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Rosacea-like demodicidosis associated with acquired immunodeficiency syndrome

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Summary We present a 35-year-old patient with acquired immunodeficiency syndrome who had demodicidosis on his face, characterized by multiple papules and papulopustules, associated pruritus, numerous mites on skin-surface biopsy and in biopsy specimens, and rapid response to topical treatment with permethrin. It seems likely that *Demodex* infestation does not manifest unless local or systemic immune function is altered, leading to the proliferation of the organism and subsequent disease.

Key words: acquired immunodeficiency syndrome, *Demodex* folliculitis, *Demodex* mites, demodicidosis, rosacea-like dermatosis

The hair follicle mites *Demodex folliculorum* and *D. brevis* are common inhabitants of the human pilosebaceous unit.[1] *D. folliculorum* is more common than *D. brevis* and is characterized by a larger size, an elongated posterior segment, and arrowhead-shaped eggs. It is usually located in the follicular infundibulum and may be present in numbers up to 10–15 per follicle. In contrast, *D. brevis* is shorter and more oval shaped. It is usually found in sebaceous glands and ducts and is solitary. Both follicular mites are most numerous in the skin of the face, scalp and upper chest. Aylesworth and Vance[2] found that 10% of 1123 skin biopsies and 12% of 1692 follicles studied contained follicular mites. Roth[3] examined 100 biopsies of eyelid skin and found follicular mites in 84% of all cases and in 100% of cases where the patients were over 70 years of age. In veterinary medicine, similar follicle mites are generally accepted as potentially pathogenic, causing severe inflammatory skin diseases by their multiplication, such as the demodectic 'red mange' in dogs.[4] Because of their prevalence on human skin, however, the pathogenic role of *Demodex* mites in human dermatopathology is still a matter of debate.

The available evidence suggests that, in certain circumstances, conditions arise which favour multiplication of follicle mites, and the presence of abnormally large numbers of mites probably induces or contributes to skin disorders, including granulomatous rosacea,[5–7] granulomatous perioral dermatitis,[7] pustular folliculitis,[8] papulopustular dermatosis of the bald scalp,[9] blepharitis,[10] follicular spicules on the face[11] and solitary granuloma.[12] We report a patient with demodicidosis (or demodicosis) associated with acquired immunodeficiency syndrome (AIDS).

Case report

A 35-year-old homosexual man with a history of AIDS (CDC C2) for 11 years was referred to our department because of a pruritic skin eruption involving his face. He stated that the eruption had manifested itself 2 years previously. He had no history of rosacea-related features such as recurrent or persistent facial erythema and flushing. He was taking zidothymidine 250 mg twice daily, saquinavir 1200 mg three times daily and delavirdin 400 mg three times daily. No systemic or topical treatment with corticosteroids had been given.

On examination, there were multiple papules and papulopustules on an erythematous base localized on the forehead and left cheek (Fig. 1). The distribution of the lesions was predominantly follicular. There was no involvement of the nose, eyelids or eyes, and no telangiectases. Bacterial and mycological cultures of skin swabs and papulopustule contents failed to grow pathogenic organisms. Mineral oil examination of skin surface biopsy from papulopustules of the forehead and left cheek revealed numerous *D. folliculorum* mites, predominantly adults (Fig. 2).

Histology of a punch biopsy from the left cheek

Figure 1. Multiple papules and papulopustules are evident on the forehead and left cheek.

Figure 2. Mineral oil preparation from lesion demonstrating many *Demodex folliculorum* mites (original magnification × 4).

revealed follicular hyperkeratosis and a dense perifollicular lymphocytic inflammatory infiltrate with an occasional multinucleated giant cell (Fig. 3). Granulomas could not be observed. The follicular infundibulum was dilated and contained several *D. folliculorum* mites. Periodic acid-Schiff staining for fungi gave negative results. Haematological findings included a CD4 cell count of 240 μL^{-1} (normal >400) and a CD4/CD8 ratio of 0·2 (normal 0·7–2·8).

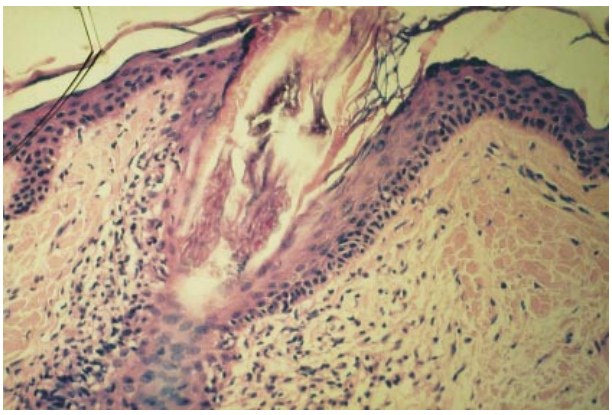

Figure 3. Photomicrograph showing follicular hyperkeratosis and dilated follicular infundibulum with perifollicular inflammatory infiltrates and *Demodex folliculorum* mites (haematoxylin and eosin; original magnification × 40).

Topical treatment with 5% permethrin cream applied twice daily gave a rapid response after 2 weeks and complete resolution of skin lesions and pruritus after 4 weeks. No adverse effects of topical treatment were noticed. At the end of the treatment, a skin surface biopsy of the forehead and left cheek showed only an occasional *Demodex* mite. No recurrence has been observed in a 10-month follow-up.

Discussion

It seems likely that, under normal circumstances, there is a control mechanism limiting the population of follicle mites, but that both local and systemic factors may create an environment encouraging their proliferation.

Ashack *et al.*[13] have reported a case of papular pruritic eruption of AIDS in which numerous *Demodex* mites were found. The eruption resolved completely following a single application of lindane. In four other patients suffering from AIDS and *Demodex*-attributed pruritic papular, papulopustular or papulonodular lesions on the scalp, face and neck, lesions responded well to topical acaricides.[14–16] In one case of papular eruption due to *D. brevis*, crotamiton treatment proved ineffective.[15] Reports of demodicidosis in children with AIDS[17,18] and acute lymphoblastic leukaemia[19–21] also suggest that the immune status of the host might have an influence on mite numbers.

Demodex mites can be demonstrated by microscopic examination of expressed follicular contents and skin scrapings, and adhesive tape, cyanoacrylate glue (skin surface biopsy)[22,23] and skin biopsy specimens. The number of mites varies greatly with the method employed and the site examined. In our experience, skin surface biopsy is a rapid and convenient method to check for *Demodex* mites.

Demodicidosis must be differentiated from staphylococcal folliculitis, human immunodeficiency virus (HIV) -associated eosinophilic folliculitis, *Pityrosporum* folliculitis and other non-specific papular eruptions of HIV disease. Our patient presented a number of clinical features that are uncommon in rosacea, including the lack of history of recurrent or persistent facial erythema and flushing, the associated pruritus, the asymmetrical distribution on the left cheek, and the absence of telangiectases. Grosshans *et al.*[5] suggested that the demonstration of a granulomatous reaction to mites, which are phagocytosed by foreign-body giant cells, may be essential to differentiate demodicidosis from rosacea. The different histopathology in most cases of AIDS-associated demodicidosis, in which well-defined granulomas were lacking,[13–17] as in our patient, may be related to the impaired immune response in these patients. However, itchy folliculitis in HIV infection does not appear to be related to *Demodex* infestation.[24]

Several agents have been used to eradicate infestation with *Demodex* mites, including metronidazole, permethrin, crotamiton, lindane, benzyl benzoate and 'Danish ointment' (sulphur). Recently, a single dose of ivermectin 200 $\mu g\ kg^{-1}$ with subsequent weekly topical permethrin led to rapid and complete recovery in a case refractory to conventional treatment.[25] In our patient, demodectic eruption was safely and effectively treated with topical permethrin. As suggested by Forton and coworkers,[22,23] the technique of standardized skin surface biopsy will enable us to monitor the *Demodex* population during treatment with metronidazole or other known acaricidal agents.

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