



# The clinical manifestations and treatment of oral lichen planus

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Lichen planus is a relatively common mucocutaneous disorder with a reported incidence equal to or greater than such diseases as psoriasis [1,2]. The exact incidence of the disease is unknown, and the determination of the number of patients affected with the disease is complicated by the fact that lichen planus has many clinical forms and may involve one or more sites. For example, lichen planus may affect the skin alone; the oral cavity alone; both the skin and oral cavity simultaneously; or one or more mucosal and nonmucosal sites, with or without skin or oral involvement.

## Extraoral manifestations

Whereas over a third of patients who present to a dermatologist with cutaneous lichen planus are noted to have oral lesions [3], only 15% of patients with predominantly oral lichen planus (OLP) develop cutaneous lesions [4]. In most instances, cutaneous lichen planus typically develops within several months after the appearance of the oral lesions. Furthermore, the severity of the oral manifestations usually does not correlate with the extent of cutaneous involvement.

Lichen planus may also involve the scalp, nails, esophagus, eyes, and genital mucosa. Often, patients have multiple sites of involvement, with over 5% developing the disease in three or more sites simultaneously [4].

Lichen planopilaris was detected in only 6 of 584 OLP patients, with the development of scalp lesions

preceding the onset of oral lesions by 1 to 3 years in five of the six women [4]. Perifollicular erythema and follicular spines accompanied by patchy alopecia are the most common findings, and histologic and often direct immunofluorescent studies are required for confirmation.

Lichen planus of the nails develops in approximately 10% of patients with cutaneous disease; however, the development of lichen planus of the nails is an infrequent finding in patients with OLP. The most common clinical manifestations include thinning, ridging, and distal splitting of the nail plate, changes that typically precede the development of oral lesions.

The clinical features of esophageal lichen planus have been well documented, and the disease seems to develop most commonly in patients with OLP [5]. Most patients with esophageal lichen planus are diagnosed as a result of symptoms brought to the attention of the gastroenterologist, with dysphagia being the predominant complaint. When patients with OLP are screened for esophageal involvement, however, the disease is detected in nearly a third of patients with asymptomatic white reticular lesions being the predominant form [6]. In all probability, esophageal lichen planus, in its erosive form and especially in its asymptomatic form, is undiagnosed in many patients and remains an unrecognized and underreported condition. Although malignant transformation has not been reported, untreated esophageal lichen planus may result in chronic pain and strictures [7]. All patients with OLP should be questioned about dysphagia and evaluated by endoscopy if symptomatic.

Fortunately, conjunctival involvement resulting in cicatrizing conjunctivitis is a rare manifestation of

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lichen planus [8]. The few reported cases have occurred in patients with OLP. Because the institution of prompt treatment has been shown to control inflammation and halt cicatrization, evaluation of conjunctival erythema and detection of the disease in its early state is of paramount importance.

Lichen planus may affect the genitalia in men and women. In fact, the most frequent extraoral site of involvement in female patients with OLP is the genital mucosa and the clinical findings are described elsewhere in this issue.

### Clinical manifestations

Oral lichen planus develops in women more than twice as often as in men, with a mean age of onset in the sixth decade of life. OLP is uncommon in children but when it develops in this age group, it is frequently erosive and painful. Children with OLP often have concomitant cutaneous disease [9,10], and those of Asian descent may be predisposed to the development of the disease [11].

#### Classification

Oral lichen planus may manifest in one of three clinical forms: (1) reticular; (2) erythematous (atrophic); and (3) erosive (ulcerated or bullous). Whereas reticular lesions occur as isolated lesions and are often the only clinical manifestation of the disease, erythematous lesions are accompanied by reticular lesions and erosive lesions are accompanied by reticular and erythematous lesions in almost all cases. This feature helps clinically differentiate OLP from other vesiculoerosive diseases, such as pemphigus and pemphigoid, which are characterized by isolated areas of erythema or erosions.



Fig. 1. Reticular lesions on the posterior buccal mucosa are characteristic of oral lichen planus (OLP).



Fig. 2. Although reticular lesions are usually asymptomatic, extensive involvement on the tongue may result in burning and dysgeusia.

Reticular lesions may be papular, plaque-like, and lacey and are the most recognized form of OLP (Figs. 1 and 2). Multiple patterns of reticular lesions in a patient are commonly noted and the degree of involvement is variable with some patients exhibiting subtle disease and others displaying diffuse and confluent lesions involving multiple sites. Reticular lesions are asymptomatic unless they develop on the dorsal and lateral borders of the tongue where they cause burning and occasional taste disturbances.

Erythematous and erosive (Fig. 3) OLP lesions result in varying degrees of discomfort. In addition to pain, the single most frequent complaint, patients also describe burning, swelling, irritation, and bleeding with tooth brushing.

#### Sites of involvement

The posterior buccal mucosa is the most frequent site of involvement followed by the tongue, gingiva,

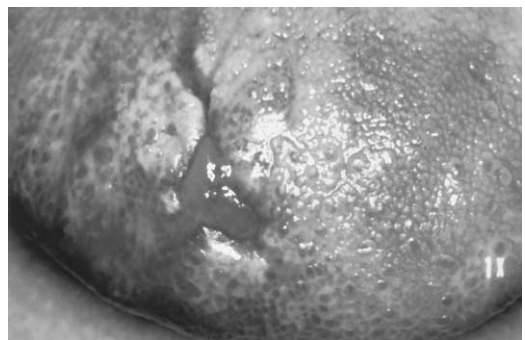


Fig. 3. Erosive lesions of lichen planus on the dorsal tongue accompanied by reticular and erythematous lesions.



Fig. 4. White reticular OLP lesions on the gingiva resembling leukoplakia.

labial mucosa, and vermillion of the lower lip. Lesions on the palate, floor of the mouth, and upper lip are uncommonly noted. Although OLP has a characteristic distribution that is bilateral and symmetric, the disease may also present a confusing array of patterns and forms clinically mimicking other disorders.

Approximately 10% of patients with OLP have the disease confined to the gingiva [12]. Gingival lichen planus presenting as small, raised white, lacy papules or plaques, may resemble keratotic diseases, such as leukoplakia (Fig. 4). Erythematous lesions affecting the gingiva result in desquamative gingivitis, the most common type of gingival lichen planus (Fig. 5). Erosive lesions resembling those observed in other vesiculoerosive diseases including pemphigoid, pemphigus, and linear IgA disease also produce desquamative gingivitis not easily identified as lichen planus unless there are coexistent reticular lesions on the gingiva or elsewhere in the oral cavity.

Lichen planus isolated to a single oral site other than the gingiva is an infrequent occurrence. Patients with isolated lip lesions [13] and tongue lesions [14] have been described although many patients who present with isolated lesions eventually develop more widespread disease. The profile of patients with OLP is as follows:

- Mean age, fifth to sixth decades of life
- Female to male ratio 2:1
- Intraoral involvement: posterior buccal mucosa > tongue > gingiva
- Two third symptomatic, one third asymptomatic
- Increased levels of anxiety and depression
- 20% of women display genital involvement
- Cutaneous involvement in 15% of patients with OLP
- Increased risk for malignant transformation

### *Precipitating factors*

Koebner's phenomenon characteristic of cutaneous lichen planus, whereby lesions develop in response to trauma, is also observed in the oral cavity. Mechanical trauma from dental procedures, heat and irritation from tobacco products, friction from sharp cusps, rough dental restorations and poorly fitting dental prostheses, and oral habits including lip and cheek chewing are exacerbating factors. Koebner's phenomenon may explain why erosive lesions develop most commonly in areas subjected to trauma, such as the buccal mucosa and lateral surfaces of the tongue. When such factors are minimized or eliminated, oral lesions either revert to the less severe forms of the disease or sometimes resolve completely.

Dental plaque and calculus can also result in worsening gingival lichen planus and are associated with a significantly higher incidence of erythematous and erosive gingival lesions [15]. Gingival OLP can ultimately result in gingival recession, advanced periodontal disease, and rarely in tooth loss. Periodontal surgical procedures, which are required to correct these defects, may themselves exacerbate OLP [16].

Patients with OLP exhibit higher levels of anxiety, greater depression, and increased vulnerability to psychic disorders [17,18]. Those with erosive lichen planus exhibit higher depression scores than patients with nonerosive lichen planus. An association with depression, however, may be anecdotal because others have largely refuted this [19]. Stress is identified as the most frequent cause of acute exacerbations of the disease and is widely held to be an important etiologic factor.



Fig. 5. OLP presenting as a desquamative gingivitis. In addition to histology, immunofluorescence of perilesional mucosa is an effective technique that can exclude other causes.

## Diagnosis

### *Histologic features*

The characteristic clinical features may be sufficient to diagnosis OLP correctly, especially if there are easily recognizable skin lesions. Because OLP is a chronic disorder often requiring long-term treatment and surveillance, biopsy is mandatory when the disease does not present with classic features. This is despite the fact that histopathologic assessment of OLP is a rather subjective and insufficiently reproducible process [20]. In addition to the superficial band-like infiltrate of lymphocytes and basal cell liquefaction degeneration, the histopathologic features diagnostic of lichen planus include focal hyperparakeratosis, irregular acanthosis, and an eosinophilic amorphous band at the basement membrane (Fig. 6) [2].

Gingival lichen planus may be more difficult to diagnose because the clinical appearance of lichen planus shares many clinical features with the vesiculoerosive diseases. Direct immunofluorescence of perilesional mucosa is an effective and accurate diagnostic technique, especially useful in excluding other causes [21]. Immunofluorescence reveals fibrin and shaggy fibrinogen in a linear pattern at the basement membrane zone (Fig. 7). Cytoids in the absence of deposition of fibrinogen are commonly detected in immunofluorescence biopsy specimens [22]. The value of direct immunofluorescence for confirmation of the disease is well

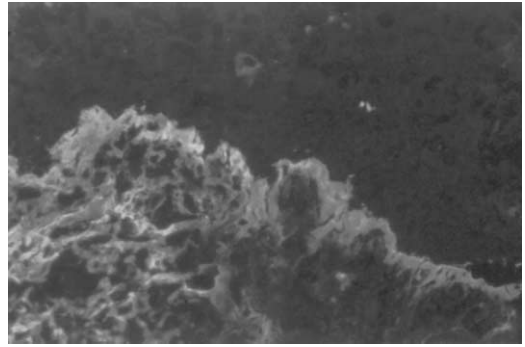


Fig. 7. Direct immunofluorescence of OLP reveals fibrin and shaggy fibrinogen in a linear pattern at the basement membrane zone. (Courtesy of Dr. James J. Sciubba, Baltimore, MD.)

accepted, especially with nondiagnostic histopathologic features and for the desquamative gingivitis form of lichen planus.

### Oral lichenoid eruptions

Although uncommon, OLP may be caused by a hypersensitivity reaction to dental restorations. An allergy or reaction to a dental filling material should be suspected when OLP lesions are confined to areas of the oral mucosa in close contact with or proximity to the filling. Amalgam restorations, especially those that are old and cracked, have been implicated most frequently, although reactions to composite and cop-

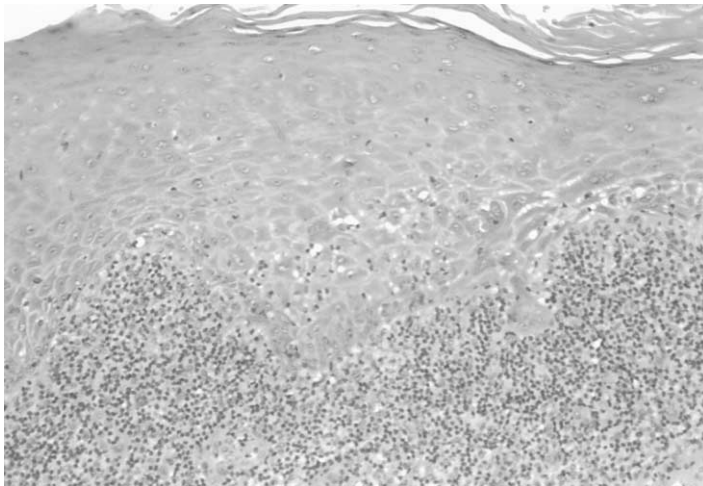


Fig. 6. Biopsy of OLP revealing superficial band-like infiltrate of lymphocytes, basal cell liquefaction degeneration, focal hyperparakeratosis, irregular acanthosis, and an eosinophilic amorphous band at the basement membrane. Hematoxylin and eosin 100X (H and E). (Courtesy of Dr. James J. Sciubba, Baltimore, MD.)

per dental materials can also develop [23,24]. Oral lesions resulting from hypersensitivity reactions to dental materials clinically and histologically resemble lichen planus closely, but have an identifiable etiology. A positive patch test reaction to more than one mercurial allergen may increase the likelihood of the correct diagnosis and may justify the removal and replacement of all amalgam fillings with those made of other materials [25]. When identified, some lichenoid lesions can show considerable improvement or complete regression after replacement with another filling material [26].

Equally uncommon are drug-induced oral lichenoid reactions. The most commonly implicated drugs are the nonsteroidal anti-inflammatory agents and the angiotensin-converting enzyme inhibitors [27,28]. Although numerous other drugs have been linked with oral lichenoid reactions, the reports have been based on a single case or poor documentation. Because the most reliable method to diagnose lichenoid reactions is impractical, namely to monitor if the reaction remits with drug withdrawal and returns on rechallenge, drug-induced oral lichenoid reactions will remain either undiagnosed or overdiagnosed.

### **Systemic associations**

After the first report by Reborá [29] linking erosive OLP with severe liver disease, a number of studies have been published suggesting this possible relationship. For example, diseases affecting the liver including Wilson's disease, hemochromatosis,  $\alpha_1$ -antitrypsin deficiency, and primary biliary cirrhosis have uncommonly been related to lichen planus [3,30]. In recent years, the attention has focused on the relationship between OLP and hepatitis. Several controlled studies have demonstrated a strong association between chronic hepatic disease or hepatitis C virus (HCV) infection and OLP [31–33]. A small but significant percentage of US patients with cutaneous lichen planus have also been shown to be HCV antibody positive [34]. Reported prevalence rates of HCV infection in OLP patients range from 20% in Spain to a high of 62% in Japan. In support of these studies, investigators have found HCV viral sequences in the serum of patients with OLP and in oral tissue samples [35,36]. Furthermore, OLP patients with HCV infection neither have evidence of autoimmune liver damage nor abnormal humoral immune responses [37]. Elevated liver function tests have also been detected most often in patients with oral erosive disease and seem to correlate with the severity of OLP [38].

The association of OLP with both HCV infection and liver disease, however, may be dependent on geographic factors. Research studies on OLP patients in the United States, Britain, France, Scandinavia, and Germany failed to confirm an association between lichen planus and liver abnormalities [39]. In a US study consisting of 195 patients with OLP, none had had detectable antibodies to HCV or abnormal liver function tests [40]. OLP may be an early marker or manifestation of hepatitis in select populations; however, routine serologic screening in Western European and American OLP patients may not be warranted.

The incidence of other systemic diseases in OLP patients including hypertension, arthritis, and diabetes is not higher than expected when compared with the incidence reported in the general population.

### **Malignant potential**

The development of squamous cell carcinoma is a feared complication of OLP with a reported frequency of 0.4% to over 5% over periods of observation from 0.5 to over 20 years [41]. Most carcinomas at presentation are reported in areas of atrophic or erosive OLP. Well-known risk factors for oral cancer, including tobacco consumption and heavy alcohol use, do not seem to be greater in OLP patients compared with the general population. Careful monitoring of patients with OLP has been demonstrated to result in the detection of carcinomas in the early stages (in situ and microinvasive) with favorable prognoses [42].

There is considerable controversy regarding the malignant transformation of OLP. Although more than 25 follow-up studies have focused on this topic, as recently reviewed by Barnard et al [43], several investigators have questioned the criteria used for diagnosing OLP in published reports [44,45]. For example, whereas some studies included patients diagnosed with OLP based on clinical and histologic criteria, others included patients that were based solely on clinical features [46]. Consequently, many published cases of OLP associated with malignant transformation diagnosed clinically as OLP may actually have been lichenoid dysplasia, a premalignant condition with lichenoid features. Patients with lichenoid dysplasia often display erythematous and erosive lesions clinically identical to OLP lesions [47]. The lack of reliable, well-defined, objective clinical criteria of epithelial dysplasia emphasizes the need to confirm by biopsy the diagnosis of patients with suspected OLP. Exceptions include

patients with classic bilateral, white reticulated lesions on the buccal mucosa and those with concomitant, easily recognizable skin lesions. Given the uncertainty of the premalignant nature of OLP and the fact that early detection of oral cancer results in improved survival, it is prudent to monitor patients with OLP carefully and long term.

## Treatment

Because no therapy for OLP is curative, the primary goal for symptomatic patients is palliation. It is tempting to speculate that aggressive treatment of patients with erosive and atrophic OLP lowers or eliminates the risk of malignant transformation; however, this hypothesis has never been substantiated or investigated. Historically, most OLP patients in large published studies have been treated with topical agents alone, which do not seem to alter the chronic and often lifelong course of this disease.

Patients with OLP who display only white, reticular lesions usually do not require treatment. This form of the disease is asymptomatic and discovered incidentally during a routine dental visit. An exception may be the burning and taste alteration that can result from reticulated lesions on the dorsal and lateral tongue, which resolve when the lesions are treated. Patients with erosive and erythematous OLP often present significant management problems. The need to reduce the morbidity associated with these forms of the disease has triggered a continuing search for novel therapies, many of which have been reported. An approach to the diagnosis and treatment of OLP is suggested in Fig. 8.

## Topical treatment

### *Corticosteroids*

Topical corticosteroids remain the mainstay of treatment of OLP. A response to treatment with midpotency corticosteroids, such as triamcinolone, potent fluorinated corticosteroids, such as flucinonide, and superpotent halogenated corticosteroids, such as clobetasol, has been reported in 30% to 75% of treated patients [48–51]. Higher-potency preparations seem to be more effective than lower-potency preparations, although there have been few studies that have compared various strength corticosteroid preparations for OLP. The great variation in efficacy of topical corticosteroids is a reflection of the heterogeneity of published reports regarding both the

experimental design of the trials and the criteria of response to therapy and differences in clinical severity of the treated lesions.

Therapy with topical corticosteroids should be initiated with a potent preparation to achieve a rapid clinical response. Because potent corticosteroids may delay wound healing, it is advisable to lower the strength of the preparation as soon as erosions heal and erythematous lesions become asymptomatic. Once the disease is controlled as defined by the absence of lesions or the presence of only white reticular lesions, therapy temporarily may be discontinued.

Patients should be cautioned about the off-label use of topical corticosteroids and the accompanying package inserts, which state for “external use only.” Although a number of studies have demonstrated the safety of topical corticosteroids when applied to mucous membranes for short intervals [52,53], the potential for adrenal suppression with prolonged use, especially for a disease that is chronic, necessitates careful and frequent follow-up examinations. Atrophy in the oral mucosa is rarely observed. As many as a third of OLP patients treated with topical corticosteroids, however, develop secondary candidiasis [54]. Because there is an increased prevalence of candidal carriage and infection among patients with OLP [55,56], antifungal therapy administered either topically or systemically is often helpful as an adjunctive therapy.

For intractable erosive OLP lesions, intralesional triamcinolone acetonide (10 to 20 mg/mL) injections can be highly effective. As with alopecia areata, treatments are repeated every 4 weeks until results are achieved. The administration of corticosteroids intralesionally is especially beneficial for lesions on the lateral border of the tongue and buccal mucosa.

### *Cyclosporine*

The topical use of cyclosporine may be used as an adjunct to topical corticosteroids [57]. The standard solution (100 mg/mL) intended for systemic use in organ transplant recipients may be used as a mouthwash for patients with OLP. Topical cyclosporine is prohibitively expensive for routine use and should be reserved for patients who are symptomatic and who fail topical corticosteroids. Patients can either swish a small amount of undiluted solution for approximately 5 to 10 minutes and then expectorate it, or hold a cotton ball saturated with solution against their lesion. Topical corticosteroids may be used immediately after using topical cyclosporine, and although controlled

## Evaluating and Treating OLP

### Diagnosis

Medical history, drug history, clinical examination, biopsy +/- immunofluorescence



### Minimizing External Factors

Tobacco and alcohol

Poor oral hygiene

Oral habits: cheek biting, tongue thrusting

Sharp cusps, fractured restorations, poorly fitting prostheses

### Medical Treatment

#### Mild disease

Observation only

Topical corticosteroids

Intralesional corticosteroids

#### Moderate disease

Topical corticosteroids

Topical cyclosporine

Tacrolimus

Hydroxychlorquine

#### Severe Disease

Topical agents

Acitretin

Azathioprine

Mycophenolate

Cyclosporine

Prednisone for acute exacerbations

Antifungals for secondary candidiasis

Combination therapy – topicals and/or systemics – most effective

Fig. 8. Evaluation and treatment of OLP.

studies are lacking, the combination of the two agents seems to be more beneficial than the use of either agent alone.

Not all investigators have found benefit from using topical cyclosporine in OLP [58,59]. As with other treatments reported to be beneficial in OLP, reports consist of small and uncontrolled trials using various doses and methods of application making comparisons difficult. For example, cyclosporine mouthwash, in doses of 500 mg three times daily for 8 weeks, resulted in improvement in all patients with atrophic and erosive OLP [60]. In a subsequent study of 13 patients with OLP, however, randomly assigned to treatment with cyclosporine (500 mg as a swish-and-spit medication for 5 minutes three times daily) or a triamci-

nolone acetonide oral paste over 6 weeks, only slight, transient clinical improvement was noted in both groups [59].

A significant reduction of pain and increased rate of healing compared with placebo was demonstrated in a controlled study of 14 patients with erosive OLP who were treated with low doses of topical cyclosporine (500 mg/d) [61], and lower cost of drug. Beneficial effects have also been claimed by using cyclosporine in bioadhesive pastes.

Systemic absorption is low with topical cyclosporine and the efficacy of the drug does not correlate with cyclosporine blood levels [62]. In patients who respond to cyclosporine, results are typically observed after 4 to 8 weeks of treatment.

### *Tacrolimus*

Tacrolimus, a new topical immunosuppressive agent approved for the treatment of atopic dermatitis, is 10 to 100 times as potent as cyclosporine. Several publications have already documented the efficacy of this agent in erosive OLP [63], although all used a specifically formulated oral cavity preparation and not the commercial preparation intended for skin. Additionally, all studies were purely anecdotal and open labeled. When commercial tacrolimus is used, a significant number of patients often complain of burning at the site of application and a smaller number note worsening of the condition. Additional studies are warranted to confirm the efficacy of tacrolimus in OLP.

## **Systemic treatment**

### *Corticosteroids*

Systemic corticosteroids are widely used in OLP and seem to be the most effective short-term treatment modality to control the disease. There have been no controlled studies, however, to evaluate their true efficacy. Furthermore, the prolonged administration of systemic corticosteroids for a chronic and often lifelong disease is unjustified because of their inherent toxicity [64]. Systemic corticosteroids should be reserved for acute exacerbations or for recalcitrant, severe erosive or erythematous lesions where topical approaches have failed. The disease often relapses when corticosteroids are discontinued and the requirement frequently to reinstitute therapy indicates the need for a corticosteroid-sparing agent. When using prednisone, a starting dose of 30 to 80 mg may be administered once daily, tapering the dose over a 2- to 3-week period. Secondary oral candidiasis may complicate therapy, especially when topical corticosteroids are used concomitantly.

### *Immunosuppressive therapies*

Systemic immunosuppressive agents have been used for the treatment of OLP patients with severe disease, unresponsive to topical agents. Although none produces long-term remission when discontinued, significant clinical benefits are achieved and maintained with long-term use. All immunosuppressive agents require administration by health care providers familiar with their adverse reactions. Monitoring for laboratory abnormalities is essential and even so, the drugs can cause undesirable adverse

effects. Topical therapy administered concomitantly is desirable and this strategy often results in a reduction of the dose of the immunosuppressive agent that is needed to achieve clinical improvement.

The aromatic retinoids have been studied in patients with erosive OLP. Documented improvement in several studies was noted in most patients undergoing treatment with etretinate, in doses ranging from 0.6 to 1 mg/kg/d [65,66]. As expected, adverse reactions, including cheilitis and dry skin, were commonly observed necessitating discontinuation of treatment in a significant number of patients. By reducing the maintenance dose to 0.3 mg/kg/d, adverse reactions were minimized but the clinical benefits were not nearly as good as those achieved with higher doses [67]. Laurberg et al [68] has replaced etretinate with acitretin, and at daily doses of 30 mg, resulted in remission or marked improvement in 64% of patients compared with 13% receiving placebo. As with etretinate, complications are frequently observed [69]. Although most dermatologists are far more familiar with the retinoid isotretinoin, its use in OLP only produces modest benefits and it is difficult to justify its use [70].

In summary, the use of systemic retinoids results in significant improvement; however, given their adverse effects and the wide range of alternatives, their use should be reserved for patients who fail other therapies.

Hydroxychloroquine at daily doses of 200 to 400 mg can be administered long term to patients with erosive disease with few adverse effects [71]. Treatment is not always effective and improvement often takes 4 to 6 months. In another anecdotal study of 30 patients with cutaneous lichen planus, four of whom had oral lesions, two with OLP had complete healing of their lesions with phenytoin [72]. Thalidomide has also been anecdotally reported to benefit patients with erosive lichen planus unresponsive to conventional therapies [73,74]. Additional studies are warranted to confirm the efficacy of these agents and furthermore, hydroxychloroquine, phenytoin, and thalidomide have all been reported to induce oral lichenoid reactions.

Azathioprine has been reported to be an effective corticosteroid-sparing treatment for cutaneous lichen planus [75,76], and its use for OLP is advocated in the dental literature as a treatment for brief 2-week periods [77]. Azathioprine does seem to be an alternative beneficial therapy for OLP, especially when there are risk factors against corticosteroid use. An initial dose of 50 mg/d may be instituted and the dose advanced to 100 to 150 mg/d if the baseline laboratory parameters remain unchanged. The response to

treatment is slow, and as many as 3 to 6 months may be needed to observe the maximum benefits. As with other diseases treated with azathioprine, no improvement should be expected after 2 weeks of therapy.

Mycophenolate mofetil used after organ transplantation seems to be a promising new immunosuppressive drug used off-label for a variety of dermatologic diseases [78]. The author's own experience indicates that at doses of 2 to 3 g/d, it is both well tolerated and effective in OLP patients unresponsive to topical agents or other immunosuppressive drugs. As with azathioprine, mycophenolate results in improvement that is achieved over a course of many months. Treatment is expensive and at doses used to control the disease averages \$600 to \$800 per month. Gastrointestinal adverse reactions sometimes require discontinuing the drug.

The systemic administration of cyclosporine in doses much less than those used to treat psoriasis (1 to 2 mg/kg/d versus 4 to 6 mg/kg/d) has been reported to be beneficial for the treatment of both cutaneous and oral lichen planus [79,80]. Systemic use of cyclosporine, however, should be reserved for severe and refractory cases because of the many potential adverse effects of this drug. Fortunately, clinical results are noted rapidly with cyclosporine, even at low doses. Once the disease is controlled, the drug should be replaced with an agent that is safer to administer long term.

## Summary

The progress in research on OLP and other autoimmune diseases has been significant. Coupled with a growing recognition of the clinical features and treatment options by dentists and physicians and fueled by the advances in immunosuppressive therapies, research will undoubtedly provide new insights into this complex disorder. It is likely that what is learned will enhance the understanding not only of OLP but also of many other mucocutaneous diseases.

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