

LETTERS TO THE EDITOR

Hypertrophic Lichenoid Eruption due to Furosemide

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Sir,

Lichenoid drug eruption (LDE) following furosemide intake has been reported once in the literature (1). Several cases of bullous eruption (2) and generalized exanthematic pustulosis (3) caused by this drug have also been described. We report here the case of a patient who developed a hypertrophic lichen planus-like eruption two months after starting furosemide for chronic renal failure.

CASE REPORT

A 65-year-old man presented with a 2-month history of cutaneous and pruriginous lesions on his trunk and limbs. His medical history included bilateral ureterohydronephrosis with chronic renal failure, hyperuricaemia, and bladder carcinoma. He had been receiving allopurinol and calcium for 2 years and had commenced furosemide 4 months previously.

Physical examination revealed a lichenoid eruption of large violaceous papules, some coalescing into plaques, with mild hyperkeratosis on his right arm, left elbow,

lumbar region (Fig. 1A), thighs, and legs (Fig. 1B). No Wickham striae or involvement of oral or genital mucosa membranes were observed, and no unguinal lesions were detected. The only laboratory finding of interest was an increase in urea and creatinine. Hepatic viral serology (hepatitis B and C) was also negative.

Microscopic study of the epidermis revealed acanthosis and hyper- and para-keratosis with abundant hyaline or Civatte bodies at different levels, and vacuolar alteration of the basal layer. Dense lymphocytic inflammatory infiltrate was detected in the papillary dermis and dermo-epidermal interface (Fig. 2A). These changes were also observed in the upper segment of the hair follicle (Fig. 2B). Inflammatory infiltrates were preferentially CD4-positive T-lymphocytes with a few B-lymphocytes.

Histological findings were compatible with lichenoid eruption, therefore the furosemide treatment was suspended and topical corticosteroid was prescribed for 2 weeks. One year later, the lesion and pruritus were significantly improved, leaving a residual grey hyperpigmentation on lumbar region at 6 months after discontinuing furosemide.



Fig. 1. Lichenoid eruption of large violaceous papules, some coalescing in plaques, with mild hyperkeratosis on (A) lumbar region, thighs, and (B) legs. (C) Residual grey hyperpigmentation on lumbar region at 6 months after discontinuing furosemide.

DISCUSSION

Lichenoid drug eruptions mimic lichen planus and have been related to numerous drugs, including angiotensin-converting enzyme inhibitors, antimalarials, β -blockers, lithium, and non-steroidal anti-inflammatory drugs (4). In general, the latency period for drug eruptions ranges from one to 2 weeks, but it is usually longer in lichenoid eruptions, depending on the drug involved (5).

Although LDE can resemble lichen planus in localization, morphology, and histology, some differences have been described. Thus, LDE do not usually have Wickham striae, there is a lesser involvement of mucous membranes and nails, they are located symmetrically in trunk and limbs, and they leave a grey hyperpigmentation after healing (6). Bullous, ulcerative, atrophic, and hypertrophic forms of LDE have been reported (4). LDE usually also show some histological differences from lichen planus: the stratum granulosum is not hypertrophic, the dermal infiltrate generally contains eosinophils and plasmatic cells, parakeratotic foci are present, and lymphohistiocytic infiltrate obscuring the dermoepidermal junction results in keratinocyte necrosis and irregular epidermal hyperplasia (1).

Despite the above differences, the differential diagnosis between LDA and lichen planus can be challenging. We followed the method proposed by Naranjo et al. (7)

to estimate the likelihood of an adverse drug reaction, finding a probable association between furosemide and the lesions. A probable association was also found according to the Edward scale (8).

The pathogenesis of LDE remains unknown, whereas lichen planus is considered to be a T-cell mediated autoimmune skin disease. Some drugs (e.g. penicillamine or captopril) can change surface antigens and the enzyme system, precipitating an immune response (9). Cases of furosemide-related bullous eruption have been reported in photo-exposed areas, suggesting a phototoxic mechanism (10). However, the LDE in the present patient involved areas that were not photo-exposed.

The diuretic furosemide is widely used to treat oedemas due to chronic renal failure, congestive heart failure, or hypertension. Many adverse reactions have been reported for this drug, including exanthema, pruritus, urticaria, purpura, acute generalized exanthematous pustulosis (3), sweet syndrome, and bullous eruption (2). To the best of our knowledge, this is the first report of hypertrophic lichenoid eruption secondary to furosemide.

Lichen planus eruption usually resolves spontaneously a few months after discontinuing the drug. Corticosteroids, ultraviolet light and vitamin A analogues can be used.

This is only the second report of furosemide-related lichenoid eruptions and the first that shows a hypertrophic pattern. An intense residual hyperpigmentation remained at one year after furosemide withdrawal. This case illustrates the need to investigate the drug treatment of patients with dermatoses.

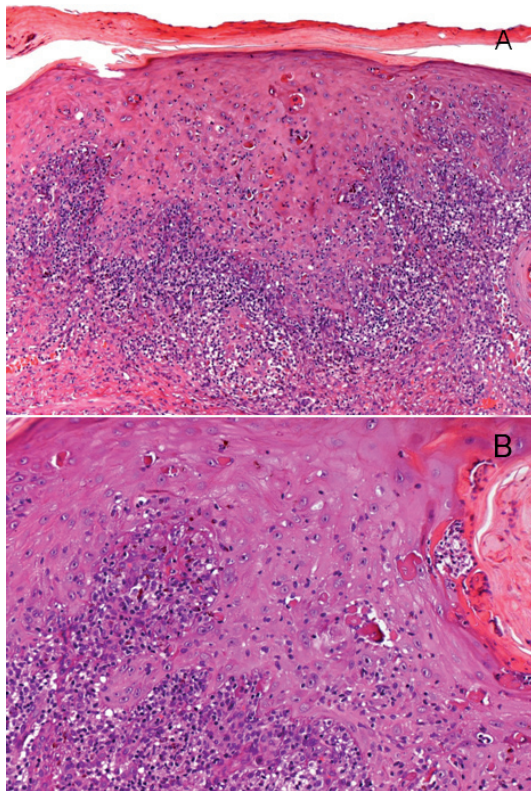


Fig. 2. (A) Intense lymphocytic infiltrate in papillary dermis obscuring the dermo-epidermal junction. (B) Abundant hyaline or Civatte bodies. Haematoxylin and eosin staining.

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