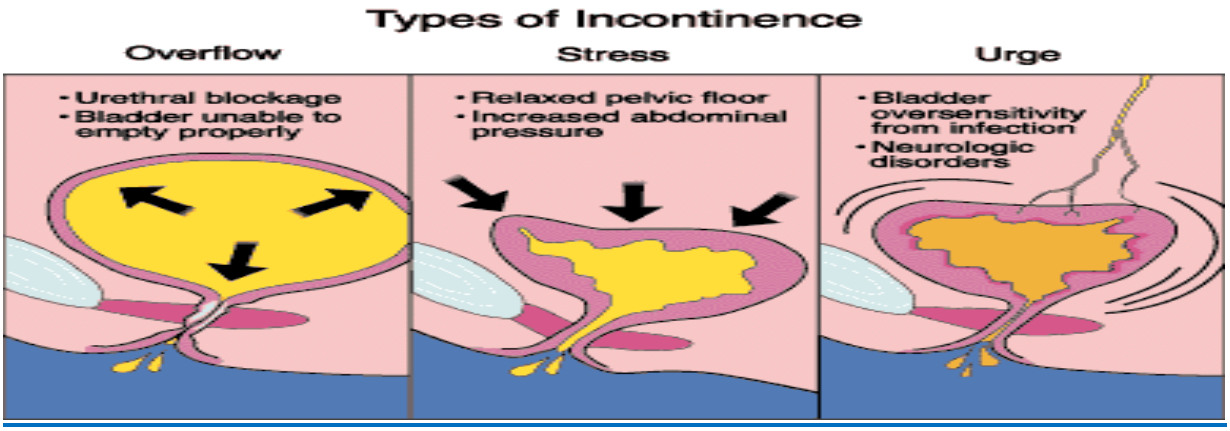
2- Sexual dysfunction – Incontinence during sexual activity (coital incontinence), which may affect up to one-third of all incontinent individuals, and fear of incontinence during sexual activity both contribute to incontinence-related sexual dysfunction.

3-Morbidity – include perineal infections (eg, candida or cellulitis) from moisture and irritation as well as falls and fractures that in turn increase overall morbidity, and health care costs .

4-Increased caregiver burden – 6%-10% of nursing home admissions in the United States are attributable to urinary incontinence.

Causes (types) of urinary incontinence:

1. Stress incontinence 50% (involuntary escape of urine due to effort)
2. Urge incontinence 25% (bladder contracts before reaching toilet)
3. Mixed incontinence (stress + urge) 25%
4. Overflow incontinence (neurogenic or automatic bladder)
5. Fistulae (Vescio-vaginal, ureterovaginal)
6. Urethral diverticulum
7. Functional (the patient cannot go to toilet (e.g. fracture lower limbs)
8. Reversible causes (certain drugs)



Stress Urinary Incontinence (SUI)

Definition : Stress urinary incontinence(SUI) is defined by the international continence society (ICS) as: "*the complaint of involuntary leakage of urine on effort or exertion, or on sneezing or coughing*" .

Genuine stress urinary incontinence (GSUI): Urinary loss which occurs with sudden elevation of the intra-abdominal pressure without detrusor contraction is called stress urinary incontinence

Symptoms:

1. Involuntary leakage of urine on effort or exertion, or on sneezing or coughing
2. Usually small amounts
3. Pressure in the bladder exceeds the urethral pressure

4. No bladder contraction

Etiology (high-risk patients):

1- Pregnancy/Childbirth (multiparity)
2- Age (mainly over 50 years, estrogen lack)
3-Obesity (BMI >40, 66%)
4-Chronic cough
5- Pelvic Organ Prolapse
6- Chronic Constipation
7-Smoking
8-Genetics

Types of SUI:

- **Type 1** *:* Incontinence due to loss of posterior urethro-vesical angle alone
- **Type 2** *:* Incontinence due to loss of posterior urethro-vesical angle *plus* urethral hypermobility
- **Type 3** *:* Incontinence due to ISD (intrinsic sphincteric deficiency).

DIAGNOSIS OF SUI:

The primary goals of evaluation of women presenting with symptoms of SUI:

1. Provide a clinical diagnosis of SUI versus overactive bladder (OAB) symptoms
2. Determine factors that may contribute to symptoms or that may require further evaluation.
3. Assess whether coexisting pelvic floor disorders, such as pelvic organ prolapse or anal incontinence, are present.
4. Establish baseline SUI severity to aid in assessing treatment effects.
5. Determine the impact of the patient's symptoms on her quality of life.
6. Determine which treatment options are acceptable to each patient.
7. Determine what her own therapeutic goals are?
8. Provide her with appropriate education regarding these goals.

Diagnosis of SUI is made via :

A- **Basic evaluation**:

1-**Careful history** **2. Physical examination** **3. Voiding dairy** **4. Simple tests**

B- **Advanced evaluation:**

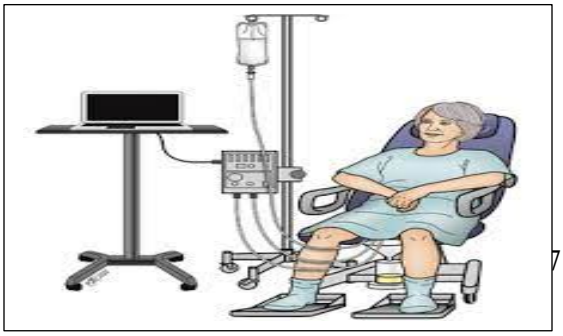
- *Further evaluation is needed after basic testing if we are still left with an uncertain diagnosis:*

1-Major discrepancies between history, the voiding diary and symptom scales
2-When the patient is considered for surgery.
3-If the patient has hematuria in the absence of an infection
4-An elevated postvoid residual volume, a neurologic condition that may complicate treatment (such as multiple sclerosis),
5-marked pelvic organ prolapse, or numerous prior surgical attempts at correction

-Advanced evaluation is done via:

1-urotho-cystoscopy
2- urodynamic studies (uroflowmetry, cytometry , urethral pressure profile)

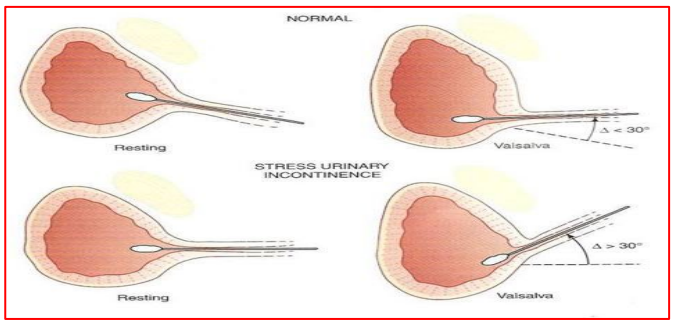
Urodynamic study:
https://youtu.be/I7pO2uZsWTE?si=wX6i2NsNRyAmN7JS



BASIC EVALUATION OF SUI:

Careful history:

1. Frequency and amount of leakage
2. Precipitating factors
3. Impact of the leakage on daily life and pad use
4. Pelvic floor symptoms



Link : https://youtu.be/61EzxtJ98P4

Physical exam :

1. Pelvic examination to rule out pelvic or abdominal masses, pelvic organ prolapse, and vaginal atrophy.
2. A positive **cough stress test**, in which leakage is visualized at the moment of the cough, is helpful to confirm the diagnosis
3. On vaginal or rectal examination, check the **pelvic floor muscles quality (symmetry and bulk)** and whether or not, and to what degree, a **woman can volitionally contract her muscles.**

4-**Cotton swab test** (test of urethral mobility); **-<<<<--(see figure & open link)**

5-To rule out urinary retention and overflow incontinence, we assess the postvoid residual volume by either direct catheterization or by ultrasonography (*Most experts agree that a postvoid residual volume less than 50 mL is normal and more than 200 mL is abnormal).*

6- Finally, a urinalysis is done to UTIs as a transient cause of stress urinary incontinence.

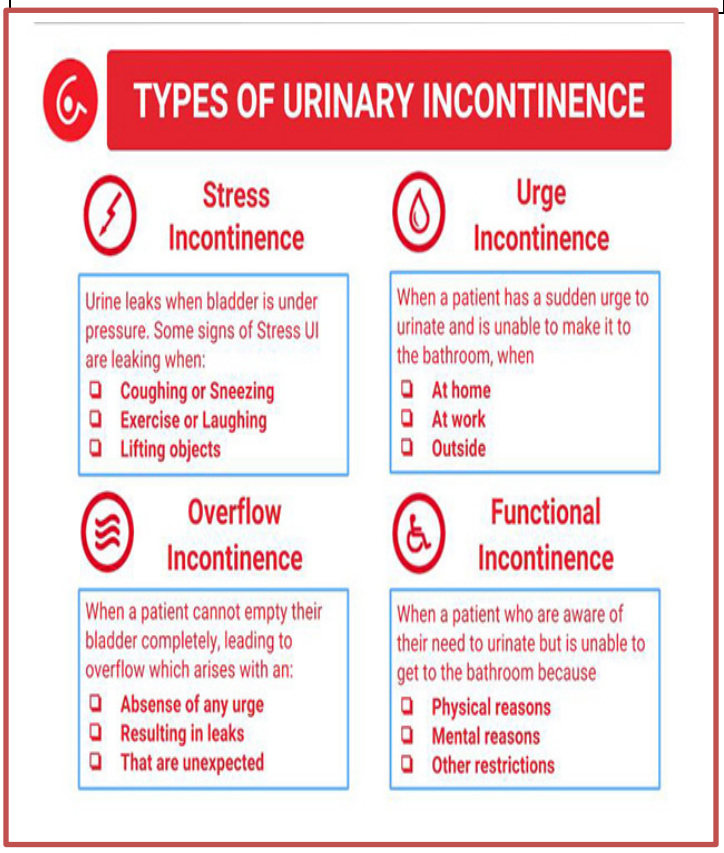


TABLE 3

Grading of stress urinary incontinence according to the clinical stress or coughing test conducted on a full bladder (about 300 mL) (20)

Severity	Definition
Stress urinary incontinence grade 0	No urine loss found
Stress urinary incontinence grade I	Urine loss in droplets while standing
Stress urinary incontinence grade II	Urine loss in a stream while standing
Stress urinary incontinence grade III	Urine loss in a stream while lying down

Management of SUI

- Women have both **non-surgical and surgical options** to treat SUI.
- Not every woman with SUI will need surgery. Some factors should be considered before deciding whether to undergo surgery include:

(a). the severity of SUI symptoms and their effect on daily activities.
(b) desire for future pregnancy as vaginal delivery can cause recurrence of SUI symptoms, which could require future surgery.

NON SURGICAL MANAGEMENT OF SUI	SURGICAL MANAGEMENT OF SUI
1. Lifestyle interventions 2. Pelvic floor muscle training 3. Medications 4. Devices	1. Use of injectable bulking agents, 2. Laparoscopic suspensions 3. Mid-urethral slings 4. Pubovaginal slings 5. Open retropubic suspensions

Lifestyle modifications:

1. Weight reduction
2. Postural changes (such as crossing legs) often prevent stress urinary incontinence.
3. Fluid intake and voiding habits (decreasing the fluid intake is helpful for patient with high fluid consumption & voiding prior to strenuous activity beneficial in mild SUI)

Pelvic floor muscle training

-Supervised pelvic floor muscle training (Kegel exercises) is an effective treatment for stress urinary incontinence.
-Should be offered as first-line conservative management to women.
-Several factors are important in maximizing the chance that pelvic muscle training will alleviate SUI:
1-The woman must do the exercises correctly, regularly, and for an adequate duration.
2-Many physical therapists recommend training sessions 3–4 times per week, with 3 repetitions of 8–10 sustained contractions each time.

Medications used for SUI:

1- Estrogen: -Estrogen has tropical effects on urethral epithelium subepithelial vascular plexus and connective tissues . Studies showed improvement of symptoms, but not urodynamic measurements.
2-Alpha-adrenoreceptor agonist: -*Ephedrine*, *Norephedrine* and *Midorine* have shown only modest effect in small trials.
3-Serotonin and norepinephrine reuptake inhibitors: -*Duloxetine chloride* is effective for the treatment of SUI.

Devices used for SUI:

Ring pessary

-The pessary compresses the urethra against the symphysis pubis and elevates the bladder neck.
-For some women this may reduce stress leakage

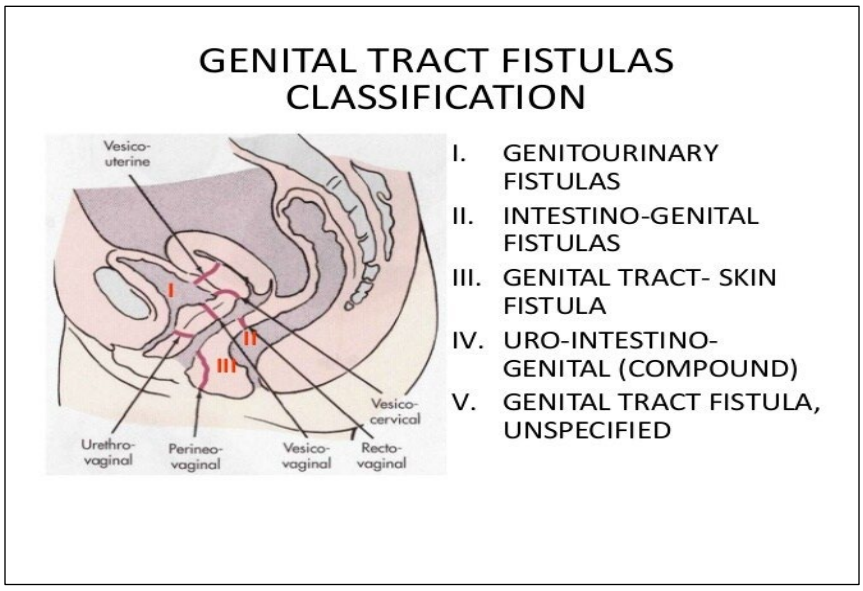
Important links : pubo-vaginal sling operation:

https://youtu.be/yqqsVS7CUXY?si=_5htUQs_8TV2vCT1

Genital Fistulas

Definition:

- *A fistula (plural: fistulas or fistulae)* in anatomy is an abnormal connection between two hollow spaces (technically, two epithelized surfaces), such as blood vessels, intestines, or other hollow organs as vagina, rectum, ureter, ……etc. Types of fistulas can be described by their location.
- *Genital fistulas* are abnormal communications between the genital tract [vagina, cervix, uterus, fallopian tube] and other hollow organs or cavities [urinary bladder, ureter, rectum, intestine, skin].
- Of all fistulas two fistulas are of concern to the gynecologist: *vesicovaginal (VVF),* and *recto-vaginal (RVF).*



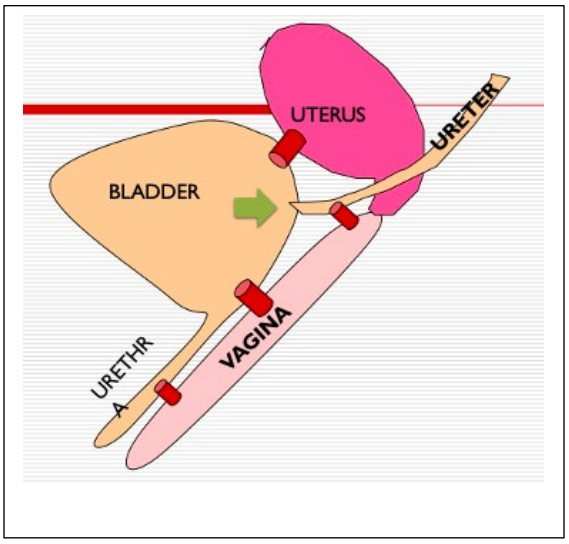
Urogenital fistulas

Definition :

Is abnormal communications urine passes from the urinary system to the genital system i.e from bladder to vagina, from ureter to vagina , … and so on. The name of the fistula follows the direction of urine e.g. vesico-vagina, uretro-vaginal, ……and so on.

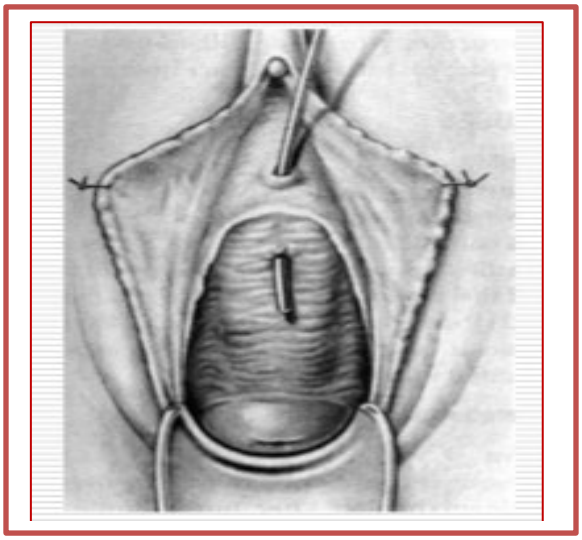
Types :

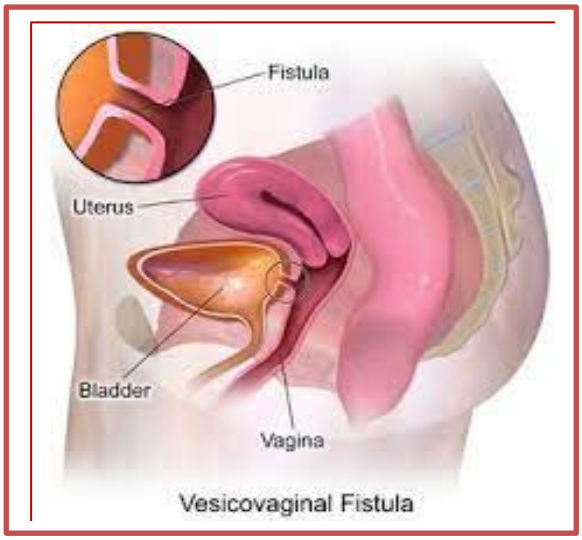
1. Vesico-vaginal (bladder→ vagina)
2. Uretro-vaginal (ureter-→ vagina)
3. Urethro-vaginal (urethra→ vagina)
4. Vesico-uterine (bladder→uterus)
5. Vesico-cervical (bladder→ cervix)
6. Uretro-cervical (ureter→ cervix)
7. Uretro-uterine (ureter→ uterus)



VESICO-VAGINAL FISTULA (VVF)

- VVF is the most common type of genital fistulas.
- It is abnormal tract communication between the urinary bladder (commonly the trigone or bladder base) and the vagina.





Etiology (causes) :

1. Congenital VVF: rare and usually associated with other urogenital malformations
2. Obstetric VVF: most common type (90%) in developing countries due to bad conduct of labor; prolonged fetal head impaction (ischemia), or traumatic (forceps, ventouse , others).
3. Surgical trauma during pelvic surgeries (gynecological, urological, or oncologic surgeries); this is the most common type of VVF in developed countries.
4. Radiation VVF; following pelvic irradiation, e.g cancer cervix, rectal cancer
5. Malignant VVF; due to bladder invasion of nearby malignancy, e.g. cancer cervix, rectal cancer.
6. Others, e.g. neglected vaginal pessary, neglected foreign body, trauma after falling astride on sharp objects.

Types of VVF :

A. According to SIMPLICITY:
1. Simple - Healthy tissues with good access
2. Complicated – Tissue loss, scarring, difficult access, associated with RVF

B. Depanding upon SITE of the Fistula:
1.*Juxtracervical* **:(** close to cx) –communication between superrational region of bladder and vagina
2.Midvaginal **:** communication between base(Trigone) of bladder and vagina
*3.Juxtraurethral***:** communication between neck of bladder and vagina

Symptoms of VVF :

A.URINARY INCONTINENCE:

1. Complete (or total) incontinence in large fistula
2. Partial (or incomplete) in small fistula ; should be differentiated from ureterovaginal fistula.

B. **Symptoms of vulvitis**: pruritus, burning pain, encrustations of vulvar skin; due to continuous dripping of urine.

C. Cystitis; due to ascending infection from the vagina.

Diagfnosis of VVF :

A. HISTORY of urinary incontinence following labor, surgery, or other trauma:

1. several days after labor or surgery if ischemic VVF
2. immediately after labor or surgery; if traumatic

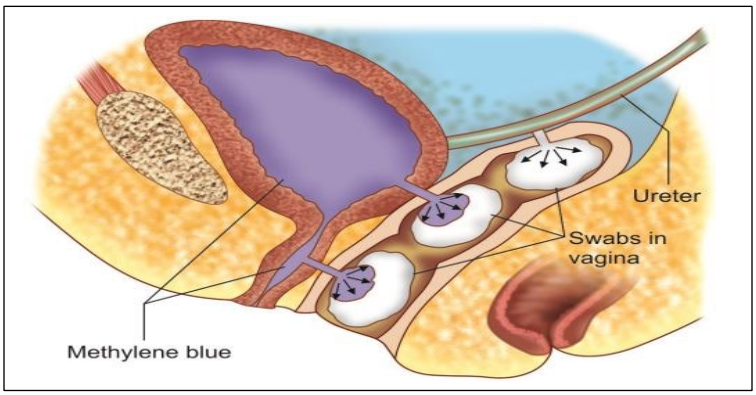
B- EXAMINATION; inspection & palpation of the anterior vaginal wall (Sims' speculum in Sims' position:

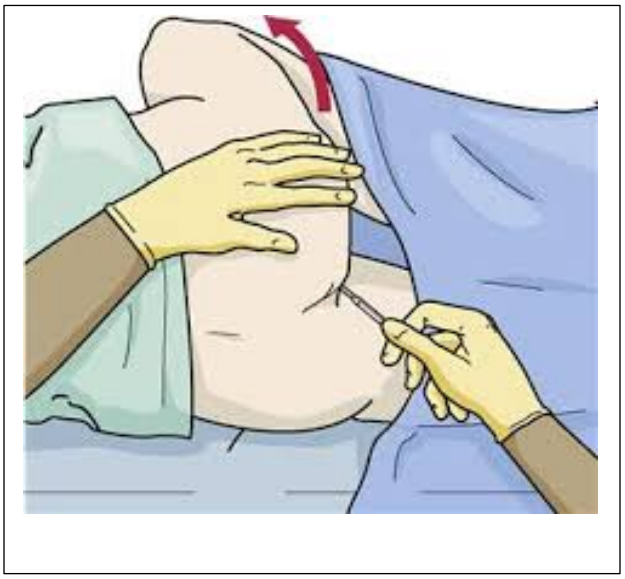
1. Large VVF can be seen & felt
2. Small VVF cannot be seen or palpated accurately but surrounding fibrosis can be seen and palpated.

C. INVESTIGATIONS :

1- Methylene Blue test *(3 cotton test);*

This test aims mainly to differentiate small VVF from ureterovaginal fistula.





2- Cystography.

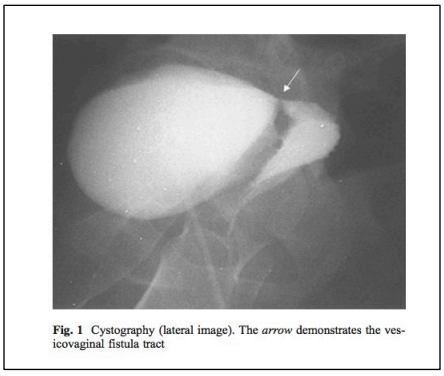


Fig. 1 Cystography (lateral image). The *arrow* demonstrates the vesicovaginal fistula tract

3- CT with dye

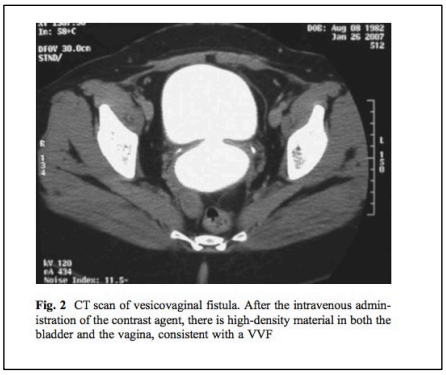
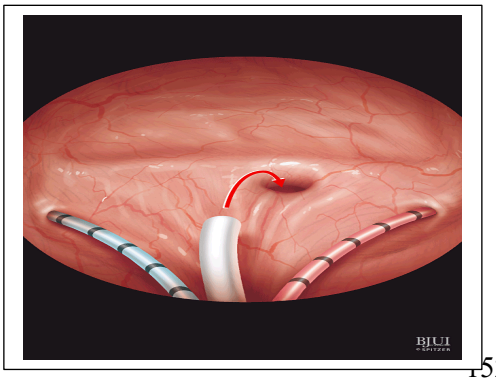


Fig. 2 CT scan of vesicovaginal fistula. After the intravenous administration of the contrast agent, there is high-density material in both the bladder and the vagina, consistent with a VVF

4- Cystoscopy



Management of VVF

[A]. Prophylaxis-

Obstetrical VVF:	Surgical VVF
-ANTENATAL: diagnosis of abnormalities that may result in VVF formation, e.g. contracted pelvis, malpresentation. - INTRAPARTUM: a). diagnosis & proper management of malpresentation, prolonged labor or contracted pelvis. b). Risky operations should be avoided such as high & mid forceps, forceps (or ventouse) with incompletely dilated cervix, and risky old fashion destructive operations.	**Preoperative measures:** - identification of high-risk cases for bladder injury , eg adhesions, endometriosis, malignancy. -Preoperative catheterization of the urinary bladder (and / or ureters) **Intraoperative:** -Meticulous dissection techniques, getting the advantage of the retropubic approaches whenever adhesions disturb the surgical anatomy; *"whenever difficult get retro".*

[B]. Conservative management

If VVF is diagnosed within the *first few days of surgery*, a *transurethral or suprapubic catheter* should be placed and maintained *for up to 30 days*. Small fistulas (< 1 cm) may resolve or decrease during this period if caution is used to ensure proper continuous drainage of the catheter.

[C]. Surgical management:

Timing of surgical repair

- The ideal time for repair of obstetric VVF is after 3 months following delivery.
- Surgical Fistula:

-If recognized <24 hrs: immediate repair
-If recognized >24 hrs : repair after 3 months

- Radiation Fistula: repair after 12 months

Surgical approaches/ techniques for VVF repair:

- The best chance for a surgeon to achieve successful repair is by using the type of surgery with which he or she is most familiar.
- *Techniques of repair includ*

VAGINAL OPERATIONS	ABDOMINAL OPERATIONS	OTHERS
1.Latzko operation 2.Dedoublement operation 3.Saucerization operation	1.extraperitoneal approach 2.Transperitoneal approach 3.combined approach	1. electrocautery 2. fibrin glue 3. endoscopic closure with fibrin glue 4. laparoscopic 5. using interposition flaps.

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PART FOUR

- **GENITAL INFECTIONS**
- **SEXUALLY TRANSMITTED INFECTIONS**

GENITAL INFECTIONS

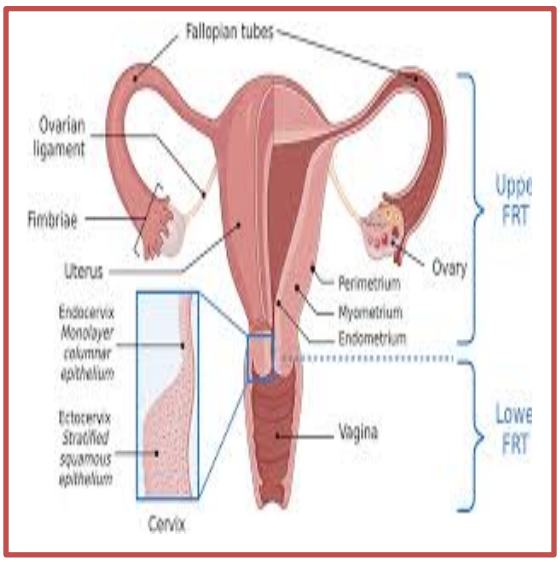
There are normal barriers protecting against genital infection:

A) Vulva:

1) Closed introitus.
2) Thick & highly vascular vulval skin.
3) Presence of hymen in virgins.
4) Antimicrobial activities of Bartholin's glands secretions.

B) Vagina:

1) Vaginal epithelium is stratified squamous (resistant to organismal invasion).
2) Acidic vaginal discharge (acidity destroys most of pathogens).
3) Vaginal flora (the commonest is lactobacilli).
4) Absence of glands (glands are important reservoir of infection).



C) Cervix:

1) Narrow cervical canal.
2) Cervical secretions contain IgA.
3) Thick cervical mucus plug closing cervix.

D) Upper genital tract:

1) Monthly shedding of endometrium.
2) Continuous ciliary movement of tubes towards uterine cavity.

Classifications of female genital tract infection:

A) Anatomical classification:

1) **Lower genital tract infection:** infection below internal os (vulvitis, vaginitis & cervicitis).
2) **Upper genital tract infection:** Infection above internal os (PID).

B) Etiological classification:

1) **Sexually transmitted diseases (STDs).**
2) **Blood born infections:** mostly TB and rare now.
3) **Endogenous infections:** caused by overgrowth of microbial agents that are normal

inhabitants (as candida albicans or bacterial vaginosis).

4) **Non-specific infections:** caused by inappropriate infection prevention measures:

a. Puerperal or post-abortive.

b. After pelvic examination.

c. Traditional practice (female genital mutilation or vaginal tampons

Vulvovaginitis

Definition: inflammation of vulva & vagina.

Incidence: commonest outpatient gynecologic problem.

Causes:

A- VULVITIS:

Primary vulvitis	Secondary vulvitis
a) Infections: STDs. **b) Chemical irritation:** soaps, powders, perfumed toilet paper & lubricant sprays. **c) Physical irritation:** by irradiation.	**a) Vaginal conditions:** vaginal infections. **b) Urinary conditions:** incontinence or fistula. **c) Rectal conditions:** fistula or complete perineal tear. **d) Skin diseases:** Dermatosis or psoriasis.

B-Vaginitis:

Primary vaginitis	Secondary vaginitis
a) Infections: candidiasis, trichomoniasis, bacterial vaginosis & STDs. **b) Estrogen deficiency (atrophic vaginitis).** **c) Chemical irritation:** soaps, powders, toilet perfumed papers, douches & vaginal contraceptives. **d) Mechanical irritation:** Vaginal tampons or neglected pessaries.	**a) Cervical conditions:** Cervicitis. **b) Uterine conditions:** Chronic uterine bleeding or intermittent pyometra. **c) Urinary conditions:** Fistula. **d) Rectal conditions:** Rectovaginal fistula or complete perineal tear. **e) General diseases:** as allergy.

Vulvovaginitis of children

Etiology:

1. Lack of cleanliness.
2. Intestinal infection with threads or round worms.
3. Foreign body in vagina.
4. Gonorrhea.

Clinical picture:

- Pain, tenderness, offensive vaginal discharge & dysuria.
- Vulva looks swollen, red & covered with purulent vaginal discharge.

Treatment:

- Treatment of the cause.
- Local cleanliness.
- Small dose of estrogen (to help eradication of infection).

Atrophic (senile) vaginitis

Etiology: Lack of estrogen (as in postmenopausal women or in prepubertal age).

Clinical picture: Vagina becomes smooth, dry, thin with itching and severe dyspareunia.

Treatment: Local conjugated estrogen cream.

Bartholinitis (*inflammation of Bartholin glands)*

Etiology: Gonorrhea or non-specific infection.

Clinical picture: Gland becomes palpable as mass in the posterior 1/3 of labia majora, tender with abscess formation in some cases.

Treatment: antibiotics and drainage (if abscess forms) OR marsupialization with recurrent cases.

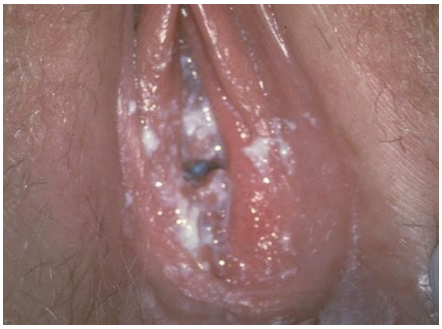
Candidiasis (Moniliasis)

Etiology: it is the second most common cause of vaginitis.

- Causative organism: candida albicans 90% of cases, c. tropicalis and c. glabrata 10% of cases.

- Flourishes in acidic media so common in diabetics, during pregnancy, with antibiotic use, with COCs, with antibiotics or steroids and immunosuppressive drugs.

Diagnosis:

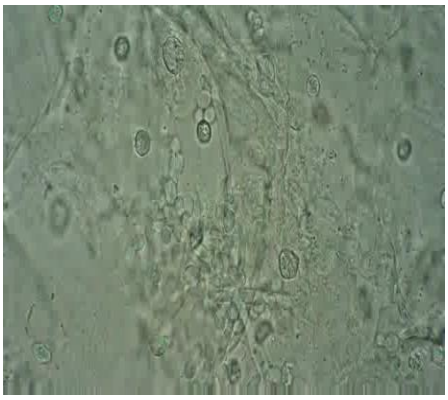


1- Symptoms:

- Discharge: is thick, scanty, white, curd-like, adherent.
- Burning sensation in the vagina.
- Itching and scratching sensation on the vulva.
- Dyspareunia and dysuria

Signs:

- Vulvitis: redness, oedema, itching marks.
- Vaginitis: red, tender vagina with adherent plaques.
- Characteristic discharge



Investigations:

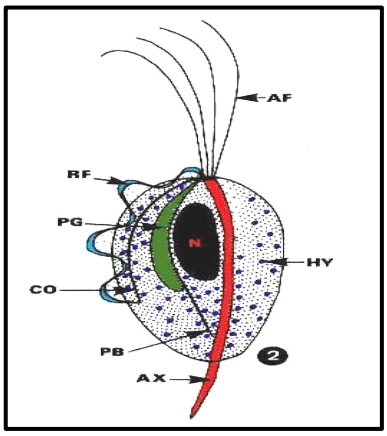
- Fresh drop examination with 10% KOH shows G+ve spores and long pseudo-hyphae.
- Culture on specific media

Treatment:

- Correction of general condition.
- Alkalinization of the vagina.
- Antifungal preparations: as by:

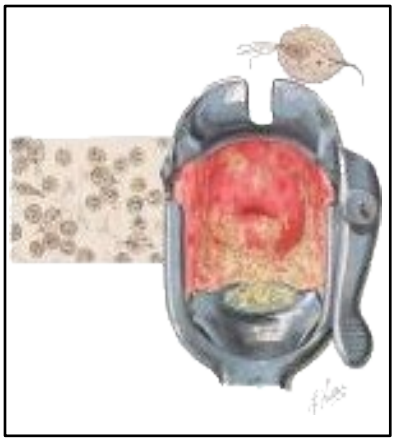
1. **Local vaginal** suppositories or intravaginal creams: e.g., clotrimazole, miconazole, and tioconazole preparations
2. **Oral antifungal** treatment: e.g., Fluconazole; and Ketoconazole

Trichomoniasis



Etiology: the third most common cause for vaginitis

- **Causative organism:** Trichomonas vaginalis, ovoid, motile, flagellated protozoon, 4 anterior flagellae and an axostyle,
- Flourishes in weak acid medium pH 5.5-6.5.
- It commonly affects vagina, urethra, Skene's tubules, bladder and cervix.
- Mode of infection: sexual intercourse and contaminated towels and instruments



Diagnosis:

Symptoms:

- Often manifests after menstruation; vaginal pH is raised.
- Profuse yellowish, frothy malodorous vaginal discharge.
- Pruritis vulvae and vaginal soreness
- Dyspareunia and dysuria

Signs:

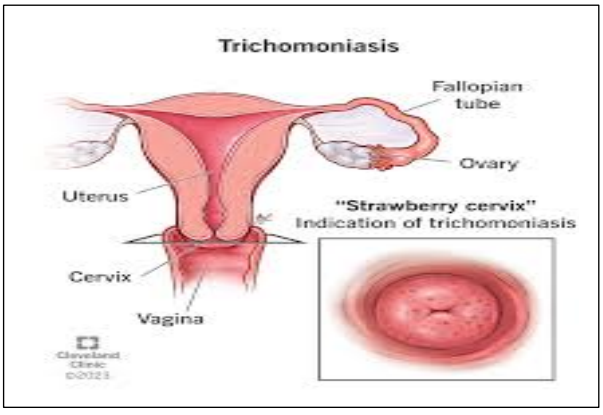
- Vulvitis (redness, hotness, oedema).
- Vagina: red, edematous, tender with punctate haemorrhage (strawberry vagina).
- Cervix: Strawberry like, sometimes eccentric erosion.
- The characteristic discharge (frothy, yellowish, malodourous ..... etc)

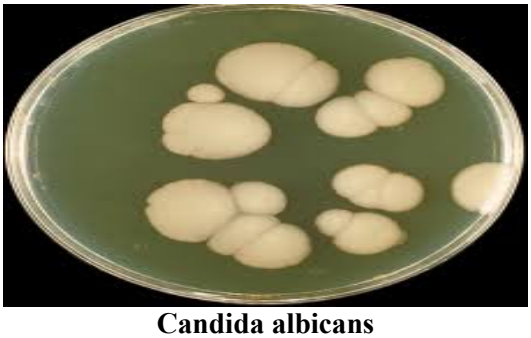
Investigations:

- Fresh smear: shows the organism and leucocytes
- Culture on specific media.

Treatment:

- Metronidazole tablets (Flagyl): 500 mg/12 h for 10 days OR 2 gm single dose
- Protozole and Tinedazole: 2 gm single dose
- The husband should be treated at the same time.





Candida albicans

Bacterial vaginosis

Etiology: it is the most common cause of vaginitis

- **Causative organism:** alternation of normal flora; decrease lactobacilli and increase Gardnerella and anaerobes.
- It is sexually transmitted.

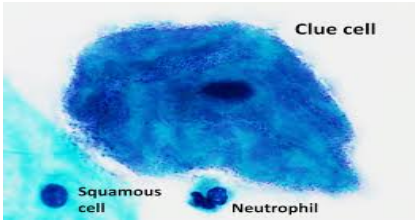
Symptoms: 50% may be asymptomatic BUT there may be thin excessive greyish frothy malodorous discharge with pruritus

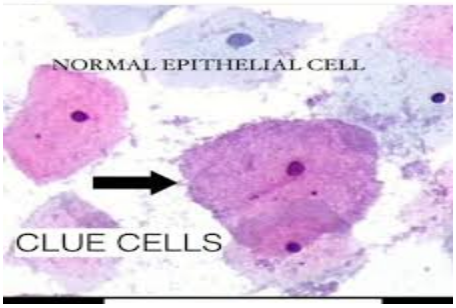
Signs: Characteristic discharge and vulvovaginitis.

Fresh drop: Clue cells appear which are aginal epithelium with obscured borders due to attachment of the organism

Culture: on specific media.

Whiff test: Discharge + 10% KOH → fishy odor





Clue cell-Bacterial Vaginosis

Treatment:

Sexual partner should be treated if infection is recurrent

1-Intravaginal preparations: Clindamycin cream

OR Metronidazole once daily for 5 days

A) Oral regimens:

- Metronidazole as a single 2 gm dose
- Clindamycin 300 mg twice daily for 7 days

c) During pregnancy.

- Clindamycin may be used throughout pregnancy
- Metronidazole may be used after the first trimester

Acute cervicitis

Etiology:

1. **Infective:** Gonorrhea & chlamydia.
2. **Non infective:** Postoperative (as D&C or cauterization).

Clinical picture:

Symptoms:

1. Mucopurulent vaginal discharge.
2. Deeply seated pelvic pain, low backache & dyspareunia.

Signs:

1. Cervix is swollen & red è tender cervical motion.
2. Discharge is seen oozing from cervix.

Treatment: Broad spectrum antibiotics, analgesics & antipyretics.

Chronic cervicitis

Etiology:

1. Non-specific: On top of acute cervicitis.

2. Specific: Chronic from the start (TB, syphilis,).

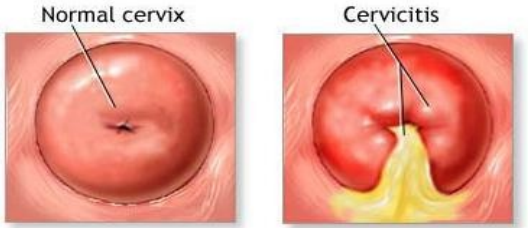
Diagnosis:

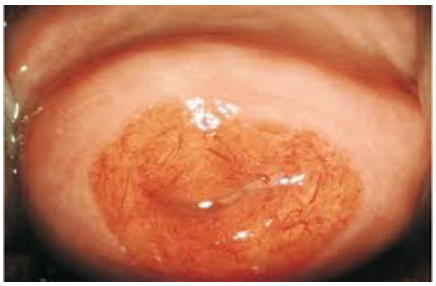
Symptoms:

- **Vaginal discharge:** the main complaint & it may lead to pruritus vulvae.
- **Pain:** low backache & sacralgia, dysmenorrhea, dyspareunia, dysuria & dyschezia.
- **Bleeding:** Menorrhagia (due to pelvic congestion) or contact bleeding.
- **Infertility.**

Signs*: there are many clinic-pathological types*

1) Mucopurulent endocervicitis.
2) Chronic endocervicitis.
3) Chronic hypertrophic cervicitis with barrel shaped cervix.
4) Cervical ectopy (erosion).
5) Cervical ectropion (bilateral cervical laceration è eversion of cervical lips).
6) Mucous polyp.
7) Nabothian follicle or cyst formation.
8) Chronic specific cervicitis.




Cervical ectopy (erosion)

Investigations:

1) Bacteriological examination of discharge.
2) **Colposcopy:** in resistant cases to exclude malignancy.

Treatment:

1. Medical treatment:

- Broad spectrum antibiotics (as tetracycline).
- Analgesics.
- Vaginal antiseptics.
- Measures to relieve pelvic congestion.

2, Cauterization: indicated mainly in cervical erosion.

Types of cautery: electrocautery, cryocautery, chemical cautery, laser cautery, end-coagulation system

3. Surgical treatment:

1) **Trachelorraphy:** in ectropion.
2) **Conization OR amputation:** in elongated hypertrophic cervicitis

Pelvic inflammatory disease (PID)

Definition: Upper genital tract infection.

Types: Often **acute** except in cases of TB or actinomycosis where it becomes **chronic**. **Causative Organisms:** PID is usually polymicrobial & the most common organisms are:

- **Neisseria gonorrhea:** 2/3 of cases (commonest).
- **Chlamydia trachomatis:** 20 % of cases (the commonest STD).
- **Aerobes:** Staphylococci, group B streptococci & E. coli.
- **Anaerobes:** Bacteroids & peptococci.
- **Mycoplasma hominis.**
- **Actinomyces israelii:** Related to IUCD.
- **TB:** Suspected if PID in virgin.

Risk factors:

- **Age:** Incidence of acute PID ↓↓ with ↑↑ age.
- **STDs:** There is strong correlation between exposure to STDs & PID.
- **Contraception:** mainly **IUCD** [Barrier methods and progesterone only types are Protective].
- **Procedures:** IUCD insertion, endometrial curettage, HSG & hysteroscopy.
- **History of acute PID.**

Pathology: **there are two types**

A) Acute PID:

1) **Endometritis.**
2) **Salpingo-oophoritis.**
3) **Parametritis (pelvic cellulitis): commonly cause by** staphylococci & E.coli.
4) **Peritonitis:** may be localized [pelvic abscess] OR generalized.
5) **Pelvic thrombophlebitis:** can results in septicemia.

B) Chronic PID (chronic salpingitis): which can present as

1) **Hydrosalpinx:** catarrhal salpingitis, the tube is filled with watery fluid, commonly occurring with gonorrhea.
2) **Pyosalpinx:** suppurative salpingitis, the tube is filled with pus.
3) **Chronic interstitial salpingitis:** Thick fibrous wall + stenosed lumen.
4) **Salpingitis isthmica nodosa.**
5) **Tubo-ovarian cyst:** hydrosalpinx communicating with ovarian cyst.
6) **Tubo-ovarian abscess:** pyosalpinx communicating with ovarian cyst.
7) **Chronic specific salpingitis:** TB salpingitis.

Diagnosis: Center for Disease Control (CDC) criteria for diagnosis of acute PID are:

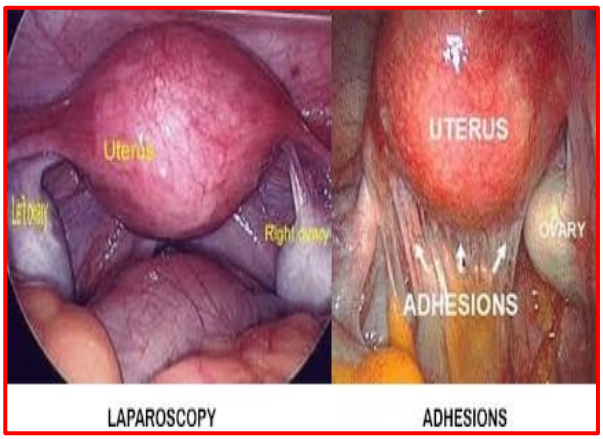
A) Minimum (major) criteria:

1) Lower abdominal pain: commonest symptom (occurs in > 90% of patients).

2) Lower tenderness ± rebound.
3) Tender cervical motion.
4) Adnexal tenderness (unilateral or bilateral).

B) Routine (minor) criteria:

1) Oral temperature > 38.3°C.
2) Abnormal cervical or vaginal discharge.
3) ↑↑ leukocytic count, CRP & ESR.
4) Positive cervical culture for N. gonorrhea or C. trachomatis.
5) Fluid in Douglas pouch or pelvic abscess.



C) Sure criteria:

1) Histopathological evidence of endometritis in endometrial biopsy.
2) Tubo-ovarian abscess on ultrasound or other radiological tests.
3) Laparoscopic abnormalities consistent with PID (gold standard for diagnosis).

Treatment:

1- Antibiotics:

- It should be started as soon as diagnosis is suspected or confirmed by culture result.
- Treatment is based on fact that PID is due to polymicrobial infection.
- Empiric antibiotic protocols should cover wide range of bacteria including N. gonorrhea, C. trachomatis & aerobic & anaerobic bacteria.

Outpatient antibiotic therapy regimens:

1. **Third generation cephalosporin (Cefotaxime)** + **Doxycycline** for 14 days
2. **Ofloxacin** + **Clindamycin** or **Metronidazole** for 14 days

2- Supportive measures:

1) Bed rest (best in Fowler's position).
2) Hydration.
3) Analgesics.
4) Removal of IUCD.
5) No intercourse + treatment of husband from STDs.

3- Reevaluation after 2-3 days: if there is no response, hospitalization of the patient.

Other indications for hospitalization are:

- Uncertain diagnosis or suspected surgical emergency (as appendicitis).
- Pelvic abscess is suspected.

- Patients are adolescent.
- Patients are pregnant.
- Immunosuppressed patients OR those with HIV infection.
- Severe systemic illness.

Inpatient (hospital) antibiotic therapy regimens:

1. **Cefoxitin** (2 gm/6 hrs IV) + **Doxycyclin** (100 mg/12 hrs orally)
2. **Clindamycin** (800 mg/8 hrs IV) + **Gentamycin** (80 mg/8 hrs IM or IV)

- Either regimen is continued for 48 hrs after clinical improvement.
- After hospital discharge, **Doxycyclin** or **Clindamycin** is given to complete 14 days ***Whether outpatient or inpatient regimen is used, the patient is cured if repeat culture revealed –ve after 2 weeks of treatment.***

4- Surgical treatment:

1. **Laparoscopy**: for diagnosis, obtaining culture and excluding surgical emergencies.
2. **Laparotomy:** indicated for patients with surgical emergencies, ruptured abscess, failed medical treatment. Via laparotomy one of the following could be done:
   - **Peritoneal toilet, drainage of pus and removal of pelvic abscess.**
   - **Removal of adhesion.**
   - **Unilateral salpingo-oopherectomy.**
   - **TAH and BSO.**

Complications of PID:

1-Chronic pelvic pain: due to hydrosalpinx or adhesions.
2-Infertility: due to peritubal or periovarian adhesions or tubal obstruction.
3-Ectopic pregnancy.
4-Parametritis, peritonitis & pelvic thrombophlebitis.
5-Hydrosalpinx & pyosalpinx.
6-Tubo-ovarian abscess.
7-Fitz Hugh Curtis syndrome: acute PID + perihepatitis.
- **It occurs due to** vascular or transperitoneal dissemination of N. gonorrhea or C. trachomatis.
- **There is:** Rt upper quadrant pain and tenderness + pleuritic pain.

8-Mortality: 5-10% in ruptured tubo-ovarian abscess.

Types of pelvic abscesses:

1) **Tubo-ovarian abscess:** as a complication of PID.
2) **Postsurgical abscess:** as post hysterectomy abscess.
3) **Puerperal pelvic abscess:** following postpartum infection.
4) **Non gynecologic abscess:** as appendicular abscess.

Sexually transmitted Infections (STIs)

Definition: diseases transmitted by sexual contact.

Classification:

A-Viral:

1-Human papilloma virus (HPV). 2-Herpes simplex virus (HSV).
3-Cytomegalovirus (CMV). 4-Molluscum contagiosum virus.
5-Human Immune Virus (HIV) the causative organism of AIDS.
6-Hepatitis B virus (HBV). 7-Hepatitis C virus (HCV).

B-Bacterial:

1-Gonorrhea.(N. gonorrhea) 2-Syphilis (T. pallidum)
3-Granuloma inguinale (Donovanosis) ; klebsiella granulomatis
4-Chancroid (soft sore); hemophilus ducryi
5-Gardnerella vaginalis. 6-Shigella vaginitis.
7-Group B streptococci.

C-Fungal:

1-Candida Albicans. 2-Torulopsis glabrata.

D-Protozoal:

1-Trichomonas vaginalis. 2-Entameba histolytica vaginitis.
3-Giardia lamblia vaginitis.

E-Parasitic:

1-Tenia pubis. 2-Sarcoptes scabiei.

F-Chlamydial: C. trachomatis.

G-Genital mycoplasma:

1-Mycoplasma hominis. 2-Urea-plasma urealyticum.

Some important Sexually Transmitted Infections

Human papilloma virus = HPV

- It is the most common viral STD caused by DNA Popova virus.
- It affects the vuvla, vagina, cervix, perineum and anus as condyloma accuminata (genital warts).
- Types 16 &18 cause flat warts with the risk of CIN.
- Types 6 &11 cause exophytic warts with no malignant potential.

● **Diagnosis:**

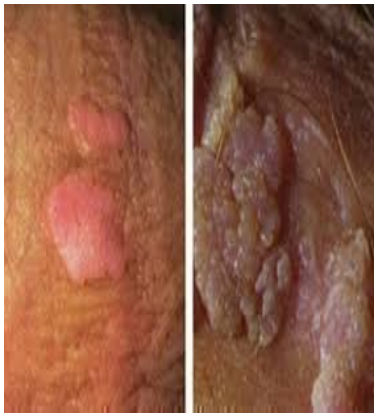
- The characteristic lesion is genital wart which may be single or multiple.
- Direct demonstration of virus by electron microscopy, detection of viral Ag & PCR.

- Viral isolation on tissue culture.
- Serology for detection of viral Abs in the patient's serum.
- Pap smear: Koilocytosis may present [some cells with atypia or dysplasia].
- Colposcopy is mandatory for all cases to exclude atypical cells.

● **Treatment:**

1) ***Small lesion:*** Topical podophyllin, trichloroacetic acid or 5-fluorouracil.

2) ***Large lesion:*** Cryotherapy, electrocautery or laser therapy.



Genital warts

Herpes simplex virus (HSV)

Causative organism: DNA herpes simplex virus type II.

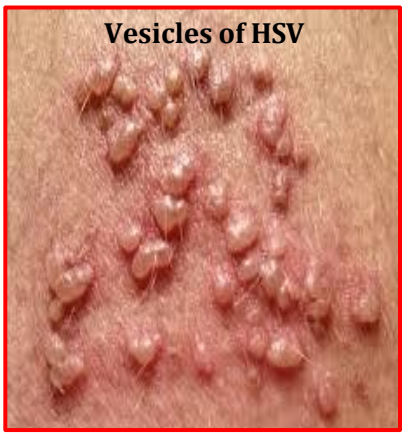
Diagnosis:

- Multiple vesicles which may coalesce together to form superficial painful ulcers.
- **Direct demonstration of virus:** after staining by Giemsa stain (presence of multinucleated giant cells is suggestive).
- **Viral isolation:** On tissue culture.
- **Serology:** for detection of viral Abs in patient's serum.

Treatment:

1) ***Antiviral drugs:*** Acyclovir, Iodoxuridine & Vidarabine.

2) ***Symptomatic treatment:*** Analgesics.



Vesicles of HSV

Acquired immunodeficiency syndrome (AIDS)

Causative Organism: Human immunodeficiency virus (HIV); RNA retrovirus. It attacks T4 lymphocytes → cell death OR T4 dysfunction with altered body immunity.

Diagnosis: the patient may be a carrier for many years.

- Aids related complex: Lymphadenopathy, oral candidiasis, pneumonias, recurrent viral infections like HSV or cytomegalovirus, T.B., toxoplasmosis.
- Persistent lymphadenopathy, fungal, bacterial and viral infections.
- ***Direct demonstration of virus:*** by PCR.
- ***Viral isolation:*** by lymphocyte culture.
- ***Serology:*** Detection of viral Abs in patient's serum by ELIZA or Western blotting.

Treatment: Mainly prophylactic to avoid acquiring infection.

Gonorrhea

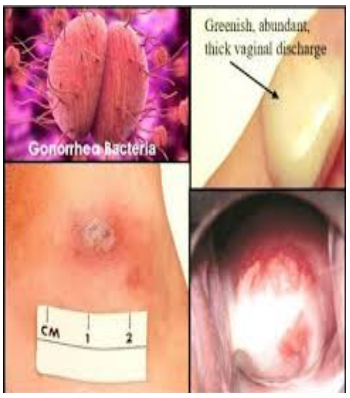
Causative Organism: Neisseria gonorrhea (gram –ve intracellular diplococci).

-It affects areas lined by thin epithelium (columnar OR transitional) not stratified epithelium.

- It may cause urethritis, Bartholinitis, cervicitis, salpingitis (PID) or arthritis (via blood spread).

Diagnosis:

- Burning urination, frequency, dysuria with urethritis.
- Discharge with Bartholinitis or cervicitis OR even picture of PID.
- ***Direct demonstration of organism:*** by Gram stain.
- ***Culture and sensitivity:*** On Thayer martin or chocolate agar.
- ***Serology:*** Detection of Abs in patient's serum.



Treatment: Penicillin or cephalosporins.

Syphilis

Causative Organism: Treponema pallidum.

Pathology: Passes in 3 phases that can be present clinically as:

1) ***Primary syphilis:*** with the characteristic lesion (chancre or hard sore = small painless ulcers) on genital or extragenital organs + enlarged regional LNs.

2) ***Secondary syphilis:*** Generalized skin rash, mucus patches in mouth & condyloma lata.

3) ***Tertiary syphilis:*** Gumma in nervous & cardiovascular systems, liver, eye, bone …..

Diagnosis:

1) ***Direct demonstration of organism:*** Dark field microscopy.

2) ***Culture:*** on Smith-Noguchi's media or rabbit epithelial cells.

3) ***Serology:*** detection of Abs in patient's serum by

- **Non-specific tests** as (VDRL = venereal disease research laboratory test) test or (RPR = rapid plasma reagin test).
- **Treponemal specific tests** as Fluorescent treponemal Ab absorption test (FTA-ABS) and Treponema pallidum immobilization test (TPI).

Treatment: Benzathine penicillin-G is the drug of choice.

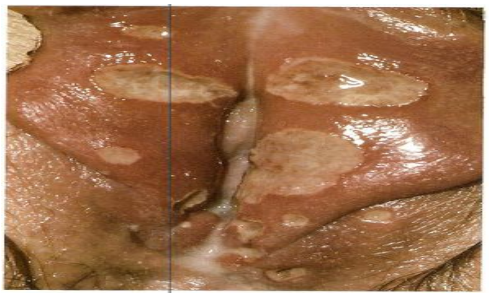
Chancroid

Causative Organism: Hemophylus ducreyi.

Diagnosis:

- Painful vulval ulcer + lymphadenopathy.
- ***Direct demonstration of organism:*** by Gram stain.
- ***Culture:*** on Chocolate agar.

Treatment: Erythromycin.


CHANCROID


Chncre of syphilis

Granuloma inguinale

Causative Organism: Campylobacter granulomatis.
Diagnosis:
- Painless vulval nodule that progresses to ulcer + **No** lymphadenopathy.
- ***Direct demonstration of organism:*** by Gram or Wright stain (presence of Donovan bodies inside macrophages are essential for diagnosis).

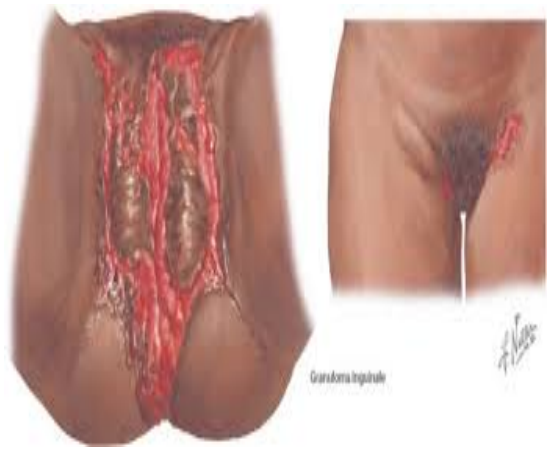
Treatment: Tetracycline.

Chlamydial infections (*lymphogranuloma venereum*)

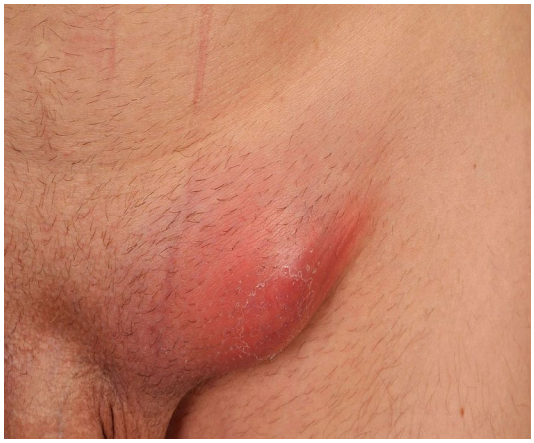
Causative Organism: **chlamydia trachomatis, the commonest STD in sexually active women.**
- It is obligate intracellular organism between viruses & bacteria.
- It is of 15 serotypes.
- Serotypes L1, L2, L3 cause ***lymphogranuloma venereum (LGV)*** i.e., chronic infection of lymphatic tissues in genital region (vulva, urethra, rectum & cervix) that passes in 3 phases:
  a) Primary phase: shallow painless ulcer on labia.
  b) Secondary phase: painful inguinal & perirectal lymphadenopathy → if untreated, LNs adhere together and overlying skin to form buboes (if both superficial inguinal LNs & femoral LNs are affected → inguinal ligament will form groove between them "groove sign" which is characteristic for LGV).
  c) Tertiary phase: rupture of buboes, formation of multiple sinuses & fistulas discharging pus.
- Serotypes D, E, F, G, H, I, J, K cause genital infections other than LGV as urethritis, Bartholinitis, cervicitis, endometritis & salpingitis (PID).

Diagnosis:
- Clinical manifestations according to the phases mentioned before.
- ***Direct demonstration of organism:*** by detection of chlamydial Ag by monoclonal Abs via ELISA (the test of choice).
- ***Culture:*** tissue culture.

Treatment: doxycycline.



Granuloma inguinale



Lymphogranuloma venerium

For more information about Sexually Transmitted Infections follow these links:

Type	Disease
Bacterial	Chancroid Chlamydial urethritis and cervicitis Gonorrhea Granuloma inguinale Lymphogranuloma venereum Syphilis
Viral	Genital herpes simplex Genital warts (caused by the human papillomavirus) Human immunodeficiency virus (HIV) infection or AIDS Molluscum contagiosum (caused by a poxvirus)
Parasitic (protozoan)	Trichomoniasis
Arthropod	Pubic lice infestation Scabies (due to burrowing mites)

PART FIVE
GYN ONCOLOGY

- **INTRODUCTION TO GYNECOLOGIC ONCOLOGY**
- **ENDOMETRIAL CARCINOMA**
- **CHORICARCINOMA**
- **CARCINOMA OF THE CERVIX UTERI**
- **VULVAR CARCINOMA**
- **OVARIAN MALIGNANCIES**
- **GYNECOLOGIC OPERATIONS**

INTRODUCTION TO GYNECOLOGICAL ONCOLOGY

Definition: it is benign OR malignant tumor arising from.

Incidence: total incidence, age incidence, parity incidence, racial incidence.

Etiology:

- All uterine tumors depend on hyper-estrogenic states.
- Cervical and vulval tumors depend on STDs especially "HPV and HSV".
- Ovarian tumors depend on ovulation trauma and dysgenetic gonads.
- In all tumors, there is pre-malignant states and genetic or familial tendency.

Naked eye: site, size, shape, surface, surroundings, consistency, cut section and capsule.

Microscopic examination:

- Cell of origin + benign criteria OR malignant criteria.
- **Benign** criteria are hyperplasia, hypertrophy, and mitotic figures 2 or less.
- **Malignant** criteria are:

1-Large nuclei of various sizes and shapes. 2-Irregularly clumped chromatin with deep basophilia.
3-Thickened nuclear membrane. 4-Prominent nucleoli.
5-Increased nuclear to cytoplasmic ratio. 6-Loss of polarity and infiltration to surroundings.

- **Histological grading of the cells in malignant type:** well differentiated tumors, moderately differentiated tumors, poorly differentiated, and undifferentiated tumors.

Spread: *by*

- **Lymphatic:** it is the main route in all tumors except choriocarcinoma **"by blood".**
- **Blood:** to LBLB "lung, bone, liver, brain". it is the main route in choriocarcinoma.
- **Direct:** to surroundings.
- **Special routes.**

Complications:

- HIND "hemorrhage, infection, necrosis, and degeneration".
- Spread local causing destruction of the organ and malignant fistula.
- Spread general leading to cachexia, loss of weight, pallor, anemia, jaundice...etc.

Staging:

Stage 0: CIS=carcinoma in-situ i.e., tumor not invading the basement membrane.
Stage 1: tumor confined to the organ of origin.
Stage 2: tumor extension to nearest surroundings by direct spread.
Stage 3 : more spread and lymph nodes involvement.
Stage 4 : metastatic tumor:

- **S 4a:** bladder and rectum involvement and
- **S 4 b:** distant metastasis

DIAGNOSIS

1- *Clinical:*

- **Symptoms** "ABCDM".
- **Signs:** general cachexia, loss of weight, pallor anemia, jaundice, abdominal and local = N/E.

2- *Investigations:*

- **Laboratory:** as usual + tumor markers.
- **Radiology:** bone survey, IVP, US, CT, MRI.

- **Endoscopy:** cystoscope, proctosigmoidoscopy, and one according to the site of the tumor [hysteroscope for uterine, laparoscope for ovarian and colposcope for vaginal, vulval and cervical tumors].
- **Histopathology**: microscopic examination mentioned before.

TREATMENT

1- **Prophylactic:** by avoidance of the causes and early detection of the tumors (by good screening in high-risk groups).

2- ***Active:***

- Correction of general condition and specific tumor therapy that may be:
  - **Surgery:** nearly in all tumors.
  - **Radiotherapy:** is the main line in cancer cervix.
  - **Chemotherapy:** is the main line in choriocarcinoma.
  - **Combined palliative treatment:** in advanced cases + pain killers, tonic, vitamins, leave the patient dying in peace NOT in pieces.

ENDOMETRIAL CARCINOMA

PATHOLOGY

Definition: it is a malignant tumor arising from the endometrium.

Incidence:

Total: the ***most common*** gynecologic cancer in the developed countries "about 1% in all postmenopausal females due to:

- Prolonged life expectancy.
- More use of post-menopausal hormone replacement therapy.
- Early and increased rate of diagnosis of the tumor.

Age: the median age 60 years [75 % of cases], some cases occur around menopause [25% of cases], rarely occur before the age of 40 years.

Parity and race: more common in nulls, white races and high classes.

Etiology:

1- *Chronic unopposed estrogen stimulation*: from either endogenous or exogenous sources.

2- *Tamoxifen therapy:* following breast carcinoma.

3- *Corpus cancer triad*: endometrial cancer is more with "D.M + hypertension + obesity".

4- *Previous pelvic irradiation*: risk for sarcoma and not carcinoma.

5- *Genetic and family history*: of cancer breast, ovary or colon "Lynch II syndrome".

6- *High socioeconomic status* and urban residence.

7- *Pre-malignant lesion*: mainly endometrial hyperplasia especially with atypia.

8- *Endometrial metaplasia*: common with senile endometritis and chronic pyometra.

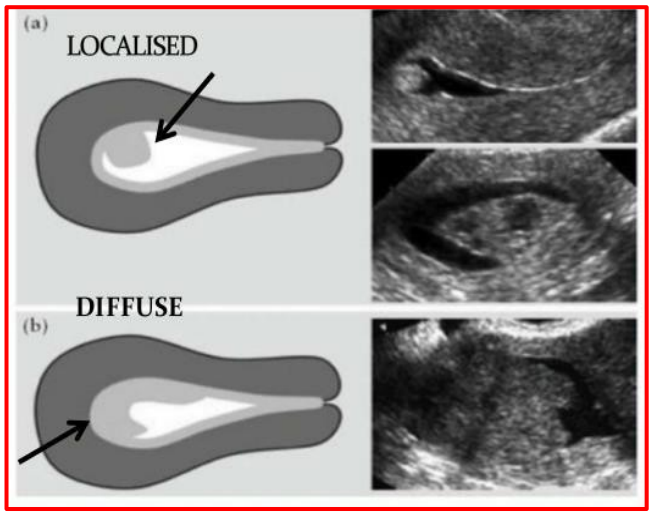
Naked Eye:

Site: Endometrial carcinoma is either focal or diffuse lining the endometrium.

Shape: It appears as pale friable and necrotic sessile or pedunculated mass.

Size: It is either limited to the endometrium or invading the myometrium and serosa.

Microscopic Examination:



1- *Endometrial adenocarcinoma (80 %):* is the usual type.

2- *Adenoacanthoma (15 %):* adenocarcinoma and benign squamous element.

3- *Pure squamous cell carcinoma.*

4- *Mucinous carcinoma.*

5- *Clear cell carcinoma.*

6- Papillary serous carcinoma.

7- Undifferentiated carcinoma.

8- Mixed carcinoma.

Spread:

- Endometrial carcinoma tends to remain limited to the endometrium for a long time and acts as a surface rider due to presence of glycoprotein layer preventing penetration.

1. **Direct** spread to the cervix, vagina, tubes ovaries…etc.
2. **Lymphatic** spread to internal and common iliac lymph nodes, then to para-aortic nodes.
3. **Blood** spread to the lung, liver, bone and brain [but rare].
4. **Special route** e.g., trans-cavitary and implantation spread.

Complications:

-HIND + spread local and malignant fistula + spread general causing cachexia, loss of weight, pallor, anemia, etc.

-There may be bleeding and pyometra.

-Intestinal obstruction.

- Main cause of death is **peritonitis**.

Staging: (surgical staging -FIGO, 2023)

(For detailed staging see next page)

Stage	Description
Stage I	**Confined to uterine corpus and ovary**
Stage II	**Invasion of cervical stroma with no extrauterine extension, or substantial LVSI b, or aggressive histological type with any myometrial invasion**
Stage III	**Local and/or regional metastasis of any histological subtype.**
Stage IV	**Spread to the bladder mucosa and/or intestinal mucosa and/or distant metastasis.**

The 2023 FIGO staging of endometrial cancer .

Stages	Description
Stage I	**Confined to uterine corpus and ovary**
IA	**Disease limited to the endometrium OR non-aggressive histological type, with < 50% myometrial involvement, no focal LVSI b , or good disease prognosis.**
IA1	**Non-aggressive histological type limited to an endometrial polyp or confined to endometrium.**
IA2	**Non-aggressive histological type with < 50% myometrial involvement with no to focal LVSI b**
IA3	**Low-grade endometrioid carcinomas limited to uterus or ovary a**
IB	**non-aggressive histological type with > 50% myometrial invasion, and no to focal LVSI b**
IC	**Aggressive histological subtype limited to a polyp or confined to endometrium.**
Stage II	**Invasion of cervical stroma with no extrauterine extension, or substantial LVSI b, or aggressive histological type with any myometrial invasion.**
IIA	**non-aggressive histological type with invasion of cervical stroma.**
IIB	**non-aggressive histological type with substantial LVSI b**
IIC	**Aggressive histological subtype with any myometrial involvement.**
Stage III	**Local and/or regional metastasis of any histological subtype.**
IIIA	**Invasion of uterine serosa, adnexa, or both.**
IIIA1	**Spread to ovary or fallopian tube (excluding lesions that meet stage IA3 criteria a).**
IIIA2	**Spread to uterine subserosa or through uterine serosa.**
IIIB	**Metastasis or direct spread to the vaginal canal and/or parametria or pelvic peritoneum.**
IIIB1	**Metastasis or direct spread to the vaginal canal and/or parametria.**
IIIB2	**Metastasis to pelvic peritoneum.**
IIIC	**Metastasis to pelvic and/or para-aortic lymph nodes.**
IIIC1	**Metastasis to pelvic lymph nodes.**
IIIC1i	**Micro-metastasis to pelvic lymph nodes.**
IIIC1ii	**Macro-metastasis to pelvic lymph nodes.**
IIIC2	**Metastasis to para-aortic lymph nodes up to the renal vessels, and/or pelvic lymph node metastasis.**
IIIC2i	**Micro-metastasis to para-aortic lymph nodes up to the renal vessels, and/or pelvic lymph node metastasis.**
IIIC2ii	**Macro-metastasis to para-aortic lymph nodes up to the renal vessels, and/or pelvic lymph node metastasis.**
Stage IV	**Spread to the bladder mucosa and/or intestinal mucosa and/or distant metastasis.**
IVA	**Spread to the bladder mucosa and/or intestinal mucosa.**
IVB	**Abdominal peritoneal metastasis beyond the pelvic peritoneum,**
IVC	**Distant metastasis, included metastasis to any extra-abdominal or intra-abdominal lymph nodes superior to the renal vessels, lungs, liver, brain, or bone.**

---

a Concurrent uterine and ovarian low-grade endometrioid endometrial carcinoma are included in this stage if the following criteria are met: (1) No more than 50% of myometrial involvement, (2) absence of extensive LVSI, (3) absence of additional metastasis, and (4) unilateral ovarian involvement without capsule rupture or invasion.
b Lympho-vascular invasion.

Diagnosis

1- Clinical:

a. Symptoms:

1- **Asymptomatic:** and accidentally discovered on screening.

2- **Vaginal bleeding** or serosanguinous discharge:

- May be the only presenting complaint in 90 % of women.
- Postmenopausal bleeding is the **commonest** complaint.
- Premenopausal irregular bleeding in up to 25% of cases.

3- **Pain:** may present with pelvic discomfort or lower abdominal pain due to uterine enlargement "Sampson's pain".

4- **Complications:** symptoms of hematometra/pyometra or, symptoms of distant metastasis.

b. Signs:

1- **General exam:** may show obesity, hypertension, cachexia, loss of weight, pallor, etc.

2- **Abdominal exam:** in advanced cases there may be;

- Hepatomegaly and ascites.
- Palpable omental or hepatic metastases OR lymph nodes.
- The uterus is usually impalpable unless associated with fibroid or pyometra.

3- **Pelvic exam:** the uterus may be:

- Normally in size as the tumor grows slowly.
- Smaller than normal due to post-menopausal atrophy.
- Larger than normal due to rapid tumor growth, pyometra, or associated myomas.

- The adnexa are palpated for masses.
- Per rectal examination is done for extension of the tumor.

2- Investigations:

1- Laboratory: CBC, renal & liver function tests, urine analysis, and tumor markers.

2- Radiology:

- Chest X-Ray and bone survey.
- Intravenous pyelography and barium enema.
- Ultrasonography, CT, MRI scans for masses, endometrial thickness and lymph nodes.

3- Endoscopes:

- Cystoscopy, Procto-sigmoidoscopy.
- Hysteroscopy: more accurate in diagnosing associated polyps and sub-mucus myomas and direct visualization of the cavity and taking biopsies

4- Histopathology: could be done by

1. **Office endometrial biopsy:**
2. **Endo-cervical curettage:**
3. **Fractional dilation and curettage (D&C):** The malignant tissues are suspected by:
   - Plentiful necrotic tissues "endless curettage".
   - It increases bleeding rather than decreasing it with no gritty sensation reached.
4. **Hysteroscopic guided biopsy [best].**

Treatment

1- Prophylactic:

1. By avoidance of the causes and proper treatment of hyper-estrogenic states.
2. Proper screening for endometrial carcinoma and early management of endometrial hyperplasia.

2- Active:

1- Correction of general condition: by tonics, blood transfusion, vitamins…

2- Specific tumor therapy: surgery is the mainstay of treatment.

Plan of treatment:

1- Surgical staging:

a)-TAH & BSO (the standard primary treatment for all cases).
b)-Peritoneal cytology (washing of the pelvis & abdomen).
c)-Intraoperative evaluation of depth of myometrial invasion.
d)-Pelvic & para-aortic LNs sampling is indicated in:

1- Poorly differentiated cancer.
2- Invasion of more than 1/2 of the myometrium.
3- Extension to cervix.

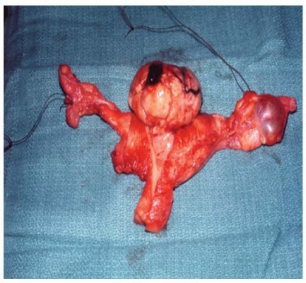


Figure 5-12 Total abdominal hysterectomy (TAH) and bilateral salpingo-oophorectomy (BSO) showing large polypoid adenocarcinoma of the endometrium with deep myometrial invasion.

- Total abdominal hysterectomy with bilateral salpingo-oophorectomy may be followed by:

2 - Radiotherapy:

Vaginal cuff irradiation, whole pelvic irradiation, extended field irradiation or whole abdominal irradiation.

3- Chemotherapy: Adriamycin, cisplatin or carboplatin.

4- Hormonal therapy: Anti-estrogens (gestagens or GnRH agonists).

In advanced Stage IV Carcinoma: surgery is followed by combined treatment of

- Adjuvant radiotherapy.
- Adjuvant chemotherapy.
- Hormonal therapy (progestins or depot provera).

Recurrent or severe disease: by combined palliative treatment

Follow up visits:

-Every 3 months for one year.
-Every 6 months for 2 years.
-Every year for years 4 & 5.

Treatment of enbdometrial carcinoma by stage

Stage IA: total hysterectomy/BSO/PPALND
Stage IB: total hysterectomy /BSO/PPALND ± adjuvant pelvic XRT
Stage II: total hysterectomy /BSO/PPALND, adjuvant pelvic XRT
Stage III: total hysterectomy /BSO/PPALND, adjuvant chemotherapy
Stage IV: chemotherapy and palliative radiation

5-year survival by stage :

Stage I: 81-91% (72% diagnosed at this stage)
Stage II: 71-78%
Stage III: 52-60%
Stage IV: 14-17% (3% diagnosed at this stage)

CHORIOCARCINOMA

Definition: it is malignant gestational trophoblastic tumor

Incidence:

Total: far east: 1/5000 while in USA and Europe 1/20000.

Age: common in middle age group "age of pregnancy.

Etiology and Risk Factors:

- **Maternal age:** increased incidence with age >35 years.
- **Parity:** vesicular mole is common in patients with low parity while choriocarcinoma is common in multipara.
- **Diet:** common in rice eating and spicy cooking population.
- **Genetic:** common association with trisomy 16.
- **Past history** of molar pregnancy as the disease is found:

1. 50% following vesicular mole.
2. 25% following abortion or ectopic pregnancy.
3. 20% following normal pregnancy.

Naked/eye: there are two types:

1. **Non metastatic choriocarcinoma** "Placental site tumor":
   - The disease is confined to the uterus, produces little amount of HCG and HPL.
   - Although locally malignant, it can invade the myometrium and blood vessels.
   - Treatment is always by hysterectomy as the tumor is *chemo resistant*.
2. **Metastatic choriocarcinoma:** which in turn is subdivided into;
   - Low risk (with good prognosis) and high risk (with poor prognosis)
   - It may be circumscribed soft dark red nodule, polypoidal mass OR diffuse mass affecting the whole uterine cavity.
   - It is usually chemo-sensitive.

Microscopic examination: there are --

- Sheets of malignant trophoblasts interspersed with blood lakes.
- There is variable degree of myometrial invasion.
- Absence of intact villi (pathognomonic).
- Areas of Arias Stella reaction may be present.

Spread: as usual but the main route is blood spread and the commonest site is the lung.

Complications: HIND, spread local, spread general and effects of HCG.

Staging (FIGO 2000):

- **Stage I:** tumor confined to the uterus.
- **Stage II:** genital extension (vagina and pelvic organs).
- **Stage III:** the disease affects the lungs with or without genital metastasis.
- **Stage IV:** distant metastasis affecting other organs.

***FIGO staging further classified each stage into*:**

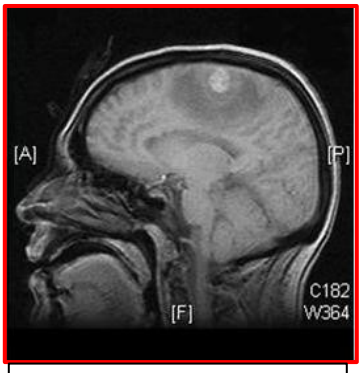
A. With no risk factors.

B. With one risk factor.

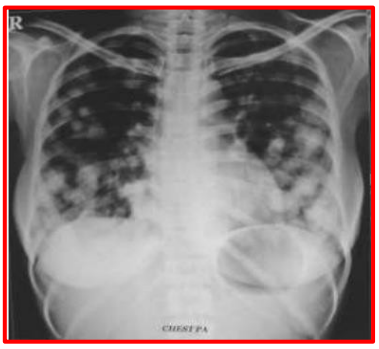
C. With two risk factors.

The risk factors were:

1. HCG level > 100,000 mIU/ml
2. Last pregnancy since > 6 months OR more.



Brain and lung metastasis



Diagnosis

1- Clinical:

(A)-symptoms:

1- *History of*: vesicular mole, abortion, full term pregnancy or ectopic pregnancy.

2- *Bleeding*: which is excessive irregular vaginal bleeding.

3- *Pain*: with uterine distension or perforation.

4- *Cachexia* with recurrent blood loss, anorexia and fever.

5- ***Complications***: e.g.

-Fever due to infection.

-Hemoptysis with lung metastasis or jaundice with liver metastasis.

-Hyperemesis, hypertension, thyrotoxicosis and increased intracranial tension.

6- **Mass**: vaginal or abdominal mass.

(B)-Signs:

7- **General**: signs of complications e.g.

-Pallor, fever, hypertension, thyrotoxicosis.

-Pleural rub. -Cachexia and jaundice.

8- **Abdominal:**

-Enlarged uterus. - Ovarian cysts. -Hepatic enlargement, ascites…etc.

9- **Local:**

-Vaginal metastasis. - Symmetrically enlarged soft, tender uterus.

- Adnexal swelling [cystic ovaries].

2- Investigations:

1- Laboratory tests: CBC, RFT, LVT, serum HCG, CSF level of HCG.

2- Radiology:

- Chest X- ray (Canon ball appearance), bone survey, isotope scan
- Ultrasonography to diagnose any uterine mass and ovarian cysts.
- CT scan and MRI on liver, brain…etc.

3- Endometrial curettage and biopsy.

TREATMENT

1- Prevention:

- Proper treatment and follow up of every case of vesicular mole.
- Prophylactic chemotherapy after vesicular mole in high-risk cases.

2- Active:

1- *Correction of general condition and* ttt of complications as hypertension, thyrotoxicosis.

2- *Specific tumor therapy*: treatment depends on WHO 2000 prognostic Scoring System.

3- *Main lines of treatment include:*

A- Stage I (non-metastatic):

i- Fertility is desired:

-Single agent chemotherapy: by **Methotrexate** that cures more than 90% of patients.
-Combination chemotherapy: by Methotrexate + Actinomycin D.
-Local resection.

ii- Fertility is not desired:

-Hysterectomy with single agent chemotherapy.
-Hysterectomy alone in placental site tumor.
-The patient is followed up by Pregnancy test OR B-HCG titer till become –ve.
-COCs or Depo provera is used for one year.

B- Stage II and III (metastatic):

i- Low risk: single agent chemotherapy: Methotrexate or Actinomycin D.

ii- High risk:

-Multimodal approach: combination chemotherapy "e.g., MEA and EMACO", surgery and radiation-therapy.
-Vaginal metastasis: local resection or embolization of internal iliac artery.
-Lung metastasis: combination chemotherapy and lobectomy may be needed.
-Hysterectomy.

C- Stage IV:

i- Primary: combination chemotherapy **"e.g., MEA and EMACO".**

ii- Liver metastasis:

-Selective arterial chemotherapy.
-Arterial embolization of hepatic artery.
Local resection.

iii- Brain metastasis:

- Whole brain irradiation.
- Combination chemotherapy and intrathecal methotrexate.
- Craniotomy and resection of the mass.

- M-EA = Methotrexate + Etoposide + Actinomycin-D.
- EMACO = Etoposide + Methotrexate + Actinomycin-D + Cyclophosphamide + Oncovin.

Other types of uterine malignancies:

- Leiomyosarcoma-----→Rapidly growing fibroid should be evaluated
- Stromal sarcoma
- Carcinosarcoma (MMMT)

CERVICAL INTRA-EPITHELIAL NEOPLASIA (CIN)

PATHOLOGY

Definition: locally malignant tumor of cervical epithelium not invading the basement membrane.

Incidence:

- The disease is more common in "young ages "25-35 years" that are sexually active".

Etiology and risk actors:

1. Sexually transmitted diseases:

- Infections with high risk are **HPV types 16 and 18** "rarely 45, 56, 31, 33, 35".
- This is evidenced by:
  1. The disease is common in those with early age of sexual activity OR 1st pregnancy.
  2. Common in those with multiple sexual partners "4 times increase"
  3. Sperm integration theory
  4. Common association between clinical manifestation of CIN and infection.
- Other infection by HSVII also can contribute.

2. Other risk factors include:

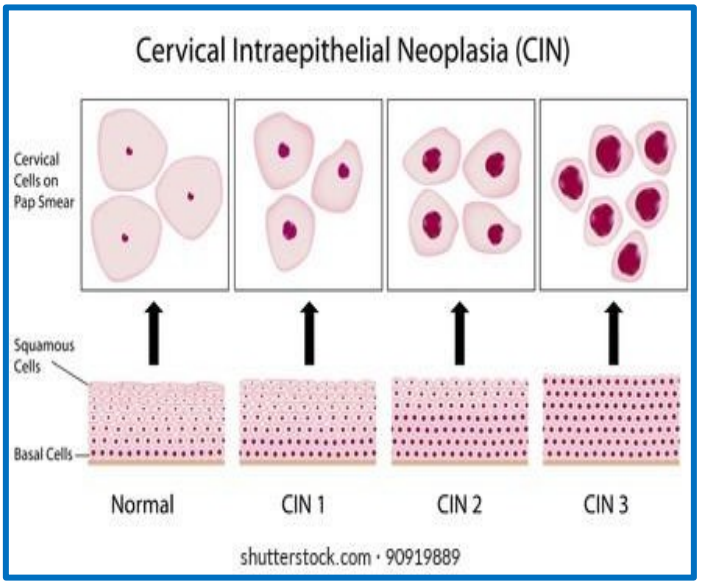
1. Smoking, alcoholics and drug abuse.
2. Prolonged hormonal contraception with HPV infection.
3. Immune-suppression and AIDS.

Naked/eye: the cervix appears normal, picture of infection, erosion like or red cervix.

Microscopic examination: there are 3 degrees of CIN

- **CIN-1** = infiltration of the lower 1/3 of the epithelium with malignant cells.
- **CIN-2** = infiltration of the middle 1/3 of the epithelium with malignant cells.
- **CIN-3** = infiltration of the upper 1/3 of the epithelium with malignant cells.

NE appearance of CIN



Diagnosis:

1- Clinical:

a- Symptoms:

- Asymptomatic in most patients and accidentally discovered on routine screening.
- Post-coital bleeding or vaginal spotting or even discharge.

b- Signs: local examination may reveal:

1. Normal cervix.
2. Contact bleeding.
3. Abnormal red OR erosion like areas.
4. Picture of infection.

2- Investigations: the main investigation is histopathology that is maintained by --

1. *Cervical cytology by Ayer's spatula OR cyto-brush*:

- The obtained specimen is stained with **Pap stain.** It is used as a screening test.

2. *Visual inspection with Acetic Acid "VIA test":*

- Application of acetic acid 3-5 % for 1-2 minutes, aceto-white epithelium is abnormal and should be biopsied for histopathological examination.

3. *Colposcopy and Colposcopic guided biopsy:* after application of acetic acid.

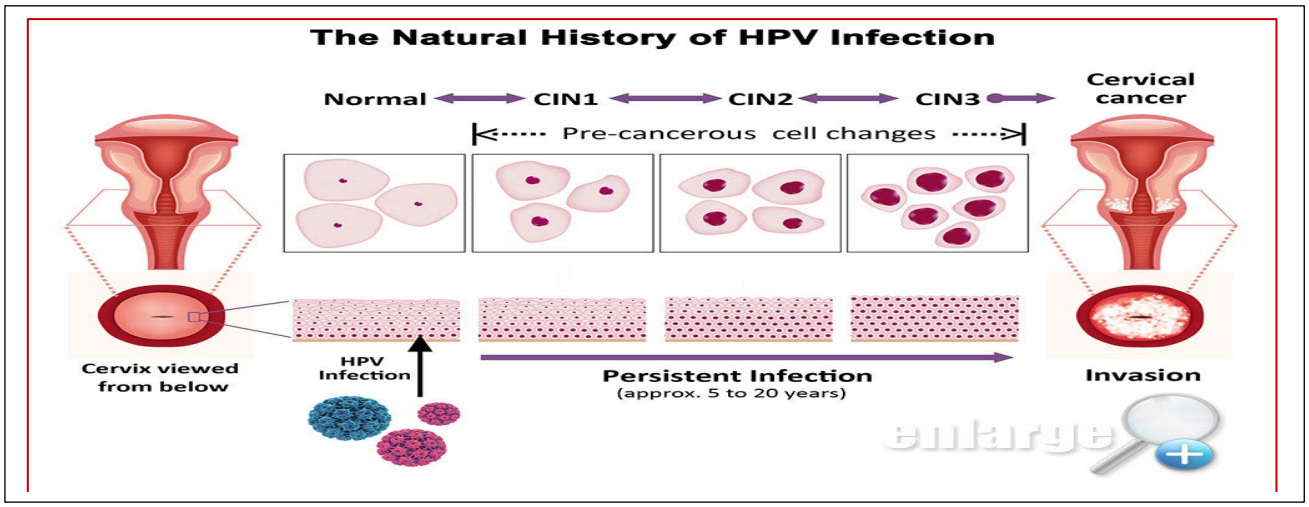
Colposcopic results finally classified into:

1. *Satisfactory*: entire transformation zone visualized and the lesion is seen in it.
2. *Unsatisfactory*: entire transformation zone not visualized or lesion seen in it.

4. **Endocervical curettage.**

- Any obtained biopsy is graded according to histo-pathological examination and degree of infiltration of the epithelium into:

1. **CIN-1** = infiltration of the lower 1/3 of the epithelium with malignant cells.
2. **CIN-2** = infiltration of the middle 1/3 of the epithelium with malignant cells.
3. **CIN-3** = infiltration of the upper 1/3 of the epithelium with malignant cells.



Treatment

1- Prophylactic:

- By avoidance of STDs, changing the sexual behavior and proper screening of those at risk.
- HPV vaccine [**Gardacil and Cervirix]**

2- Active: Current treatment modalities include:

1- Ablative procedures "failure rate 5-10%"**:**

- Cryotherapy, Laser ablation OR diathermy coagulation.

2- Excisional procedures "failure rate less than 5 %":

- Loop electo-surgical excision procedure "LEEP".
- Laser conization, Cold knife conization OR Surgical conization of the cervix.

3- Follow-up and treatment of recurrent disease:

- The recommended follow-up interval is: every **3-6** months for the first **2** years then if persistently **negative** become **annually** thereafter for **5** years.
- Most of the recurrence occurs during the first 2 years following therapy.
- Treatment of recurrence includes:

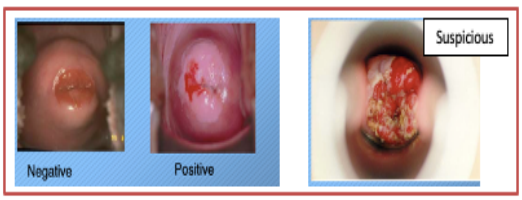
1. **CIN-I:** another ablation OR excision therapy "better" is done.
2. **CIN-II, II:** LEEP, deep conization OR hysterectomy "better".

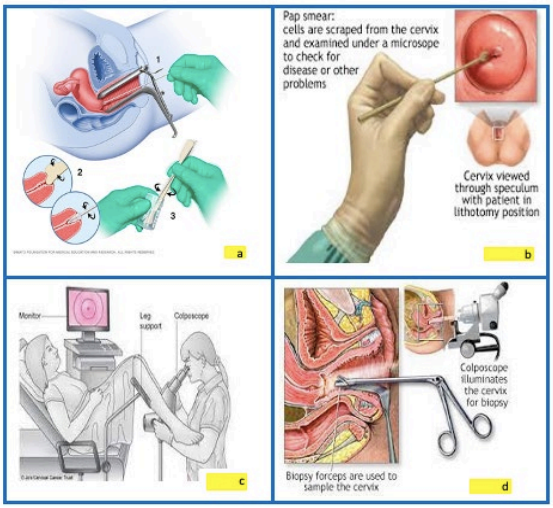
Natural history of CIN according to degree.

	Regress	Persist	Progress
CIN1	60%	30%	10%
CIN2	40%	40%	20%
CIN3	33%	55%	>12% invasive cancer

Table (4-4): VIA -test findings:

VIA CATEGORY	CLINCAL FINDINGS
Test -Negative	No acetowhite lesions or faint acetowhite lesions; polyp, cervicitis, inflammation, Nabothian cysts.
Test-positive	Sharp, distinct, well-defined, dense (opaque/dull or oyster white) acetowhite areas—with or without raised margins touching the squamocolumnar junction (SCJ); leukoplakia and warts.
Suspicious for cancer	Clinically visible ulcerative, cauliflower-like growth or ulcer; oozing and/or bleeding on touch.





CARCINOMA OF THE CERVIX

Pathology

Definition: it is malignant tumors arising from the cervical epithelium mainly the etocervix.

Incidence:

Total: the second most common female cancer after endometrial carcinoma.

Age: median age at diagnosis is 45-50 years [usually in HPV infected females). There may be increased risk at 80 years or more [increased cancer incidence with age].

Race: low socioeconomic class (due to early marriage, high parity, black race).

Parity: more in high parity and rare in nulls.

Etiology and risk factors:

1- Sexually transmitted diseases: HPV types 16 and 18 and other infections "mentioned before with CIN".

2- The other risk factors include:

1. Smoking.
2. Other STDs: herpes genitals, HIV, chlamydial infection.
3. Immune deficiency.
4. Use of hormonal contraception.
5. CIN: up to 30% of CIN-3 can develop to cancer in about 10-15 years.
6. Genetic and familial tendency.

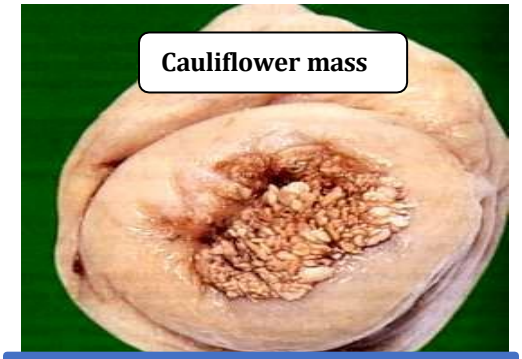
Naked/eye: invasive cancer of the cervix may arise from:

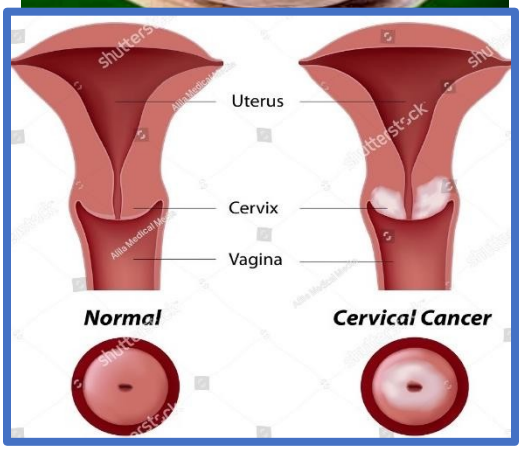
(1)- The portiovaginalis = ectocervix: which may be -

1. Micro-invasive: seen only by microscopy.
2. Invasive: seen by the naked eye and may be:
   - i. **Exophytic (cauliflower mass):** most of ectocervical cases.
   - ii. **Ulcerative type:** typical malignant ulcer.

(2)- Supravaginal portion = endocervix:

1. Microinvasive: seen only by microscopy.
2. Invasive: seen by naked eye and may be
   - i. **Endophytic:** comprise most of endocervical cases giving a barrel shaped cervix.
   - ii. **Ulcerative type:** crater like ulcer.





Microscopic examination:

1- Squamous cell carcinoma: about 80-90% of all cervical carcinoma and include: All ectocervical carcinoma ang 50% of endocervical carcinoma.

- Types of Squamous cell carcinoma: it may be →

1. Large cell keratinizing.
2. Large cell non keratinizing.
3. Small cell non keratinizing: of bad prognosis, either anaplastic or poorly differentiated.

2- Adenocarcinoma: about 10-20% and mostly from endocervical carcinoma.

3- Rare types:

1. Adeno-squamous carcinoma.
2. Sarcoma.
3. Malignant melanoma "very rare".
4. Metastatic from endometrial carcinoma and rarely from vaginal cancer.

Spread:

1- Direct spread to:

1. Cervical stroma.
2. Upwards to the body of the uterus or downwards to the vagina.
3. Parametric spread resulting in ureteric obstruction.
4. Anteriorly to the bladder or posteriorly to the rectum.

2- Lymphatic spread to:

1. Parametrial, paracervical, obturator, hypogastric, external iliac and sacral lymph nodes.
2. Secondary group of lymph nodes to common iliac and para-aortic nodes.

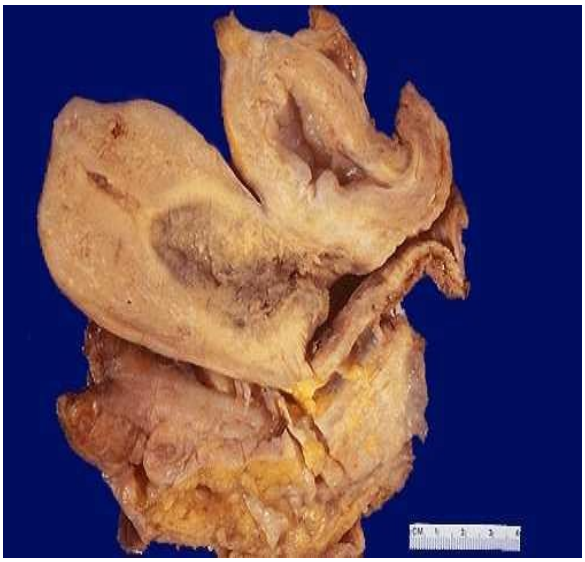
3- Blood borne metastasis: rare to lung, bone, brain and ovaries.

4- Implantation:

1. During Wertheim's operation to retroperitoneal space.
2. Vaginal fornices, lower vagina and vulva.

Complications:

1. Hemorrhage, infection "causing pyometra" necrosis and degeneration.
2. Spread local: causing renal failure and uremia (60 %) most common cause of death [due to ureteric stenosis, hydronephrosis and fistula formation].
3. Spread general causing cachexia, loss of weight, anemia, pulmonary embolism...etc.



Endophytic carcinoma with extension to the bladder

Staging:

The (FIGO) 2018 revised staging of cervical carcinoma

Stage I ;The carcinoma is strictly confined to the cervix (extension to the corpus should be disregarded)

IA- Invasive carcinoma that can be diagnosed only by microscopy with a maximum depth of invasion ≤5 mm
IA1 -Measured stromal invasion ≤3 mm in depth
IA2- Measured stromal invasion >3 mm and ≤5 mm in depth

IB- Invasive carcinoma with measured deepest invasion >5 mm (greater than Stage IA); lesion limited to the cervix uteri with size measured by maximum tumor diameter.
IB1 Invasive carcinoma >5 mm depth of stromal invasion and ≤2 cm in greatest dimension
IB2 Invasive carcinoma >2 cm and ≤4 cm in greatest dimension
IB3 Invasive carcinoma >4 cm in greatest dimension

Stage II The cervical carcinoma invades beyond the uterus, but has not extended onto the lower third of the vagina or to the pelvic wall

IIA- Involvement limited to the upper two-thirds of the vagina without parametrial invasion
IIA1- Invasive carcinoma ≤4 cm in greatest dimension
IIA2- Invasive carcinoma >4 cm in greatest dimension
IIB- With parametrial invasion but not up to the pelvic wall

Stage III The carcinoma involves the lower third of the vagina and/or extends to the pelvic wall and/or causes hydronephrosis or non-functioning kidney and/or involves pelvic and/or paraaortic lymph nodes

IIIA -Carcinoma involves lower third of the vagina, with no extension to the pelvic wall
IIIB -Extension to the pelvic wall and/or hydronephrosis or non-functioning kidney (unless known to be due to another cause)
IIIC- Involvement of pelvic and/or paraaortic lymph nodes (including micro-metastases), irrespective of tumor size and extent (with r and p notations)
IIIC1 -Pelvic lymph node metastasis only
IIIC2- Paraaortic lymph node metastasis

Stage IV: The carcinoma has extended beyond the true pelvis or has involved (biopsy proven) the mucosa of the bladder or rectum. A bullous oedema, as such, does not permit a case to be allotted to Stage IV

IVA Spread of the growth to adjacent organs
IVB Spread to distant organs

Diagnosis

1- Clinical:

a- Symptoms:

1- Asymptomatic: in early pre and micro-invasive stages.

2- Bleeding (most common):

- Contact bleeding after intercourse, douching or examination "usually the first symptom".
- Irregular or intermittent bleeding.
- Heavy postmenopausal bleeding: in ulcerative type.

3- Pain:

- Backache due to infiltration of utero-sacral ligaments OR supra-pubic pain in pyometra.
- Ureteric colic and pain with ureteric infiltration.

4- Complications:

- Cachexia, anorexia, loss of weight, anemia…. etc.
- Hemorrhage.
- Infection and FAHM group.
- Renal failure.
- Malignant fistula.

5- Dys group:

- Dysuria and hematuria with bladder involvement and dyschazia with rectal involvement.
- Discharge "watery or blood tinged".

b- Signs:

1- General examination: cachexia**,** anemia, loss of weight, signs of renal failure.

2- Abdominal examinations: signs of metastasis, hydronephrosis, and pyometra.

3- Local examination: there may be

- Cauliflower mass.
- Malignant ulcer which is characterized by -
  - Easily bleeds on touch with necrotic surface and indurated base.
  - Friability and necrosis lead to easy probing and uterine sounding (Krobacke's sign).
- Barrel shaped cervix (endocervical type).
- Genitourinary fistula in advanced cases.
- Rectal examination: to detect infiltration of the lateral pelvic wall.

2- Investigations:

1. **Lab:** CBC, renal function, liver function, tumor marker and preoperative preparation
2. **Radiology:** bone survey, abdominal U.S, MRI, C.T, IVP.
3. **Endoscopies:**
   - *i. **Colposcopy and colposcopic guided biopsy.***
   - *ii. **Cystoscopy for bladder involvement***
   - *iii. **Proctosigmoidoscopy for rectal involvement.***
4. **Histopathology:** by taking biopsy to determine the microscopic type as mentioned before.

Treatment

1- Prevention:

- Proper screening program for CIN and use of HPV vaccine.
- Change of sexual attitude and behavior.
- Treatment and follow up of CIN lesions.

2- Active treatment: after correction of general condition the treatment may be

1. Radiotherapy: is the main line and can be used in all stages.

2. Surgery alone: limited to stages I and IIa.

3. Combined surgery and radiotherapy: as postoperative radiotherapy for patients with L.N metastasis and para-cervical spread.

4. Adjuvant therapy:

i. Neo-adjuvant chemotherapy before surgery or radiotherapy [as cisplatin].

ii. Radiation sensitizers i.e., chemo-radiation before radiotherapy using cisplatin or hydroxyurea as radiation sensitizer to shrink the tumor.

5. Palliative therapy in advanced cases:

1. Pain killers, analgesics, tonics, vitamins, blood transfusion and ICU for critical patients.
2. Palliative dose of radiotherapy, chemotherapy, surgery…etc.

Surgical management of cervical carcinoma

Indications:

1- Younger women for whom hormonal ovarian function and vaginal preservation is needed as therapeutic doses of radiation may cause.

(A)-Ovarian failure. (B)-Vaginal stenosis and adhesions and dyspareunia.

2- Pregnancy "radiotherapy cannot be used".

3- Associated adnexal disease.

4- Pelvic inflammatory disease "as radiotherapy flares up infection".

5- Good physical health.

6- Lesions ≤ 4 cm (lesions 4-6 cm better by radiation, lesions > 6 cm by combined therapy).

7- Adenocarcinoma as it is radio-resistant.

Surgical Modalities: that may be

1- Conservative surgery: strict follow up is mandatory

i-large loop excision of the transformation zone (LLETZ) OR Conization of the cervix: Indicated in: Stage Ia1 in young women wishing to preserve their fertility.

ii-Radical trachelectomy "removing the cervix only":

(Indicated in: Stage Ia2-Ib in young women wishing to preserve fertility.)

2- Radical surgery: *Wertheim's hysterectomy* = radical hysterectomy:

-It is the standard surgical procedure and indicated in stages I and IIa.

-The uterus, tubes and ovaries along with parametrial tissue and upper third of the vagina are removed together with pelvic lymph node dissection.

-One or both ovaries can be conserved in young women.

-Common complications of radical hysterectomy:

1-Blood loss. 2-Fistula formation: ureteral and Vescio-vaginal. 3-Pulmonary embolism.
4-Intestinal obstruction (adhesions). 5-Febrile morbidity (infection). 6-Lymphocele formation.
7-Bladder dysfunction (denervation). 8-Ureteric stricture.

Radiotherapy modalities:

-External irradiation [teletherapy] plus brachytherapy and the tumor lethal dose is 5000-8000 rads.

-Common radiation-associated morbidity are:

-Problems with vagina as fibrosis causing sexual difficulties.

-Bowel and bladder dysfunction. -Premature ovarian failure.

Combined RT and surgery:

– Preoperative brachytherapy followed 6–8 weeks later by surgery.

- Postoperative RT if the resection margins or pelvic lymph nodes are involved.

VULVAR INTRA-EPITHELIAL NEOPLASIA

Pathology

Definition: it is pre-invasive vulval cancer.

Incidence:

Race: more in Western countries due to widespread infection by (HPV).

Age: 40 years.

Etiology and risk factors:

1-HPV infection 2-History of other genital tract dysplasia or malignancy.
3-Smoking. 4-Immune suppression.

Naked eye:

- Lesions appear as pigmented, erythematous, black, brown, white patches or picture of infection.
- They are sharply demarcated and raised from the surrounding epithelium.
- After the application of acetic acid, they turn white **(Acetowhite lesions).**

Microscopic examination:

- Application of 3–5% acetic acid for 1-2 minutes to the vulva, perineum, and perianal areas, then colposcopy guided punch biopsies from suspicious areas, the result may be:

VIN-I: infiltration of the lower 1/3 by malignant cells.

VIN-II: infiltration of the middle 1/3 by malignant cells.

VIN-III: infiltration of the full thickness by malignant cells.

Diagnosis:

1- Clinical: patients with VIN and vulval cancer can be divided into two groups:

The first group is usually premenopausal women and with HPV-positive multifocal disease.

The second group is postmenopausal patients, HPV-negative and only unifocal disease.

- The main symptom in both is pruritus "most common in 60% of patients and asymptomatic in 40% of patients diagnosed at routine gynecologic examination.

2- Investigations:

The affected areas are tested with acetic acid and biopsied for histopathological examination. It shows show degrees mentioned before.

TREATMENT

1- Prophylaxis: by avoidance of the predisposing factors and proper treatmentof infection.

2-Active: by one of the followings *(Laser ablation, Skinning vulvectomy, OR Wide local excision in patients > 45 years as incidence of micro-invasive cancer increases).*

CARCINOMA OF THE VULVA

PATHOLOGY

Definition: it is malignant tumor arising from the vulval skin.

Incidence:

Total: uncommon neoplasms account only 4% of all gynecologic cancers.

Age: vulval carcinoma tends to have two peaks of distribution:

-In postmenopausal women: develop as solitary lesions, often in association with chronic vulval dystrophies.

-In younger women: develop usually in association as multifocal carcinoma in association with human papillomavirus (HPV) infection.

Parity and race: more common in high para and Nigro.

Etiology and Risk Factors:

1-Local lesions in the genital tract and chronic vulval irritation: e.g.

i-Human papilloma virus and herpes simplex virus infection.
ii-Vulval condylomas of syphilis and other granulomatous venereal disease.
iii-Prior genital-tract neoplasia (cervix or vagina). iv-Vulvar dystrophies and chronic pruritus vulvae.
v-Vulval carcinoma in situ. vi-Vulval Paget's disease.
vii-Exposure to radiation or use of epilators, deodorants and synthetic underwear.

2-Chronic medical illnesses: as DM, HTN, obesity, and immune-suppression.

3-Personal/lifestyle factors: cigarette smoking and multiple sexual partners.

Naked/eye:

Site: usually affecting labia majora more than labia minora but the worst affecting the clitoris.

Size: usually small.

Shape: may be nodule, cauliflower mass, or malignant ulcer that takes one of three forms:

1-Malignant squamous cell carcinoma ulcer. 2- Melanometous ulcer.
3- Basal cell carcinoma ulcer “rodent ulcer”.

Microscopic/examination:

1-*Squamous cell carcinomas*: about 90% of cases.
2-*Malignant melanoma*: the second most common cancer “5-10%”.
3-*Basal-cell carcinoma.*
4-*Adenocarcinomas:* from sweat and Bartholin's glands, ectopic breast tissue OR Paget's disease.
5-*Sarcomas* arising from connective tissue.
6-*Metastases from adjacent organs*: Rectum, Vagina, Cervix Bladder.

Spread.

1- Local direct spread is to:

- The vulval skin and supporting soft tissue.
- Vagina, urethra, anus, bladder base, rectum and pubic bones.

2- The lymphatic spread:

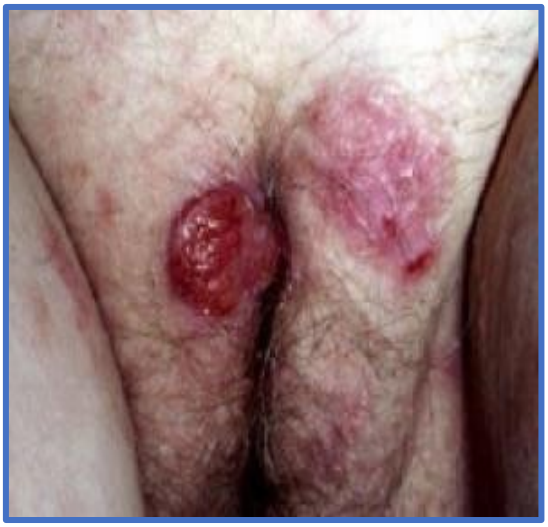
- To the ipsilateral superficial inguinal lymph nodes.
- From superficial inguinal to deep inguinal, femoral and pelvic lymph nodes.
- Cancer Clitoris drains directly to the deep femoral lymph node (gland of Cloquet) then to the pelvic lymph nodes.
- Finally to common iliac and to para-aortic nodes.
- There are cross-lymphatics between both sides.

3- Blood spread: to LBLB group but very rare in malignant melanoma.

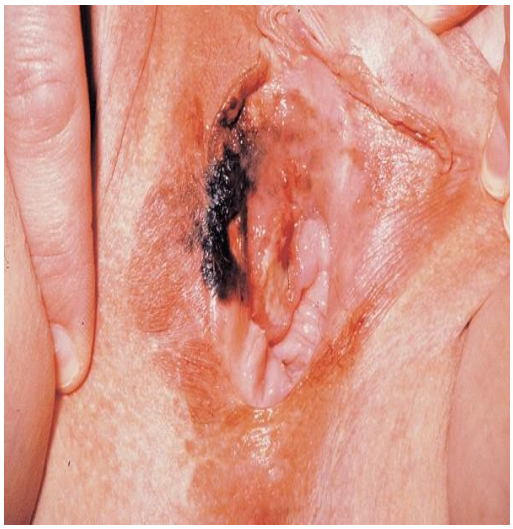
4- Special route: kissing ulcer from one side to the other was considered to be direct spread but now considered to be blood and lymphatic spread from one side to the other.

Complications:

HIND + spread local + spread general + complication of treatment "being radical and destructive surgery".



Squamous cell carcinoma -



Malignant melanoma -vulva

Staging:

Stage	Description	
I	**Tumor confined to the vulva**	
	IA	Tumor size ≤2 cm and stromal invasion ≤1 mm.
	IB	Tumor size >2 cm or stromal invasion >1 mm.
II	Tumor of any size with extension to **lower one-third** of the urethra, lower one-third of the vagina, lower one-third of the anus with negative nodes	
III	**Tumor of any size with extension to upper part of adjacent perineal structures**, or with any number of **non**-fixed, **non**-ulcerated lymph node	
	IIIA	**Tumor of any size with disease extension to** upper two-thirds of the urethra, upper two-thirds of the vagina, bladder mucosa, rectal mucosa, or regional lymph node metastases ≤5 mm
	IIIB	Regional lymph node metastases >5 mm
	IIIC	Regional lymph node metastases with extracapsular spread.
IV	**Tumor of any size fixed to bone, or fixed, ulcerated lymph node metastases, or distant metastases**	
	IVA	Disease fixed to pelvic bone, or fixed or ulcerated regional lymph node metastases
	IVB	Distant metastases

DIAGNOSIS

1- Clinical:

a- Symptoms:

-Asymptomatic and accidentally discovered.

-Chronic vulval irritation or soreness and pruritic vulvae **(the most common).**

-Bleeding may be present if surface ulceration occurred.

-Symptoms of complications.

-Dys group, mass OR ulcer at the vulva.

b- Signs:

1-General: picture of cachexia, loss of weight, pallor, anemia,etc.

2-Abdominal: signs of metastasis.

3-local: pictures of etiology can be detected, and in addition to;

- **Malignant ulcer:** with raised everted edges, necrotic floor and indurate base.
- **Kissing ulcer:** involving both sides of the labia.
- **Vulval mass:** nodule, or cauliflower mass, with evidence of local invasion.
- **Degree of extension of the tumor and staging can be assessed.**

Investigations:

1- Lab: as usual and tumor markers.

2- Radiology:

a-Chest X-ray and bone survey. b-Computed tomography (CT).

c-Magnetic resonance imaging (MRI). c-Barium enema.

3- Endoscopes:

i- Colposcopy: following application of dilute acetic acid (3-5%) to show abnormal areas and help in selecting sites for biopsy.

ii- Cystoscopy and proctoscopy.

Treatment

4- Biopsy: the microscopic picture mentioned before.

1. Prophylactic: by avoidance of the risk factors and proper treatment of VIN.

2. Active:

- Correction of general condition.
- Surgery is the mainstay for treatment.
- Radical vulvectomy was done in the past but due to its complications no place for the extensive form of radical surgery and skin excision nowadays.

Treatment of Stages I and II Vulvar Cancer

- Surgery (wide local excision).
- Surgery (radical local excision with removal of lymph nodes in the groin and upper thigh).
- Surgery (modified radical vulvectomy with removal of lymph nodes in the groin and upper thigh). Radiation therapy may be given.
- Surgery (radical local excision and removal of sentinel lymph node) followed by radiation therapy in some cases.
- Radiation therapy alone.

Treatment of Stage III Vulvar Cancer

- Surgery (modified radical vulvectomy or with removal of lymph nodes in the groin and upper thigh) with or without radiation therapy.
- Radiation therapy or chemotherapy followed by surgery.
- Radiation therapy with or without chemotherapy.
- In-lateralized lesions: modified radical hemi-vulvectomy and ipsilateral inguinal and femoral lymphadenectomy OR bilateral lymphadenectomy.

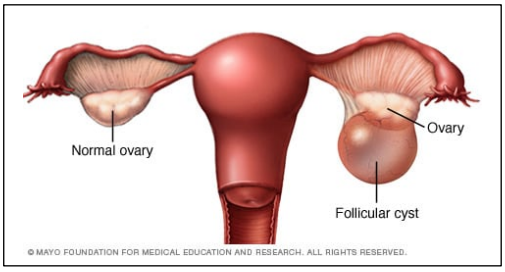
Treatment of advanced disease:

- Palliative surgery "followed by skin and muscular grafting for extensive lesions".
- Palliative radiotherapy and palliative chemotherapy for metastasis.

OVARIAN TUMORS

- According to WHO classification, ovarian tumors are classified into neoplastic and non- neoplastic swellings.

A. NON-NEOPLASTIC SWELLINGS:



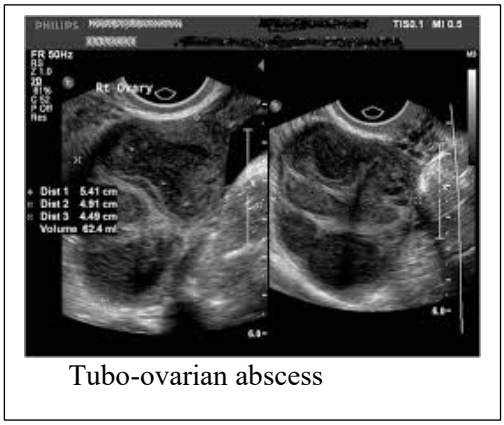
1- Functional cystic swelling:

i. -Follicular cyst.
ii. Granulosa lutein (luteinized) cysts.
iii. Theca-lute in cyst.
iv. Corpus luteum cyst.
v. Corpus albicans cyst.
vi. Corpus hemorragicum cyst.

-All of these cysts are small and do not exceed 10 cm in diameter.

-All are usually asymptomatic and accidentally discovered and resolve spontaneously.

-Usually no treatment, but COCs, cyst aspiration OR rarely cystectomy may be used.

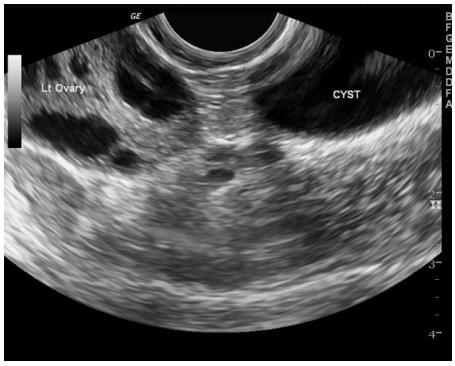


Tubo-ovarian abscess

2- Pathological cystic OR solid:

a- Cystic e.g.

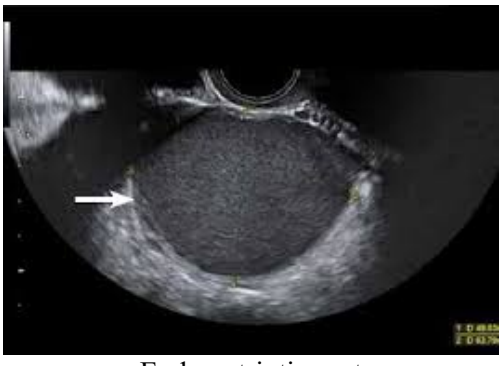
ı. Polycystic ovary syndrome "discussed before".
ıı. Endometriotic cyst "discussed before".
ııı. Tubo-ovarian cyst OR abscess

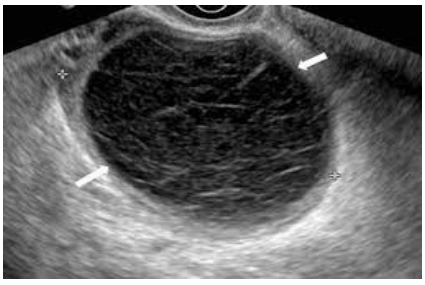


Para-ovarian cyst

b- Solid e.g.

ı. Hilar cell hyperplasia.
ıı. Edema of the ovary.
ıı. Pregnancy luteoma.


Endometriotic cyst


Corpus luteum cyst

B. NEOPLASTIC SWELLINGS: they are classified according to the cell of origin into:

1- Surface epithelial tumors:

-The surface epithelium has the potential capacity to be converted into any type of epithelium.
-It may be benign, borderline (low malignant potential) or malignant types.

1- Benign:

a-Serous: simple ,serous cystadenoma, papillary serous cystadenoma, surface papilloma "all arise from tubal- like epithelium".

***b-Mucinous*:** mucinous cystadenoma "cervical like epithelium".
***c-Endometrioid tumors*:** "endometrial like epithelium".
***d-Brenner tumor*:** transitional like epithelium = bladder like epithelium.
e-Mesonephroid tumors.
f-Mixed benign group.
g-Unclassified and Undifferentiated group.

2- Borderline (of low malignant potential): at younger age, bilateral with local metastasis.

3- Malignant types: e.g. serous cystadenocarcinoma, mucinous cystadenocarcinoma ….etc.

2- Sex cord stromal tumors:

1- Feminizing group: ganulosa - theca cell tumors and fibroma.

2- Virilizing group: androblastoma, Sertoli cell and Leydig cell tumors.

3- Combined feminizing and virilizing tumors: gyn-andro-blastoma.

4- Stromal tumors [connective tissue tumors] fibroma, lymphoma, sarcoma, lipoid cell tumors.

3- Germ cell tumors:

1- Tumors of undifferentiated germ cells: Dysgerminoma.

2- Tumors of embryonic origin:

A- Moderately differentiated tumor: e.g. embryoma and embryonal carcinoma.

B- Well differentiated tumors: e.g.

a. Tumors of embryonic cells:

i. **Teratomas:** benign cystic teratoma (dermoid cyst) OR malignant teratoma.

ii. **Yolk sac tumor = Carcinoid tumor "intestinal adenocarcinoma".**

b. Tumors of extra-embryonic cells:

1. **Endodermal sinus tumor.**
2. **Struma ovarii "as thyroid gland adenocarcinoma".**
3. **Choriocarcinoma.**

4- Mixed tumor: e.g. Gonadoblastoma.

5- Unclassified tumors.

6- Metastasis (secondary) to the ovary: That may be typical "resemble the original tumor" OR atypical "not resembling the original tumor =Kruckenberg's tumor".

SURFACE EPITHELIAL TUMORS

Common criteria for all surface epithelial tumors:

- It is the commonest ovarian tumors "about 80 % of ovarian tumors".
- Usually occurs at old age 60 years OR more.
- The commonest predisposing factor is ovulation trauma, but other factors may play a role e.g. exposure to radiation, mump oopheritis.
- There is genetic and familial tendency "Lynch syndrome II" and usually occurring 10 years earlier than non-hereditary type.
- The main route of spread in malignant type is trans-celomic spread.

1- Serous Cystadenoma: it is the commonest type.

N/E:

- Usually small to moderate dimension (20 -30 cm).
- Usually unilateral single tumor, soft in consistency.
- Usually uni-locular or comprises a small number of locules.

Types:

1. **Non papillary:** simple serous cyst or cystoma simplex.
2. **Papillary:** Containing intra-cystic papillae or occasionally papillae on the external surface.

M/E:

- Layers of cuboidal epithelium similar to that of the endosalpinx with benign criteria.
- **Psammoma bodies** are calcified granules may present giving gritty sensation of the papillae.

Serous Cystadenocarcinoma:

N/E:

- Usually bilateral, large, fixed tumor, solid OR heterogenous consistency.
- Cut section shows HIND group.

M/E: layers of tubal like epithelium with malignant criteria, **Psammoma bodies also** may present.

2- Mucinous Cystadenoma: it arises from cervical like epithelium "mucin secreting".

N/E:

- Usually single, unilateral, soft with smooth surface.
- It may reach a huge dimensions filling and distending the abdominal cavity due to mucin content which is usually thick, clear, bluish or colorless fluid.
- Usually multi-locular (honeycomb) with thin wall.
- It may perforate or rupture resulting in pseudomyxoma peritonei.

M/E:

- Lined by tall columnar cells with clear cytoplasm similar to the epithelium of the endocervix or the intestinal mucosa with benign criteria.
- It may be well differentiated, moderately differentiated, OR undifferentiated tumors.

Mucinous cystadenocarcinoma: "less common than serous type"

N/E:

- Usually bilateral, large filling the abdominal cavity, fixed tumor.
- Solid OR heterogonous consistency and cut section shows HIND group.

M/E: adenocarcinoma like that of the cervix with malignant criteria.

3- Endometrioid Cystadenoma:

- Arises from endometrial like epithelium.
- When benign so small, single and unilateral tumor.
- If malignant has the same criteria mentioned before.

4- Brenner Tumor: "usually benign and bladder like epithelium":

N/E:

- Usually benign: single, unilateral **BUT solid.**
- Rarely produce estrogen, resulting in postmenopausal bleeding.

M/E:

- Islands of epithelioid cells and hyperplastic fibrous tissue.
- The nuclei show a "coffee-bean appearance" caused by longitudinal grooving of the nuclei.
- If malignant it appears as transitional cell carcinoma.

5- Clear Cell (Mesonephroid) Tumor: "mesonephric like epithelium".

N/E:

- When benign "the same criteria" and when malignant “the same criteria" as mentioned.

M/E:

If benign: layers of benign mesonephric like epithelium and may arrange in tubule like forms.

If malignant: the same and malignant criteria. Focal areas of endometriosis and endometrioid carcinoma sometimes occur.

Important notes:

1- Pseudomyxoma peritonei:

- It is a rare condition associated with benign OR malignant mucinous ovarian tumor.
- The peritoneal cavity is filled with mucinous material and this causes extensive adhesions.
- The condition commonly recurs after laparotomy and evacuation with excision of the ovarian tumor "may be due to chemical peritonitis".
- It may need radiotherapy or intra-peritoneal instillation of radio-active isotope.

2- In mesonephric tumors

Both benign and malignant mesonephric types may secret parathyroid hormone → hypercalcemia and hypercalciuria with great liability for renal stones.

SEX-CORD STROMAL TUMORS

Common criteria of sex cord-stromal tumors:

- Usually occurs in middle age group but very rarely in premenarchal age.
- Usually tumors are functioning as they secret hormones.
- Usually have commonly yellow color due to hormonal content.
- According to the type of hormone they may be:

1- **Feminizing group:** that include;

1. **Granuolsa cell tumor.**
2. **Theca cell tumor = thecoma** "if the secreted androgen is completely turned to estrogen".
3. **Fibroma.**

- They Secrets estrogen and causes clinical presentation of hyperestrognic states.
- Therefore, these tumors may present clinically by: precocious puberty, abnormal uterine bleeding and estrogen dependent tumor OR as postmenopausal bleeding.

N/E:

1. ***Granulose-theca cell tumor:*** **if benign, its criteria and if malignant, criteria of malignancy.**
2. ***Fibroma:***

- Small to moderate in size.
- It is rounded or lobulated.
- Despite it is benign but it is firm solid tumor.
- The cut surface is fibrous white-yellow or grayish in color and gives whorly appearance.

M/E:

1. ***In granulosa cell tumor:***

- Granulosa cell appear as rounded or ovoid cells with scanty cytoplasm.
- The granulosa cells arrange themselves in small clusters or rosettes around a central cavity, so they resemblance to primordial follicles **(Call-Exner bodies).**

2. ***Fibroma:***

- It is formed of fusiform fibroblast, with fibrous matrix, commonly benign **BUT solid** tumor.
- All types of degeneration in uterine fibroid can occur **EXCEPT** red degeneration.

Meigs' Syndrome:

- Comprises ovarian fibroma, ascites and right hydrothorax.
- The cause is unclear but may be due to lymphatic irritation by the tumor.
- Hydrothorax results from rich lymphatic connections across the diaphragm.
- This spontaneously disappears after removal of the ovarian fibroma.
- Pseudo Meigs' syndrome: with tumors other than fibroma (ovarian or uterine) e.g. Brenner tumor or Krukenberg secondaries or uterine fibroma, with ascites and right hydrothorax.

2- **Virilizing group:** that include-

1. Theca cell tumor if there is an excessive amount of androgen secretion.
2. Sertoli-Lydige cell tumor.

- They secrets androgen and clinical presentation of hyper-androgenism e.g. defeminization OR virilization syndromes.

GERM CELL TUMORS

Common criteria of germ cell tumors:

- Commonly occurring in young age below 20 years **EXCEPT** dermoid cyst occurs at 20-30 years.
- The most important predisposing cause is Dysgenetic gonads e.g.:
  - Pure gonadal dysgenesis (46 XY, bilateral streak gonads).
  - Mixed gonadal dysgenesis (45 X0/46 XY, unilateral streak gonad, contra-lateral testis).
  - Androgen insensitivity syndrome (46 XY = testicular feminization).
- They are usually functioning tumors as they secrets hormones.
- The main route of spread is lymphatic spread.
- Common types are:

1- Dysgerminoma: "undifferentiated germ cell tumor"

- It is the commonest germ cell tumor "about 80 % of germ cell tumor before 20 years".
- Usually locally malignant tumor.

N/E:

- Usually large in size, has capsule and is slightly bosselated.
- Consistency firm to solid and the cut surface shows HIND and is gray-brown in color.

M/E:

- It shows the basic germ cell, and the mitotic figures are usually numerous.
- The cells are arranged as lobules and nests separated by fibrous septa.
- The cells usually secret **α-feto protein, placental alkaline phosphatase, lactate dehydrogenase (LDH), and □-1 antitrypsin.**

2- Dermoid Cyst: (benign cystic teratoma)

- It occur at 20-30 years, therefore it is the commonest tumor that complicates pregnancy.

N/E:

- It is usually benign tumor, small to moderate size, single, unilocular but may be bilateral.
- It has a long pedicle with increased liability to torsion and free mobility in the abdomen **(mouse of the abdomen)** so commonly felt anterior to the uterus **"in the utero-vesical pouch".**
- It has thick and opaque yellow capsule.

On cut section:

- There is greasy sebaceous fluid secreted from the skin.
- An embryonic nodule projects from the wall.
- To this nodule a tuft of hair is attached.
- From this embryonic nodule" all tumor tissues arise "as skin, skin appendages, teeth, bone, cartilage, thyroid tissue, gastrointestinal epithelium and neural tissue".

M/E:

- Dermoid cyst is commonly lined by stratified squamous epithelium and contains other normal body tissues that are commonly benign.
- Very rarely malignant change occurs resulting in Squamous cell carcinoma.
- Other malignant changes have reported (leiomysarcomas & mixed mesodermal tumors).

3- Immature Teratomas:

- It is the second most common germ-cell malignancy, commonly occurs before 20 years of age.

M/E:

- Immature teratomas are classified according to a grading system **(grades 1-3)** that is based on the degree of differentiation and the quantity of immature **neural tissue.**

4- Endodermal sinus tumor: "yolk sac carcinomas".

N/E:

- It is **unilateral in 100 %** of cases despite it is malignant.
- It is soft grayish-brown in color, with cystic areas and the capsule is usually intact in most cases.

M/E:

- The characteristic feature is intestinal like epithelium with villi **"Schiller-Duval body".**
- It secretes □-1-antitrypsin and □-fetoprotein.

5- Embryoma and Polyembryoma:

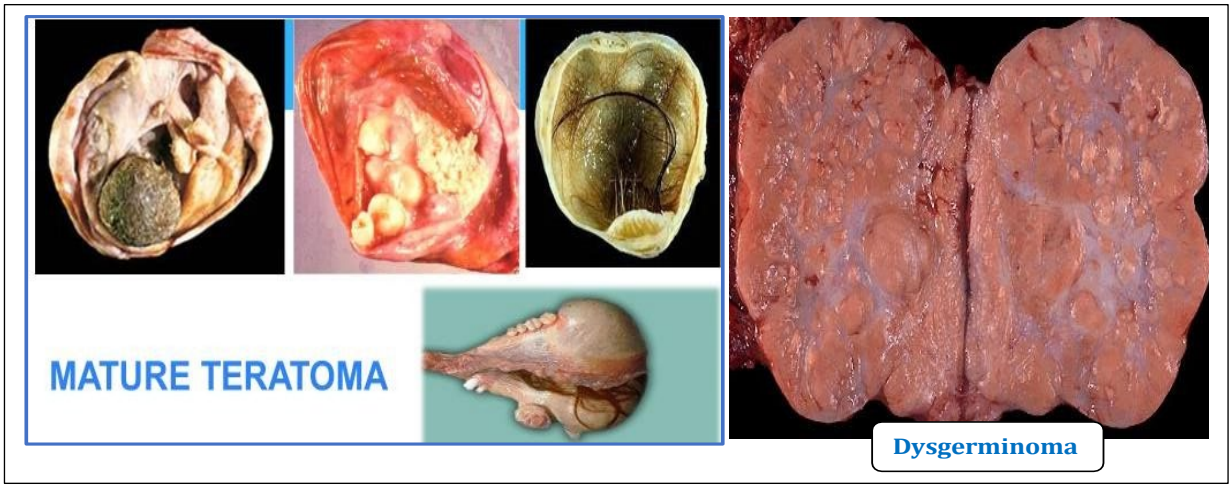
- Extremely rare ovarian tumor which is composed of embryoid bodies.
- This tumor structures of early embryonic differentiation (i.e. endoderm, mesoderm and ectoderm).
- These tumor usually secret □-fetoprotein and hCG titers.

Embryonal Carcinoma:

- It shows criteria of malignancy "by N/E and ME".
- It may secrete estrogens and the patient exhibits ymptoms and signs of precocious pseudopuberty or irregular bleeding.
- Also frequently secrete □-fetoprotein and hCG.

6- Choriocarcinoma:

- Show criteria of malignancy as malignant sheets of trophoblastic tissues "the same appearance as gestational choriocarcinoma metastatic to the ovaries".
- It secrets much hCG that ++ the ovaries □ precocious puberty in about 50 % of patients.



METASTASIS TO THE OVARY

- About 5- 10 % of ovarian tumors are metastatic coming from other organ malignancies e.g. from genital tract, breast, or the gastrointestinal tract.
- The ovary is considered as a common site for metastasis due to:
  - Rich blood and lymphatic supply.
  - Present as intra-peritoneal structure not covered by peritoneum.
- Mode of spread to the ovary may be ;
  1. Direct extension
  2. Hematogenous spread.
  3. Lymphatic spread.
  4. Transcelomic dissemination.
  5. Surface implantation.
- Sites of primary tumors may be :
  - **Local genital tract tumors:** all genital tract malignancies.
  - **Extra-genital tract tumors:** Cancer breast, cancer stomach, colon, and less commonly, the small intestine, Lymphoma and Leukemia.
- Involvement is usually bilateral.
- On histological examination: the tumor may be ;
  - **Typical** "resemble the primary tumor".
  - **Atypical** "not resembling the original tumor= Krukenberg Tumor ". Clusters of mucin filled cells arise in the ovarian stroma and have the characteristic signet ring appearance.

SPREAD OF MALIGNANT OVARIAN TUMORS

1- Direct spread: to the surrounding genital and extra-genital organs.

2- Trans-celomic spread:

- The most common and earliest mode of spread in epithelial cancer.
- It rarely penetrates intestinal lumen but progressively may agglutinate loops of bowel together leading to intestinal obstruction.

3- Lymphatic spread:

- It is the main route of spread in germ cell tumors.
- To pelvic and para-aortic lymph nodes.
- Also to the **supraclavicular** lymph nodes through the lymphatic channels of the diaphragm.

4- Hematogenous:

- Hematogenous spread at the time of diagnosis is uncommon.
- It involves lung, liver, bone, brain.

STAGING OF OVARIAN CANCER:

- For proper staging exploratory laparotomy should be done. **FIGO staging 2014 include:**

Stage IA: Tumor limited to 1 ovary, capsule intact, no tumor on surface, negative washings.

Stage IB: Tumor involves both ovaries otherwise like IA.

Stage IC: Tumor limited to 1 or both ovaries

- **Stage IC1:** Surgical spill.
- **Stage IC2:** Capsule rupture before surgery or tumor on ovarian surface.
- **Stage IC3:** Malignant cells in the ascites or peritoneal washings.

Stage II A: Extension and/or implant on uterus and/or Fallopian tubes

Stage II B: Extension to other pelvic intraperitoneal tissues

Stage III A1: Positive retroperitoneal lymph nodes only

- **Stage IIIA1-i:** Metastasis ≤ 10 mm.
- **Stage IIIA1-ii:** Metastasis > 10 mm.

Stage III A2: Microscopic, extra-pelvic peritoneal involvement ± positive retroperitoneal l. nodes.

Stage III B: Macroscopic, extra-pelvic, peritoneal metastasis ≤ 2 cm ± positive retroperitoneal lymph nodes. Includes extension to capsule of liver/spleen.

Stage III C: Macroscopic, extra-pelvic, peritoneal metastasis > 2 cm ± positive, retroperitoneal lymph nodes. Includes extension to capsule of liver/spleen also.

Stage IVA: Pleural effusion with positive cytology.

Stage IVB: Hepatic and/or splenic parenchymal metastasis, metastasis to extra-abdominal organs (including inguinal lymph nodes and lymph nodes outside of the abdominal cavity).

COMPLICATIONS OF OVARIAN TUMORS

1. Torsion:

- Usually occurs in small to moderate sized tumors with a long pedicle (as dermoid cyst).
- It is precipitated by straining during defecation, labor or sexual intercourse.
- The tube is usually involved with torsion of ovarian tumors.

Clinically:

- There is acute pain associated with feeling a tender mass in the lower abdomen.
- Presence of fluid in the peritoneal cavity.

Treatment: immediate laparotomy, then:

- **If the ovary looks gangrenous:** Salpingoophorectomy is done.
- **If the ovary looks healthy:** Cystectomy and conserving viable ovarian tissue.

2 Rupture:

- Predisposing factors include torsion or hemorrhage or external trauma.
- According the content of the tumor there may be:
  - Septic peritonitis if contain pus.
  - Chemical peritonitis, with sebaceous secretion in dermoid cyst.
  - Ascetic like fluid if serous tumors.
  - Psudomyxoma peritonii with mucinous tumors.

3. Hemorrhage: this is predisposed to by:

- Torsion of the tumor or trauma to the abdomen.
- Infection, degeneration or malignant nature.

Clinically: the condition results in acute, intra peritoneal hemorrhage, collapse, and peritonitis.
Treatment: immediate laparotomy is needed.

4. Infection:

- Infection may result from torsion, hemorrhage or tapping ovarian cyst.
- It is common during puerperium.

Clinically: "FAHM" group, severe pain and systemic toxemia due to pus.

5. Impaction "incarceration":

- This is predisposed to by long pedicle and small size this results in:
  - Pelvic pain, and urinary retention.
  - Rectal pressure and constipation
  - Obstructed labor if the patient gets pregnant.

6. Intestinal Obstruction:

- This rarely occurs with benign tumor **"due to adhesion"** but common with malignant tumors "due to adhesion, infiltration, compression or amalgamation of intestinal loops".
- Intestinal obstruction is **"the commonest cause of death"** from ovarian cancer.

7. Malignant change: may occur in some benign ovarian tumors.

8. Metastasis of malignant one: leading to cachexia, loss of weight, palor, anemia …etc.

9. Meig's and psudo Meig's syndrome: mentioned with fibroma.

10. Pregnancy complications: abortion, preterm labor, obstructed labor.

11. Pressure symptoms: that may cause

- Dysuria, urine retention, dyschazia, dyspepsia, dyspnea if huge reaching the diaphragm.
- Intestinal obstruction.

12. Degeneration:

- Commonly occurring in fibroma which may be asymptomatic or result in a dull aching pain.

DIAGNOSIS OF OVARIAN TUMOR

1- Clinical:

a- Symptoms:

1. **Asymptomatic** and accidentally discovered
   - Ovarian tumor is commonly called **"silent killer"** as the patient is late discovered and commonly present by atypical symptoms.
2. **Abdominal distention due to:**
   - Tumor mass.
   - The presence of ascites.
   - Omental metastases, bowel metastases, liver metastasis.
   - Bloating, indigestion or constipation.
3. **Pain:** diffuse vague abdominal pain, pelvic pain or severe with torsion, rupture, hge….etc.
4. **Bleeding:** that may present as:
   - Pre-menarchal bleeding and precocious puberty.
   - Pre-menopausal menorrhagia, OR metrorrhagia.
   - Post-menopausal bleeding.
   - Bleeding is usually due to:
   - Direct extension to the uterus.
   - Feminizing ovarian tumor and estrogen secretion.
   - Pelvic congestion by large tumors.
   - Complicated ovarian tumors as torsion.
   - Extension to the liver with disturbed liver functions.
5. **Symptoms of complications e.g.:**
   - Urinary frequency with pressure on the bladder.
   - Constipation with pressure on the rectum.
   - Cachexia, loss of weight pallor, anemia…..etc.
   - Effect of hormones e.g.
     1. **Estrogen** → estrogen related condition.
     2. **Androgen** -→ de-feminization then, virilization syndrome.
     3. **HCG and thyroxin** ---□ hypertension and hyperthyroidism.
     4. **Serotonin** → circulatory disturbances and syncopal attacks.
6. **Dys group.**

b- Signs:

1. **General:**

-There may be nothing characteristic in benign tumor "OR effects of hormones".

- OR malignant cachexia, loss of weight pallor, anemia, effect of hormones in malignancy.
- There may be enlarged supraclavicular L. nodes in malignant conditions.

2. Abdominal:

- Diffuse abdominal enlargement.
- In Benign cases -→ benign criteria.
- In malignant cases -→ malignant criteria and in addition:
  1. Sister Josef nodules.
  2. Dilated veins.
  3. Abnormal pigmentation on the abdominal wall.
  4. Presence of ascites.
  5. Hepatomegally, omental cakes, intestinal obstruction….etc.

3. Local: there may be normal tissues OR Benign OR malignant criteria.

2- Investigations:

1. **Laboratory:** the basic investigations and tumor markers "remember all".
2. **Radiology:** bone survey, U.S, MRI, C.T.
3. **Endoscopes:** laparoscopy, cystoscopy and proctosigmoidoscopy in malignant cases.
4. **Histopathology:** via laparoscope OR laparotomy.

Criteria suspecting malignant ovarian tumors

A. Symptoms:

1. Age:
   - The older the patient, the higher the chance of malignant tumor.
   - More than 50 % of ovarian tumors above the age of 50 years are malignant.
2. Rate of Growth: rapidly growing tumors are usually malignant.
3. Dull aching continuous pain.
4. Back Pain: it suggests advanced malignant disease involving the roots of sacral plexus.
5. Malignant cachexia and symptoms of metastasis.
6. Hormonal effects: functioning tumors are usually malignant.

B. Signs:

1. Bilaterality: bilateral tumor is suggestive of malignancy BUT dermoid and mucinous tumor may be bilateral.
2. Solidity: solid tumor or heterogeneous consistency suggests a malignant nature.
   - Some cases of benign ovarian tumors may be solid e.g. ovarian fibroma and Brenner's tumor.
3. Fixation.
4. Presence of ascites.
5. Unilateral edema OR varicosities: may occur in the lower limbs and vulva.
6. Presence of metastases as evidenced by
   - Presence of left supra-clavicular lymph nodes (Virchow nodes).
   - Omental mass
   - Nodules in the Douglas pouch
   - Masses OR nodules in and around the umbilicus.
   - Palpable Para- aortic lymph nodes.
   - Effect of blood spread to distant sites e.g. lung, liver, bone, or brain "rarest site".

C- On laparotomy:

-Weak friable tissues. -Hemorrhagic ascites OR +ve peritoneal wash.

- Bilaterality, solidity, fixity of the tumor….etc.
- Presence of exophytic growth on the capsule or its infiltration by the tumor.

Treatment of Apparently Benign Ovarian Tumors

1- Prophylactic:

- By avoidance of predisposing factors e.g. avoid unnecessary induction of ovulation, removal of dysgenetic gonads at the age of puberty …..etc.

2- Active: the treatment may be conservative surgery OR radical surgery.

A- Conservative surgery:

Indicated in:

- Young patient "aiming to preserve fertility".
- Small cystic not complicated tumors.
- Suspicious or malignant tumor confined to one ovary when further fertility is required.

Lines are:

- **Cyst aspiration** OR tapping in order to reduce its size.
- **Ovarian Cystectomy**.
- **Unilateral salpingo-ophorectomy.**
- **Laparoscopic surgery:** cyst aspiration, de-roofing and removal.

B- Radical surgery:

Indicated in:

- Benign tumors old age above 45 years "not needing the ovaries for fear of malignancy".
- Recurrence after removal OR bilateral tumors.

Lines are: total hysterectomy with bilateral salpingo-ophorectomy

Treatment of Malignant Ovarian Tumors

1- Prophylactic:

- Avoidance of predisposing factors and proper treatment of benign tumors.
- Proper screening of cancer ovary and early detection of high risk by getting good detailed history.
- Annual screening of this group by TAS, TVS, Doppler study OR tumor markers [remember all].

2- Active: The main line is surgery.

i. In borderline ovarian tumors: surgical resection of the primary tumor OR TAH and BSO.

ii. In malignant tumors:

Stage	Treatment
Stage Ia Fertility desired	Unilateral oophorectomy or salpingoopherectomy.
Stage Ia Fertility NOT desired	TAH & BSO.
Stage Ib Fertility desired	Unilateral oophorectomy + postoperative single agent chemotherapy
Stage Ib Fertility NOT desired	TAH & BSO + postoperative single agent chemotherapy [cisplatin, pacletaxol….].
Stages Ib G2&3, Ic, IIa	TAH & BSO + postoperative combination chemotherapy ± radiotherapy
Stages IIb, IIc	Radical oophorectomy + postoperative combination chemotherapy ± radiotherapy.
Stages III, IV	Debulking + postoperative combination chemotherapy ± radiotherapy [intraperitoneal or whole abdomen irradiation] ± immunotherapy.

GYNECOLOGICAL OPERATIONS

Dilatation of the Cervix

Types of dilators:

1. **Hegar** dilator has a uniform thickness throughout its whole length "numbered on both sides".
2. **Fenton** dilator tapers gradually towards the tip "numbered on one side only".

The number indicates the diameter in mm e.g. Hegar number 4 = 4 mm in diameter.

Indications:

1. Dilatation alone:
   1. Spasmodic dysmenorrhea "up to no 14"
   2. Cervical stenosis OR drainage of pyometra or hematometra.
2. Dilatation preliminary to another procedure:

Cervical procedures	Uterine procedures
- Amputation (including Fothergill operation). - Trachelorrhaphy. - Cautery in Nullipara.	- Curettage & evacuation or MVA. - Polypectomy. - Introduction of radium. - Introduction or removal of IUD.

Dilatation of the cervix:

- Usually start by dilator number 3 or 4, held like a pencil and pushed gently to the cervical canal.
- The 1st resistance is felt at the internal os & then it is pushed through it to the uterus.
- The dilator should be left for 1/2 minute to allow the circular fibers of the cervix to relax.
- It is then removed and the next larger size is introduced until the required dilatation:
  - *Number 8-10 for endometrial curettage.*
  - *Number 12 before amputation of the cervix.*
  - *Number 14 for treatment of spasmodic dysmenorrhea.*

Curettage

Types of curettes:

1. **Loop curette** "sharp or blunt".
2. **Biopsy curette** e.g. Novak or Sharman curettes:
3. **Fundal curette:** it has a tapering end to curette the fundus and angles.
4. **Flushing curette:** to curette and to wash the decidua after evacuation.

Indications:

1. Diagnostic (endometrial biopsy):

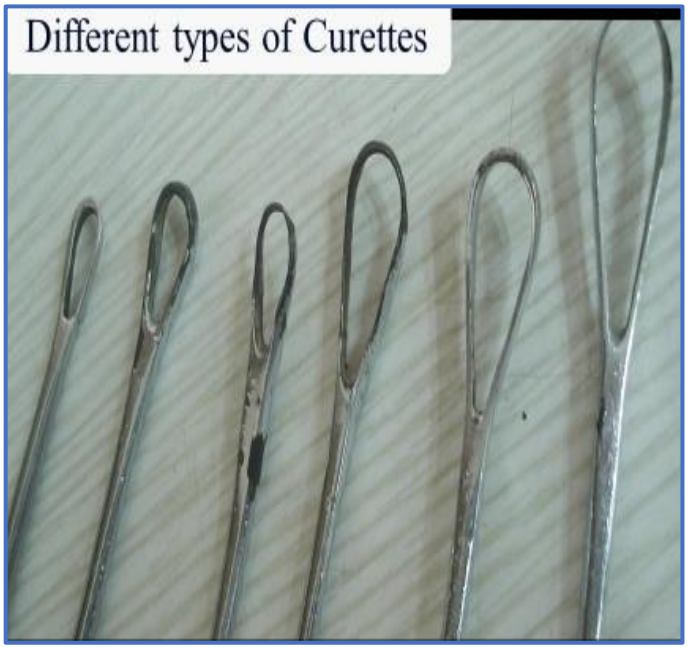
a. Detection of ovulation by dated premenstrual endometrial biopsy.
b. Detection of luteal phase defect by a dated premenstrual endometrial biopsy.
c. Detection of TB endometritis
d. Detection of cancer of the endometrium or endo-cervix by fractional curettage.
e. Differentiation of DUB & organic causes of uterine bleeding.

2. Therapeutic:

a. Postpartum & post-abortive hemorrhage to remove retained products.
b. Endometrial & cervical polyps.
c. Dysfunctional uterine bleeding (DUB).
d. after evacuation to remove the decidua.
e. membranous dysmenorrheal.
f. Before Fothergill's operation to remove congested endometrium.
g. Removal of missed IUD.
h. Cutting of adhesions in Asherman's syndrome.

Complications of dilatation & curettage

1. **Shock:** due to dilatation without anesthesia (vasovagal attack) or excessive bleeding.
2. **Injury and perforation** of the uterus or cervical lacerations --□ hemorrhage.
3. **Infection as** cervicitis, endometritis, PID....
4. **Complications of anesthesia.**
5. **Remote complications:** incompetent isthmus OR Asherman syndrome.



Hysterectomy

Indications:

a. Obstetric:

1. Irreparable rupture uterus especially if the patient completed her family.
2. Couvelaire's uterus if atonic & failed to contract with uterine massage & ecobolics.
3. Uncontrolled post-partum hemorrhage.
4. Placenta accreta if failed conservative treatment.
5. Vesicular mole "in invasive or recurrent mole or suspicious of choriocarcinoma if the patient is above 40 years".

b. Gynecologic:

1. Benign neoplasm: e.g. fibroid, above 40 years with large number of myomas , complicated or if malignancy is suspected.
2. Benign ovarian tumors if the patient is above 45 years.
3. Malignant neoplasms of the cervix, body and ovary.
4. Dysfunctional uterine bleeding in patients above 40 years with adequate number of children, if hormonal treatment & curettage failed to control bleeding.
5. Second and third degree uterine prolapse in menopausal patients.
6. Endometriosis and adenomyosis in patients above 40 years.
7. Genital tuberculosis if medical therapy fails in cases of large masses

Types = routes:

a. Abdominal hysterectomy:

1. Total hysterectomy: the body and cervix are removed.
2. Subtotal hysterectomy: the body is removed and the cervix is left.
3. Pan-hysterectomy: total hysterectomy & bilateral salpingo-oophorectomy
4. Radical hysterectomy e.g. Wertheim's operation.
5. Ultra-radical hysterectomy "Pelvic excentration".

b. Vaginal hysterectomy:

Indications:

1. Uterine prolapse (second or third degree in postmenopausal patients).
2. Chronic inversion.
3. Some cases of fibroids.
4. Schauta operation (in cancer cervix with prolapse).
5. Some cases of dysfunctional uterine bleeding.

c. Laparoscopic assisted vaginal hysterectomy: by using laparoscope to divide the uterus then pushing it vaginally.

Hysteroscopy

Definition: visualization of the uterine cavity by an endoscope introduced through the cervix.

Indications:

1. Infertility or recurrent abortions:

a. Diagnostic	*b. Therapeutic*
- Congenital uterine anomalies as septate uterus.	- Resection of a septum
- Asherman syndrome	- Lysis of intrauterine adhesion
- Sub-mucous myoma	- Hysteroscopic myomectomy

2. **AUB (HMB/ IMB):**
   - **a.** Diagnostic e.g. endometrial hyperplasia.
   - **b.** Therapeutic: endometrial ablation for some cases of DUB.
3. Uterine masses:
   - **a.** Diagnostic e.g. tumor or polyp & taking biopsy.
   - **b.** Therapeutic: Removal of a polyp or myoma.
4. Removal of a missed IUD.
5. Sterilization: hysteroscopic occlusion of the tubal ostium by cautery or chemicals.

Contraindications:

1. Pregnancy.
2. Infections e.g. acute cervicitis
3. Moderate to severe bleeding.
4. Extensive stenosis in the cx as fibroid blocking it.
5. Endo-cervical carcinoma blocking the lumen.

Laparoscopy

Definition: visualization of the peritoneal cavity by endoscope introduced through the abdomen.

Indications:

1. **Infertility:** diagnosis and treatment of tubal, uterine, ovarian factors, endometriosis and pelvic adhesions.
2. **Amenorrhea:** diagnosis and treatment of some cases as
   - **a.** PCO (Stein-Leventhal syndrome).
   - **b.** To detect streak gonads in Turner syndrome.
   - **c.** uterine anomalies as aplasia, hypoplasia…
3. **Pelvic masses:** for diagnosis and treatment of sub-serous myoma & ovarian rumors, endometriosis.
4. **Pelvic pain:**
   - **a.** Acute pain: to differentiate ectopic pregnancy from acute salpingitis.
   - **b.** Unexplained chronic pelvic pain e.g. endometriosis or pelvic adhesions.
5. **Extraction** of a missed extra-uterine **IUD.**
6. **Tubal sterilization** by application of rings or cauterization of the tubal ostia.

Catheterization of the bladder

Indications in gynecology:

1. Before local examination if the patient cannot evacuate her bladder.
2. Before any vaginal or abdominal operation, also post-operative till the patient can evacuate.
3. Collection of urine for bacteriological investigation.
4. To differentiate full bladder from pelvi-abdominal mass.
5. Diagnosis of genitourinary fistula, stone bladder.
6. Ascending cystogram and methylene blue test in fistula.

Indications in obstetric:

1. during labor if the patient cannot evacuate the bladder.
2. before instrumental delivery OR C.S.

Types of catheters:

1. **Neleton** non-self-retaining plastic catheter.
2. **Foley's** self-retaining rubber catheter.
3. **Metal** catheters "straight or S-shaped".

Hysterosalpingography & Ultrasound

Indications of HSG:

a. Detection of uterine abnormalities:

1. Congenital anomalies of the uterus e.g. septum, bi-cornuate uterus, hypoplasia.
2. Intrauterine synechiae.
3. Incompetent isthmus.
4. Sub-mucous fibroid or uterine polyps.
5. Localization of retained intrauterine device i.e. missed IUD.

b. Infertility: diagnostic & therapeutic "mentioned in infertility".

c. Diagnosis of genital T.B.

d. Before and after tubal surgery e.g. tuboplasty.

e. To confirm the relation of a pelvic mass to the uterus.

Indications of ultrasound: visualize the following:

1. The uterus: to detect --

    1. Uterine anomalies e.g. hypoplasia, aplasia or bicornuate uterus.
    2. Endometrial thickness to detect endometrial hyperplasia, cancer or polypi.
    3. An IUD is intra-uterine.
    4. Uterine fibroids or adenomyosis.

2. The ovaries: to detect --

    1. Polycystic ovaries.
    2. Ovarian masses.
    3. Follicular growth.
    4. Oocyte retrieval for IVF, GIFT or ICSI.
    5. Aspiration of ovarian cyst under ultrasound guidance.

3. The tubes: ectopic pregnancy, hydrosalpinx or tubo-ovarian abscess.
4. Douglas pouch: to detect hematoma, abscess or peritoneal fluid.