

CLINICAL VIGNETTE

Lichenoid Dermatitis with Olmesartan-hydrochlorothiazide Use

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Abstract

Lichenoid dermatitis also known as lichenoid drug eruption is an uncommon cutaneous side effect associated with many classes of medications. The time frame between the initiation of the offending drug and the presentation can vary depending on the class of the drug, dose and other medications used.¹ Angiotensin-converting enzyme inhibitors, Angiotensin receptor blocker, thiazide diuretic and beta-blocker have been associated with cutaneous lichenoid dermatitis, which usually spare the mucous membrane and is typically distributed in sun exposed area.² We report lichenoid dermatitis in response to use of antihypertensive medication. The diagnosis of the skin lesions was clinical with typical presentation of symmetric eruption of violaceous patches in the setting of presence of offending drug and was confirmed with biopsy. Clinical course showed resolution of the lesions upon drug withdrawal and reoccurrence upon re-challenge.

Case Presentation

A 53-year-old female presented with an 8-month history of an itchy violaceous skin eruption on both hands, ankles and feet. Lesions were not responsive to low potency topical steroids. There was no personal or family history of eczema or other allergic diseases. She has high blood pressure that was under control with olmesartan-hydrochlorothiazide 40-12.5 mg which she has been taking for more than a year. She was also taking levothyroxine 125 mcg for hyperthyroid and hyperlipidemia treated with atorvastatin for elevated cardiac risk. On skin examination there were multiple scattered purplish polygonal plaques with slight overlying scale and a few annular plaques with dusky center along the dorsal hands/wrists and dorsal feet/ankles and was referred to Dermatology. She was suspected to have lichenoid planus versus granuloma annular and after discussion with the dermatologist biopsy was performed which confirmed the diagnosis of Lichenoid dermatitis. It was thought that this condition was triggered by the Thiazide diuretic and it was discontinued without significant change in her rash. Halobetasol 0.05% ointment BID for 2 weeks with minimal improvement in symptoms. The size of the lesions decreased but she developed new lichenoid eruption in the elbows area. Review of medical literature noted that eprosartan,² an angiotensin II receptor blocker use associated with lichenoid dermatitis and Olmesartan was discontinued and patient was switched to metoprolol succinate. She was also counselled about the possible association of beta blocker with lichenoid dermatitis before initiation. After 4 weeks there was near reso-

lution of her lesions. Because her blood pressure remained above goal she was started on Lisinopril. She declined amlodipine due to prior ankle swelling. After more than 2 months she remains free of lesions.

Discussion

Lichenoid drug eruption is characterized by uniform development of flat-topped, erythematous or violaceous patches or papules on the trunk and extremities that resemble lichen planus lesion.³ The time frame between initiation of the offending drug and development of cutaneous manifestation varies from weeks to years and varies with the class of the triggering drug, dosage and other concurrent medications.¹ Olmesartan is an angiotensin II receptor blocker used in treatment of hypertension. The incidence of cutaneous side effect is very low.⁴ It is frequently prescribed in combination with thiazide diuretics which commonly cause cutaneous adverse effects. Eczematous reactions and lichenoid changes have been commonly reported.⁵ For that reason the most likely culprit associated with lichenoid dermatitis, was hydrochlorothiazide. The olmesartan was continued to maintain blood pressure. But the persistence of the lesions required switching to deferent class which resulted in patient's rash resolution. Lichenoid dermatitis has been reported more widely with other medications like antimalarial medications, gold and phenothiazines with various degree of response after discontinuation of the triggering agent.²

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