



# Diagnosis and Management of Oral Lichen Planus

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**ABSTRACT** Oral lichen planus is a relatively common mucosal autoimmune disease that may be initially detected and diagnosed in the dental office. For asymptomatic patients, clinical characteristics including a generalized involvement of the oral mucosa are often sufficient to establish a working diagnosis. Symptomatic presentations of oral lichen planus, however, can mimic a variety of other potentially serious conditions and scalpel biopsy is recommended to determine an accurate diagnosis. Treatment strategies for the symptomatic patient are discussed.

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Lichen planus is a chronic, immunologically mediated condition first described as a disease of the skin that can also affect mucosal surfaces, including those that line the oral cavity. Oral lichen planus has been estimated to affect from 0.1 percent to 4 percent of the population.<sup>1</sup> Interestingly, while more than one-third of patients with cutaneous lichen planus will reportedly have oral involvement, only about 15 percent of patients with oral lichen planus ever develop skin lesions.<sup>2,3</sup> Although the etiology is unknown, most authorities agree it represents a form of autoimmune disease in which dysregulation of T lymphocyte function results in damage to, or destruction of, basal cells of the surface epithelium.<sup>4,5</sup>

The relatively high prevalence of oral lichen planus makes it likely that virtually every dentist who treats adult patients will encounter this condition. The fact that the mucosal changes in oral lichen planus

can occasionally mimic oral precancerous lesions or other significant conditions makes it important for all dentists to be aware of its clinical features. Practitioners should also know the additional steps that can be taken to confirm a clinical diagnosis of oral lichen planus, including incisional biopsy for routine histopathologic evaluation and direct immunofluorescent examination. Finally, as some patients with oral lichen planus are symptomatic and desire treatment, clinicians should be aware of current management strategies.

## Clinical Presentations of Oral Lichen Planus

Since a significant percentage of oral lichen planus patients will also have cutaneous involvement, skin lesions can be used to help support the clinical or working diagnosis. The classic skin lesions of lichen planus have been described as purple, polygonal, pruritic papules that are usually found in small clusters on the flexor aspects of the extremities (FIGURE 1).



**FIGURE 1.** Erythematous cutaneous papules and plaques of lichen planus on the lower leg of a female patient. (Courtesy of Doug D. Damm, DDS, Lexington, Ky.)

Fine, interlacing whitish lines known as Wickham's striae can occasionally be observed on the surface or periphery of the flat-topped papules and plaques. Dys-trophic nail changes develop in some patients and females can have vulvo-vaginal involvement that may be symptomatic.<sup>3,6</sup>

Oral lichen planus usually develops in middle-aged adults, and women are affected more often than men. It is quite uncommon in childhood, although affected patients often have associated cutaneous disease and a predisposition among children of Asian descent has been reported.<sup>7-9</sup> Several variants of oral lichen planus have been described, however, two major forms are recognized: reticular and erosive.

#### RETICULAR

Reticular oral lichen planus represents the most common clinical pattern of this disease. The word reticular refers to the net-like or lacy pattern of interlacing keratotic lines (also denoted as Wickham's striae) that is characteristic of oral lichen planus. Reticular oral lichen planus is usu-

ally asymptomatic and bilateral involvement of the posterior aspects of the buccal mucosa that may extend into the vestibules is virtually pathognomonic for this condition (**FIGURES 2A-B, FIGURE 3**). Some cases are predominated by small keratotic papules that may be interconnected by thin keratotic striae. With involvement of the dorsal aspect of the tongue, a lace-like quality may not be present and lesional tissue will often appear as single or multiple keratotic plaques with loss or coalescence of the filiform papillae (**FIGURE 4**).

The lesions of oral lichen planus tend to wax and wane in their clinical severity without any treatment. Many patients report nothing more than a vague awareness of tissue "roughness." Concomitant involvement of other mucosal sites, most often the gingivae, the dorsal and lateral aspects of the tongue and vermilion border, may be noted.

#### EROSIVE

The erosive form of oral lichen planus is much less common than the reticular form and differs in that most patients report symptoms with their oral lesions. Affected mucosa usually presents as an area of atrophy and erythema with variable zones of central erosion or ulceration and a peripheral border of fine, radiating keratotic striae. Affected sites are similar to those seen with reticular oral lichen planus and it is not uncommon to see both forms of the disease

manifest in the same patient (**FIGURES 5A-B**). Occasionally, lesional changes are relatively confined to the attached gingival or alveolar mucosa, producing a clinical pattern that has been termed "desquamative gingivitis" (**FIGURE 6**). Rarely, the erosive aspect of the disease is so severe that epithelial separation may occur and vesicle or bulla formation may be observed clinically.

As with the reticular form, erosive oral lichen planus tends to have a bilateral or multifocal mucosal presentation with periods of remission and exacerbation rather than steadily progressing course (**FIGURES 5A-B**). Symptoms can vary from mild discomfort to severe pain that interferes with normal mastication or speaking.

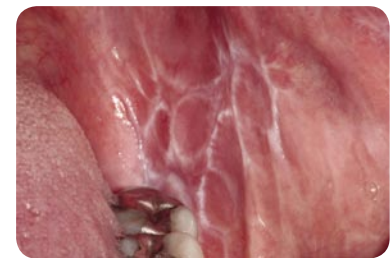
#### Diagnosis: Clinical

Even without a history or evidence of cutaneous lichen planus, reticular oral lichen planus with bilateral involvement of the buccal mucosa has such a characteristic pattern that clinical diagnosis alone is usually sufficient. It should be emphasized that even in "classic" cases, periodic patient re-evaluation would be warranted to detect any progressive tissue changes, and the patient should be advised to consider tissue biopsy in order to provide a firm, baseline histopathologic diagnosis.

The finding of a single area or an isolated mucosal lesion with a reticular or lichenoid appearance is not characteristic of oral lichen planus and is



**FIGURE 2.** Reticular oral lichen planus affecting right (a) and left (b) buccal mucosa. Note scattered small whitish papules and interconnected keratotic striations (Wickham's striae).



**FIGURE 3.** Reticular oral lichen planus of the posterior right buccal mucosa with well-defined lace-like pattern.



**FIGURE 4.** Reticular oral lichen planus of the dorsal tongue. Keratotic plaques can be seen on patient's right side and mid-dorsum while fine, internal striations are visible within the lesion on the left dorsolateral aspect.

more suggestive of conditions such as a lichenoid drug or contact hypersensitivity reactions (see related manuscript in this issue). To complicate matters, some oral lichen planus patients with generalized mucosal involvement may also have similar lesions localized to areas in direct contact with amalgam restorations (lichenoid amalgam reaction).<sup>10</sup> Careful history taking and clinical correlation may be helpful in assigning a working diagnosis and a biopsy is usually warranted. In presentations limited to keratotic plaque(s) of the dorsal, and especially dorsolateral, tongue, a biopsy would be mandatory to exclude the possibility of dysplasia (precancerous epithelial change) or squamous cell carcinoma.

For patients with suspected erosive oral lichen planus, the differential diagnosis can be quite broad. A biopsy should be recommended to support or confirm the clinician's working diagnosis and exclude other and potentially more serious conditions. Depending upon the precise clinical setting, the differential could

include epithelial dysplasia, squamous cell carcinoma, lichenoid reactions to drug, foreign body, amalgam, or other contact agents (such as artificial cinnamon flavoring), lupus erythematosus and chronic ulcerative stomatitis.<sup>11,12</sup> In patients with a history of bone marrow transplantation, the complication known as graft versus host disease can closely mimic the clinical features of oral lichen planus.<sup>12</sup>

If a desquamative gingivitis-like presentation predominates, conditions such as lichenoid foreign body reaction (possibly to dental prophylaxis materials), mucous membrane (cicatrical) pemphigoid, chronic ulcerative stomatitis and pemphigus vulgaris would need to be considered. Therefore, a biopsy should be considered for any case of persistent desquamative gingivitis that does not respond to conservative local hygiene measures. Submission of tissue for both routine and direct immunofluorescent examination will permit the exclusion or confirmation of a specific autoimmune disease, such as pemphigus vulgaris, as quickly as possible.

It should also be noted that oral lichen planus, reticular and erosive forms alike, may become complicated by the acquisition of superficial fungal microorganisms, usually *Candida albicans*. In most cases, this probably represents an opportunistic infection since *Candida* consume keratin and this substance is readily available in the keratotic papules

and striae produced by oral lichen planus.

Superimposed candidiasis may lead to mild "burning" discomfort of the affected mucosa, even in reticular oral lichen planus, and can further complicate the diagnosis by masking the classic net-like pattern of the keratotic striae. Cytologic or culture studies can aid in the management of these cases by providing positive identification of the microorganisms. Even without diagnostic tests, an empirical course of appropriate antifungal therapy (such as clotrimazole troches or fluconazole tablets) may unmask the characteristic clinical features of the underlying oral lichen planus and help reduce candidiasis-related symptoms.

#### Diagnosis: Routine Biopsy and Direct Immunofluorescence

The final diagnosis of oral lichen planus, especially in cases of erosive disease, often rests with a tissue biopsy of affected mucosa. Following appropriate local anesthesia, an elliptical wedge should be obtained that extends from lesional tissue into adjacent normal mucosa. Use of cautery methods is not recommended for this purpose due to artifactual changes they often induce within the specimen. In addition, erosive or ulcerated lesions must be handled gently to minimize the chance of peeling or splitting the surface epithelium from the underlying connective tissue, greatly degrading the diagnostic usefulness



**FIGURE 5.** Erosive oral lichen planus affecting left buccal mucosa (a) and same area seven months later (b). Bilateral involvement was noted at both time periods and patient reported a waxing and waning course. (Courtesy of Carl M, Allen, DDS, MS, Columbus, Ohio.)



**FIGURE 6.** Erosive oral lichen planus presenting as desquamative gingivitis in the canine-molar region of the right maxilla. All quadrants were similarly affected.

of the specimen. When it is important to exclude specific vesiculobullous conditions such as mucous membrane pemphigoid, a separate sample must be obtained for direct immunofluorescent examination because the routine formalin fixative interferes with direct immunofluorescent processing.

This can be accomplished with two separate biopsies, but can also be managed through careful planning and harvest of a single incisional specimen. Ideally, a “double-duty” biopsy should extend from just within the border of lesional tissue to several millimeters into normal-appearing mucosa. An overall length of 8 mm to 10 mm ensures adequate sampling for both studies. Once the tissue is removed, it can be carried to a table or sterile gauze and split across the short axis with a sharp scalpel. The “lesional” half of the specimen should be placed in formalin for routine histopathologic examination. The “normal” half can then be placed in Michel’s solution, a special liquid medium designed for direct immunofluorescence.

Oral lichen planus has several characteristic histopathologic features, including hyperkeratosis, vacuolar degeneration of the basal cell layer and degenerating keratinocytes termed colloid or Civatte bodies. Rete ridges may be absent or elongated with a pointed or “saw-tooth” appearance. A band-like infiltrate of small lymphocytes is seen immediately subjacent to the epithelium, occasionally destroying the epithelial-connective tissue interface. Unfortunately, these features are not specific to oral lichen planus and can be seen in several other conditions, such as lichenoid amalgam reaction, lichenoid drug reaction, mucosal cinnamon reaction, lupus erythematosus, graft versus host disease, and chronic ulcerative stomatitis. As a result, oral lichen planus is a diagnosis that demands careful correlation of the clinical setting with the results of routine biopsy examination.

Many practitioners are familiar with

oral vesiculo-bullous diseases like mucous membrane (cicatricial) pemphigoid or pemphigus vulgaris. In contrast, most dentists and physicians are unfamiliar with chronic ulcerative stomatitis, a specific mucocutaneous autoimmune disease first described in 1990 that can mimic the clinical features of oral lichen planus.<sup>12-14</sup> Chronic ulcerative stomatitis is associated with the development of

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circulating autoantibodies to a nuclear antigen in stratified squamous epithelium known as p63. For this reason, chronic ulcerative stomatitis has also been compared to both oral lichen planus and lupus erythematosus, another autoimmune disease that is characterized by the production of anti-nuclear antibodies.

The majority of chronic ulcerative stomatitis patients have been older adult women, and some patients have also presented with erosive or bullous skin lesions. Intraorally, the most commonly affected site is the tongue, followed by the labial or buccal mucosa and gingiva.<sup>14</sup> Similar to erosive oral lichen planus, lesions appear as shallow, irregular ulcerations but peripheral keratotic striae, if present, are usually abbreviated or vaguely formed. Gingival involvement produces a clinical presentation of desquamative gingivitis.

Direct immunofluorescent testing of oral lichen planus specimens is similar to routine histopathologic examination in that the results can be suggestive of or consistent with the diagnosis of oral lichen planus, but they are not specific to oral lichen planus alone. Most lesions demonstrate an irregular linear band of fibrinogen deposition at the basement membrane zone, a feature shared with other forms of lichenoid mucositis (see related manuscript in this issue), graft versus host disease, lupus erythematosus and chronic ulcerative stomatitis. The distinguishing feature for chronic ulcerative stomatitis patient specimens is the additional finding of punctuate (dot-like), intranuclear deposits of IgG in the basilar cells of the surface stratified squamous epithelium.

Patients with chronic ulcerative stomatitis have been shown to respond best to treatment with hydroxychloroquine (Plaquenil) and are usually resistant to initial treatment measures recommended for oral lichen planus patients. This provides a persuasive rationale for obtaining both routine and direct immunofluorescent examination in all cases of erosive oral lichen planus. Although chronic ulcerative stomatitis has been described as an uncommon or even rare autoimmune disease, the number of cases masquerading as oral lichen planus could be substantial due to similarities in their clinical and even routine histopathological features. Patients should be advised that the benefit of a correct diagnosis (including exclusion of other forms of autoimmune disease like pemphigoid or pemphigus) and early initiation of effective treatment for the patient more than justifies the added cost of baseline direct immunofluorescent testing.

### Management

Unlike cutaneous lichen planus, which is usually self-limited and spontaneously resolves within one to two years,

oral lichen planus is more commonly a chronic condition that often persists for multiple years, if not decades.<sup>11,17</sup> As with most forms of autoimmune disease, there is no cure for oral lichen planus. The primary goals of treatment are to reduce the length and severity of disease during periods of activity and, if possible, increase the periods of disease quiescence.

As mentioned, patients with asymptomatic reticular oral lichen planus do not require therapeutic intervention. Conservative measures to improve oral hygiene and minimize local tissue irritation may help reduce periods of notable tissue “roughness.” These could include decreasing the interval between professional dental prophylaxis (every four months instead of every six months), recommending the use of bland toothpaste or mouthrinse formulas and smoothing/repairing sharp or broken teeth, restorations, or prostheses. In the case of superimposed candidiasis, antifungal therapy would be appropriate to relieve associated symptoms.

Treatment of symptomatic erosive oral lichen planus is largely based on the use of topical corticosteroids, especially the higher potency formulations such as fluocinonide (Lidex) 0.05 percent, augmented betamethasone (Diprolene) 0.05 percent and clobetasol (Temovate) 0.05 percent. Gel formulations are preferable to creams or ointments as the latter are more hydrophobic and adhere poorly to the normally moist oral mucosa. Patients should be advised to apply the corticosteroid gel in a thin film directly to the lesional tissue four to five times daily. Emphasis should be placed on the use of tiny amounts of the gel multiple times a day rather than large amounts less often. After symptoms subside, patients can simply stop applying the gel without tapering the dosing schedule. Since oral lichen planus has a natural waxing/waning course, patients should be instructed

to re-institute their topical therapy at full strength whenever symptoms return. Dentists and hygienists should also encourage patients to improve or maintain excellent oral hygiene measures as this step leads to decreased disease activity, with or without topical corticosteroid treatment.<sup>16,18</sup>

In addition, it is important to inform the patient that while this treatment has not been approved in the United States by

**PATIENTS WITH asymptomatic reticular oral lichen planus do not require therapeutic intervention.**

the Food and Drug Administration, it is considered a well-documented “off-label” use for formulations originally marketed to treat skin conditions such as cutaneous lichen planus. More than three decades of scientific studies have shown these agents to be safe and efficacious in managing patients with oral lichen planus, yet no pharmaceutical company has pursued the costly process required by the FDA to receive formal approval for this application. It can be pointed out that significant complications from topical corticosteroid treatment of oral lichen planus have been rare, and only in cases where the patient substantially and improperly overused their medication. On the other hand, clinicians should also be aware that oral candidiasis is not an uncommon minor complication of topical corticosteroid therapy. These opportunistic infections (probably

resulting from mild local immunosuppression), however, are readily resolved with concomitant antifungal therapy.

For patients with widespread symptomatic disease or who have limited manual dexterity, possibly secondary to underlying conditions such as arthritis, aqueous corticosteroid solutions may be an effective alternative to gel formulations. Options include dexamethasone (Decadron) elixir, 0.5 mg/5 ml and prednisolone (Prelone) syrup, 15 mg/5 ml. Patients should be instructed to swish the solution over affected areas for a minute or so and expectorate without rinsing after meals and before bedtime.

A variety of other medications have been used in treating oral lichen planus, including other topical immunosuppressives (tacrolimus, retinoids, cyclosporine), systemic agents (corticosteroids, retinoids, dapsone, azathioprine, griseofulvin, thalidomide, levamisole), and PUVA (oral psoralen and low-dose ultraviolet A) or laser therapy.<sup>1,6,11,12,16</sup> Although encouraging results have been reported, these agents are typically more expensive than topical corticosteroid therapy without clear evidence of superior efficacy. Currently, their use should be reserved for erosive oral lichen planus patients who prove recalcitrant to topical corticosteroid treatment and prescribed under the guidance of a dental (i.e., an oral and maxillofacial pathologist) or medical specialist, i.e., a dermatologist.

### **Does Oral Lichen Planus Represent a Premalignant Condition?**

Numerous studies have addressed this important question; however, a definitive answer remains elusive.<sup>11,16,19</sup> Evidence from some reports indicates that patients with oral lichen planus, particularly those with erosive or atrophic forms, have an increased risk for the development of oral squamous cell carcinoma. Others have suggested that case reports or case series

of oral lichen planus that have undergone “malignant transformation” probably represent cases of oral epithelial dysplasia (precancerous change) that were misdiagnosed (clinically, microscopically or both) as oral lichen planus. In their recent review, Lodi et al. pointed out that oral lichen planus could be confused, both clinically and microscopically, with the condition known as proliferative verrucous leukoplakia.<sup>16</sup> Patients with proliferative verrucous leukoplakia may present with multiple leukoplakic areas throughout the oral cavity. Lesions of proliferative verrucous leukoplakia are considered precancerous with a significant rate of malignant transformation.

Obviously, the distinction between oral lichen planus and premalignant lesions is critical. For this reason, oral biopsy specimens should be interpreted by oral and maxillofacial pathologists, who are specifically trained in both the microscopic and clinical diagnosis of mouth diseases. With their experience in clinico-pathologic correlation, oral and maxillofacial pathologists are uniquely suited to provide an accurate diagnosis for these challenging cases and, if needed, to assist in patient management or follow-up.

Science has known for years that cancer is essentially a genetic disease that results from nonlethal damage to cellular DNA. Different patterns of damage can be seen in different forms of cancer and several chromosomal sites have been recognized as important to the development of epithelial dysplasia and oral squamous cell carcinoma. To date, the only molecular studies to address the issue of DNA damage in oral lichen planus have been presented by Zhang et al. using comparative genetic analysis of biopsy material to detect evidence of allelic loss or loss of heterozygosity at three different chromosomal sites related to oral squamous cell carcinoma.<sup>20</sup> Analysis of multiple examples of different oral mucosal lesions, including cases of oral lichen planus, benign

reactive hyperplasia, various degrees of dysplasia and oral squamous cell carcinoma was performed. Among the oral lichen planus specimens, evidence of loss of heterozygosity was lower than that for reactive hyperplasia (6 percent versus 14 percent) and was significantly lower in comparison to mild, moderate, or severe dysplasia/carcinoma-in-situ (40 percent, 46 percent, and 81 percent, respectively) as well as oral squamous cell carcinoma (91 percent). The

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follow-up study examined dysplastic lesions that mimicked oral lichen planus under the microscope (so-called lichenoid dysplasia) and found high levels of loss of heterozygosity in these cases that were essentially identical to dysplastic lesions lacking a resemblance to oral lichen planus.<sup>21</sup>

Confirmation of these results by other scientists is needed. It is possible that DNA damage occurs in oral lichen planus, but not in areas of the chromosomes that would have been detected by the panel of probes used by Zhang and co-authors. Overall, however, their molecular findings would argue that oral lichen planus is probably not a premalignant condition. The problem, particularly with erosive oral lichen planus, is that lesional tissue can occasionally resemble areas of erythroplakia, a clinical presentation that is suspicious for precancerous or cancerous change. As

mentioned previously, baseline biopsy with direct immunofluorescent is recommended in all cases of erosive oral lichen planus to establish the diagnosis. Subsequently, any lesional tissue that appears to worsen progressively despite appropriate therapy should be viewed with suspicion and undergo biopsy (or re-biopsy) as soon as possible. Oral lichen planus may not be a premalignant condition, but neither does it preclude a patient from developing a second disease, including oral cancer.

## Conclusion

In patients with classic reticular oral lichen planus, the diagnosis can often be made on the basis of clinical features alone. Patients should be advised as to the chronic nature of their disease and its tendency to exhibit periods of activity that alternate with times of relative quiescence or remission. Biopsy confirmation of oral lichen planus should be considered, especially with symptomatic erosive disease, and the use of direct immunofluorescent is strongly recommended to exclude more specific forms of autoimmune disease. Most cases of oral lichen planus can be managed through the use of topical corticosteroids and good oral hygiene measures. While the most current molecular evidence does not suggest oral lichen planus to be a precancerous condition, clinicians are advised to closely monitor their oral lichen planus patients for any intraoral lesion that does not respond to normal therapeutic measures. Regardless of a previous diagnosis of oral lichen planus, tissue biopsy and histopathologic evaluation should always be recommended for any persistent or progressive area of mucosal abnormality. ■■■■

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