Vulvar neoplasia and search for human papillomavirus 16 and 18 genetic information.

Short communication

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Summary
The presence of HPV 16 and 18 is frequent in cases with vulvar carcinomas and intraepithelial neoplasias.

Key words: Vulvar cancer; Vulvar intraepithelial neoplasia; Human papillomavirus.

Introduction
In recent years considerable interest has developed in the role of the human papillomavirus (HPV) and its effects on the relationships with vulvar intraepithelial neoplasia (VIN) and vulvar cancer [1, 2]. The presence of HPV 16 and 18 was reported in vulvar lesions before the development of invasive carcinoma, and also described in specimens with vulvar invasive squamous cell carcinomas [3, 5]. The purpose of the present study was to detect possible genetic information for HPV 16 and 18 in vulvar neoplasias [4, 6, 8].

Material and Methods
Specimens from 15 cases with vulvar intraepithelial neoplasia (VIN 3) and 21 cases with invasive vulvar carcinomas were analyzed for the presence of human papillomavirus genetic expression (Group A).

All cases were detected for the presence of homology to HPV 16 and 18 DNA transforming sequences with Southern blot hybridization. A search for HPV 16 E6 protein was also carried out with an immunoperoxidase assay in all cases. Specimens from 18 normal vulvas were used as controls (Group B).

Results
Our results showed that 4 invasive vulvar carcinomas (19%) contained HPV 16 DNA and 3 (14%) expressed the E6 protein, while 11 (52%) contained HPV 18 DNA. Also, 7 vulvar intraepithelial neoplasias [VIN 3] (47%) contained HPV 16 DNA and 2 (13%) expressed the E6 protein, while 3 (20%) contained HPV 18 DNA. Two specimens from normal vulvas (11%) were positive for HPV 16 DNA and E6 protein and 1 (5%) for HPV 18 DNA.

The difference between the two groups was highly significant (p<0.01).

Discussion
The association of HPV 16 and 18 with neoplasms of the lower female genital tract includes evidence that those HPV DNA types can transform epithelial cells in vitro. Cumulative evidence strongly implicates HPV in the genesis of squamous neoplasia of the vulva [9]. It would appear, however, that viral integration and the development of aneuploids is essential for the progression of VIN toward an invasive carcinoma.

The data of our study suggest that the presence of HPV 16 and 18 is more frequent in cases with vulvar carcinomas and VINs compared with normal vulvas. However, there have recently been reports that almost 60% of invasive and 47% of preinvasive lesions lack HPV 16 and 18, as judged by the extremely sensitive nonspecific PCR technique [7]. This has given rise to suggestions that there is a subset of VIN3 and invasive cancers that has a nonviral etiology (Husseinradeh et al. 1989, Pilotti et al. 1990, Rush et al. 1991, Li Vigni et al. 1992).

Nevertheless, epidemiologic evidence points to additional factors, including habitual, environmental and immunological factors that may play an equally important role in the development of vulvar carcinomas.

References


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