Pathology - Topic 29 - Female Genital Tract

Cervicitis

- Cervicitis is inflammation of the cervix.

Infective and Noninfective Causes

- Infective causes include sexually transmitted organisms and other disturbances of the local microbial environment.
- Chlamydia trachomatis and Neisseria gonorrhoeae are important infectious causes; Trichomonas vaginalis, primary genital herpes and Mycoplasma genitalium are also associated with cervicitis.
- Many cases lack an identified pathogen, and chemical irritation or altered vaginal flora can contribute in selected settings.

Inflammatory and Reactive Morphology

- Acute inflammation contains neutrophils and may produce discharge and epithelial injury.
- Chronic inflammation contains lymphocytes and plasma cells in the cervical stroma.
    - Obstruction of mucus glands during repair can form benign nabothian cysts.
- Reactive epithelial changes must be distinguished from dysplasia.
- Cytological atypia in an inflamed sample requires assessment of the whole pattern rather than one enlarged nucleus.

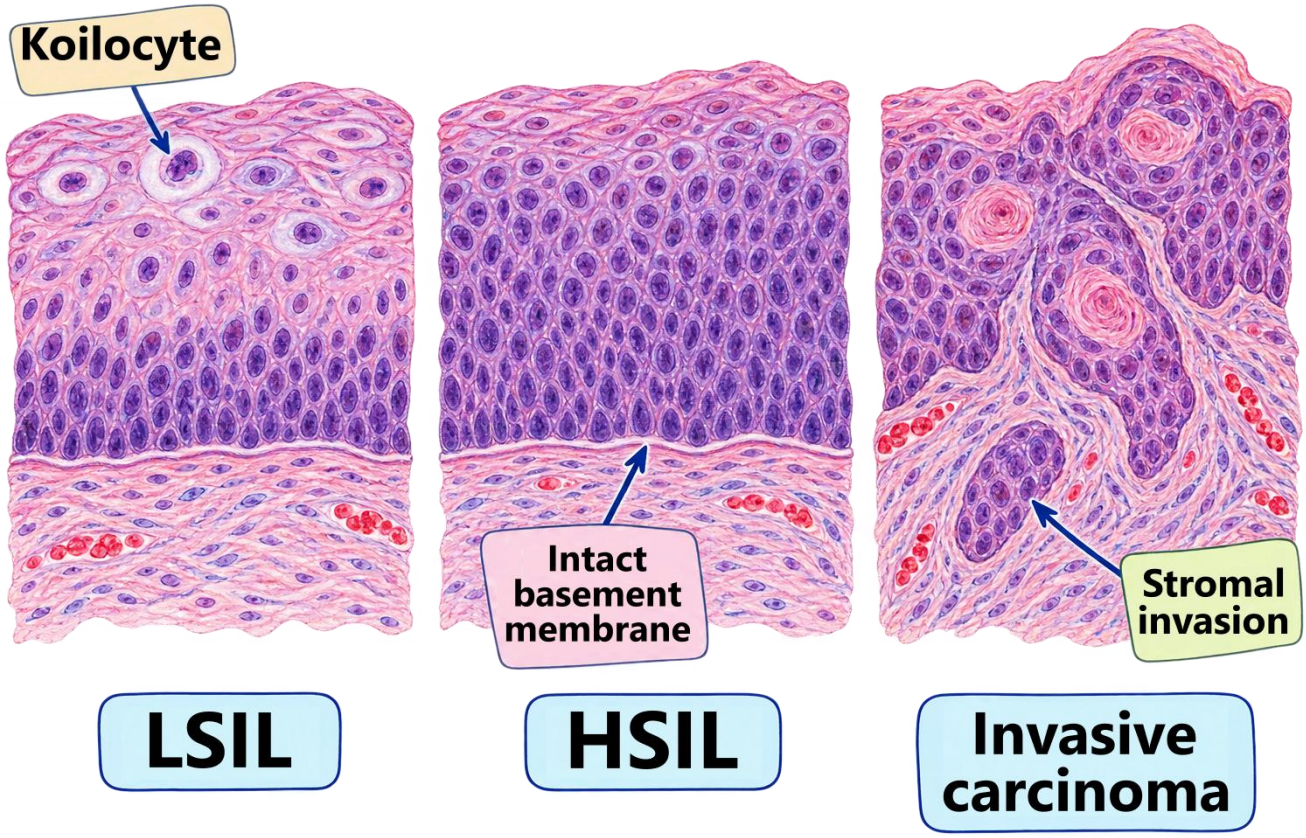
Clinical Findings and Etiological Testing

- Mucopurulent endocervical discharge and easily induced cervical bleeding are important examination findings.
- Nucleic-acid amplification tests for chlamydia and gonorrhoea support the aetiological assessment, with evaluation for other infections and upper genital-tract involvement according to the clinical findings.

Carcinoma of the Cervix

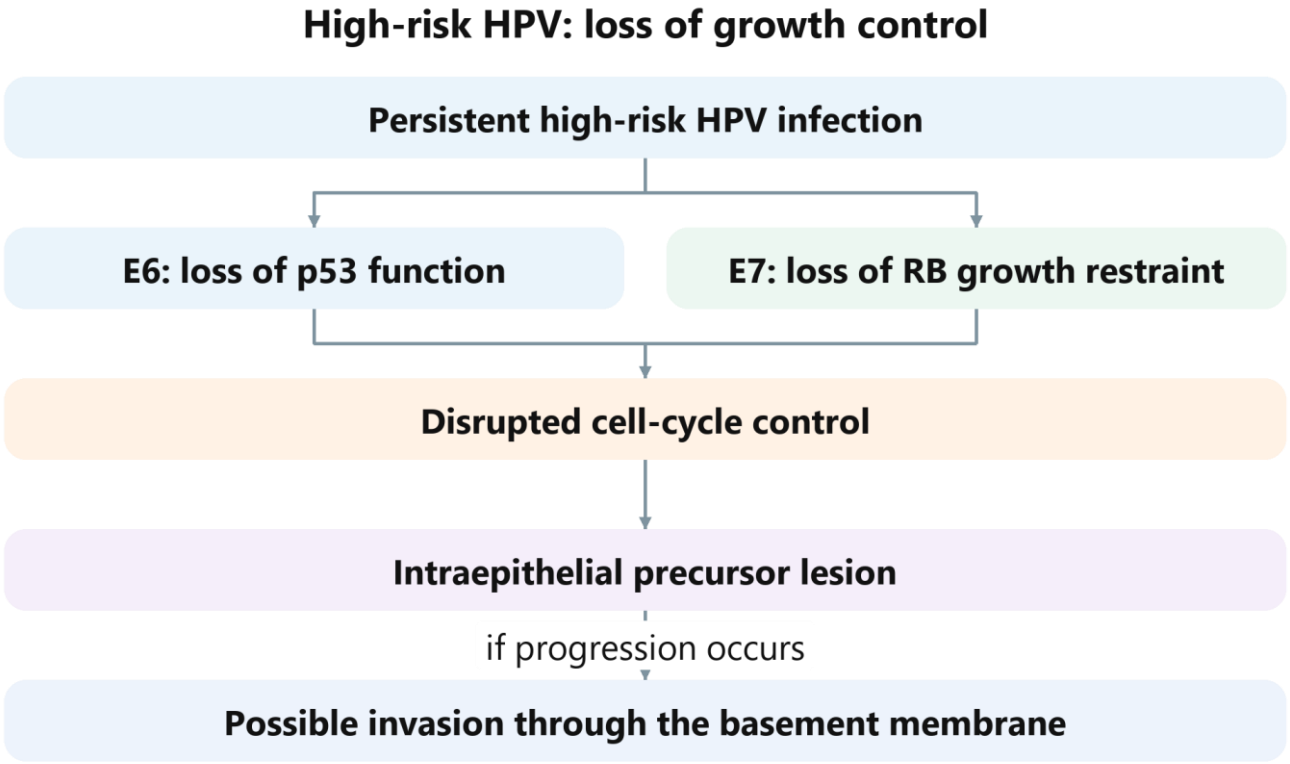
- Cervical carcinoma is a malignant epithelial tumour of the cervix, most often associated with persistent high-risk HPV infection.

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Cervical squamous intraepithelial lesions and stromal invasion

- Persistent infection with high-risk human papillomavirus is the central cause of most cervical carcinomas.
- Many HPV infections clear, so infection alone does not mean that cancer will develop.
- The transformation zone is especially susceptible because it contains actively changing epithelium near the squamocolumnar junction.



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- High-risk viral proteins interfere with normal cell-cycle control. E6 promotes loss of p53 function, while E7 disrupts RB-related growth restraint.

- Smoking, immunosuppression and failure to access effective screening increase the risk that persistent infection progresses. HPV-associated cancer usually develops through a prolonged precursor phase.

Squamous Precursors and Invasive Carcinoma

1. Low-Grade Squamous Intraepithelial Lesion

- Low-grade squamous intraepithelial lesion broadly corresponds to CIN 1.
- Atypical immature cells mainly occupy the lower third of the squamous epithelium.
- Koilocytes have enlarged irregular nuclei with surrounding cytoplasmic clearing.
- Many low-grade lesions regress, particularly when HPV infection clears.
    - Koilocytic change supports HPV-related injury but must be distinguished from non-specific cytoplasmic vacuolation.

2. High-Grade Squamous Intraepithelial Lesion

- High-grade squamous intraepithelial lesion includes CIN 2 and CIN 3.
- Atypical immature cells extend further through the epithelial thickness, with reduced maturation and abnormal mitoses.
- The basement membrane remains intact in an intraepithelial lesion.
- Strong block-type p16 staining can support classification in appropriate diagnostically difficult settings.
    - p16 is a surrogate marker in a defined context and should not replace morphological assessment.

3. Invasive Cervical Carcinoma

Invasion and Squamous Carcinoma

- Invasion occurs when malignant cells cross the basement membrane and enter the cervical stroma.
- Squamous cell carcinoma is the most common histological type.
- Grossly, the tumour may be exophytic, ulcerative or diffusely infiltrative.
- Microscopy shows malignant squamous nests with variable keratinisation and a stromal response.

Glandular Types and Hidden Endocervical Disease

- Adenocarcinoma arises from glandular epithelium and has distinct precursor and invasive patterns.
- Some uncommon cervical adenocarcinomas are HPV-independent.
- A normal-looking ectocervix does not exclude an endocervical lesion.

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Screening and Diagnostic Confirmation

- Screening detects high-risk HPV infection or cellular precursors before symptoms develop. HPV testing and cervical cytology serve different but complementary roles in screening pathways.
- An abnormal screen is evaluated according to an established programme, often using colposcopy and directed tissue sampling.
- Cytology is not a substitute for histological proof of invasion.
    - Visual assessment and biopsy can miss disease if the transformation zone is not adequately evaluated.

Clinical Presentation

- Invasive carcinoma may cause postcoital bleeding, irregular bleeding, discharge or pelvic symptoms.
- Early disease can be asymptomatic.

Local Extension, Nodal Spread and Obstruction

- Spread occurs through the cervical stroma into parametrial tissues and vagina, and through lymphatics to pelvic and para-aortic nodes.
    - Advanced invasion may obstruct ureters or involve bladder and rectum.
    - Hydronephrosis is an important consequence of pelvic extension.

Endometrial Hyperplasia

- Endometrial hyperplasia is excessive proliferation of endometrial glands relative to stroma.

Hormonal Mechanism

- Prolonged oestrogen stimulation without adequate opposing progesterone is an important cause.
    - Anovulation, obesity, some hormone-producing tumours and unopposed oestrogen therapy can create this hormonal setting.
- Obesity can increase oestrogen exposure through peripheral conversion of androgens in adipose tissue.
- Hyperplasia commonly presents with abnormal uterine bleeding.

Morphological Categories

- Hyperplasia without atypia shows increased and sometimes cystically dilated glands without the cytological features of a precancerous clone.
- Atypical hyperplasia, also termed **endometrial intraepithelial neoplasia**, contains crowded abnormal glands with cytological differences from the surrounding endometrium.

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- This lesion has a higher risk of progression and may coexist with an already established endometrioid carcinoma.

- Sampling must distinguish a precursor from invasive carcinoma and from benign mimics such as a polyp.
- Hormonal and clinical information aids interpretation.
- A limited biopsy may underestimate a heterogeneous lesion; associated carcinoma cannot be excluded simply because one fragment contains only a precursor.

Endometrial Carcinoma

- Endometrial carcinoma is a malignant epithelial tumour arising in the uterine endometrium.

Clinical Presentation

- Endometrial carcinoma commonly presents after menopause, although younger patients can be affected.
- Abnormal bleeding is an important presenting feature.

Pathogenesis and Main Histological Types

1. Endometrioid Pathway and Hereditary Risk

- Endometrioid carcinoma often arises in a background of prolonged oestrogen exposure and atypical hyperplasia.
- Alterations in PTEN, mismatch repair and related pathways can contribute.
  - Lynch syndrome increases inherited risk through pathogenic mismatch-repair gene variants.
- Squamous differentiation can accompany malignant endometrioid glands and should not automatically be interpreted as a separate cervical primary.

2. Serous and Other Histological Types

- Serous carcinoma commonly arises in an atrophic endometrium and is strongly associated with abnormal p53-related biology.
- Clear cell carcinoma and other types have separate morphological patterns.
  - The older type I/type II division is a useful historical simplification but does not capture all biological groups.
- Extrauterine spread can occur despite a small uterine lesion, making surface appearance an unreliable measure of biological risk.

Gross and Microscopic Features

- The tumour may be a polypoid mass or a diffuse thickening of the endometrial lining.
- It can infiltrate the myometrium and extend beyond the uterus.

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