cal drainage with debridement of necrotic tissue may be mandatory when extensive pyomyositis occurs.

In conclusion, a high index of suspicion and selection of the proper culture conditions should help clinicians identify cases of pyomyositis due to \textit{M. haemophilum} and initiate appropriate treatment.

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References


Detection of Human Papillomavirus Type 35 in a Nodular Cutaneous Tumor in a Patient Infected with Human Immunodeficiency Virus

Numerous studies of HIV-seropositive and HIV-seronegative women and homosexual men have shown an increased incidence of human papillomavirus (HPV) infection and HPV-related neoplasia in HIV-positive individuals \cite{1}. The neoplasms described in HIV-infected patients include basal cell carcinoma, squamous cell carcinoma, bowenoid papulosis, Bowen’s disease, cloacogenic carcinoma, and malignant melanoma \cite{2}. The degree of immunodeficiency (CD4 cell counts) is an important factor with respect to the prevalence of HPV-associated dysplastic and neoplastic lesions. The relative risk for invasive anal cancer among homosexual men, compared with the general population, increases significantly, especially after the diagnosis of AIDS \cite{1}. High-risk HPV types 16 and 18 are detected most frequently, but other HPV types are also found in high-grade squamous intraepithelial lesions (SILs) in patients with AIDS \cite{1}. Because of their immunocompromised status, HIV-infected patients are less able to suppress cutaneous premalignant and malignant diseases; therefore, histological examination of any suspicious lesion has been suggested for these patients \cite{2}.

We describe an HIV-infected patient with markedly impaired cellular immunity (1996 CD4 cell count, 40–70/μL), but no other evidence of AIDS-defining disease. A 49-year-old male had had deep dermal nodules of the scrotal skin since January 1996 (figure 1). In May 1996, histopathologic examination of a scrotal biopsy specimen was performed. The epidermis showed irregular acanthosis with many enlarged and hyperchromatic nuclei, as well as dyskeratotic and vacuolated keratinocytes. Mitoses could be detected above the basal layer. A second biopsy specimen examined in November 1996 again displayed pronounced acanthosis and papillomatosis with mitoses present above the basal layer. Koilocytic cells could be detected in the upper portions of the epidermis, and many nuclei were hyperchromatic and irregular, sometimes with prominent nucleoli.

By use of PCR with consensus primers MY09/MY11 \cite{3}, HPV DNA was detected both in a scrotal-skin swab specimen obtained in January 1996 and in the biopsy specimen from May 1996. By restriction fragment length polymorphism (RFLP) analysis of the PCR product and subsequent hybridization with a generic oligonucleotide probe \cite{4}, HPV 35 was identified in both specimens. The presence of HPV 35 was confirmed by sequence analysis of the PCR product. In November 1996, the nodular tumor lesions were excised and the patient subsequently received treatment with IFN. Up to 6 months after surgery, no recurrence of symptoms was observed.

The clinical manifestations of the scrotal skin lesions described herein are unusual for HPV-associated anogenital lesions. Typical morphological features of condyloma acuminatum, SILA, carcinoma in situ (bowenoid papulosis or Bowen’s disease), or invasive carcinoma were not detectable by histopathologic evaluation, although the dysplastic epithelial changes somewhat resembled bowenoid papulosis. However, the koilocytic epithelial changes indicated etiologic involvement of HPV infection.

Among carcinomas in situ (bowenoid papulosis or Bowen’s disease) and cancers of the anogenital tract, HPV 16 is the HPV type detected most frequently \cite{5}. HPV 35 was first detected in a cervical adenocarcinoma \cite{6}. Later, it was detected in other sites of the lower anogenital tract (i.e., vulva, vagina, penis, and anus). HPV 35 has also been identified in bowenoid dysplasia of the periurethral area \cite{7}. HPV 35 had been commonly referred to as an HPV type with intermediate risk for tumor induction that is most prevalent in high-grade SILs \cite{8}; however, due to its detection in cervical and laryngeal carcinomas, HPV 35 is now considered...
lymphocytes and Langhans’ cells. In addition, because extracellular HIV-1 Tat protein has been shown entering HPV 16—infected cells and transactivating the HPV long-control region, a direct induction of HPV oncogene expression by HIV may also be possible [10]. Thus, depending on the HPV type detected, HIV-infected patients should be monitored carefully with respect to both histopathology and HPV infection, and surgical treatment should even be considered.

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References