Benign squamous papillomatosis: case report

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SUMMARY Squamous papillae in the vestibule are common. They were once considered to be normal variants of female anatomy, but reports of the viral aetiology of such lesions are emerging. When they are symptomatic, squamous papillae can lead to problems in sexual relationships between healthy partners. Here we report a case that responded well to treatment.

Benign lesions in the lower genital tract, which were once considered as normal, are now known to relate to the wide range of disease caused by human papilloma virus (HPV). Despite strong circumstantial evidence suggesting an association between HPV and lower genital tract neoplasia, final proof that HPV is the causative agent of cervical cancer still remains to be established. It is important to be aware of the various clinical manifestations and take adequate steps to screen the patients.

Case report

A nulliparous woman aged 29 presented with a two year history of roughness around the vulval skin. She had noticed occasional itching in the vulva and superficial dyspareunia, and an occasional non-offensive brownish discharge mainly after menstruation. She had regular sexual intercourse with her fiancé, who was her only sexual contact during the previous five years. She was in good health until this episode, which caused her great anxiety as she was due to be married four weeks later.

Examination showed a pearly white papillomatous area in the introitus, on the inside of labia minora (fig 1). The extent of the lesions was well defined on hand lens examination. There was minimal clear mucoid discharge. The rest of the external genitalia, vagina, and cervix showed no abnormality. A wet film, Gram stained smears, culture, and serology gave negative results for gonorrhoea, chlamydial infection, anaerobic vaginal infections, trichomoniasis, candidiasis, and syphilis. A biopsy specimen taken from the papillomatous area in the introitus showed fibroepithelial polyps covered by hyperplastic squamous epithelium, the core of fibrovascular tissue showed slight chronic inflammatory infiltrate, and there was koilocytosis indicating HPV infection.

Cervical exfoliative cytology showed no abnormality. As the patient had had normal results for two cervical smears in the previous year and the cervix looked healthy, colposcopy was not undertaken.

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Trichloroacetic acid 10% and podophyllin 25% in spirit were applied to the lesion weekly. The application was left for three hours in the first instance, and then for longer periods lasting up to six hours, before being washed off. After three weeks of treatment the residual areas were treated with cryocautery.

Examination, with a hand lens but without acetic acid, of her fiancé showed no clinical evidence of HPV infection.

Discussion

Squamous papillae of the vulva are well recognised, but their aetiology is controversial. Even in recent anatomical descriptions they are said to be usually asymptomatic variants of normal anatomy. Many women, however, experience pronounced symptoms, with pruritus, burning localised in the vestibule, and post-coital irritation. Local superficial dyspareunia may be present, as in our patient. In a normal sexually active woman these symptoms may cause anxiety. Patients often seek treatment having noticed a roughness in the vestibule or some change in local anatomy; many are told that the lesions are normal. Recent deoxyribonucleic acid (DNA) hybridisation and immunocytochemical studies have shown HPV in some of these lesions. As in other HPV lesions there is koilocytosis, although squamous papillomatosis is said to differ from condylomata acuminata histologically in that the papillary fronds are isolated and individually supported by a fibrovascular stroma. This was not observed in the biopsy specimen taken from our patient, in which the appearance was typical of condylomata acuminata (fig 2).

HPV causes a variety of macroscopic lesions in the lower genital tract. The appearance varies with such variables as local environment, type of epithelium affected, and presence or absence of moisture. To date HPV types 6, 11, 16, 18, and 32 are recognised as causing genital lesions. DNA-DNA hybridisation studies show that HPV 6 and 11 (which are very similar and may be grouped as one) sequences are found in 90% of condylomata acuminata and 30% of cervical intraepithelial neoplasia (CIN). HPV 16 or 18 is found in 66% of CIN and 90% of malignant disease of the cervix. HPV 32 has also been found in malignant disease of the cervix. The oncogenic potential leading to carcinoma varies according to the site, the cervix having the highest potential for such change.

In the past decade the incidence of genital wart
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Virus infection has doubled, and increasing numbers of sexually active healthy young women with warts are attending departments of genitourinary medicine. In these patients vulval squamous papillomatosis is a clinical entity to be recognised. As up to a third of women with simple vulval warts may develop premalignant cervical lesions, patients with squamous papillomatosis require careful follow-up. Once the viral aetiology is identified, treatment is by conventional methods; we used podophyllin and trichloroacetic acid treatment with cryotherapy to resistant areas, which produced good results. Lesions resistant to conventional treatment have been treated effectively with laser. Male sexual contacts should also be examined, in view of the sexual transmission of genital warts, and treated if necessary. It is, however, important to note that there are other vulval papillary lesions of unknown aetiology; each patient should be carefully assessed before treatment.

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References