Vulvar Adenosis after Diathermy Treatment for Condylomas

Sir.

The condition in which glandular epithelium or its secretory products are present in the vaginal wall is defined as vaginal adenosis (VA). This disorder can occasionally be observed on the vulva (1). VA is usually related to prenatal exposure to diethyl-stilboestrol (DES) (2). However, it may also occur after mucosal injury. In this report we describe a case of vulvar adenosis occurring in a woman not exposed to DES after diathermy of condylomas.

CASE REPORT

A 25-year-old woman presented in our clinic with vulvar erosions. Three years before, the patient had been treated by diathermy for condylomas in another clinic. This diagnosis was confirmed by histologic examination. The in situ DNA hybridization test was able to detect HPV 6/11. After that, erosive papules appeared in the sites of the previous condylomata and remained unchanged until our observation. There was no history of prenatal DES exposure or of oral contraceptives intake.

Physical examination revealed a group of erosive papules, 3 to 10 mm in diameter, sharply demarcated and partially confluent, involving the vestibulum (Fig. 1). The patient complained about vulvar discharge, burning, pain, dyspareunia and post-coital bleeding. No condylomata were detected. The gynaecological visit did not reveal any other abnormalities. Histological examination of specimens of a biopsy showed a squamous epithelium, partially replaced by numerous papillary projections with little stroma, lined by columnar cells. Their nucleus was oval with faintly eosinophilic cytoplasm, with a thin PASpositive brush border. Some of them were ciliated. Several cystic invaginations extended downward from the vulvar epithelium. No squamous metaplasia and no evidence of malignant transformation were present. The stroma was oedematous, with a dense cellular infiltrate composed nearly entirely of plasma cells. In addition, the capillaries were dilated, with extravasation of erythrocytes and deposits of hemosiderin.

A complete resection of these lesions was performed.

DISCUSSION

Although VA is considered a clinical rarity, autopsy studies indicate that its frequence in women unexposed to DES is 13% to 15% (3), whereas in DES-exposed women it is 34% (4). Clinically, VA consists of erythematous, smooth or erosive papules, 1–3 mm in diameter, grouped in a cluster or confluent in plaques. The lesions can be focal or extensive on the entire surface of the vaginal wall. Excessive vaginal discharge, dyspareunia and bleeding, especially after sexual intercourse, are the most commonly reported complaints. However, VA can be present as an asymptomatic palpable nodule or represent an incidental finding on colposcopic or microscopic examination (3).

Histologically, two forms of VA can be identified: surface and glandular (2–4). Surface VA, as in our case, is characterized by the presence of a columnar surface epithelium, which replaces the squamous epithelium of the vagina or vulva; glandular VA is observed in occult and cystic forms, in which there are vaginal glands in the lamina propria covered by normal surface squamous epithelium. VA may pose problems of clinical differential diagnosis with other chronic erosive vulvo-vaginal diseases, such as lichen planus, cicatricial pemphigoid, plasma cells mucositis, and vulvo-vaginal intraepithelial neoplasias.

In our case, surface VA arose in a woman unexposed to



Fig. 1. Erosive papules on the vulvar vestibule.

DES or other estrogenic preparations after diathermy of condyloma acuminata. Trauma could induce VA; in fact this disorder has been reported in patients with vaginal condylomatosis, dysplasia and lichen sclerosus treated by CO, laser and topical 5-fluorouracil (5-FU) cream alone or in combination (1,5-8). Development of VA following Steven-Johnson syndrome is also reported (9). Probably the damage of the vaginal or vulvar epithelium may induce the development of columnar metaplasia or unmask a clinically undetectable form of VA. Dungar & Wilkinson retain that columnar metaplastic changes induced by topical 5-FU therapy are related to the alterations of stromal factors, which control vaginal epithelial differentiation and growth (6). Usually, symptomatic VA requires surgical treatment. However, no specific treatment is required for the asymptomatic forms of this condition, because the lesions seem to decrease over time with squamous metaplasia. A careful follow-up of women exposed and unexposed to DES is nevertheless recommended, because in some cases malignant transformation (usually clear-cell carcinoma) has been described (7). In conclusion, dermatologists should consider the possible occurrence of VA after destructive therapy of condylomata acuminata as a side-effect.

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