

## Localized Crusted Scabies in a Patient with Acquired Immunodeficiency Syndrome

Sir,

Crusted (Norwegian) scabies (CS), is a rare variant of infestation with *Sarcoptes scabiei var. hominis*, where the skin lesions are extensive, and the thickened horny layer is riddled with innumerable parasites. This condition was first described in lepers by Danielssen & Boeck in 1848 (1).

This rare variant of scabies occurs mainly when the host's immune response is impaired and in patients who have a decreased sensation of itch, resulting in less scratching and less destruction of burrows.

In 1986, CS was reported for the first time in infected patients with human immunodeficiency virus (HIV) (2). Subsequently, some additional cases have been described in the literature (3). One case of localized crusted lesions on the toe has earlier been reported (4). We describe a patient with acquired immunodeficiency syndrome (AIDS), who developed an unusual form of CS, involving exclusively the genital area.

### CASE REPORT

In June 1995, a 31-year-old HIV-seropositive intravenous drug-abuser was admitted to hospital because of pneumonia. His CD4 count was 14/ $\mu$ l. He had a 1-month history of a generalized intensely pruritic eruption and had been found to have AIDS 3 years earlier. Physical examination on admission showed erythematous papules and excoriations involving predominantly the abdomen, thighs and buttocks. Burrows were found on the interdigital webs of his hands. A biopsy specimen revealed the presence of a scabies mite. The patient was treated with topical gamma benzene hexachloride lotion 1% for 3 days and improved rapidly.

Two months later, he was admitted again to hospital because of

pneumonia. His CD4 count was only 7/ $\mu$ l. He had also a keratotic lesion on the penis, which produced local pain and intense pruritus, and which had appeared 1 month earlier. Physical examination revealed extensive thick and confluent keratotic plaques with some greyish crusts, exclusively on the penis (Fig. 1). No other skin lesions were seen. A biopsy specimen showed multiple carved burrows within the cornified layer, of which some extended into the malpighian stratum. These burrows were occupied by great numbers of adult *Sarcoptes scabiei*, with eggs and larvae. Prominent hyperkeratosis with parakeratosis and a hyperplastic epidermis (psoriasiform) were also observed (Fig. 2). Treatment was started with a 5-day course of topical gamma benzene hexachloride lotion 1% in addition to a 10% salicylic acid ointment. Initially, a significant clinical improvement was noted, but the patient was lost for follow-up.

### DISCUSSION

HIV infection is today probably the most common disease triggering CS. However, atypical forms of scabies often complicate diagnosis in HIV/AIDS patients and predispose to nosocomial transmission. Scabies should therefore be suspected in any HIV/AIDS patient with itching or non-itching eczematous or keratotic lesions. Besides early diagnosis, prompt treatment and strict control measures are extremely important.

The treatment for CS is as for ordinary scabies, although repeated applications may be required with scabicides, and sometimes the sequential use of several agents. Permethrin cream is the preferred scabicide to begin the therapy. The entire skin should be treated, including under the nails (5).



Fig. 1. Extensive thick and confluent keratotic plaques with some greyish crust on the penis.

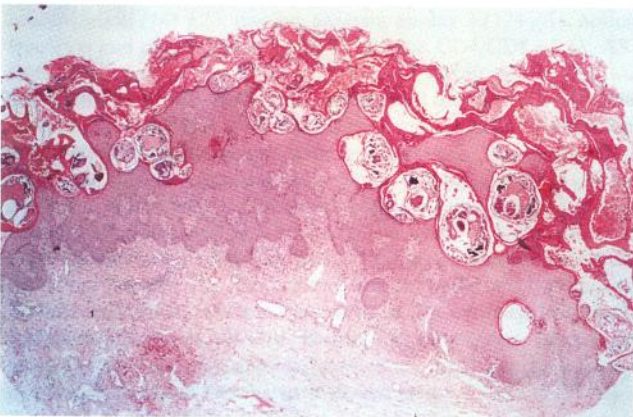


Fig. 2. Histology of a biopsy from the lesion, showing multiple carved burrows within the cornified layer. The burrows are occupied by great numbers of adult *Sarcoptes scabiei*, with eggs and larvae. Prominent hyperkeratosis with parakeratosis is also observed (hematoxylin-eosin stain; original magnification  $\times 150$ ).

Keratolytic agents may be needed, because the scabicide alone will not penetrate through the hyperkeratotic crusts. A recent report describes 2 patients with CS who were successfully treated with a single oral dose of ivermectin, in addition to a 3% topical ointment of salicylic acid (6). Even more recently, Meinking et al. reported a series of 22 patients effectively treated with ivermectin. Only 3 of the 11 HIV patients needed two or more oral doses of ivermectin (200  $\mu\text{g}$  per kilogram). Two of the HIV patients had CS; one of them was cured after a single dose of ivermectin, but the other needed three doses of ivermectin, the last one in addition to a topically applied 5% permethrin cream (7).

In HIV patients with CS no established therapeutical regimen exists, and the therapy applied is usually similar to that of HIV-negative patients. However, compliance in these patients is often low, and treatment is usually applied incorrectly. Orally taken ivermectin may be the future treatment of choice in these patients, because of its effectiveness and easy management. However, optimal treatment must await results from studies of larger numbers of patients.

As these patients are highly contagious, strict measures for control of infection are essential and should include the following: isolation of the patient; use of disposable gloves and gowns by staff; washing of clothes, linen and towels of patients; cleaning of the room, and prophylactic treatment of contacts (8). We have here reported a patient with AIDS, who developed a localized genital form of CS. We would like to emphasize the importance of its early diagnosis, which may prevent a possible bacterial sepsis and the transmission to other persons.

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