Tissue erosion with perianal warts in HIV infection

Mayura Nathan, Steve Beck

Abstract
A case of painful tissue erosion due to perianal warts is described with HIV infection. Such a development can be avoided by frequent and careful monitoring.

Case history
A 25 year old male referred himself with persistent gnawing perianal pain associated with his perianal warts, of two weeks duration. The pain gradually worsened and was intense at presentation. A year previously his perianal warts were surgically removed in his residential district but recurred 2 weeks later. Over two years earlier he had been found to be Hepatitis B antigen and HIV antibody positive.

On examination there were multiple large warts, some measuring 5 cm. Two warts measuring 3 cm were found to be eroding into the perianal tissues. These warts were clearly circumscribed but were buried into the apposing perineal tissues. There was fresh blood oozing from the eroded area. His “gnawing” pain was located to the eroded tissue of the perineum. His CD4 count was 0.54 x 10^9/L (27%). Laser excision of the larger warts with ablation of the smaller warts (<1cm) were carried out under local anaesthesia with immediate relief of pain. Extension of the exophytic warts into the anal canal (4 cm spread) was noted and the treatment extended to the anal mucosa. Biopsy specimens were obtained from the eroding warts, adjoining anal mucosa and the skin overlying the eroded tissues. Histology demonstrated typical features of condylomatous wart virus infection with an overall exophytic growth pattern. There was no evidence of invasion of the underlying stroma by the proliferating squamous epithelium. There were extensive changes of high grade (grade 2–3) anal intraepithelial neoplasia but no evidence of invasive carcinoma (fig).

HPV typing was performed using in situ hybridisation and HPV types 6 and 11 were detected. Subsequent follow-up showed excellent healing with minimal scarring and the patient remained free of pain. He developed further small exophytic warts (<1 cm) both at the anal mucosal site and at the perianal skin. The treatment was extended with systemic interferon injections in addition to laser ablation of new exophytic warts.

Discussion
Most anogenital warts are mucocutaneous outgrowths precipitated by HPV infection. The mechanisms by which the HPV stimulus lates the formation of such appendages are not fully understood. On occasion the mucocutaneous proliferation is directed downwards into the deeper tissues leading to invasion of the underlying organs, so-called Buschke-Lowenstein tumour. Although it is suspected that some form of immune deficiency or other trigger factors for downward growth is not known. Recent observations indicate the possibility of altered virulence as one of the mechanisms concerned. The latter was seen in association with HPV Type 6.

We describe here a case of perianal warts with localised pain in which there was no invasion of the underlying tissues. Histological examination of the pedicle of the warts did not demonstrate any proliferation into the underlying tissues, yet clinical examination demonstrated penetration into the deeper tissues that were pressing on some of the warts. There were no carcinomatous changes seen either in the offending warts or the tissues eroded by them. However, intraepithelial neoplasia changes have been noted in the anal mucosa. Such changes are well documented in HIV infection. This patient's CD4 counts indicate mild to moderate immune suppression and he was asymptomatic otherwise. There has been some evidence of increased presence of HPV DNA in the anal mucosal cells with increasing
Tissue erosion with perianal warts in HIV infection

Immune deficiency, in addition to anal dysplasia by cytological evidence, under such circumstances. A possible explanation in this patient for tissue invasion may well be the rapidity of the growth of the warts and the pressure on the surrounding tissues. Rapid growth of anogenital warts in HIV infected individuals is well recognized. Such considerations will emphasise the need for early intervention. In addition, close monitoring may be necessary in homosexual men with HIV infection and perianal warts, as intraepithelial neoplasia and squamous carcinoma may occur under such conditions. A number of previous reports have suggested that such changes are closely related to HPV 16 infection. In one study, of HIV infected individuals the most common presenting symptoms and signs for squamous cell carcinoma of the anus were anal mass, pain and bleeding. In addition, the increased risk of intraepithelial neoplasia in the presence of infection with multiple HPV types will call for increased vigilance in such patients.

It is suggested that close and frequent monitoring of patients with perianal warts and HIV infection will be helpful in avoiding patient discomfort and tissue invasion.

The authors thank Dr Michael Wells, Dept. of Pathology, University of Leeds for his help with the HPV typing.