A Patient With AIDS With Fungating Lesions of the Face and Scalp
(See pages 1029–1030 for the Photo Quiz.)

Diagnosis: Giant molluscum contagiosum.

Molluscum contagiosum is a benign skin tumor caused by molluscum contagiosum virus. It is a parapoxvirus with 4 subtypes described [1]. Lesions >1 cm diameter are known as giant molluscum [2]. At onset of infection, the virus replicates in the lower layers of the epidermis. This is then followed by an incubation period of 2–7 weeks but can be up to 6 months. The epidermis then hypertrophies and extends into the dermis, and molluscum bodies (inclusion bodies characteristic of molluscum contagiosum) are formed. They were first described in 1841 by Henderson and Paterson and are also known as Henderson–Paterson bodies [3]. Figure 1 shows well-circumscribed intraepidermal lobules containing molluscum bodies. Molluscum bodies enlarge and rise to the surface as cells age. The hypertrophied epidermal cells project above the skin and appear tumor-like [4]. The characteristic appearance molluscum contagiosum skin lesions may be sufficient to make a clinical diagnosis. However, histopathologic evaluation of biopsy specimens, with the identification of molluscum bodies, is required to confirm diagnosis.

The typical lesion of molluscum contagiosum is smooth, dome-shaped, pearly or flesh-colored, often umbilicated, and always spares the palms and soles. Approximately 10%–20% of patients with symptomatic HIV infection or AIDS have molluscum contagiosum [5]. Patients with AIDS may have atypical presentations of molluscum contagiosum, such as molluscum without umbilication, molluscum as an abscess, tender molluscum, or erythematous nodular molluscum. The differential diagnosis of such lesions in AIDS could be cutaneous cryptococcosis, basal cell carcinoma, cutaneous horn, keratoacanthoma, histoplasmosis, coccidioidomycosis, and verruca vulgaris. Transmission routes include skin contact, fomite contact, and sexual transmission [6]. The disease lasts for 6–9 months, although individual lesions tend to regress after ~2

Figure 1. Hematoxylin and eosin image taken at 10× magnification, showing a transverse section taken from the exophytic skin lesions (A and B). The image shows the presence of acanthosis and numerous epidermal infoldings. The most striking feature is the presence of round to ovoid homogeneous eosinophilic intracytoplasmic inclusions, representing molluscum bodies (arrow). These structures, which are composed of viral particles and cellular debris, are pathognomonic for this condition.
months [7]. The cell-mediated immune system is most responsible for the defense against and subsequent spontaneous resolution of these lesions [8]. The cellular immunodeficiency in AIDS therefore predisposes to such infections. The natural history is significantly different in HIV-infected and immunocompetent hosts [9]. In HIV-infected individuals, lesions occur on the face (sometimes with ocular involvement) and/or intertriginous areas (axilla, groin, and buttocks) and tend to be prolonged, severe [10], and very resistant to treatment [11, 12]. The number of lesions is inversely related to the CD4 cell count; thus, it is a marker of immunocompromise [13]. The most effective approach to treating this condition is to restore the underlying immunodeficiency. In HIV-infected individuals, the lesions tend to regress with highly active antiretroviral therapy and immune reconstitution [14], which has been slow for our patient. Topical 3% cidofovir and imiquimod have both been shown to be beneficial [15, 16]. Other chemical treatments include 5-flourouracil, silver nitrate, phenol, podophyllin, cantharidin, iodine, and tretinoin, all of which can cause irritation [17]. Cryotherapy and mechanical curettage are also effective [17].

After establishment of the diagnosis, topical cidofovir was chosen as the treatment for our patient [18]. However, he could not afford intraleosomal cidofovir, and attempts to obtain the drug free of cost were unsuccessful.

Subsequently, shave excisions followed by electrodesiccation were performed by dermatology on several lesions on our patient’s forehead, temple, glabella, and preauricular areas, without recurrence. The treated sites healed with mild hypopigmented, hypertrophic scars, which the patient found cosmetically acceptable. He was scheduled for further shave excisions and electrodesiccation by plastic surgery. Unfortunately, more extensive excision subsequently performed resulted in profound scarring, and he was admitted to our hospital for intravenous cidofovir therapy.

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