



Vulvar Squamous cell carcinoma

Factors	HPV-associated vulvar squamous cell carcinoma	Non-HPV-associated vulvar squamous cell carcinoma
Differentiation (degree of similarity compared to normal cells)	Poor	Good
Precursor Lesion	High grade squamous intraepithelial lesion. Intraepithelial – within the lining	Differentiated vulvar intraepithelial neoplasia (dVIN) VIN – abnormal overgrowth of the vulva
Risk factors	Age: 30 to 50 years Causative agents: HPV subtype: 16 and 17 People who smoke. Overgrowth of vulva (vulvar intraepithelial neoplasia – VIN)	Age: 40 to 60 years Lichen sclerosis: A chronic skin disease that affects the vulva. It has ivory white skin that is followed by lesion.
Description under the microscope (histology)	Multifocal (more than one area or foci separated by intact epithelium.	Abnormal cells (cytologic atypia) to the basal layer. Abnormal keratin protein pearls. Basal cell atypia Unifocal (one area affected) Mature neoplastic cells. High levels of eosinophilic cytoplasm. Eosinophils (type of immune cell whose nucleus appears red-orange when stained). Cytoplasm- the space inside the cell. Enlarged nuclear atypia. Nucleus contains genetic material that helps control the cell. Mitotic activity – high rate/ability to divide cells and grow.
Immunohistochemistry markers (protein markers that help detect) Block positive staining helps distinguish between types	P16 abnormalities (positive staining) P16 is a cyclin dependent kinase inhibitor for VIN. It accumulates/increases when transforming HPV.	Tumour suppressor gene (p53) abnormalities in basal layer It is difficult in small biopsy samples as normal epithelium cannot be found. CK17 better expression in differentiated VIN over undifferentiated VIN and lichen simplex chronicus
Prognosis (assessment of course of disease)	Good prognosis compared to non-HPV vulvar carcinoma	Poor prognosis compared to HPV-associated carcinoma