DERMATOPATHOLOGY

MOLECULAR MARKERS EXPRESSION IN THE TRANSITION FROM NORMAL EPITHELIUM TO INVASIVE CANCER IN VULVAR SQUAMOUS CELL CARCINOMA

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Introduction: Vulvar Carcinoma is a rare neoplasm affecting elderly women. Hormonal factors may exert a relevant role in its pathogenesis.

Objective: We investigated some molecular markers, including steroid hormones receptors, in the transition from normal epithelium (NE) through pre-neoplastic associated lesions (PRE) to invasive cancer (K).

Materials and Methods: Immunohistochemical (IHC) expression of Androgen Receptor (AR), Estrogen Receptor α (ERα), Progesterone Receptor (PR), Ki67, p53, Cyclin D1 was investigated on NE, PRE and K. Only patients treated with curative-intended surgery and with no previous therapies (hormonal, chemotherapy or radiation therapy) were considered.

Results: 29 patients affected by VSCC and treated at Ospedale Policlinico San Martino between 2013 and 2014 were considered eligible (mean age 74 years; range: 46 – 94). Associated lesions were: Squamous Hyperplasia (SH) in 7 cases, Lichen Sclerosus (LS) in 15 cases, usual VIN (uVIN)
in 9 cases and differentiated VIN (dVIN) in 19 cases. Comparing NE to PRE, NE showed a significantly ($p<0.05$) higher expression of AR (61.8±32.5 vs. 13±22.8) and Cyclin D1 (20.4±15 vs.
12.5±11.7) and lower expression of Ki67 (11.2±5.1 vs. 22.1±11.1). Comparing NE to K, NE showed a significantly ($p<0.05$) higher expression of AR (61.8±32.5 vs. 2.5±8.3) and ERα (16.4±27.3 vs. 1.4±4.3) and lower expression of Ki67 (11.2±5.1 vs. 37.1±16) and p53 (1.6±0.3 vs.
26.2±5.1). Comparing PRE to K, PRE showed a significantly ($p<0.05$) higher expression of AR (13±22.8 vs. 2.5±8.3) and lower expression of Ki67 (11.2±5.1 vs. 37.1±16) and p53

(11.4±2.1 vs.
26.2±5.1). No significant differences for PR were observed.

Conclusions: VSCC confirmed higher expression of ki67 and p53 and showed insensitivity to steroid hormones, losing its receptors in the transition from NE to K. Therefore, the gradual and progressive loss of ERα and AR may exert a relevant role in the pathogenesis of VSCC.