Ovarian tumour

normally cells division occur only when additional cells

are required for normal body function .

At certain times the control that regulate cells division is lost , result in accumulation of more & more cells without order , eventually these cells grow into cell masses and termed tumour

Types :

1. **benign**
2. **Border line**
3. **malignant**

Benign

Not cancer

Only rarely life threatening

Do not invade or spread other tissue

Can be removed & rarely grow back

Borderline

Groupes of tumour which are intermedate in both behavour & histological feature between benign & malignant, (semimalignant or low grade malignancy)

Most of them benign

Few spread & progress esp. lymphatic

Prognosis is good but depend on spread

Malignant

Are cancer

spread beyond ovary , invading & damaging other organs (metastasis)

Grow back

Ovarian cancer

is a disease of old women predominantly over age of 50,

Classification

1. **epithelial (mullerian in origin)**
2. **sex cord stromal tumour**
3. **germ cells tumour**

4. **Metastatic**

Epithelial : serous, mucinous, endometriod, clear cell and Brenner

Sex cord : androdlastoma, thecoma- fibroma, granulosa cell tumour

Germ cell tumour : teratoma , endodermal sinus tumour, non-gestational choriocarcinoma and dysgerminoma

Epidemiology

Age : increase with advancing age (sixth decades)

Smaller family size

Industrialized country lowest in Japan , ↑ Sweden

Previous pelvic irradiation

Null Para or low parity

Early age of first pregnancy

Breast feeding is protection

More common in women who ovulates more

Infection (mumps) is protective

Use of fertility enhancing drugs

Oral contraceptive pills is protective

Hysterectomy with unilateral oopherectomy is protective

Tubal ligation decrease risk by 33%

Use of talc product in feminine hygiene

Genetic predisposition : high risk family (those who have one or more close relative affected) BRCA1 , BRCA11 (gene locus on chromosome no. 17) carries 40 % risk of developing ovarian cancer

CLASSIFICATION: FIGO

♣**Stage 1** **growth limited to the ovaries**

1a one ovary , no ascitis , no tumour on external surface , intact capsule

1b two ovaries no ascitis , no tumour on external surface , intact capsule

1c 1a or 1b with ascitis or tumour on surface or rupture capsule

♣**Stage 2** **growth involve one or both ovaries with pelvic extension**

2a extension to uterus or tubes

2b extension to other pelvic orgens

2c 2a or 2b with ascitis , tumour on surface…

,

♣Stage 3 involve one or both ovaries with peritoneal implants outside the pelvis and or positive retroperitoneal or inguinal implants

3a microscopical seeding of abdominal peritoneal

3b macroscopical disease outside the pelvis less 2cm

3c abdominal implants greater than 2cm and or positive nodes

♣Stage 4 growth involve one or both ovaries with distent metastasis including liver & lung

screening

Ovarian cancer is the fifth leading cause of death in women & most cancer of the femal reproductive system

Screening is indicated in order to detect the disease at early stages

Indication :

1. Mother & or sister with ovarian cancer
2. H\O breast cancer
3. Over 50 years
4. No children
5. Other risk

3 methods :

1. Routine pelvic examination
2. Vaginal USS
3. tumour marker

-Suspicious feature on USS :
Bilateral
↑size of ovary
Internal septa
Partly solid & partly cystic
↑ blood flow by doppler
-CA 125:
Glycoprotein shed by 85% of epithelial tumour
Cutoff level 30 - 65 iu\l

Lack sufficient specificity if used alone but can be used as primary screening to identify candidate for USS

Require no expertis

False positive occur with other malignancy (liver & pancreas) , endometriosis , PID , pregnancy

Specificity increased by serial measures

Tumour marker

Protein selectively secreted by tumour cells, which upon released into circulation can be detected in peripheral

blood , using immunological methods

Ideal marker

Specific protein produce by tumour

In the earliest stage of disease

Allow rapid diagnosis

Concentration falls following treatment

Rises again with recurrence

Three types :

1.Carcinoplacental antigen : HCG , placental alkaline phosphates

2. Carcinoemberyonic antigen : alfa feto protein , beta- oncofetoprotein which secreted by all epithelial tumours

3. Tumour associated antigen : glycoprotein on the surface of ovarian cancer e.g CA125

Advantage of screening

ovarian tumou metastasizes so fast , so early so early detection helps to

- limite the disease

-make treatment easy

-cure rate high

Clinical features

-Early stages ..... asymptomatic

-Symptom may appear 6 - 12 months before ovarian cancer is found

-Mostly due to pressure on surrounding tissue e.g bladder & rectum

Lower limbs edema , varicosity & hemorrhoids

-Pain in abdomen or pelvis (cramps)

-Increase abdominal size

Symptoms related to complication (torsion , hemorrhage , rupture)

Hormone secreting tumour (either masculinizing or feminizing)

Malignant changes (ascitis , vaginal bleeding weight loss)

Investigation

1. **Physical & pelvic examnation**
2. **Radiological (pelvic & abdominal USS) : features suggest malignancy multilocular , opaque fluid , solid in consistency , papillary projections , ascites)**
3. **Doppler USS**

4. **CT scan & MRI (identify L.N)**
5. **No paracentesis**
6. **Blood test (tumour markers)**
7. **Definitive diagnosis laparatomy & biopsy (ascitic fluid or tissue)**

treatment

Benign → cystic : cystectomy

→ solid or patient age more than 45 : oopherectomy

1. **Surgery : is primary step in management .**
2. **Chemotherapy : adjuvant .**
3. **Radiotherapy : of little role .**

Surgery : optimal or debulking

role of surgery → daignostic

→staging

→therapy

Aim : to leave no more tumour deposite greater than 2cm in diameter

Optimal (surgery which leaves no tumour deposite than 2cm)

Debulking (done when optimal surgery is difficult)

Stage 1 , 11a

→ primary treatment is TAH+BSO+ omentectomy

→pelvic & paraoartic L.N ectomy

→appendicectomy

→ no chemotherapy 1a , 1b

midline incision : adequate exposure

Take ascitic fluid for cytology Or install 100c.c of normal salin

Check liver & diaphragm

Biopsy for enlarge L.N

5- year survival 67% - 89%

Advance ovarian tumour : debulking + chemotherapy

Chemotherapy

Play central central role , given after surgery

Platinum based drugs (single drug)

Cisplatinum , carboplatinum are currently mainstay of treatment

3 – 4 weeks interval for 6 courses

Side effect : nausea , vomiting neurotoxicity & nephrotoxicity & B.M depression

Other combination : hexa , 5fu , cycloph , adriamycin

New drug : paclitaxel

Response rate 60- 80 %

Radiotherapy

Adjuvant when debulking performed

As consolidation therapy added to chemo & surgery

Used for local deposite

Used esp . For germ cell tumour

Types

1. **Intraperitoneal radioactive isotopse**
2. **External beam radiotherapy**

Second look laparatomy

Indication :

Original surgery disease is too extensive or fixed

After chemotherapy

Undertaken 12 months after surgery

Same procedure

Immunotherapy

non specific

specific

corynebacterium parvum , BCG , stimulate immune system (cell mediated immunity)

Association between immune system & prognosis

Specific tumour associated antigen

allogenic irradiated tumour cell

Role of cytokine & interferon

Borderline tumour

can be treated by unilateral oopherectomy in young & wish to preserve fertility

Spread

1. **Local : peritoneal cavity**
2. **Lymphatics : pelvic & paraoartic , 6-8 chanal accompany ovarian vessles , broad ligament to obturator L.N**
3. **Hematogenses : bone , brain , lung , liver**

Serouscystadenoma :

Commonest cystic tumour

Usually bilateral

Lined by cuboidal epithelium

Unilocular with papillary projection (malignant)

Wall contain calcified granules psammoma body

Fluid serous

Mucinouse

Usually multiloclar

Large size up to 100kg

Fluid is mucin

unilateral

Lining epithelium is tall columnar secretory

Little tendency to form papillary

Less likely to become malignant

Granulosa cell tumour

Arise from granulosa cell

Produce oestrogen

3% of all ovarian tumour

25% exhibit malignancy

Can be presentedat any age

Symptoms depend on age (precocious puberty , endometrial hyperplasia , PMB)

Arrhenoblastoma

Arise from sertoli- ledig cell

Secret androgen

20- 30 years

symptoms (amenorrhea, virilism)

Mature teratoma

Dermoid cyst

12-15%

Contain large number of emberyonic elements e.g hair , skin , bone , teeth ,

Can be recognized radiologically

Asymptomatic unless complicated

2% only malignant

Immature teratoma

Occur in children & young adult

Usually unilateral & solid

Content similar to mature

Highly malignant

Dysgerminoma

Most common germ cell tumour

Occur in second & third decades

Highly sensitive to radio & chemotherapy

20% daignosed during pregnancy

Hormon secreting tumour (oestrogen , HCG , androgen)

Yolk sac tumour

Endodermal sinuses tumour

Second most common germ cell tumour

Always unilateral

Secret alfa fetoprotein

Highly malignant

Choriocarcinoma

Rare

Children & young

Secret HCG

Highly malignant

Meig's syndrome

Characterizied by :

1. **ovarian mass (ovarian fibroma) benign tumour**
2. **ascites**
3. **pleural effusion**