oral lichen planus- a review

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ABSTRACT
Lichen Planus (LP) is an immune-mediated inflammatory situation which can affect the oral hollow space, skin, nails, hair, eyes, esophagus, and different mucous membranes. The clinicians ought to be familiar with the scientific features of LP, which include oral LP’s (OLP) etiologies, danger elements, and possible treatments. This comprehensive review discusses LP/OLP etiologies, such as dental materials allergies, prescribed drugs¹ aspect results, genetics predisposition, and systemic sicknesses, such as autoimmune and dietary deficiencies. In addition, possible institutions with OLP and viruses are mentioned. The authors additionally summarized OLP’s remedy with glucocorticosteroid remedy or other modalities that would reduce the affected person’s signs and symptoms even as investigating the opportunity of getting an underlying cause.
Keywords: Oral Lichen Planus, immune mediated dysfunction, reticular type, saw tooth rete peg, corticosteroids.

INTRODUCTION
Lichen planus is a persistent inflammatory mucocutaneous disorder that evolves in outbreaks, affecting the skin, mucous membranes, hair, eyes, and nails. It is first described in 1869 by way of British health practitioner Wilson Erasmus. When the lesions are present in the oral cavity, it’s called oral lichen planus (OLP). Previous studies imply that OLP is a T-cell dysfunction-precipitated localized autoimmune disease. OLP usually influences buccal mucosa, tongue, and gingiva. It continually has a bilateral and symmetric distribution of the oral lesions. It happens often in elderly female patients with a female-to-male ratio of 1.5:1. The diagnosis of OLP is done by clinical and histological examination. However, in classical lesions, it is possible to achieve the analysis based on clinical appearance. OLP lesions normally last for years with alternating intervals of exacerbation. There are several feasible causative agents for OLP, the maximum common being prescribed drugs and dental materials that produce a lichenoid response. If the lesions clear up whilst the causative agent is eliminated, it confirms a lichenoid response. If the lesions hold, then it’s miles given the analysis of OLP. The oral lesions in Lichen planus are normally persistent, don’t display spontaneous remission and have the propensity to emerge as cancerous. Even though oral lichen planus is often asymptomatic, it once in a while causes symptoms ranging from burning sensations to intense pain, which interferes with talking, ingesting and swallowing.

ORAL LICHEN PLANUS AND LICHENOID LESION
Oral lichenoid reactions or lesions (OLL) are phrases used to explain lesions that clinically and histologically resemble OLP however have an identifiable etiology. Very few epidemiological studies on the frequency of OLL exist and viable precipitants encompass chronic graft-as opposed to-host sickness (cGVHD), some dental substances, a range of drugs and some viruses. Lichenoid lesions have a propensity to be unilateral and erosive with histological examination displaying a greater diffuse lymphocytic infiltrate with eosinophils, plasma cells and growth in colloid bodies in contrast with classic OLP. However, regularly OLL is indistinguishable from idiopathic OLP.

ETIOLOGY
Oral LP is possibly a T-cellular-mediated immunological reaction to an brought about antigenic exchange inside the pores and skin or mucosa in predisposed sufferers. A key, early event in LP is the genetically prompted elevated production of Th1 cytokines. Another key event in OLP pathogenesis is the recruitment of different subsets of dendritic cells (DCs), which include Langerhans cells, stromal DC-SIGN⁺ DCs and plasmacytoid, likely via the expression of the chemotactic agonist chemerin through endothelial cells lining blood vessels. Attraction and migration of the activated T cells to the oral epithelium is further greater by way of intercellular adhesion molecules (ICAM-1 and VCAM), upregulation of epithelial basement membrane extracellular matrix proteins (collagen sorts IV and VII, laminin and integrins), and probably CXCR3 and CCR5 signaling pathways. Binding of T cells to keratinocytes and IFN-gamma, and subsequent upregulation of p53, matrix metalloproteinase (MMP)1 and MMP38 results in apoptosis, culminating in destruction of the epithelial basal cells. The chronic path of OLP may be due to the activation of the inflammatory mediator NF-κB, and the inhibition of the TGF manage pathway (TGF-beta/SMAD), which might also purpose keratinocyte hyperproliferation, leading to improvement of the white lesion.

DENTAL RESTORATIVE MATERIAL
The most usual dental substances related to OLP that purpose a lichenoid reaction are amalgam, composite resins, nickel, and gold, which motive a touch sensitivity response. Another possible reason for oral lesions associated with dental restorations can
be an immunological or toxic reaction to plaque accumulation on the floor of the recuperation, and such lesions may disappear after improvements in oral hygiene. A patch test can determine those allergens.

MEDICATIONS

Drugs inducing OLP/OLL are classically NSAIDs and angiotensin-converting enzyme inhibitors. Withdrawal and reintroduction of the drug is the most reliable technique for diagnosing a drug reaction based on the decision or reactivation of the lesion. However, that is frequently no longer sensible and can be probably dangerous. A systematic assessment said that there's enough evidence that b-blockers, methyldopa, penicillamine, quinidine and quinine play a function in LP even as NSAIDs ought to also be taken into consideration causative. Empirical withdrawal of the drug in query, and its substitution with every other, can be warranted as it could take months for a decision of the lichenoid reaction.

PSYCHOLOGICAL STRESS

The role of mental strain on the immune system and inflammatory process affects the neuroendocrine, cardiovascular, gastrointestinal, and crucial structures, along with contamination psychosocial events, autoimmune ailment, and at the end cancer by viral mediation. Stress-associated release of cortisol, which produces inflammatory mediators along with cytokines with a T helper cell 1 [Th1], ought to contribute to OLP.

INFECTIOUS AGENTS

Several infectious agents together with H. Pylori and some viruses, were implicated in the precipitation of the cell-mediated response that results in OLP. The function of human herpes viruses (herpes simplex virus 1, Epstein–Barr virus, cytomegalovirus, human herpesvirus [HHV]-6 and HHV-7) in the pathogenesis of OLP, within the case of HHV-7, is unclear and may be secondary to oral erosive LP lesions. The exceptional to be had evidence of the involvement of hepatotropic hepatitis C virus (HCV). HCV is one of the primary reasons of chronic liver sickness internationally, but its morbidity is likewise because of a whole lot of extrahepatic manifestations inclusive of mixed cryoglobulinemia, non-Hodgkin lymphoma, diabetes, porphyria cutanea tarda and LP.

GENETICS

Familial OLP has an early age onset, and it may come to be severe and continual. It has a tendency to have a peculiar and extensive clinical presentation. The maximum widely spread genes associated are HLA-DR1, DR2, DR3, DR4, DR7DR9, HLA- DRB1, HLA-DQA1, and HLA-DQB1 and HLA-DR6. A meta-analysis showed that the A allele and AA genotype in IL 10-592 C/A increased OLP susceptibility. Other polymorphisms related to the susceptibility of OLP consist of TNF-alpha (-308A), TNF-B (+252A/G), and IL (-10G/A, -819C/T, and -592C/A).

TYPES OF ORAL LICHEN PLANUS

Clinically, six varieties of OLP, specifically reticular, plaque-like, papular, atrophic/erosive, ulcerative, and bullous sorts, can be recognized.

- Reticular OLP is the prevalent form OLP. It contains "Whickham striae," an asymptomatic white lace-type pattern at the cheek or buccal mucosa. It may be erythematous or non-erythematous.
- Papular OLP is an unprecedented shape that incorporates small white raised (papular) regions and may generate fine striae. It commonly carries some other variation described.
- Plaque-like OLP seems similar to leukoplakia. It has a whitish color generally observed at the cheek and dorsum of the tongue. Papular, plaque-like, and reticular are generally asymptomatic and hyperkeratotic (white in coloration).
- Erosive OLP is defined as an ulcerative purple, an inflamed region which could incorporate a white lacy pattern. It is commonly sore or painful to the patient. It is likewise defined as burning or uncomfortable.
- Atrophic is an extraordinary shape of OLP which has diffuse red lesions. It may have two one-of-a-kind editions of OLP within the lesion, which include white striae (reticular) within the middle and redness (erythematous) surrounding it.
- Bullous OLP has a painful ulcerative floor. It incorporates blisters that usually rupture, causing ulcerations. It can be high-quality for Nikolsky’s signal. Erosive, atrophic, and bullous are the "purple form" of OLP and are painful.

PATHOGENESIS

The pathogenesis of Lichen planus is poorly understood. But oral lichen planus is of autoimmune beginning. Both antigen-specific and non-particular mechanisms are worried in OLP. Antigen-particular mechanisms consist of antigen presentation with the aid of keratinocytes and Langerhans cells to CD4+ helper and CD8+ cytotoxic T lymphocytes to set off those two styles of T cells. The activated helper T cells can secrete interleukin (IL)-2 and interferon (IFN)-γ which in turn activate the cytotoxic T lymphocytes and promote their proliferation. Finally, the activated cytotoxic T lymphocytes can cause the apoptosis of basal keratinocytes and result in the liquefaction degeneration of basal epithelial cells generally located in OLP lesions. Non-precise mechanisms encompass mast cellular degranulation and release of tumor necrosis factor (TNF)-a and chymase. The TNF-a can help the T cells emigrate from the capillaries into the surrounding extracellular matrix. Chymase can activate the matrix metalloproteinase (MMP)-9 which subsequently destroys the basement membrane and ends in the migration of CD8+ cytotoxic T lymphocytes into the epithelium of OLP lesions. The intraepithelial cytotoxic T lymphocytes can further result in the apoptosis of basal and parabasal epithelial cells. The above findings recommend that OLP is a T-lymphocyte-mediated chronic inflammatory oral mucosal sickness. The above findings propose that OLP is a T-lymphocyte-mediated localized autoimmune sickness. Serum autoantibodies like Specific serum anti-nuclear (ANA), anti-clean muscle (SMA), anti-mitochondrial (AMA), gastric parietal cellular (GPCA), thyroglobulin (TGA), and thyroid microsomal autoantibodies (TMA, also known as thyroid peroxidase antibody or TPO) are located in OLP.
CLINICAL FEATURES

Usually, OLP has exclusive medical functions and distribution, characterized with the aid of more than one, bilateral but not continually symmetrical, mucosal lesions. There are 4 medical manifestations of OLP: papular, reticular, erythematous (atrophic) and erosive (ulcerated, bullous). Classification is determined by means of the worst presenting shape; thereby, an affected person offering with reticular and erosive adjustments might be high-quality labeled as having erosive OLP. However, some authors consider erosive OLP to consist predominantly of erosive lesions. Papular/reticular lesions, regularly the simplest medical manifestation of the disease, arise in isolation and usually take the shape of minute white papules that expand and coalesce to shape both a reticular, annular, or plaque-like pattern. These reticular lesions seem like an interconnected, overlapping network of white lines (so-referred to as ‘Wickham’s striae’). Lesions of erythematous and erosive OLP bring about various diplomas of pain. The length, area and quantity of ulcerated regions are variable, spontaneous remission is rare and confusion with the autoimmune illnesses pemphigus vulgaris (PV) and mucous membrane pemphigoid (MMP), which proportion comparable medical features, is not unusual. However, concomitant reticular lesions useful resource clinical differentiation of OLP from PV and MMP, that are characterized simplest with the aid of regions of erythema and/or erosion. The posterior buccal mucosa observed through the tongue, gingival, labial mucosa and vermilion of the decrease lip are the maximum frequently worried sites, whilst lesions of the palate, ground of mouth and upper lip are rare. Approximately 15% of sufferers with OLP broaden cutaneous lesions, with 20% of women with OLP developing lesions of the genital mucosa, the most common more-oral site in girls. The affiliation of LP of the vulva, vagina and gingiva is identified as vulvovaginal–gingival syndrome. Symptoms encompass burning, pain, vaginal discharge and dyspareunia and are regularly stated in sufferers with erythematous and erosive genital ailment. On occasion, patients with slight oral involvement display intense erosive vulvovaginal disease, and sufferers with excessive oral involvement broaden handiest slight asymptomatic genital disorder. Esophageal and conjunctival involvement in OLP patients has currently been reported, and in each instance cicatricial lesions aren’t unusual.

HISTOPATHOLOGICAL FEATURES

The histopathologic capabilities of OLP are feature and composed of hyperkeratosis (hyperorthokeratosis or hyperparakeratosis) of the epithelium, hydropic or liquefaction degeneration of basal epithelial cells, atrophy or acanthosis of spinous epithelial cells, saw-tooth epithelial ridges, a homogeneous eosinophilic deposit on the epithelium connective tissue junction, and a band-like lymphocytic infiltrate inside the superficial lamina propria. Degenerating keratinocytes may be seen in the place of the epithelium and connective tissue interface and had been termed colloid, cytod, hyaline or Civatte bodies. Compared to cutaneous LP lesions, OLP lesions less regularly display saw-tooth epithelial ridges and greater often show off epithelial atrophy with unobvious epithelial ridges. Moreover, epithelial dysplasia ought to no longer be observed in OLP lesion.

DIRECT AND INDIRECT IMMUNOFUORESCENCE FINDINGS FOR ORAL LICHEN PLANUS

DIF is a diagnostic adjunct for OLP lesion. The feature DIF finding for OLP lesion is a deposit of fibrinogen in a shaggy pattern on the basement membrane region in the absence of immunoglobulin and complement. DIF is regularly essential to distinguish OLP from mucous membrane pemphigoid and pemphigus vulgaris. DIF finding for mucous membrane pemphigoid is a linear deposition of immunoglobulins (IgG, IgA or IgM) or complement three (C3) at the basement membrane region of the epithelium, and that for pemphigus vulgaris is a lattice-like (or chook wire) deposition of immunoglobulins (IgG or IgM) or C3 within the intercellular areas among epithelial cells. The oblique immunofluorescence (IIF) locating for OLP is terrible.

DIAGNOSIS

The medical features on my own, specially when providing inside the ‘classic’ reticular form, may be sufficiently diagnostic. However, given the chronic nature of OLP and the requirement for long time treatment and tracking, biopsy could be prudent scientific practice, specifically previous to beginning an active treatment, as a common purpose of remedy failure is inappropriate analysis. Despite the fact that the histopathological assessment of OLP can be subjective, an oral biopsy with histopathological examination is suggested to verify the scientific diagnosis while the sickness does not gift with its normal manifestations, especially to exclude dysplasia and malignancy. Direct immunofluorescence trying out can be useful whilst there are distinct gingival and/or predominantly erosive/ulcerative lesions to exclude an autoimmune bullous ailment.

DIFFERENTIAL DIAGNOSIS

- Leukoplakia
- Sponge Cheek Nevus
- Erythematodes
- Mechanical Damage to Oral Epithelium
  - Candidosisa
  - Pemphigus Vulgarisa
  - Pemphigoid mucosae oris
  - Lingua Geographica

TREATMENT AND MANAGEMENT

The examination of complete blood count number and serum tiers of iron, nutrition B12, folic acid, and homocysteine is also very essential for OLP affected person before giving remedies for OLP sufferers. The NEOLP (reticular, papular or plaque-like OLP) lesions, if the clinical manifestations are standard (bilateral and symmetric lesions), we may or might not do the biopsy for affirmation of medical diagnosis. For the EOLP (atrophic/erosive, ulcerative or bullous OLP) lesions, biopsy have to be completed particularly for those lesions with the suspicion of dysplastic exchange or
malignant transformation. Both visual inspection and palpation are critical for exam of OLP lesions. If tissue induration is detected on the peripheral place of OLP lesion via palpation, biopsy should be carried out to rule out the opportunity of a malignancy. The remedy for OLP relies upon the types and signs of the ailment. Since OLP is an immunologically mediated disorder, corticosteroids are the medication of desire for treatment of EOLP. If the EOLP lesion is small and the oral signs and symptoms are mild, topical software of corticosteroid (dexamethasone or triamcinolone) ointment as a thin film 2-3 times consistent with day to the most symptomatic regions is typically sufficient to result in recuperation of the EOLP lesions inside 2-3 weeks. If the EOLP lesions are large and the oral signs are mild or excessive, topical spray of corticosteroid powders (e.g., sealcoat cap for spray, containing beclomethasone dipropionate) to the lesional mucosal regions 2-3 times in step with day is likewise effective for induction of recuperation of EOLP lesions. Alternatively, huge and severe EOLP lesions can be dealt with through intralosomal and submucosal injection of kenacort A (forty mg triamcinolone acetonide divided into two doses with each for one-facet EOLP lesion as soon as weekly for 2-3 weeks) plus oral management of prednisolone (15-30 mg of prednisolone once each day for 2 weeks; the oral administration of prednisolone is tapered to five mg consistent with day and stopped inside the 0.33 week). For the severest multifocal EOLP lesions with massive areas of ulceration, systemic management of prednisolone (40 mg of prednisolone according to day for 5 days observed via 10-20 mg of prednisolone per day for every other 7-10 days) can gain a big improvement of the EOLP lesions.

CONCLUSION

OLP is a multifactorial ailment of the oral mucosa and is categorized as a potentially malignant disorder which could rework into OSCC in 1.10 - 1.40% of the instances. While a definitive causative agent for OLP remains unknown, there are some of viable etiologies, inclusive of psychological and bodily pressure, facet effects from medicines, irritation and hypersensitive reactions from dental materials, genetic predisposition, systemic illnesses, viral exposures, and nutritional deficiencies. Certain medications and dental restorative materials were proven to each result in or exacerbate OLP or purpose a lichenoid response. Specific genetic polymorphisms and systemic diseases (which includes diabetes, hypertension, dyslipidemia, thyroid and liver disease, and a bunch of autoimmune situations) had been shown to be related to OLP. Nutritional and nutrition deficiencies (in particular iron, Vitamins B12, A, C, and E) are frequently found in OLP cases. Corticosteroids are the ‘gold standard’ for the remedy of OLP but have shown limited efficacy. Alternative medications and/or treatments had been proven to enhance OLP. Some evidence factors to viruses as risk elements for OLP.

REFERENCES