A Curious Case of Drug-induced Subacute Cutaneous Lupus Erythematosus

Sir,

Drugs causing drug-induced systemic lupus erythematosus (DI-SLE) are different from drugs drug-induced subacute causing cutaneous lupus erythematosus (DI-SCLE) except for tumor necrosis factor antagonists.[1] Isoniazid is known to cause DI-SLE. However, isoniazid causing DI-SCLE has been infrequently described in the literature.

A 60-year-old female, a known case of tubercular osteomyelitis, presented with multiple erythematous scaly lesions over the body for the last 2 months. The patient was on antitubercular drugs HRZE (isoniazid, rifampicin, pyrazinamide, and ethambutol (HRZE) for 2 months and isoniazid, rifampicin, and ethambutol (HRE) for 2 months. The lesions developed after nearly 8 weeks of intake of HRZE. The cutaneous examination showed multiple annular erythematous scaly plaques over the face, trunk, and limbs with accentuation in the photo-exposed sites along with multiple targetoid lesions [Figure 1a and b]. The complete hemogram showed eosinophilia of 11%. The liver function test, renal function test, chest X-ray, 24-hour urine protein, and electrocardiogram (ECG) were within normal limits. Antinuclear antibody (ANA) was positive (1:90, homogenous pattern). The anti Ro/La, antihistone, anti dsDNA, antismith antibodies, and rheumatoid factor were negative. Skin biopsy revealed basal cell vacuolization, colloid bodies, pigment incontinence, infiltration of the vessel wall by inflammatory cells, and karyorrhectic debris [Figure 2a]. The patient was diagnosed as a case of SCLE and started on hydroxychloroquine 300 mg daily along with oral prednisolone 30 mg daily for 2 weeks, topical mometasone, and sunscreen; however, the lesions persisted. Because of the refractory nature of lesions, the drug was suspected as a possible trigger. As isoniazid is more commonly incriminated in systemic lupus erythematosus, isoniazid was stopped and replaced by oral

a

Figure 1: Multiple erythematous annular scaly plaques over the gluteal area (a). Close-up photograph of the thigh showing annular scaly plaques and targetoid lesions (b)

levofloxacin. Rifampicin, ethambutol, and levofloxacin were continued along with levocetrizine and topical mometasone, and oral steroids were stopped. After 2 weeks of stopping isoniazid, there was a significant improvement in cutaneous lesions with complete subsidence of lesions at 4 weeks [Figure 2b]. The patient is on follow-up and has not reported any new lesions or systemic symptoms since then.

DI-LE has predominantly systemic symptoms such as fever, arthritis, and serositis, whereas LE-specific skin changes are relatively uncommon.^[2-4] However, in DI-SCLE, there are specific cutaneous lesions and systemic features are minimal or absent. Anti-histone antibodies are frequently present in DI-LE,^[3] whereas anti Ro/SSA antibodies are more frequently positive in DI-SCLE,^[2]

It is also important to identify and differentiate this subset of patients from idiopathic SCLE (I-SCLE) as removal of the culprit drug is the mainstay of management in DI-SCLE. DI-SCLE usually has an older age of onset than I-SCLE. [3,4] Latent period between exposure to the drug and the onset of symptoms is 4 to 8 weeks. [2] The disease is more widespread, and involves areas that are usually spared by I-SCLE. Bullous, erythema multiforme—like lesions, purpura, necrotic-ulcerative lesions, malar rash, and the involvement of lower legs are more common in DI-SCLE. [3,4]

Recently, one case of isoniazid-induced SCLE was described in a HIV-positive female. [5] In our patient, temporal correlation with the intake of drugs, widespread involvement including legs, erythema multiforme—like lesions, and eosinophilia suggested drug as a possible etiology. The patient did not meet the criteria for Rowell syndrome, [6] and SLE was ruled out because of lack of systemic involvement.

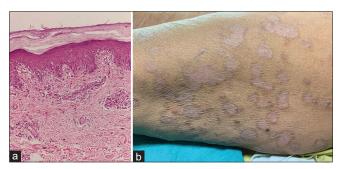


Figure 2: (a) Skin biopsy showing hyperkeratosis, parakeratoses, basal layer vacuolization, colloid bodies, and infiltration of the vessel wall by inflammatory cells. (H & E 10×). (b) Subsiding lesions over the thigh after 2 weeks of stopping isoniazid

The mainstay of treatment is the discontinuation of the culprit drug which leads to the resolution of lesions in a period of nearly 6 to 12 weeks.^[4] Other treatment modalities are topical steroids, topical tacrolimus, hydroxychloroquine, and oral steroids.^[2]

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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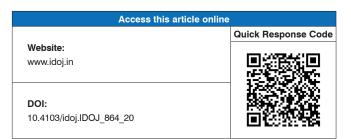
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