Ashy Dermatosis in an HIV Antibody-positive Patient

Sir,

Ashy dermatosis, or erythema dyschromicum perstans (1), is a chronic skin disorder characterized by hyperpigmented macules of various size on the trunk, face and extremities (2). It has rarely been described in HIV patients. We describe here the third known case of ashy dermatitis in a long-term HIV-infected patient.

CASE REPORT

A 38-year-old Spanish male, an ex-intravenous drug abuser, who was diagnosed with HIV infection 10 years previously, presented with an asymptomatic skin eruption on the buttocks and sacral region, which had been present for the past 2 months. No medication or prior disease was reported. The patient had not received anti-retroviral drugs or prophylactic medication against *Pneumocystis carinii*. Physical examination revealed the presence of hyperpigmented (brownish-grey) oval-shaped macular lesions with well-defined margins (Fig. 1), less than 30 mm in diameter. In the sacral region some of the lesions were confluent and formed patches. Tactile, thermal and pain sensitivities remained intact in the affected zone. The rest of the physical exploration was normal.

A biopsy of one of the lesions revealed a discretely acanthotic epidermis, with moderate compact hyperkeratosis. Epidermal basal layer was normal, without vacuolar degeneration. The upper dermis presented a predominantly lymphocytic perivascular infiltrate with many melanophages (Fig. 2).

The blood tests showed a CD4-positive count of 104 cells/mm^3, with positive anti-HVB and HVC serology. The rest of the analytical parameters (including the proteinogram) were normal. Serological tests for syphilis were negative.

DISCUSSION

Ashy dermatosis is a rare disorder pertaining to the lichenoid dermatitis group (3). This chronic dermatitis is of insidious onset, and is characterized by the appearance of oval or round-shaped bluish-grey macular lesions. The initial lesions may consist of raised erythematous plaques that progressively acquire a brownish-grey colour. The margins tend to be well-defined and lesion confluence can form extensive plaques. The eruption is generally asymptomatic, though moderate pruritus may develop. The lesions are generally located on the trunk and upper limbs. The palms, soles, scalp, nails and mucosal surfaces are not affected. Most cases reported to date are of Latin American and Indian patients (4).

The histological findings include pigmentary incontinence with sparse perivascular mononuclear infiltrate and numerous melanophages located in the papillary dermis. Basal cell layer vacuolization and Civatte bodies may also be present.

The aetiology of the disease is not known, though it has been associated with the ingestion of ammonium nitrate (5), exposure to environmental contaminants and worm infestation (6). A number of immunopathological studies of active skin lesions have shown that ashy dermatosis may involve immune mediation (7). The findings supporting this hypothesis are the expression of Ia antigen by the epidermal keratinocytes, the presence of OKT4 and OKT6 antigens in the epidermal dendritic cells, the dermal infiltrate consisting of OKT4- and OKT8-positive lymphocyte subpopulations and the presence of colloid bodies with IgG deposits as determined by direct immunofluorescence (7, 8). The disorder could represent a variety of lichen planus, in addition to exhibiting a similar immunopathological mechanism, the histological findings point to the existence of important similarities (7, 9, 10). Moreover, some cases have been reported (fundamentally involving dark-skinned individuals) in which ashy dermatosis preceded, followed or was concomitant to the presence of established lichen planus (11).

The pigmented macules closely resemble the lesions of the late pinta, but the negative dark-field examinations, negative serological tests for syphilis and lack of response to penicillin are important features that allow the dermatologist to exclude this treponematosis (12).

No treatment of choice is presently available. A number of treatment modalities have been attempted, including antibiotics and topical corticoids, keratinolytic agents, isoniazid, chloroquine and psychotherapy, but all with poor responses. Griseofulvin has been reported to induce complete resolution of the disease, though the lesions tend to recur upon suspending treatment. Likewise, 7 of 8 patients exhibited an

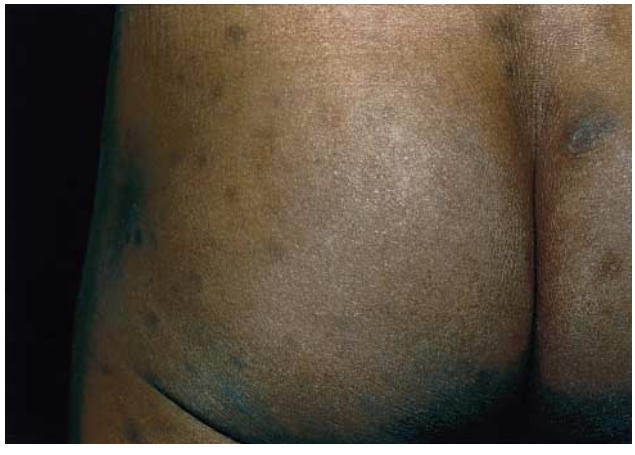

Fig. 1. Hyperpigmented oval-shaped macules on the buttock area.

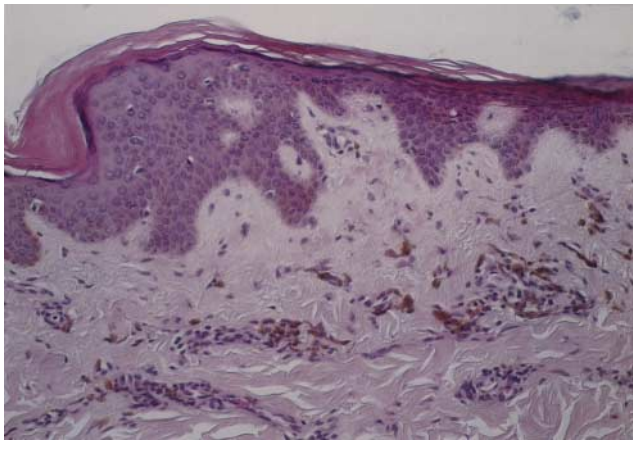

Fig. 2. Epidermis with discrete acanthosis and moderate compact hyperkeratosis. Lymphocytic perivascular infiltrate with many melanophages is seen in papillary dermis. (Haematoxylin and eosin stain × 100).

excellent response to clofazimine in an open non-controlled study (13). The underlying mechanism of action could involve a modulating effect on the cell immune response. In the future, this drug may become the treatment of choice, though further studies are required.

The development of ashy dermatosis in HIV-positive patients has been reported previously in 2 other cases. The first was a 41-year-old male (14) with no antecedents of AIDS defining or associated disorders, with a clinical context similar to that of the case described here. The second was a boy with haemophilia B, who developed the eruption following HIV seroconversion (15). The authors suggested that HIV infection should be ruled out in all patients with this skin disorder. We agree with Nelson et al. (14), in that the lack of cases published involving HIV-infected individuals is surprising, because down-regulation of the immune system may be an important element in the aetiology of this condition. The benign and asymptomatic nature of ashy dermatosis could explain why most cases go unnoticed.

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ANNOUNCEMENTS

Annual Meeting of the British Society for Investigative Dermatology in Edinburgh, UK on **April 5–7, 2000**. On April 5 meetings with the British Photodermatology Group and the Scottish Skin Biology Group will be held.

International Congress EUROGIN in Paris, France on **April 5–9, 2000**. Inf: BAXON, N. Duraincie, C. Boussin, 220224, boulevard Jean Jaurés, F-92773 Boulogne Cedex, France. Tel: 33-155202383. Fax: +33-155202393. duraincie.baxon@covos.fr. http://www.eurogin.com.

XXIst Course of Pediatric Dermatology in Arcachon, France on **April 25–28, 2000**. Inf: Prof. Alain Taïeb, Unité de Dermatologie, Hôpital Pellegrin Enfants, FR-33076 Bordeaux, France. Tel: +33-556795622. Fax: +33-556795987. alain.taieb@dermatol.ubordeaux2.fr.

27th Annual Meeting of the Society for Ultrastructural Cutaneous Research and *12th Analytic Morphology Group of the German Society for Dermatological Research (ADF)* in Bochum Germany on **May 4–6, 2000**. Inf: Joint Meeting /co Piwek, Department of Dermatology, The RuhrUniversity Bochum, Gudrunstr. 56, D-44791 Bochum, Germany. Tel: 0049 234 5093450. Fax: 0049 234 5093445. JointMeeting@derma.de.

International Dermato-Epidemiology Association (IDEA) Annual Meeting in Chicago, USA on **May 10–14, 2000**. Inf: Carolyn Charman, Honorary Secretary of IDEA, Department of Dermatology, Queen's Medical Centre, Nottingham NG7 2UH, UK. Fax: +44-115 9709003. carolyn.charman@nottingham.ac.uk.

Clinical Dermatology 2000 in Vienna, Austria on **May 18–20, 2000**. Inf: Sherryll Eady, Congress Secretariat. s.eady@ccltd.unet.com.

11th Scandinavian Society for Genitourinary Medicine (SSGM) Meeting in Kaunas, Lithuania on **May 19–21, 2000**. Inf: Andrius Ladavicius, UAB "Adveritas ir Ko" Draugystes 19, LT-3031 Kaunas, Lithuania. Tel/Fax: 00370 7 70 69 43. adveritas@takas.lt.

3rd Congress and Postgraduate Course of the European College for the Study of Vulval Diseases (ECSVD) in Jena, Germany on **May 25–27, 2000**, and *European College of HPV-associated Deseases* in Jena, Germany on **May 25, 2000**. Inf: A. Bauer or C. Greif, Department of Dermatology, Friedrich Schiller University Jena, Erfurter Str. 35, D-07740 Jena, Germany. www.kukm.de/ecsvd2000.