

Case report

Crusted scabies in a patient with the acquired immunodeficiency syndrome*

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Summary. A case of crusted (Norwegian) scabies in a patient with the acquired immunodeficiency syndrome (AIDS) is described and seven previous reports of this association are reviewed. Injury to epidermal Langerhans' cells by the human immunodeficiency virus or cytotoxic cells may explain the appearance of this unusual form of scabies in patients with AIDS.

Key words: Scabies – AIDS – Langerhans' cells

Introduction

The cutaneous lesions described in patients with the acquired immunodeficiency syndrome (AIDS) include diverse inflammatory conditions, neoplasms and infections (Warner and Fisher 1986), the latter often caused by agents frequently found in immunocompromised patients. As a consequence of an imbalanced host-parasite relationship secondary to infection with the human immunodeficiency virus (HIV), patients with AIDS suffer from severe and atypical forms of cutaneous infections, many of which are difficult to recognize clinically. Such is the case in crusted (Norwegian) scabies, an uncommon variant of scabies usually associated with immunosuppression or neurologic impairment (Espy and Jolly 1976; Wolf and Krakowski 1987) and recently described in patients with AIDS (Sadick et al. 1986; Rau and Baird 1986; Drabick and Tompkins 1986; Glover et al. 1987). Crusted scabies may be the first evidence of an underlying severe immunologic derangement as occurred in the patient with AIDS reported herein.

Case report

This 30-year-old man presented to the General Hospital of Mexico City with an 11 months history of increasing weakness, weight loss, a generalized eruption, and diarrhea. He denied homosexuality and intravenous drug abuse. He had not received blood or blood products but donated several blood units before screening for HIV was available.

Physical examination revealed an acutely ill, emaciated and dyspnoeic patient with an generalized papular hyperkeratotic eruption that was more prominent on the forearms, hands and in the axillary folds. Antiscabietic treatment was started after a dermatology consultant established the diagnosis of crusted scabies, confirmed by microscopic examination of scrapings obtained from one of the lesions. The patient was found to be HIV positive by Elisa and Western blot techniques. His overall condition rapidly deteriorated and he died 6 days after admission.

Autopsy revealed a predominantly interstitial pneumonia and a central nervous system lymphoma. Microscopic examination of the cutaneous lesions showed numerous mites in the stratum corneum, hyperkeratosis and a discrete lymphocytic infiltrate in the upper dermis (Fig. 1).

Discussion

The majority of the cases of crusted scabies occur in immunodeficient or neurologically impaired patients. Since the initial communication in 1986, 8 patients (including the present case) with HIV infection and "exaggerated" or crusted scabies have been reported (Table 1). Of these patients, 3 developed "atypical" scabies as the initial form of the disease while in other two ordinary scabies evolved into the crusted form (Rau and Baird 1986; Glover et al. 1987). Lack of recognition of the disease prompted the improper use of topical steroids in three patients (Sadick et al. 1986).

Crusted scabies is extremely contagious. Outbreaks of scabies in closed communities such as residential homes are almost invariably associated with an unno-

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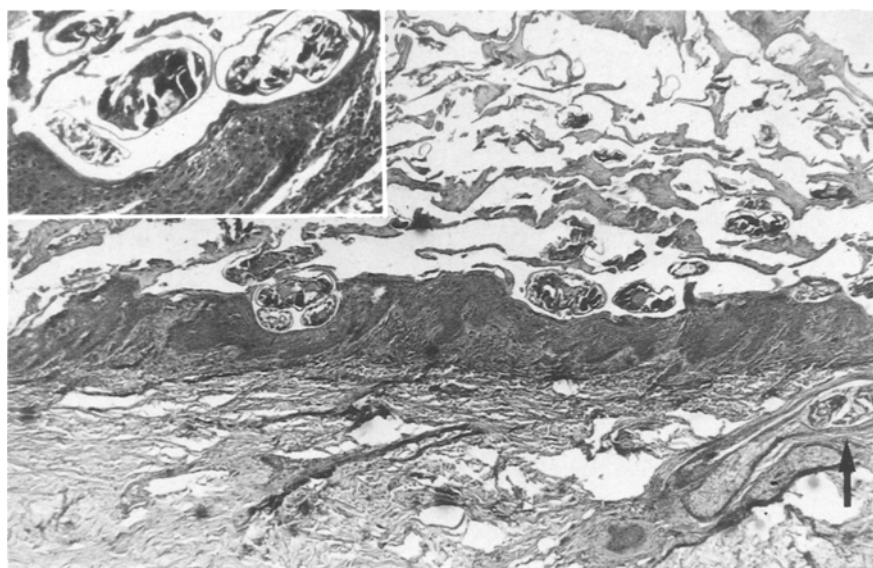


Fig. 1. Several mites are present within a hyperkeratotic epidermis. A mild lymphocytic infiltrate is observed in the upper dermis. (Haematoxylin-eosin stain $\times 45$). *Insert:* Detail of two mites. (Haematoxylin-eosin stain $\times 150$)

Table 1. Reported cases of scabies complicating HIV infection

Case	Age*/Sex	Risk group	Clinical lesions	Reference
1	22/M	Hemophiliac	Scaling, Papules, Burrows	Sadick et al. 1986
2	36/M	IVDA	Papular dermatitis	Sadick et al. 1986
3	31/M	Homosexual	Scaling, Papules, Nodules	Sadick et al. 1986
4	6/M	Hemophiliac	Eroded, crusted papules	Sadick et al. 1986
5	19/M	Homosexual	Scaling, Hyperkeratotic papules, burrows	Rau and Baird 1986
6	39/M	Bisexual	Erythematous papules	Drabick and Tompkins 1986
7	35/M	Homosexual	Hyperkeratotic plaques, fissures	Glover et al. 1987
8	30/M	None	Hyperkeratotic papules	Present case

HIV = human immunodeficiency virus

* age in years, M = male

ticed or misdiagnosed index case (Burns 1987). This problem may be particularly severe in hospitals or medical wards that provide care for immunodeficient patients.

The clinical presentation, appearance of the lesion and distribution of crusted scabies varies from that of common scabies. In the latter, the classic lesion is the burrow which is predominantly located on the hands, interdigital areas, wrists and elbows. In contrast, crusted scabies is characterized by generalized hyperkeratotic, scaly plaques or papules (Meyers and Connor 1976).

Due to the atypical clinical presentation, biopsies of the lesions are frequently performed. Unlike ordinary scabies where mites or ova are difficult to find, in crusted scabies the stratum corneum is filled with parasites. In addition, the epidermis becomes hyperkeratotic (Meyers and Connor 1976). The dermis shows a predominantly perivascular mononuclear cellular infiltrate where T lymphocytes predominate (Flak and Matre 1982; Van Neste 1988). Immunohistochemical studies performed on one patient with crusted scabies showed C3 deposition at the dermal-epidermal interface; (Van Neste and

Lachapelle 1981) however other investigators have reported negative results (Salo 1982).

A tempting speculation is that proliferation of the parasites in the stratum corneum is secondary to Langerhans cell injury. These antigen-presenting cells of the skin carry the CD 4 molecule on their surface (Wood et al. 1983), the receptor for HIV (Ho 1987), and HIV infection of Langerhans cells has been detected in patients with clinically established AIDS-related complex or AIDS (Tschachler et al. 1987). Reduction in the number of Langerhans cells, has been shown to occur in patients with AIDS, (Belsito 1984) and may represent a consequence of cytopathic effects of direct viral infection, or target cell injury by effector cells with cytotoxic potential (Ringler et al. 1987). The clinical presentation of superficial fungal infections in patients with AIDS may also be atypical and resemble other conditions (Penneys and Hick 1985); the same is clearly true in scabies.

This case reiterates the need to perform serological tests for HIV infection in any patient with crusted scabies without other cause of immunodeficiency (Drabick and Tompkins 1987).

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