



Clinical Insights from A Case Series of lichen Planopilaris: Variability and Management Strategies

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ABSTRACT:

Lichen planopilaris (LPP) is a cell-mediated scarring alopecia that causes inflammation of the scalp and the eventual destruction of hair follicles in affected areas. Current literature on treatment of LPP remains limited with no definitive treatment approach being recognized, although a combination of topical/intralesional steroids and orally administered hydroxychloroquine remains the most utilized option. Low-level light therapy (LLLT) is an expanding technology shown to be effective in a variety of dermatologic conditions. We report here four patients with LPP who show a dramatic response to LLLT, including a reduction of inflammation, disappearance of symptoms, and evident hair regrowth with no side effects. We review the possible role of LLLT in LPP and other lichenoid conditions.

Introduction

Lichen planopilaris (LPP) is a rare chronic inflammatory scalp disease and considered a prototype for lymphocytic cicatricial alopecias. Although the precise pathophysiology of LPP is unknown, it is believed to be related to the permanent killing of follicular stem cells in the hair bulge by cells, which results in the loss of the ability for hair follicles to regenerate [1]. A fast developing treatment option for many illnesses that need to reduce pain and inflammation in order to eventually restore function is low-level light therapy (LLLT) [2]. The US Food and Drug Administration (FDA) has approved LLLT for the treatment of both acute and chronic musculoskeletal pain as well as male and female androgenic alopecia (AGA). Additional dermatological disorders such vitiligo, hypertrophic scarring,

inflammatory acne, and skin ageing have also been partially resolved by LLLT [2–5]. LLLT is thought to offer a variety of advantages and potential therapeutic applications for LPP sufferers. Thus far, LLLT has been directly tested in two trials [6, 7] for the treatment of scarring alopecia, such as LPP, and has been tested in other studies [8–10] for oral lichen planus.

Here we present three cases – first classic case of LPP, second is Lichen planopilaris with Koebner phenomenon and Folliculitis Decalvans And Lichen Planopilaris Phenotypic Spectrum.

Case 1- Classic Case of LPP

In 2022, a 40-year-old lady visited to Department of Dermatology, Venereology and Leprosy, Meenakshi Medical College, Hospital and Research Institute,



Kanchipuram who had no notable family medical history or past medical history initially appeared with symptoms of hair loss, thinning hair, and itching on her scalp. She got a scalp biopsy at that time, and the results revealed miniaturisation and scarring alopecia typical with LPP. Upon inspection, the patient was found to have several peripilar casts on trichoscopy, scarring patterned alopecia, and absence of follicular openings. After

receiving a diagnosis of FAPD, the patient was prescribed naltrexone (3 mg) once daily, clobetasol 0.05% lotion once daily, and LLLT (272 pulsed laser diode cap with 1360 mW total output) for six minutes each day. Follow-up with photography showed significant improvement with evident regrowth of hair after 3 and 6 months.

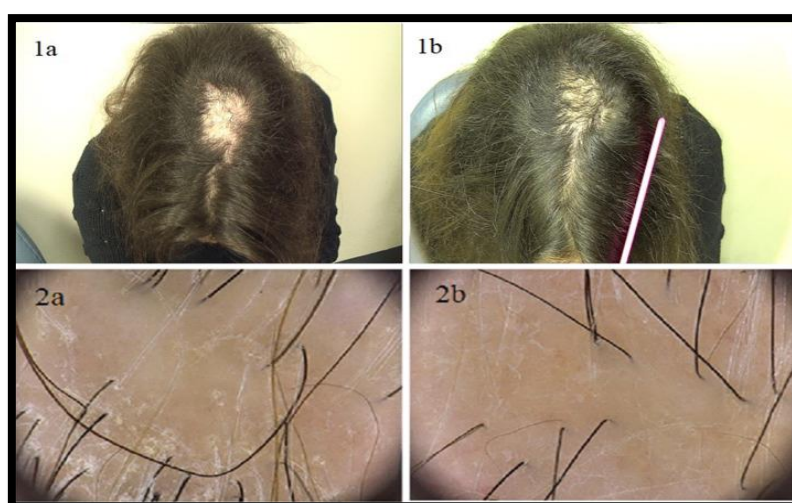


Fig. 1 A 40-year-old patient **1a** before and **1b** after 18 months of treatment with LLLT with evident hair regrowth. Scalp under $\times 50$ magnification trichoscopy **2a** before and **2b** after 4 months of treatment with LLLT, showing reduction of peripilar casts.

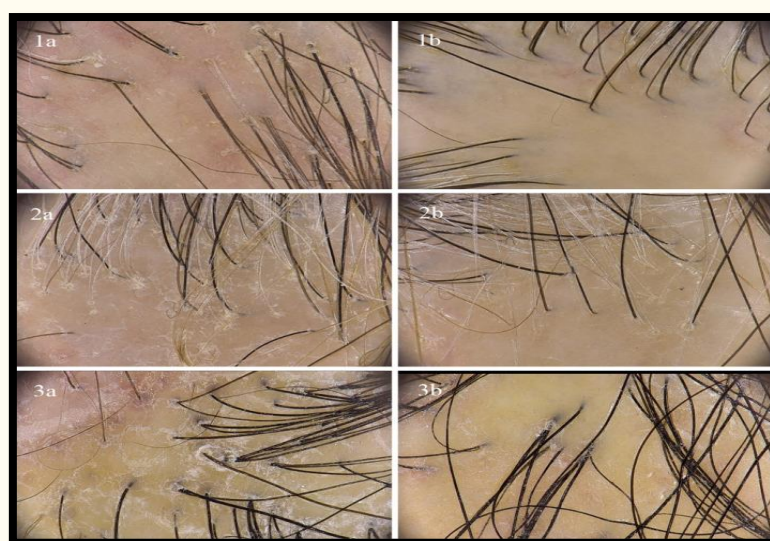
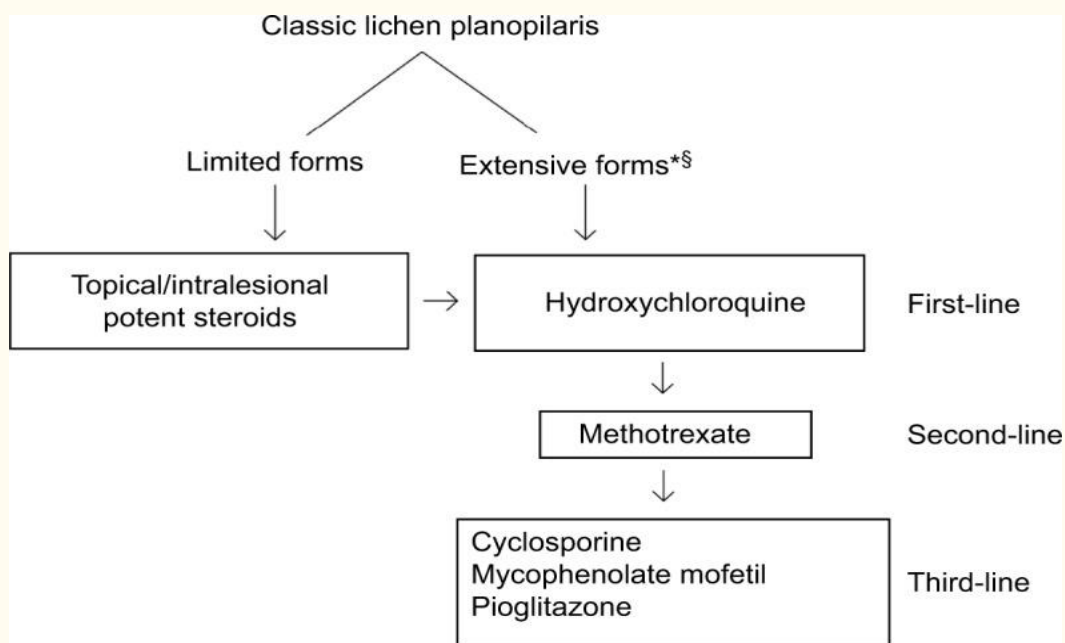


Fig. 2-Scalp under $\times 50$ magnification trichoscopy. A 60-year-old patient **1a** before and **1b** after 6 months of LLLT treatment. A 65-year-old patient **2a** before and **2b** after 6 months of LLLT treatment. A 28-year-old patient **3a** before and **3b** after 12 months of LLLT treatment.



Management



Case 2- Lichen Planopilaris With Koebner Phenomenon

A 25-year-old healthy woman presented to our Department of Dermatology, Venereology and Leprosy, Meenakshi Medical College, Hospital and Research Institute, Kanchipuram with a 5-month history of bilateral focal temporal hair loss with concomitant pain, redness, itching, and burning sensation of the scalp. She stated that she had a long history of traction applied to the balding spots. Before her hair started to fall out, she denied taking any oral or topical medications. Her family has an unremarkable history of autoimmune diseases or other hair disorders, but she tests positive for alopecia areata. Examining her scalp

revealed striking perifollicular erythema and scaling together with symmetrical bitemporal irregular patches of scarring alopecia (Fig 1, A and B). The axilla, groyne, or eyebrow did not experience hair loss. The lichen planus-related alterations to the mucocutaneous and nail structures were absent. The results of her laboratory testing, which included tests for liver and renal function, thyroid function, serum ferritin level, and total blood count, were all within normal limits. The rate of erythrocyte sedimentation was not increased. Reduced hair follicle density, perifollicular fibrosis, and a lymphatic infiltrate of the hair follicle's isthmus section were observed in a scalp biopsy specimen that was consistent with LPP (Fig 2, A and B).

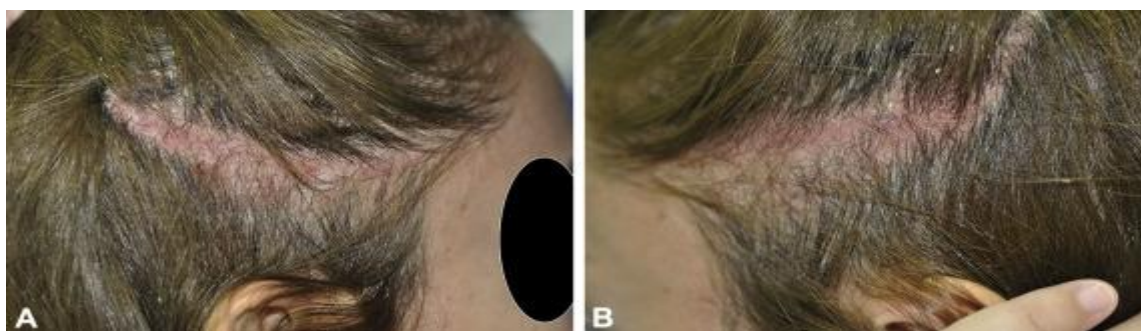


Fig 1 A and B, Bilateral linear patch of scarring alopecia following the line of hair traction

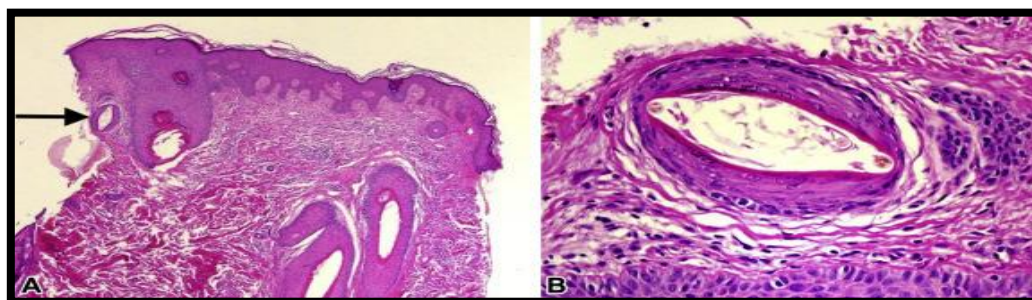


Fig 2A, Low-power view shows a low follicular density. **B**, A higher magnification shows an altered follicle with focal perifollicular fibrosis with a few lymphocytes at the level of the infundibulum. (A and B, Hematoxylin-eosin stain.)

Management

In order to avoid their hair loss getting worse, the patient was recommended not to use traction hair care products. Her pain, redness, and itching remained even after she was started on clobetasol solution and monthly injections of triamcinolone Acetonide (10 mg/mL). The start of prednisolone (20 mg/d) systemic treatment was made. At the time of follow-up, the patient's symptoms had subsided and additional hair loss had stopped, therefore the prednisolone dosage had been gradually reduced to 5 mg/d. Additional systemic drugs were recommended, such as 200 mg twice day of hydroxychloroquine; however, since the patient did not take the prescription as directed, it was stopped. A trial of tetracycline (500 mg twice a day) was attempted, but the patient was noncompliant because of the gastrointestinal side effects. The patient then experienced a complete remission, as she became symptom free for around 3 months, so all medications were stopped.

Case 3-Folliculitis Decalvans And Lichen Planopilaris Phenotypic Spectrum

Background- Folliculitis decalvans (FD) and lichen planopilaris (LPP) are classified as neutrophilic and lymphocytic cicatricial alopecias according to the North American Hair Research Society. Recently, a clinical phenotype combining concomitant or sequential features for both was described as a FD LPP phenotypic spectrum (FDLPPPS).

Case- A 33-year-old female patient with alopecia, follicular erythema, follicular scales, and intermittent pustules on her scalp for 30 years was seen at the Meenakshi Medical College, Hospital and Research Institute, Kanchipuram, Department of Dermatology, Venereology, and Leprosy. Thirty years ago, she had

suffered from pustules on her scalp for an undisclosed reason before experiencing significant hair loss. After receiving systemic antibiotic therapy, her condition gradually improved and she started growing new hair. On her scalp, though, follicular erythema and scaling began to show, accompanied by sporadic pustules. Ten years ago, the symptoms started to become more noticeable; symmetrical cicatricial alopecia lesions with follicular papules, pustules, hemorrhagic crusts, perifollicular erythema, and scale accumulation occurred on the scalp. She had multiple diagnoses, including discoid lupus erythematosus, FD, psoriasis, and LPP. Among other therapies, she was given topical and oral antibiotics, topical corticosteroids, and oral hydroxychloroquine in order of diagnosis. She still experienced recurrent pustules, scaling, and increasing follicular erythema as her symptoms gradually worsened. The patient denied having any relevant family medical history and denied having ever had a chronic illness or sunburn.

The findings of the hair pull test were negative, and a dermatological examination revealed two patches of symmetrical cicatricial alopecia on the scalp, measuring 7 by 10 cm and 8 by 10 cm, with perifollicular erythema, hyperkeratosis, and yellowish scales.

No lesions were found on the face, extremities, mouth, nails, or the remaining (Figure 1). A histopathological study of the hair follicle revealed hyperkeratosis, moderate epidermal hyperplasia, and damage of the epithelial basal layer. The hair follicle was evidently surrounded by extensive lymphocyte and histiocyte infiltration, with notable plasma cell infiltration in certain regions. Only the arrector pill muscle's structure was visible on horizontal sections, where there was partial hair follicle loss and surrounding fibrosis (Figure 2). Upon trichoscopy, the following findings were



observed: pustules, follicular tufts, dilated blood vessels, follicles encircled by patches of milky red, and partial loss of follicular opening (Figure 3). These trichoscopic, histological, and clinical results supported the diagnosis of FDLPPS.

Throughout a 6-month follow-up, the patient showed no signs of hair loss progression and no erythematous lesions or scaling after using oral prednisone tablets (20 mg/day, tapered over 2 months) and azithromycin (500 mg thrice weekly for 1 month). The patient also stopped having pustules throughout this time.



Figure 1-Clinical manifestations of the patient. (A) Two symmetrical cicatricial alopecia patches on the scalp; (B) Recurrent follicular papules, pustules, and hemorrhagic crusts in the early stage (photographed by the patient herself); (C) Perifollicular erythema, keratosis, scales, and follicular tufts in the late stage.

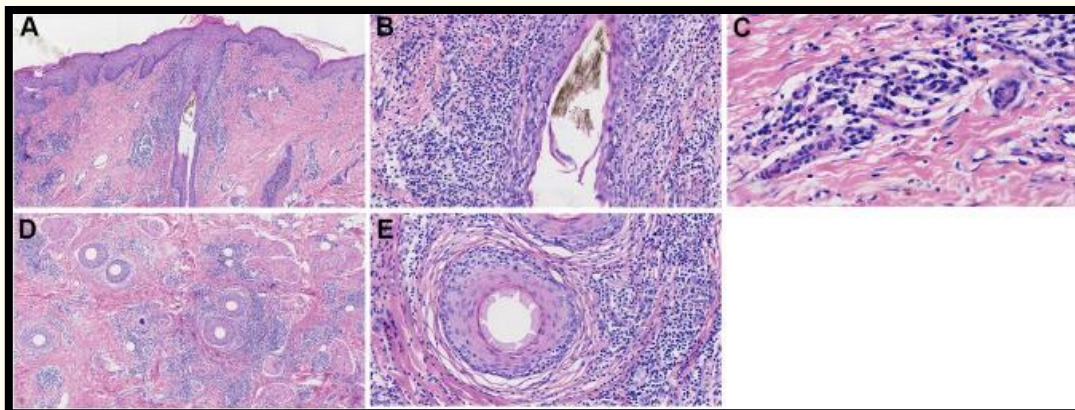


Figure 2-Histopathological findings in a vertical section showing (A) hyperkeratosis and mild epidermal hyperplasia (HE×40), (B) epithelial basal layer destruction of the hair follicle with surrounding infiltration of dense lymphocytes and histiocytes (HE×200); (C) significant plasma cell infiltration (HE×400); Horizontal section showing (D) partial destruction of hair follicles (HE×40); (E) only the structure of the arrector pill remains (HE×200).



Figure 3-Trichoscopy showing follicular tufts, pustules and dilated blood vessels in an area of scarring alopecia.



Management

- The treatment aim is to stop inflammation and further irreversible destruction of hair follicles. Antibiotics remain the first-line therapy, due both to their anti-inflammatory and antimicrobial properties. Although topical fusidic acid is widely used as adjuvant treatment, there are few data regarding its oral use. We report a case of folliculitis decalvans successfully treated with oral fusidic acid. Our patient was a 33-year old.
- Direct microscopic examination and mycological culture showed no fungal element. A diagnosis of folliculitis decalvans was established and the patient was started on oral fusidic acid at a dose of 500 mg three times a day.
- Betamethasone dipropionate 0.05% and salicylic acid 3% lotion as well as azelaic acid 5% lotion were also applied to the affected area once daily.
- After two months of treatment, the patient showed clinical improvement, with less erythema and suppuration of the affected scalp.
- A partial hair regrowth was noted, mainly at the periphery. Subsequently the patient maintained only topical therapy, and no recurrences were observed after 6-months of follow-up.
- During the following year, recurrence was observed in only one patient after ending zinc sulfate therapy.
- Oral antibiotics are frequently used to treat folliculitis decalvans. Tetracyclines and the combination of clindamycin with rifampicin are the most commonly used.
- However, the disease usually progresses when treatment is stopped. Fusidic acid is an anti-staphylococcal drug with few adverse effects. It is highly bioavailable orally, and has a long plasma half-life. Despite years of clinical use in numerous countries, resistance rates remain at low levels to date.

Discussion

Various forms of LPP, such as "classical LPP," frontal fibrosing alopecia (FFA), and FAPD, a form of LPP distinguished by the presence of miniaturisation, can manifest as patchy, marginal, or patterned alopecia. With a peak onset age of between 30 and 60 years old, LPP is more frequent in women than in males [11]. Patients frequently suffer more hair shedding, scalp burning,

itching, and discomfort at first. Trichoscopy reveals peripilar casts around hair tufts in cases of active illness. Additionally, scalp erythema is typically seen. As the condition progresses and follicular openings in affected areas eventually disappear, hair loss becomes more noticeable [12]. LPP generally has a slow and insidious course of disease, although less frequently, extensive hair loss can occur within months in a more rapid disease course [13].

Currently, as a result of the infrequency of the disease and limited literature availability, no definitive treatment approach has been recognized. There remains no curative therapy and the main goal of treatment is reducing inflammatory symptoms and slowing the progression of hair loss. Treatment commonly involves the use of high potency topical and/or intralesional corticosteroids and orally administered hydroxychloroquine [14]. Other systemic treatment options include tetracyclines, pioglitazones, cyclosporine, mycophenolate mofetil, methotrexate, or systemic corticosteroids. A systematic review concluded that topical/intralesional steroids or hydroxychloroquine can be seen as first-line agents for treating classic LPP, although this is not based on direct comparisons and the quality of evidence for many therapeutic options is low [15]. In recent years, naltrexone has been shown to have anti-inflammatory properties with the potential to be used as a treatment modality for autoimmune conditions [16]. A case series of four patients on low dose naltrexone for treatment of LPP is the only study on the subject and has shown therapeutic benefits including a decrease in inflammation and inflammatory symptoms with slowed disease progression [17].

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