

Endometriosis

→ Presence of ectopic benign endometrial gland and stroma outside of the uterus.

Types

- superficial peritoneal
- ovarian endometriosis
- deep infiltrating endometriosis

Sites: → Ovaries, Pouch of Douglas, Fallopian tube, uterine ligament, Periumbilical tissue, Laparotomy scars.

PATHOGENESIS:

two main sources

- from the uterine endometrium
- from outside the uterine

Main theories

- ① Regurgitation theory: due to Retrograde menstruation
- ② Benign metastasis theory: spread through blood & lymph
- ③ Metaplastic theory: embryonic development coelomic epithelium
 ↓
 Mullerian duct
 ↓ develop
 endometrium
 ∴ Metaplasia of coelomic epithelium.

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④ Extracutaneous stem theory: endometrial tissue is ectopic.

character of endometrial tissue that undergo endometriosis.

⇒ ↑ level of proinflammatory and Angiogenic factor
eg: PGE₂, bFGF (Basic Fibroblast Growth Factor)

⇒ Secretion of Aromatase by endometrial stromal cells
⇒ ↑ local estrogen production from Androgens.

∴ These two factors ↑ survival of endometrial tissue in foreign location.

t/t → COX2 Inhibitor.

MORPHOLOGY:

⇒ Lesions bleed periodically

⇒ Cystic nodules with Red-blue to yellow brown.

⇒ Endometriosis of ovary → chocolate cyst

↓
ovaries may be distorted by cystic space

filled with dark brown fluid.

Repeated hemorrhage from large cysts contains inspissated chocolate cysts.

MICROSCOPY:

shows both endometrial glands and stroma with or without hemosiderin-laden macrophages.

C/F

- ⇒ dysmenorrhea
- ⇒ pelvic pain
- ⇒ dyspareunia
- ⇒ Irregular disturbance
- ⇒ dysuria.

Consequence → Infertility is 30-40%.

Endometrial hyperplasia:

- Abnormal proliferation of endometrial gland relative to stroma → ↑ thickness of the endometrium.

- Imp. cause of abnormal uterine bleeding
- Precursor of endometrial carcinoma. (soil for it)

Pathogenesis:

⇒ ↑ levels of estrogen → hyperplasia.

sources → endogenous: obesity
menopause

cortical steroid hyperplasia
chronic anovulation.

Exogenous: Administration of estrogenic substances

Mutches P⁵³ gene

CLASSIFICATION:

- ① Endometrial hyperplasia without Atypia
- ② Endometrial atypical hyperplasia.

I without Atypia progresses to Adenocarcinoma
→ proliferation of endometrial gland
of irregular size and shape without significant
cytological Atypia.

MORPHOLOGY:

→ Highly thickened - with spongy cysts.

MICRO

→ 3 criteria:

- ↑ endometrial gland to stroma Ratio.
- cystically dilated endometrial gland.

Irregular
distribution of gland

Glands lined
by simple epithelium.

- Uniform distribution of nuclear features.

→ High level of estrogen cause Round nuclei.

II with Atypia progresses to endometrial carcinoma.

ENDOMETRIAL CARCINOMA:

- ↑ number of endometrial glands compared with the stroma.
- occurs as expansion of mutated gland.
- genetic mutation of $CTNB1$, $PTEIN$, $KRAS$.

MORPHOLOGY:

- ⇒ focal thickening resembling a polyp.

MICROSCOPY:

- ⇒ crowded Aggregates of cytologically atypical tubular or branching gland.
- show both architectural and cytological Atypia.

- ⇒ Gland to stroma Ratio ↑ → back to back Arrangement.

- ⇒ nuclear Atypia → Alteration in nuclear size, shape of