

Amlodipine-Induced Subacute Cutaneous Lupus

Dear Editor,

A 62-year-old man was seen in Cardiology clinic, where he complained of a worsening asymptomatic skin eruption on both arms and shoulders for 3 months duration. He had no history of cutaneous disease and was concerned about a possible drug reaction. The patient had a 4-year history of coronary artery disease; he had status post vein graft percutaneous coronary interventions and multiple stent implantations. Four weeks prior to the onset of the skin eruption he was started on amlodipine. His medications also included, isosorbide dinitrate, lisinopril, metoprolol, aspirin, clopidogrel, finofibrate, simvastatin and ranitidine. He was referred to Dermatology with non-pruritic, erythematous, scaling plaques on his arms and chest. The total body surface area involved was 7% [Figure 1]. Skin biopsy revealed perivascular inflammation with disruption of the epidermal-dermal junction consistent with subacute cutaneous lupus erythematosus (SCLE) [Figure 2]. Laboratory studies were positive for anti-RO (SS-A), negative for antinuclear antibodies (ANA) and anti-LA (SS-B), with a normal blood count and metabolic panel. He was instructed to apply topical triamcinolone 0.1% and to stop amlodipine, while increasing his dose of isosorbide. He had a 4 week follow-up and the skin eruption was much improved. When seen in Cardiology clinic 6 months later his eruption had disappeared.

SCLE is a subset of cutaneous lupus erythematosus described in 1979.^[1] It presents as a non-scarring, annular, papulosquamous eruption on sun exposed skin, primarily on the upper torso, arms, and lateral neck, in conjunction with anti-Ro/SS-A antibodies.^[2] Antinuclear antibodies were positive in about 60-80% of the patients,^[3,4] more often found in the annular presentation rather than the papulosquamous form.^[4] About one-third of all SCLE cases could be attributed to a previous drug exposure.^[5]

Drug-induced subacute cutaneous lupus erythematosus (DI-SCLE) occurs as an adverse reaction to certain medications, especially anti-hypertensives and anti-fungals.^[3] Within the anti-hypertensive group diuretics, beta-blockers, angiotensin conversion enzyme inhibitors and calcium channel blockers have been



Figure 1: Drug-induced subacute lupus erythematosus: Erythematous and scaly plaque on a left posterior arm

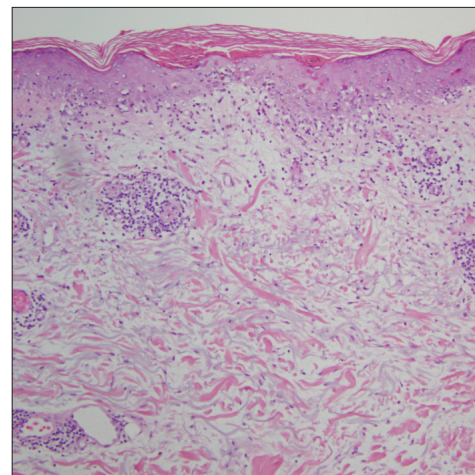


Figure 2: Drug-induced subacute lupus erythematosus (×20): Slight parakeratosis with mild epidermal atrophy, liquefaction of the basal layer and perivascular and periadnexal lymphocytic infiltrate

reported as possible causes of DI-SCLE. Within the calcium channel blocker group diltiazem, verapamil, nifedipine, and nitrendipine have been implicated.^[2,3,5] There is no previous report of amlodipine and DI-SCLE in the literature; however, there is a single report of a patient in one study, where the combination of benazepril/amlodipine was associated with DI-SCLE,^[6] but this does not rule out the possibility that benazepril was the culprit. In addition to this, our patient's lesions resolved upon withdrawal of amlodipine, which makes lisinopril and simvastatin less likely culprits.

Amlodipine is one the most common medications used to treat hypertension and angina, making this adverse event potentially significant. Although, there are reports of DI-SCLE persisting after withdrawal of the suspected drug, DI-SCLE is usually reversible

once the offending drug is discontinued. Thus, it is important to include drug-induced SCLE in the differential diagnosis of eruptions in patients taking anti-hypertensive medications and obtain a complete medication history.

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