PRETERM LABOUR

Prof. maysoon sharief

Objectives:-

1- Definition

2- Incidence

3- Classification

4- Etiology

5- Clinical features of preterm labour

6- Management of high –risk asymptomatic women

7- Management of symptomatic women

Definition

In pregnancy, term refers to the gestational period from 37+0 to 41+6 weeks. Preterm births occur between 24+0 and 36 +6 weeks.

Births earlier than this are referred to as miscarriages; occasional survivors are seen after delivery at 23 weeks, which has become the "grey zone" for viability.

Early births occur either (spontaneous deliveries) or (induced deliveries in 25% of preterm birth).

Incidence

The incidence in the developed world is 7 -12 %. The rate of preterm birth prior to 32 weeks has remained relatively stable at 1 -2 %.

30% of preterm birth is associated with preterm pre-labour rupture of membranes.

Classification:-

1- Mildly preterm births at 32+0 to 36 +6 weeks (incidence 6.1 %).

2- Very preterm births at 28+0 to 31+6 weeks (incidence o.9 %).

3- Extremely preterm births at 24+0 to 27+6 weeks (incidence 0.4 %).

Etiology:-

1- Infection

Subclinical intrauterine infection of the choriodecidual space and amniotic fluid by ascending bacterial infection lead to

increase prostaglandin release and trigger contractions or lead to rupture membranes.

2- Over-distension

The commonest cause of uterine over distension is multiple gestations, polyhydramnios.

3- Vascular

Ante partum hemorrhage like placental abruption.

4- Abnormal uterine cavity

Congenital malformation may be less able to accommodate the developing pregnancy, uterine fibroids.

5- Cervical weakness (incompetence)

Congenital or pervious surgical defect leads to cervical shorten and open prematurely.

6- Intercurrent illness:-

Serious maternal infective illnesses such as pyelonephritis, appendicitis and pneumonia. In these conditions preterm labour is presumed to be due either to direct blood-borne spread of infection to the uterine cavity or indirectly to chemical triggers, such as end toxins or cytokines.

7- Idiopathic :-

Risk factors for preterm labour:-

- Maternal weight (both underweight women < 20 BMI and overweight are at risk)
- Maternal age: mother with age < 16 and more than 40.
- Parity: primgravidae and women with parity more than 5.
- Poor socioeconomic status.
- Smoking.
- Women with previous history of preterm delivery increases the risk fourfold.

Clinical features:-

History

Uterine contractions (intensity and frequency)
Vaginal discharge or bleeding
Pelvic pressure and or backache are some times reported

EXAMINATION:-

General : assess overall health, maternal pulse, blood pressure and temperature**.**

Abdominal:

May reveal the presence of uterine contraction, tenderness which suggests abruption or chorioamnionitis**.**

Pelvic: A careful speculum examination may yield valuable information; pooling of amniotic fluid, blood and/ or abnormal discharges may be observed. Digital exams should be limited, as they are known to stimulate prostaglandin production and may introduce organisms into the cervical canal.

Investigations:

Fetal fibronectin is a glue like protein binding the choriodecidual membranes. It is rarely present in vaginal secretions between 23 – 34 weeks and possible detection in the cervicovaginal secretions in patient with symptomatic labour.

Cervical length : Transvaginal ultrasound measurement , normal cervix measures 35mm in length and serial U/S measurement is needed started at 18 -22 wks.

Management of high –risk asymptomatic women:

1- Early dating scan which ensures the assessment of gestational age.

2- Treatment of urinary tract infection, asymptomatic bacteriuria.

3- Screening and treatment of antenatal bacterial vaginosis .

4- Detection of fetal fibronectin test at age of 24 weeks increase the risk of preterm labour to the half.

5- Progesterone therapy has been recognized in support of the pregnancy.

6- Lifestyle modification.

7- Elective cervical cerclage may be advised in women with previous history of preterm labour.

Management of symptomatic women

1- Communication and support: This include communication with the mother and her family ensures that they are fully understanding of the risk and management plan , communication with the neonatal unit staff ensures adequate resources are available include neonatal care unit.

2- Maternal steroid : Single course of maternal steroids (two injection 8 mg/12hs apart) given between 28- 34 wks of gestation and received within 7 days of delivery result in markedly improved neonatal outcomes by reduction of (RDS).

3- tocolytics: Suppression of uterine contractions by tocolytics is delaying of preterm labour allows time for steroid administration and in utero transfer that improve outcome.

Types of tocolytics:-

- **Sympathomimetics**

With introduction of beta- sympathomimetics into obstetrics practice in the 1970s associated with great efficacy in inhibiting preterm contractions. More modern studies have shown that ritordine will delay preterm delivery in minority of patients for 24-48hs , beside are associated with significant life – threatening maternal side effects (particularly if given in combination with corticosteroids) that include fluid overload, pulmonary oedema , myocardial ischaemia, hyperglycaemia and hypocalaemia.

- **Non – steroidal anti- inflammatory drugs**

The NSAIDS most widely studied as an acute tocolytic is indometacin , at present there is no evidence that any type of NSAIDS has any advantage as a first –line tocolytic beside it has high major effect on effect on fetal renal function ,fetal cardiovascular system in particularly on ductus arteriosus.

- **Magnesium sulphate**

Beside its use for management of per-eclampsia can be used in preterm labour with equal efficacy findings with sympathomimetics.

Oxytocin antagonists

Atosiban which is oxytocin antagonists was no better than placebo in prevention of preterm delivery.

Calicum channel blockers

The central role of calcium in the biochemistry of myometrial contractions led to the exploration of the use of ca channel blockers specifically nifedipine as a tocolytic drugs.

Antibiotics :

Meta analysis of the use of antibiotics in symptomatic preterm labour show that do not delay delivery or improve any aspect of neonatal morbidity or mortality. Its benefit is reduction of maternal infection if associated with rupture membrane.

Neonatal outcome after preterm delivery

1- Respiratory distress syndrome : Introduction of surfactant therapy improved the neonatal respiration.

2- Hypocalcaemia

3- Hypoglycemia

4- Hypomagnesaemia

5- Hypothermia

6- Failure of thrive

Conduct of preterm delivery

Rates of neonatal morbidity and mortality are higher in babies transferred ex utero to neonatal intensive care units compared with those born in a tertiary referral centre.

1- Every effort should therefore be made to transfer a woman to an obstetric unit linked to a neonatal intensive care unit prior to a preterm delivery.

2- Continuous electronic fetal heart rate monitoring once preterm labour is clearly established.

3- Cesarean section indicated in breech presentation.

4- availability of pediatrician and expert midwife and obstetrician at time of labour.

Preterm pre-labour rupture of membrane

Prof. maysoon sharief

Objectives:

1- Definition

2- Incidence

3- Diagnosis

4- Management

DEFINITON:-

Means Rupture of fetal membranes before 37 wks.

Incidence: 2 % of all pregnancies and accounts for up to one-third of preterm deliveries. 50% of PROM will deliver within a week, 75 % of PROM within 2 weeks and 85 % within 1 month. It appears to be an inverse relationship between gestational age and the interval between membrane rupture and preterm labour.

Causes: Over distention of the uterus, cervical incompetence, silence genital infection, uterine malformation.

Clinical features:

History

The most reliable diagnosis feature is "gush of fluid" vaginally, usually followed by a more or less continuous dribble. This must be distinguished from leaking urine (ask about frequency, urgency, leakage and dysuria). Fetal movements may be reduced and occasionally uterine irritability or contractions may be reported.

Examination

Infection may lead to an increased pulse and temperature. Abdominal examination may reveal a clinical suspicion of oligohydramnious or tenderness is present. The definitive diagnosis of PROM Can only be made by performing sterile speculum examination and observation of pool of amniotic fluid in the posterior vagina with visualize the fluid through the external os " Digital examination should be avoided".

Investigations

1- Nitrazine testing： Amniotic fluid is alkaline ,whereas the vaginal secretion are usually are acidic **.**An elevated PH turns a nitrazine stick black indicate amniotic fluid positives occur with high predictive value .

2- Genital tract swabs: May guide antibiotic therapy, screening for group Streptococcus (GBS).

3- Fetal fibronectin testing: The presence of fibronectin in vaginal secretions indicates PROM.

4- Ultrasound: It gives valuable information about the amniotic fluid volume.

Management

The management is balance between the risks of prematurity if delivery is encouraged and the risk of maternal and fetal infection if conservative management.

Indication of termination of pregnancy:

1- After 34 weeks to confirm lung maturity

2- Presence of chorioamnionitis which is diagnosis by maternal fever, tachycardia, rising WBC count and CRP level beside uterine tenderness, fetal tachycardia with offensive odor. Lower genital tract swabs are routinely taken in women with PROM . Positive cultures indicate termination.

3- Established uterine contraction and labour .

4- Confirmation of fetal anomalies.

5- Fetal death.

Expectant management

1- Maternal well-being: Daily assessment of the mother's blood pressure, pulse and temperature.

2- Fetal well-being: Serial ante partum CTG to exclude fetal distress.

3- Serial ultrasound assessment of lung hypoplasia(a pool of amniotic fluid greater than 2 cm is associated with a low incidence of pulmonary hypoplasia.
4- Use of erythromycin improves neonatal outcome.
5- Use of corticosteroid for enhance lung maturity is controversy.

Refrences: 1- Obstetrics by ten teachers, 19th edition.
2- Dewhurst for obstetrics and Gynecology.

NAUSEA AND VOMITING IN PREGNANCY

PROF. MAYSOON SHARIEF

Objectives:

- Definition
- - Incidence
- - Etiological factors
- - Clinical presentation
- - Management
- - Complications

Incidence:

50-80% of pregnant women experience nausea and, 25% of them had vomiting and 35% of all pregnant patients are absent from work on at least one occasion through nausea and vomiting.

The symptoms are usually begun between 4^{TH}- 6^{th} weeks of pregnancy**.**

Types of nausea and vomiting (NVP):-

- 1-Physiological type called (MORNING SICKNESS)
- 2-Pathalogical type called HYPEREMISIS GRAVIDIURM (HG) is Greek word: hyper = excessive, gravidarum= pregnant woman.

The nausea range from mild with or without once attack of vomiting that not interfere with patient daily work.

Morning sickness need no treatment just reassurance and self limiting.

NVP negatively impacts family relationships early recognition and management of NVP prevent progression to HG

HG is defined as vomiting in early pregnancy sufficient to warrant hospital admission. Incidence:- 0.5 – 2 %

Etiology:-

The exact cause is unknown.

- Genetic factor

- Hormonal:

1- HCG- the peak of HCG is between 8- 12 weeks which is the same time of NVP It may be due to increase level of HCG like

multiple pregnancy & hydatiform mole OR sensitivity to HCG in primagravida

2- High level of oestrogen & progesteron

3- Thyroid hormone

4- Immunology:- increase Cytokines (necrosis factor alfa protein and interleukin 6) both are release from T helper cell type1 .

5- Psychological, socio -cultural factors

6-Gastrointestinal infection with Helicobacter pylori .

7- Nutritional deficiencies:- vitamin K, thiamine ,Zinc deficiency

Clinical features:

are usually non- specific, result from serious sequel following vomiting like:

. Dehydration, ketosis , which aggravated by hungry ,iron intake, smell of food(hyperalfaction)

- Loss of 5% body wt.
- Nutritional disorder like B1, B6 ,B12 deficiency.
- Metabolic imbalance like ketoacidosis.

Laboratory test:

1- Non specific lab. Tests for diagnosis of NVP apart from dehydration include:

CBP, WBC, blood urea, GUE, LFT, TSH

2- Specific investigation to the diagnosis the underlying cause

Differential diagnosis

Infection:- Urinary tract infection ,hepatitis, meningitis ,gastrointestinal infection

Gastrointestinal disorder:- Appendicitis ,cholycystitis, fatty liver, gastritis, bowel obstruction.

Metabolic :- Thyrotoxcicosis, DM ketoacidosis

Drugs: - iron therapy

Management

Aims:- 1- Rehydration**.**

2- Minimizing risks to the mother and fetus.

TYPES OF TREATMENT:-

1- Non pharmacological treatment:

- Dietary and life style recommendation , emotional support, use of herbal remedies like ginger syrup 1g per day .

Drink small amounts of liquid .

- Fluids better than water(- diluted fruit juice).

-Small amounts of food more than large meals

- Avoid having an empty stomach; early morning nausea may be helped by biscuit before getting out of bed.

- Avoid fatty, rich or spicy foods. - Make the most of her best time of day-eat well when she feels best or whenever she feel hungry.
- - If the smell of hot food makes her feel ill-try having cold food instead .avoid cooking ask for help from family.
- - Lie down when nauseated.
- - Avoid stress.

Pharmacological therapy:

- pyridoxine (vitb6) which is given in dose of 25mg/8hrs.
- - Combination of pyridoxine (10mg) and 25mg of doxylamine(antihistamine).
- - Dopamine antagonists which inhibit gastric motility by block dopamine receptors like metoclopramide.
- - Corticosteroids in severe cases life treating. Prednisone orally 3 times daily.

Rehydration:

1- First line includes **oral rehydration** by oral fluid.

2- **Parental rehydration** and nutritional support:-

A- Intravenous fluid which supply energy(glucose) , electrolyte(Na, K,CL).

- Type of fluid like ringer lactate (Na ,k , cl ,suger ,Ca), 5% dextrose solution ,9% normal saline .

- Amount of fluid depend on daily requirement which is about 4-6 liter per day.

B- I.V thiamine or B complex in infusion

Management of sever condition

Enteral tube feeding in sever cases.

INDECATION OF TERMINATION:-

1- Hepatic fatty degeneration

2- Hepato renal encephaly

Lower genital tract infection

Prof. maysoon sharief

Objectives:-

- DEFINITION
- INCIDENCE
- TYPES OF INFECTION
- CLINICAL PRESENTATION
- INVESTIGATION
- TREATMENT

Lower genital infection

PHYSIOLOGY:

The vaginal epithelial state changes in different phases of life

- **Neonatal period-----** vaginal epithelium is stratified squamous due to the effect of maternal oestrogen.
- **Young female-------**thin layer of simple cuboidal epithelium.
- **Puberty--------** at the menarche the epithelium regenerates to form stratified squamous epithelium pH is decrease 3.5-4.5
- **Menapause**----- the vaginal epithelium undergoes atrophic changes pH more than 7.

Physiological vaginal discharge:

Every woman may had history of vaginal discharge which regard as physiological which is White, odorless, asymptomatic.

Content= desquamated epithelial cells from vagina and cervix, mucus, vaginal transudate, lactobacilli

Excessive discharge which is called leucorrhea which can occur in the following conditions:

- At the time of ovulation.

- At premenstrual period.
- At time of sexual excitement.
- During pregnancy , contraceptive pills users.

Types of vaginal infections

- 1- Fungal infection.

- 2- Viral infection : genital warts

  Herpes simplex

- 3- Bacterial infection: nonspecific infection

  Syphilis

  chancroid

  Lyphogranuloma venereum

- 4- Protozoa infection.
- 5- parasitic infection.

• 1- TRICHOMONIASIS

It is sexually transmittable infection

Causes by trichomonas vaginalis which is flagellate organism (protozoan). Its appearance ,four flagellae anteriorly and

membranous stylus posteriorly with typical movements make easy on microscopy of a saline preparation.

Clinical features:

- Yellow or green vaginal frothy discharges with soreness of vulva and adjacent skin , dysuria , abdominal discomfort
- o/E punctate haemorrhages on vaginal epithelium and redness of the cervix(straw berry cervix). The PH is 5.0 or higher.

DIAGNOSIS:

Microscopy of saline preparation shows the typical movement, ,culture by finebery media.

Treatment:

Oral metronidazole 2 g as single dose or 400 mg twice daily for 5 days both partners.

Bacterial vaginosis

- **CAUSATIVE ORGINISUM**: Gardnerella vaginalis , bacteriodes , mycoplasma hominis but predominantly anaerobic species that are usually present in the vagina at low concentration increase in concentration up to a thousand fold pH more than 4.5-7,lactobacilli are deficient and the cells may be heavily coated with the small bacteria involved giving them a finely stippled or hazy appearance known as clue cells .The condition is related to sexual activity with non- barrier contraception.
- **CLINICAL FEATURE:**

Offensive fishy smelly discharge, thin .homogenous adherent to the walls of the vagina with burning sensation with grey color discharge

Microscopy gram stain presence of clue

Vaginal PH of more than 4.5 .Release of fishy odor on addition of 10%KOH.

Culture = mixed anaerobic and high aerobic media.

TREATMENT:

- 400 mg /2 times day for 5 days of metronidazole, or 2g single dose as single dose.

CANDIDA ALBICANS VAGINITIS

Is around 80-90% of genital infection

Causative organism: Candida (monilia) albicans is a yeast- like fungus commonly affecting the vagina.

Clinical features: Characterized by light white discharge with curd-like patches adherent to the epithelium .Symptom are of discharge with severe vulvae itching ,edema , soreness

Diagnosis: direct microscopy shows the mycelia filaments a preparation in 10-20% KOH.

Predisposing factors: 1- pregnancy 2- use of oral contraception 3- Diabetes mellitus 4- taking of wide spectrum antibiotics 5- corticosteroid usage 6- condition include HIV ,immune compromised women.

Treatment: use of antifungal preparations such as clotrimazol and miconazole both are fungacidal single dose 500mg pessary or 100mg pessary for 6 -14 days, oral fluconazole single dose 150mg .

In chronic infection like patient with pill user , the topical treatment extend to 2 weeks

- Recurrent infection: oral fluconazol 100 mg in three doses every 72 hrs followed by maintenance dose of 100mg weekly for six months, or clotrimazole 500mg pessary weekly for 6 months.

CHLAMYDIAL INFECTION

- It is pelvic inflammatory disease start from cervix lead to endometritis, salpingitis , oophoritis, pelvic peritonitis and subsequent tubo-ovarian and pelvic abscesses long term complication tubal adhesions and infertility

Signs and symptoms: 1- pelvic pain, mucopurulent discharge, pyrexia, pelvic examination reveals tender adnexal mass. Lab. Test: elevation of WBC , C reactive protein ,ESR.

Adnexal masses may be seen by ultrasound examination.

TREATMENT: In mild condition can be treated as outpatient by oral floxacin 400mg twice+ oral metronidazole 400mg twice daily for 14 days. In severe cases need admission to the hospital and start parental therapy as ceftriaxone250mg+azithromycin1g/week for 2 wks with strong analgesia

GONORRHOEAL INFECTION:

Is sexually transmitted disease, caused by gram-negative diplococcus Niesseria gonorrhoea . Gonorrhoea cause about 5% of PID . N. gonorrhoeae initially infects the cervix but ascends to the upper genital tract in 10- 20 % of untreated cases

Signs and symptoms: 1- Half of women with gonorrhea are asymptomatic.

2- Symptomatic women are present with thick Mucopurulent discharge with dysuria , uretheral discharge , pelvic discomfort.

Diagnosis: A swab for gram stain and culture medium, nucleic acid amplification tests

Treatment: Single dose of cefixime 400mg.

Single dose of ciprofloxacin 500mg, ampicillin 2g

Single dose of amoxycillin 1g

ATROPHIC VAGINITIS

Vaginal atrophy presents into conditions

1- postclimacteric vaginitis, bleeding may occur and discharge from secondary infection . Correction of these problems has for long been easily achieved by oestrogen applied locally.

2- prepubertal children must always be suspected of foreign bodies or Pinworm (enterobius vermicularis) infestation It can be treated by topical oestrogen cream.

Condylomata acuminate:

Is venereal warts cause by papilloma viruses especially type 6 and 11.

Treated by local application of the resin podophyllin or by thermocautery ,cryo cautary ,lazer

HERPES SIMPLEX INFECTION:

Cause by type 2 herpes virus it is common in young ,unmarried women , it is due to sexual contact.

Incubation is less than a week, vesicles appear which proceed to ulcers with erythema and lymphadenopathy with severe pain tenderness.

Resolution take place spontanseously but recurrence is common representing a flare up of latent infection.

A direct smear from the vagina shows multinucleated giant cells with antinuclear inclusion, direct viral culture of vesicular fluid and antibodies studies on serum confirm the diagnosis.

Local antiseptic treatment and antibiotics for secondary infection along with symptomatic measures antiviral therapy both for local and systemic use like acyclovir

Caesarean section has been advocated to avoid risk of neonatal infection Primary chancre occur in the vulva it is painless rounded ulcer with raised borders, the floor is indurate and covered by gray slough

Dark ground microscopy reveals spirochetes treponema pallidum

Secondary syphilis called condylomata lata are coarse flat topped moist necrotic in contract to condylomata acuminata

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References: 1- Gynecology by Ten teachers 19th edition.

2- Dewhurst in obstetrics and gynaecology.

Premalignant disease of the cervix

Prof. maysoon sharief

Introduction:

Intraepithelial disease occurs in the cervix , vagina and vulva.It is rising in young women.

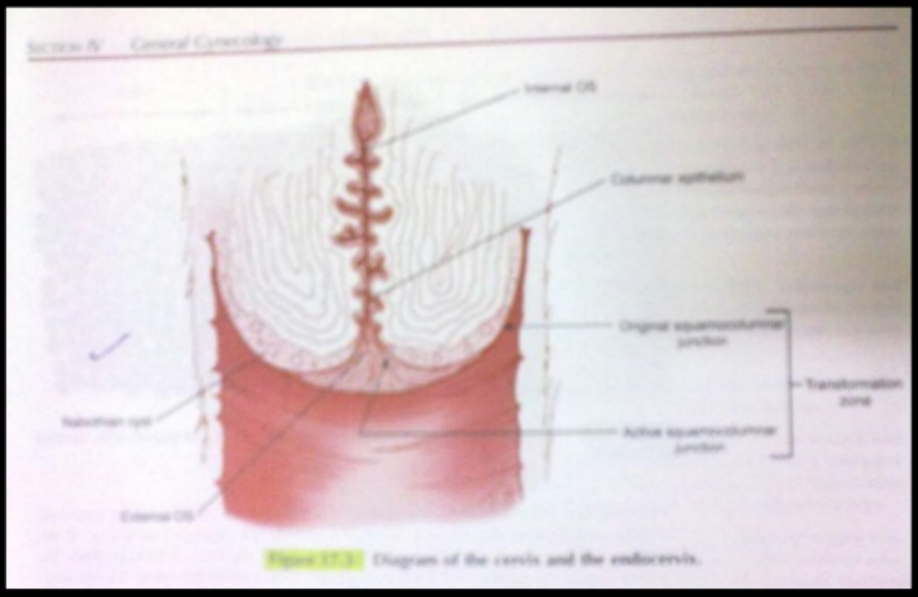
There are 450,000 cases of cervical cancer/year.

There are 300,000 death/year. It is the second most common cancer.

- It is a preventable disease (screening programmed-cervical smear).

Cervical anatomy: The cervix is composed of columnar epithelium, which lines the endocervical canal , and the sequamous epithelium, which covers the exocervix. The point at which they meet is called the squamocolumnar junction (SCJ).

CUT SECTION OF CERVIX



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CERVIX AND TRANSFORMATION ZONE

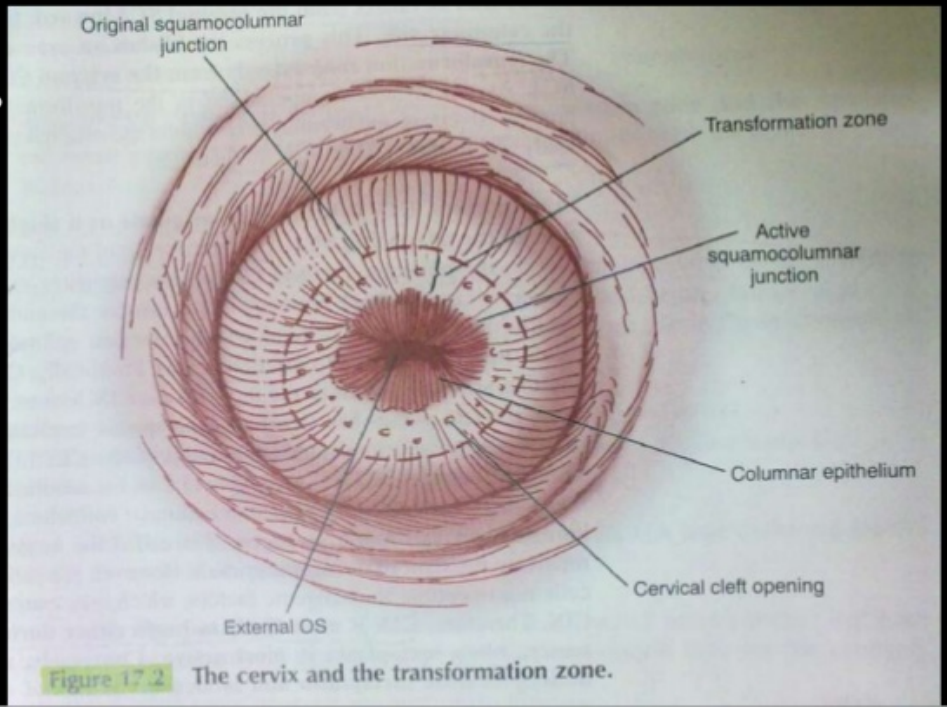


Figure 17.2 The cervix and the transformation zone.

Squamocolumnar Junction

- The SCJ rarely remains restricted to the external os. Instead, it is a dynamic point that changes in response to puberty. Pregnancy, menopause and hormonal stimulation.
- In neonates, the SCJ is located on the exocervix. At menarche, the production of estrogen causes the vaginal epithelium to fill with glycogen.
- Lactobacilli act on the glycogen to lower the pH, stimulating the subcolumnar reserve cells to undergo *metaplasia*.

LOCATION OF-SCJ

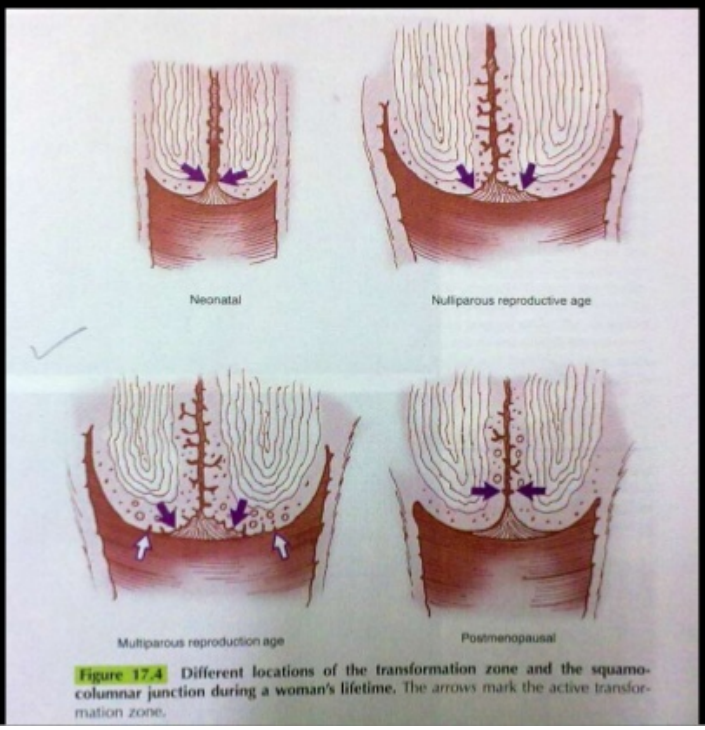


Figure 17.4 **Different locations of the transformation zone and the squamocolumnar junction during a woman's lifetime.** The arrows mark the active transformation zone.

SCJ

- Metaplasia advances from the original SCJ inward, toward the external os and over the columnar villi. This process establishes an area called the *transformation zone.*
- The transformation zone extends from the original SCJ to the physiologically active SCJ.
- As the metaplastic epithelium in the transformation zone matures. It begins to produce glycogen and eventually resembles the original squamous epithelium, colposcopically and histologically.

❖ Definition of cervical intraepithelial neoplasia(CIN):

- CIN is a pre-invasive change of the cervical epithelium where the epithelium thickness is replaced by abnormal cells in varying degree without breaching the cell membrane. Most of the cases are symptomless.

❖ Abnormal cytological changes of CIN (immature and disorganized cells) are:

- Increased Nuclear/Cytoplasm ratio.
- Prominence of nuclear chromatin.
- Multinucleate.

GRADES OF CIN

CIN is graded according the proportion of epithelium occupied by the abnormal cells.

- **CIN 1 (mild dysplasia):**

- *One-third or less is occupied by the abnormal cells.*

- Progress to (CIS) in 6%.

- Regressed or disappeared in 62%,

- **CIN 2(modrate dysplasia):**

- Between 1/3-2/3 of the epithelium is occupied by the abnormal cells.

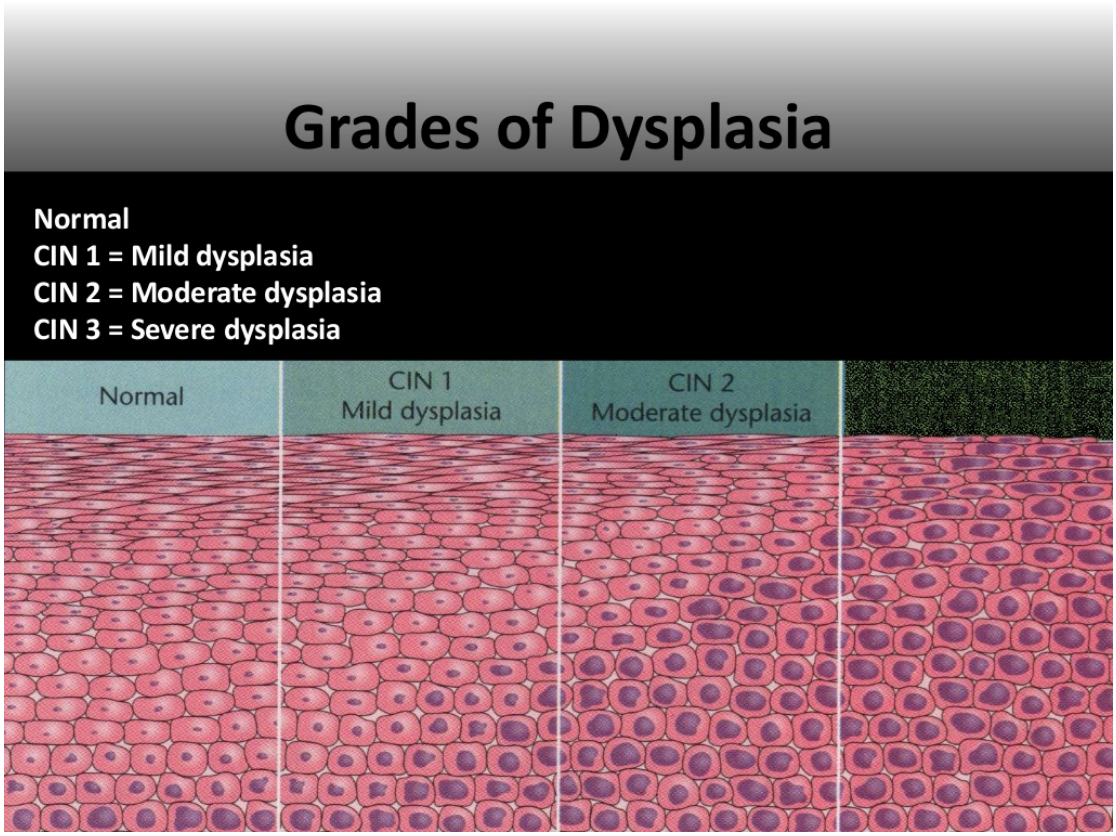
- Become invasive in 13%.

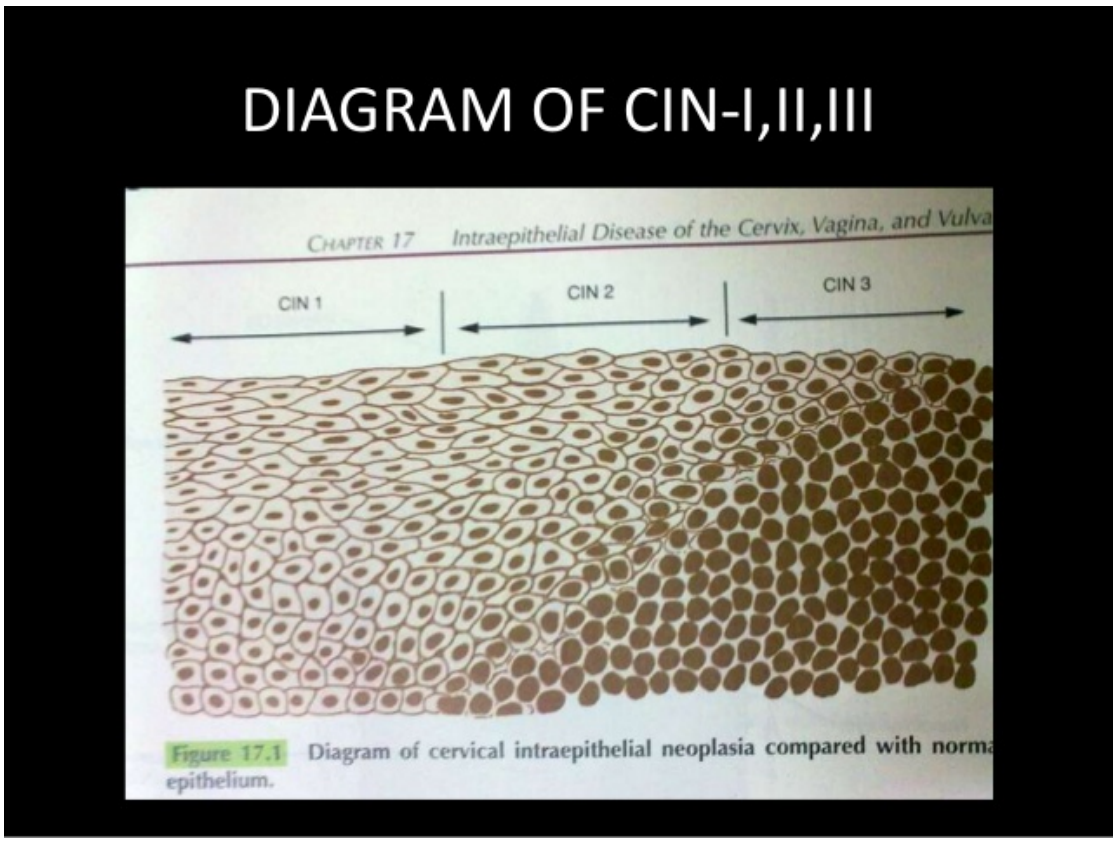
- **CIN 3 (severe dysplasia):**

- **T**he whole thickness of the squamous epithelium is occupied by the abnormal cells.

-It is regarded as carcinoma-in-situ (CIS).

- It could arise as CIN 3 or progress from CIN 1or CIN 2.
- Become invasive in 29%.





Incidence of CIN

- The incidence of CIN is 4 to 5% of Pap tests.
- The incidence of CIN vary according to the: (1) population studied, as the peak incidence being between 25 and 29 years of age, (2)socioeconomic factors, and (3) risk-related behaviours.
- The true incidence and prevalence can only be estimated, as screening cytology and colposcopy lack complete sensitivity.

Risk factors (Epidemiology):

1- Early age of first intercourse is the most important factor.

2- Multiple partners

3- Smoking

4- Race, studies reported that low incidence in women with their partners had been circumcised.

5- Hereditary factor may play a role.

6- Low social class and poverty may have a role.

7- High incidence among prostitutes.

8- More common in women with herpes simplex virus type 2 , and human papilloma virus.

9- Immunosuppression.

Cervical Cytology (The Pap test):

Initiation of screening: 3 years after onset of vaginal intercourse; no later than age 21.

Screening intervals: (1) age < 30 years: annually

(2) age > 30 years: : every 2 to 3 years after 3 consecutive negative tests.

(3) Patients with HIV or other immunocompromised state: 2 tests during the first year, then annually.

Discontinuation of screening: Women with age 65 to 70 years, women not at high risk (history of cervical cancer, HPV, HIV, & immunocompromised state).

Management of women with abnormal cervical smear:

If smear changes resulting from infection are recognized then the specific infection should be treated and further smear should be taken.

- Atrophic smear: give estrogen then repeat the smear.
- All women with abnormal smears should have a colposcopic examination.

Colposcopy:

- The patient is examined in the lithotomy position for exposure of the cervix using bivalve speculum. The coloposcope which is binocular microscope view the cervix telescopically at magnification range of 6 to 40 times.

4% of acetic acid is applied to the cervix which coagulates proteins of the epithelial cells and abnormal epithelium appears white; biopsies should be taken from that area.

Schiller test which is done by adding iodine solution(Lugols iodine) applied ,it had observed that squamous carcinoma of the cervix were lacking in glycogen and as a result failed to take up an iodine solution, biopsies should be taken from that area. Squamo-columnar junction should be seen entirely.

CIN Management

If the examination has been incomplete, if microinvasion or invasion is suspected, or if the colposcopic examination revealed no abnormality in spite of further positive smears being obtained then a cone biopsy.

Cone biopsies:

- **<u>indications</u>:**

- squamo-columnar junction is not seen by coloposcopy.

- negative colposcopic examination with positive repeated cervical smear.

- microinvasion or invasion is suspected.

Size of the cone should be kept to a minimum with normal tissue, tailored & cut with a knife. D/C is should be done.

- Complication: 1- Primary and secondary bleeding is achieved by using Dexon at 3 & 9 0'clock of the cervix.
- 2- Scarring of the cervix which lead to stenosis and impaired fertility, cryptomenorrhoea, abortion, preterm labour & cervical dystocia.

Types of management

1- Excisional therapy:

1- Cone excision(laser and diathermy loop excision)

2-Total hysterectomy

Indications:

- Persistent lesion after conization.
- Lesion extends to the upper vagina (colposcopy).
- Coexisting indication (menorrhagia or prolapse).

2- Local destruction (ablation): (for young, unmarried or wanting children):

- Cryocautery: freezing the tissue.
- Electrocautery: burning the transformation zone.
- Large loop excision of the transformation zone: using electrodiathermy.
- Cold coagulation: destruction of the transformation zone with a probe heated to 100 to 120Ċ.

malignant disease of the cervix

prof. maysoon sharief

Histology:

1- squamous carcinoma is 80-85%

2- adenocarcinoma is 15-20%

Clinical presentation

Early stage :1- asymptomatic.

2-postcoital bleeding ,postmenapausal bleeding, offensive blood-stained vaginal discharge.

Late stage: backache, leg pain/oedema , haematuria , bowel changes, malaise, weight loss.

Diagnosis

1- History

2- Cervical cytology

3- colposcopy + biopsy

4- Ultrasound

5- MRI, lymphoangiogram.

Staging

Staging include an assessment of disease extent and sites of spread by:-

1- Clinical examination.

2- cystoscopy, sigmoidoscopy

3- Chest x ray, Intravenous uragram

4- CT, MRI

FIGO staging of cervical cancer(2009)

stage 0 :carcinoma in situ.

stage 1: invasive carcinoma confined to the cx

a- micro invasive.

b- Visible lesions.

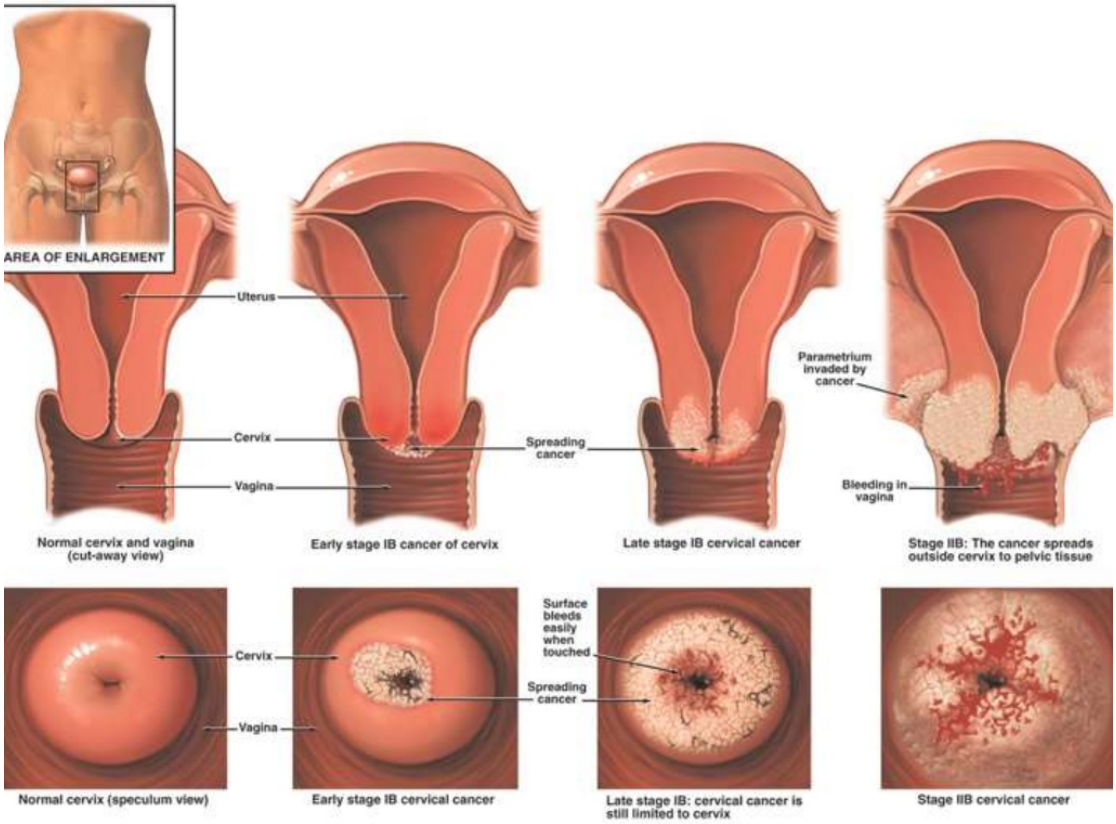
stage 2: cervical carcinoma invades beyond the uterus ,but not to the pelvic wall or to the lower third of the vagina with parametrical invasion.

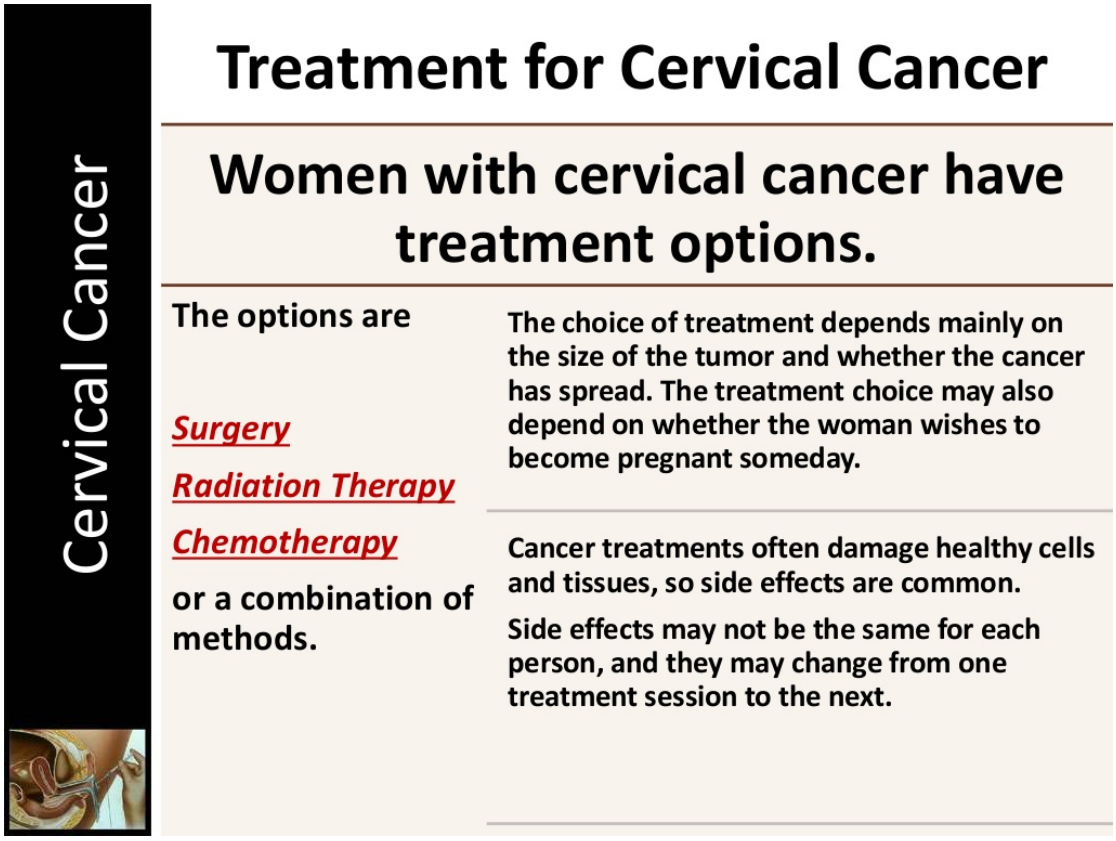
- **stage 3**: the growth extends to the pelvic wall and/or involves the lower third of the vagina and/or causes hydronephrosis or non functioning kidney.

- **stage 4**: the carcinoma has extended beyond the pelvis.

a- spread to adjacent organs.

b- Spread to distal organs.


AREA OF ENLARGEMENT
Uterus
Cervix
Vagina
Spreading cancer
Parametrium invaded by cancer
Bleeding in vagina
Normal cervix and vagina (cut-away view)
Early stage IB cancer of cervix
Late stage IB cervical cancer
Stage IIB: The cancer spreads outside cervix to pelvic tissue
Cervix
Vagina
Surface bleeds easily when touched
Spreading cancer
Normal cervix (speculum view)
Early stage IB cervical cancer
Late stage IB: cervical cancer is still limited to cervix
Stage IIB cervical cancer



Management

1- curative treatment

2- pallative treatment.

Management

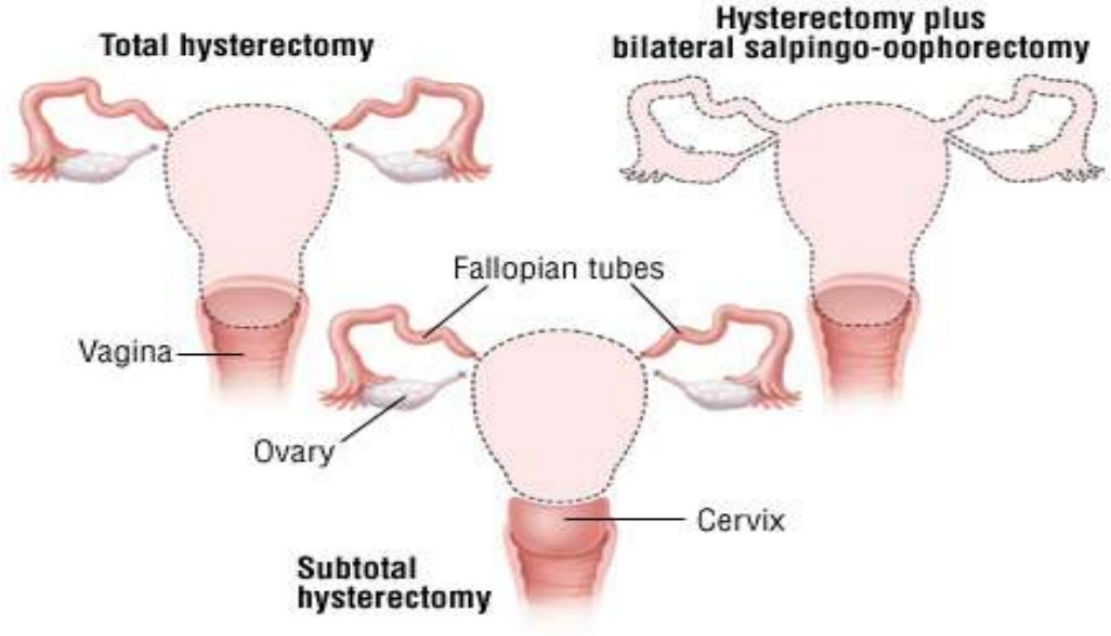
stage 1 : a- microinvasive need simple hysterectomy.

b- radical hysteractomy

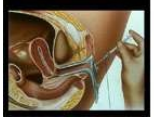
stage 2 : radical hysterectomy + radiotherapy.

stage 3 ,4 : radiotherapy +chemotherapy

Hysterectomy



Cervical Cancer



Radiation Therapy

Doctors use two types of radiation therapy to treat cervical cancer. Some women receive both types:	
▪ ***External Radiation Therapy***:	A large machine directs radiation at the pelvis or other tissues where the cancer has spread. The treatment usually is given in a hospital or clinic. External radiation usually takes place 5 days a week for several weeks. Each treatment takes only a few minutes.
▪ ***Internal Radiation Therapy***:	A thin tube is placed inside the vagina. A radioactive substance is loaded into the tube. The patient may need to stay in the hospital while the radioactive source is in place (up to 3 days). Or the treatment session may last a few minutes, and the patient can go home afterwards. Once the radioactive substance is removed, no radioactivity is left in the body. Internal radiation may be repeated two or more times over several weeks.

Complication of radiotherapy

Bowel and bladder urgency, due inflammatory effects.

Skin reythema-like burn.

Vaginal stenosis in long term complication.

Damage to ovaries, fistula formation.

Palliative treatment

1- Sedation for pain due to nerve infiltration.

2- Anemia correction.

3- Physiotherapy.

4- Urinary or bowel diversion.

References: Gynaecology by ten teachers.

Dewhurst for obstetrics and gynaecology.

Urinary incontinence in the female

prof. maysoon sharief

Objectives:

1- Definition

2- Types

3- Clinical presentation

4- Investigations

5- Management

Urinary incontinence

Is a distressing condition that, although rarely life threatening, but severely affects of a woman life. Though ignorance, embarrassment, belief and a belief that loss of bladder control is a normal result of childbirth and ageing, many women suffer for years before seeking help.

Anatomy of lower urinary tract

Bladder is a hollow muscular organ of smooth muscle fibers (detrusor) innervated by parasympathetic's.

Trigone (area between the two ureteric orifices and the urethral orifice) composed of two smooth layers which is continue with the smooth layer of the urethra. The superficial layer are devoid of parasympathetic's.

The normal urethra is 3-5 cm length, line by transitional cells in its proximal half and the distal part by str. Squamous epithelium.

Beneath this a rich vascular plexus and rich layer of collagen (contributes 1/3 of urethra pressure.

The longitudinally smooth muscles (its contraction lead to urethra shorting and opening).

The striated muscle is located in the middle third of the urethra circularly arranged (rhabdosphincter urethrae).

All the previous stractures called intrinsic sphincter.

The extrinsic sphincter consisted of the levater ani which had no direct connection with the urethra situated at the middle and lower thirds of the urthra

Both intrinsic and extrinsic sphincters called positive closure pressure and maintenance of continence.

Continence control

- intrauretheral pressure exceeds the intavesical pressure.
- The proximal 1/3 of the urethra should be intra abdominal organ for closure of urethra.
- Intact both sphincters
- The urethra is support by pudourethral ligament and its surrounding fascia

Physiology of lower urinary tract

Storage phase :

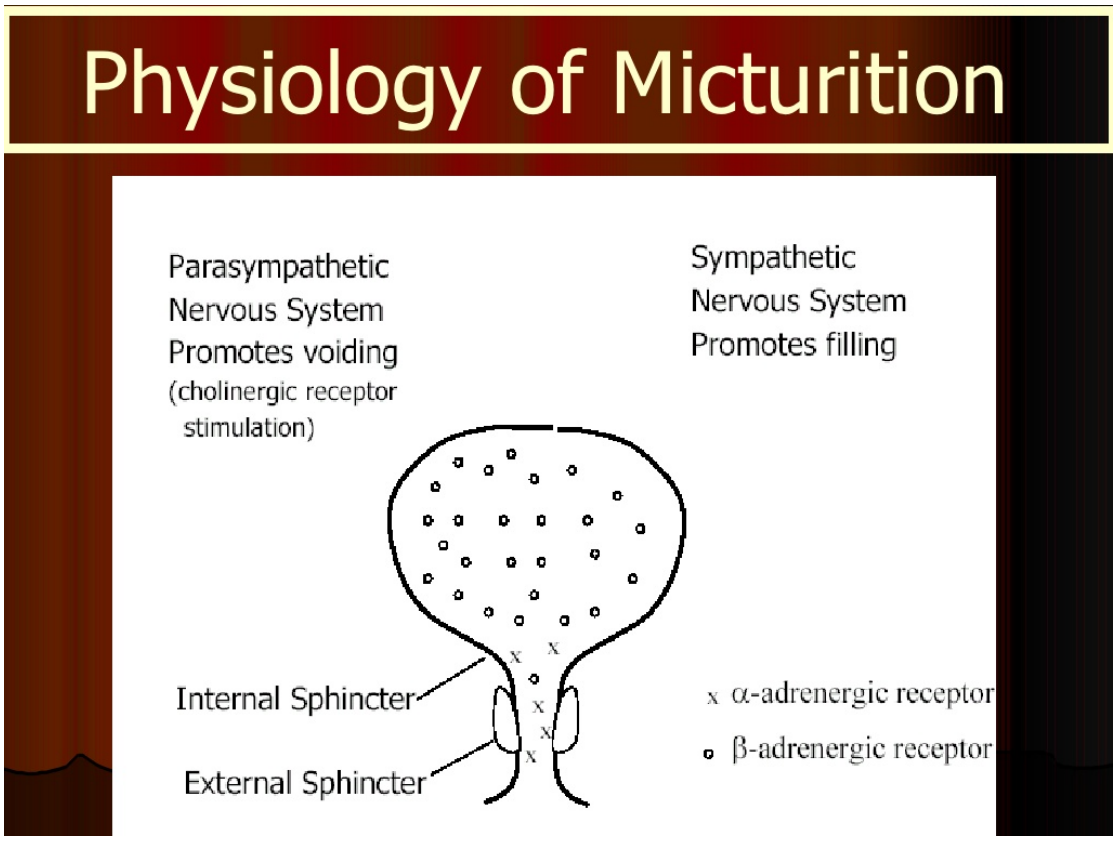
The 1st sensation of bladder filling is 150-250 ml. Bladder capacity is 400-500ml.

During filling the bladder pressure should not normally rise by more than 10 cm water in 300ml or 15cm water in 500ml urine.

Innervations: impulse from stretch receptors to sacral roots S2-S4 to spinal cord-----basal ganglia-----increase bladder filling lead to cortical stimulation.

Voiding phase:

At suitable time and place cortical inhibition is released and relaxation of pelvic floor occur and relaxation of intrinsic sphincter.



Definitions of urinary incontinence

Involuntary loss of urine which is objectively demonstrable & is a social or hygienic problem.

, **Incidence:** It increase with age and increase with degree of pelvic relaxation.

The prevalence of incontinence of 5% in (15- 44 years),10% (45-60 years).

CLASSIFICATION

1-URETHERAL CAUSES

-stress incontinence

Detrosal instability(unstable bladder)

Retention with overflow

2-EXTRA URETHERAL CAUSES

-congenital ectopic ureter

- fistula vesicovaginal

Path physiology of stress incontinence

Intravesical pressure exceeds urethral pressure because of weakness of urethral sphincter mechanism i.e. **Genuine Stress Incontinence**

GRADES

1- **Mild :** Incontinence with severe stress such as coughing, sneezing ,jogging.

2- **Moderate:** Rapid movement or walking up and down stairs.

3- **Severe:** Under mild stress like standing, the patient is continent in the supine position.

EITIOLOGY: 1- Childbirth may result in damage to the pelvic floor. The association between increasing parity, the risk increase by 5 fold in woman with vaginal delivery, forceps delivery, fetal macrosomia.

2- Menopause.

3- Pregnancy

4- Race

5- Quality of life

Physical examination

- The patient should be examined with full bladder to demonstrate the uncontrollable leakage of urine under stress.
- Local- excoriation of vulval skin due to local irritation of urine.
- Demostration of atrophic changes, cystocele, prolapsed in the vagina and vulva.
- Bladder neck elevation test (bonny test)- To see whether surgery will benefit or not.
- Assessment of mental state.
- Assessment of developmental anomalies.

- Assessment of neurological examination.

Investigations:

- **General**

i. Mid stream urine (MSU) for Routine and microscopic examination.

ii. Urine for culture and sensitivity.

iii. Frequency/volume chart or urinary diary by this chart provides an objective assessment of a patient fluid input and urine output, numbers of voids and incontinence episodes.

Pad test : Incontinence can be confirmed by asked the patient to drink 500ml of water. She then applies a preweighed perineal pad to the perineum and spends the next hour walking around, household duties. A weight gain of more than 1g in 1h normally represents urinary incontinence.

● Basic Urodynamics:

1-Uroflowmetry: The measurement of urine flow rate, is a simple test that can exclude the presence of outflow obstruction. The normal flow rate is 15-25ml/sec.

2- Cystometry-: Which measures the pressure- volume relationship within the bladder, can differentiate between stress incontinence and detrusor overactivity. The bladder is filled with physiological saline via a blood- giving set and

urethral catheter. The information that can be obtained is the intravesical pressure during filling , if > 15cm water after 250 ml of urine indicate detrusal instability.

Normal residual urine is less than 5oml.

First sensation of urge is 250ml. If earlier indicate urge incontinence.

Normal bladder capacity is 500-600ml. If increased indicate neurologic disease.

3- Videocystourethrography :- Which Combines cystometry, uroflowmetry & radiological screening of bladder & urethra.

Most informative, but expensive/time consuming.

4- Cystourethroscopy: is normally carried out under general anaesthesia , it may reveal abnormalities of the bladder by inspection of urethra , urethrovesical junction, and bladder walls ,and ureteral orifices

Observation: 1- the amount of residual urine

2-bladder capacity(400- 500cc)

3-exclude inflammation ,diverticula,

4-mobility of the urethrovesical junction in response to rectal squeeze, cough.

Management of stress incontinence

- **Conservative**

a. Pelvic floor muscle training (PFMT)Kegel's exercises and physiotherapy.

b. Weight reduction in obese patients.

c. Treatment of chronic cough, Urinary tract infection.

d. Faradism- interrupted current to stimulate muscles & nerves.

Medical therapy:- In the past estrogen, α adrenergic agonists have been used .Duloxetine is potent serotonin (5 – hydroxytryptamine) and noradrenaline reuptake inhibiter that enhances urethral striated sphincter activity via centrally mediated pathway in dose 20 mg -40mg.

Surgical management

Surgical treatment is usually the most effective way with curative rate of 90%

Types

1- Vaginal approach:- anterial colporraphy

2- Abdominal approach :- It is suprapubic operation in which the paraurethral tissue at the level of bladder neck is sutured to the periostium it is called (marshall-marchetti) or to the iliopecinal ligament it is called(burch coloposuspension) .

3- Laparoscopic colposuspension

4- Sling procedures.

5- Artificial sphincter implant.

6- Retro pubic mid – urethral tape like tension- free vaginal tape(TVT). Trans obturator tension tape (TOT)

7- Urinary diversion- (terminal stage).

Detrusor Instability

It is define as spontaneous detrusor contractions during the filling phase when the patient. Is attempting to inhibit micturition , with urgency, enuresis, frequency especially nocturia.

No specific clinical signs.

CAUSES

- Idiopathic.
- Emotional, psychosomatic.
- Upper motor neuron lesion like multiple sclerosis (neurogenic detruser overactivity).
- Radiotherapy, recurrent urinary tract infections.

Management:-

The treatment for detrusor over activity aims to re-establish central control or to alter peripheral control via bladder innervations. Various behavioral interventions have been used, it shown to improve symptoms in up to 80% but it is time consuming.

1- Psychotherapy:- (give normal volume of fluid, keep fluid balance chart, increase voiding interval between 1.5-2hs, give encouragement).

2- Drug therapy :-

a- inhibit bladder control :- anticolinergic agent

propantheline 15-30\3 time .

Tricyclic antidepressent Impramine 25-50\2-3 times daily

b- Drugs increase outlet resistance: - alpha adrenergic stimulated, estrogen

c- Reduce urine production-- desmopressin.

d - Bladder training.

e- Denervation- bladder transaction.

vaginal denervation, sacral neurectomy.

f- Cystoplasty- clam ileocystoplasty.

RETENTION WITH OVER FLOW

Detroser hypotonic: Retention of urine due to failure of bladder to contract , after time and when the amount of urine exceed the maximum bladder capacity lead to over flow leakage of urine .

Women with present in variety of ways ,they may complain of dribbling urine or small amount at frequent intervals .The diagnosis is usually made by the discovery of a large bladder on clinical examination, exclude pelvic mass or cystocele. this can be confirmed by a postmicturation ultrasound scan to assess the residual urine volume more than 50 cc deside reduced peak flow rate of less than 15 ml/s.

Causes 1- lower motor neuron disease.

2- spinal cord injuries .

3- autonomic neuropathy.

4 – urethral obstruction.

5-postoperative retention.

Diagnosis 1- poor stream.

2- Incomplete bladder emptying.

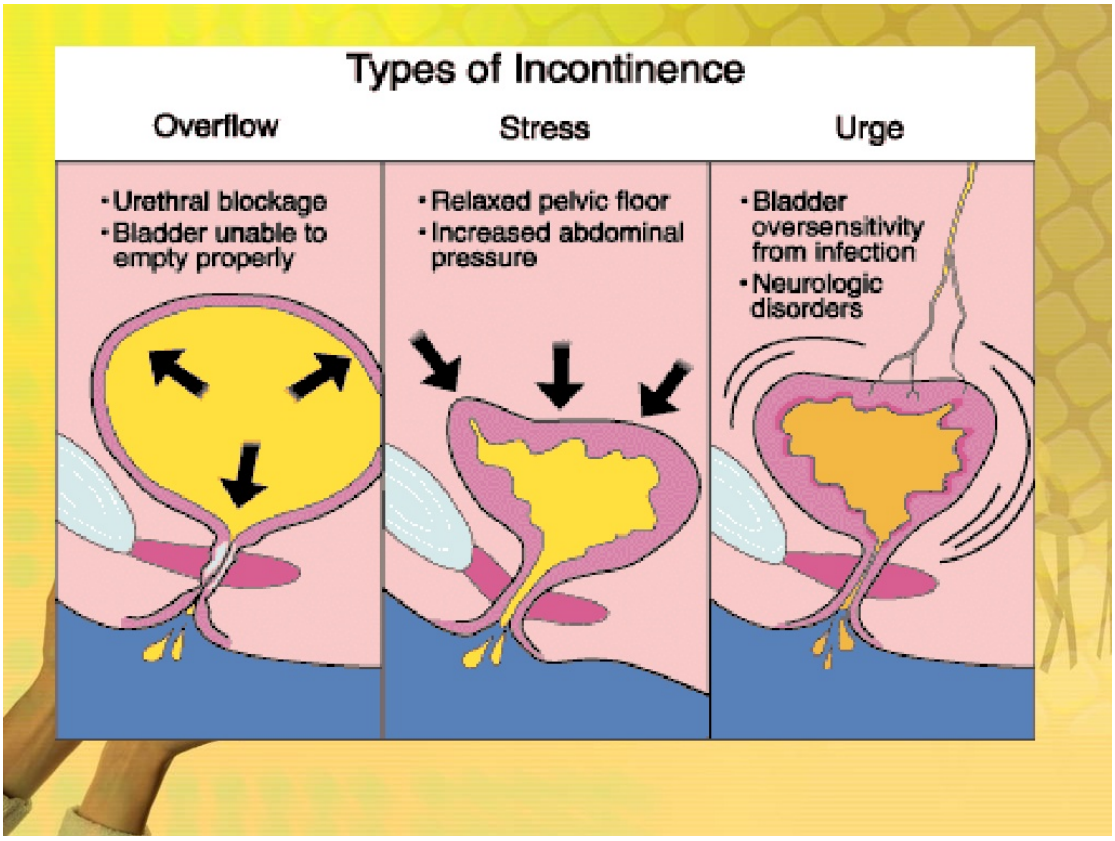
3- Straining to void.

Management: -

Depend on underlining causes, intermittent cauterization

Bladder training.

cholinergic agent bethanechol 25 mg\3 time, dilatation of urethra

- 

TRUE INCONTINENCE

1- CONGENITAL :-

- Ectopic ureter single or bilateral open inside the urethra or upper vagina.

- Bladder exstrophy.

2 -FISTULA :- is abnormal opening between the urinary tract and the outside. May be ureterovaginal , vesico vaginal fistula.

Causes **A** - obstetric like obstructed labor.

B- gynecological :- pelvic surgery.

Pelvic malignancy. Radiotherapy.

DIGNOSIS :- They are usually visible on speculum examination and Methylene blue dye test.

Cystouretheroscopy.

Intravenous cystourethrography.

Treatment - 1- conservative:-

Catheterization for 2-3 months in acute conditions

2- Surgical repair is advisable if no response to the conservative treatment or in chronic conditions through abdominal approach or vaginal approach.