Episode 75 – Oral Lichen Planus December 30, 2022

Overview

- Lichen planus (LP) is a chronic inflammatory autoimmune disease that affects the skin, hair, nails, and mucous membranes (e.g., oral, vulvovaginal, esophageal, laryngeal, conjunctival mucosa). LP has a broad range of clinical manifestations, with the skin and oral cavity being the major sites of involvement.
- LP lesions most commonly present on the skin (cutaneous LP), in the oral cavity or on the lips. Less commonly, LP may also involve the genitals, scalp (lichen planopilaris), ears, nails, eyes, and esophagus. The disorder itself is not infectious.
- Skin hypertrophic and mucosal lesions are considered a potential premalignant condition since incidence of squamous cell carcinoma in these variants is ~1%. [1] [2] [3] [4] [5]

Clinical subtypes of LP

Several variations of LP have been described according to:

- Distribution and configuration of lesions,
- Morphology of an individual lesion, and/or
- Site of involvement.

The various clinical forms of LP are divided into general categories of cutaneous, mucosal, appendageal, and other forms. [1]

Lichen planus subtypes [1] [6] [7]

Category	Subtypes
Cutaneous LP	Localized cutaneous lesions of LP Generalized cutaneous LP Palmoplantar LP Hypertrophic LP Atrophic LP Actinic LP Vesiculobullous LP Erosive and ulcerative LP Annular LP LP pigmentosus Lichen planus pemphigoides Linear LP
Mucosal LP	Oral LP (OLP) ○ Reticular OLP

Category	Subtypes
	○ Plaque-like OLP ○ Papular OLP ○ Erosive OLP ○ Atrophic OLP ○ Bullous OLP Genital LP ○ Papular genital LP ○ Hypertrophic genital LP ○ Chronic erosive LP lesions in genitalia Esophageal LP Laryngeal LP
Appendageal LP	Lichen planopilaris (LPP) ○ Classic LPP ○ Frontal fibrosing alopecia ○ Graham-Little-Piccardi-Lassueur syndrome Nail LP
Other forms of LP	Drug-induced lichen planus Overlap syndromes: LP erythematosus Lichenoid reaction of graft-versus-host disease Lichenoid keratosis Ocular LP Aural and urethral LP

Etiology

- LP is a mucocutaneous disease affecting stratified squamous epithelia. Etiology is not known, but is likely multifactorial. It is thought to be caused by a T cell-mediated autoimmune response against basal epithelial keratinocytes in individuals with genetic predisposition.
- Stress, infections (e.g., human papilloma virus [HPV],[1] herpes simplex virus [HSV][2]), changes in the mucosal microbiome (e.g., *Candida* sp, various bacterial infections) and dental metals (contributing to OLP), play a role in disease manifestation. Metals associated with oral LP may include amalgam, copper, and gold.
- LP may be found with other autoimmune conditions (e.g., ulcerative colitis,[3] alopecia areata,[4] vitiligo,[5] myasthenia gravis,[6] morphea,[7] lichen sclerosus[8]).

[1] Refer to Episode 7, 12, 53, 58, & 61 for additional information on HPV.
[2] Refer to Episode 61 for additional information on herpes simplex virus.
[3] Refer to Episode 60 for information on ulcerative colitis.
[4] Alopecia areata is an autoimmune disorder that usually results in unpredictable, patchy hair loss.
[5] Vitiligo is a condition that causes patchy loss of skin pigmentation. Hair on these regions of skin can also lose pigment and appear white.
[6] Myasthenia gravis is a chronic autoimmune, neuromuscular disease primarily characterized by muscle fatigue and weakness.
[7] Morphea, also called localized scleroderma, is an autoimmune disease that causes sclerosis, or scarlike, changes to the skin.
[8] Lichen sclerosus is a chronic inflammatory skin disorder that most often affects genital and perianal areas.

- Associations with hepatitis (hepatitis B infection, hepatitis B vaccine,[9] and, particularly, hepatitis C-induced liver insufficiency) and primary biliary cholangitis[10] have been reported.
- Certain medications are considered responsible for drug-induced LP, especially beta-blockers, nonsteroidal anti-inflammatory drugs (NSAIDs), angiotensin-converting enzyme (ACE) inhibitors, sulfonylureas, gold, antimalarial drugs, penicillamine, and thiazides.[11] [1] [6] [8]

Drugs associated with lichen planus* [1] [6]

Drug type	Examples
Anticonvulsants	Carbamazepine, phenytoin, oxcarbazepine, valproate sodium
Antidiabetics	Chlorpropamide, glyburide, glipizide, insulin, tolazamide, tolbutamide
Antifungal drugs	Ketoconazole, amphotericin B, griseofulvin
Antihypertensives	ACE inhibitors, beta-blockers, nifedipine, methyldopa, diuretics (hydrochlorothiazide, furosemide, spironolactone, chlorothiazide)
Antimalarial agents	Hydroxychloroquine, chloroquine, pyrimethamine, quinidine, quinine
Chemotherapeutic agents	Hydroxyurea, 5-fluorouracil, imatinib, olmutinib
Lipid-lowering drugs	Gemfibrozil, orlistat, pravastatin, simvastatin
NSAIDs	Acetylsalicylic acid, diflunisal, ibuprofen, indomethacin, leflunomide, mesalamine, naproxen, rofecoxib, sulindac, sulphasalazine, tolbutamide
Sulpha drugs	Sulphonylurea, hypoglycaemic agents, dapsone, mesalazine, sulphasalazine
Metals	Gold salts, arsenic, mercury, bismuth, palladium
Psychiatric drugs	Antipsychotics (chlorpromazine, levomepromazine, methopromazine, thioridazine), benzodiazepines (lorazepam), lithium, selective serotonin reuptake inhibitor (escitalopram), tricyclic antidepressants (amitriptyline, imipramine)
Tuberculosis drugs	Ethambutol, isoniazid, rifampicin
Tumour necrosis factor antagonists	Infliximab, etanercept, adalimumab
Other drugs	Allopurinol, iodides and radiocontrast media, interferon-α, omeprazole, penicillamine, tetracycline, levamisole, clopidogrel, palifermin, mercaptopropionyl glycine, misoprostol, nandrolone, furyl propionate, norflex, omeprazole, pyrithioxin, sildenafil, tiopronin, isotretinoin, zidovudine, vaccines, solifenacin

*Nonexhaustive list

Epidemiology

- Incidence of LP is estimated between 0.14-1.27% of the general population.

[9] Refer to Episode 53 & 61 for information on hepatitis B virus and vaccine.
[10] Primary biliary cholangitis (formerly known as primary biliary cirrhosis) is an autoimmune liver disorder where the bile ducts are slowly destroyed, leading to cholestasis (i.e., flow of bile from the liver is reduced or blocked), cirrhosis, and liver failure. [16]
[11] Refer to Episode 62 for intitial discussion on drugs associated with lichen planus.

- At least two-thirds of cases occur in individuals aged 30-60 years, but LP can occur at any age. Children are rarely affected.
- No sexual or racial prevalence is evident in the cutaneous form, whereas 60-75% of individuals with oral LP are female.
- Familial cases are rare. [1] [8]

Signs & symptoms

- Cutaneous lesions classically present as pruritic, violaceous (purple), polygonal, flat-topped papules[12] that may coalesce into rough scaly plaques.[13] Lesions are initially 2-4 mm in diameter, with angular borders and a sheen when light is direct across the lesions. Lesions are often accompanied by oral and/or genital lesions.
- Papules are often covered by lacy, reticular, white lines (i.e., Wickham striae). These fine lines are considered highly characteristic and can be more easily observed after applying oil or water to the skin when visualizing lesions with a magnifying lens or dermatoscope.
- Lesions on nonkeratinized epithelium (e.g., buccal mucosa, tongue, esophagus, genitalia) are often nonpruritic and may be asymptomatic or present with pain or burning. Mucosal lesions often become erosive.
- Lesions are usually symmetrically and bilaterally distributed, most commonly on inner surface of the wrists, forearms, legs, torso, genitalia, and oral and vaginal mucosae but can be widespread. The face is less often affected.
- LP onset is usually acute with initial lesions almost always appearing on the extremities. A generalized eruption, in approximately one-third of the cases, may develop after one week or more with maximal spreading within 2-16 weeks.
- During the acute phase, new papules may appear at sites of minor skin injury (Koebner phenomenon), such as a superficial scratch. Lesions may coalesce or change over time, becoming hyperpigmented, atrophic, hyperkeratotic (hypertrophic LP), or vesiculobullous.[14]
- Sometimes hyperpigmentation remains after lesions heal.
- LP affecting the hair follicles often leads to permanent scarring resulting in islands of alopecia (lichen planopilaris).
- Nails are involved in up to 10% of cases. Findings vary from mild symptoms (e.g., discoloration of the nail beds, thinning of the nails, and formation of nail ridges) to complete loss of the nails and scarring from the cuticle to the skin under the nail.
- Drug-induced LP (also called lichenoid drug eruption) may be indistinguishable from nondrug-induced LP or may appear more eczematous. [1] [2] [5] [8]

Diagnosis

- Diagnosis is usually by clinical evaluation, supported by biopsy.
- If LP is diagnosed, laboratory testing for liver dysfunction, including hepatitis B and C infections, should be considered. [8]

[12] Papule is a solid raised lesion <1 cm.
[13] Plaque is a solid lesion >1 cm, raised or depressed compared to the skin surface.
[14] Vesiculobullous formation of both vesicles (raised, clear fluid-filled blisters <1 cm) and bullae (raised, clear fluid-filled blisters >1 cm).

Management

- Treatment is aimed at reducing length and severity of symptomatic outbreaks.
- Treatment usually requires topical or intralesional corticosteroids. Severe cases may require phototherapy or systemic corticosteroids, retinoids, or immunosuppressants.
- In mild cases, symptoms may be minimal or absent, requiring no therapy.
- Drugs suspected of triggering LP should be stopped. It can take weeks to months after the offending drug has been stopped for lesions to resolve. [8]

Prognosis

- LP can last for more than one year and can recur. Many cases resolve without intervention, presumably because the inciting agent is no longer present.
- Recurrence after years may be due to reexposure to the trigger or some change in the triggering mechanism.
- Drugs possibly causing the LP should be avoided.
- Classical cutaneous LP is self-limiting and usually resolves within 6 (>50%) to 18 months (85%). [1]
- Chronic disease is more typical in hypertrophic cutaneous lesions, oral lichen planus, and with nail or scalp involvement.
- Vulvovaginal lichen planus may also be chronic and refractory to therapy, causing decreased quality of life and vaginal or vulvar scarring. [1] [5] [8]

Oral lichen planus

- Li et al. (2020) conducted a systematic review and meta-analysis[15] examining the global prevalence and incidence of oral lichen planus (OLP). The overall estimated pooled prevalence of OLP was 0.89% among the general population and 0.98% among clinical patients. A higher prevalence of OLP was found in non-Asian countries, among females, and among individuals ≥40 years. However, the findings should be considered with caution because of the high heterogeneity of the included studies. [9]
- Oral mucosa is involved in ~50% of cutaneous LP cases. Oral lesions may occur without cutaneous lesions. [10]
- Up to 50% of females with oral mucosal findings have undiagnosed vulvar LP. [8]
- A meta-analysis by Li et al. (2017) found a significantly high prevalence of thyroid disease among individuals with OLP, suggesting routine screening for thyroid disease may be beneficial to those with OLP. However, due to the small number of studies included, further studies are needed to confirm the results. [1] [11]

Signs & symptoms

- Oral lesions are usually bilateral and often involve the gingiva[16] and present as desquamative gingivitis causing pain and discomfort during eating and toothbrushing.

[15] Refer to Episdode 73 for information on interpreting research.

[16] Note: According to the AAP classification of periodontal diseases and conditions, when OLP presents with gingival manifestations, it is classified as a non-plaque-induced gingival disease of autoimmune origin. [17]

- Buccal mucosa and tongue are also commonly involved.
- Periods of remission occur where symptoms and lesions appear and regress at intervals.
- Oral lesions may present alone or with skin lesions or other mucous lesions. [10] [12]

Risk factors

Risk factors associated with OLP include:

- Psychological stress and anxiety; however, this association remains controversial. Psychological support may be beneficial to some individuals with recurrent OLP.
- Certain drugs (e.g., NSAIDs, beta blockers, sulfonylureas, ACE inhibitors, antimalarials).
- Dental materials (e.g., amalgam, gold, cobalt, palladium, chromium, composite resins, prolonged use of denture wear).
- Various viruses (e.g., Epstein Barr virus [EBV], HPV, human herpes virus 6 [HHV-6], HHV-7, varicella zoster virus [VZV], hepatitis C virus [HCV][17]).
- Systemic diseases (e.g., dyslipidemia, thyroid dysfunction)**.**
- Autoimmune disorders (e.g., Sjögren's syndrome,[18] systemic lupus erythematosus[19] (SLE), celiac disease,[20] ulcerative colitis,[21] Hashimoto's thyroiditis).
- Nutritional deficiencies (e.g., vitamin A, B12, C, E, iron). [1] [10] [13]

OLP subtypes [2] [10]

Subtype	Description
Reticular OLP	Most common form of OLP. Presents as a lacy pattern of interconnecting keratotic white lines (Wickham striae). Typically located bilaterally on buccal mucosa, mucobuccal fold, gingiva, and less commonly on tongue, palate, and lips. Usually asymptomatic.
Plaque-like OLP	Clinically resembles leukoplakia. Ranges in presentation from smooth, flat areas to irregular, elevated areas. Commonly found on dorsum of the tongue and buccal mucosa. Usually asymptomatic. Seen with higher frequency among tobacco smokers.
Papular OLP	Rare form of OLP. Appears as papules on the oral mucosa, especially the buccal mucosa, often occurs concurrently with reticular OLP.
Erosive OLP (ulcerative)	Second most common type of OLP. Presents as mix of erythematous and ulcerated areas that may contain finely radiating keratotic striae. Lesions migrate over time and tend to be multifocal. Gingival lesions produce desquamative gingivitis. Symptoms of erosive form range from episodic pain to severe burning discomfort that can interfere with eating and oral selfcare.
Atrophic OLP (erythematous)	Rare form of OLP. Appears as diffuse, erythematous patches surrounded by fine white striae and can cause significant discomfort. Primarily affects the attached gingiva. Buccal mucosa can also be involved, particularly in the posteroinferior areas adjacent to the second and third molars.

[17] Refer to Episode 61 for information on hepatitis C.
[18] Refer to Episode 62 for information on Sjögren's syndrome.
[19] Refer to Episode 62 for information on systemic lupus erythematosus.
[20] Refer to Episode 60 for information on celiac disease.
[21] Refer to Episode 60 for information on ulcerative colitis.

Subtype	Description
Bullous OLP	Usually presents as bullae[22] on buccal mucosa and lateral borders of the tongue; bullae rupture soon after they appear causing painful ulcerations, resulting in classic appearance of erosive OLP. May be positive for Nikolsky's sign.[23]

Diagnosis

Diagnosis is usually based on clinical and histologic features. A biopsy is recommended to differentiate from other lesions. [13]

Differential diagnosis

OLP may resemble conditions such as leukoplakia, cheek chewing/frictional keratosis, candidiasis,[24] carcinoma, aphthous ulcers,[25] pemphigus,[26] mucous membrane pemphigoid,[27] lupus erythematosus,[28] and chronic erythema multiforme. [8] [14]

Management

- OLP is often difficult to treat, particularly when ulcerations and erosions are present. For years, treatment for OLP had been aimed at palliation rather than cure of oral symptoms. However, current treatments should intend to eliminate symptoms and potentially reduce risk of malignant transformation.
- Clients should be advised to maintain good oral hygiene and avoid mucosal trauma.
- Excellent oral hygiene is thought to reduce severity of symptoms, but can be difficult to achieve during periods of active disease.
- Depending on the severity of the disease, regular professional oral healthcare, avoidance of smoking, spicy food, and alcohol may be indicated for some individuals with OLP.
- If the cause of OLP is suspected to be a systemic drug, the medical provider should change the implicated drug to different medication.
- Long-term follow-up is necessary to monitor disease activity and to exclude malignant transformation of erosive lesions. [1]

Treatment

- Topical or systemic corticosteroids are used depending on lesion severity.
- Topical corticosteroids are the first line of treatment and do not have as many side effects as systemic corticosteroids.
- Systemic corticosteroids are used for more aggressive forms, multisite disease, diffused disease, or cases unresponsive to topical therapy.
- Treatment with both topical and systemic corticosteroids are used for the most severe cases.

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[22] Bulla is a raised, clear fluid-filled blister >1 cm.
[23] A positive Nikolsky sign occurs when firm sliding pressure separates normal-appearing epidermis at the basal layer and produces an erosion.
[24] Refer to Episode 61 for additional information on candidiasis.
[25] Refer to Episode 61 & 62 for information on aphthous ulcers.
[26] Refer to Episode 30 & 62 for additional information on pemphigus.
[27] Refer to Episode 62 for information on mucous membrane pemphigoid.
[28] Refer to Episode 62 for additional information on lupus erythematosus.

- Localized steroid injections can be used in cases unresponsive to topical or systemic treatment.
- Systemic retinoids, such as acitretin initially, followed by isotretinoin have been used to treat OLP. Topical retinoids (isotretinoin) or other forms of vitamin A derivatives can eliminate white lesions, but relapse was reported 2-5 weeks after treatment discontinuation.
- Systematic use of cyclosporine has been found to be effective in different studies.
- In OLP recalcitrant to topical corticosteroids, the use of topical calcineurin inhibitors, such as tacrolimus and pimecrolimus, is suggested as second-line treatment.
- Lesions may respond to oral dapsone, hydroxychloroquine, or cyclosporine. Cyclosporine rinses may also be useful. [1] [8] [10]

Complications

- OLP and the drugs used to treatment OLP (e.g., corticosteroids) may predispose to *C. albicans* infection.
- OLP can be very painful, and ulceration may lead to scarring.
- Research indicates OLP reduces oral health-related quality of life (OHRQoL) leading to physical, social, and psychological consequences that affect everyday life, especially social interactions. The clinical form of OLP is an important indicator for assessing the impact of OLP on quality of life. Individuals with reticular OLP generally report a higher quality of life than those with other clinical forms.
- A 1% incidence of squamous cell carcinoma has been reported among individuals with OLP, but is more prevalent in atrophic and erosive lesions, emphasizing the need for long-term follow-up of the condition. Proposed reasons for increased risk of carcinoma include:
    - Oral mucosa affected by OLP may be more sensitive to *C. albicans* and to the exogenous mutagens found in tobacco and alcohol.
    - In individuals with OLP, the chronic inflammatory response and simultaneous healing response of epithelial wounds may increase the likelihood of cancer-forming gene mutations. [1] [15]

Prognosis

Oral mucosal lesions usually persist for life. [8]

Take home messages

- Oral lichen planus is a chronic condition. Currently, there is no cure, so management of the condition focuses on helping severe lesions heal and reducing pain or other discomfort.
- Multidisciplinary care is necessary for clients with systemic lichen planus.
- OLP must be closely monitored with referrals to appropriate specialists considering the potential risk of malignant transformation.

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OLP Support Group
The Texas A&M School of Dentistry's online International Oral Lichen Planus Support Group serves as a resource for clients, family members, and practitioners.
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Additional Resources

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