

## Case Report

# Telmisartan-induced lichenoid drug eruption: a case report

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## ABSTRACT

Lichenoid drug eruption (LDE) is a rare adverse reaction to various medications, including antihypertensives like telmisartan. Early identification and withdrawal of the offending agent are key to management. A 52-year-old hypertensive male on telmisartan developed pruritic, violaceous lesions over the scalp and extremities, later spreading to the face, neck, and back, with oral involvement. Initial treatment offered partial relief. A diagnosis of telmisartan-induced lichenoid eruption was made, and the drug was replaced with amlodipine. Betamethasone mini-pulse therapy and azathioprine were initiated, leading to full resolution of lesions, leaving only post-inflammatory hyperpigmentation. Although uncommon, telmisartan can cause LDEs. Timely discontinuation and appropriate immunosuppressive therapy ensure favourable outcomes.

**Keywords:** Telmisartan, Lichenoid drug eruption, Hypertension, Adverse drug reaction, Case report

## INTRODUCTION

Lichenoid drug eruption (LDE) is a rare, immune-mediated cutaneous adverse reaction that clinically and histopathologically mimics idiopathic lichen planus. Various medications have been implicated in its development, including beta-blockers, angiotensin-converting enzyme inhibitors, non-steroidal anti-inflammatory drugs (NSAIDs), and antimalarials.<sup>1</sup> Telmisartan, a long-acting angiotensin II receptor blocker (ARB), is widely prescribed for the management of hypertension and cardiovascular risk reduction. It acts by selectively blocking the angiotensin II type 1 (AT1) receptors, leading to vasodilation, reduced aldosterone secretion, and lower blood pressure.<sup>2</sup> Telmisartan is generally well-tolerated; however, commonly reported adverse drug reaction include dizziness, hypotension, fatigue, hyperkalaemia and gastrointestinal disturbances such as diarrhoea and abdominal pain.<sup>2</sup> Cutaneous side effects are rare but may manifest as LDEs, which require high clinical suspicion for early diagnosis and management.

## CASE REPORT

A 52-year-old male with a two-year history of hypertension, well-controlled on telmisartan 40 mg once daily, presented with pruritic, purplish, raised, and painless lesions over the scalp and dorsum of the hands and feet. These symptoms developed four months prior to consultation.

Initially, a dermatologist prescribed oral antihistamines and topical clobetasol, resulting in partial relief over 20 days. However, the patient subsequently experienced new skin lesions and enlargement of existing ones, along with a burning sensation in the oral cavity. Over the next month, the lesions spread to involve the scalp, face, V-area of the neck, upper back, and extensor aspects of the extremities.

Upon further dermatological evaluation, the patient's medication history revealed the use of telmisartan. Based on clinical presentation and temporal relation with the drug intake, a diagnosis of LDE was made.

Consequently, telmisartan was discontinued and replaced with amlodipine 5 mg once daily as an alternative antihypertensive. Betamethasone mini-pulse therapy (5 mg on two consecutive days per week) was initiated to manage the skin and mucosal lesions. After one month of therapy, azathioprine 50 mg once daily was added to the treatment regimen to aid in lesion resolution.

At the last follow-up visit on February 2025, the patient showed significant improvement, with only post-inflammatory hyperpigmentation remaining; all other skin lesions had entirely resolved.

As per the world health organization-Uppsala monitoring centre (WHO-UMC) causality assessment system, the reaction was classified as probable. The severity of the adverse drug reaction was graded as moderate (Level 3) according to the modified Hartwig and Siegel scale. The preventability assessment, based on the Modified Schumock and Thornton criteria, categorized the reaction as non-preventable.<sup>6</sup>



**Figure 1: LDE on mucosal surface of mouth.**



**Figure 2: LDE on hands and abdomen**

## DISCUSSION

LDE is a rare cutaneous adverse drug reaction that differs from classical lichen planus (LP) by its atypical clinical presentation, often involving acral areas, extensor surfaces, and photo-distributed sites, whereas classical LP predominantly affects flexural regions. Typically, LDE arises after a variable latency period ranging from weeks to several years following drug exposure, making clinical recognition more challenging.<sup>3</sup> In our case, the patient developed pruritic violaceous plaques predominantly over the scalp, dorsum of hands and feet, face, V-area of the neck, upper back, and extensor aspects of the extremities a distribution pattern consistent with LDE.

Although telmisartan is widely used for hypertension management, reports of telmisartan-induced LDE are scarce in the literature. The exact pathogenesis is not fully understood but is believed that LDE is marked by the chronic activation of CD8<sup>+</sup> cytotoxic T lymphocytes targeting epidermal cells. These lymphocytes can trigger apoptosis in basal keratinocytes and secrete various cytokines, elevating the expression of class II major histocompatibility complex molecules and facilitating antigen presentation to CD4<sup>+</sup> T cells.<sup>4</sup>

Once LDE planus is identified, management involves immediate withdrawal of the suspected drug, followed by the application of high-potency topical corticosteroids for cutaneous lesions. In cases unresponsive to topical therapy, calcineurin inhibitors may serve as effective alternatives, while extensive or severe eruptions may necessitate a short course of systemic corticosteroids.<sup>5</sup> In our patient, telmisartan was discontinued and replaced with amlodipine. Initial symptomatic management with topical corticosteroids and antihistamines provided inadequate relief.

Therefore, betamethasone initiated mini-pulse therapy, which involves the intermittent administration of systemic corticosteroids. Additionally, azathioprine, an immunosuppressive agent that inhibits purine synthesis and suppresses T-cell-mediated inflammation, was introduced.

## CONCLUSION

This case highlights the importance of considering drug-induced LDE when a patient develops new skin lesions, especially if they are taking medicines known to cause such reactions. Early diagnosis, stopping the suspected medicine quickly, and giving proper treatment help in faster recovery and better results.

In patients who have a past history of drug allergies, LDE can sometimes be prevented by choosing medicines carefully and monitoring them closely. Good communication between dermatologists and primary care doctors is very important for proper treatment, follow-up, and to prevent the problem from coming back.

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