Female Genital System Cervix and GTD.

DR.EMAN KREISHAN

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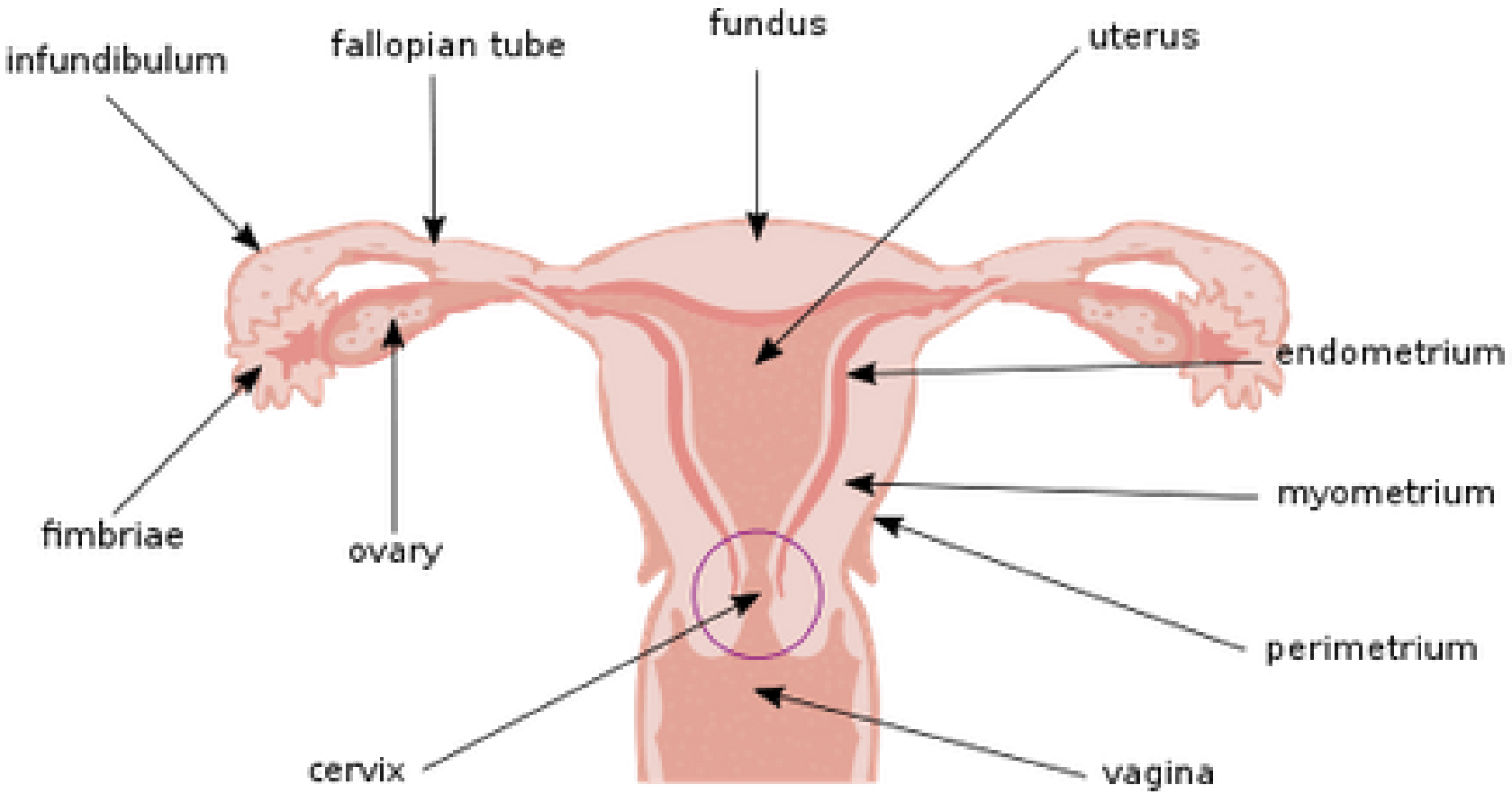
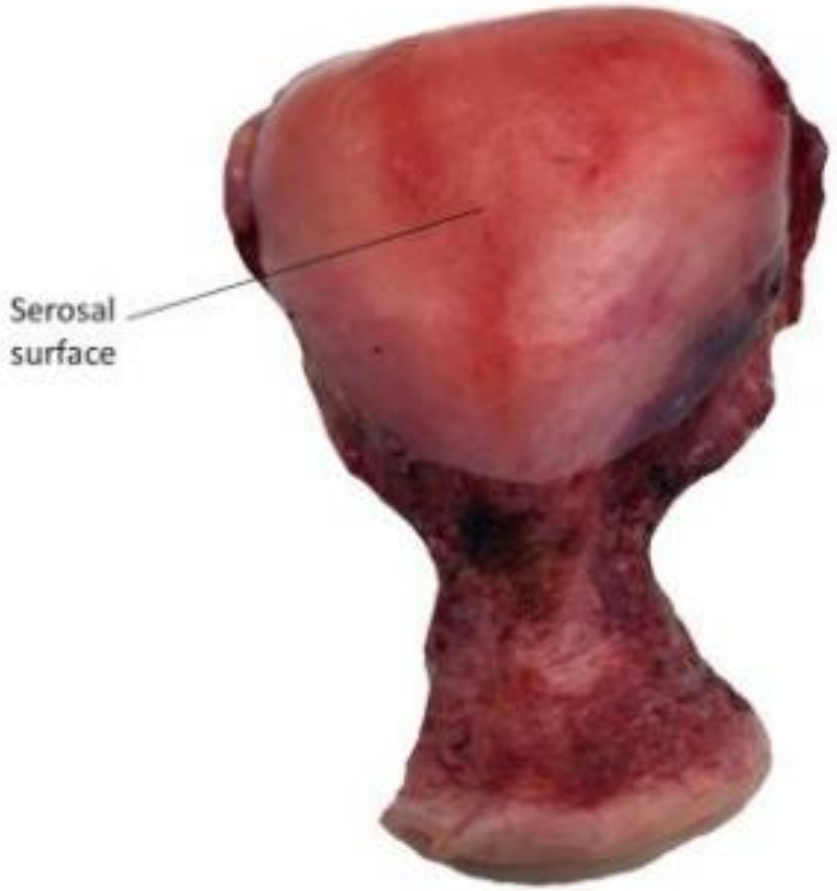
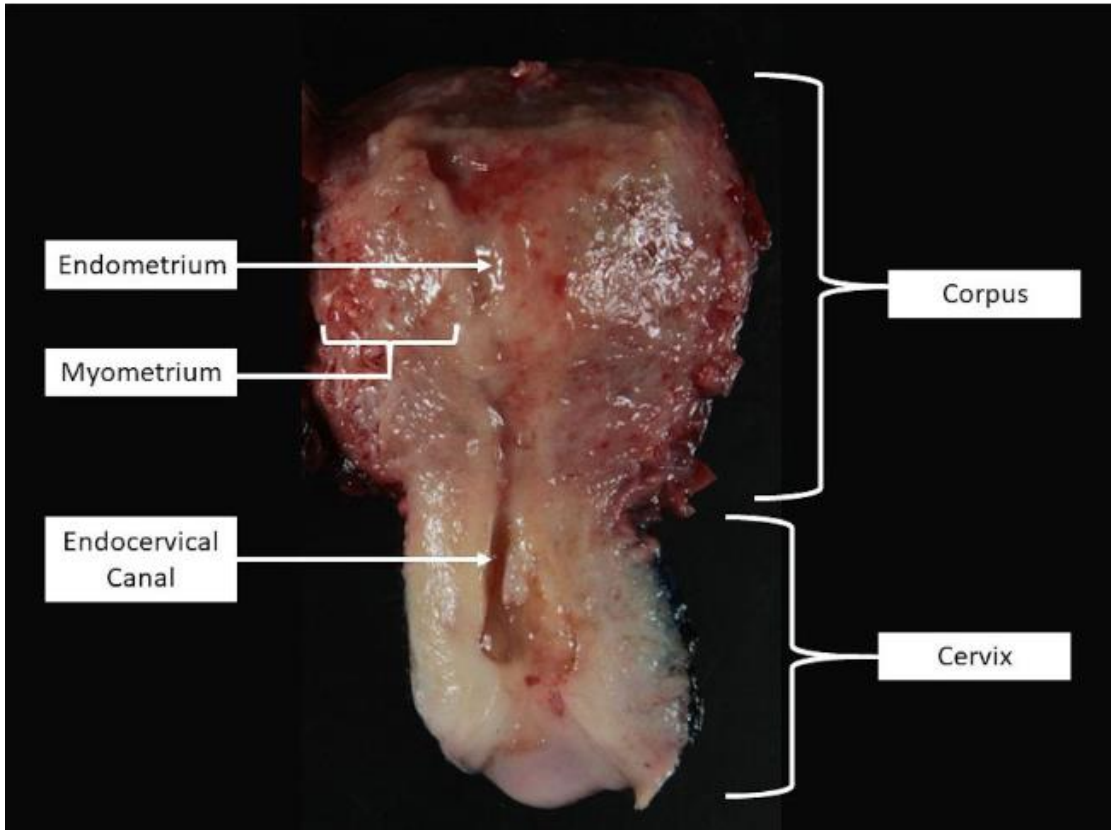
Anatomy

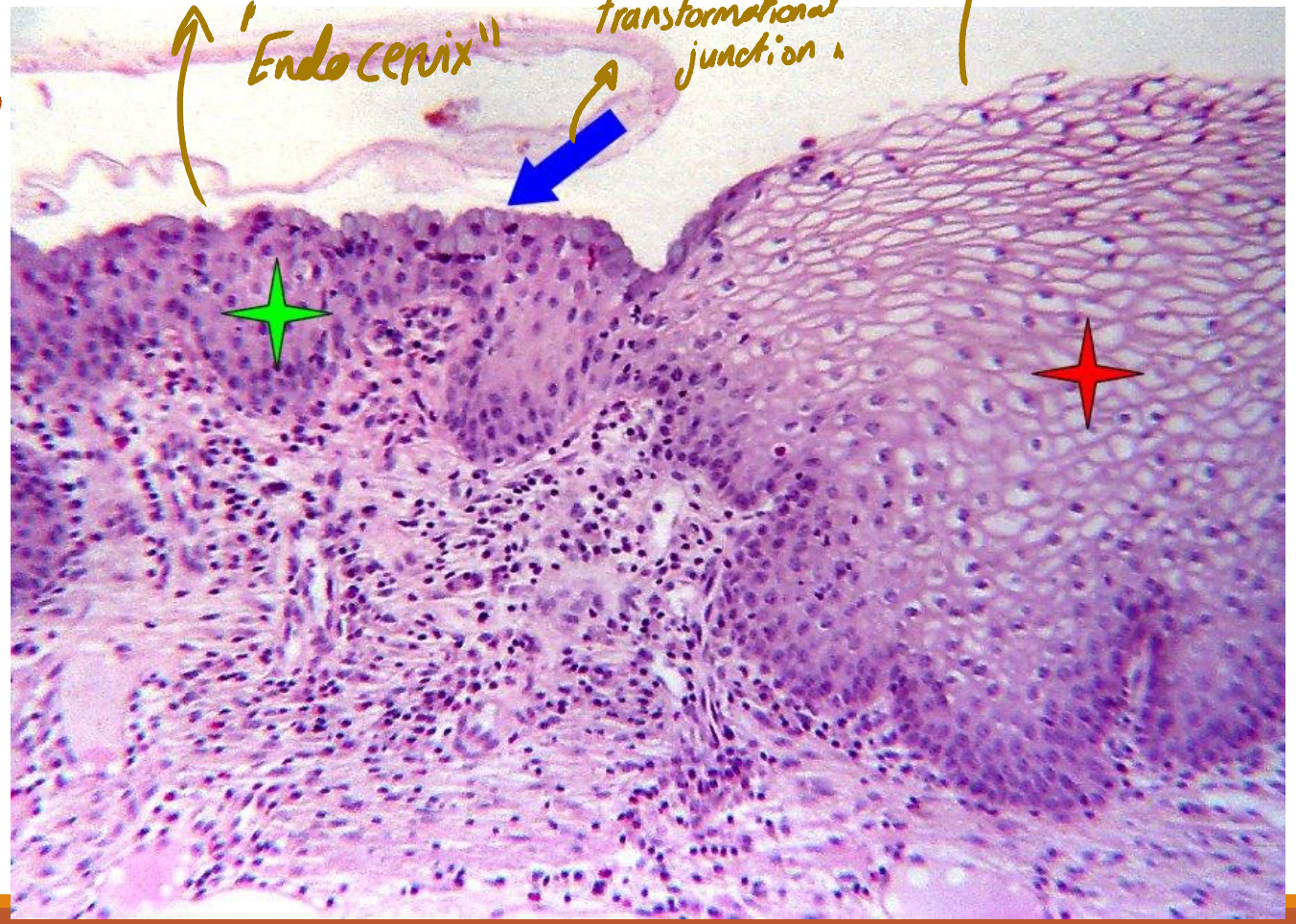

infundibulum
fallopian tube
fundus
uterus
endometrium
myometrium
perimetrium
fimbriae
ovary
cervix
vagina

Gross anatomy





Histology



during puberty → "squamus metaplasia in transformational junction"

vic consist from part :-
upper "Endocervix"
lower "Ectocervix"

I. Cervix: Cervicitis.

- ✓ inflammation of the cervical epithelium and stroma, with varying degrees of cellular infiltration.
- ✓ Clinically characterized by: purulent vaginal discharge.
- ✓ Cervicitis can be sub classified as:

- ❖ infectious: Chlamydia trachomatis, Ureaplasma urealyticum, T. vaginalis, Neisseria gonorrhoeae, HSV-2.

- ❖ Noninfectious. autoimmune + iCUD

II. NEOPLASIA OF THE CERVIX

- Most tumors of the cervix are of epithelial origin and are caused by oncogenic strains of HPV and commonly arising from the transformation zone.

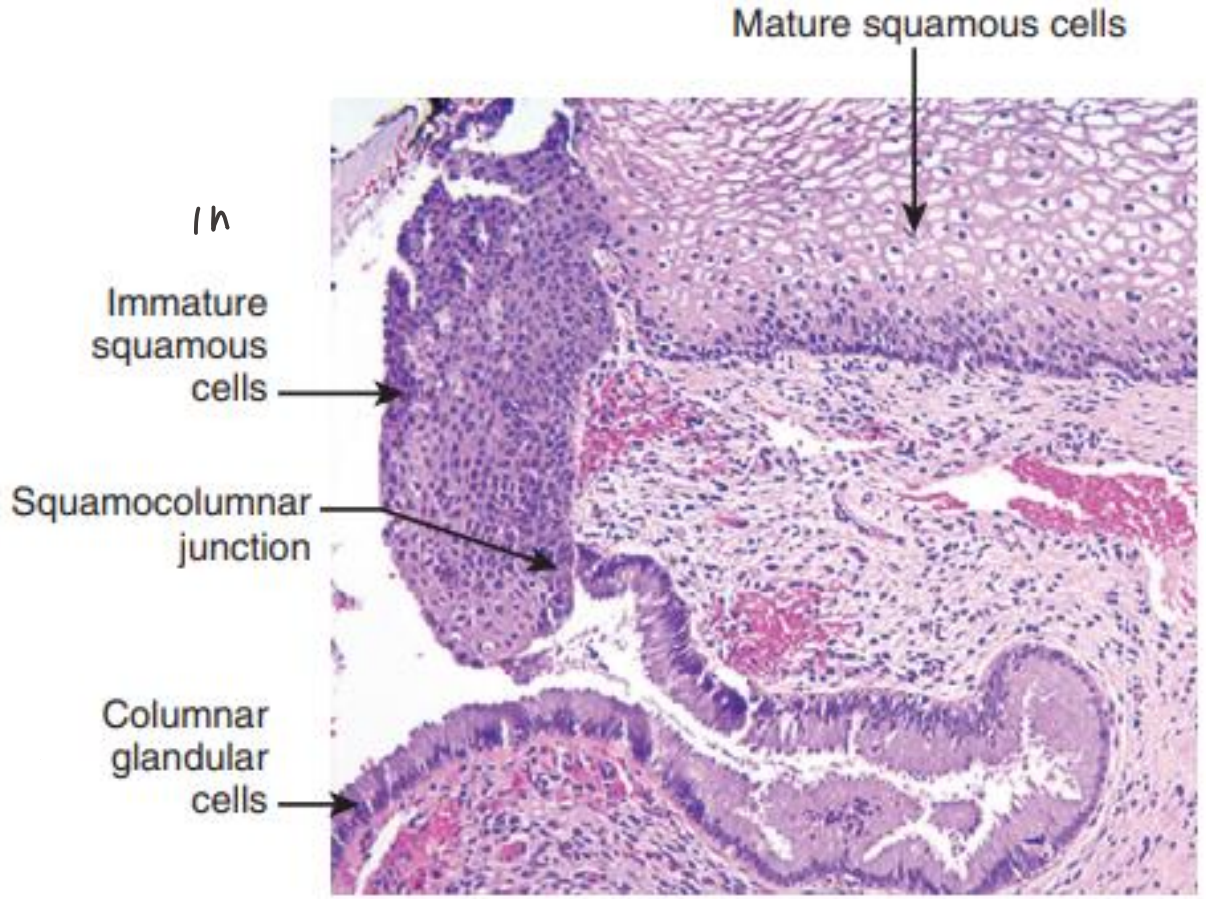
- HPV, the causative agent of cervical neoplasia

has a tropism for the immature squamous cells

of the transformation zone

During puberty squamus area extend upward and metaplagio to form immature squamer cell

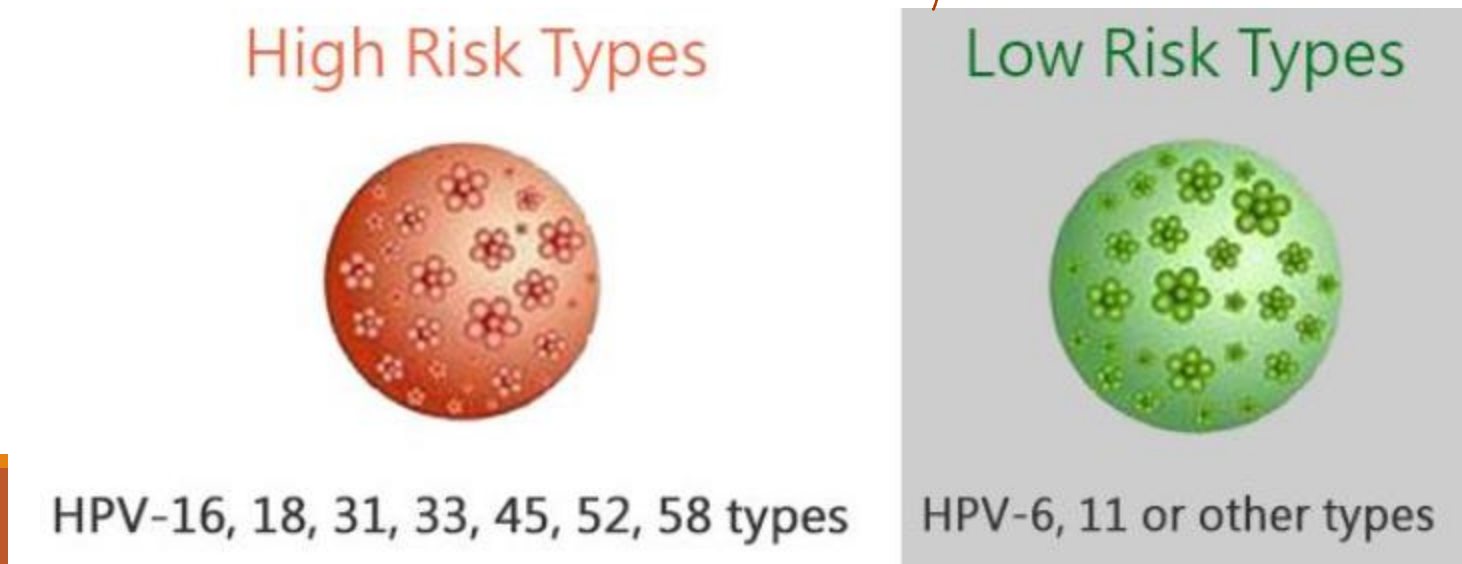
where HPV affected in transformation junction.



Then?

- Most HPV infections are transient and are eliminated within months by the host immune response.
- A subset of infections persists leading to:

- cervical intraepithelial neoplasia (CIN).
- Invasive cervical carcinoma.

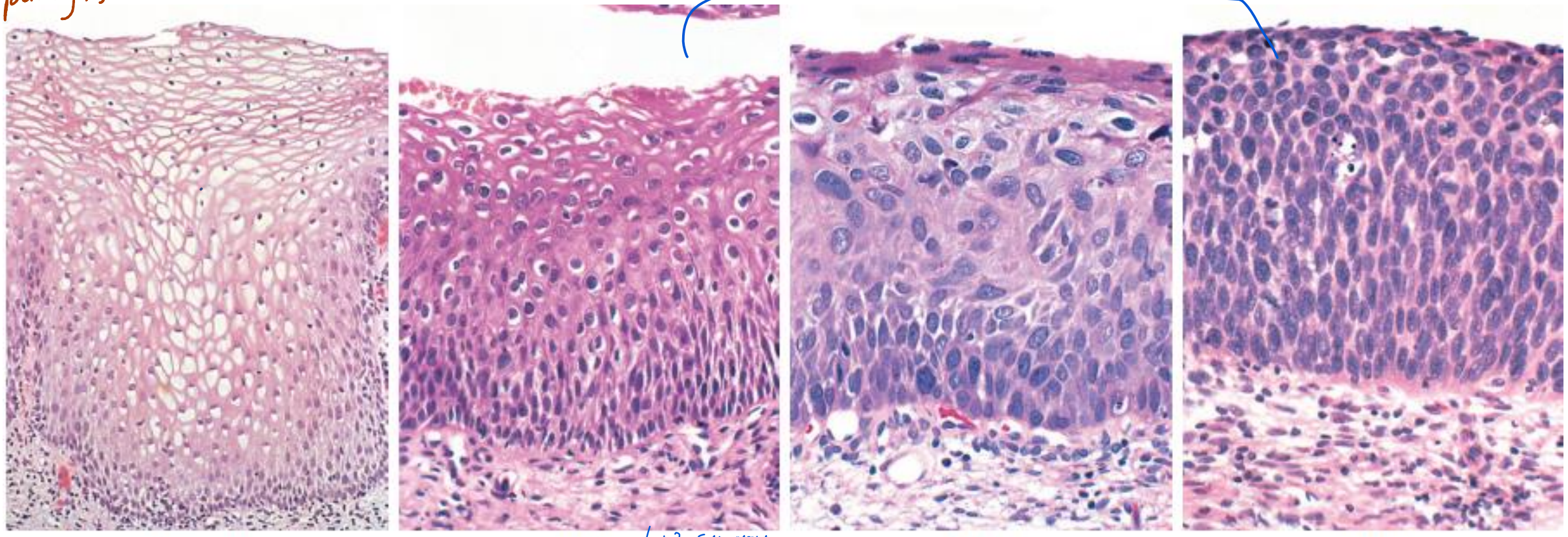


Cervical intraepithelial neoplasia (CIN)

- Precancerous squamous proliferative lesion with variable thickness nuclear atypia and varying degrees of cytoplasmic maturation

- Graded depending on the extent of epithelial involvement:
- CIN I: Mild dysplasia (involves a third or less of thickness)
- CIN II: moderate dysplasia (involves 2/3 of thickness).
- CIN III: severe dysplasia (involves full thickness) → carcinoma in situ

CIN


polarity, small nuclear
significant polymorphism, hyperchromatic
nearly all thickning or all
Normal
LSIL
low squamous intraepithelial lesion
HSIL
high
HSIL

Clinical features, diagnosis and treatment

CIN I(LSIL):

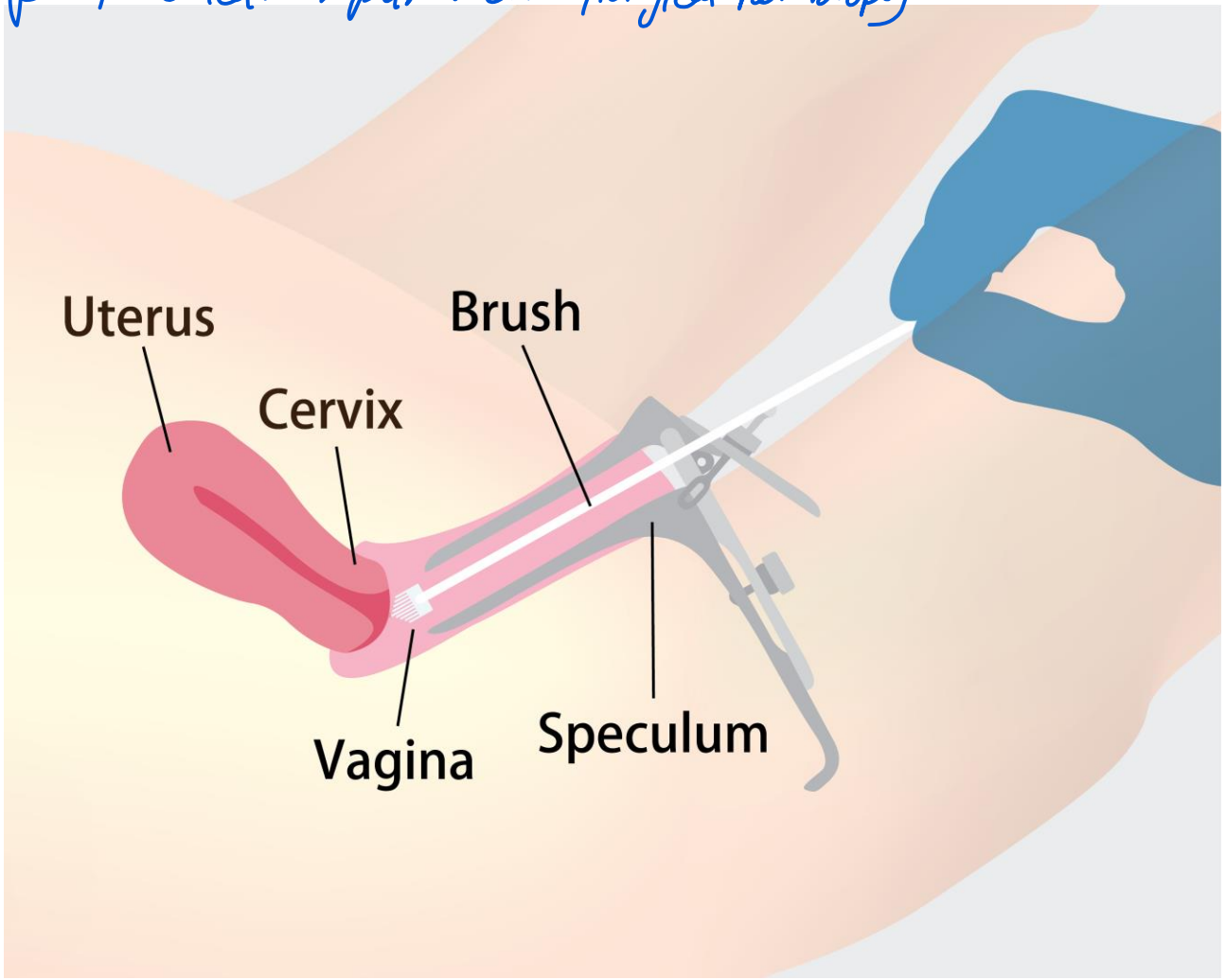
remove all uterus

- Asymptomatic : Found incidentally on Pap smear screening, cervical biopsy or hysterectomy
- Diagnosis: Pap smear or Cervical biopsy.
- Management : colposcopy with careful observation.

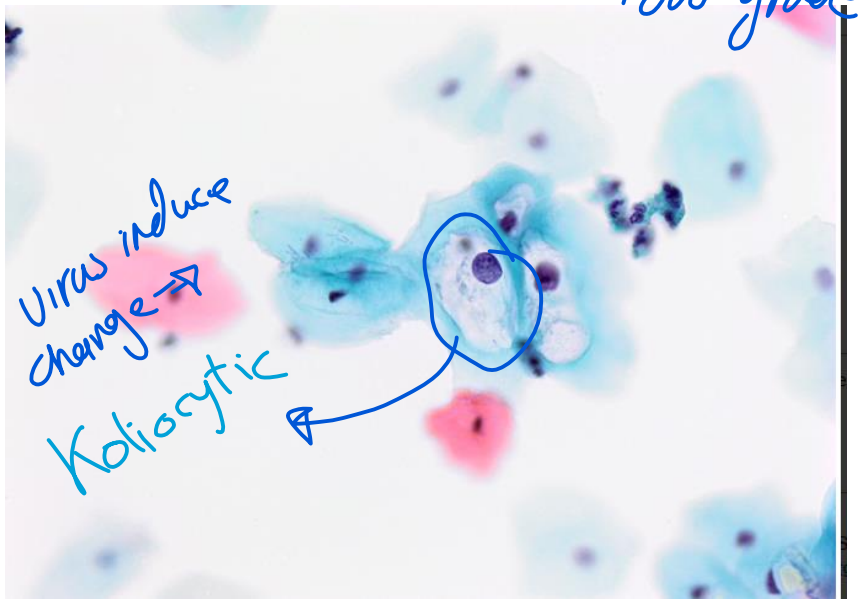
CIN II, III (HSIL):

- Asymptomatic or presented as abnormal colposcopy.
- Diagnosis: Pap smear or Cervical biopsy.
- Management: Often treated with local excision. → high rate convert to cancer

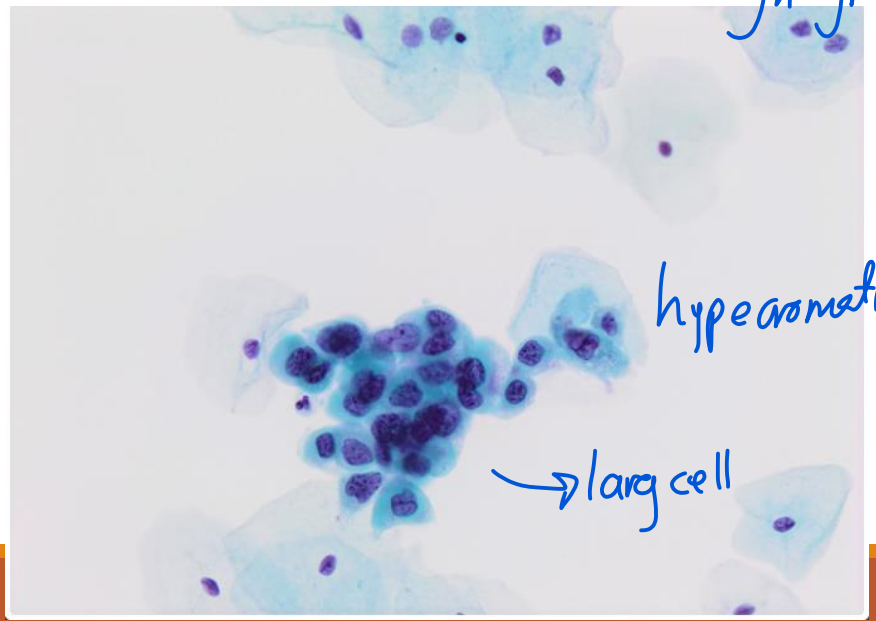
Pap smear => part from cytological not biopsy



low grade



high grad



Invasive Carcinoma of the Cervix

- Progression of SIL to invasive carcinoma is variable & unpredictable. (smoking is a risk factor).

- Most often is seen in women who have never had a Pap smear or who have not been screened for many years.
- Most common form is SCC 75%, adenoCa. & adenosquamous (mixed) Carcinoma 20%, & neuroendocrine Carcinoma 5%.
- All cases are associated with HPV infection.

Table 22.2 Natural History of Squamous Intraepithelial Lesions With Approximate 2-Year Follow-Up

"clear"

Lesion	Regress	Persist	Progress
LSIL	60%	30%	10% to HSIL
HSIL	30%	60%	10% to carcinoma[a]

HSIL, High-grade squamous intraepithelial lesion; *LSIL*, low-grade squamous intraepithelial lesion.

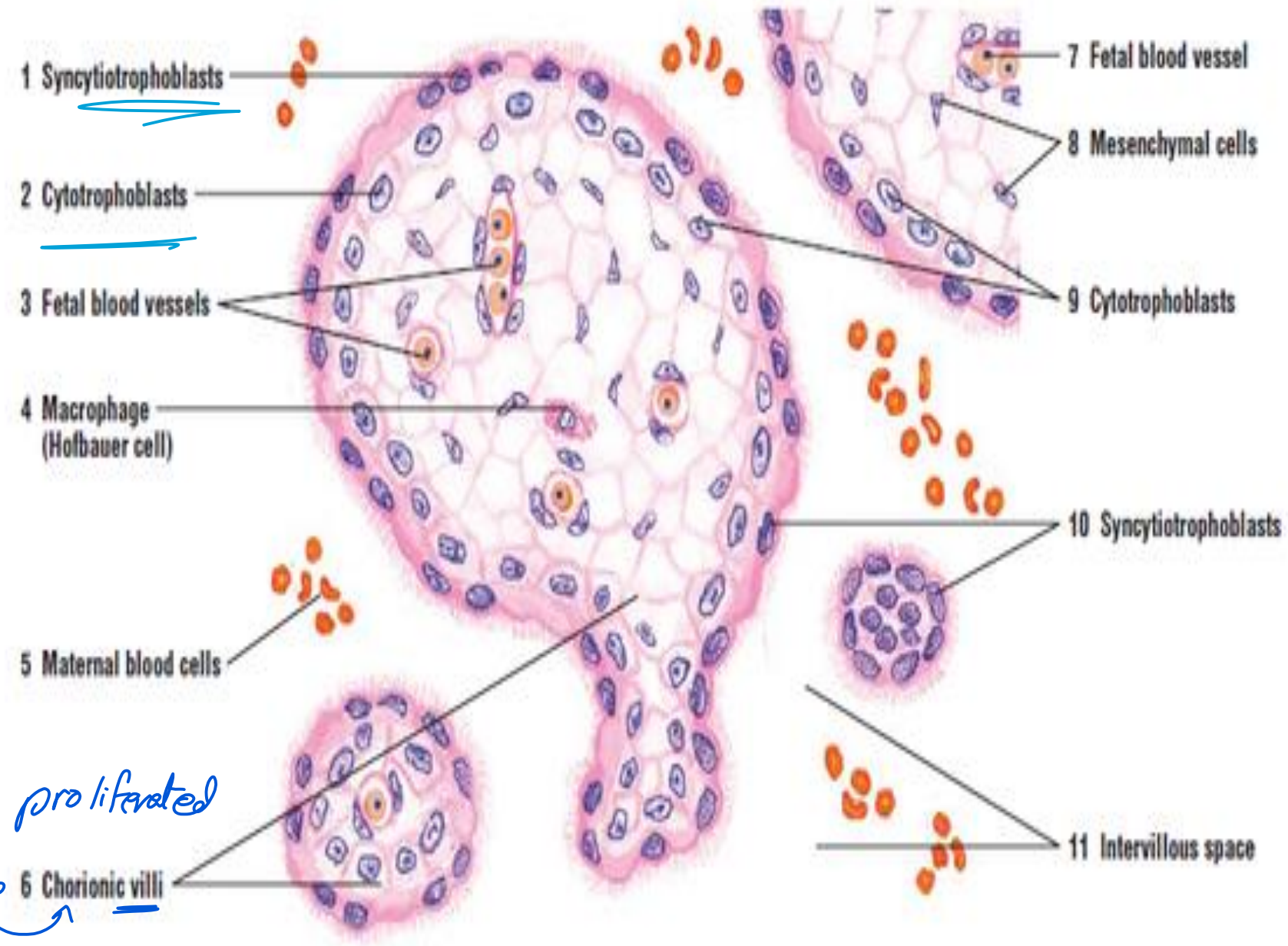
[a]Progression within 2 to 10 years.

during pregnancy

Gestational trophoblastic disease:

- Hydatidiform Mole .
- Gestational Choriocarcinoma

Placenta histology



Placenta grossly

Gestational trophoblastic disease (GTD)

- An abnormal proliferation of fetal trophoblast cells. (normal cells of placenta in pregnancy).

- All GTD elaborate human chorionic gonadotropins (hCG) → detected in the blood & urine at levels higher than those found during normal pregnancy. So it used as a tumor marker for diagnosis and follow up.

also elevated in twins pregnant

1. Hydatidiform Mole

- An abnormal gestational process due to abnormal fertilization with an excess of paternal genetic material.
- Incidence of complete hydatidiform mole is about 1 to 1.5 per 2000 pregnancies.
- Most common before 20 & after 40 years.
- History of Mole increases the risk for molar disease in subsequent pregnancies.

- Two forms:

Complete mole.

Partial mole.

A. Complete Hydatidiform Mole

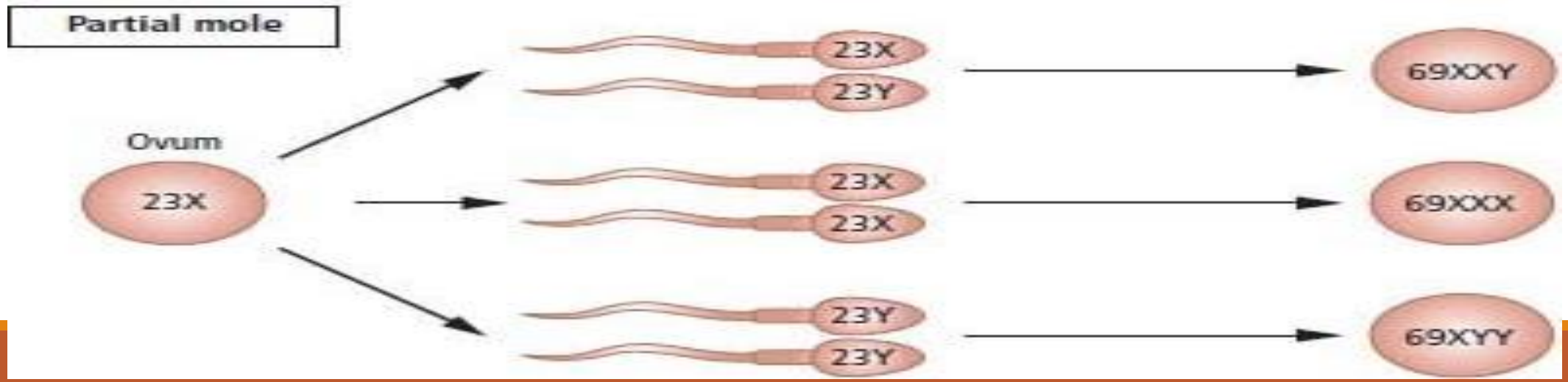
an empty egg fertilized by two spermatozoa (or a diploid sperm) , diploid karyotype containing only paternal chromosomes.

Complete mole are not compatible with embryogenesis & **does not contain fetal parts.** The chorionic epithelial cells are diploid (46,XX or, uncommonly, 46,XY).

B. Partial Hydatidiform mole.

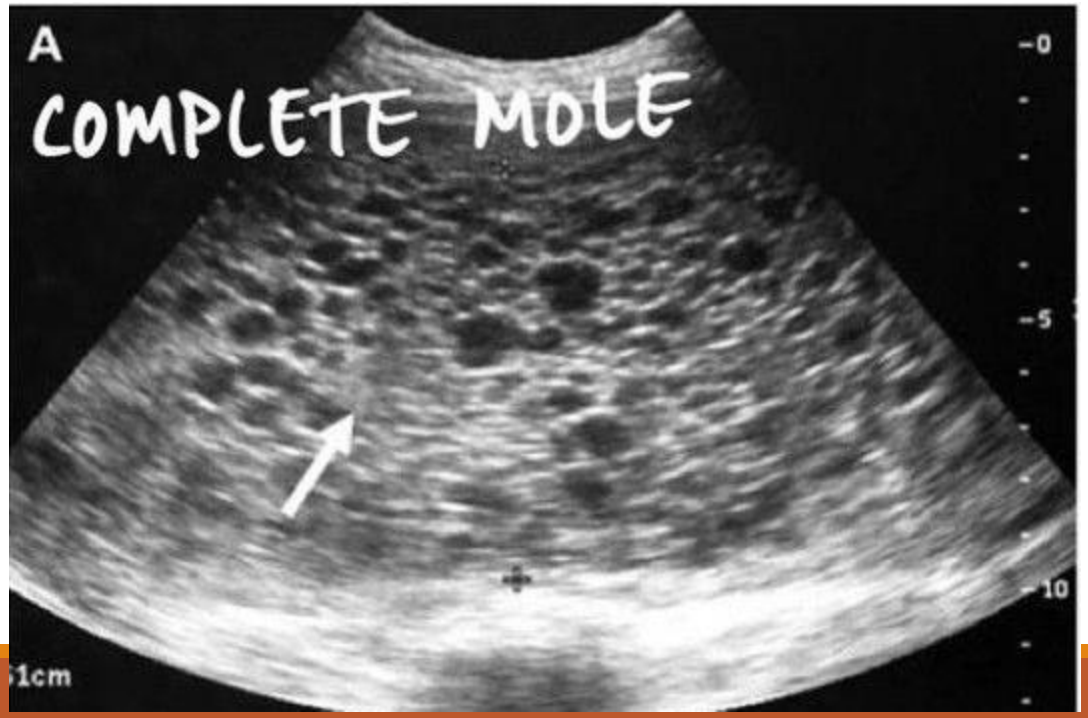
- A normal egg is fertilized by two spermatozoa (or a diploid sperm) , triploid karyotype with a dominance of paternal genes.

Partial mole is compatible with early embryo formation → may contain fetal parts & some normal chorionic villi. Chorionic epithelial cells almost always triploid (e.g., 69,XXY)



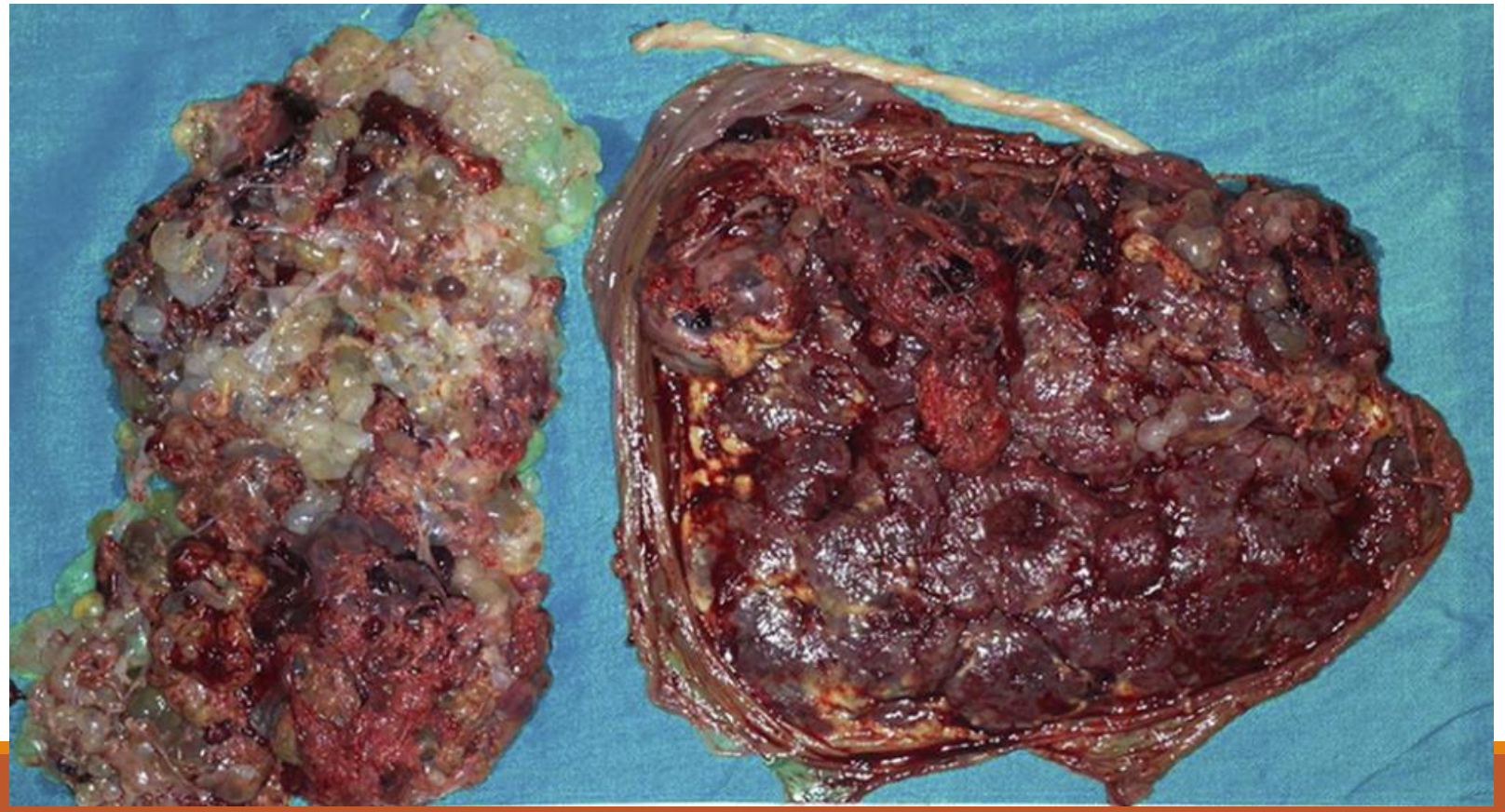
Clinical presentation

- Hyperemesis. → vomiting
- Elevation of hCG in maternal blood & no fetal heart sounds.
- Large for dates fetus on ultrasound examination
- snow storm appearance on ultrasound



Gross morphology

Uterine cavity is expanded by friable mass (**Grape-like villi**) composed of thin-walled, cystically dilated chorionic villi .



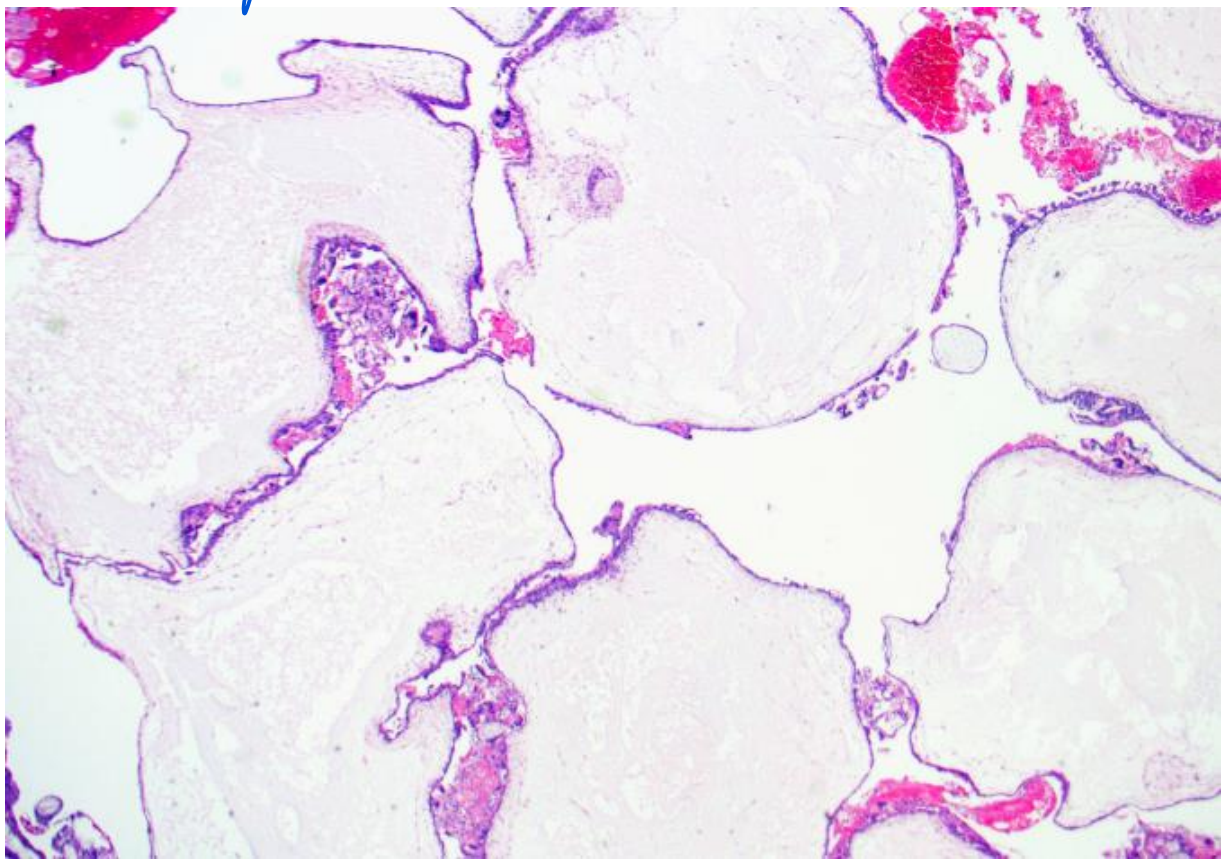
Histology

Complete hydatidiform mole :Diffuse villous enlargement and Circumferential trophoblastic hyperplasia.

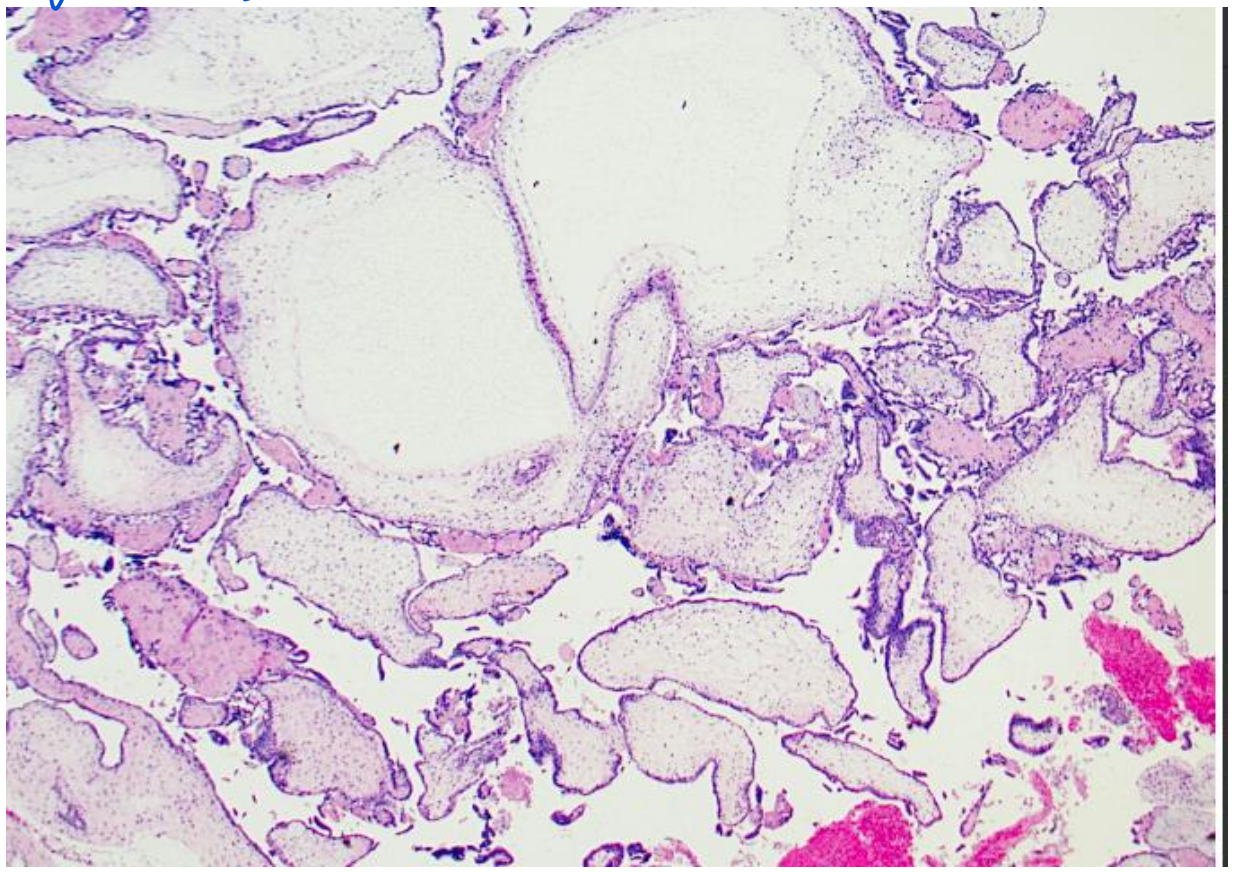
Partial hydatidiform mole: 2 villous populations seen: large hydropic and small fibrotic villi

Microscopic features

Complet



partialy



Treatment & prognosis

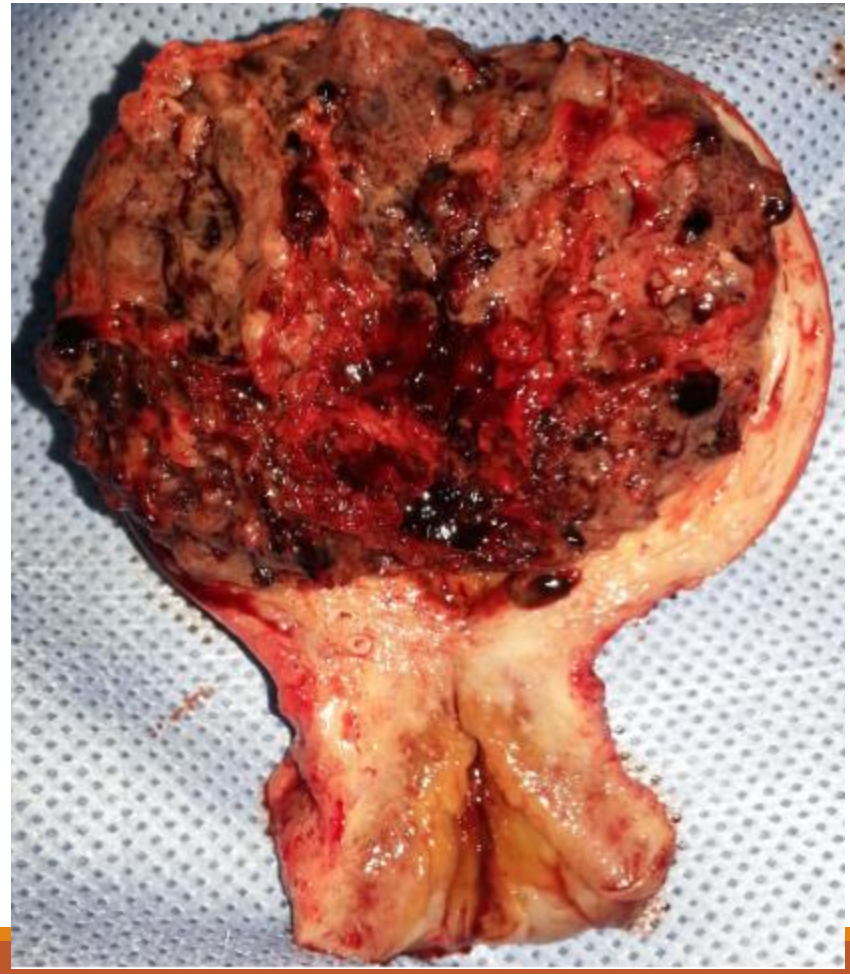
- Treatment : surgical evacuation of the uterine cavity & close follow up with serum hCG.
- The majority of moles do not recur after thorough curettage, 10% of complete moles are invasive
- No more than 2-3% give rise to choriocarcinoma (usually complete, rarely partial).
- So partial mole has much better prognosis

2. Gestational Choriocarcinoma

- Aggressive form of gestational trophoblastic neoplasia composed of syncytiotrophoblast, cytotrophoblast and intermediate trophoblast.
- Most common before 20 & after 40 years.
- 50% result from complete moles; 25% after an abortion, 25% after an apparently normal pregnancy
- Serum human chorionic gonadotropin (hCG) is a reliable tumor marker
- High cure rates with chemotherapy.
- Clinically:
- - vaginal bleeding.
- - Can present initially with metastatic disease

Gross morphology

•Dark red, solid, friable tumor, with areas of hemorrhage and necrosis



Microscopic features

•Solid sheets of atypical syncytiotrophoblast, cytotrophoblast and intermediate trophoblast.

•Hemorrhage and necrosis.