

Lichenoid drug eruption induced by erenumab



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Key words: adverse cutaneous; drug eruption; drug reaction; erenumab; lichenoid; migraine.

INTRODUCTION

Erenumab, a neuropeptide calcitonin gene-related peptide receptor antibody, is approved by the Food and Drug Administration for migraine prophylaxis. This new therapy specifically targets the trigeminal pain system. Given its mechanism and specificity, the drug has been well tolerated with few reported adverse effects. To our knowledge, a lichenoid drug eruption (LDE) secondary to erenumab has not yet been described.

CASE REPORT

A 66-year-old Hispanic man with a medical history of uncontrolled migraines despite multiple therapies presented with a pruritic eruption without any systemic symptoms, recent illness, or infection. He had no prior dermatologic history, including no similar eruptions in the past. The only recent adjustment to his medication regimen was the addition of erenumab for migraine prophylaxis. His other medications were amitriptyline, valproate, methocarbamol, sumatriptan, omeprazole, losartan, amlodipine, citalopram, and atorvastatin. The patient reported that the eruption began on his upper chest and forearms 2 to 3 weeks after starting therapy with subcutaneous injections of erenumab. Following a second injection in the subsequent month, the eruption considerably worsened to involve nearly the entire body surface area, with associated intense pruritus. The patient completed a 14-day course of a prednisone taper prescribed by his primary care physician that initially helped, but the rash flared after completion. Treatment with erenumab was ceased following the second

Abbreviation used:

LDE: lichenoid drug eruption

injection. The patient continued migraine abortive treatment with sumatriptan but did not receive alternative prophylactic therapy.

On initial evaluation, physical examination was remarkable for planar, violaceous papules coalescing into plaques involving the trunk and extremities, especially on the inner arms, volar wrists, dorsal feet, and lower legs and abdomen (Fig 1). There was no mucosal involvement. A 4-mm punch biopsy sample from a characteristic lesion on the left arm was obtained. Microscopic examination of the biopsy specimen revealed an acanthotic epidermis with jagged rete ridges, hypergranulosis, and scattered dyskeratosis, as well as a lichenoid band of lymphocytes with rare eosinophils (Fig 2). A diagnosis of LDE was made and treatment with triamcinolone cream twice daily was initiated. The patient demonstrated substantial clinical improvement approximately 10 to 14 days after starting the topical steroid application, with complete resolution at a 5-week follow-up.

DISCUSSION

LDE, or drug-induced lichen planus, is a rare, T-cell mediated, cutaneous adverse event of a myriad of drugs. The most common culprits include antihypertensives, diuretics, antimalarials, non-steroidal antiinflammatory drugs, and antitubercular drugs.¹

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Fig 1. Lichenoid drug eruption due to erenumab. Planar, violaceous papules on the (A, B) extremities and (C) trunk.

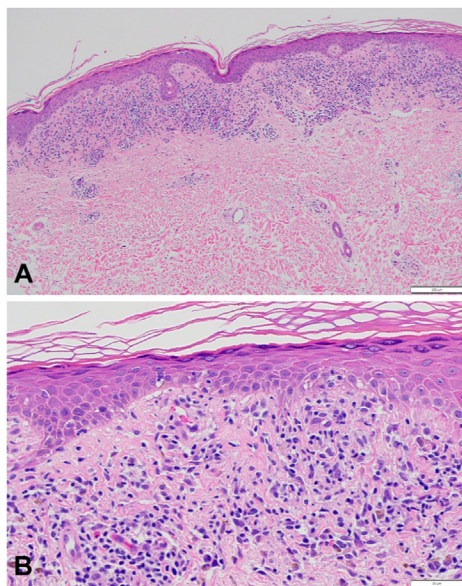


Fig 2. Acanthotic epidermis with jagged rete ridges, hypergranulosis, and scattered dyskeratosis, as well as a lichenoid band of lymphocytes with rare eosinophils. (Hematoxylin-eosin stain; original magnifications: A, $\times 100$; B, $\times 400$.)

Although similar to lichen planus, LDE commonly presents with symmetric, violaceous to hyperpigmented papules, often photo-distributed and devoid of Wickham striae or mucosal involvement.² Histologically, the presence of an eosinophilic infiltrate in LDE helps to differentiate it from lichen planus.² Onset of LDE is variable, with reports ranging from 10 days to several years after the initiation of a drug.³ Drug cessation is the mainstay of treatment, with resolution typically within weeks, but topical steroids may also be used to hasten resolution.³ Additionally, oral steroids, antihistamines, and topical calcineurin inhibitors have been reported to be useful.⁴

For patients with migraines, there are various treatment options with unique mechanisms of action. However, poor efficacy and tolerability to existing treatments limit options for patients. Erenumab is a new, useful alternative, with efficacy in patients who have failed 2 to 4 previous medication treatments with a favorable side effect profile.⁵ Apart from injection site reactions, the only cutaneous adverse effects reported due to erenumab are a case of symmetrical drug-related intertriginous and flexural exanthema and impaired wound healing.⁵⁻⁷

LDE due to biological therapy is rare, though tumor necrosis factor- α inhibitors are the most frequently implicated.⁸ In cases of biological therapy-induced LDE, the median onset time is 3 months.⁸ Rechallenge with biological therapy typically results in the recurrence of lesions but class-switching is a viable alternative.⁸ The exact mechanism for biological therapy-related LDE is not fully understood but is thought to be due to shifts in cutaneous immune response patterns.⁹ Although few adverse cutaneous effects due to erenumab have been reported, clinicians should consider the possibility of LDE with erenumab.

Conflicts of interest

None disclosed.

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