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Lichen Planus: An Update and Review

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Lichen planus (LP) is a papulosquamous eruption of the skin, scalp, nails, and mucous membranes. Although LP is more common in adults, it has become an established pediatric disorder. Its classic presentation is characterized by 4 p's: purple, polygonal, pruritic papules. Histopathologic examination reveals characteristic interface dermatitis. Although its pathogenesis is not fully understood, there is evidence that an imbalance of immunologic cellular reactivity is central. Lichen planus usually resolves within a few months. Treatment that primarily consists of topical and/or oral steroids will expedite recovery and alleviate symptoms. Resolution of this cutaneous disease often is accompanied by postinflammatory hyperpigmentation. Long-term sequelae of LP in the pediatric population are rare, but cutaneous atrophy and pterygium unguis may occur.

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Lichen planus (LP) is a papulosquamous eruption characterized by small, violaceous, pruritic, flat-topped, polygonal papules. This distinctive dermatosis often is idiopathic but may be induced by medications or other chemicals. Lichen planus chiefly affects individuals aged 30 to 60 years, but pediatric LP is an established though less prevalent condition.¹ Lichen planus tends to resolve within a few months with an expedited recovery if the appropriate treatment is used.

History

Sir William James Erasmus Wilson² was most likely the first to describe LP in his review in 1869. He characterized the disease as “an eruption of pimples remarkable for their color, their figure, their structure, their habits of isolated and aggregated development.” In 1892, Kaposi³ reported the first clinical variant of the disease, lichen ruber pemphigoides. In 1895, Wickham⁴ noted the characteristic reticulate white lines on the surface of LP papules. Today the white lines are recognized as Wickham striae. Darier is credited with the first formal description of the histopathologic changes associated with LP.⁵

Epidemiology

The exact incidence and prevalence of LP is unknown. In 1895, Kaposi⁶ noted the disease as “rather frequent” with 25 to 30 cases presenting annually. In the United States, the incidence of LP is reported to be approximately 1% of all new patients seen at health care clinics.⁷ Internationally, the frequency of disease varies but may be slightly more prevalent among men.⁸ The Indian subcontinent has a particularly high incidence of disease.¹ Pediatric cases are uncommon, representing only 2% to 3% of patients with LP.⁹

Pathophysiology

Although the pathogenesis of LP is not fully understood, there is strong evidence that the disease development involves an imbalance of immunologic cellular reactivity. At the dermoepidermal junction of a lesion, activated T lymphocytes are found with an abundance of CD4⁺ cells in established lesions.¹⁰ The recruited lymphocytes induce apoptosis in basal keratinocytes; this interaction is enhanced by an increased expression of basal keratinocytes in intracellular adhesion molecule 1.¹¹ Molecules shown to influence this interaction include tumor necrosis factor α , IFN- γ , nuclear factor κ B-dependent cytokines, fas/apolipoprotein 1, and B-cell chronic lymphocytic leukemia/lymphoma 2.¹²⁻¹⁵

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Immunizations for hepatitis B virus infection and influenza virus may trigger the cytotoxic lymphocyte-mediated reaction that produces LP.^{16,17} In pediatric patients, a bullous variant of LP has been reported following administration of hepatitis B virus vaccine.¹⁸ Lichen planus also has been associated with hepatitis C virus infection,¹⁹ which induces aberrations in cytokine expression that may predispose patients to LP development.²⁰ Onset of LP also has been linked to various pigments; metals; and medications including, among others, beta-blockers, nonsteroidal anti-inflammatory drugs, and antimalarial agents.²¹ The manner by which these agents promote LP is unknown. There also is evidence for a possible genetic contribution in the development of the disease. Cases of familial LP have an increased frequency of HLA-B7.²² Associations also have been made for idiopathic LP and HLA-DR1 and HLA-DR10.²³

Clinical Manifestation

Classic LP is characterized by 4 *p*'s: purple, polygonal, pruritic papules. Initially, LP is evident as a cutaneous and mucosal eruption, though rarely it can manifest with only oral or nail findings. Lichen planus usually begins as discrete, flat-topped papules that are 3 to 15 mm in diameter with Wickham striae evident on their surfaces (Figure 1). The papules often are located on the flexor surfaces of limbs (Figure 2) and also may appear on lines of trauma, reflecting the Köbner phenomenon (Figure 3). In 1 week, a generalized LP eruption occurs, with the most intense spread from weeks 2 to 16.²¹ The severity of pruritus varies, with hypertrophic lesions presenting as the most severe. The morphologic variants of cutaneous LP in pediatric patients include linear, hypertrophic, annular, follicular, oral, actinic (Figure 4), vesiculobullous,



Figure 1. Purple polygonal papules with Wickham striae found in lichen planus.



Figure 2. Eruptive lichen planus on the trunk and flexor surfaces of the upper extremities.



Figure 3. Lichen planus lesions appearing at the site of a prior injury, reflecting the Köbner phenomenon.



Figure 4. Actinic lichen planus on the chest of an adolescent girl.

and pemphigoidlike (Table).²⁴ Resolution of cutaneous LP often is associated with postinflammatory hyperpigmentation, which also may be accompanied by cutaneous atrophy in hypertrophic and annular variants.

When LP involves the mucous membranes, it commonly appears on the tongue and buccal mucosa. Other sites for the disease include the conjunctivae, larynx, esophagus, tonsils, bladder, vaginal vault, vulva, and anus. Within the oral cavity, LP can induce a burning sensation or cause painful erosions. The lesions are characteristically tender, white, reticulated patches or plaques on a violaceous background. Oral LP is uncommon in younger patients, with an incidence of less than 1% of all pediatric LP cases.^{25,26}

Ungual findings are rare in pediatric LP. Affected nails may appear dull and show thinning of the nail plate, longitudinal fissuring, and distal splitting.²⁷ Dorsal pterygium unguis resulting from irreversible

damage to the nail matrix is the most specific nail abnormality for LP. Lichen planus also has been associated with childhood idiopathic nail atrophy and may overlap with 20-nail dystrophy of childhood.

Lichen planopilaris (LPP) refers to LP involving the hair follicles. Lichen planopilaris may be focally tender and/or pruritic; hyperkeratosis also may be present. If left untreated, LPP can lead to scarring alopecia. There is a correlation between LPP and nutritional deficiencies.²⁸

Lichen planus sometimes is found with other diseases of altered immunity such as discoid lupus erythematosus, ulcerative colitis, vitiligo, dermatomyositis, morphea, primary biliary cirrhosis, lichen sclerosus et atrophicus, myasthenia gravis, and alopecia areata.^{1,29-32} Although once believed to have a risk for malignant transformation, cutaneous LP has not been shown to promote squamous cell carcinoma or other skin cancers³³; however, oral LP may have a small malignant potential,²⁶ and LP of the

Variants of Pediatric Lichen Planus

Variant	Clinical Features
Linear	Isolated linear lesions that may be zosteriform or appear in prior sites of trauma, reflecting the Köbner phenomenon
Hypertrophic	Intensely pruritic, scaly, hypertrophic nodules often found on extensor surfaces of lower extremities, especially around ankles
Annular	Purely annular papules are rare; buccal mucosa may have violaceous plaques with atrophic centers
Follicular ^a	Keratotic papules of the scalp that may coalesce into plaques; it is more common in women; may result in scarring alopecia
Oral	Painful eroded or ulcerated lesions often found on mucosal surfaces; may result in scarring
Actinic	Mildly pruritic photodistributed lesions; characteristic nummular patches with hypopigmented zone surrounding hyperpigmented center
Vesiculobullous	Vesicles or bullae found in existing lichen planus lesions; presents mostly on the lower extremities or in the mouth
Pemphigoidlike	Blisters that develop into papules of lichen planus; clinical, histologic, and immunologic features of both lichen planus and bullous pemphigoid

^aLichen planopilaris.

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genitalia also has been linked to a low incidence of squamous cell carcinoma.³⁴⁻³⁷ These patients must be examined annually, possibly for life, to promote early diagnosis of malignant degeneration.

Diagnosis

Clinical findings of LP often are specific enough to begin treatment. Hepatitis C virus infection should be considered in the differential diagnosis, and if suggested, serologic testing also should be done. In cases of LPP, nutritional deficiencies also may need to be assessed. It is important to review the patient's medications, especially if there have been recent changes in regimen.

Skin biopsy specimens may be valuable to confirm the diagnosis, particularly in uncertain cases. Histologic examination reveals interface dermatitis with orthokeratosis, hypergranulosis, acanthosis, sawtoothing of the epidermal rete ridges, pigmentary incontinence in the superficial dermis, and absence of parakeratosis.^{7,38} Hyperorthokeratosis in the presence of hyperparakeratosis and eosinophils in the dermis suggests a lichenoid drug eruption.^{39,40} In the lower epidermis, degenerative keratinocytes known as Civatte bodies or colloid bodies are found. The basal layer shows liquefactive degeneration. At the dermoepidermal junction, there is a characteristic bandlike infiltrate of lymphocytes, consisting primarily of helper T cells and histiocytes.^{7,38} The Civatte bodies have immunoglobulins, which are mostly IgM and complement deposits. Direct immunofluorescence (DIF) testing visualizes these molecules at the basement membrane zone.⁴¹ This test has a sensitivity of 75% for detecting LP and aids in the differentiation of LP from immunobullous disorders.⁴²

Differential Diagnosis

In diagnosing LP, one may need to consider other papulosquamous diseases. Although cutaneous LP has a classic presentation, it can be confused with psoriasis, secondary syphilis, lichen nitidus, lichen simplex chronicus, prurigo nodularis, lichen striatus, papular granuloma annulare, papular sarcoidosis, or epidermal nevi.^{7,38} Vaginal LP can similarly present as lichen sclerosus et atrophicus, bullous disorders, and atrophic vaginitis. Oral LP has been misdiagnosed as leukoplakia, discoid lupus erythematosus, candidiasis, aphthous stomatitis, and herpes simplex virus. The patient's history, presence of Wickham striae, and skin biopsy findings can help to identify LP.

To distinguish LP from lupus erythematosus, one may notice that the Civatte bodies in lupus are numerous and deeper, there is vacuolization on both sides of the basement membrane zone,

and DIF testing shows a granular linear arrangement at the basement membrane zone.^{7,38} Antinuclear antibodies and other autoantibodies may be found in lupus erythematosus and other collagen vascular disorders. Lichenoid drug eruptions have lesions with increased eczematization, hypertrophy, hyperpigmentation, and scaling. Graft-versus-host disease can present with lichenoid papules that are indistinguishable from idiopathic LP under light microscopy and DIF.⁴³⁻⁴⁵ History of bone marrow transplant is essential in the differentiation of the two entities.

Management

Although the symptoms of LP are discomforting, the disease often resolves within 8 to 12 months. In cutaneous LP, topical steroids are the initial treatment; systemic steroids can be used as second-line treatment. To minimize the side effects of potent steroids, close supervision by the administering physician is mandated. A combination of oral and topical corticosteroids is useful for widespread cutaneous LP.⁷ Intralesional triamcinolone is effective for the treatment of hypertrophic LP in older children who do not respond to topical corticosteroids.⁴⁶

In LP that involves the oral mucosa, topical steroids are administered as first-line therapy. Although cyclosporine and other immunomodulating agents have been successful in some adult patients,⁴⁷⁻⁵² these drugs are relatively contraindicated for use in pediatric patients. Adequate oral hygiene under the guidance of a dentist is recommended. For unresponsive cutaneous or oral LP, retinoids may need to be considered.⁵³⁻⁵⁵ Acitretin was shown to be effective in a double-blind placebo-controlled trial.⁵⁵

Phototherapy primarily with narrowband UVB has been employed to treat LP for several years. Although no controlled studies assessing the efficacy of this therapeutic option are available, several case series reported relief of symptoms and even remission of the disease with the application of phototherapy.⁵⁶⁻⁶¹ Psoralen plus UVA therapy also has been found to be effective in the treatment of LP.^{62,63} However, it carries long-term risks for squamous cell carcinoma and cataracts as well as other phototoxic reactions. Medications that do not have strong evidence regarding effectiveness and may be considered as third-line treatments of LP include griseofulvin, oral metronidazole, thalidomide, phenytoin, and dapsone.^{47,52,64-69}

Lichen planopilaris is difficult to treat, so ultra-potent topical steroids or intralesional steroids are first-line treatments.²⁸ Alopecia from untreated disease may be permanent. In pediatric patients, LP of

the nail is treated with oral corticosteroids or oral retinoids.⁷⁰ Intralesional triamcinolone injected into the nail matrix is an option for adult patients. When LP is generalized and recalcitrant to other therapy, intermittent megadose corticosteroid therapy may be considered.⁷¹

Prognosis

Most children with LP show full clearance of the disease within 6 months of treatment.²⁴ Although long-term sequelae in the pediatric population are uncommon, cutaneous atrophy following the resolution of hypertrophic LP, permanent alopecia from untreated LPP, and pterygium unguis with chronic nail disease may occur. Recurrence of LP is rare; the noninfectious nature of the disease as well as the side effects of the treatment options should be emphasized to the patient's family.

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