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Naproxen-induced Lichen Planus Bullosus

Sir,

Certain drug provocations have been suggested in the aetiology of lichen ruber planus. We describe here a 46-year-old woman who has breast carcinoma with clinical and histological findings of bullous lichen planus, which was induced by naproxen.

CASE REPORT

A 46-year-old woman presented with a 2-month history of pruritic, violaceous maculopapular eruption. The lesions had started on her knees and elbows, then gradually spread all over the body. She had been using naproxen for osteoarthritis since 1 month before the beginning of her eruptions.

Dermatological examination revealed small, flat-topped, polygonal erythematous and violaceous papules and plaques on her trunk, arms and legs. Two weeks after the first examination, tense blisters began developing at the sites of pre-existing lesions (Fig. 1). General physical examination was normal. The patient had been diagnosed as having breast carcinoma 9 years previously. She had then undergone total mastectomy, chemotherapy and radiation therapy.

The result of laboratory tests, including urinalysis, complete blood cell count, biochemical analysis, AFP, CEA, CA-125, X-ray examination of the lung, ultrasonographic examination of abdomen, computerized tomography of the abdomen-chest and cranium, were normal. Erythrocyte sedimentation rate was 54 mm/h. Hepatitis markers and serological syphilis reactions were negative.

No casual or other provocative factors were detected other than naproxen.

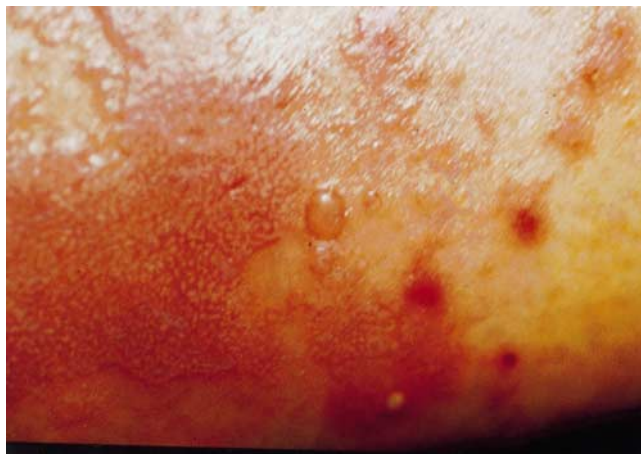


Fig. 1. Tense blister localized on typical lichen planus lesion on the leg.

A biopsy from 1 of the bullous lesions, which arose on an erythematous lesion, was taken. Hematoxylin-eosin staining showed compact orthokeratosis, epidermal hyperplasia, focal hypergranulosis, vacuolar degeneration of the basal layer, subepidermal vesicle formation and dense band-like infiltration of mononuclear inflammatory cells in the upper dermis. Direct immunofluorescence studies showed granular deposition of IgG, IgA, IgM and fibrinogen at the basement membrane zone.

Naproxen administration was stopped. She was then treated with topical corticosteroid and the lesions gradually disappeared. After the cessation of therapy, she was followed-up regularly (at 3, 6, 9 and 12 months) with no evidence of relapse.

DISCUSSION

The bullous form of lichen planus has been divided into 2 groups; lichen planus bullosus (or vesiculosus) and lichen planus pemphigoides. In the former group the blisters occur on pre-existing lichen ruber planus lesions. Conversely, the blisters appear on clinically normal skin as well as on lesional skin in the latter group. The clinical and histological findings for distinguishing bullous lichen planus and lichen planus pemphigoides are supported by immunohistological and immunobiochemical techniques (1).

Direct immunofluorescence findings of lichen ruber planus consist of deposition of IgM, IgG and C₃ on colloid bodies. In contrast to bullous lichen planus, lichen planus pemphigoides shows clump-like inflammatory infiltrate, which may also contain eosinophils. Linear deposition of C₃ and immunoglobulins at the dermoepidermal junction and colloidal bodies are detected by direct immunofluorescence microscopy (2). The clinical, histopathological and direct immunofluorescence findings of the case described here are consistent with bullous lichen planus.

The relationship between malignancy and some bullous dermatoses, such as pemphigus group, dermatitis herpetiformis and lichen planus pemphigoides, has been proposed for many years (2–4). Lichen ruber planus have also been reported in association with neoplasia (5). Although the case presented here also has a history of breast carcinoma, no relapses or metastasis were detected, so the patient is not fulfilling the reported paraneoplastic criteria (4).

Lichen planus caused by naproxen has rarely been reported (6, 7). To our knowledge this is the first case of lichen planus bullosus induced by this drug. It is reasonable to consider that any form of lichen planus might be possibly induced by naproxen.

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Shave Excision as an Adjunct to the Therapy of a Rhinophyma-like Complication in Post-kala-azar Dermal Leishmaniasis

Sir,

Post-kala-azar dermal leishmaniasis (PKDL) is an uncommon sequel in patients with a previous history of kala-azar (KA). We describe here a hitherto unreported mode of surgical excision for an unusual complication that did not regress following successful antimonial therapy.

CASE REPORT

A 40-year-old man from the eastern part of India presented with eruptions of 15 years' duration. They had commenced on the nose and cheeks, initially as transient erythema, leading to persistent induration and papules. Later, lesions appeared on the trunk, external genitalia and extremities, in that order. After a few years lesions had appeared on the tongue and glans penis. Five years prior to the onset of the eruptions he had been treated for KA elsewhere. Six years ago he had been diagnosed with PKDL and been prescribed injections of sodium antimony gluconate, but had never completed the course. The condition had then progressed and disfigured his face, which led to him presenting to us.

On examination erythematous induration was prominent on the face, studded with papules and nodules on the eyebrows, malar area, lips, chin and a few on the ears. On the nose they had coalesced, forming a large irregular nodule (Fig. 1). Well-defined, irregular hypopigmented macules were present on the back and trunk. Scattered papular lesions were seen in the axillae, trunk, upper limbs, penis and thighs. Nodular plaques were seen on the dorsa of the hands and scrotum. Papules were seen on the tongue and glans penis. The palms and soles appeared normal.

Systemic examination revealed no abnormality. Routine haemogram, urinalysis, and liver and renal function tests showed no abnormality. ECG and chest skiagram were within normal limits. Slit-skin smears stained with Giemsa and Ziehl-Neelsen technique revealed no organisms. Skin biopsy from the nose was taken for histopathology and culture. The former revealed a diffuse infiltration of the dermis by a dense mixed inflammatory infiltrate of lymphocytes, histiocytes and numerous plasma cells. Leishman-Donovan bodies (LDB) were identified in a few histiocytes. The epidermis showed hyperkeratosis and pronounced follicular plugging. Culture in Medium 199 containing 10% foetal calf serum, penicillin 10 u/ml, streptomycin 10 µg/ml and Hepes buffer pH 7.4 grew spindle-shaped, flagellated promastigote form of the parasites after 8 days of incubation at 24°C.

The patient was treated with 10 ml (1 g) of sodium antimony gluconate daily i.m. The nodules showed considerable regression after 6 weeks. Monthly ECG was done to keep a watch on cardiac function. After completing a total of 120 g in 125 days, he was asked to stop therapy. The indurated sites had completely regressed. The papules and nodules had subsided well in all areas except over the nose where the lesion had shrunk but still remained prominent. On the advice that the hypochromic macules and the nasal lesion would eventually disappear, he was kept under follow-up. Three months later he returned, stating that the lesion on the nose hampered social interactions (Fig. 2). Re-examination of the rest of the skin showed signs of subsided disease. The desire was to achieve a cosmetically acceptable result that was not socially disabling. We chose shave excision to clear the lesion on the nose. This was performed using a no. 11 scalpel blade under local anaesthesia. The bleeding points were controlled with pressure and light electrodesiccation. After completion, firm bandage was applied and changed every fourth day. Prophylactic broad-spectrum oral antibiotics were given for a week. The lesions healed without scarring in 10–14 days without any disfigurement of the nose



Fig. 1. Large irregular nasal nodule.