patches in the face. Since the age of 2 years she had been suffering from atopic eczema. She was otherwise healthy. There was especially no previous history of vitiligo, thyreopathy or other autoimmune diseases in her nor in her nearest family. The depigmentation had started when visiting Eritrea. As a prophylactic against malaria, she had been given 250 mg chloroquine weekly. After 3 weeks of therapy the parents noticed the first skin changes in their daughter. The girl experienced no subjective complaints. There were no signs of visual disturbances; she played as usual and got a lot of sun and fresh air. In all she received ten tablets of chloroquine before the family returned to Norway. Here the parents discontinued the medication. After an initial attempt with antimoniotic ointment, she was seen in our department showing the classical picture of vitiligo (Fig. 1). Clinical controls at 1, 3, 6 and 18 months (Fig. 2) showed almost complete repigmentation of the skin. The repigmentation did not seem to start perifollicularly; instead patchy repigmentation was seen.

DISCUSSION

Hypopigmentation of the skin during chloroquine therapy has been reported in 4 patients only (3–5). All of the patients were Africans with dark skin, who had been exposed to the African sun. After discontinuation of the drug therapy, the skin rapidly repigmented. In 2 of the patients, the retinopathy which developed during the malaria-prophylaxis did not improve (3). One patient had a reduced minimal erythema dose in the UVB range during the chloroquine therapy and the photopatch testing showed positive reactions to chloroquine and UVA (4). The 4th patient received chloroquine for rheumatoid arthritis. He also experienced repigmentation after cessation of therapy, but he received ascorbic acid 100 mg daily as placebo (5). Interestingly, factors which influence the binding of chloroquine to melanin are those typical of cation exchange. It has been shown that the free radical content of melanin is reduced by pretreatment with ascorbic acid (6), which could explain the clinical improvement in this patient. Abnormal reactions to sunlight have previously been observed in patients on chloroquine therapy.

In conclusion, the skin depigmentation in our patient resolved almost completely upon discontinuation of the chloroquine therapy. Although, like the other 4 patients described, she was exposed to intense sunlight, one may only speculate whether the underlying mechanism for the depigmentation is a photosensitizing reaction.

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Bullous Scabies

Sirs,

Scabies is an infestation caused by Sarcoptes scabiei. It is characterized by polymorphous lesions, like burrows, papules, nodules, excoriations and crusts, while vesicular and bullous lesions are very rare (1–11). We here report a case of bullous scabies which, on the basis of the clinical and histopathological picture, mimicked bullous pemphigoid.

CASE REPORT

A 66-year-old woman was admitted because of a widespread eruption which had appeared approximately 2 months before and was accompanied by intense pruritus. The patient was in good general condition, and before admission to our institute, she had not been given any topical or systemic treatment.

Dermatological examination showed the presence of widespread polymorphous lesions (erythema, papules, vesicles) in the axillae and on the sides, pubis, buttocks and thighs. Furthermore, bullae containing clear serous or hemorrhagic fluid, erosions and crusts on an erythemato-oedematous base were present on the arms, forearms (Fig. 1) and legs. There were also widespread linear excoriated lesions.

General physical examination was normal. Laboratory tests demonstrated a moderate increase in the inflammatory indices (ESR: 32 mm at the 1st hour; alpha 1 acid glycoprotein: 129 mg/dl) and slight leukocytosis (9,660 WBC/mm3) with eosinophils (12%). Bacteriological and mycological examinations of 2 lesions, one vesicular and the other bullous, proved negative. A Tzanck preparation was negative for acantholytic cells. The patient was subjected to 2 biopsies: one on a papulo-vesicular lesion and the other on a bullous lesion. Histopathological examination of the first biopsy showed mild acanthosis, focal spongiosis and oedema of the papillary dermis. A superficial diffuse and perivascular lymphocytic infiltrate with many eosinophils and plasma cells was also present. In the second biopsy we observed several dermo-epidermal clefts filled with fibrin and focal spongiosis. In the papillary dermis a slight perivascular lymphocytic infiltrate with many eosinophils was seen. Direct immunofluorescence (DIF) showed on two occasions granular deposits of C3 at the dermo-epidermal junction (DEJ) and in the vessel walls of the superficial dermis. Indirect immunofluorescence (IF) was negative.

On the basis of the clinical and histopathological picture, and in spite of the results of the DIF, a diagnosis of suspected bullous pemphigoid was made.

The patient was about to be treated with systemic corticosteroids when she declared that her brother too had developed a papular eruption accompanied by intense pruritus. He presented with a classical form of scabies and was in fact positive for Sarcoptes scabiei.

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The patient was then subjected to a search for the mite, which was found in both papulo-vesicular and bullous lesions.

Both patients were successfully treated with 1:10,000 potassium permanganate (1 bath/day) and hydroxyzine (40 mg/day orally), followed by 20% benzy1 benzoate cream (1 application/day). At follow-up (6 months), no recurrences were observed.

DISCUSSION

Vesiculo-bullous lesions during the course of scabies are very rare: as far as we are aware, there are only 17 published cases (1–11). The lesions are usually multiple and widespread, in only one case was a single bulla localized to the chest observed (8). Vesicular and bullous lesions appear on a more or less erythematous base. They are not always accompanied by the characteristic lesions of scabies, like burrows, papules, nodules, excoriations and crusts. The bullae can be haemorrhagic (6, 11). The localizations may be the same as those of classical scabies, but the lesions may occur in areas seldom involved in classical scabies, like the lower limbs. In particular, scabies with a prevalently vesicular component often involves the buttocks and the thighs and can mimic herpetiform dermatitis (1, 2). Prevalently bullous scabies often involves the limbs; one of its characteristics is the clinical resemblance to other bullous diseases, like bullous pemphigoid ("bullous pemphigoid-like scabies") (1, 4, 6, 7, 9), pemphigus (1), allergic contact dermatitis (1), impetigo (1), drug reactions (1), erythema multiforme (1) and epidermolysis bullosa (1, 3). From the histopathological point of view, vesicular-bullous scabies is characterized by eosinophilic spongiosis, a subepidermal cavity containing eosinophils (or a dermo-epidermal cleft at the top of the papillae) and a lymphohistiocytic infiltrate with numerous eosinophils (2, 4, 6–9, 11). The mite is only rarely found (2, 5).

On DIF the findings were: a) linear deposits of IgG and C3 (6) or IgM and C3 (9) or C5 (7) at the DEJ; b) granular deposits of IgG (6) or C3 (11) at the DEJ and c) deposits of IgM and C3 in the vessel walls (7). In case 2 DIF was negative (4, 8). The IF was carried out in 5 patients; in 4 it was negative (4, 6, 9–11), while in one case anti-basal membrane IgG at a titre of 1:10 was observed (7).

As far as the pathogenesis of vesicular-bullous scabies is concerned, 2 hypotheses have been put forward (7): a) the mite or its lytic secretions could produce an alteration of the antigen of bullous pemphigoid with the production of anti-basal membrane antibodies. These antibodies could determine an activation of the complement system with recall of inflammatory cells, including eosinophils, and release of enzymes that would produce a dermo-epidermal cleft; b) a component of the mite could act as an antigen which may cross-react with the bullous pemphigoid antigen and thereby stimulate the production of autoantibodies.

In conclusion, the case we have described demonstrates that scabies can present in multiple clinical forms and that the development of vesicular-bullous lesions is possible, although very rare.

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