Endometrial caner

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ILO

- **K:1,4,5**
- **S1,3,4,7,13, 17. 18, 21, 18, 21, 23, 24**
- **AB : 1, 3, 4, 5,**
- **Spcefic objective:**
- **To define endomaterial cancer**
- **To know clinical staging of disease**
- **To know different modality of treatment and follow up**

Malignant disease of the uterus:

Endomaterial carcinoma (CA) has good prognosis in which (5 years survival rate is 60%)

Epidemiology:

Median age of presentation is (60 years old).
*(75-80%) of cases are postmenopausal.
*(3-5%) of cases are premenopausal (< 40 years).

Aetiology:

1-excess unopposed oestrogen stimulation of the endometrium.

2-premenapausal female which have high incidence of anovulation due to PCOS.

3-obesity because the main circulating oestrogen in postmenopausal women is derived from aromatization of peripheral androgen in fat & muscles & also reduce SHBG.

4-disturb carbohydrate intolerance but in the cause is unclear in postmenopausal women with diabetes have increase oestrogen independent of body weight so there may had altered oestrogen metabolism independent of the effect of the weight.

- 5-women with personal history of breast or colonic cancer.
- 6-women who take tamoxifen are exposed to a risk of oestrogenic type effect on the uterus.
  7-ovarian tumor like granulosa theca cell tumor in which there is (10% endometrial cancer & 50% endometrial hyperplasia).
- 8-family history of breast, colon & endometrial cancer.
- 9-nulliparity.
  10-early menarche & late menopause.

Endometrial hyperplasia:

This partly because these lesion cannot be identified clinically & their detection is depend on blind biopsy.

Pathology:

a-simple hyperplasia: (cystic hyperplasia): is characterized by increase number of glands that are dilated with irregular outline , some degree of crowding & reduce in amount of stroma but no cytological atypia.

b-complex hyperplasia:(adenomatous hyperplasia): the glands have irregular outline show marked structural complexity, the glands show(back-back) crowding with little stroma.

c-atypical hyperplasia: the glands show nuclear atypia & abnormal mitosis figures & sever structural abnormalities.

Aetiology:

No obvious predisposing cause may found but the most common cause is excess oestrogen unopposed by progesterone either arise from an ovulatory cycles or oestrogen secreting tumor & tamoxifen affect.

Natural history:

*cystic hyperplasia: is common in postmenopausal women & an ovulatory teenager, it rarely seen with endometrial CA & the risk of progression is (0.5- 1%).

*adenomatous hyperplasia: the risk of progression is (3 – 4%).

*atypical hyperplasia: CA may exist in (25 – 50 %) or concurrent endometrioid ovarian CA.

Clinical features:

In premenopausal women they present with abnormal bleeding, in simple hyperplasia the patient present with infrequent heavy period but the complex & atypical hyperplasia does not give characteristic pattern of bleeding, the largest group is postmenopausal & per menopausal.

Investigation:

1-outpatient biopsy.

2-hystroscopy.

3- U/S.

4-EAU.

5-D&C.

Treatment:

1-discontiue oestrogen treatment & remove the oestrogen secreting tumor.

2-cystic hyperplasia: does not require special follow up & may manage on the basis of subsequent symptoms.

3-adenomatous hyperplasia: have low risk of progression to CA so there is no indication for hysterectomy or progesterone therapy & subsequent management depend on further symptoms.

4- atypical hyperplasia: TAH& BSO because of high risk of coexistence CA, younger women who wish to preserve fertility can manage by progestogen (20mg/day) may stop after (8-12 weeks), long term follow up is require because of risk of recurrence.

Pathology of endometrial CA:

1-endometrial adeno CA.

2-adenosequmous CA.

3-papillary serous CA.

4-endometrial stromal SA.

5-clear cell CA.

6-malignant mixed Mullerian tumors.

Spread of endometrial CA:

*Endometrial CA invade to myometrium some time extend over the endometrium before penetrating the myometrium.

*also invade the lymph nodes (para-aortic LN).

*spread to the cervix by extension but infiltration through the lymph & cervical stroma is more common.

*direct infiltration into paramatrium is uncommon except when the cervix is involved.

*spread to ovary is common, transpetoneal spread occur spread when myometrial invasion reach the serosa or via fallopian tubes.

Staging system of endometrial CA:

Ia: tumor limited to the endometrium.

Ib: invasion < half of myometrium.

Ic: invasion > half of myometrium.

II a: endocervical glands involvement only.

II b: cervical stroma invasion.

III a: tumor invades serosa or adnaxia or +ve peritoneal cytology.

III b: vaginal metastasis.

III c: metastasis to pelvic & para-aortic lymph nodes. IV a: tumor invade bladder or bowel mucosa.

IV b: distant metastasis including intra-abdominal or inguinal lymph nodes.

Clinical feature:

1-abnormal bleeding & discharge or pain or abnormal screening tests.

2-post menopausal bleeding because (75 – 80%) of disease in this age.

3- vaginal spotting & this should not related to atrophic vaginitis unless investigation is normal.

4-cervical smear & endometrial biopsy should be performed in all cases

5-diagnostic curettage in patients with pyometra reveal cancer in 5% of cases.

6-pain due to nerve compression or constant dull pain or cramping due to pyometra.

7-in premenopausal there is irregular bleeding or heavy but regular periods.

Clinical examination:

1-enlarge lymph in the groin or supra clavicular lymph nodes.

2-metastic focus in the vagina commonly on the anterior wall.

3-the uterus enlarged or spread to adnexa or parameterium.

4-breast cancer may present with uterine or ovarian spread.

Investigations:

1-out patients :

1- endometrial biopsy

2- vaginal & abdominal U/S.

3-hystroscopy.

4-colposcopy.

2-in patients:

1-EUA.

2-fractional curettage.

3- hysteroscopy.

3-general tests:

1- CXR.

2-IVU.

3-CBP.

3-RFT

4-MRI.

6-S. electrolytes.

Treatment:

a-stage I:

1- surgery: TAH & BSO.

* the adnexa should be removed because of risk of subclinical metastatic tumor rather than to eliminate any hormonal influence.

* removal of vaginal cuff not reduce the recurrence or improve survival

* occlusion of the cervix & fallopian tube to prevent intra operative spillage of tumor are unnecessary. *the role of lymph adenectomy is to identify those women without nodal disease who not need radiotherapy to avoid the risk of complication, if pelvic LN involved so dissection is performed.

2-radiotherapy:

brachytherapy to vaginal vault or teletherapy to the whole pelvis, post-operative radiotherapy to vault reduce recurrence & mortality rate.

Teletherapy used in women with poor prognostic factors such as:
1-invasion > half way through the myometrium.

2-high grade tumor & large tumor.

Stage II:

The treatment depend on microscopic & macroscopic involvement of the cervix, if the disease is occult in the cervix so the treatment is the same as stage I with same prognosis, the need for radio therapy is depend on depth of myometrial invasion & tumor grade.
Prognosis not improve by post-operative teletherapy but complication increase, if the disease is obvious in the cervix so prognosis is worse (5 years survival rate is 30-60 %) so treatment by TAH & BSO & bilateral pelvic lymphadenectomy & para-aortic LN sampling.

Stage III:

CT scan should be performed, if the disease confirm to the pelvis so the radiotherapy is the treatment of choice. If the disease in the adnexa laparotomy should be performed to see the extension of disease & to remove as much as possible from the tumor.

Stage IV:

The aim is for symptoms control & local tumor control so radiotherapy cytotoxic & hormonal treatment may be required.

Recurrent disease:

(70%) of recurrence following primary treatment occur within the 1st (2-3 years), early recurrence carry poor prognosis & vault recurrence is more common in the non-irradiated patient . if there is single site of metastasis so radiotherapy cure (30-60%) of recurrence & the radiotherapy is of value to relief pain & discomfort due to bone & nodal disease.

Progesterone treatment the response rate is (15-20%) with grade 1 tumor than grade 3 tumor, Medroxy progesterone acetate oral (200 mg) twice or three times / day.
Cytotoxic treatment is less attractive option because the patient is old & medically unfit the drugs is Adriamycin, cisplatin & cyclophosphamide.

Uterine sarcoma:

Highly malignant tumor, the incidence rate is (3-5%), it is common in black women & those with history of previous pelvic irradiation.

Endometrial stromal SA:

*low grade ESS: it arise from stroma & from adenomyosis & endometriosis. It is look like a fibroid but in (20-30%) of cases spread into broad or cardinal ligament, adnexa & other abdominal organs .
treatment: TAH& BSO with wide excision of parametrium, the recurrence is high as (50%) & can be detected by CA 125 markers.

*high grade ESS: it is highly aggressive tumor occur after the menopause & presented with post-menopausal bleeding or discharge or pain.
treatment: TAH & BSO & radiotherapy.

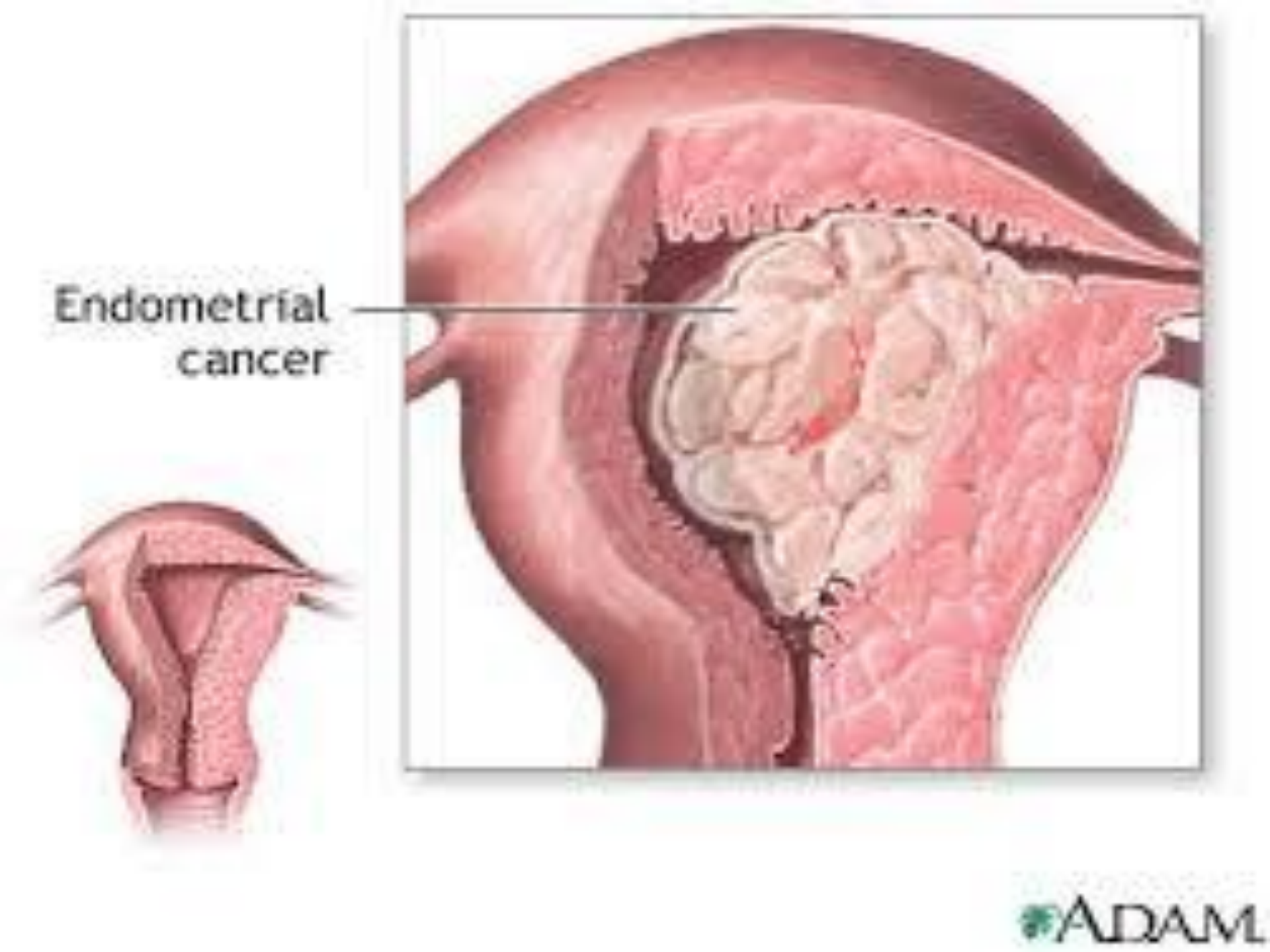
Malignant mixed mullerian tumor:

It is composed from malignant glands & malignant stroma. It is called carcino sarcoma if homologous elements are found & called mixed mesodermal tumor if heterologous elements are found.

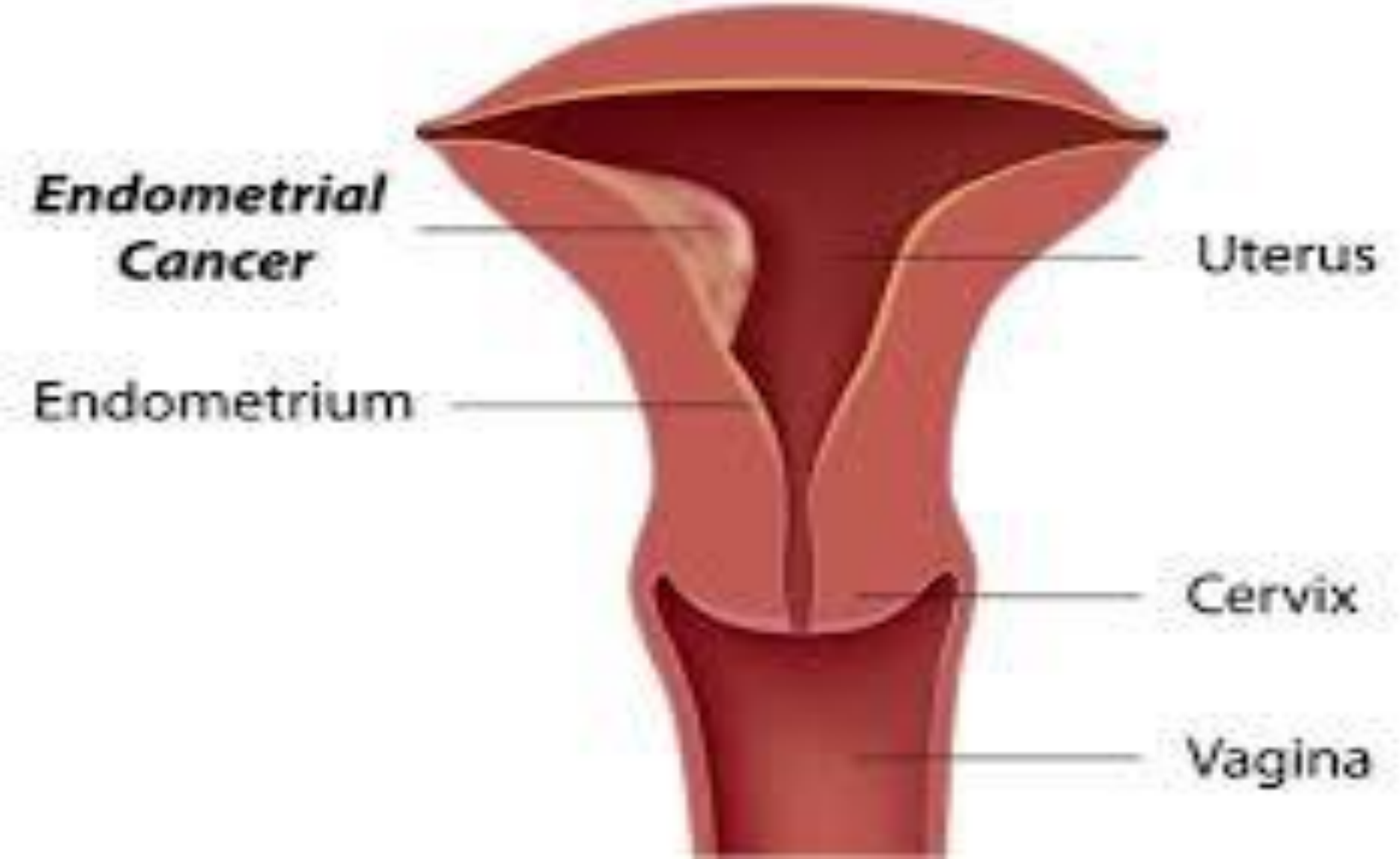
CLF: it is presented with abnormal bleeding, pain & mass, the average age is (60 years) & its highly aggressive tumor.

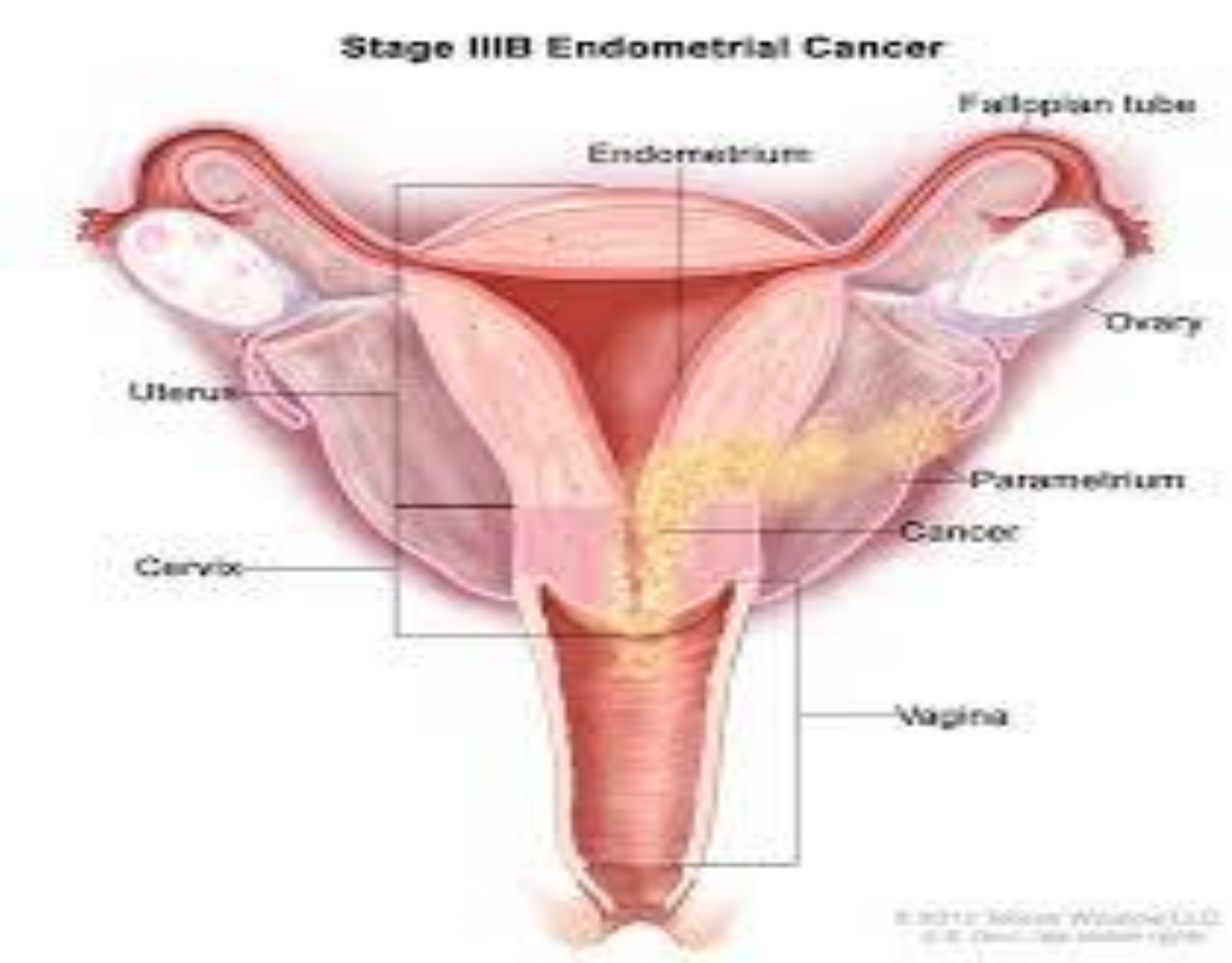
Treatment: as endometrial CA with radiotherapy.

- ***Myometrial tumor:***
- Lieomyosarcoma: look like fibroids but the cut surface yellowish more than of fibroid with areas of hemorrhage & necrosis. The (5-10%) of lieomyo SA that arise from fibroid have better prognosis than those arise directly from normal myometrium.
- Treatment : TAH & BSO.


Endometrial cancer

Endometrial Cancer




Stage IIIB Endometrial Cancer
Fallopian tube
Endometrium
Ovary
Uterus
Parametrium
Cancer
Cervix
Vagina

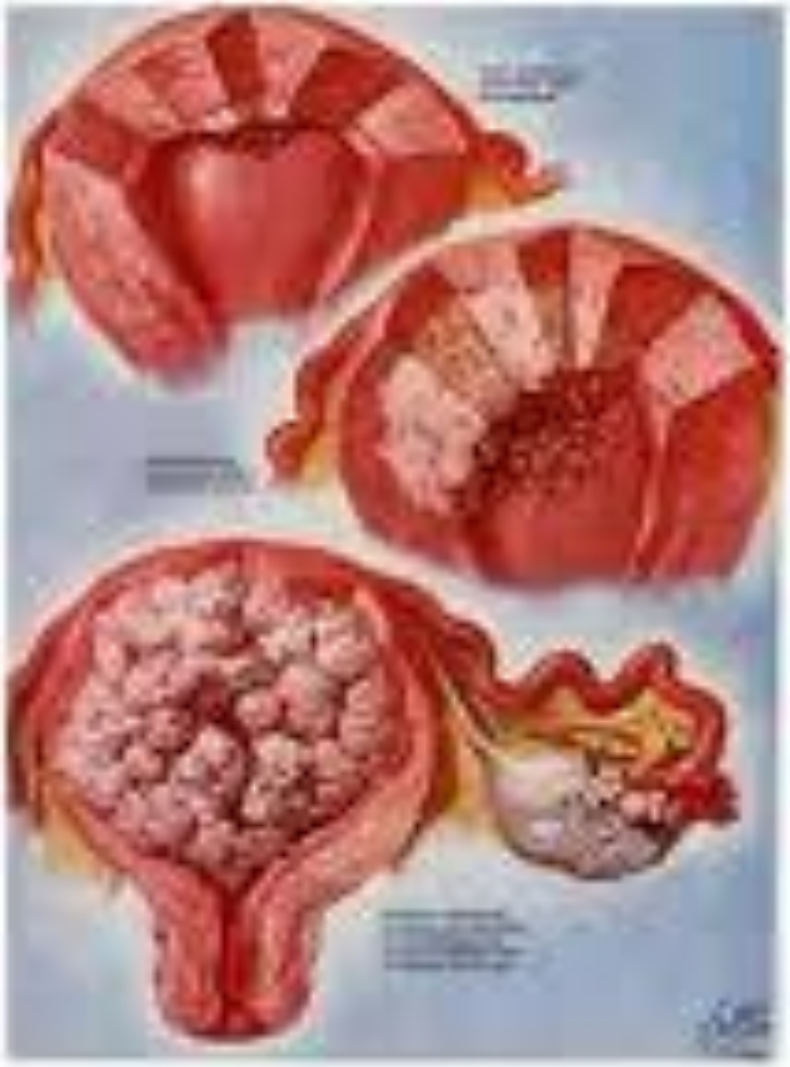
Endometrial Carcinoma

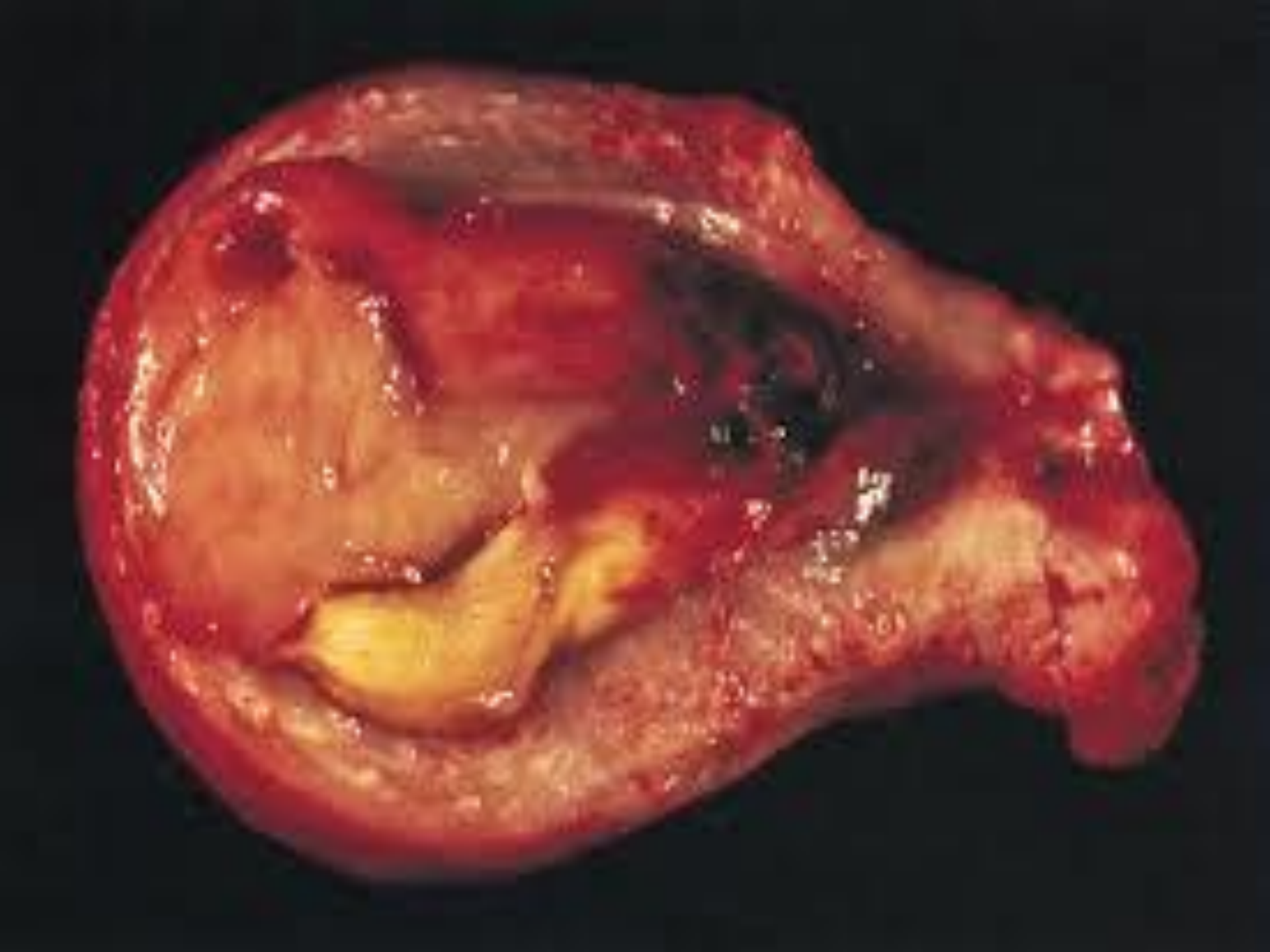
Etiology

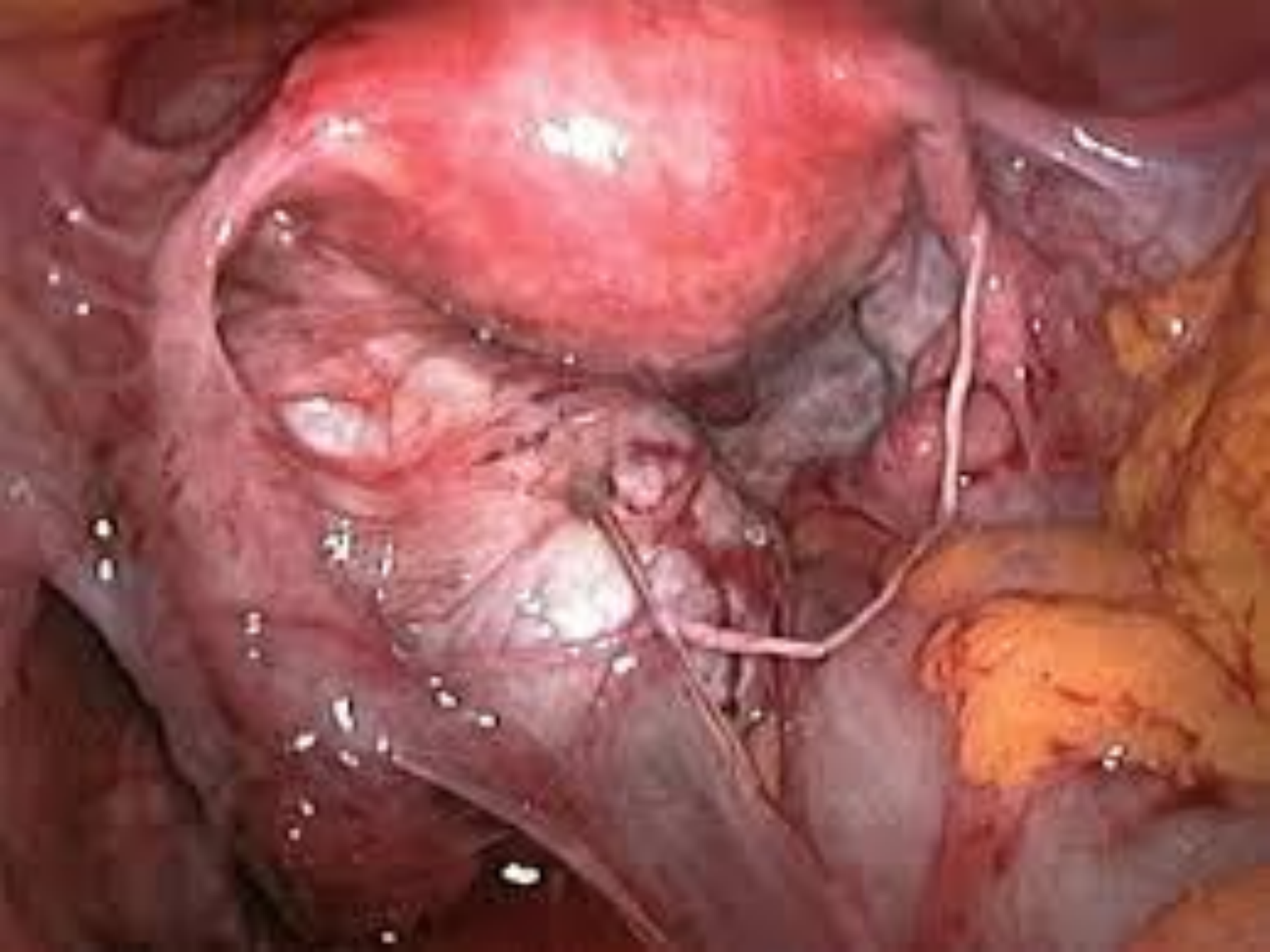
- Unopposed estrogen hypothesis: exposure to unopposed estrogens

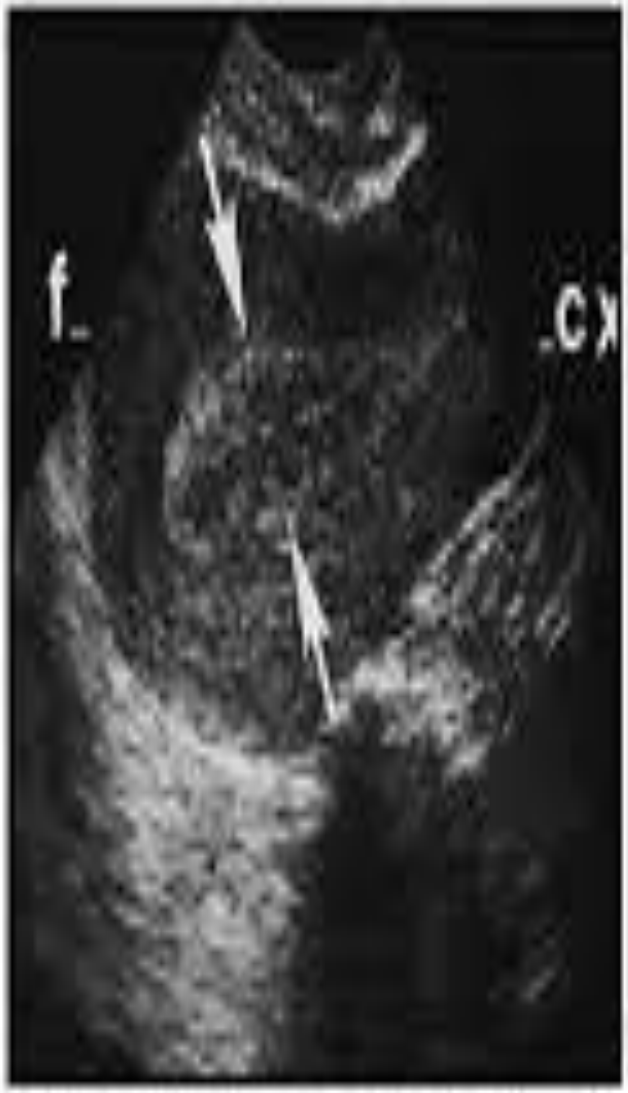
Pathology

- Spreads through uterus, fallopian tubes, ovaries and out into peritoneal cavity
  - Metastasizes via blood and lymphatic system

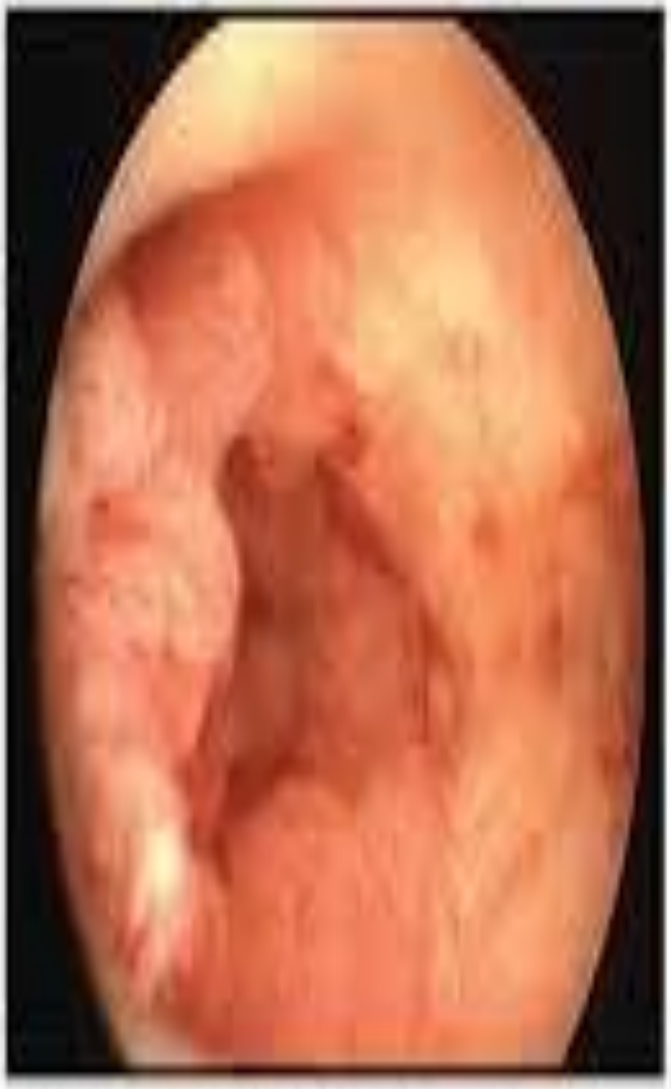




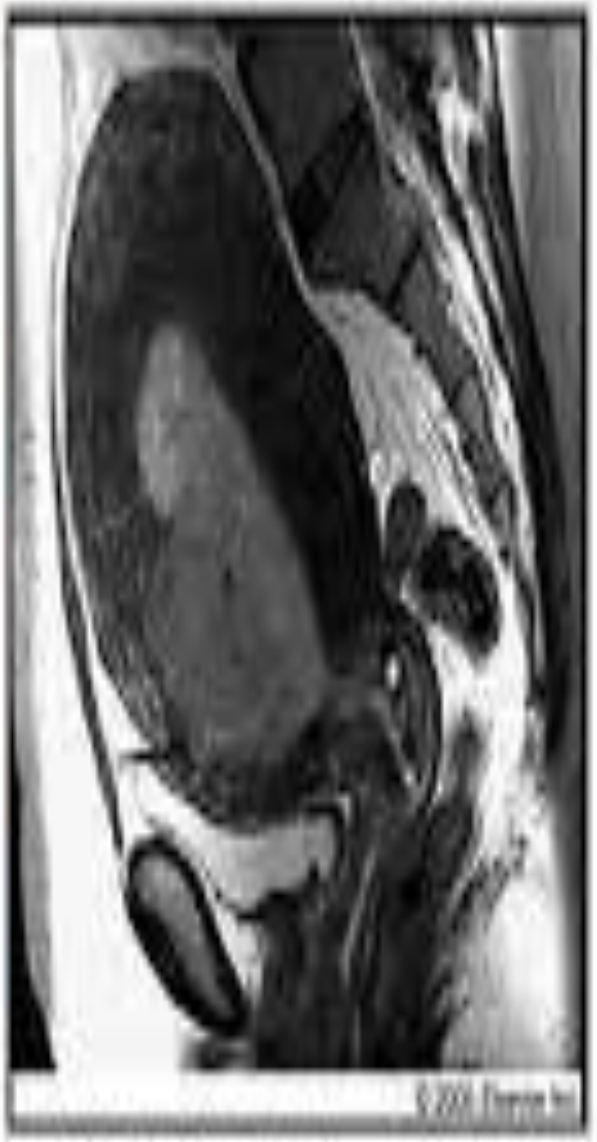




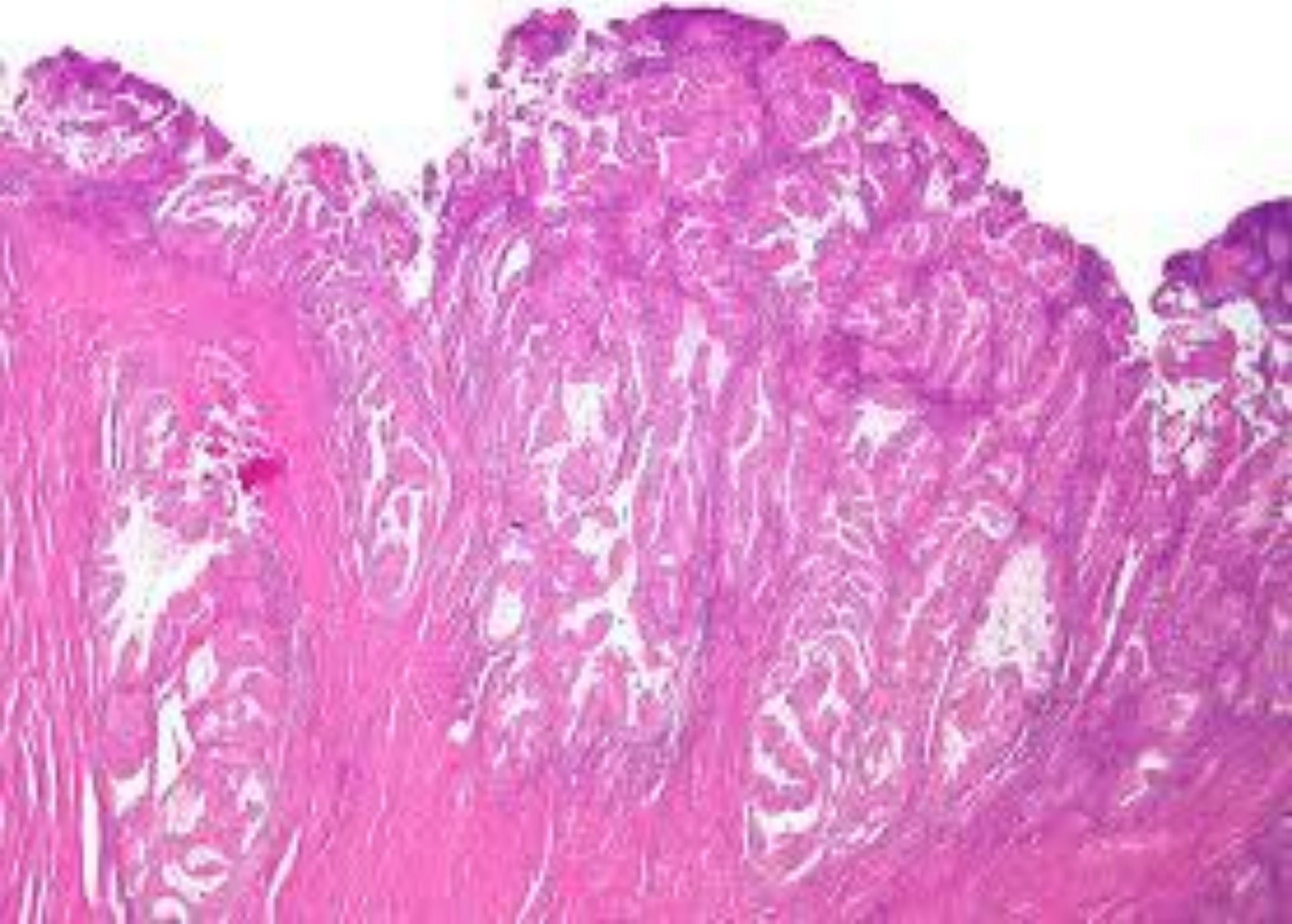
Ultrasound scan showing thickened endometrium (between arrows) (image from GLOWM.com)

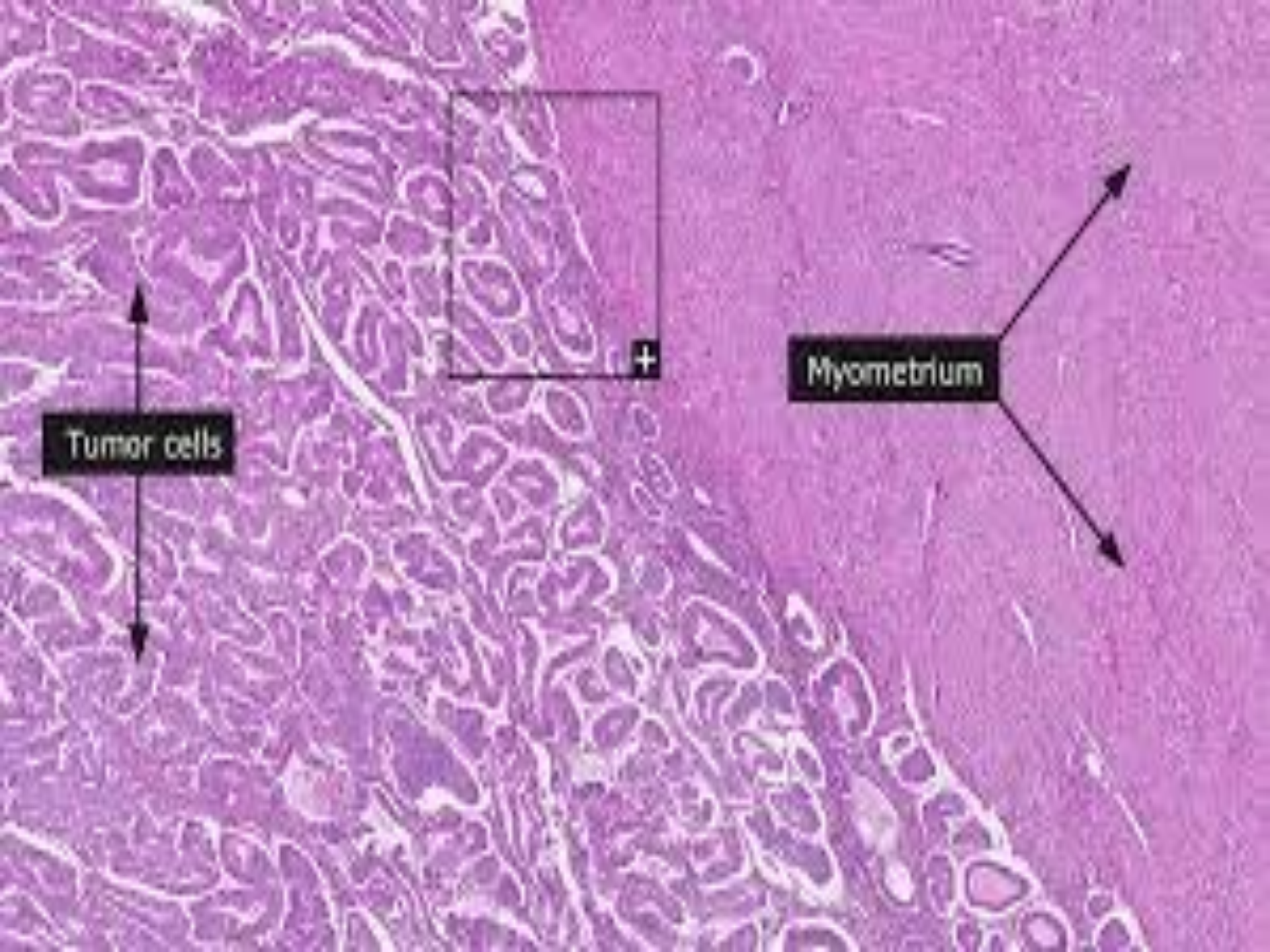


Hysteroscopic view of a malignant endometrial growth (image from J Gynec Endosc Surg)



Pelvic MRI scan showing a large endometrial cancer (image from Elsevier Imaging Consult)




Tumor cells
Myometrium

Thank you

Any questions