

Images in Allergy

Proton Pump Inhibitor–Induced Subacute Cutaneous Lupus Erythematosus

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Drug-induced subacute cutaneous lupus erythematosus (DI-SCLE) secondary to proton pump inhibitor (PPI) is a rare phenomenon, though its prevalence and incidence have risen in recent years. From 2009 to 2016 alone, reported cases of PPI-induced SCLE have increased by 34.1%.¹ Several authors have speculated that this is likely due to changes in PPI-prescribing practices. For instance, Hoffman et al² found that, over the years 2005 to 2013, the prescribing prevalence of PPIs almost doubled from 8.2% to 16.2%. Furthermore, PPIs are now available for over-the-counter use. Given this remarkable increase in the prevalence and incidence of DI-SCLE, we hope our report will improve the awareness surrounding this phenomenon.

Here, we present the case of a 79-year-old white male with a history of coronary artery disease, hypertension, diabetes mellitus, and Barrett esophagus, who was admitted for large-volume hematemesis requiring intubation. A pantoprazole drip was initiated for a presumed upper gastrointestinal bleeding source and was continued for 8 days. After esophagogastroduodenoscopy, the patient's bleeding was discovered to be secondary to esophageal ulcers. The patient was discharged on oral pantoprazole 40 mg twice daily.

After 2 weeks on oral pantoprazole, he noticed scattered erythematous, pruritic papules with scaling on his shoulders, upper back, forearms, head, thighs, and feet—all sun-exposed areas (Figure 1, A and B). There was no mucosal involvement or blistering of the lesions. His primary care doctor discontinued the pantoprazole approximately 4 weeks after initiation and switched to ranitidine. On further investigation, the patient recalled a similar skin eruption after taking omeprazole and rabeprazole 20 years earlier that resolved on discontinuation of these drugs. He had avoided PPIs until this recent hospitalization.

Subsequently, he was evaluated in our clinic. Because of the suspicion of drug-induced rash, he was told to continue strict avoidance of PPIs and sent to Dermatology for a skin biopsy. Skin biopsy demonstrated severe vacuolar interface dermatitis composed of a brisk lymphocytic infiltrate with associated dermal edema and focal hyperkeratosis. Laboratory tests were notable for positive antinuclear antibodies with a titer greater than 1:1280 (speckled pattern), positive anti-Ro antibodies, and positive antihistone antibodies. The presence of positive antinuclear antibodies, anti-Ro antibodies, and anti-La antibodies was each strongly associated with DI-SCLE. In comparison, the presence of antihistone and/or

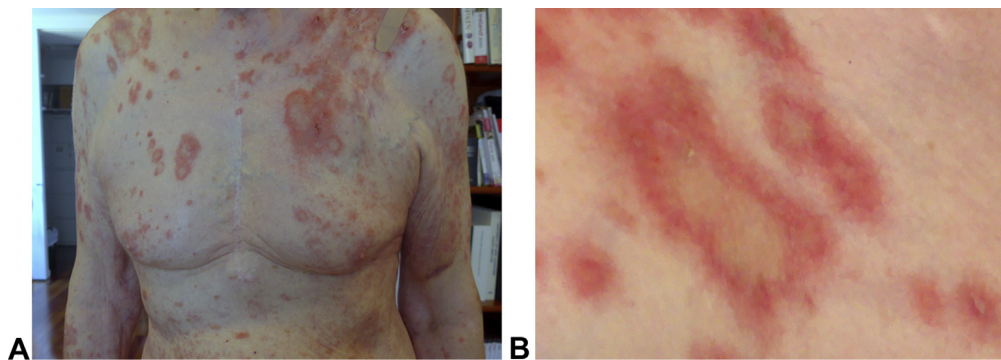


FIGURE 1. (A) Diffuse annular lesions characteristic of DI-SCLE present on the chest of our patient. (B) A close-up view of the classic annular lesions noted in DI-SCLE.

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anti–double-stranded DNA antibodies is more suggestive of drug-induced systemic lupus erythematosus. In our patient, complete blood cell count, chemistry panel, liver function tests, and complement levels were all normal. Symptomatic relief was achieved with oral cetirizine and topical triamcinolone cream applied to the affected areas. On a subsequent visit 4 weeks later, the patient noted complete resolution of the rash.

We conclude by emphasizing that prompt evaluation is key to the treatment of DI-SCLE secondary to PPI use. Although it is a

rare phenomenon, resolution of symptoms can be achieved with removal of the offending agent.

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