

# Lichen planopilaris

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**ABSTRACT:** Lichen planopilaris is a chronic scarring alopecia characterized by follicular hyperkeratosis, perifollicular erythema, and loss of follicular orifices. The scalp lesions may be single or multiple and commonly involve the vertex and parietal area. The hair follicles at the margin of the alopecic patches reveal perifollicular erythema. Anagen hairs can be pulled out easily in active lesions. Associated cutaneous, nail, and mucous membrane lichen planus may be present. Commonly encountered symptoms and signs are increased hair shedding, itching, scaling, burning, and tenderness. Differentiation from other cicatricial alopecia can be performed through meticulous evaluation of the clinical, histopathologic, and immunohistopathologic findings. Treatment strategies depend on the disease activity and physician expertise. Although there are no definitive curative modalities, some new discoveries and conceptual advances continue to broaden our treatment options of this complex condition.

**KEYWORDS:** alopecia, cicatricial, lichen planopilaris, scarring

## Introduction

Lichen planopilaris (LPP) is an uncommon inflammatory hair loss disease characterized by autoreactive lymphocytic destruction of the hair follicle and progressive scarring alopecia of the scalp. Perifollicular erythema, scaling, and groups of keratotic follicular papules are commonly encountered clinical findings of scalp LPP. In the early stages, diagnosis is made through both clinical and histopathologic findings. However, in the later stages, not many specific signs are present.

Since Pringle's first description of LPP (1), many reports have elaborated on its precise clinical features. LPP is a variant of lichen planus that can be classified as morphologically follicular lichen planus. The cause and pathogenesis of this disorder is poorly understood. According to the cicatricial alopecia classification of the North American Hair Research Society (2), LPP is classified as a primary lymphocytic cicatricial alopecia. In primary scarring alopecias, the hair follicle itself is destroyed with relative sparing of the interfollicular dermis.

Most patients with cicatricial alopecia have an intense concern and are distressed by this permanent hair destruction (3). An exact diagnostic approach as well as a stepwise treatment program is crucial. An update on LPP and the practical diagnostic and therapeutic approaches will be discussed.

## Background

Lichen planopilaris can be subdivided into three groups, including classic LPP, frontal fibrosing alopecia (FFA), and Graham-Little syndrome. They share clinical, histopathologic, and immunohistopathologic findings. Classic LPP presents as scalp hair involvement and is sometimes accompanied by extracranial lichen planus lesions. FFA is characterized by progressive band-like scarring alopecia of the frontal hairline that usually affects middle-aged women (4). Graham-Little syndrome, also known as Graham-Little-Piccardi-Lassueur syndrome, has a triad of cicatricial alopecia of the scalp, lichen planus of the skin with widespread follicular papules (lichen planus spinulosus), and nonscarring hair loss of axillary and pubic area (5). This article will focus mostly on classic LPP. Other variants of LPP also will be discussed briefly.

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## Etiology and pathogenesis

Although the cause and pathogenesis of LPP remain poorly understood, the most widely accepted theory states that it is a hair-specific autoimmune disorder in which activated T lymphocytes target follicular antigens (6). LPP is one of the clinical variants of lichen planus. Cell-mediated immunity plays a major role in triggering the clinical expression of LPP. In lichen planus, T lymphocytes destroy keratinocytes, which express an unknown antigen on their surface. In LPP, most infiltrated T lymphocytes are located around the bulge area (7), which is a specialized portion of the outer root sheath epithelium defined as the insertion site of the arrector pili muscle. Lineage studies have proven that bulge area cells are multipotent and that their progeny generate the new lower anagen hair follicle (8). The failure of affected follicles to regenerate is thought to be due to destruction of follicular stem cells located in the bulge area. T lymphocytes seem to be activated by Langerhans cells that are increased in the LPP lesions (9).

The inflammatory process involving the bulge area and lymphocytes can be subdivided into three groups, including antigen recognition, lymphocyte activation, and keratinocyte apoptosis. These complex reactions are carried out by antigen-presenting cells, adhesion molecules, and inflammatory cytokines. Recent work on integrin expression emphasize the importance of adhesion molecular expression patterns (10).

It is proposed that a cell-mediated reaction is potentially initiated by the action of an endogenous or exogenous agent, such as medication, virus, or contact sensitizers, which bind to keratinocytes as well as follicular epithelium. Thereafter, keratinocyte and hair follicle may act as signal transducers, capable of converting these stimuli into producing cytokines and chemotactic factors for initiation of inflammation. This inflammation activation may accelerate further inflammation and stimulate production of interferon-gamma, tumor necrosis factor-alpha, and antigen-presenting cell interactions (11-13).

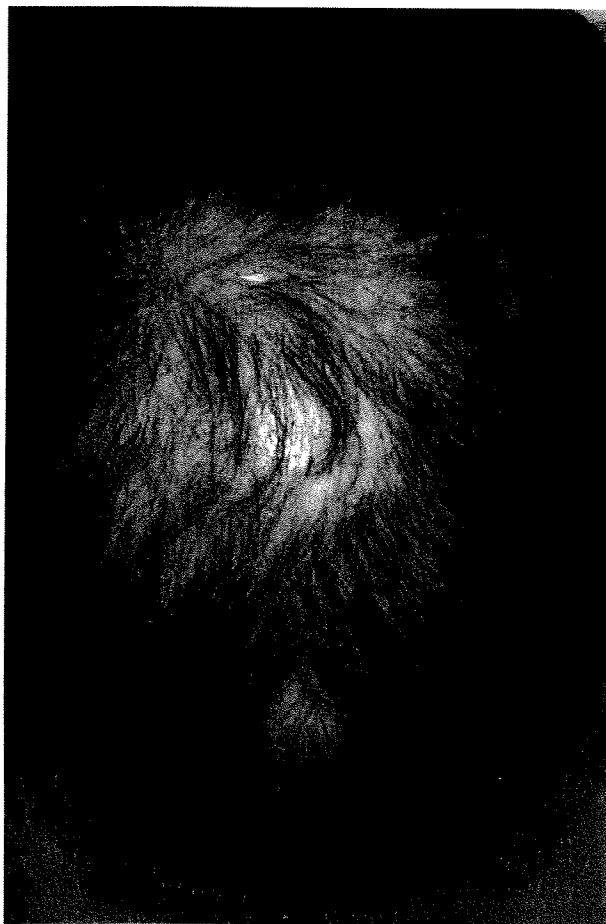
Contact sensitizers such as metals could act as haptens and can evoke an inflammatory reaction. Commonly encountered metals are gold, mercury, and cobalt. The role of infection in the development of lichen planus and LPP has been raised over the last several years. These infection or microorganisms include hepatitis C virus, human immunodeficiency virus, herpes simplex virus 2, *Helicobacter pylori*, human papillomavirus, and syphilis. Lichenoid

drug eruptions, which exhibit similar cutaneous and histopathological findings of lichen planus, are reactions that may occur after exposure to various medications. Most common agents include antimalarial agents, gold, beta-blockers, thiazide, quinidine, and angiotensin-converting enzyme inhibitors (14,15). Lichenoid lesions can be observed in cases of chronic liver disease, chronic bowel disease, diabetes, thyroiditis, thymoma, malignancy, trauma, and occasionally stress (16). Although the pathogenesis of LPP show extensive similarities to that of lichen planus, some reports point out differences in immunoreactant deposition (17). Further detailed investigation is needed to delineate the distinctive characteristics of LPP.

## Clinical features

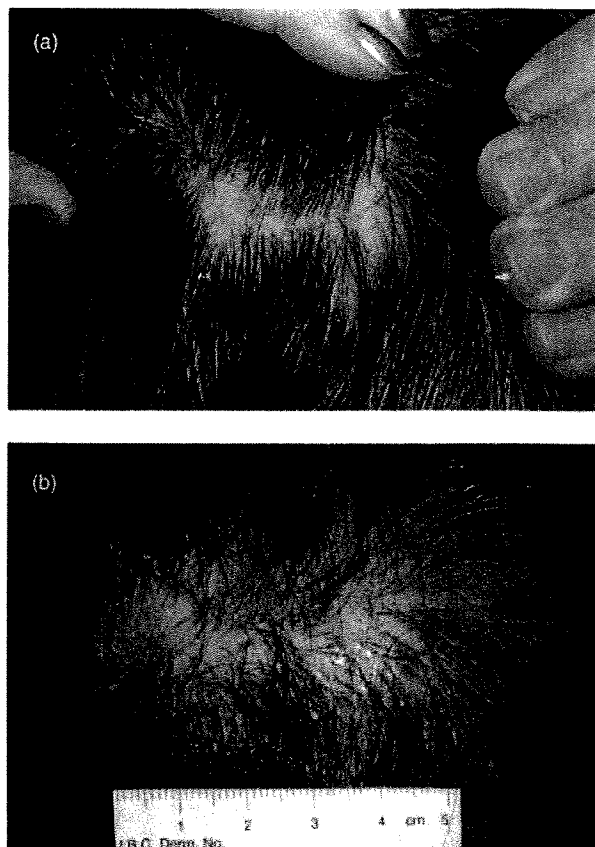
Lichen planopilaris more frequently occurs in Caucasian and East Indian females. The University of British Columbia study found a lower incidence in the Asian population compared to other ethnic groups (18). The female-to-male ratio is 1.8 : 1 (18). The majority of patients are adults between 25 and 70 years, although it occasionally can be observed in children (18-20). This disease may develop alone or in association with other forms of cutaneous, nail, or mucous membrane lichen planus. The known incidence rate of LPP with lichen planus is variable according to reports, although 17-28% of LPP patients may have other forms of lichen planus (18). The scalp lesions may be single or multiple, focal or extensive and mostly involve the vertex and parietal area (FIG. 1) (18,21). Small alopecic patches of LPP can slowly progress and become interconnected with each other, which can lead to a reticulated pattern. Furthermore, these patches may coalesce to produce larger scarring patches, and sometimes the entire scalp may be involved (22,23). Characteristic polygonal, erythematous, flat-topped papules of cutaneous lichen planus are rarely found on the scalp. Early stage or mild lesions of classic LPP may be difficult to diagnose and a scalp biopsy is recommended to confirm the diagnosis.

The classic lesion of LPP is characterized by whitish atrophic or scarring patches on the scalp with complete loss of follicular orifices (23,24). The surrounding marginal hair follicles and residual small hairy islands within the patch reveal perifollicular erythematous macules and scale (FIG. 2). Acuminate keratotic plugs can be frequently observed in the margins of the expanding area of alopecia. Positive pull test of anagen hairs indicate



**FIG. 1.** Scarring alopecic patches mostly involving vertex and parietal portions of the scalp with interconnections and polycyclic borders.

disease activity. Scalp ulceration may be found infrequently. Commonly encountered symptoms are increased hair shedding, severe itching, scaling, burning, and tenderness. These symptoms can be aggravated by ultraviolet light, irritation, sweating, and stress. Disease activity can be evaluated by examining hairs at the rim of patches. Stability of the condition usually reveals asymptomatic, noninflammatory lesions and a negative hair pull test. At this stage, it clinically may not be distinguished from other lymphocytic inflammatory disorders that completely destroy the hair follicles. The only visible remnants are whitish scars and rarely some tufted hairs. The clinical features of classic LPP overlap with those of discoid lupus erythematosus and other primary lymphocytic cicatricial alopecia (1). Scleroderma, erythema dyschromicum perstans, dermatitis herpetiformis, and hyperthyroidism may be seen in association with LPP (22,23,25). Fibrosing alopecia in a pattern distribution, which has characteristic features of progressive miniaturization of central



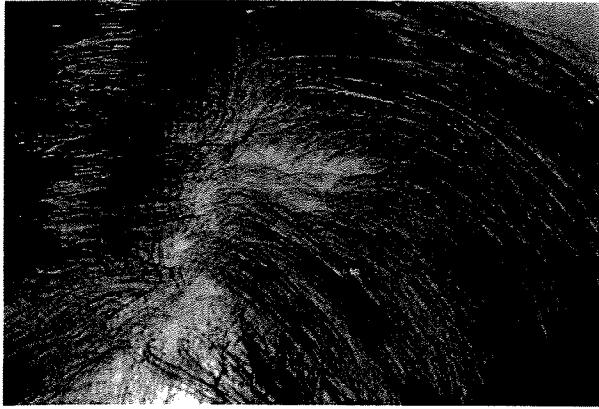
**FIG. 2.** Irregularly shaped atrophic patches with perifollicular erythema, scaling, and loss of follicular orifices.

scalp hair, often presents with perifollicular erythema, follicular hyperkeratosis, and eventually complete loss of follicular ostia (26).

Frontal fibrosing alopecia is a progressive band-like scarring alopecia mostly confined to the frontal hair line. Postmenopausal women are predominantly affected even though it can occur occasionally in premenopausal women and men (27). Its characteristic clinical findings include: follicular hyperkeratosis with perifollicular erythema, progressive recession of frontal and temporal hair line, and loss of follicular ostia. Fifty-two percent of FFA patients also have eyebrow hair loss (28). In contrast to classic LPP, FFA has no multifocal areas of scarring alopecia and is very rarely associated with lesion of lichen planus.

### Differential diagnosis

The diagnosis of LPP is suggested when patients present with cicatricial alopecia, loss of follicular orifices, perifollicular erythema, and hyperkeratotic follicular papules in the margins of expanding areas of alopecia. A sudden onset of patchy hair



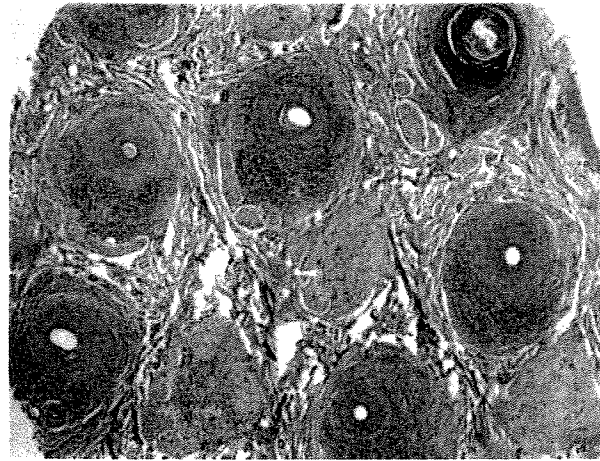
**FIG. 3.** Dark discoloration of the alopecic patches in lichen planopilaris making the diagnosis more difficult to differentiate from discoid lupus erythematosus.

loss on the scalp may be diagnosed as alopecia areata. Careful examinations for the existence of follicular orifices, perifollicular erythema and scaling, as well as keratotic follicular papules are mandatory to rule in or out LPP. When pruritus with erythematous scaly patches is present, seborrheic dermatitis should be considered in the differential diagnosis.

The distinction of classic LPP from other primary cicatricial alopecias, especially lymphocyte-associated conditions, such as discoid lupus erythematosus, pseudopelade of Brocq, and FFA, can be difficult and requires histopathologic and/or immunohistopathologic studies. In contrast to LPP, discoid lupus erythematosus lesions show linear deposition of immunoreactants on the dermoepidermal junction and superficial blood vessels (29). Sometimes dark discoloration of the alopecic patches in LPP makes it difficult to clinically differentiate from discoid lupus erythematosus (FIG. 3). Lesions in patients with pseudopelade of Brocq represent mild clinical features, consisting of less inflammation, mild atrophy, and irregular patches like the "footprints in the snow" pattern. Some authors have suggested that pseudopelade of Brocq is the end stage of other inflammatory cicatricial alopecias such as LPP (30); however, there is still continued debate about whether it is or not. There may be some difficulty in differentiating FFA from alopecia areata. A scalp biopsy is strongly recommended to differentiate among these conditions (31).

### Histopathologic features

Lichen planopilaris shows scarring as defined by the destruction and fibrosis of hair follicles (32).



**FIG. 4.** Perifollicular lymphocytic infiltrate with prominent lichenoid reaction of the follicular epithelium and partial absence of sebaceous glands (hematoxylin and eosin,  $\times 40$ ).

Histopathologic findings are variable according to disease progression and sometimes it is difficult to finalize a diagnosis. The histopathologic changes in the active and early-stage disease process provides more information that can lead to the diagnosis of LPP. In cases of late stage or "burned out" stage, the histopathologic findings may not reveal more than a follicular scar. The biopsy site selection is an important process to establish an exact diagnosis. An active lesion that includes a site in the hair-bearing margin of the alopecic patch is recommended for biopsy. Active inflammatory areas usually reveal scaling with erythema and are symptomatic. However, some patients present with diffuse hair thinning and scaling over the frontal and parietal scalp, absence of follicular orifices, and gradual extension to the entire scalp. In these cases, a biopsy should be taken from the most symptomatic area.

The histopathologic feature in early stages is a lichenoid lymphocytic infiltrate affecting the infundibulum and isthmus with the lower portion of hair follicle being spared (FIG. 4) (7,33). Thereafter, an interface dermatitis occurs between the follicular epithelium and adjacent dermis with loss of the basement membrane zone. Sebaceous glands are lost in early lesions and the root sheaths of the hair follicles are destroyed (32,33). Occasionally wedge-shaped hypergranulosis within the affected infundibula may be found. There is no increase in dermal mucin and abnormal vascular plexus structure changes as in discoid lupus erythematosus (29). Finally, the follicular structure will be replaced with extensive perifollicular lamellar fibrosis, especially around the isthmus, and horizontal band-like scarring beneath

the papillary dermis. Direct immunofluorescence may show positive colloid body staining with anti-immunoglobulin M (IgM), anti-IgA, anti-IgG, or C3, at the dermoepidermal junction or around the infundibulum (34). FFA has similar histopathologic characteristics to LPP, which show a lymphocytic infiltrate around the isthmus and infundibulum, but it is less severe than LPP (35,36). In advanced lesions, the hair follicles are completely destroyed and replaced by thick fibrous tracts.

## Treatment

Management of the LPP generally begins with efforts to reduce subjective symptoms and prevent inflammation and spread. Treatment strategies depend on the severity of disease activity, age of the patient, as well as physician experience. Various forms of treatment regimens have been developed in the past several decades. They have mostly been developed empirically, and as with all other diseases of unknown cause, new regimens are being tried constantly. Although there are no definitive curative modalities, some new discoveries and conceptual advances continue to broaden our treatment options of this complex condition.

Like that of other cicatricial alopecias, treatment guidelines can be made on the basis of clinicopathologic diagnosis, age, and clinical severity. Clinical severity is determined by symptoms, signs, rapidity of disease progression, and extent of scalp involvement (37). There are limited reports about available effective options.

### Corticosteroids

In mild to moderate LPP, if scalp involvement is less than 10%, intralesional triamcinolone acetonide at a concentration of 10 mg/mL for a total of 2 mL every 4–6 weeks is recommended at the University of British Columbia Hair Clinic (18,38).

Intralesional corticosteroid injections frequently reduce inflammation significantly. However, close attention should be paid to the occurrence of scalp denting/atrophy. If this occurs, injections should be skipped for one or two treatment sessions. Intralesional corticosteroid injections can be maintained until activity has been stabilized. If there is no significant reduction of symptoms and clinical signs of inflammation or if there is progression of the disease within 3 months, other treatment options should be considered or added.

Topical treatments with high potent corticosteroids (class I and class II corticosteroids) can

be used in conjunction with the intralesional injection to provide symptomatic relief (38). Some patients display an overall good response to topical corticosteroids (19). In our clinic, clobetasol lotion or cream are recommended as first-line topical corticosteroids applied twice daily. Ancillary clobetasol (Clobex®, Galdern, Canada Inc., Tharnhill, Ontario) shampoo twice a week is effective to reduce some of the severe itching and burning sensation. Fluocinolone acetonide in an oil formulation (Derma-Smoothe/FS®) is helpful to remove scales and to calm down the inflammation. Patients are advised to use this preparation at night time on severely affected scalp areas under a shower cap.

Oral corticosteroids are reserved for rapidly progressing, aggressive disease, and severe subjective symptoms. Prednisone is added at 1 mg/kg daily and it can be tapered over 2–4 months (39,40). Moreover, prednisone can be used as bridge therapy until other agents show effectiveness (41).

### Hydroxychloroquine

Hydroxychloroquine has been utilized in the treatment for LPP (33,37). First noticeable effects of treatment may be found within 2–3 months and maximal clinical efficacy may take up to 6–12 months (42). Although the exact mechanism of action in which hydroxychloroquine exerts a treatment effect in LPP remains unclear, it is possible that it interferes with antigen presentation, production of cytokines such as tumor necrosis factor-alpha and interferon-gamma, and stimulation of toll-like receptor 9 family receptors (43,44).

In patients with more than 10% scalp involvement and/or little responsiveness to topical or intralesional corticosteroids, oral hydroxychloroquine (200 mg twice daily) is commenced after an ophthalmologic examination and blood test including complete blood count and liver function tests (40,41). Adverse reactions are very rare, but include abdominal pain, anorexia, nausea, myalgia, skin hyperpigmentation, hematologic changes, and ophthalmologic damage. Regular checkups, usually every 3–6 months, are required (37). Smoking is known to decrease efficacy of hydroxychloroquine and should be discouraged (45).

### Immunomodulating agents

If symptoms and signs of the disease persist after 3–6 months of corticosteroids and hydroxychloroquine therapy, another systemic medication should

be considered. Recently two kinds of immunomodulating agents, cyclosporine and mycophenolate mofetil, have been advocated for the treatment of uncontrollable LPP. Oral cyclosporine has been used for severe refractory lichen planus of the skin (46,47). As a result, it may be worthwhile to try in refractory LPP. Cyclosporine has been reported to be effective in LPP unresponsive to hydroxychloroquine and corticosteroids. A significant decrease in pruritus, burning, and pain, as well as perifollicular erythema and hyperkeratotic follicular plugs were noted for 12 months in three out of three cases (37,48). Usual recommended cyclosporine dosage is 3–5 mg/kg per day. Liver and kidney function tests and blood pressure must be monitored.

Mycophenolate mofetil has been successfully used for the treatment of some clinical variants of lichen planus (49,50). According to its relative safety and effectiveness compared to other immunomodulators, it may be indicated for several autoimmune-related skin diseases. Decreased LPP lesional thickness and effective prevention of new lesions were observed by 4 weeks of mycophenolate mofetil treatment in one case (51). The main mechanism of action is the specific inhibition of the proliferation of activated T and B lymphocytes through noncompetitive, reversible inhibition of inosine monophosphate dehydrogenase (52). The recommended dosage for LPP treatment is 500 mg twice daily for 4 weeks and sustained for 5–6 months at a dose of 1 g twice daily (37,51). Baseline liver function test and complete blood count should be checked before therapy is started.

### Miscellaneous

Other third-line therapy reported for refractory and aggressively progressive LPP include systemic retinoids, tetracycline, griseofulvin, thalidomide, dapsone, topical tacrolimus, and minoxidil. However, their effectiveness is still controversial.

Systemic isotretinoin has been reported to have beneficial effects in generalized or oral erosive lichen planus (53,54). This experience has suggested isotretinoin and acitretin use in patients with refractory LPP. Isotretinoin is usually started at a dose of 1 mg/kg per day and can be maintained for 8 months (18). Acitretin also can be tried at a low dose and slowly increased up to 0.6 mg/kg per day (55). Although the mechanism of action of retinoids is unknown, it may involve normalization of hair follicular keratinocyte antigen expression or suppression of the inflammatory cellular infiltration. However, it should be

noted that relapses may occur shortly after discontinuation of therapy. Side effects of these medications include cheilitis, xerosis, conjunctivitis, desquamation, epistaxis, paronychia, and reversible alopecia (56). Griseofulvin, 500 mg twice daily, has been reported to be helpful in LPP (57). However, there was no clinical improvement with griseofulvin in a patient with LPP accompanied by dermatitis herpetiformis (58). Topical minoxidil 5% solution can be added to recruit and prolong the life of anagen hairs as well as to aid in any underlying pattern hair loss that may be concomitant with the LPP (18). Thalidomide may have a beneficial effect on the control of LPP. However, its side effects and limited effectiveness need to be considered (59,60).

Topical tacrolimus has been proven an effective treatment option for hair growth in animal models of hair loss due to its induction of early anagen (61). Topical tacrolimus has shown some effectiveness, especially in the reduction of symptoms in FFA patients (62) and in refractory LPP patients when combined with other treatment options such as topical and intralesional corticosteroids. Dapsone can be another alternative (63,64). Tetracycline at 1 g per day showed statistically significant improvement as published by the Cleveland Clinic Foundation; however, antibiotic efficacy in LPP is still controversial (65).

If active inflammation has not been present and the hair pull test is negative for more than 2 years, hair transplantation and/or scalp reduction can be attempted cautiously (41). Please refer to Dr. Unger's article in this issue.

Patient support and education is important for many patients with cicatricial alopecia. We recommend two websites for patient education: [www.carfintl.org](http://www.carfintl.org) and [www.nahrs.org](http://www.nahrs.org).

### Conclusions

Lichen planopilaris is a prototype of cicatricial alopecia characterized by a reticulated pattern of scarring with perifollicular erythema and scales that may be single or multiple, and mostly involve the vertex and parietal area. The histopathologic findings are a crucial clue to help clarify this disorder. Early diagnosis and treatment are key to prevent widespread involvement and differentiate from other nonscarring alopecia. Treatment options depend on age and disease severity in terms of symptoms and extent of scalp involvement. Topical, intralesional, and systemic therapies singly or in combination may be necessary. Although there

are no definitive curative modalities, new discoveries and conceptual advances continue to broaden our treatment options of this rather complex condition.

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