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Case Report

Isoniazid-Induced Lichenoid Drug Eruption: A Rare Case Report

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ABSTRACT

Lichenoid Drug Eruption (LDE) is a rare, immune-mediated adverse cutaneous reaction that closely mimics idiopathic lichen planus. Isoniazid, a first-line anti-tubercular drug, has been infrequently implicated as a causative agent. We report the case of a 55-year-old male with hypertension and pulmonary tuberculosis receiving multidrug anti-tubercular therapy, who developed multiple hyperpigmented macules, patches, and plaques over the trunk, limbs, and oral mucosa. Histopathological examination revealed features consistent with lichenoid dermatitis. The patient was managed with systemic corticosteroids, immunosuppressants, and supportive therapy while continuing anti-tubercular treatment. This case emphasizes the importance of recognizing isoniazid-induced cutaneous adverse reactions early, managing them effectively without discontinuing essential therapy, and ensuring multidisciplinary collaboration between dermatology, pulmonology, and pharmacy teams to achieve optimal outcomes.

Keywords: Anti-tubercular Therapy, Cutaneous Adverse Drug Reaction, Isoniazid, Lichenoid Drug Eruption.

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INTRODUCTION

Lichenoid Drug Eruption (LDE) is a rare, immune-mediated adverse cutaneous reaction that mimics idiopathic lichen planus both clinically and histopathologically (Pathave *et al.*, 2022). A group of chronic inflammatory diseases that affect the stratified squamous epithelium are collectively referred to as lichen planus (Shanmukhappa *et al.*,2024). The spectrum of severe tuberculosis-associated cutaneous, ADRs is wide and includes Stevens-Johnson syndrome, drug hypersensitivity syndrome and Lichenoid Drug Reactions (LDRs) (Lehloenya *et al.*,2014). Numerous medications, such as thiazides, antimalarials, diuretics, beta blockers, ACE inhibitors, gold salts, penicillamine, and lipid-lowering medications including fibrates and statins, can result in Lichenoid Drug Eruption (Singh *et al.*, 2020)

Large, symmetrical, flat-topped violaceous plaques distributed over the trunk and extremities are characteristic features of Lichenoid Drug Eruption. Approximately 10% of patients receiving first-line anti-tubercular therapy develop this reaction (Chacko *et al.*, 2022). In the general population, the prevalence of lichenoid eruptions is around 2.4%, with a female predominance occurring nearly three times more often in women than in men (Suryana, 2020). It is rare for isoniazid to cause Cutaneous Adverse Drug Reactions (CADR), such as maculopapular rashes, purpuric skin eruptions, exfoliative dermatitis, toxic epidermal necrosis, and Lichenoid Drug Eruptions. The most typical Lichen Planus symptoms comprise flat, polygonal, symmetrical, erythematous, violaceous papules on the trunk and extremities (Ali, 2020).

According to laboratory standards, absolute eosinophil counts that are higher than 450-550 cells/μL are considered elevated. A drug allergy can result in mild to severe eosinophilia, which frequently goes away rapidly. It decreases more slowly; eosinophilia brought on by a medication allergy may not go away for months (Grossman *et al.*, 1995).

Although the precise process by which a medicine causes lichenoid eruptions is not entirely understood, it is believed to be connected to a secondary reaction brought on by interactions between the drug and the immune system and is treatable with sunscreens, calcineurin inhibitors, and corticosteroids (Ali, 2020).

Lichenoid Drug Eruption (LDE) is thought to result from a T cell-mediated autoimmune reaction in which drugs act as haptens, binding to host proteins and triggering a type IV hypersensitivity response. This leads to $CD8^+$ cytotoxic T-cell-induced keratinocyte apoptosis, as the immune system

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misidentifies keratinocytes and Langerhans cells as foreign (Pathave *et al*.,2022).

The present case highlights a patient with Lichenoid Drug Eruption in the background of hypertension and pulmonary tuberculosis receiving multidrug therapy.

CASE DESCRIPTION

A 55-year-old male with a known history of hypertension for the past three years and pulmonary tuberculosis under Anti-Tubercular Therapy (ATT), which was initiated on 29th January 2025, presented with complaints of itching lesions over both lower limbs. On Day 1 (2 September 2025), he noticed the first coin-sized erythematous patches on his lower limbs. By Day 2, these lesions began to increase in number, and over the following days they progressively spread, eventually involving the entire body within 15 days. He also reported the gradual appearance of dark, blackish sores on his face, which worsened with sun exposure and were associated with significant itching. For these skin complaints, a dermatologist prescribed oral prednisolone for 15 days and azathioprine for 20 days on Day 2, which provided only temporary symptomatic relief. On general examination, the patient was conscious, oriented, and moderately built.

Mucocutaneous examination revealed normal genitalia, scalp, and nails. However, numerous hyperpigmented macules, patches, and plaques were observed over the abdomen, back, chest, bilateral lower limbs (with erythema), malar area, and oral cavity (refer to Figures 1-3). Based on the clinical presentation and medical history, a provisional diagnosis of lichenoid drug eruption was made, with differential diagnoses including lichen planus pigmentosus and irritant contact dermatitis, particularly in the context of hypertension and tuberculosis.

A skin biopsy specimen measuring 0.3 × 0.1 cm, grey-white to grey-brown in color, was examined histopathologically. Microscopic findings showed vacuolar degeneration of basal cells, hyperkeratosis, irregular acanthosis, and a band-like lymphocytic infiltrate beneath the epidermis, along with pigment incontinence. No eosinophils were observed. The overall findings were consistent with lichenoid dermatitis.

The patient was managed with a combination of systemic, topical, and supportive therapies as summarized in Table 1. Regular follow-up was advised to monitor cutaneous outcomes, emphasizing the importance of cautious drug monitoring during multidrug therapy.

DISCUSSION

Lichenoid Drug Eruptions associated with anti-tubercular therapy are uncommon but clinically significant. This case underscores the importance of considering concurrent or alternative medications as potential contributing factors while recognizing ATT, particularly isoniazid, as a frequent causative agent of LDE.

In the present case, the patient developed widespread hyperpigmented macules, patches, and plaques involving the face, trunk, oral cavity, and extremities approximately three months after initiating ATT. Similar findings have been documented in earlier reports. Ali *et al*. described a 55-year-old woman in whom isoniazid was identified as the causal drug responsible for LDE (Ali, 2020), whereas Chacko *et al*. reported a 63-year-old man who developed lichenoid plaques following ATT (Chacko *et al*., 2022). These observations are consistent with the current case, in which the temporal association and clinical presentation suggest that the lichenoid eruption was most likely induced by continued ATT.

Comparable manifestations were also reported by Pathave. *et al*., who described a 35-year-old woman developing violaceous papules and plaques over the lips, trunk, and extremities (predominantly extensor aspects) one month after starting ATT. Histopathological examination confirmed LDE, and the patient responded favorably to tapering doses of systemic corticosteroids during the course of treatment (Pathave *et al*., 2022). In contrast, our patient exhibited a photodistributed pattern of hyperpigmented macules, patches, and plaques affecting the extremities, trunk, malar region, and oral cavity, with histopathology confirming lichenoid dermatitis. However, the therapeutic response was less favorable compared to previously reported cases.

Grossman *et al*. further documented a 67-year-old patient who developed lichenoid papules with a distinct photo-distributed pattern on the hands and forearms three months after initiation of ATT. Histopathology revealed perivascular inflammation without eosinophils, necrotic keratinocytes, and a superficial band-like mononuclear infiltrate at the dermo-epidermal junction (Grossman *et al*.,1995). The histopathological findings in our case were in agreement with this report, supporting the diagnosis of isoniazid-induced LDE.

Therapeutically, the present patient required a more intensive management approach. Treatment included intravenous dexamethasone (1.5 mL), oral hydroxychloroquine 200 mg, iron supplementation (ferrous sulphate), and multivitamin capsules. Despite these measures, the patient showed only partial improvement, suggesting a more resistant course compared to previous reports. Singh P, *et al*. described a 63-year-old patient who responded satisfactorily to potent topical corticosteroids and gradually tapered oral prednisolone (30 mg), highlighting the variability in clinical response among different cases (Singh *et al*., 2020).

Based on the temporal association and lack of improvement following dechallenge, the adverse reaction to isoniazid in the present case was classified as Possible according to the WHO Causality Assessment Scale. This case thus reinforces the need for

vigilance during ATT, with prompt recognition and appropriate management of cutaneous adverse reactions to ensure optimal therapeutic outcomes. This case highlights the management of isoniazid-induced lichenoid drug eruption while continuing anti-tubercular therapy. It underscores the importance of identifying isoniazid as the causative agent, maintaining effective TB control without discontinuing therapy, and optimizing immunosuppressive treatment. The case also emphasizes the vital role of clinical pharmacists in coordinating care between dermatology and pulmonology teams.

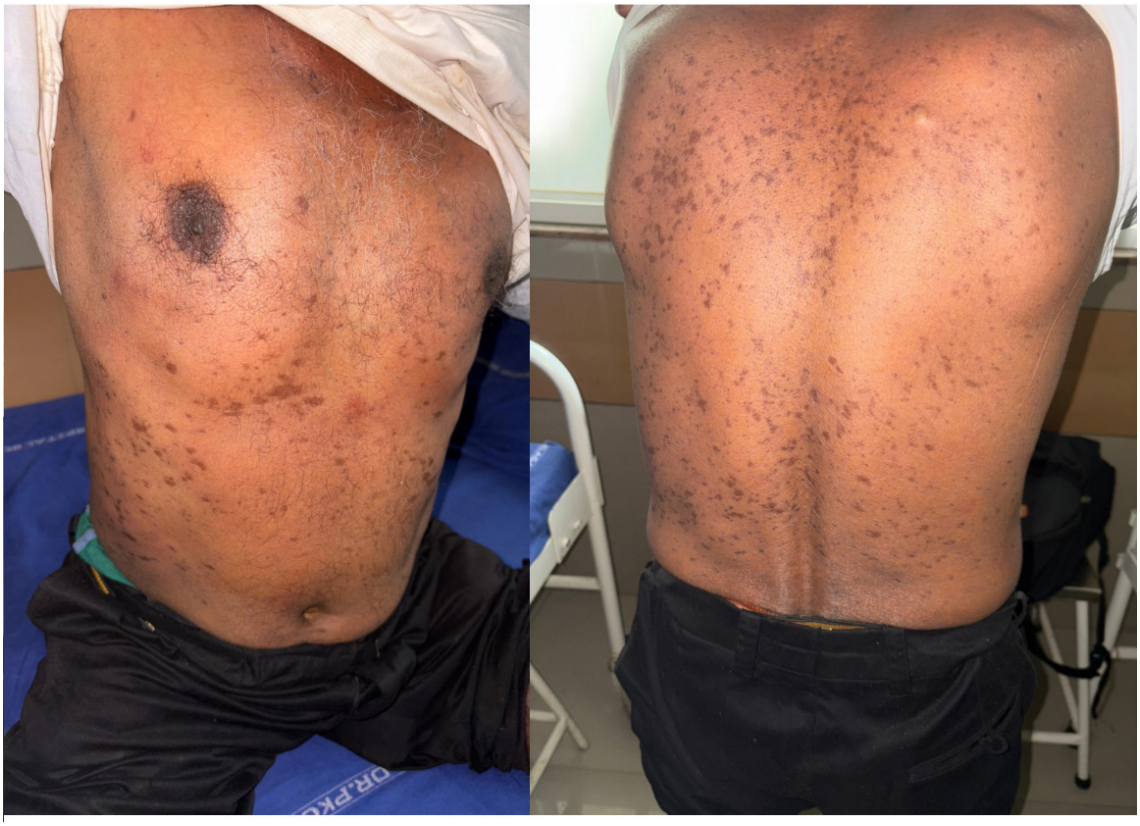

Figure 1: Multiple well defined hyper pigmented macules, Patches over abdomen, back and chest.

Table 1: Management for Lichenoid Plaques.

Medication	Dose	Route	Frequency
Dexamethasone	1.5 mL	INTRAVENOUS	Once daily
Pantoprazole	40 mg	INTRAVENOUS	Once daily
Chlorpheniramine Maleate	4 mg	INTRAVENOUS	Twice daily
Hydroxychloroquine	200 mg	ORAL	Once daily
Rifampicin	450 mg	ORAL	Once daily
Isoniazid	300 mg	ORAL	Once daily
Ethambutol	800 mg	ORAL	Once daily
Calcium + vitamin D3	500 mg	ORAL	Once daily
Atenolol	50 mg	ORAL	Once daily
Liquid paraffin L/A		TOPICAL	Twice daily
Ferrous sulphate	200 mg	ORAL	Once daily
Multivitamin + B complex + Vitamin C		ORAL	Once daily
Benzalkonium Chloride + Choline Salicylate		TOPICAL	Thrice daily
Beclomethasone+ fusidic acid	20 g	TOPICAL	Once daily
Povidine-iodine	1:1 dilution	ORAL	Thrice daily

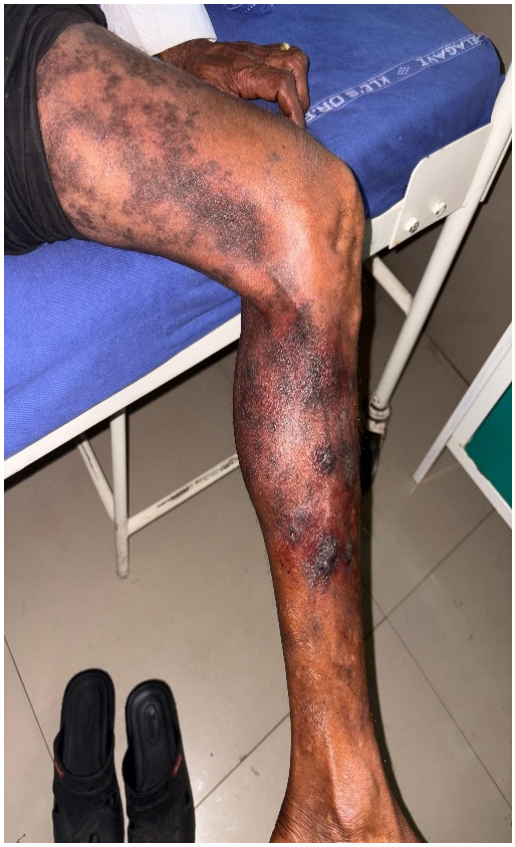

Figure 2: Multiple well defined hyper pigmented macules, Patches over bilateral lower limb with Erythema seen.

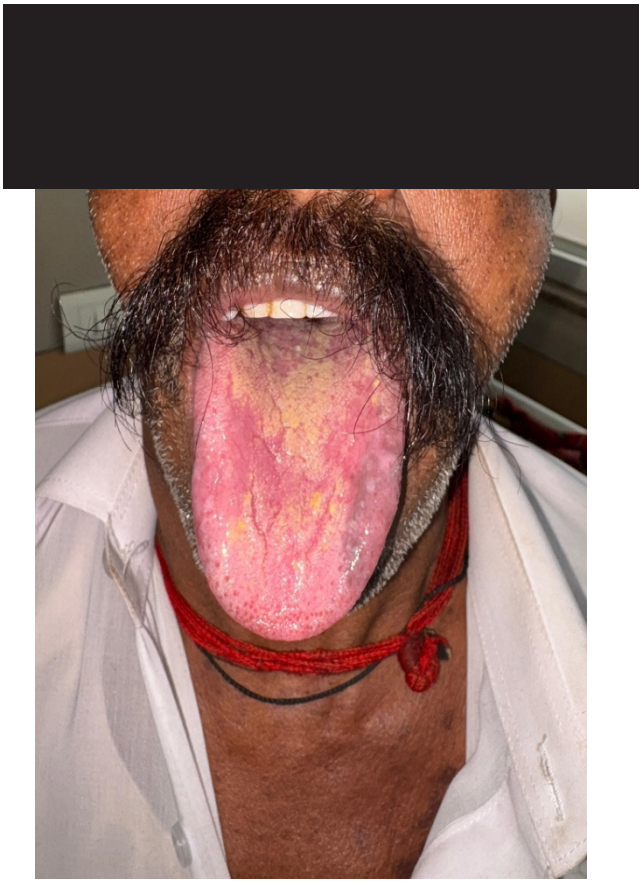

Figure 3: Multiple well defined hypo pigmented plaques over tongue.

CONCLUSION

Despite being uncommon, isoniazid-induced Lichenoid Drug Eruption necessitates early detection and close monitoring during anti-tubercular therapy. Severe reactions can be minimized through routine skin examination, patient education regarding early signs such as new rashes or itching, and appropriate symptomatic management without compromising essential isoniazid therapy. Multidisciplinary collaboration ensures safer patient care and improved therapeutic outcomes.

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ABBREVIATIONS

LDE: Lichenoid Drug Eruption**; ATT:** Anti-Tubercular Therapy; **ADR:** Adverse Drug Reaction; **CADR:** Cutaneous Adverse Drug Reaction; **WHO:** World Health Organization.

PATIENT CONSENT

Written informed consent was obtained from the patient for publication of the clinical details and accompanying images. The patient was assured of confidentiality, and identifying information has been withheld to protect privacy.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

SUMMARY

This case report describes a rare presentation of isoniazid-induced lichenoid drug eruption in a 55-year-old male undergoing anti-tubercular therapy. The patient developed widespread hyperpigmented macules, plaques, and oral lesions, with histopathology confirming lichenoid dermatitis. Management involved systemic and topical therapies while continuing essential anti-tubercular treatment. The case highlights the importance of early recognition and multidisciplinary care in managing cutaneous adverse drug reactions without interrupting tuberculosis therapy.

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