Unilateral vancomycin-induced linear IgA bullous dermatosis

Sir,

We report a new case of vancomycin-induced linear IgA bullous dermatosis with unilateral distribution of lesions. A 77-year-old man with a medical history of diabetes, hypertension, and coronary artery disease was admitted for infective endocarditis. He was treated with vancomycin, gentamicin, and ampicillin. Twenty days later, the patient developed multiple, tense, bullous lesions on an erythematous base and few erosions, involving the right side of his body: neck, trunk, wrist, and thigh [Figures 1 and 2]. Nikolsky sign was negative. No other areas were affected. No mucosal involvement was noted. Initially, we suspected that he was suffering from a drug eruption. So, we discontinued all the drugs other than vancomycin. This drug was continued because the infectious endocarditis and sepsis had not yet been adequately controlled. New lesions appeared, leading to the withdrawal of vancomycin. There were no additional lesionsafter the withdrawal of vancomycin. The skin eruption improved rapidly without any treatment. A biopsy specimen of involved skin showed subepidermal bullae and an upper dermal neutrophilic infiltrate with some eosinophils [Figure 3]. Direct immunofluorescence revealed a linear band of IgA at the dermoepidermal junction [Figure 4]. The diagnosis of vancomycin-induced linear IgA bullous dermatosis was established.

In our patient, this diagnosis was made based on the chronological plausibility, a Naranjo score of 6 (probable linear IgA bullous dermatosis), and previous reports concerning this reaction. Induced linear IgA bullous dermatosis eruption starts two to 21 days after initiating the drug and remission occurs 1–21 days after drug withdrawal. Vancomycin is the most common drug that induces this dermatosis, though there are. other drugs that have been incriminated (penicillins, cephalosporins, antiepileptic drugs, calcium channel blocker nonsteroidal anti-inflammatory drugs, etc.).[1]

Clinically, it presents with a generalized vesiculobullous eruption involving generally the trunk and extremities, with herpetiform arrangement of bullae on an erythematous base or on normal-appearing skin. This is commonly known as the cluster of jewels sign. Mucosal involvement is possible, but it is more frequently seen in idiopathic presentations. Histology reveals a subepidermal bulla with neutrophil predominance at the dermoepidermal junction. In perilesional skin, direct immunofluorescence demonstrates linear deposition of IgA along the dermoepidermal junction.

Drug-induced linear IgA bullous dermatosis is characterized by significantly more atypical and severe forms. It may show heterogeneous presentations such as: urticarial eruption, eczematous patches, morbilliform rash, toxic epidermal necroslysis, bullous pemphigoid like eruption, and erythema multiforme-like eruption. Further, localized vancomycin-induced linear IgA bullous dermatosis confined to the palms has been reported.[2] There is also another report of vancomycin induced linear IgA bullous dermatosis occurring at the infusion site, caused by its extravasation.[3]

The unique feature in our case is the unilateral distribution of lesions. Our patient was bedridden in the same position for several days. Lesions predominated in pressure zone, and could be probably attributed to a Koebner phenomenon. Three cases of vancomycin-induced linear IgA bullous dermatosis, with skin lesions occurring at the sites of trauma or friction in previously uninvolved skin, have been reported in the literature[4,5]. Trauma may generate an increase in blood

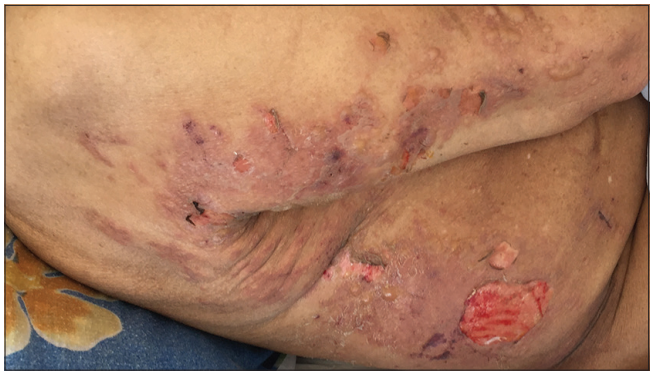

Figure 1: Unilateral tense bullous lesions before VCM withdrawal with large erosions on the right side of trunk; pressure zones

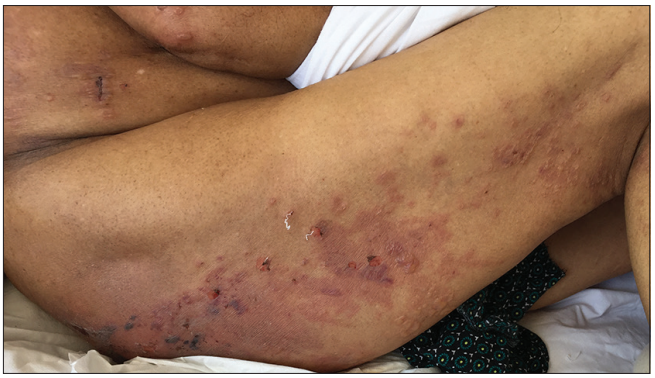

Figure 2: Unilateral bullous lesions on an erythematous base and some erosions involving only the patient's right thigh

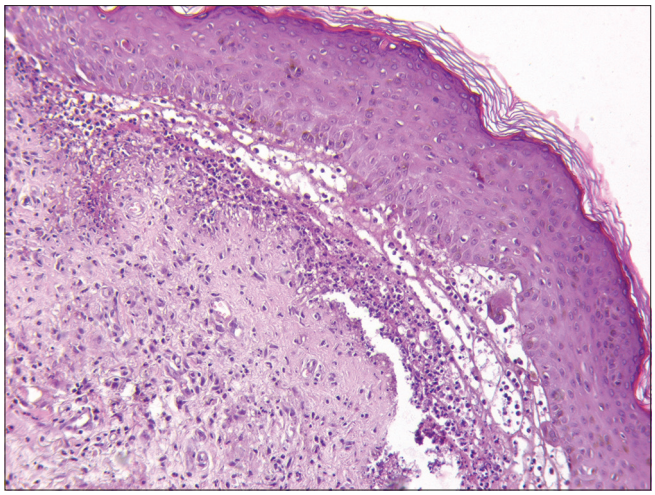

Figure 3: A biopsy specimen of Involved skin from the patient showed subepidermal bullae and an upper dermal neutrophilic infiltrate with some eosinophils (H and E, ×100)

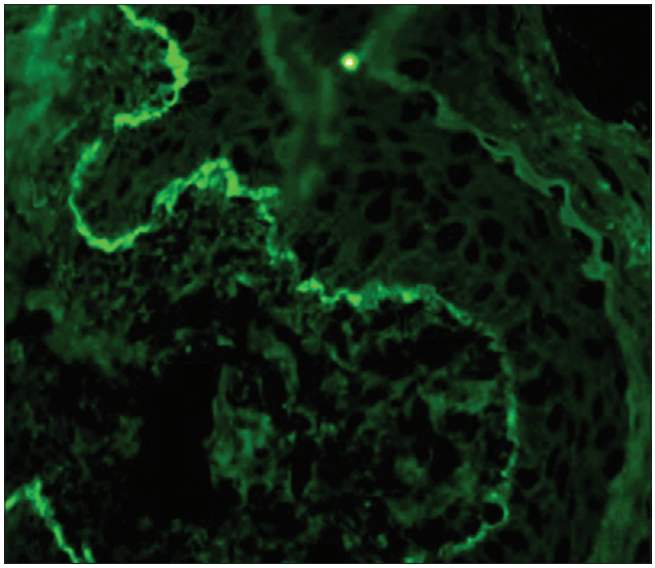

Figure 4: Direct immunofluorescence shows strong linear staining of the basement membrane along the dermoepidermal junction with IgA

flow locally, which attracts more autoantibodies to the site and thus results in blister-like lesions.[5]

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Conflicts of interest
There are no conflicts of interest.

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