



4. HPV STATUS AND ASSOCIATED PRECURSOR LESION IN VULVAR SQUAMOUS CELL CARCINOMA

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Introduction. There are distinctive precursor lesions related to vulvar squamous cell carcinoma (VSCC) that are associated with high-risk human papillomavirus (HPV) type 16,18 specifically vulvar intraepithelial neoplasia (VIN) and those with vulvar dermatoses not associated with HPV such as lichen sclerosus (LS). Risk factors are cigarette smoking, immunodeficiency, poor hygiene, lichen plan (LP).

Aim of study. Vulvar cancer is highly relevant in the context of being the most common invasive malignant tumor that affects the vulva epithelium and the fifth most common type of cancer among women.

Methods and materials. This literature review was based on exploring scientific articles from PubMed, Research Gate, Medscape to identify studies examining histologically verified and HPV-tested vulvar cancer.

Results. HPV-associated VSCC arise from high-grade squamous intraepithelial lesions, also referred to as vulvar intraepithelial neoplasia of usual type (HSIL/uVIN) in 30% of cases. HPV-independent VSCC derives from a premalignant lesion as differentiated vulvar intraepithelial neoplasia (dVIN) and is associated with chronic inflammatory dermatoses in 70% of cases. HPV-independent VSCC are well differentiated and highly keratinized and arise on an autoimmune background of T-lymphocyte-mediated inflammatory disorder among patients at 60-80 years old. In younger women age 30-40, the tumor has a warty or basaloid pattern. HSIL has a relatively low risk of progression to VSCC. The prognosis is worse in patients with VSCC associated with dVIN than in patients with uVIN.

Conclusion. HPV status is a risk factor for VSCC, which is correlated with the survival rate. HPV-negative VSCC have a worse survival rate than HPV-positive VSCC. The role of HPV as a potential biomarker for early cancer diagnosis and predictor of prognosis and cancer treatment is significant.