Papulonodular Genital Growths in an HIV-Infected Woman

(See page 1585 for Photo Quiz)

Diagnosis: giant molluscum contagiosum (MC).

Microscopic examination of Giemsa stained curetted material from the patient’s lesions (figure 1) demonstrated molluscum bodies (figure 2). The patient was treated with liquid nitrogen cryospray, and there was a reduction in the size of the lesions on subsequent follow-up.

Altered presentations of MC are seen among patients with different immunological aberrations. Defects in T lymphocyte count or functions are most commonly encountered [1]. The prevalence of MC lesions among HIV-infected individuals is 10%-30% [2]. Occurrence of extensive or giant MC lesions among these patients is a marker of an advanced stage of immunosuppression. Most of these patients have established AIDS, with a CD4+ T cell count of <100 cells/mm³ [2]. Statistically significant correlation between low CD4+ T cell count and the severity of the MC lesions supports the association between advanced HIV infection and the extent of MC lesions [3].

In immunocompetent hosts, MC lesions are dome-shaped, pearly white umbilicated papules of 2–4 mm in diameter. The course of the lesions is usually self-limited, with or without the need for minimal intervention. In patients with HIV infection, MC lesions are extensive and recalcitrant to treatment [1]. In these patients, lesions are more common on the upper body parts (i.e., face and neck) [4] than on the genital region, which is the usual site of occurrence in sexually active, immunocompetent, adult individuals [2]. In patients with HIV infection, there is a tendency for large lesions to form, often ≥1 cm in diameter; such lesions are designated as “giant MC.”

Compared with immunocompetent patients, patients with HIV type 1 (HIV-1) infection are more commonly infected with subtypes other than MC virus (MCV) type 1 (which is the usual infective agent in immunocompetent hosts) [2, 4]. There are several postulations to explain the pathogenesis of giant MC lesions in patients with advanced HIV infection. It may be because of a higher production of epidermal growth factors by the strains of MCV that are commonly seen in HIV-1–infected patients [4]. Microscopical changes suggestive of MCV infection have been found in perilesional skin with a normal appearance (up to 0.5–1 cm in diameter) in HIV-infected patients [5]. Koilocytic atypia in a hyperkeratotic and acanthotic epidermis is the characteristic finding in hematoxylin and eosin–stained histopathological preparations. Electron microscopy revealed occasional keratinocytes in the spinous layer with intracytoplasmic viral particles and concurrent keratinization. The epithelium adjacent to the lesions is unremarkable in immunocompetent individuals [5]. This is indicative of widespread existence of the virus in these immunocompromised hosts, even in clinically healthy skin. A decrease in the CD4+ T lymphocyte count and
depleted cutaneous Langerhans cells in HIV-infected patients [1, 5] may explain such findings and contribute to the pathogenesis of widespread and larger lesions. Moreover, an epidermal hyperproliferative response is evident in the skin biopsy specimens of different dermatoses in patients with advanced HIV infection, compared with the response in skin biopsy specimens obtained from asymptomatic HIV-infected patients [6]. This effect may be contributory to the hyperkeratotic, large lesions and recalcitrant nature of the infection in patients with AIDS.

The principal mode of transmission of MCV in adults is close sexual contact [2]. In this case, the lesions might have localized in the genital region because our patient was a commercial sex worker. The patient’s advanced stage of immunosuppression due to HIV infection was likely responsible for the fact that her lesions were large and widespread.

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