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Pathogenesis And Histopathological Characteristics of Oral Lichen Planus: A Review Article

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Abstract

Oral lichen planus is a disorder of great clinical and pathological significance. Lesions may be minor reticular to very painful erosive lesions. Pathogenic mechanisms are multifactorial. T-cell-mediated immune responses, cytokine imbalance, genetic susceptibility, and environmental influences have been evidenced as the most factors contributing to the development of this disease. These ultimately result in basal keratinocyte apoptosis followed by chronic inflammation attacking the epithelial cells. Basal cell degeneration and hyperkeratosis with a dense band of lymphocytes characterize OLP at the level of the epithelial-connective tissue interface. That is how much relevant it is toward an accurate diagnosis. This paper will not rise much high but shall ensure updated knowledge toward better comprehension resulting in early diagnosis with apt management of so complicated a disease by reviewing recent articles oriented toward the clinical overview, pathogenesis, and histopathological features of OLP.

Keywords: *Keratinocyte apoptosis, pathogenesis, chronic inflammatory disorder, OLP*

Introduction

Millions of various groups denote major oral pathologies, key public health issues worldwide. Chronic inflammatory and premalignant conditions are greater clinical and scientific challenges. These may be lesions presenting with pain reducing oral function increasing probability for malignant transformation hence mechanism exploration and histopathological characterization become important topics. Relatively common among these is oral lichen planus (OLP) a chronic disease of unidentifiable etiology with extremely wide clinical presentation which has been discussed as being at risk for malignant transformation-involved interest (Gupta & Jawanda 2015).

The pathology discipline always tried to integrate the mechanism of disease as it develops at the molecular

level together with the microscopic view. Oral pathological conditions can be elicited by genetic, immune, and environmental factors. Much distinction is made by leukoplakia, erythroplakia, and oral lichen planus diseases in pathogenesis and histology pattern with a certain amount of overlappings existing between a few of them; hence these can add up diagnostic plus treatment differences. Most importantly, oral lichen planus falls under the WHO categorization group for potentially malignant oral diseases (OPMD) leading towards transformation risk into oral squamous cell carcinoma (OSCC). Consequently, research about its pathogenesis and histopathological characteristics can provide new systems for early detection through accurate diagnoses followed by therapeutic intervention (Speight et al., 2018).

The pathogenesis is the subject of intensive studies, and accurate etiopathogenesis is not known. However, it is generally considered a T-cell-mediated autoimmune disease. At present, evidence available indicates that cytotoxic CD8+ T lymphocytes are the main participants in recognizing antigenic peptides presented by basal keratinocytes. Once identified, they become apoptotic to these epithelial cells, resulting in tissue damage (Gupta & Jawanda 2015; Lucchese et al., 2020). This immune-mediated epithelial destruction is inflammatory with chronicity and even more enhanced epithelial atrophy precipitated by interleukin (IL)-6, IL-17, tumor necrosis factor-α (TNF-α), and interferon-γ (IFN-γ) proinflammatory cytokines (Peng et al., 2021). Other precipitating factors include genetic predisposition psychological stress microbial infection and systemic comorbidities that can influence both the initiation and persistence of the disease (Shengyuan et al., 2020). The diagnosis of OLP is essentially a clinicopathological correlation. Hyperkeratosis, jagged epidermal ridges, liquefactive degeneration of the basal cell layer, and a dense band-like lymphocytic infiltrate are typically seen in lesions. Other lichenoid conditions may enter into the differential diagnosis with OLP; these include contact reactions (amalgam), drug-induced lichenoid reactions, and lupus erythematosus. These conditions do not have, however, microscopic features as primary pathology but rather have overlapping features. Tissue specimens vary considerably across different sites within one lesion of an individual case; consequently, subjectivity of observers further complicates matters in eliciting typical presentations from histological slides. Thus, clinical correlation is imperative for an appropriate diagnosis.

Beyond diagnosis, histopathological evaluation of OLP is imperative. Recent studies have strongly established the fact that variations in histology can be considered as premonition towards malignant transformation. Dysplastic changes, increased proliferation of epithelium as well as molecular alterations (mutations of p53, altered Ki-67 expression, and abnormal E-cadherin signaling) have already been reported in some subsets of lesions of OLP which precipitates a biological continuum from chronic inflammation to carcinogenesis (García-Pola et al., 2017; Zhou et al., 2021). The actual rate of malignancy is much debated however most long-term studies wager a percentage between 1 and 2%. This risk makes vigilance obligatory hence here comes the tissue study as a futuristic tool.

Besides being a suitable disease for the investigations of pathology-immunology relations, it is from OLP that lessons are drawn on how a misbalance at epithelial-connective tissue interplays can trigger chronic inflammatory pathways with ultimate carcinogenesis. This brings to the fore yet another imperative merger of classic histopathology with molecular pathology toward reading patient risks more accurately to interest. Trends have developed to involve high technology in probing into the investigation of OLP, including immunohistochemistry, molecular analysis, and digital pathology as steps toward better understanding its biological behavior (Bodle et al., 2021).

Overview of Oral Lichen Planus

Oral lichen planus is defined as a chronic inflammatory disease of the oral mucosa and has gained much popularity because of its heterogeneous clinical picture, chronicity, and relatively high potential for malignancy. The World Health Organization (WHO) defined this pathology under the rubric of potentially malignant oral diseases to reflect, both clinically and pathologically, its great importance (Warnakulasuriya et al., 2020). Therefore, an adequate understanding of OLP requires a description of its clinical presentation, prevalence rate, demographic profile, and risk factors that will help define its pathogenesis and histopathological characteristics (Ismail et al., 2022).

Definition and Clinical Features

Lichen planus is a chronic mucocutaneous condition that may present with diseases involving the skin, nail, and scalp, genital mucosa or oral tissues. When it occurs solely in the oral cavity it is termed specifically as oral lichen planus (OLP). Clinical forms of OLP include reticular, papular and plaque types; atrophic (erythematous), erosive and bullous conditions. White striae is the most common presentation with striae forming a pattern of interlacing lines usually seen on buccal mucosa. Less common presentations include atrophic and erosive forms of OLP that are of greater clinical concern since they are symptomatic, presenting burning and pain, and the lesions that most frequently relate to malignancy. Other forms of OLP include plaque type that often resembles leukoplakia and bullous OLP that is relatively infrequent. Such heterogeneity in the clinical presentation complicates diagnosis and proves the value of an integration between clinical findings and histopathological examination (Carrozzo & Porter, 2021).

Epidemiology and Prevalence

Around the globe, oral lichen planus has been recorded as something that involves about 0.5%–2.0% of the population. Methodologies and standards shared a difference in rates of prevalence from various regions used to diagnose this condition (Mollaoglu, 2021). It is seen more commonly in older adults, typical presentations between the age of 40 to 60 years old. Women do have higher numbers with two or three times more cases being reported than men. This sex difference may be related to hormonal influences and differences in the immune system. Geographically, OLP has been reported worldwide, but the prevalence is higher in certain populations, particularly in Asia and the Middle East. These differences in prevalence may reflect lifestyle factors, genetic susceptibility, and differences in epidemiological survey methods (Scully et al., 2021).

Risk Factors and Predisposing Factors

Even though the exact etiology of OLP has not been fully elucidated, several possible risk factors and their associations are known. Stress and psychological factors are commonly involved. Patients commonly report exacerbations in periods of emotional or physical stress. Systemic diseases that have associations with OLP include diabetes, hypertension, and hepatitis C virus (HCV) infection. However, strength of these associations varies in different populations (Gupta et al., 2022).

The association of OLP with HCV is seen in areas of high prevalence, that is, Southern Europe, Japan, and the Middle East. High prevalence led some researchers to propose it as part of the etiology through immune mechanisms mediated by the virus. Other predisposing factors include habits; however, it does not directly cause OLP. It increases mucosal irritation which perhaps heightens the danger of malignancy transformation of preexisting lesions (González-Moles et al., 2021).

Clinical Course and Symptoms

The clinical course is most of the time chronic, observed with relapsing seen in many patients accompanying remissions- exacerbations. Great diversity exists regarding symptomatology based on various clinical subtypes. Reticular and papular OLP mostly remain asymptomatic; hence found accidentally during routine oral checkups. It may be as painful as eating food or cleaning the mouth, giving a substantial effect on the quality of life of the patient (Ismail et al., 2022).

A key issue regarding the clinical management of OLP relates to its potential for malignant transformation as OSCC. Malignant transformation has been reported between 1% and 2% of cases, depending on several factors such as diagnostic criteria used, length of follow-up period, and study population. Variations have been noted. It is the erosive and atrophic forms that are most predisposed to such risk; regular follow-up assessments should be instituted (Warnakulasuriya et al., 2020).

Diagnosis and Differential Diagnosis

Diagnosis of oral mucositis is based on clinical and histopathological evidence. According to WHO, major criteria include bilateral symmetry of the lesions for biopsy and histology showing band-like lymphocytic infiltrate, basal cell degeneration, and saw dental ridges. It is difficult to differentiate oral mucositis from other diseases that affect the oral mucosa (van der Meij & van der Waal, 2003).

Oral lichenoid reaction (OLR), leukoplakia, lupus erythematosus, and chronic candidiasis may clinically and histologically simulate oral mucositis. Oral lichenoid lesions (OLL) were described as clinically similar to oral lichen planus (OLP); however, most of the time they occur as a hypersensitivity reaction to dental restorative materials, drugs, or contact allergens. Thus, other causes that can be elicited have to be excluded to arrive at a diagnosis (Scully et al., 2021).

Public Health and Clinical Relevance

The clinical and public health significance of OLP is not confined to its chronic painful nature but as it falls under the categorization of oral lichen planus (OPMD). because OLP carries a malignancy potential, detailed clinical follow-up and patient education are highly recommended. This disease also creates a large psychosocial burden since most patients become anxious and depressed and have reduced quality of life related to oral health. Treatment of OLP has its roots in palliatives. Control of inflammation and management of symptoms rather than control of the disease itself are mainstays in its treatment. Topical corticosteroids form the first line of treatment but they bear a very high relapse rate, thus pushing more studies with regards to etiology, biomarkers, and usable therapeutic targets of this disease (Gupta et al., 2022).

Pathogenesis of Oral Lichen Planus

Oral Lichen Planus represents a chronic inflammatory mucocutaneous disease with a complex and multifactorial pathogenesis. Even though the exact etiology of this disease has not been described, immune dysregulation presents increasing evidence as the main factor. More specifically, it is a T-cell–mediated immune response against basal keratinocytes of the oral epithelium that seems to be most commonly involved in pathogenic mechanisms. Other factors include genetic predisposition, microbial, and environmental factors together with psychosocial stress acting in concert both on the vulnerability and course of the disease. Molecular and cellular mechanisms are highly relevant as they contribute to explaining clinical heterogeneity, histopathological characteristics, and malignant potential in OLP (Carrozzo & Porter, 2020).

Immune-Mediated Mechanisms

The main pathogenic mechanism of OLP is immune-mediated cytotoxicity directed toward basal keratinocytes of the oral epithelium. It thereby involves the recruitment and activation by an unknown antigen expressed by basal keratinocytes of CD8+ cytotoxic and CD4+ helper T lymphocytes to the subepithelial connective tissue (Sugerman et al., 2002; Gupta and Jawanda, 2015).

Keratinocytes in OLP lesions express upregulated levels of major histocompatibility complex (MHC) class I and class II molecules, thereby enabling antigen presentation and recognition by T lymphocytes. CD8+ T cells when activated lead to keratinocyte apoptosis through the expression of perforin, granzyme B, and tumor necrosis factor-α (TNF-α). This cytotoxicity leads to basal cell degeneration, a characteristic histological feature of OLP (Lavanya et al., 2011).

CD4+ T helper cells further promote disease progression by producing proinflammatory cytokines, such as IL-2, IFN-γ, and TNF-α, which maintain a chronic inflammatory environment (Farhi & Dupin, 2010). Regulatory T cells, normally crucial for maintaining immune tolerance, appear to be functionally impaired in OLP, thereby enhancing autoreactive immune responses (Carrozzo & Porter, 2020).

Cytokine Dysregulation

Cytokines, chemokines. Children play and break the bread of life across the immune response in OLP. Through proinflammatory cytokines IL-17, IL-6, IL-1β, and TNF-α; consistent expression within both tissue and serum samples derived from patients diagnosed with OLP. These cytokines basically further enhance the perpetual inflammatory response by attracting more immune cells into activation (Ghallab, 2017).

The Th1 cytokines IFN-γ and TNF-α are the major players in upholding the activity of cytotoxic T cells (Sugerman et al., 2002). More recently expressed by Th17 cells, these molecules attract even more neutrophils to increase the already impressive epithelial barrier dysfunction into a situation where chronic inflammation is connected with carcinogenic potential (Scully & Carrozzo, 2008). Underexpression of anti-inflammatory cytokines such as IL-10 signaling imbalanced pro-and anti-inflammatory pathways (Payeras et al., 2013).

The Role of Apoptosis and Keratinocyte Alterations

Keratinocyte death is a key event in the pathogenesis of OLP. The role of the Fas/Fas ligand (FasL) signaling pathway has been apprised. In this mechanism, keratinocytes expressing Fas interact with cytotoxic T cells expressing FasL leading to apoptosis by means of caspase activation (Farhi & Dupin, 2010). Other pathways that have also been suggested to be involved in basal keratinocyte death include the pathway related to TRAIL (TNF-related apoptosis-inducing ligand) and the mitochondrial apoptosis pathway (Ghallab, 2017).

Keratinocytes are not passive bystanders; they secrete cytokines. IL-1 and TNF-α produced by keratinocytes enhance the inflammatory response and upreges the recruitment of other immune cells (Lavanya et al., 2011). There is involvement of Oxidative stress too. Reactive oxygen species (ROS) generated in OLP lesions can further intensify DNA damage as well as apoptosis and even malignancy transformation (Agha-Hosseini et al., 2012).

Antigenic and Microbial Factors

While the exact antigenic stimuli responsible for T cell activation in OLP are not known, a large number of endogenous and exogenous triggers have been postulated. Self-antigens such as heat shock proteins (HSPs) may become targets through molecular mimicry or by aberrant expression in stressed keratinocytes (Sugerman et al., 2002). Viral infections have been highly associated particularly hepatitis C virus (HCV) with strong regional associations making a putative viral role important in susceptible population areas (Carrozzo, 2014). Human papillomavirus (HPV) has also been studied; however, its role remains controversial. Another component thought to be involved in the pathogenesis of OLP is microbial dysbiosis of the oral microbiome. Change in the oral microbiome increases antigen presentation capability as well as sustaining local immune activation (Cheng et al., 2016).

Genetic and epigenetic influences

Genetic susceptibility is one factor in OLP susceptibility. Certain human leukocyte antigens (HLA) have been reported to relate to OLP. Alleles, including HLA-DR6 and HLA-DR10, provide a pathway through which genetics can be involved in faulty antigen recognition and immune activation (Carrozzo, 2014).

Epigenetic mechanisms-DNA methylation and microRNA dysregulation are equally important. MicroRNAs, miR-155, and miR-146a reveal the regulation of immune response modulation and keratinocyte apoptosis in OLP. This further emphasized that besides the immune-mediated assault, transcriptional and post-transcriptional regulatory mechanisms play a significant role in sustaining and advancing the disease (Yang et al., 2017).

The Role of Stress and Neuroimmune Interactions

Psychological stress has been related to the initiation and flaring up of OLP. Stress influences neuroendocrine pathways, mainly the hypothalamic-pituitary-adrenal (HPA) axis, by changing cortisol levels which eventually leads to immune regulation (Agha-Hosseini et al., 2012). Mediators of stress like substance P and catecholamines can even more assist mast cell degranulation to increase vascular permeability and also enhance T cell activity in OLP lesions (Ghallab, 2017).

Chronic Course and Malignant Potential

The persistent immune-mediated epithelial damage is what makes the oral lichen planus (OLP) condition a chronic disease. This means that since keratinocyte apoptosis and inflammatory cell infiltration followed by epithelium repair continue, genomic instability can easily set in within the OLP condition. Therefore, clinically OLP can be placed in the group of pre-malignant conditions with a relatively lower but still significant probability of transformation into frank oral squamous cell carcinoma (OSCC), as described by Scully & Carrozzo in their 2008 publication. Some patients may be induced into carcinogenesis by cytokines IL-6 and IL-17 as well as oxidative stress and epigenetic alterations (Yang et al., 2017).

Histopathological Characteristics

Oral lichen planus (OLP) is a chronic, immune-mediated mucocutaneous disease presenting most frequently with oral symptoms and has well-defined histopathological features. Histopathology becomes very important in the diagnosis of OLP because through it one can understand the pathophysiology of the disease, differentiate it from other lesions occurring in the oral cavity, and its route to malignant transformation. Microscopically, OLP involves a dynamic interplay of epithelial and connective tissue changes as a consequence of immune pathology and chronic inflammation. Thus, the knowledge of these histopathological features will be very useful in making an accurate diagnosis, and management of the patient, as well as when estimating the risk for malignancy (Alrashdan et al., 2016; Gupta and Jawanda, 2015).

Classic Histopathological Features

The histopathological diagnosis of OLP is reliant upon a combination of epithelial and subepithelial changes that go toward making up a classic microscopic pattern. A very early and characteristic feature is represented by band-like lymphocytic infiltrates in the superficial lamina

propria, besides the basement membrane of epithelium. Infiltrates are primarily composed of T lymphocytes; these cells play a central role in the pathogenesis of the disease (Kramer et al., 1978; Farhi & Dupin, 2010).

There is also hydrogen degeneration of the basal cell layer, basically due to immune-mediated cytotoxicity attacking the basal keratinocytes. It usually forms apoptotic keratinocytes. These are seen as eosinophilic round bodies and termed Civatte bodies or colloid bodies. Basal cell liquefaction results in the production of colloid bodies which are believed to be the remnants of necrotic basal cells (Lavanya et al., 2011).

There is seen irregular thickening of the stratum spinosum (acanthosis) with jagged epidermal ridges emphasized as a feature in classic descriptions of OLP. It may show hyperkeratosis or parakeratosis that might explain reticular or plaque-like clinical presentation. Epithelial architectural changes prove altered turnover of keratinocytes due to chronic irritation plus immune-mediated damage (Gupta & Jawanda, 2015).

Diversity of Histological Features

Though classic features are strong in evidence, the histopathology of OLP may vary concerning clinical subtype, chronicity of the disease, and host response. In atrophic OLP, the epithelium becomes thin, reduced, and sometimes totally atrophic epidermal ridges (Mollaoglu, 2000). This strain manifests more clinically with lesions that are erythematous and painful. In comparing the two diseases, it is noted that hyperkeratotic OLP comprises thickening of the epithelium with marked hyperkeratosis or parakeratosis which corresponds to reticular or plaque lesions observed clinically. Erosive or ulcerative types present destruction of the epithelium and ulceration within the usual subepithelial lymphocytic zone-with fibrin laying often and mixed inflammatory cell infiltrate wherein neutrophils are present. These varying findings must be interpreted with caution since they may appear similar to other mucosal diseases (van der Meij & van der Waal, 2003).

Basement Membrane Zone Alterations

Basement membrane zone alterations are some of the major histopathological changes in OLP. At light microscopy, one may describe the BMZ as being disrupted or obscured, due to degeneration of the basal cells and also because it is masked by inflammatory infiltrate. Fibrinogen and immunoglobulins (by immunofluorescence and immunohistochemistry) further support an immune etiology for the disease. Besides giving a diagnosis, these facts reiterate the pathologic role of autoimmunity in epithelial-connective tissue interaction(Scully & Carrozzo, 2008).

Immunohistochemical and Molecular Insights

Immunohistochemistry may be applied as frequently as histopathology to even better appreciate the cellular compositions and mechanisms of diseases. The inflammatory infiltrate in lesions of OLP is comprised predominantly by CD8+ cytotoxic T cells with limited support provided by CD4+ helper T lymphocytes. Proinflammatory cytokines wherein tumor necrosis factor-α (TNF-α) cytokine, IL-6, and interleukin (IL)-2 most frequently expressed increased levels in these tissue samples have correlated with activity of disease (Ghallab, 2017).

The expression of keratinocyte apoptosis markers, Fas ligand (FasL) and p53 in OLP lesions indicates the possible involvement of immune-induced apoptosis in the pathogenesis of cancer (Zhou et al., 2009Increased expression of adhesion molecules, and mainly intercellular adhesion molecule-1 (ICAM-1), helps T cell movement and holding at the epithelial interface thus helping in chronic inflammatory state. These molecular changes are visualized by immunostaining in tissue sections that have extended the diagnostic and prognostic significance of traditional microscopic features (Carrozzo & Thorpe, 2009).

Differential Diagnosis

Oral lichenoid lesions and a great number of other mucosal diseases with overlapping characteristics come into differential diagnosis with OLP. Oral lichenoid lesions resulting from dental restorations, drugs, or even systemic diseases usually look like OLP but present a more diffuse perivascular inflammatory infiltrate rather than the characteristic ribbon-like structure seen in true cases of OLP (van der Meij et al., 2009). Similarly, lupus erythematosus can reveal basal cell degeneration and subepithelial lymphocytes but generally also has deeper perivascular inflammation accompanied by deposits of villous or granular immunoglobulins along the BMZ. Other conditions to be included in the differential diagnosis are leukoplakia, erythroplakia, graft-versus-host disease, and chronic candidiasis. Therefore, the

histopathological report must be correlated with clinical features for making a final diagnosis. (Eisenberg, 2000).

Malignant Potential and Dysplastic Features

A key factor in the histopathology of oral lichen planus is that OLP may transform into malignancy. The actual rate at which this transformation occurs is still a matter of speculation but OLP has been described as a premalignant disorder by numerous studies. Some cases of OLP, particularly its atrophic and erosive forms may be malignant. In pediatric, loss of maturation, epithelial nuclear pleomorphism, hyperchromatism, and mitotic abnormality, can be seen. However, the presence of epithelial dysplasia in combination with classic OLP creates a diagnostic dilemma because it makes more sense to think of lichenoid dysplasia rather than classic OLP (González-Moles et al., 2021).

Molecular markers further underscore the malignant potential of OLP. Overexpression of p53, Ki-67, and other proliferation markers in histological sections supports the hypothesis that chronic inflammation and epithelial damage contribute to the carcinogenesis of OLP. Therefore, careful long-term surveillance of patients is warranted, particularly in cases with atypical or dysplastic features (Zhou et al., 2009).

Conclusion:

From a histopathological perspective, oral lichen planus (OLP) is marked by epithelial hyperkeratosis, degeneration of basal cells, and a dense, band-shaped lymphocytic infiltrate along the epithelial–connective tissue junction, all of which are essential diagnostic indicators. Although its malignant potential is relatively low, the possibility of transformation highlights the need for careful monitoring. This paper reviews current evidence concerning the clinical characteristics, underlying mechanisms, and microscopic features of OLP, with the objective of enhancing knowledge, facilitating early recognition, and guiding appropriate therapeutic strategies for this challenging condition.

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