Levofloxacin-Induced Purpura Annularis Telangiectodes of Majocchi

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PRACTICE **POINT**

- Purpura annularis telangiectodes of Majocchi, a type of pigmented purpuric dermatosis, may on occasion be triggered by a medication; therefore, a careful medication history may prove to be an important part of the workup for this eruption.

To the Editor:

Purpura annularis telangiectodes of Majocchi (PATM) is a type of pigmented purpuric dermatosis (PPD). Patients present with nonblanchable, annular, symmetric, purpuric, and telangiectatic patches, often on the legs, with histology revealing a perivascular lymphocytic infiltrate and extravasated erythrocytes.[1,2] A variety of medications have been linked to the development of PPD. We describe a case of levofloxacin-induced PATM.

A 42-year-old man presented with a rash on the arms, trunk, abdomen, and legs of 1 month's duration. He reported no associated itching, bleeding, or pain, and no history of a similar rash. He had a history of hypothyroidism and had been taking levothyroxine for years. He had no known allergies and no history of childhood eczema, asthma, or allergic rhinitis. Notably, the rash started shortly after the patient finished a 2-week course of levofloxacin, an antibiotic he had not taken in the past. The patient resided with his wife, 3 children, and a pet dog, and no family members had the rash. Prior to presentation, the patient had tried econazole cream and then triamcinolone acetonide cream 0.5% without any clinical improvement.

A complete review of systems was unremarkable. Physical examination revealed scattered, reddish brown, annular, nonscaly patches on the back, abdomen (Figure 1), arms, and legs with nonblanching petechiae within the patches.

A punch biopsy of the left inner thigh demonstrated patchy interface dermatitis, superficial perivascular inflammation, and numerous extravasated red blood cells in the papillary dermis (Figure 2). The histologic features were compatible with the clinical impression of PATM. The patient presented for a follow-up visit 2 weeks later with no new lesions and the old lesions were rapidly fading (Figure 3).

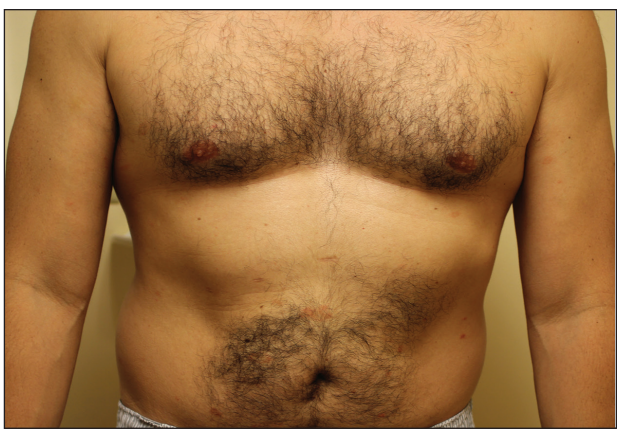

FIGURE 1. Purpura annularis telangiectodes of Majocchi with scattered, reddish brown, annular, nonscaly patches on the trunk and nonblanching petechiae within the patches.

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The authors report no conflict of interest.

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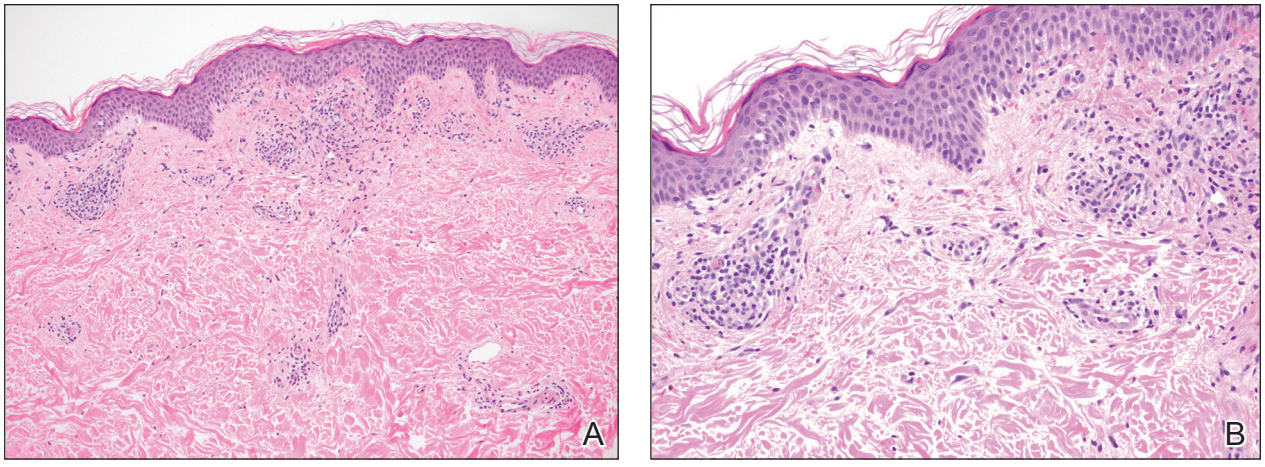

FIGURE 2. Purpura annularis telangiectodes of Majocchi histology demonstrated patchy interface dermatitis, superficial perivascular inflammation, and numerous extravasated red blood cells in the papillary dermis (A and B)(both H&E, original magnifications ×10 and ×20).

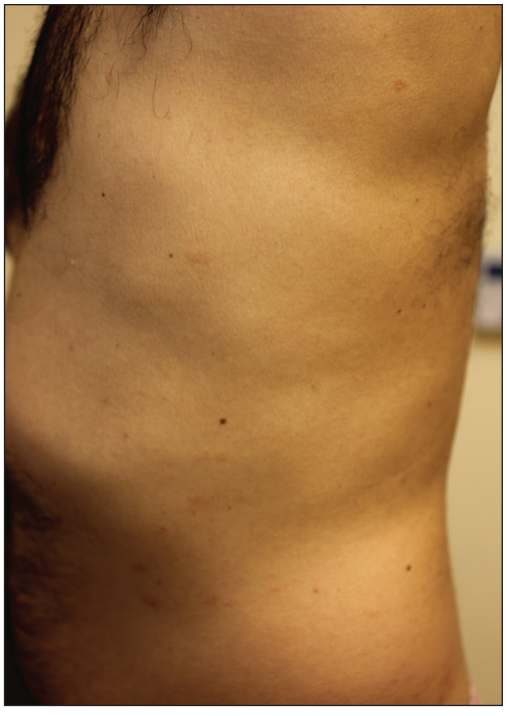

FIGURE 3. Clearance of purpura annularis telangiectodes of Majocchi lesions on the abdomen after discontinuation of levofloxacin.

Pigmented purpuric dermatoses are a group of conditions that have different clinical morphologies but similar histopathologic examinations.[2] All PPDs are characterized by nonblanching, nonpalpable, purpuric lesions that often are bilaterally symmetrical and present on the legs.[2,3] Although the precise etiology of these conditions is not known, most cases include a perivascular lymphocytic infiltrate along with the presence of extravasated erythrocytes and hemosiderin deposition in the dermis.[2] Of note, PATM often is idiopathic and patients usually present with no associated comorbidities.[3] The currently established PPDs include progressive pigmentary dermatosis (Schamberg disease), PATM, pigmented purpuric lichenoid dermatosis of Gougerot and Blum, lichen aureus, and eczematidlike purpura of Doucas and Kapetanakis.[2,4]

The lesions of PATM are symmetrically distributed on the bilateral legs and may be symptomatic in most cases, with severe pruritus being reported in several drug-induced PATM cases.[3,5] Although the exact etiology of PPDs currently is unknown, some contributing factors that are thought to play a role include exercise, venous stasis, gravitational dependence, capillary fragility, hypertension, drugs, chemical exposure or ingestions, and contact allergy to dyes.[3] Some of the drugs known to cause drug-induced PPDs fall into the class of sedatives, stimulants, antibiotics, cardiovascular drugs, vitamins, and nutritional supplements.[3,6] Some medications that have been reported to cause PPDs include acetaminophen, aspirin, carbamazepine, diltiazem, furosemide, glipizide, hydralazine, infliximab, isotretinoin, lorazepam, minocycline, nitroglycerine, and sildenafil.[3,7-15]

Although the mechanism of drug-induced PPD is not completely understood, it is thought that the ingested substance leads to an immunologic response in the capillary endothelium, which results in a cell-mediated immune response causing vascular damage.[3] The ingested substance may act as a hapten, stimulating antibody formation and immune-mediated injury, leading to the clinical presentation of nonblanching, symmetric, purpuric, telangiectatic, and atrophic patches at the site of injury.[1,3]

Levofloxacin is a broad-spectrum antibiotic that has activity against both gram-positive and gram-negative bacteria. It inhibits the enzymes DNA gyrase and topoisomerase IV, preventing bacteria from undergoing proper DNA synthesis.[16] Our patient's rash began shortly after

a 2-week course of levofloxacin and faded within a few weeks of discontinuing the drug; the clinical presentation, time course, and histologic appearance of the lesions were consistent with the diagnosis of drug-induced PPD. Of note, solar capillaritis has been reported following a phototoxic reaction induced by levofloxacin.[17] Our case differs in that our patient had annular lesions on both photoprotected and photoexposed skin.

The first-line interventions for the treatment of PPDs are nonpharmacologic, such as discontinuation of an offending drug or allergen or wearing supportive stockings if there are signs of venous stasis. Other interventions include the use of a medium- or high-potency topical corticosteroid once to twice daily to affected areas for 4 to 6 weeks.[18] Some case series also have shown improvement with narrowband UVB treatment after 24 to 28 treatment sessions or with psoralen plus UVA phototherapy within 7 to 20 treatments.[19,20] If the above measures are unsuccessful in resolving symptoms, other treatment alternatives may include pentoxifylline, griseofulvin, colchicine, cyclosporine, and methotrexate. The potential benefit of treatment must be weighed against the side-effect profile of these medications.[2,21-24] Of note, oral rutoside (50 mg twice daily) and ascorbic acid (500 mg twice daily) were administered to 3 patients with chronic progressive pigmented purpura. At the end of the 4-week treatment period, complete clearance of skin lesions was seen in all patients with no adverse reactions noted.[25]

Despite these treatment options, PATM does not necessitate treatment given its benign course and often self-resolving nature.[26] In cases of drug-induced PPD such as in our patient, discontinuation of the offending drug often may lead to resolution.

In summary, PATM is a PPD that has been associated with different etiologic factors. If PATM is suspected to be caused by a drug, discontinuation of the offending agent usually results in resolution of symptoms, as it did in our case with fading of lesions within a few weeks after the patient was no longer taking levofloxacin.

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