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CASE REPORT

Sulfasalazine-Induced Linear Immunoglobulin A Bullous Dermatositis with DRESS[☆]

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KEYWORDS

Sulfasalazine;
Rheumatoid arthritis;
Bullous dermatosis;
Adverse skin reaction

PALABRAS CLAVE

Sulfasalazina;
Artritis reumatoide;
Dermatosis
ampollosa;
Reacción adversa
cutánea

Abstract Linear immunoglobulin (Ig) A dermatosis is an immune-mediated bullous disease characterized by linear deposits of IgA along the basal membrane. While usually idiopathic, it can occasionally be induced by drug exposure. We report the case of a 60-year-old woman with rheumatoid arthritis being treated with sulfasalazine who developed linear IgA dermatosis and drug rash with eosinophilia and systemic symptoms (DRESS). The dermatosis and associated symptoms resolved following withdrawal of the drug and treatment with systemic corticosteroids for 2 months. This is the first report of sulfasalazine-induced linear IgA dermatosis in association with DRESS and we believe that sulfasalazine should be added to the list of drugs that can cause linear IgA dermatosis.

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Dermatosis ampollosa inducida por inmunoglobulina A lineal con clínica de síndrome DRESS por sulfasalazina

Resumen La dermatosis IgA lineal es una enfermedad ampollosa mediada inmunológicamente que se define por presentar un depósito lineal de IgA a lo largo de la membrana basal. Habitualmente es idiopática y ocasionalmente se asocia con algunos fármacos. Describimos el caso de una mujer de 60 años con artritis reumatoide en tratamiento con sulfasalazina, que desarrolló un cuadro de dermatosis IgA lineal con clínica de DRESS (drug-rash with eosinophilia and systemic symptoms) el cual respondió al suspender el fármaco causal más tratamiento con corticoides sistémicos durante dos meses. Este es el primer caso descrito de dermatosis IgA lineal con clínica de DRESS relacionado con la sulfasalazina. Creemos que es importante tener en cuenta esta asociación para poder incluir a la sulfasalazina en el listado de fármacos que pueden producir dermatosis IgA lineal por fármacos.

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Introduction

Sulfasalazine is a sulfonamide-derived drug with anti-inflammatory properties that is used in the treatment of rheumatoid arthritis and inflammatory bowel disease. Although a maculopapular rash is the most common cutaneous reaction caused by this treatment,¹ other potential reactions should not be ignored.

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We treated a case of linear IgA dermatosis (LAD) secondary to sulfasalazine treatment which presented as drug-rash with eosinophilia and systemic symptoms (DRESS) in a patient with rheumatoid arthritis.

Case Description

The patient was a 60-year-old woman with a history of rheumatoid arthritis, systemic hypertension, and dyslipidemia, who was receiving treatment with deflazacort (18 mg/d), diclofenac (50 mg/d), omeprazole (20 mg/d), and a combination of hydrochlorothiazide and valsartan (150/12.5 mg). She was prescribed treatment with sulfasalazine (2 g/d) for joint pain. Three weeks later the patient presented to the emergency department with fever and skin lesions on the trunk and limbs that had appeared 2 weeks previously. Physical examination revealed general malaise, fever, hypotension, and tachycardia. Confluent erythematous macules and papules that formed moderately infiltrated plaques with poorly defined borders affected 70% of the body surface area. Tense blisters filled with a clear fluid were present on both legs and the dorsum of the hands (Figures 1 and 2), but the Nikolsky sign was negative. No mucosal lesions or lymphadenopathy were detected. Blood tests revealed a white blood cell count of $15\,700/\mu\text{L}$ (normal range, $4500\text{--}11\,000/\mu\text{L}$) (54.5% neutrophils [normal range, 40.0%–70.0%] and 8.4% eosinophils [normal range, 0.00%–5.00%]) and eosinophilia of $1320/\mu\text{L}$; erythrocyte



Figure 2 Tense blisters filled with clear fluid on the dorsum of the hands.

sedimentation rate, 42 mm/h; alanine aminotransferase, 44.5 U/L (normal range, 7–40 U/L); γ -glutamyltransferase, 452.3 U/L (normal range, 12–54 U/L); lactate dehydrogenase, 716.1 U/L (normal range, 230–460 U/L); and C-reactive protein, 9.88 mg/dL. Serology for Epstein-Barr virus (EBV) was positive (IgM and IgG) and abdominal ultrasound showed no abnormal findings. Skin biopsy revealed a blister caused by dermoepidermal detachment with a neutrophil-rich infiltrate (Fig. 3) and on direct immunofluorescence there were



Figure 1 Erythematous papules coalesced to form firm plaques on the lower limbs, accompanied by blisters filled with clear fluid.

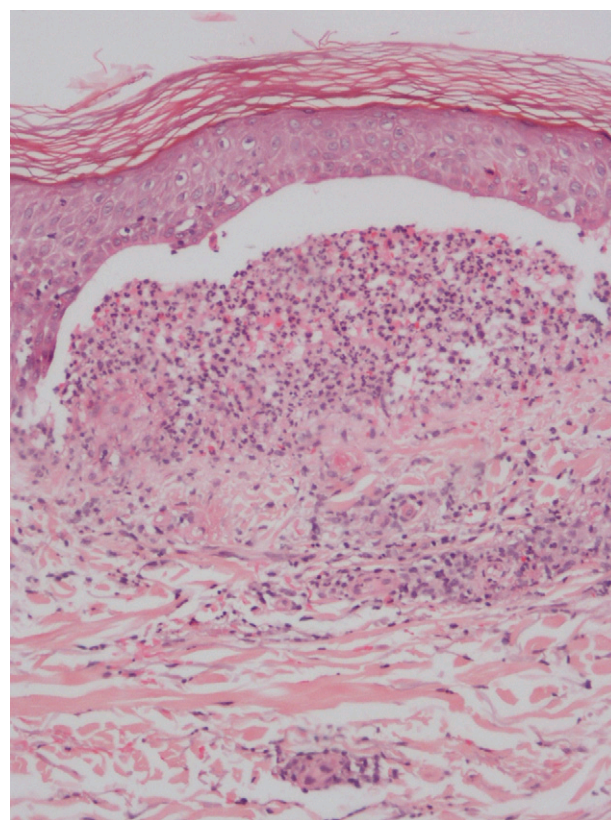


Figure 3 Dermoepidermal detachment with a neutrophil-rich inflammatory infiltrate. Hematoxylin-eosin, original magnification $\times 10$.

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