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Clinical And Morphological Diagnostic Criteria Of Oral Lichen Planus

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Abstract: Oral lichen planus (OLP) is a chronic immune-mediated disease of the oral mucosa with diverse clinical manifestations and variable severity. This study aimed to assess the correlation between clinical forms of OLP and the expression of immunohistochemical markers Ki-67, p53, CD4, CD8, and CD1a.

Twenty-five patients with clinically and histologically confirmed OLP were examined. Clinical findings were correlated with histological and immunohistochemical parameters. Severe clinical forms, particularly the erosive-ulcerative variant, demonstrated significantly increased expression of Ki-67 and p53, indicating enhanced epithelial proliferation and cellular stress. Immunohistochemical analysis revealed a predominance of CD8-positive cytotoxic T lymphocytes and increased CD1a-positive antigen-presenting cells in clinically aggressive forms of OLP.

The results demonstrate a significant association between clinical severity of OLP and immunohistochemical marker expression, supporting the value of combined clinical and immunohistochemical assessment for improved diagnosis and evaluation of disease activity.

Keywords: Oral lichen planus; immunohistochemistry; Ki-67; p53; CD4; CD8; CD1a; oral mucosa; inflammatory infiltrate; premalignant disorders.

Introduction: Oral lichen planus (OLP) is a chronic inflammatory disease of the oral mucosa with an immune-mediated pathogenesis, characterized by a polymorphic clinical presentation and a prolonged recurrent course. According to epidemiological data, the prevalence of OLP ranges from 0.5% to 2% of the general population, with a predominance among middle-aged and elderly individuals and a higher incidence in women. Despite extensive research, the diagnosis of OLP remains a significant clinical challenge due to the diversity of clinical forms and the similarity of its manifestations to other inflammatory and potentially malignant disorders of the oral mucosa.

Clinically, OLP presents in several forms, including typical (reticular), hyperkeratotic, erosive-ulcerative and bullous variants. The typical form is the most common and often asymptomatic, whereas erosive-ulcerative and atrophic forms are associated with pronounced discomfort, pain, and impaired quality of life. The variability of clinical manifestations necessitates careful differential diagnosis, particularly with leukoplakia, oral lichenoid reactions, pemphigoid, pemphigus vulgaris, and early stages of oral squamous cell carcinoma.

Histopathological examination plays a crucial role in confirming the diagnosis of OLP. Typical morphological features include hyperkeratosis or parakeratosis, degeneration of the basal cell layer, a band-like lymphocytic infiltrate in the subepithelial connective tissue, and the presence of Civatte bodies. However, routine histological evaluation alone may be insufficient to fully characterize disease activity, severity, and potential malignant transformation risk.

In recent years, immunohistochemical (IHC) analysis has gained increasing importance as an adjunctive diagnostic tool in the assessment of OLP. The

evaluation of cellular proliferation, apoptosis regulation, and immune response using specific molecular markers allows for a deeper understanding of the pathogenetic mechanisms underlying different clinical forms of the disease. Markers such as Ki-67, p53, Bcl-2, CD4, CD8, and CD1a provide valuable information regarding epithelial turnover, apoptotic imbalance, and the composition of the inflammatory infiltrate.

The integration of clinical findings with morphological and immunohistochemical criteria may significantly improve diagnostic accuracy, enable better differentiation between clinical forms of OLP, and contribute to the assessment of disease progression and potential malignant transformation. Therefore, a comprehensive approach combining clinical examination, histopathology, and immunohistochemical analysis is essential for the modern diagnosis and management of oral lichen planus.

METHODS

Study design and patients

The present study was designed as an observational clinical and morphological investigation. A total of 25 patients diagnosed with oral lichen planus (OLP) were included in the study. The study group consisted of 17 women (68%) and 8 men (32%). The mean age of the patients was 51.4 ± 8.6 years. The average duration of the disease at the time of examination was 3.2 ± 1.7 years.

The diagnosis of OLP was established based on clinical presentation and confirmed by histopathological examination. Patients with systemic autoimmune diseases, malignant neoplasms of the oral cavity, or a history of immunosuppressive therapy were excluded from the study.

Clinical examination

All patients underwent a comprehensive clinical examination of the oral mucosa. The assessment included evaluation of lesion localization, extent, color, surface characteristics, and the presence of subjective symptoms such as pain or burning sensation. Based on clinical findings, patients were classified according to the clinical forms of oral lichen planus. Special attention was paid to distinguishing between reticular, atrophic, and erosive forms due to their different clinical behavior and prognostic significance.

Histological examination

Incisional biopsies were obtained from representative lesions of the oral mucosa under local anesthesia. Tissue samples were fixed in 10% neutral buffered formalin, routinely processed, and embedded in paraffin. Sections of 4–5 µm thickness were stained with hematoxylin and eosin for histopathological evaluation. Morphological assessment focused on epithelial keratinization patterns, basal cell layer integrity, presence of Civatte bodies, and the characteristics of the subepithelial inflammatory infiltrate.

Immunohistochemical analysis

Immunohistochemical examination was performed on paraffin-embedded tissue sections using standard protocols. The expression of immune and proliferation-related markers, including CD1a, CD4, CD8, Ki-67, and p53, was evaluated. Immunoreactivity was assessed in epithelial cells and inflammatory infiltrates. The intensity and distribution of positive staining were analyzed to characterize immune response patterns, proliferative activity, and potential alterations in epithelial homeostasis associated with different clinical forms of OLP.

Statistical analysis

Statistical processing of the obtained data was carried out using standard methods of descriptive statistics. Quantitative variables were expressed as mean values with standard deviations (mean ± SD). Comparative analysis was performed to identify associations between clinical forms of OLP and histological as well as immunohistochemical findings. A p-value of < 0.05 was considered statistically significant.

RESULTS

Based on the patient survey, subjective symptoms associated with oral lichen planus were frequently reported. Pain of the oral mucosa was noted in 29% of patients, which is considered a significant symptom in the context of potentially premalignant disorders of the oral mucosa. A burning sensation was reported by 24% of patients. Additionally, 32.7% of patients experienced discomfort during speech and mastication. A feeling of tightness of the oral mucosa or xerostomia was observed in 12.8% of cases.

Analysis of predisposing factors revealed that disorders of the nervous system were the most common contributing condition, identified in 25% of patients. Galvanic phenomena were detected in 23% of cases, while tobacco smoking was reported by 22% of patients. Alcohol consumption was noted in 15% of cases, and concomitant somatic diseases were present in 10% of patients. Less frequently identified factors included malocclusion (3%), overhanging restoration margins (1%), and chronic mechanical trauma of the oral mucosa (1%).

The localization of lesions predominantly involved the buccal mucosa, which was affected in 60% of patients.

Lesions of the tongue and gingiva were observed in 16% of cases each, while involvement of the lips was noted in 8% of patients.

Clinical classification of oral lichen planus revealed that the typical (reticular) form was the most prevalent, accounting for 52% of cases. The erosive-ulcerative form was diagnosed in 20% of patients, the hyperkeratotic form in 24%, and the bullous form in 4% of cases.

Histological examination demonstrated characteristic morphological features consistent with oral lichen planus. Saw-tooth-shaped epithelial rete ridges were observed, representing one of the hallmark histopathological signs of the disease. These changes reflect epithelial dystrophy and architectural distortion associated with chronic inflammatory processes. A dense band-like lymphocytic infiltrate was present beneath the epithelium, accompanied by vacuolar (hydropic) degeneration of the basal cell layer. Damage to basal epithelial cells with pronounced vacuolization indicated disruption of basal layer integrity and ongoing immune-mediated epithelial injury.

Immunohistochemical findings

Immunohistochemical analysis revealed distinct differences in immune cell composition and epithelial activity depending on the clinical form of oral lichen planus. In patients with the erosive-ulcerative form, a marked increase in immune cell infiltration was observed. The expression of CD1a-positive cells reached 31.4%, while CD8-positive cytotoxic T lymphocytes accounted for 72.5% and CD4-positive T helper cells for 68.4%. In contrast, the typical (reticular) form demonstrated significantly lower values, with CD1a expression of 18.9%, CD8 expression of 39.7%, and CD4 expression of 46.2%.

Quantitative assessment of immune cells indicated a pronounced predominance of CD8-positive lymphocytes in the erosive-ulcerative form, reflecting activation of a cytotoxic immune response. The increased number of CD1a-positive cells suggests enhanced antigen-presenting activity, which is consistent with ongoing immune-mediated epithelial damage. Additionally, increased expression of pro-inflammatory cytokines, including TNF-α and IL-6, further supports the presence of an active inflammatory and immune response in more severe clinical forms of the disease.

Evaluation of epithelial proliferative activity demonstrated a gradual and statistically significant increase in Ki-67 expression across different clinical forms of OLP. In normal oral mucosa, Ki-67 expression was limited to 4.3%. In patients with the typical form of OLP, Ki-67 expression increased to 12.4%, while the hyperkeratotic form showed a further rise to 18.6%. The highest proliferative activity was observed in the erosive-ulcerative form, with Ki-67 expression reaching 27.3%, indicating intense epithelial turnover and regenerative stress.

A similar pattern was observed for p53 expression. In the typical form of OLP, p53 expression was relatively low (8.1%), whereas higher values were recorded in the hyperkeratotic form (14.7%). The erosive-ulcerative form demonstrated the highest p53 expression (22.9%), suggesting activation of cellular stress response mechanisms and potential dysplastic alterations within the epithelium.

Based on the obtained data, a set of combined clinical, morphological, and immunohistochemical diagnostic criteria was proposed. It was established that pronounced proliferative activity (Ki-67 > 20%), elevated p53 expression (> 15%), and a shift in the CD4/CD8 ratio toward cytotoxic CD8-positive cells are characteristic markers of unfavorable clinical forms of oral lichen planus. These criteria allow for more precise diagnosis, assessment of disease activity, and identification of patients at increased risk for progressive or complicated disease course.

Correlation analysis demonstrated significant associations between clinical severity and immunohistochemical parameters. Elevated Ki-67 and p53 expression levels, along with increased CD8-positive cell infiltration, correlated with more severe clinical manifestations, particularly in the erosive-ulcerative form. These findings indicate that the integration of clinical evaluation with immunohistochemical profiling provides the most accurate assessment of pathological activity and potential risk of adverse disease progression.

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