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 diethylstilbestrol (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 condylomata.

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 condylomata 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 stroma, lined by columnar cells. Their nucleus was oval with finely cosinophilic cytoplasm, with a thin PAS-positive 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 was 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 frequency in women unexposed to DES is 15% to 18% (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 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 oescent and cystic forms, in which there are glandular 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 periphphinx, plasma cells mucitis, and vulvo-vaginal intraepithelial neoplasia.

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

DES or other estrogenic preparations after diathermy of condylomata acuminata. Trauma could induce VA; in fact this disorder has been reported in patients with vaginal condylomatosi, dysplasia and lichen sclerosus treated by CO2 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. Dungan & 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.

REFERENCES

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Lymphangioma Circumscription of the Penis

Sir,

Lymphangioma circumscription (LC) is a rare, benign seccular dilatation of thin-walled lymph vessels, presenting as a local eruption of persistent, grouped, translucent vesicles. Usually it is present at birth, but it may become apparent at any age. It is more common in females and any cutaneous site may be affected (1). We here report its unusual penile localisation in a 27-year-old man.

CASE REPORT

In 1991 the patient had noted a few vesicular, occasionally oozing lesions localized around the urethral meatus. Two years later he began complaining of haemorrhagic diarrhea. Diagnosis of ulcerative colitis was made and treatment with mesalazine 1.6 g/day was started.

Clinical examination revealed a few translucent, thick-walled vesicles filled with a clear fluid and grouped in an area of about 1 cm² (Fig. 1).

A specimen from the excisional biopsy revealed dilated lymph vessels in the superficial dermis, lined by a simple layer of endothelial cells with moderate acanthosis and hyperkeratosis.

An ultrasound scan of the penis and perineal area showed no involvement of deep tissues. Diagnosis of LC was made.

DISCUSSION

Cutaneous lymphangiomas can be due either to a developmental abnormality of the lymphatics or to their obstruction secondary to surgical operation, irradiation, Crohn’s disease, tuberculosis and other inflammatory diseases (2).

Developmental abnormalities are named LC. In this case the origin of the vessel malformation remains unknown.

According to Whimster, the underlying defect of LC consists essentially of a collection of large-muscular coated lymphatic cisterns in the deep subcutaneous tissue, which do not communicate directly with the lymphatic system but represent a sequestrated part of it. Instead, they would communicate via dilated dermal lymphatics with the superficial vesicles. The latter are sacular dilatations of superficial lymphatics secondary to raised pressure transmitted from the pulsating cisterns beneath. Cisterns connecting vesicles and vesicles represent a closed system, in which there is no real lymph circulation.

The absence of lymphedema in LC is explained by the possible coexistence of normal lymphatics with proper central connections that ensure efficient lymph drainage (3).

The cases secondary to obstruction of the lymphatics are referred to as lymphangiectasias or acquired lymphangioma. Both clinically and histologically, LC and lymphangiectasias are indistinguishable. Only the medical history may help (4).

REFERENCES


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