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Oral Lichen Planus: A Various Forms Disease

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Abstract

Oral lichen planus is a chronic inflammatory disease that can appear in several clinical subtypes as reticular, papular, plaque like, vesiculo-bullous and the most evolved from the erosive type. These lesions are often met in oral pathology leading to different levels of pain and difficulties in everyday life that is why it is important to be able to take care of these patients.

The aim of this study is to describe the different clinical patterns, to have the key for a correct diagnosis and the alternative medical examinations leading to confirm the diagnosis. Number of treatments are available from topical or systemic medication to laser ablation depending on the subtype and the evolution level.

Keywords: Oral Pathology ,Oral Lichen Planus, Reticular Oral Lichen Planus, Papular Oral Lichen Planus, Plaque Like Oral Lichen Planus, Vesiculo-Bullous Oral Lichen Planus, Erosive Oral Lichen Planus, Oral Lichen Planus Therapy, Corticosteroids, Calcineurin Inhibitors, Retinoids, Laser Therapy

Lichen Planus (LP) is a condition that can cause swelling and irritation in the skin, hair nail and mucous membranes, due to an abnormal immune response. It a chronic inflammatory disease that can be very painful and difficult to live with.

Cutaneous forms commonly involve the classical 6 P's as Pruritic, Purple, Polygonal, Planar, Papules and Plaques; and are typically symmetric and bilateral. The oral impairment can be the unique clinical presentation of a lichen planus, and may be coupled to cutaneous or mucosal manifestations [1].

Oral lichen planus is a chronic inflammatory disease of immune origin whose etiopathogenesis has not been completely disclosed. Factors such as stress, genetic background, certain dental materials, several drugs, infectious agents or an association with autoimmune disorders have been involved. It is often questioned whether malignant transformations occur on OLP lesions or on epithelial dysplasias of lichenoid appearance. OLP eruptions usually have a distinct clinical morphology and a characteristic distribution, but OLP may also present a confusing array of patterns and form.

Epidemiology

Depending the studies, the average prevalence of LP is in between 0,22% to 5% worldwide. Some studies report a slight women predominance particularly over the age of 40 years. Prevalence of concomitant cutaneous and buccal lesions is 20% to 50%, cutaneous lesions exist in 15% of patients suffering from LP but oral lesions are still the most frequent with 30% to 70% of patients affected. Oral lichenoid reactions gather: OLP (Oral Lichen Planus), contact lichenoid reactions, lichenoid drug reactions, Lichenoid reactions of graft-versus-host disease (GVHD) [2].

Olp Clinical Patterns

OLP has several clinical subtypes as reticular, papular, erosive, atrophic, bullous, and plaque-like subtypes. Eisen suggested that mechanical trauma of dental procedures, smoking, and bad oral habits may exacerbate OLP. The incidence and spreading of lesion in the oral mucosa are 80% on gums and inside cheeks, 65% on the tongue, 20% on the lips and ≤ 10% in floor of mouth or palate [3].

Subtypes

The reticular form commonly found on the cheeks is the most common and is characterized by Wickham's striae, described to be fine whitish points or lacy lines surrounded by well-defined erythematous borders and can have an evolution to severe subtypes as erosive OLP. Note however in reticular patterns spontaneous remission is more common (Figure 1) [4, 5].

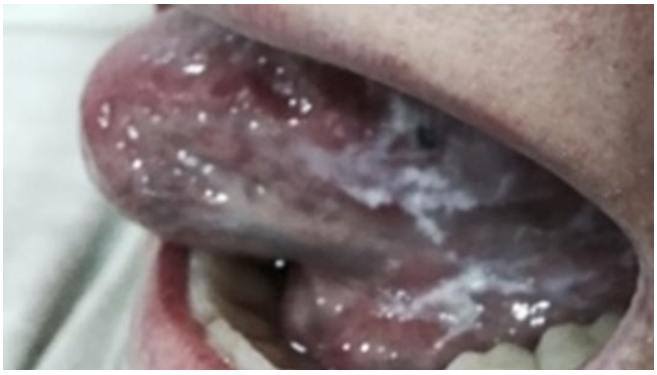

Figure 1: Reticular oral lichen planus on the lateral border of the tongue

Papular OLP is characterized by small white point papules difficult to see and asymptomatic, that is why it is a rare diagnosis: frequently located on the lateral border or dorsum surface of the tongue (Figure 2).

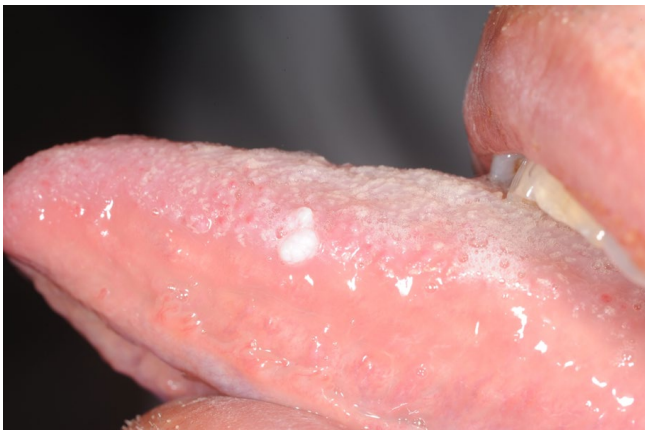

Figure 2: Papular oral lichen planus on the lateral border of the tongue

Plaque like forms show large and homogeneous irregularities white patches similar to leucoplakia, more common with tobacco smokers : it mainly involves the dorsum of the tongue and the mucosa of the cheek (Figure 3) [6].

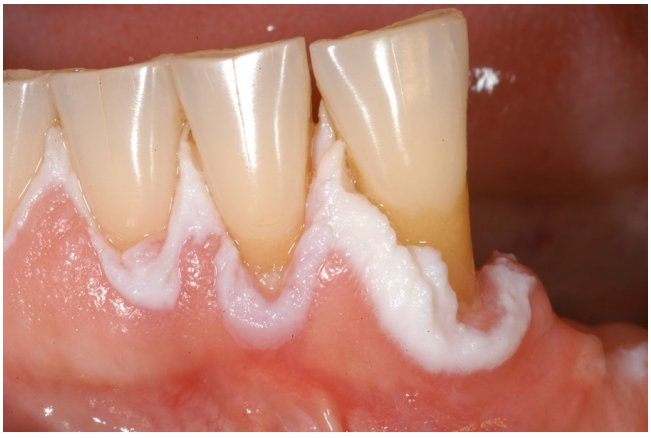

Figure 3: Plake like oral lichen planus on the gum of the lower jaw

FiVesiculo-bullous form is the most unusual clinical form, exhibiting that increase in size and tend to rupture, leaving the surface ulcerated and painful (Figure 4) [7].

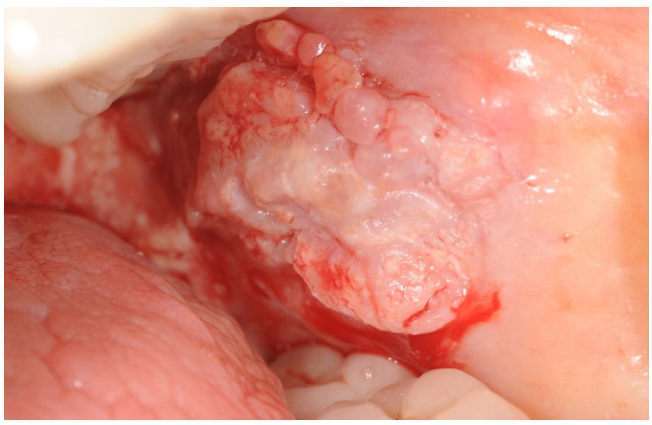

Figure 4: Vesiculo-bullous form on the mucosa of the left cheek

The most evolved form, Erosive OLP, will show symptomatic lesions as atrophic or erythematous ulceration and erosion areas of the mucosa, and Wickham's striae with a network appearance [8].

Etiology

OLP etiology is uncertain, but we are sure that the immune system is the key in the development of this disease. We can still say that it is a T-cell mediated autoimmune disease in which the auto-cytotoxic CD8+ T cells trigger apoptosis of the basal cells of the oral epithelium [4].

Autoimmunity OLP may sporadically be related with autoimmune disorders: concomitant pathologies have been shown as chronic ulcerative colitis, alopecia, alopecia areata, vitiligo, dermatomyositis, primary biliary cirrhosis, coeliac disease, Crohn's disease, thymoma, myasthenia, or agammaglobulinemia [9].

In recent studies, an association between OLP and hepatitis C virus (HCV) has been described in different ethnicities (Japanese and Mediterranean populations). This is partially explained by the observation that specific alleles of the major histocompatibility complex, such as HLA-DR6, are more prevalent in Italian patients with HCV-related OLP. However, no scientific based knowledge has described association between OLP and HCV [10].

Dental reconstruction materials (usually amalgam) in close contact to a lichenoid-like area can evoke an etiology based on these materials and suggest their removal. Therefore, factors that influence response of the immune system may be important for evolution of this pathology. Stress and psychological problems, as depression and anxiety, are etiological factors in the development of LP, without agreement considering them as major or minor etiological factors in development of LP [11].

Diagnosis

Clinical diagnosis

More commonly the distribution of oral lesions is bilateral and symmetric.

Correct OLP diagnosis before starting any treatment is essential (Figure 5). Various subtypes of OLP may be concomitant in a patient and the appearance can change over time. Clinical examination, and patients history will often be enough to conclude a good diagnosis. OLP may be diagnosed, in certain types, only by its clinical presentation, specially the reticular form. Biopsy and histopathological examination confirm the diagnosis; we often proceed these examinations even though they are not recommended in all cases of oral lichen planus suspicion [12].

Even if OLP lesions are generally bilateral and symmetrical, the cases with etiology based on dental materials have an asymmetrical and unilateral situation coinciding with the restorations [13].

Many cases do not present any symptoms, particularly when the reticular form is alone. Some patients report a roughness of the lining of the mouth, sensitivity or pain of the oral mucosa to any stimuli as medication or food. Certain cases show painful and inflamed gums which prevent good toothbrushing, red or white patches on the oral mucosa or painful ulcers. It is possible to find depapillated areas on the dorsum of the tongue, bringing to modify taste and feeling of scorch. Studies have shown that OLP really affects daily life [14,15].

The establishement of a clinical diagnosis, reticular or papular textures must be present. If, in addition, plaque- like, bullous, erythematous, or ulcerative areas are present, the OLP lesion confirmed. If the lichen planus lesion is concentrated to the gingiva it could only be an erythematous lesion needing to be confirmed by biopsy.

Gingival erythematous lesions may avoid observation of papules or striae. Atrophic types usually affect the gingiva and should be differentiated from pemphigoid due to the same desquamative appearance of the mucous surface [16].

Biopsy contribution to the diagnostic

A biopsy is necessary to confirm diagnosis of oral lichen planus especially for atypical cases. In case of doubt a removed abnormal tissue by incision or punch offers a definitive diagnosis. Standard Hematoxylin Eosin Safran process is usually sufficient to avoid neoplasms [17].

All the lesions are described histologically by:

- A distinctive "lichenoid tissue reaction" presenting a bandlike lymphohistiocytic infiltrate that fills the chorion and liquefactive degeneration of the basal keratinocytes. It is the same pathologic process where T-lymphocyte directed, immune mediated, attack the oral epithelial basal cells
- An hyperortho-or parakeratosis
- Presence of a marked stratum granulosum [18]

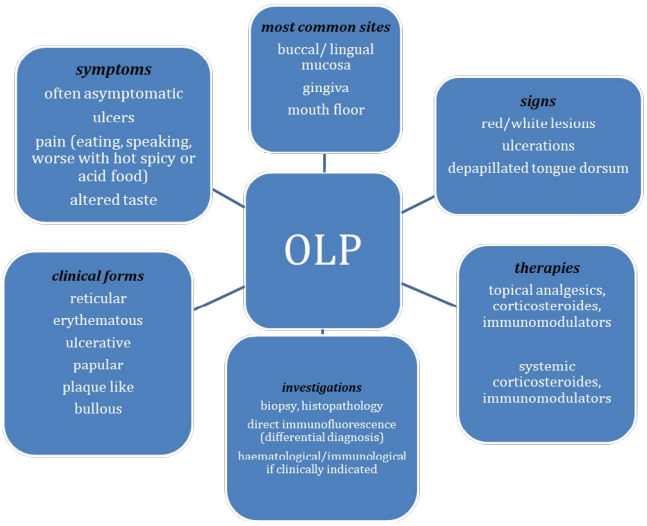

Figure 5: Oral Lichen Planus overview diagnosis

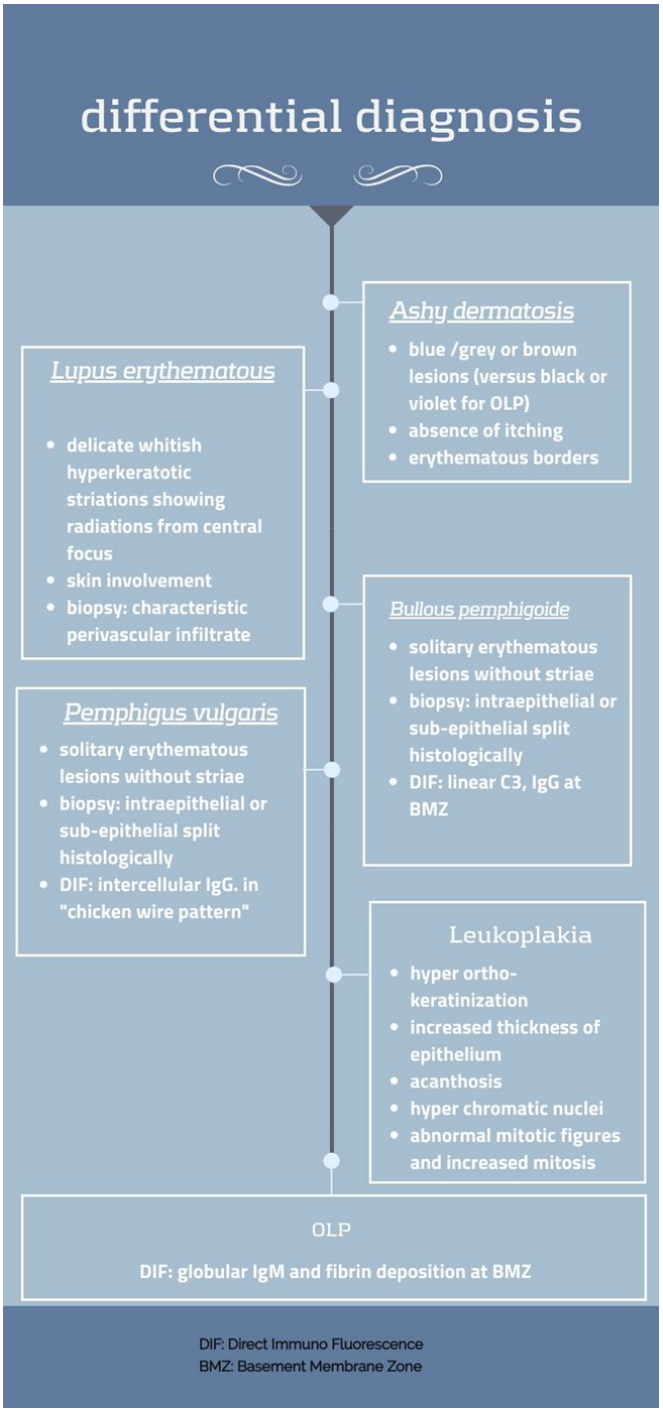

Figure 6: Differential diagnoses of the oral lichen planus [19].

Treatments

A large range of treatments are available from medication to laser ablation. The treatment regarding the symptomatology should be topical or systemic.

Pain leads to the need to start treatment. The goal of treatment is to reduce pain and improve the healing of oral lesions. Currently there is no curative treatment for the moment. The 2020 Cochrane literature review evaluating the success of corticosteroids in the treatment of OLP concludes that they may be effective in treating the pain produced by OLP but these conclusions are very limited due to the small number of studies available and the small number of participants. Corticosteroids are the base of OLP care; still, different treatments as calcineurin inhibitors, retinoids, tacrolimus, and laser therapy have shown

effectiveness on these lesions. Recent studies on the mechanisms of the disease suggests that blocking IL-12, IFN-γ, TNF-α, RANTES, or MMP-9 activity or upregulating TGF-β1 activity in OLP may be the future oriented path to find a treatment [20].

Corticosteroids

The lesions are most often resistant to local treatments. The first-line treatment for OLP is corticosteroids (40mg of Prednisolone per day for 5 days followed by 20 mg of Prednisolone for 10 days) [21].

There is no evidence that corticosteroids for topical use can eliminate the lesions of OLP and side effects are not known. There is no evidence that any corticosteroid studied is more effective than another (Flucinonide, Triamcinolone acetonide, Clobetasol proprionate, Betamethasone, Dexamethasone, and Prednisolone).

The Cochrane systematic review (2020) shows that investigations into the effectiveness of corticosteroid treatments must be carried out. A Cochrane systematic review of other possible treatments for LPB is currently being developed. The application of corticosteroids with an adhesive base (sodium carboxymethylcellulose) may be recommended even if the Lo Muzio study does not find any difference with the application of corticosteroids alone. Other possible treatments are: calcineurin inhibitors (tacrolimus, cyclosporine, pimecrolimus), retinoids, UVA therapy, photodynamic therapy or LASER CO2. A therapeutic gradient will have to be respected due to the side effects of certain therapies [22].

Calcineurin inhibitors

Calcineurin is a protein phosphatase which is involved in the activation of transcription of IL-2, stimulating growth and differentiation of T-cell response. In immunosuppressive therapy, calcineurin is inhibited by cyclosporine, tacrolimus and pimecrolimus. These drugs are called calcineurin inhibitors. Cyclosporine is used as a mouth rinse or topically with adhesive bases in OLP [23].

Retinoids

Topical retinoids (tretinoin, isotretinoin, fenretinide), are able to modulate the immune response, they have shown effectiveness against OLP. Elimination of white striae is possible with topical retinoids, but the effect can be reversible. Different degrees of success are shown with use of systemic retinoids in cases of severe OLP. A balance between positive effects and side effects like cheilitis, elevation of serum liver enzymes and triglyceride levels and teratogenicitymust leed to the choice of using retinoids [24,25].

Tacrolimus

Tacrolimus, is a topical immunosuppressive agent without steroids witch has proved it's effects to treat atopic dermatitis. It is 10–100 times as potent as cyclosporine and has greater percutaneous absorption than cyclosporine. Efficience has been proved on resistant OLP forms [23].

Laser therapy

In cases of painful erosive OLP without any positive results to topical corticosteroid treatments, cryosurgery and laser therapies have been studied. A 980-nm Diode laser, CO2 laser evaporation, biostimulation with a pulsed diode laser using 904 nm pulsed infrared rays and low-dose excimer 308-nm laser with UV-B rays have been tried. Laser destroys the superficial epithelium containing the target keratinocytes by protein denaturation. A diode laser will destroy the underlying connective tissue with the inflammatory component along the epithelium by producing a deeper penetrating beam, however a to poor number of studies have been published to prove their effectiveness [26].

Conclusion

According to the literature review, most authors agree on the malignant transformation potential of OLP as well as contact lichenoid reactions. It is therefore necessary to perform an annual follow-up of these patients as a minimum.

This paper illustrates the difficulty of making an accurate diagnosis of oral lichen planus. A clinical examination is not enough, an anatomo-pathological examination can rule out certain differential diagnoses, but the diagnostic wandering may last many months or years.

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