

## A review on oral lichen planus

M E Islam<sup>1</sup>, D Jahan<sup>2</sup>

### Abstract

*Oral lichen planus (OLP) is a chronic inflammatory disease of unknown etiology. It is a T-cell mediated autoimmune disease in which the cytotoxic CD8+ cells trigger apoptosis of the basal cells of the oral epithelium. It can present in a number of forms, commonly reticular, plaque-like, erosive and atrophic forms. The diagnosis of OLP can be made from the clinical features if they are sufficiently characteristic, particularly if typical oral and skin lesions are present, but biopsy is recommended to confirm the diagnosis and to exclude dysplasia and malignancy. There is a controversy in misdiagnosis of OLP with lichenoid reactions and lichenoid dysplasias. Corticosteroids in various forms remain the mainstay of treatment but newer agents and techniques are having an increasing role. Patient education may improve the outcomes of OLP therapy and further reduce the risk of oral cancer in OLP patients. In this paper we review the pathogenesis of OLP, clinical and histological features of OLP, process of OLP diagnosis and management of OLP patients.*

**Key words:** Oral lichen planus, autoimmune disease, lichenoid reaction, corticosteroids

### Introduction

Lichen planus is a chronic inflammatory disease of unknown etiology that affects the skin and the mucous membrane.<sup>1, 2</sup> Oral lichen planus (OLP) is more common than the counterpart of skin lesions.<sup>3</sup> It pursues a chronic course, being resistant to therapy, with a low order of resolution.<sup>4</sup> It was first described by Wilson in 1869.<sup>5</sup> Andreasen, in 1968, pointed out that patients with lichen planus were found to be in conditions of stress, anxiety, and emotional changes.<sup>6</sup> In 1978, Krutchkoff et al critically reviewed 223 previously published cases of malignant transformation in OLP.<sup>7</sup> The importance of this disease is related to its frequency of occurrence, its occasional similarity to other mucosal diseases, its occasionally painful and persistent nature and its possible relationship to oral squamous cell carcinoma.<sup>8</sup>

### Epidemiology

It is thought to affect 0.1-2% of the general population.<sup>5, 8, 9, 10</sup> The prevalence rates may differ among races and geographic areas.<sup>10</sup> Oral lichen planus occurs predominantly in adults over 40 years, although young adults and children may be affected.<sup>1, 11, 12</sup> It affects women more than men in a ratio of 1.4:1.1, 3, 13 The condition can affect either the skin or mucosa or both. About half of the patients with skin lesions have oral lesions, whereas about 25% present with oral lesions alone.<sup>14</sup>

### Etiology and Pathogenesis

Although the cause of lichen planus is unknown, it is generally considered to be an immunologically mediated process that microscopically resembles a hypersensitivity reaction. The role of autoimmunity in the pathogenesis is supported by many autoimmune features of oral lichen planus, including its chronicity, onset in adults, predilection for females and association with other autoimmune diseases.<sup>13</sup> In a majority of patients, possible initiators include dental materials (amalgam), emotional stress, drugs (antihypertensives, NSAIDs) and infectious agents.<sup>8</sup> If a specific causative agent is identified, the condition may be referred to as a lichenoid hypersensitivity reaction or lichenoid drug reaction. These lesions appear clinically identical to OLP at the site of contact with the offending agent and generally resolve following removal of the causative agent.<sup>11</sup>

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1. Dr Mohammad Emranul Islam, BDS (DU), MS (OMS), Assistant Professor and Head, Department of Oral and Maxillofacial Surgery, City Dental College

2. Dr Dilshad Jahan, Senior Lecturer, Department of Community Dentistry, Faculty of Public Health, Bangladesh University of Health Sciences

### Address of Correspondence:

Dr Mohammad Emranul Islam BDS, MS (OMS), Assistant Professor and Head, Department of Oral and Maxillofacial Surgery, City Dental College  
Email: milubd@gmail.com

The postulated pathogenic mechanism for OLP is that native proteins are falsely identified as foreign antigens. This may result from basal cells (keratinocytes) expressing surface proteins that bear a close structural resemblance to a foreign protein, thus stimulating a delayed cell mediated hyper immune response. It may also result from the exposure of basal cell proteins to lymphocytes or macrophages which can then process them as antigens.<sup>15</sup> Then the T-cells (mostly CD8+ as destroyer and CD4+ as helper cells) migrate into the epithelium either due to random encounter of antigen during routine surveillance or a chemokine mediated migration toward basal keratinocytes. The CD8+ cells are activated directly by antigen binding to major histocompatibility complex MHC-I on keratinocyte or through activated CD4+ cells. In addition, the number of Langerhan cells in OLP lesions is increased along with up regulation of MHC-II expression. Subsequent antigen presentation to CD4+ cells and interleukin-12 activates CD4+ helper T-cells which activate CD8+ T-cells. The activated CD8+ cells in turn kill the basal keratinocytes through tumour necrosis factor (TNF- $\alpha$ ) by activating apoptosis.<sup>3, 5, 8, 14, 16</sup>

### Clinical presentation

The patients may complain of symptoms, which are highly variable, but often consist of sensitivity to toothpaste, acidic substances, alcohol, carbonated beverages, hot, spicy or salty food and abrasive foods. The majority of the patients are otherwise typically symptom free.<sup>11</sup> Some patients report a roughness of the lining of the mouth, dryness of mouth, painful oral mucosa, sore gums, red or white patches on the oral mucosa, red gums, or oral ulcerations.<sup>13</sup> Descriptions of its classic clinical features evoke a familiar constellation of lacy, white, keratotic papules and striae on an erythematous background, distributed bilaterally on buccal mucosa, gingiva, tongue, the lips, and other oral locations.<sup>7</sup>

The most common type of oral lichen planus is the reticular form. Characteristically, it presents as a series of fine, radiant, white striae known as 'Wickham striae', which may be surrounded by a discrete erythematous border. The buccal mucosa is the site most commonly involved. The striae are typically bilateral in a symmetrical form on the buccal mucosa. They may also be seen on the lateral border of the tongue and less often on the gingiva and the lips. Reticular lichen planus is likely to resolve in 41% of cases.<sup>14</sup>

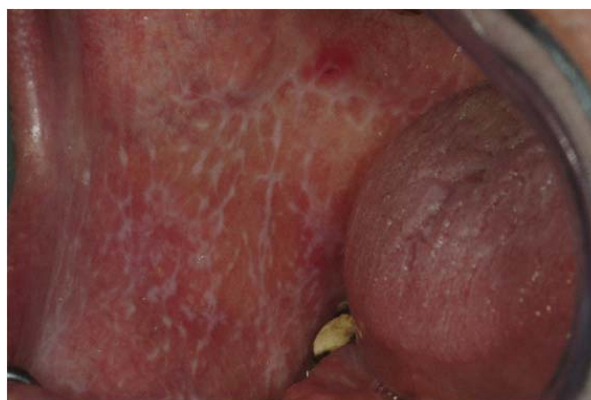


Fig 1: Reticular lichen planus

Plaque-like lesions resemble leukoplakia and occur as homogenous white patches ranging from a slightly elevated and smooth to a slightly irregular form and may be multifocal. The primary sites for this type are over the dorsum of the tongue and the buccal mucosa. Plaque-like oral lichen planus resolves in only 7% of cases. This form is significantly more common among tobacco smokers.<sup>14</sup>



Fig 2: Plaque like lichen planus

The atrophic or erythematous type of oral lichen planus is diffuse, red and can cause a burning sensation. White striae around the lesion that radiate peripherally are usually evident at the margins of the atrophic zones of the lesion. The attached gingiva is often involved and the condition is commonly referred to as 'chronic desquamative gingivitis'. The atrophic form can display a symmetrical patchy distribution over all four quadrants. About 12% of atrophic lesions will resolve spontaneously.<sup>14</sup>





Fig 3: Atrophic lichen planus

Erosive or ulcerative oral lichen planus is the second most common type. The lesions are usually irregular in shape and covered with a fibrinous plaque or pseudomembrane. The periphery of the lesion is usually surrounded by reticular or finely radiating keratotic striae. It is painful when the pseudomembrane or fibrinous plaque is disturbed. It has been reported that erosive oral lichen planus has a greater potential to undergo malignant change.<sup>14</sup>



Fig 4: Erosive lichen planus

### Histopathology

The microscopic criteria for lichen planus include hyperkeratosis, basal layer vacuolization with apoptotic keratinocytes and a lymphohagocytic infiltrate at the epithelium-connective tissue interface. Over time, the epithelium undergoes gradual remodeling, resulting in reduced thickness and occasionally a saw tooth rete ridge pattern. Within the epithelium, there are increased numbers of Langerhans cells. Discrete eosinophilic ovoid bodies representing the apoptotic keratinocytes are noted at the basal zone. These colloid or civatte bodies are seen in other conditions such as drug reactions, contact hypersensitivity or nonspecific inflammatory reactions.<sup>8</sup>

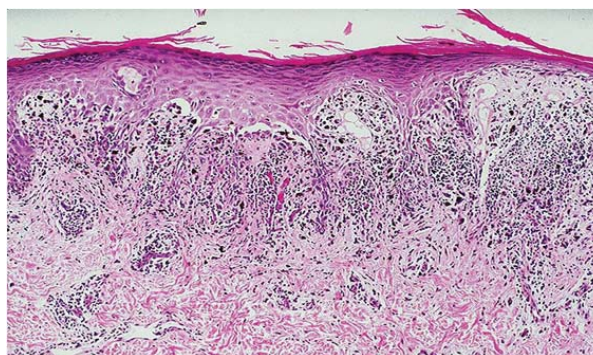


Fig 5: Histopathology of OLP

The histologic features of lichen planus vary according to the age of the lesion. Following regeneration of the basal layer, a discrete eosinophilic band that separates the epithelium from the underlying lymphocytic infiltrate is often seen. Secondary to the destruction of basal cells, desquamation of the epithelium may occur or a bulla may develop. In erosive lesion, the inflammatory infiltrate is extremely dense and more pleomorphic.<sup>15</sup>

### Diagnosis

A complete history and physical examination by a multidisciplinary group of health care providers may be required to investigate oral, skin, nail, scalp, genital, oesophageal, laryngeal and conjunctival involvement. The history, typical oral lesions and skin or nail involvement are usually sufficient to make a clinical diagnosis of OLP.<sup>1</sup> As OLP is a chronic disease, the patient's medical history, psychological state, and treatment compliance, as well as possible drug interaction, must be considered when evaluating the cost effectiveness of any treatment modalities. When oral lichenoid lesions are suspected to be related to the use of a given drug, the medication should be discontinued whenever possible. Mechanical trauma of dental procedures, friction from sharp cusps, rough dental restorations, and poorly fitting dental prostheses can be exacerbating factors of symptomatic OLP and should receive attention. Furthermore, dental amalgam restorations can cause oral lichenoid lesions which may improve following replacement of amalgam with other restorative materials. The psychological profile of the patient should also be taken into account. Studies have reported higher levels of anxiety, greater depression and increased psychic disorders in OLP compared with a control group, and stress is one of the most frequent causes of acute exacerbations in oral lichen planus patients.<sup>2, 14</sup>

However, biopsy is required to differentiate between OLP and other chronic white or ulcerative oral lesions including reactive keratoses, chronic hyperplastic candidosis, epithelial dysplasia, discoid lupus erythematosus, gastro-intestinal disease (including oral Crohn's disease) or anaemic states. Malignancy must also be excluded. Direct immunofluorescence can help distinguish erosive, ulcerative or the very rare bullous form of OLP from pemphigus vulgaris, benign mucous membrane pemphigoid, dermatitis herpetiformis and linear IgA bullous dermatosis.<sup>1</sup>

### Treatment

Although oral lichen planus generally cannot be cured, some drugs can provide satisfactory control.<sup>17</sup> Corticosteroids are the single most useful group of drugs in the management of lichen planus. The rationale for their use is their ability to modulate inflammation and the immune response. Topical and local injection of steroids has been used successfully in controlling but not curing his disease. In circumstances in which symptoms are severe, systemic steroids may be used for initial management.<sup>2, 3, 4, 8, 16, 17</sup>

The steroids used to treat painful, erythematous, or erosive oral lichen planus lesions include betamethasone, fluocinolone, triamcinolone, clobetasol and prednisolone (systemic). Immunosuppressants like azathioprine are also used for painful, erythematous, or erosive oral lichen planus that is recalcitrant to topical corticosteroids.<sup>2, 13, 16, 17</sup>

The addition of antifungal therapy to a corticosteroid regimen typically enhances clinical results by eliminating secondary candidal growth in lichen planus involved tissue. Antifungals also prevent the overgrowth of candida that may be associated with topical corticosteroid use. Tacrolimus and pimecrolimus can be used in steroid resistant cases, although the response tends to be less dramatic than that with topical steroids.<sup>8, 17</sup>

Systemic and topical vitamin A analogs (retinoids) have been used in the management of lichen planus. Reversal of white striae can be achieved with topical retinoids, although the effects may be only temporary. Systemic retinoids have been used in cases of severe lichen planus with various degrees of success.<sup>2, 8, 17</sup>

In patients who are suffering from painful erosive OLP and are unresponsive to even topical corticosteroids, surgical management using cryosurgery and different types of laser have also been tried. All types of laser destroy the superficial epithelium containing the target keratinocytes by protein denaturation but still their effectiveness is yet to be proven.<sup>3</sup>

### Prognosis

Erosive lichen planus responds well to systemic corticosteroids but a milder, residual clinical disease often persists and consequently a drug free remission is less common than a maintenance control remission. Often the disease can be suppressed with prednisolone to a point at which topical fluocinolone or triamcinolone can maintain the remission. Current immunosuppressive therapies usually control oral mucosal erythema, ulceration and symptoms in OLP with minimal side effects, although a range of therapies may need to be trialled. The typical clinical course of OLP is lesion persistence with periods of exacerbation and quiescence. Oral lichen planus patients are at increased risk of oral cancer, although the risk of oral cancer in OLP patients may be reduced. In this context, the prognosis for the majority of OLP patients is excellent.<sup>1, 15</sup>

### Malignant transformation

There are differing views about the malignant potential of oral lichen planus. It may be that there is no longer any controversy and that oral lichen planus must be thought to give rise to squamous cell carcinoma of the oral cavity in a small proportion of cases, the risk lying between 0.5% and 2.5%. However, an opposing view is that there is a distinct and unrelated condition, lichenoid dysplasia, which is frequently misdiagnosed as lichen planus, and which shares a malignant potential with other dysplastic lesions.<sup>7</sup> Cutaneous lichen planus has no malignant tendency. Molecular biological techniques suggest that oral lichen planus itself is not precancerous and that malignant transformation is associated with the coincidental development of dysplasia in the lesions. This uncertainty must be viewed in the light of the many reported cases of malignant transformation in oral lichen planus. Recent reports have suggested that atrophic, erosive, ulcerative or, desquamative are the clinical variants most likely to develop malignant change. There is no consensus as to whether this uncertainty seems to makes it necessary for such cases to be followed for a long as the condition is present.<sup>2, 7, 17, 18</sup>

In summary, 'risk for malignant transformation' or 'malignant potential' seem to be better terms for describing OLP. This is in deference to 'pre-malignant', which infers that all OLP will eventually transform, which we know does not happen. Although a rate for malignant transformation has not been reproducibly estimated, the risk appears to be low.<sup>18</sup>

## Conclusion

OLP is a common oral mucosal lesion and one of the most common mucosal pathosis encountered by dental practitioners. It is imperative that the lesion is identified precisely and proper treatment be administered at the earliest. A proper understanding of the pathogenesis of the disease becomes important for providing the right treatment. Clinical overdiagnosis of lichen planus, coincidental occurrence of lichen planus and oral cancer and microscopic confusion with dysplasias that have lichenoid features have contributed to the controversy over malignant potential of this disease. Malignant transformation is more likely to be associated with erosive and atrophic forms and those who also have a history of alcohol and tobacco misuse. As OLP is a chronic condition, patients should be observed periodically and should be offered education about the clinical course, rationale of therapy and possible risk of malignant transformation.

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