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Case Study

Lichen planopilaris with drug-induced lichen planus: A Case Report

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Abstract:

Background:

Lichenoid drug eruption is a known adverse reaction to several medications, including anti-tubercular therapy, and may clinically resemble idiopathic lichen planus. Lichen planopilaris is a scarring inflammatory disorder of hair follicles that can result in permanent alopecia. The coexistence of these two conditions is rare and presents diagnostic and management challenges.

Case presentation:

A 25-year-old female receiving first-line anti-tubercular therapy for pulmonary tuberculosis developed progressive scarring alopecia of the scalp along with pruritic violaceous papules over the neck, trunk, and axillae. Dermoscopy and histopathological examination confirmed a lichenoid drug eruption with coexisting lichen planopilaris. Given the near completion of tuberculosis treatment, anti-tubercular drugs were continued, and the patient was managed with topical corticosteroids, emollients, and oral antihistamines, with planned initiation of hydroxychloroquine after therapy completion.

Conclusions:

Early identification of drug-induced lichenoid reactions is crucial to prevent irreversible scarring. A conservative, multidisciplinary approach can allow continuation of essential anti-tubercular therapy while effectively managing cutaneous manifestations.

Keywords: *Lichenoid drug eruption, lichen planus, lichen planopilaris, anti-tubercular therapy, scarring alopecia, drug-induced skin reaction, cutaneous adverse effect, tuberculosis treatment.*
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Background

Lichen planopilaris (LPP) is a chronic inflammatory disorder affecting the hair follicles and is characterized by scarring alopecia, which may result in permanent hair loss [1]. Certain medications, including anti-tubercular drugs such as isoniazid and rifampicin, are known to induce lichenoid drug eruptions (LDEs), which closely resemble idiopathic lichen planus in their clinical appearance but are drug-related in origin [2]. Unlike classic lichen planus, LDEs are less commonly associated with mucosal involvement or Wickham striae and typically present as symmetrical, violaceous papules [3].

The simultaneous occurrence of LPP and LDE is uncommon, as both entities are usually encountered independently, making clinical diagnosis challenging [4]. Early recognition is critical, as delayed intervention may lead to irreversible scarring alopecia, particularly when management decisions involve continuation of essential systemic therapies such as anti-tubercular treatment [5]. Therefore, clinicians must carefully balance the risk of adverse cutaneous reactions against the necessity of maintaining life-saving medications during treatment planning [6].

CASE PRESENTATION

A 25 years old, female patient, who is under treatment of anti-tubercular drugs against pulmonary tuberculosis during the last 5 months, presented in the outpatient department

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with a complaint of progressive thinning of her hair on the scalp, which was observed during the last 4 months, and then was driven by the appearance of violaceous, scaly lesions in the area of the loss of the scalp hair. Also, during the prior two months, she had developed pruritic and violaceous flat-topped lesions on her neck, trunk, and both axillae. She did not report any previous locations and/or family history of this type of problem. There were no indications of photosensitivity, joint pain, alopecia (loss of hair) in other areas of the body, or dangerous behaviour. Based on dermatological observation, it was found that there were not a few flat-topped papules, which were well-defined to poorly defined, violaceous to hyperpigmented, and a little bit scaly. The papules were initially uniformly spread on the body and later on became coalescent to form plaques. The two plaques covered the surface area of the body about 6 percent; the largest, 3 by 2 cm, was on the breast, and the smallest, 0.5 by 0.5 cm, was on the back. The mucosal surfaces, faces, arms, and legs were not affected much. Temperature did not rise; the lesions were painless on touch. The area of alopecia was evident on the scalp (fig. 5), and it was approximately 4 x 3 cm wide and glossy at the vertex. Violaceous to hyperpigmented patches were also observed; however, no discharge, semi-adherent, white, non-odorous scales were present. No tenderness, reduced pinchability, or abnormal temperature on palpation of the alopecic area was either felt or experienced.

Under microscopic examination, the skin lesions depicted brownish-black macules on a light brown background that exhibited reticular or pseudo-network distribution (fig. 6). Granular pigmentation with brown spots and globules, as well as a less peculiar network of small white lines resembling Wickham striae, was additionally observed but was unevenly distributed. The erythema and vascular structures were not evident, and the pigmentation was deep but patchy and with ill-defined margins. Dermoscopy evaluation of the scalp lesion (figure 7) showed the following: there was the presence of scaling and redness around hair follicles, the hair follicles showed plugging, the hair follicles were sparse, the background pigmentation varied between violet and reddish with white interspersions at any point, and there was no phenomenon of black or yellow spots.

The histopathological picture of skin lesions is represented in Figure 8. The shallow dermis appeared normal, the deeper dermis contained thick perivascular and periadnexal lymphocytic infiltrates, and the epidermis became thin with moderate spongiosis. The histopathology of the scalp lesion was atrophic, hyperkeratotic epidermis and shrinkage of rete ridges (fig. 9). There was mild dermal fibrosis, pigment incontinence, focal vacuolar degeneration, periumbal and superficial lymphocytic infiltrate, as well as a loss of sebaceous gland and hair follicles. On histopathological and clinical evidence, the patient was determined to have lichenoid drug eruption, which coexisted with lichen planopilaris. The patient was referred to the pulmonary medicine section to review her tuberculosis treatment protocol. She was completing her treatment in the last month, and it was not intended to drop her anti-tubercular treatment too early, but the patient remained on anti-tubercular treatment under the watchful eye of a dermatologist. With regards to her symptoms, the doctor prescribed her oral Bilastine 10 mg once per day and Levocetirizine 10 mg at bedtime, to reduce itching. Topical therapy was used to keep the skin hydrated, and emollients such as liquid paraffin and white soft paraffin were applied to the skin. Mometasone Furoate lotion was applied to the trunk and parts of axillae, and betamethasone dipropionate 0.05 per cent was applied on the scalp; the cutaneous lesions. Once the patient completes her anti-tubercular therapy, she will start using hydroxychloroquine, and she is currently monitored every two weeks.

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FIG 1

FIG 2

FIG 3

FIG 4

FIG 5

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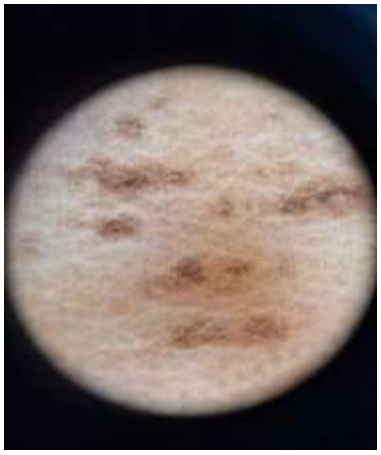

FIG 6

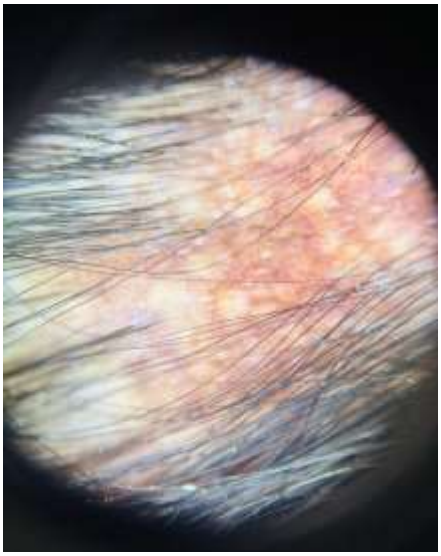

FIG 7

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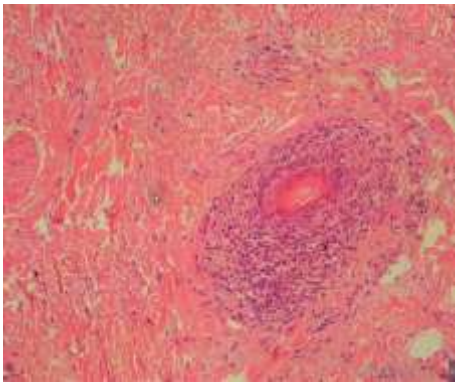

FIG 8

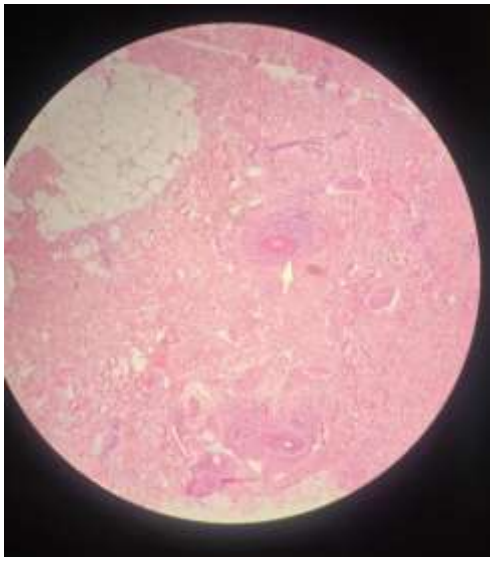

FIG 9

DISCUSSION

This case describes a rare coexistence of lichenoid drug eruption (LDE) and lichen planopilaris (LPP) in a young female receiving anti-tubercular therapy. The characteristic features observed in this patient included patchy scarring alopecia of the scalp, pruritic flat-topped papules over the neck, trunk, and axillae, and violaceous scaly plaques involving the scalp. The close temporal relationship between initiation of anti-tubercular medications and the onset of cutaneous manifestations raised a strong clinical suspicion of a drug-induced lichenoid reaction.

Lichen planus (LP) is a chronic inflammatory disorder affecting the skin, hair, nails, and mucous membranes and is mediated by a T-cell–driven immune response targeting basal keratinocytes [7]. Lichen planopilaris represents a follicular variant of LP and is characterized by perifollicular inflammation, hyperkeratosis, and progressive scarring alopecia, most commonly involving the scalp [8]. Lichenoid drug eruptions may closely resemble idiopathic LP both clinically and histologically; however, they are typically triggered by medications and often lack mucosal involvement or classical Wickham striae [9].

LDE has been associated with a wide range of drugs, including antihypertensives, antimalarials, non-steroidal anti-inflammatory drugs, and, more recently, anti-tubercular therapy (ATT). Among ATT agents, isoniazid, ethambutol,

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and pyrazinamide have been most frequently implicated [10–12]. Several reports have documented isoniazid-induced lichenoid eruptions and LPP, highlighting the relevance of ATT as a precipitating factor, particularly in tuberculosis-endemic regions [13–15]. A review published in 2020 also identified ATT as one of the commonly reported causes of drug-induced lichen planus in such settings [16].

The underlying pathophysiology of both LDE and LPP is thought to involve a type IV hypersensitivity reaction mediated by CD8+ cytotoxic T-cells, leading to apoptosis of basal keratinocytes expressing altered self-antigens, likely drug metabolites [17]. In susceptible individuals, this immune response may result in persistent inflammation and irreversible follicular damage. Management of drug-induced LP or LPP, therefore, requires careful risk–benefit assessment. While discontinuation of the offending drug is often recommended, this may not be feasible when the medication is essential for the treatment of a serious systemic disease such as tuberculosis. Previous case reports have demonstrated benefit with topical corticosteroids, calcineurin inhibitors, oral antihistamines, systemic corticosteroids, retinoids, and immunosuppressive agents, including hydroxychloroquine, methotrexate, and mycophenolate mofetil [18–20].

In the present case, continuation of anti-tubercular therapy was considered appropriate, as the patient was nearing completion of her six-month treatment course and her systemic condition was stable. The cutaneous manifestations were managed conservatively with topical betamethasone dipropionate 0.05% for the scalp, mometasone furoate lotion for truncal lesions, oral antihistamines for pruritus, and regular use of emollients to maintain skin barrier function. Hydroxychloroquine has been planned for long-term management of LPP following completion of ATT. The coexistence of LDE and LPP in association with anti-tubercular therapy is uncommon and poses diagnostic and therapeutic challenges. In this case, a balanced approach allowed effective management of cutaneous adverse effects without interruption of essential systemic therapy. Regular follow-up enabled close monitoring of disease progression and treatment response, which is crucial to prevent irreversible scarring alopecia and preserve quality of life.

Among anti-tubercular drugs, isoniazid is considered a likely contributor to the lichenoid reaction observed in this patient, based on existing literature. Early recognition of drug-induced cutaneous reactions, prompt initiation of symptomatic treatment, and close collaboration between dermatology and pulmonary medicine teams were key to successful management. This case underscores the importance of individualized treatment strategies in patients with overlapping autoimmune dermatoses and drug-induced reactions, particularly when continuation of life-saving medications is required.

Conclusions

This case highlights a rare coexistence of lichen planopilaris with anti-tubercular drug-induced lichenoid eruption. Early clinical suspicion, supported by dermoscopic and histopathological findings, is essential to prevent irreversible scarring alopecia. Careful multidisciplinary management allowed continuation of essential anti-tubercular therapy while effectively controlling cutaneous manifestations. Prompt recognition and individualized treatment strategies are crucial when managing drug-induced dermatologic reactions associated with life-saving medications.

Ethics declarations.

Ethical approval for this case report was obtained from the Institutional Ethics Committee, School of Medical Sciences and Research, Sharda University, Greater Noida, India. Written informed consent was obtained from the patient before participation and publication of clinical details and images. The study was conducted in accordance with institutional ethical standards and the Declaration of Helsinki.

Availability of data and materials

All data generated or analyzed during this case report are included within the article. The study was conducted at the School of Medical Sciences and Research, Sharda University, Greater Noida, India. No additional datasets were generated or are publicly available.

Authors Contributions

Rabia Alam was involved in patient evaluation, clinical diagnosis, data collection, and manuscript drafting.

Harsh Tyagi contributed to the literature review, clinical documentation, and manuscript editing.

Shitij Goel supervised the clinical management of the patient, reviewed the manuscript critically, and approved the final version for publication.

All authors read and approved the final manuscript.

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List of Abbreviations
1. ATT – Anti-tubercular therapy
2. LDE – Lichenoid drug eruption
3. LPP – Lichen planopilaris
4. LP – Lichen planus

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Conflict of Interest
The authors declare that there is no conflict of interest regarding the publication of this article.

Authors' Biography
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