
The British Society for Oral Medicine - Guidelines for the Management of Oral Lichen Planus In Secondary Care



Preamble

Oral lichen planus is a relatively common disorder, affecting 1-2% of the population. The disease ranges in severity from an asymptomatic condition to one of severe discomfort that may adversely impact on patient quality of life.

Lichen planus, typically arises in females of middle age but also affects males. There are as yet no ethnic groups identified as being of particular risk, the condition arising in all ethnicities¹.

Disclaimer

These guidelines have been prepared for use by clinicians specialising in the practice of Oral Medicine. They represent current knowledge and opinion at the time of their preparation, and their recommendations are clearly subject to change with emerging data, evidence for the efficacy of treatment and the development of novel therapies. It is acknowledged that the evidence base for the treatment regimes is to date limited in terms of rigorously conducted randomised clinical trials. Although the guidelines aim to highlight current knowledge and practice, they should not be regarded as being prescriptive. There will be occasions where due to individual circumstances, deviation from these guidelines may well be appropriate and as such should not be regarded as evidence of inadequate patient management. Equally, adherence to the guidelines may not always be evidence of best practice in individual circumstances.

Definition

Oral lichen planus is a common chronic inflammatory mucocutaneous disorder that typically affects the oral mucosa and additionally, in some cases the skin. Lichen planus can affect other non-oral mucosal surfaces such as the genitals, anus and pharynx. Conjunctival and oesophageal involvement may rarely occur.

Clinical presentations

Oral lichen planus has a characteristically bilateral distribution, typically involving the buccal mucosa, dorsum and ventral surfaces of the tongue and/or gingiva, when it often presents as desquamative gingivitis. Palatal and labial involvement is unusual.

Oral lichen planus is often asymptomatic, although when there are areas of ulceration, the patient experiences varying degrees of discomfort, exacerbated by eating spicy or acidic foods.

The variable clinical presentations of oral lichen planus comprise white patches, ulcers and, very rarely, blisters².

Reticular oral lichen planus – this is the most common presentation, manifesting as a lacy network of white striations. These lesions are often painless, although patients may complain of a slight roughness or dryness of the affected sites.

Papular oral lichen planus – this manifests as small white raised areas approximately 1-2mm in diameter. These again typically arise on the buccal mucosa and dorsum of tongue, although may also present on other mucosal surfaces. This variant may represent an early manifestation of the condition.

Plaque-like oral lichen planus – this manifests as areas of homogenous whiteness. This typically arises on the buccal mucosa or dorsum of tongue and may be more prevalent amongst those who are smokers.

Atrophic oral lichen planus – here areas of mucosal atrophy occur within the white patches. The clinical picture is one of red and white areas, but not speckled as is seen in chronic hyperplastic candidosis. Patients with this type of disease often complain of oral soreness.

Ulcerative lichen planus – frank ulcers exist often within the hyperkeratotic areas. On occasions there may be no white striae apparent, making clinical diagnosis difficult. Patients complain of soreness, particularly with spicy or acidic foods.

Bullous lichen planus – this rare presentation manifests as small vesicles or blisters (bullae) within the white patches. This may cause diagnostic confusion with mucous membrane pemphigoid or pemphigus

Patients with disease involving the gingiva may have areas of white patches or striae superimposed on a red base. Desquamative gingivitis is also a common presentation.

There is little predictability as regards the frequency of extraoral disease in patients with oral lichen planus. Likewise the oral features may precede, accompany or follow lichen planus affecting other sites.

Aetiopathogenesis

The aetiology of oral lichen planus remains unclear. It is believed that oral lichen planus represents a cell-mediated immunological reaction within the affected tissues, the precise trigger for this being unknown³.

A minority of patients may have disease that closely mimics lichen planus, both clinically and histologically⁴, and are described as ‘lichenoid lesions’. Examples include lichenoid drug reactions [eg anti-hypertensive agents including beta blockers, thiazide diuretics, angiotensin converting enzyme inhibitors⁵ and calcium channel blockers, sulphonylureas, anti-malarials, gold, penicillamine, allopurinol⁶ and non-steroidal anti-inflammatory agents⁷], lichenoid reactions seen in close proximity to amalgam restorations⁸ [and other metallic and also non-metallic dental restorations] and chronic graft versus host disease (GvHD)^{9, 10}

These guidelines will not differentiate between idiopathic lichen planus and lichenoid reactions from the point of view of clinical presentation.

Lichen planus-like disease can arise in patients with autoimmune liver disease including primary biliary cirrhosis¹¹ and chronic active hepatitis¹². There are reported associations between oral lichen planus and hepatitis C virus infection which appear to occur in individuals living in areas of southern Europe, particularly Portugal, Spain and Italy¹³. Such an association has also been observed in Japan. These observations may relate to the carriage rates of Hepatitis C in these parts of the world, rather than necessarily being of aetiological significance. Additionally there is the possibility that at least in Italian patients, HLA-DR6 may be linked to the reported association of oral lichen planus with hepatitis C¹⁴. At present there is no evidence that patients, at least in Northern Europe, require routine screening for possible hepatitis C virus infection or other hepatic disease.

The large majority of patients, however, with oral lichen planus do not have disease that is either due to drug therapy or dental restorations.

Diagnosis

The diagnosis of oral lichen planus is initially based upon the clinical presentation of bilateral white patches with or without ulcers or blisters, typically affecting the buccal mucosa, ventral, lateral and dorsal surfaces of the tongue and gingiva.

Consideration should be given to extra-oral sites of involvement

- Skin
- Nails
- Scalp
- Other mucosal sites such as the genitalia.

Comorbidity: autoimmune liver disease, hepatitis C, chronic GvHD, and possible ulcerative colitis should also be considered during the patient interview. It is not usual to routinely investigate patients for underlying disease.

Biopsy and histopathological examination of affected tissue may be needed to exclude other disease that may mimic oral lichen planus – eg discoid lupus erythematosus and to identify possible epithelial dysplasia. The need for biopsy in all cases of suspected lichen planus is debated but it would be appropriate in cases that are atypical in presentation, atrophic or ulcerating¹⁵.

Smears and swabs for mycology may be helpful in some instances as oral lichen planus is not infrequently superinfected with candidosis, particularly when treated with topical corticosteroid therapy¹⁶.

Haematological/immunological investigations are not routinely indicated but should be undertaken where clinically appropriate.

Skin testing for allergy to mercury amalgam may be undertaken where there is a suspicion that there may be a lichenoid reaction in response to this dental material. However there is debate as to the value of such an investigation.^{17, 18}

Differential Diagnosis:

- Drug induced "lichenoid" reactions
- Discoid / Systemic lupus erythematosus
- Non specific ulceration
- Candidosis
- Leukoplakia
- Hairy Leukoplakia
- Mucous Membrane Pemphigoid
- Pemphigus
- White sponge naevus and other genodermatoses
- Cheek biting

General Aspects of Treatment

Patients with reticular lichen planus generally have no painful symptoms associated with their disease, although they may complain of mild oral mucosal roughness, soreness or dryness of the oral mucosa. This generally does not require treatment.

Individuals with ulcerative or bullous disease tend to have oral discomfort, particularly with spicy or acidic foods, and thus require treatment.

The treatment aims of symptomatic oral lichen planus are to heal areas of painful ulceration or blistering. A stepwise approach should be adopted.

Topical corticosteroid therapy is the mainstay of treatment for ulcerative disease. There is limited evidence from randomised controlled trials as to the precise efficacy of the various preparations that are in common usage¹⁹.

As an adjunct to therapy, patients should also be advised of the need to maintain a high standard of oral hygiene and any causes of mucosal trauma such as ill fitting dentures, sharp cusps and poor dental restorations should be eliminated.

Patients should be informed that there is a very small risk of malignancy associated with oral lichen planus and that long term monitoring is appropriate.

Detailed Aspects of Treatment

A useful summary of the various treatment modalities, including the evidence base for the use of topical corticosteroids in particular, can be found in the report of a working party of the World Workshop on Oral Medicine 2007¹⁵.

1. Asymptomatic non ulcerative lichen planus.

Explanation of the nature of the condition, including its aetiology, clinical features, treatment, follow up and prognosis. Advice on avoidance of risk factors [tobacco and alcohol]. Empirical dietary advice regarding intake of fresh fruit and vegetables. Reassurance and referral back to the GDM or other appropriate clinician for monitoring.

2. Symptomatic non ulcerative lichen planus.

Topical anaesthetic and /or barrier agents for symptomatic relief of pain eg: Benzydamine hydrochloride (0.15%) spray or mouthrinse. Alternatively, it may be appropriate for the patient to apply 2% lidocaine gel to painful areas. Some patients may gain benefit from antiseptic mouthwashes such as chlorhexidine gluconate. Following clinical improvement, the patient should be referred back to primary care for monitoring, as above.

If possible obviate any provoking factors e.g. toothpastes, medication, [amalgam restorations].

Short term [6 monthly] specialist review

If symptoms are in excess of clinical signs consider other co-existing disease [eg Burning Mouth Syndrome]

3. Atrophic/ulcerative oral lichen planus

Topical corticosteroid preparations:

Soluble prednisolone tablets, 5mg dissolved in 15ml of water and used as a mouthrinse 3-4 times daily

Betamethasone sodium phosphate (500mcg dissolved in 10-15ml of water) used as a mouthrinse up to 4 times daily.

Fluticasone propionate spray (50mcg per puff), directed to affected areas up to 3-4 times daily

Beclometasone spray (100mcg per puff), sprayed 3-4 times daily on affected sites

Clobetasol ointment (0.05%) applied to painful areas 3-4 times daily

Fluticasone cream (0.05%) applied to painful sites 3-4 times daily

Appropriate topical antifungal preparations if indicated [eg clinical signs of candidosis]

Ensure good oral hygiene

Consider lichenoid reaction to amalgam particularly if localised lesion[s].

Skin patch tests may be requested although the correlation between positive test results and the presence of lichen planus is unclear; empirical amalgam replacement]

Specialist review until condition has shown a stable improvement. Counselling re tobacco/alcohol.

Treatment dosage and duration should be titrated according to patient need.

4. Severe, symptomatic atrophic/ulcerative oral lichen planus, unresponsive to topical measures

Systemic steroids [prednisolone, deflazacort], short reducing dose with maintenance with topical corticosteroids

+ antifungals if appropriate

Long term review and counsel re tobacco/alcohol

5. Recalcitrant oral lichen planus

Low dose, short term corticosteroids [appropriate surveillance for hypertension, diabetes and bone prophylaxis - DEXA scan, calcium +D3 supplements/ bisphosphonates as appropriate²⁰].

Azathioprine [\pm corticosteroids, with prior assessment of TPMT levels and appropriate monitoring]

Topical ciclosporin [100mg/ml] as a mouthwash twice daily [consider checking blood levels].

Topical tacrolimus^{21 22} [Protopic 0.03%; 0.1%; monitoring of blood levels of tacrolimus is no longer considered necessary] Whilst concerns have been expressed regarding the potential of tacrolimus to encourage malignant transformation of the mucosa, the evidence for this is very limited. However, it is prudent to use tacrolimus with caution, for a limited duration, at the minimum dosage commensurate with efficacy. Empirically avoid where there is evidence of epithelial dysplasia.^{23 24}

Long term review and counsel re tobacco/alcohol.

The value of mycophenolate mofetil in managing recalcitrant oral lichen planus is as yet unconfirmed²⁵. More recently there has been interest in the use of methotrexate²⁶ in such cases.

Titrate treatment dosage and duration according to patient needs in all cases, being mindful of treatment complications and contraindications as appropriate

6. Desquamative gingivitis

Intensive oral hygiene programme

Occlusive corticosteroid therapy [fluocinolone acetonide]

Counsel re tobacco/alcohol

Long term review, once condition controlled and stable, to be undertaken within a primary care setting.

Review intervals – There is neither evidence base nor consensus as to the optimum review interval²⁷. An annual review interval of 1 year would seem reasonable for mild forms of oral lichen planus, reducing to 6 monthly for more severe forms and 3 monthly if dysplasia is present on biopsy. The use of ‘open appointments’ is also of value in follow up of lichen planus, with patients being informed of the clinical signs about which to be aware. Where appropriate, and certainly for cases of lichen planus that are well-controlled, monitoring can and should be undertaken within a primary care setting, with re-referral to secondary care as necessary. ‘NICE’ guideline 27 [Referral Guidelines for Suspected Cancer²⁸] clearly states that patients with oral lichen planus should be monitored for oral cancer as part of the routine dental examination.

Extra-oral lesions – liaise with dermatology or other specialty, as appropriate.

Prognosis

Oral lichen planus may typically persist for many years [15-20]. The main long term concern regarding this condition is that of its malignant potential, an issue which remains controversial²⁹. Although various studies have examined the malignant potential of oral lichen planus, most have been retrospective, and some lack clarity regarding the initial diagnosis. In addition patients have developed oral squamous cell carcinoma at sites distant to those affected by lichen planus. However it is

presently suggested that patients with long-standing oral lichen planus may develop a squamous cell carcinoma of the mouth at a rate of 0.5 - 2% over a 5 year period³⁰. Whether this reflects a common cause or that lichen planus does indeed predispose to oral squamous cell carcinoma remains unclear – lichen planus is common, and an association with squamous cell carcinoma could be coincidental.

In view of the controversy regarding the malignant potential of lichen planus, it is recommended that all patients are kept under regular review by their general dental practitioner. Isolated areas of increasing whiteness, speckling (areas of redness and whiteness) or solitary ulceration unlikely to reflect local trauma require further specialist opinion and possible biopsy.

All patients with oral lichen planus should be advised of the controversy regarding the malignant potential of oral lichen planus, and provided with appropriate advice – particularly the avoidance of tobacco and alcohol, and empirically a diet rich in vitamins A, C and E and the maintenance of good oral hygiene with regular dental care.

Prevention

Currently, as the cause of lichen planus remains unknown, there are no specific preventative regimes for this disorder. However, regular clinical review is deemed prudent in view of the controversy regarding the malignant potential of this condition.

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