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A Case of Benidipine-Induced Lichenoid Drug Eruption

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Introduction & Objectives:

Lichenoid drug eruptions (LDE) are rare adverse cutaneous reactions mimicking lichen planus (LP) both clinically and histologically. However, key distinctions such as the absence of Wickham striae and characteristic histological findings, including eosinophilic infiltrates and vacuolar degeneration, aid in differentiating LDE from idiopathic LP.

Although various drugs, including beta-blockers and angiotensin-converting enzyme inhibitors, are commonly implicated, this case represents the first reported instance of LDE induced by benidipine, a calcium channel blocker with known renoprotective and cardioprotective properties.

Materials & Methods:

A 76-year-old female presented with a three-week history of intensely pruritic erythematous and violaceous papules and plaques primarily localized to the lower extremities. The eruption developed 3 weeks after initiating benidipine for the management of hypertension. A punch biopsy confirmed the diagnosis of LDE, revealing interface vacuolar degeneration and band-like lymphocytic infiltration. Considering the temporal relationship, benidipine was discontinued based on the suspicion of benidipine-induced LDE and doxazosin was initiated. Since the patient had widespread lesions, systemic prednisolone treatment (40 mg daily) was started in addition to the discontinuation of benidipine and complete resolution of the lesions was observed within two months. No recurrence was observed in the 5-month follow-up.

Results:

Although LDE may have several clinical and histopathological findings that help in the differential diagnosis from idiopathic LP, these two entities still represent very similar clinical pictures, making the diagnosis of LDE challenging. LDE has a characteristic delayed onset, often appearing weeks to months after initiating the offending medication. In the present case, the latent period was approximately three weeks, which aligns with the variability reported in the literature. The diagnosis of LDE was supported by the temporal association between the onset of symptoms and initiation of benidipine, as well as the resolution of lesions following discontinuation of the drug and the introduction of systemic corticosteroid therapy. Histopathological findings, including band-like lymphocytic inflammation with sparse eosinophils and interface vacuolar degeneration, further corroborated the clinical diagnosis. Treatment for LDE generally involves cessation of the causative drug, with most cases resolving within weeks to months. In severe or persistent cases, systemic corticosteroids may be required, as was employed successfully in this patient. The absence of new lesions and complete resolution of existing lesions at the two-month follow-up underscores the importance of early recognition and prompt discontinuation of the offending agent.

Conclusion:

This case expands the spectrum of adverse cutaneous reactions associated with calcium channel blockers, underscoring the importance of prompt recognition and management of LDE. Clinicians should remain vigilant for

LDE in patients presenting with compatible eruptions following antihypertensive therapy, particularly when involving less commonly implicated agents like benidipine.