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Papular Eruption in AIDS: Role of Demodectic Mites?

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We report 3 cases of pruriginous papular eruption of HIV disease, in which demodectic mites were found in the pilosebaceous follicles which were surrounded by a granulomatous inflammatory infiltrate. Papular eruption of HIV disease is a distinctive clinicopathological entity, whose pathogenicity is not known. Our observations suggest that demodectic mites might be sometimes the etiologic agents of this eruption. ***Key words: folliculitis; HIV; demodicidosis.***

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A peculiar papular eruption occurring in HIV-infected patients was reported by James et al. in 1985 (1). It has been named "papular eruption of HIV disease" or "papular eruption of AIDS" (1–3). The pathogenicity of this distinctive clinicopathological entity is not known, and no specific etiologic agent has been identified. However, the role of demodectic mites has been noted in this disease, since some cases of demodicidosis presenting as a similar papular or papulo-nodular eruption have been described (4–6). We report 3 new cases of papular eruption of HIV disease, in which demodectic mites might be the causative agents.

CASE REPORTS

Case 1

A 40-year-old man, homosexual, seropositive for HIV (CD4 100/mm^3), was treated for Kaposi's sarcoma by radiotherapy and intramuscular bleomycin. He also had psoriasis vulgaris treated by acitretin and was receiving primary prophylactic treatment for pneumocystosis with pentamidine aerosols. Within less than one week, follicular erythematous pruriginous papules appeared on his trunk, neck and face (Fig. 1). Cutaneous biopsies of the follicular papules showed numerous demodectic mites within the pilosebaceous follicles (Fig. 2). The hair follicles were surrounded by an inflammatory infiltrate, predominantly made of lymphocytes, which penetrated the external epithelial sheath. Severe spongiosis was present. There was also a perivascular dermal infiltrate consisting predominantly of mononuclear cells, as well as a dermal fibrosis. Treatment with 1% lindane lotion, including the face, rapidly resolved the clinical symptoms. Three milder relapses were treated successfully in the same way.

Case 2

A 28-year-old man, previously a drug addict, HIV positive, (CD4 50/mm^3), was treated by acyclovir for a chronic genital herpes virus infection, and by fluconazole for a chronic buccal candidiasis. He was also receiving zidovudine and a primary prophylactic treatment for pneumocystosis with pentamidine aerosols. For 10 months, he had had recurrent pruriginous erythemato-papular lesions, follicular in certain areas, on the thorax, the shoulders, and the neck. Treatment combining antihistamines and topical steroids only slightly improved the symptoms. A cutaneous biopsy showed the presence of numerous demodectic mites within the pilosebaceous follicles, with a dermal perivascular inflammatory polymorphous infiltrate, predominantly mononuclear. This diffuse infiltrate entered between the collagen layers, which themselves presented small, fibrinoid, necrotic centres. Topical treatment with crotamiton cream was carried out with success.

Case 3

A 37-year-old heterosexual man seropositive for HIV (CD4 100/mm^3) had had a papular follicular and non-follicular pruriginous eruption on the thorax and the neck for 4 months. The lesions did not improve with topical steroids. The clinical aspect of the lesions suggested a papular eruption of HIV. A cutaneous biopsy showed a granulomatous polymorphous inflammatory dermal infiltrate, mainly lymphohistiocystic with multinuclear giant cells. Follicles containing demodectic mites were at the centre of the inflammatory reaction, which destroyed the follicular habitat of demodectic mites, with spongiosis and exocytosis of the infundibular epithelium (Fig. 3). Topical treatment with 1% lindane lotion was carried out with success.

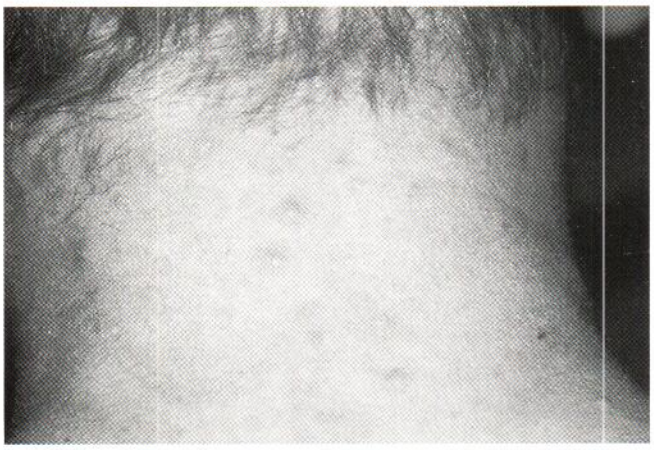

Fig. 1. Erythematous papules of 2–5 mm in diameter on the neck in Case 1.

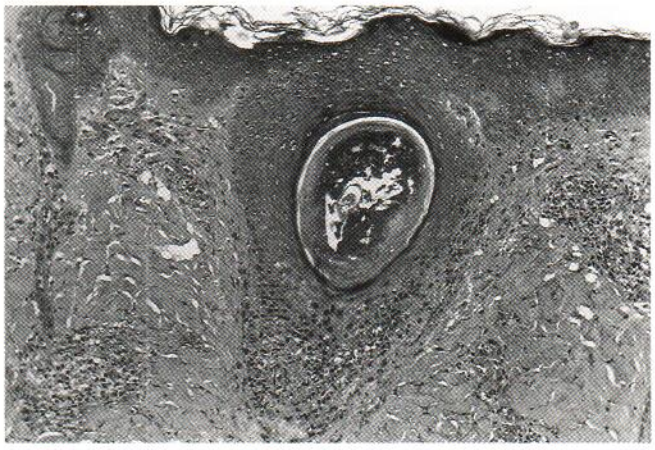

Fig. 2. Presence of demodectic mite within a pilosebaceous follicle in Case 1 (HE ×250).

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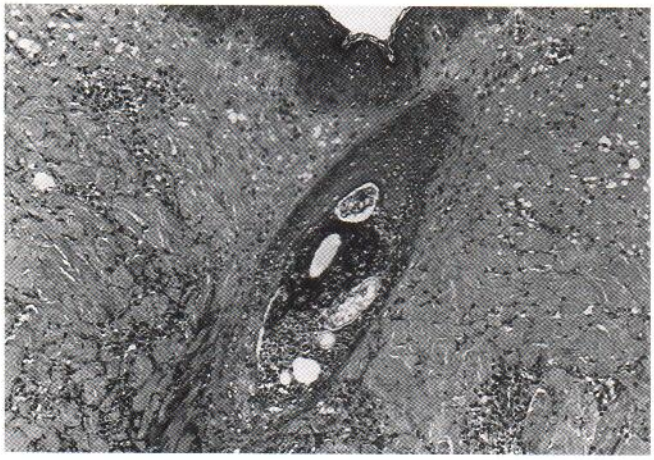

Fig. 3. Perifollicular inflammatory reaction with spongiosis and exocytosis of the infundibular epithelium in Case 3 (HE ×125).

DISCUSSION

In humans, cutaneous infestation by demodectic mites (an ectoparasite of the arthropod family) is generally asymptomatic; it is found in approximately 10% of all cutaneous biopsies. Demodex folliculorum and Demodex brevis are, amongst the 56 species of demodectic mites, saprophytes of the pilosebaceous follicles of the face, particularly in the nasolabial region, on the nose and on the eyelids. Demodex folliculorum is a parasite of the infundibulum, Demodex brevis of the sebaceous glands. Demodectic mites preferentially invade areas rich in sebaceous glands (face, thorax, neck, scalp), which explains why they are not found in children, whose sebaceous glands are immature (7). Their pathogenicity in the immunocompetent host is disputable, but they have been suspected in some cases of acne rosacea (9) as well as pruriginous blepharites (10), perioral dermatitis (11), follicular pustules of the scalp and face (12), and even macular hyperpigmentation of the face (8). They have also been suspected to be responsible for eosinophilic pustular folliculitis of AIDS (13) via a cutaneous hypersensitivity reaction. Their role has also been underlined by Cutler in eosinophilic pustular folliculitis (14), based on a major positive reaction of a RAST-IgE test for Dermatophagoides pteronyssinus (a house-dust acarian, the RAST for Demodex folliculorum not being available).

More recently, the question of their role in papular eruption of HIV infection has been raised. Several authors have described a papulonodular eruption made up of non-coalescent, pruriginous papules, 2 to 5 mm in diameter, follicular or non-follicular, of the head, the neck and the trunk, clinically similar to the papular eruption of AIDS (2, 4–6). Cutaneous biopsies revealed the presence of a large number of demodectic mites in the pilosebaceous follicles, associated with a perivascular lymphoplasmocytic infiltrate of the dermis accompanied in some cases by eosinophils and dermal fibrosis. Based on clinical, histological, and, above all, evolutive arguments (disappearance of the eruption after topical treatment with benzyl benzoate, lindane or crotamiton), these authors stated that demodicidosis was one of the etiologies of the HIV papular eruption. A proliferation of the parasite, due to a local and systemic immune deficiency and a hypersensitivity reaction induced by its presence might be responsible for the cutaneous lesions. Recently, Smith et al. (15) presented a clinical histological and immunohistochemical study of the papular eruption of HIV disease. They concluded that this dermatosis was a distinct anatomoclinical entity, which could be secondary to a hypersensitivity reaction to an antigen, mediated by the dermal dendrocytes. It must be noted that this study eliminated the histological sections showing bacterial, mycological, or parasite follicular involvement (in particular demodectic mites). These antigens could be multiple (bacteria, mycoses, parasites) and include demodectic mites, since it is known that cutaneous responses to various agents may be exacerbated in HIV-infected patients (16–18).

The clinical and histological similarity between the papular eruption of AIDS and the "papular demodicidosis" of HIV suggests that demodicidosis could be a possible etiology, not to be ignored, of the papular eruption of HIV infection, in particular when a follicular component is clinically present. This possibility justifies, in our opinion, a systematic treatment of these eruptions with an acaricidal, after a cutaneous biopsy has been carried out on principle.

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