

Definition

 Lichen Planus is a common inflammatory disorder of skin characterized clinically by distinctive, violaceous, flat topped papules; and histologically by a band like lymphocytic infiltrate at the dermo-epidermal junction.

EPIDEMIOLOGY

Epidemiology:

- Risks for the condition include:
 - Exposure to medicines, dyes, and other chemicals (including gold, antibiotics, arsenic, iodides, chloroquine, quinacrine, quinide, phenothiazines, and diuretics)
 - Diseases such as hepatitis C
- Race: No racial predispositions.
- Sex: Lichen Planus effects women more compared to men (3:2) ratio.
- Age: More than two thirds of lichen planus patients are aged 30-60 years; however, lichen planus can occur at any age

Etiology

- T-cell-mediated autoimmune disease
- CD8+ T-cells trigger the apoptosis of oral epithelial cells.
- Antigen associated with the major
 histocompatibility complex (MHC) class I on
 keratinocytes
- Grinspan's syndrome: lichen planus, diabetes mellitus and vascular hypertension



Commonly affected areas

- Lower legs, inner aspect of thighs
- Forearms, arms
- Lower back
- Oral cavity
- Genitals
- Nails



TYPES OF CUTANEOUS LICHEN PLANUS

HYPERTROPHIC PLAQUES



VESICULAR LICHEN PLANUS

LICHEN PLANUS PEMPHIGOIDES

Mechanism involves keratinocyte antigen expression or unmasking of an antigen that may be a self-peptide or a heat shock protein

T cells (mostly CD8+, and some CD4+ cells) migrate into the epithelium

These migrated CD8+ cells are activated directly by antigen binding to major histocompatibility complex (MHC)-1 on keratinocyte or through activated CD4+ lymphocytes

The activated CD8+ T cells in turn kill the basal keratinocytes through tumor necrosis factor (TNF)-α

(N Lavanya, P Jayanthi, Umadevi K Rao, and K Ranganathan. Oral lichen planus: An update on pathogenesis and treatment. J Oral Maxillofac Pathol. 2011 May-Aug; 15(2): 127–132.)

SYNDROME STATEMENT

Signs and Symptoms:

- The following may be noted in the patient history:
 - Lesions initially developing on flexural surfaces of the limbs, with a generalized eruption developing after a week or more and maximal spreading within 2-16 weeks
 - Pruritus of varying severity, depending on the type of lesion and the extent of involvement
 - Oral lesions that may be asymptomatic, burning, or even painful
 - In cutaneous disease, lesions typically resolving within 6 months (>50%) to 18 months (85%); chronic disease is more likely oral lichen planus or with large, annular, hypertrophic lesions and mucous membrane involvement

LICHEN PLANUS - THE 6 Ps'

- · P Pruritic
- •P Planar
- P Polygonal
- •P Purple
- •P Plaques
- •P Papules





Diagnostic tests

- Histological exam
 - ☐ Requires biopsy
 - Varies based on the type of lesion
 - Typically: epithelial hyperplasia, orto and para keratosis, acanthosis, atrophic areas w/ loss of rete pegs, dense accumulation of T-lymphocytes in the basilar cell layer
- Direct Immunofluorescent examination
 - Requires biopsy
 - □ Differentiates between other autoimmune conditions
 - Detects shaggy deposition of fibrinogen along the basement membrane

MANAGEMENT

Management

- Lichen planus is a self-limited disease that usually resolves within 8-12 months.
- Mild cases can be treated with fluorinated topical steroids.
- More severe cases, especially those with scalp, nail, and mucous membrane involvement, may necessitate more intensive therapy.

MANAGMENT

Treatments and Rationale

- Treatments may include:
 - Antihistamines
 - Medicines that calm down the immune system, such as cyclosporine (in severe cases)
 - Lidocaine mouthwashes to numb the area and make eating more comfortable (for mouth sores)
 - Topical corticosteroids (such as clobetasol) or oral corticosteroids (such as prednisone) to reduce swelling and lower immune responses
 - Corticosteroids shots into a sore
 - Vitamin A as a cream (topical retinoic acid) or taken mouth (acitretin)
 - Dressings placed over skin medicines to protect from scratching
 - Ultraviolet light therapy for some cases

LICHEN PLANUS

MEDICOWESDME

EPIDEMIOLOGY

- Age: peak at 30-60 years Sex: F>M (3:2)
- Prevalance: 0.1-4% (rare)

PATHOPHYSIOLOGY

- Sawtooth lymphocytic infiltrates at dermoepidermal junction
- Increased thickness of a. stratum corneum (with parakeratosis)
 - b. stratum granulosum
- c. stratum spinosum (with colloid bodies)
- Liquefactive degeneration of stratum basale

ASSOCIATIONS

- Hepatitis C
- Ulcerative colitis
- Aplastic anaemia Vitiligo, diabetes and other autoimmune conditions

CLINICAL FEATURES

Skin lesion (6 Ps):

- Pruritic, Polygonal, Purple, Planar, Papule and Plaque
- Most common site: scalp + flexor aspects of wrists. Can involve lower
- back, genital and shins. Associated with scarring alopecia
- Mucosal lesion

- White lace-like/reticulae (Wickham striae)
- Most common site: buccal mucosa

DIAGNOSIS Mainly clinical

- Supported by histopathology

TREATMENT

- Spontaneous remission in 6 months to 2 years
- 1st line: topical steroids
- 2nd line: topical cyclosporine, tacrolimus, isotretinoin
- Diffuse involvement: oral steroids