

Lichen Planus (Lichen Ruber Planus) — Causes and Symptoms

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The primary pathophysiology of lichen planus is CD8 cell-mediated damage to keratinocytes. Lichen planus can be found in various sites on the body. Topical steroids remain the mainstay of the treatment, but there are many other medications (e.g., retinoids) and therapies (e.g., UV-B) that have been tried. In this article, we will study the etiology, pathophysiology, clinical features, diagnostic techniques and the common treatment modalities for this condition.

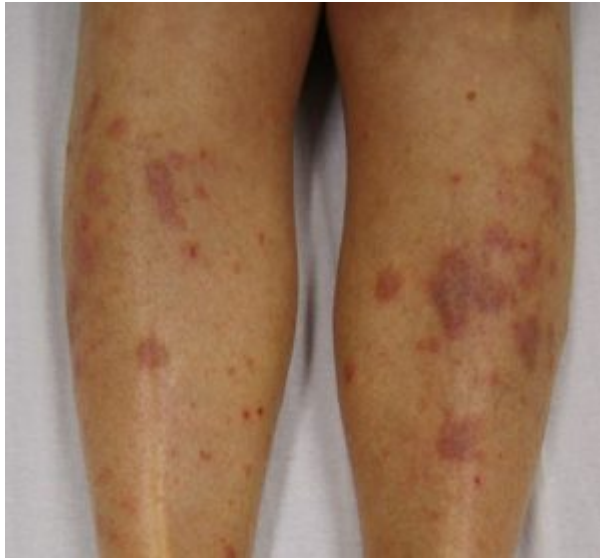


Definition and Epidemiology of Lichen Planus

Lichen planus is an acquired, chronic, immune-mediated disease, manifesting as polygonal, purple, **pruritic, planar papules** or **plaques** on the skin and other lesions on the mucosal membranes, such as in the oral cavity.

Lichen planus is reported in less than 1% of the population. The incidence may increase in December and January. No significant geographical variation and no racial or sex predispositions have been observed. Children are rarely affected; most patients affected by lichen planus lie in the range of 30 - 60 years.

Etiology of Lichen Planus



[Image](#): "Lichen plants affecting the shins." by James Heilman, MD
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- Genetics and familial.
- Chronic [hepatitis C](#) virus infection.
- Human herpesvirus type 7.
- Mercury (in dental amalgam, for oral lichen planus).
- Radiotherapy.
- Psychogenic factors, such as anxiety, depression, and stress.

Pathology and Pathophysiology of Lichen Planus

Lichen planus is thought to be an immunologically mediated disorder. It seems to be a **CD8 cell**-mediated response against antigens in the basal cell layer and the dermo-epidermal junction (mainly keratinocytes). The cause of this response is unknown, and exposure to certain viruses (e.g., **hepatitis C** and **hepatitis B**) or medications have been considered.

The immune-mediated damage to **keratinocytes** releases melanin into the dermis, leading to **hyperpigmentation**. The main pathology of the lesion is primarily found at the interface of the **squamous epithelium** and **papillary dermis** (interface dermatitis). At this junction, microscopically, a dense **lymphocyte infiltration** is seen.

The damaged **basal keratinocytes** often atrophy or become necrotic; they appear like the mature cells of the **stratum spinosum** (**squamization**). This, in turn, causes sawtoothing (angulated contour) of the interface. In the dermis, anucleate, necrotic basal cells (colloid or **Civatte bodies**) are seen. In addition to these changes, other features of lichen planus include **epidermal hyperplasia**, **hypergranulosis**, and **hyperkeratosis**.

Symptoms of Lichen Planus

The characteristic skin lesions of lichen planus are **polygonal, purple, pruritic, planar papules**, (remember four Ps) which may coalesce to form **plaques**. The lesions may be close together or widely separated from each other. They may have white dots or lines

called **Wickham striae**; they may also be **hyperpigmented**.



Image: "Wickham's striae." by James, Candice, Mai - <http://bohone09.wikispaces.com/Group+1>. License: [CC BY-SA 3.0](https://creativecommons.org/licenses/by-sa/3.0/)

The lesions are usually symmetrically distributed, often in extremities, particularly around the elbows and wrists. However, they may be found anywhere on the body, including on the **glans penis** and **vulva**. Usually, papules flatten in a few months and remain as areas of **hyperpigmentation**.

Mucous membrane (e.g., oral) lesions can be also found in patients with lichen planus; in 15% of cases, the involvement is predominant mucosal with minimal skin lesions.

In all, 50 - 70% of patients have oral lesions. These lesions can be **whitish papules** or **plaques** and/or white striae. They generally do not ulcerate, but this may occur due to **epitheliomatous transformation**. In women, sometimes, lichen planus manifests as **desquamative inflammatory vaginitis**.

Other variants of lichen planus include the following:

- **Hypertrophic**: Warty lesions, mainly on the lower limbs, which can persist for many years.
- **Follicular (lichen planopilaris)**: Multiple, small, spiny lesions around hair follicles.
- **Linear**: Isolated linear lesions made up of papules close to each other.
- **Actinic (lichen planus subtropical)**: Distinct discoid lesions with deeply hyperpigmented center surrounded by hypopigmented region; seen mainly in dark skin and young adults in tropical areas.
- **Lichen planus pigmentosus**: Diffuse or reticular macular hyperpigmentation.
- **Annular**: A few, large, discrete lesions, mainly on the penis and buccal mucosa.
- **Atrophic**: Few lesions, because of resolution or fading.
- **Guttate**: Wide scattered, small, discrete lesions.

Diagnosis of Lichen Planus

The diagnosis is usually established by **clinical examination**, although in doubtful or erosive cases, **histopathology** can help in differentiating from other similar diseases. In many cases, there may be comorbid conditions such as **diabetes** or **oral candidiasis**. These conditions should be checked for and treated accordingly. **Wickham striae** are usually seen in the oral lichen planus.

Differential diagnosis

The differential diagnosis can be complicated if the lesions are **atypical**. The differential diagnosis will vary depending on the location of the lesion, and often the presence of lesions in other parts (skin or mucosa) can help rule out some other diseases. Some of the more common ones are listed below:



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- Lichenoid drug eruptions
- [Warts](#)
- Scratching-induced eczematous eruptions
- [Lupus erythematosus](#)
- Lichen sclerosis
- Secondary [syphilis](#)
- [Psoriasis](#)
- **Erythema multiforme**
- **Leukoplakia**, gingivitis, oral candidiasis, smoker's patches (for oral lesions)
- Bullous diseases

Therapy of Lichen Planus

Most cases of lichen planus can be treated with **topical class I and II corticosteroids** (first-line treatment). **Systemic corticosteroids** are given when topical treatment fails or the disease is severe due to the involvement of the nails or scalp. However, systemic therapy is rarely needed due to the self-limiting nature of lichen planus.

For oral lesions, **topical triamcinolone paste** (0.1%) can be prescribed. Alternatively, 0.05% **fluocinonide gel or ointment** can also be applied bid or tid.

Oral prednisolone (0.5 mg to 1.0 mg/kg of body weight/day) or **intramuscular triamcinolone** (40-80 mg) for 4 to 6 weeks can be prescribed for severe mucosal lichen planus or nail lichen planus.

Oropharyngeal candidiasis and **epidermal atrophy** are common side effects of

corticosteroids.

Retinoids are also prescribed for the treatment of oral lichen planus (OLP) with papular and plaque-like form. Retinoids may be prescribed alone or with the corticosteroids. Most commonly used retinoids are:

- Isotretinoin 0.1% gel
- Tretinoin 0.025%
- Tazarotene
- Oral acitretin, 30 mg/day for 8 weeks for cutaneous lichen planus (second-line treatment)

Tretinoin is effective only in OLP, but not effective in the cutaneous lesion.

Common adverse effects of retinoids are **dyslipidemia**, **teratogenicity** and **liver enzyme elevations**.

Body lesions in lichen planus can be treated with 0.05% of **betamethasone dipropionate** or **diflorasone diacetate** cream.

Several studies have recommended the usage of **calcineurin inhibitors** (tacrolimus or pimecrolimus) in steroid-unresponsive cases; however, they have the risk of causing lymphoma and skin cancers in adults and children.

For **genital lesions**, topical corticosteroids or calcineurin inhibitors are preferred.

Some other treatments have also been reported, but their efficacy is uncertain because of limited studies:

- Sulfasalazine, 2.5 g/day for 6 weeks
- Mycophenolate mofetil, 1–1.5 g, twice daily
- Apremilast (inhibitor of phosphodiesterase 4)
- Oral metronidazole (the mechanism is unknown)

However, relapses occur despite these effective treatments. For extensive, widespread disease, **UV-B therapy** or **psoralen with UV-A (PUVA) therapy** for 8 weeks can be given; however, PUVA therapy can be **carcinogenic**, as can be some medications.

Progression and Prognosis of Lichen Planus

The prognosis is good. Skin lesions usually resolve spontaneously within 1 year. The oral lesions may take longer to resolve. Recurrence/relapse is not uncommon. Ulcerative lesions on the oral mucosa can undergo **malignant transformation** (because of the high expression of cyclooxygenase-2), but the rate is very low (<2%).

Vulvar lichen planus can undergo transformation to or may be associated with **squamous cell carcinoma**. Very rarely, even skin lesions can give rise to squamous cell cancer. The following factors increase the risk of transformation of lichen planus lesions to **squamous cell carcinoma**: smoking, heavy alcohol consumption, erosive or atrophic lesions, erythroplakic lesions, and mucosal involvement.

Review Questions on Lichen Planus

The correct answers can be found below the references.

1. **A patient has oral lichen planus which is refractory to topical steroid**

therapy. The doctor tries oral retinoids in this patient. Which of the following parameters should be closely monitored?

- A. Kidney function tests
- B. Lipid levels
- C. Serum electrolytes
- D. Hemoglobin
- E. Tumor markers

2. A patient has lichen planus lesions that have been present for more than 5 years despite adequate therapy. The lesions are flat and not itchy. Which of the following type of lesions can they be?

- A. Follicular
- B. Linear
- C. Annular
- D. Guttate
- E. Hypertrophic

3. Oral prednisolone is prescribed for severe mucosal lichen planus for?

- A. 4-6 weeks
- B. 1 week
- C. 2 weeks
- D. 3 days
- E. 8 weeks

References

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Correct answers: 1B, 2E, 3A

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