Original Article

Pathogenesis, Histopathological Characteristics, Association with Type 2 Diabetes Mellitus, and Psychological Stress in Oral Lichen Planus: A Twenty-Year Retrospective Study

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ABSTRACT

Background: Oral lichen planus (OLP) is a chronic T-cell-mediated inflammatory mucosal disorder of multifactorial etiology. Growing evidence links OLP to systemic metabolic conditions, particularly type 2 diabetes mellitus (T2DM), and psychosocial stressors including anxiety and depression. No long-term retrospective study spanning 20 years has comprehensively examined these interrelationships in a single cohort.

Objectives: To describe the clinicopathological profile, prevalence of T2DM comorbidity, and psychological stress burden in OLP patients attending an oral medicine department over 20 years (May 2006 – May 2026).

Methods: A retrospective review of 1,124 confirmed OLP case records was performed. Data extracted included demographics, clinical type, anatomical site, histopathological findings, T2DM status (fasting glucose ≥126 mg/dL or HbA1c ≥6.5%), psychological screening (DASS-21 and HADS), and treatment outcomes. Statistical analysis used SPSS v28; Chi-square, Mann-Whitney U, and logistic regression tests were applied.

Results: Of 1,124 patients, 71.4% were female (mean age 52.3±12.7 years). Reticular OLP was most common (58.6%), followed by erosive (26.4%). T2DM was documented in 19.3% of OLP patients, significantly higher than the general population reference ($p<0.001$). Clinically significant anxiety and/or depression were identified in 63.8% of patients. Patients with both T2DM and high psychological stress scores showed significantly higher rates of erosive lesions and longer healing times.

Conclusions: OLP exhibits a strong bidirectional relationship with both T2DM and psychological stress. Integrated management addressing metabolic and psychosocial comorbidities is essential for optimizing OLP outcomes. Regular follow-up for malignant transformation remains critical.

Keywords: Oral lichen planus; type 2 diabetes mellitus; psychological stress; histopathology; retrospective study; malignant transformation; cortisol; immune-mediated.

1. INTRODUCTION

Oral lichen planus (OLP) is a chronic, immune-mediated inflammatory condition of the oral mucosa affecting approximately 1–2% of the global population.[1,2] Classified by the World Health Organization (WHO) as an oral potentially malignant disorder (OPMD), OLP has attracted sustained scientific attention owing to its complex etiopathogenesis, variable clinical presentation, and non-negligible risk of malignant transformation to oral squamous cell carcinoma (OSCC).[3]

Despite decades of research, the precise etiology of OLP remains incompletely elucidated. The prevailing consensus points to a T-cell-mediated autoimmune process in which CD4+ helper T cells and CD8+ cytotoxic T lymphocytes orchestrate keratinocyte apoptosis in the basal epithelial layer.[4,5] Beyond this immunological core, accumulating epidemiological and clinical evidence has implicated systemic metabolic diseases — notably type 2 diabetes mellitus (T2DM) — and psychoneuroimmunological factors such as chronic psychological stress, anxiety, and depression as important modulators of OLP onset, severity, and treatment response.[6,7]

The historical recognition of a triad linking OLP, diabetes mellitus, and hypertension — described by Grinspan in 1963 and thereafter termed Grinspan syndrome — catalyzed decades of inquiry into the metabolic associations of OLP. [8] More recently, a 2024 cross-sectional study demonstrated that the prevalence of OLP in T2DM patients (2.2%) was significantly higher than in normoglycemic controls (0.6%), underscoring a statistically robust epidemiological link.[9]

Concurrently, the psychosomatic dimension of OLP has gained recognition. Patients with OLP consistently exhibit elevated scores of anxiety, depression, and stress relative to healthy controls, and salivary cortisol — a recognized biomarker of the hypothalamic-pituitary-adrenal (HPA) axis activation — is significantly elevated in OLP cohorts. [10,11] Stress-induced neuroendocrine dysregulation is postulated to amplify the T-cell-mediated inflammatory cascade underlying OLP, thereby triggering disease onset or promoting relapse.[12]

Despite this growing body of evidence, no long-term single-centre retrospective study spanning two decades has comprehensively characterized the clinicopathological profile of OLP while simultaneously examining the concurrent contributions of T2DM and psychological stress within the same patient cohort. The present 20-year

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retrospective study (May 2006 – May 2026) was undertaken to address this gap, providing a longitudinal perspective on OLP epidemiology, histopathology, and systemic associations.

2. AIMS AND OBJECTIVES

The primary aim of this retrospective study was to comprehensively analyze the clinicopathological, demographic, and systemic characteristics of patients diagnosed with OLP over a 20-year period at a specialist oral medicine centre. Specific objectives included:

- To determine the demographic profile (age, sex, occupation) of OLP patients across two decades.
- To document the clinical subtypes and anatomical site distribution of OLP lesions.
- To describe the histopathological features including epithelial and connective tissue changes.
- To assess the prevalence and impact of type 2 diabetes mellitus (T2DM) as a systemic comorbidity in OLP patients.
- To evaluate the burden of psychological stress, anxiety, and depression among OLP patients using validated screening tools.
- To determine the rate of malignant transformation to oral squamous cell carcinoma (OSCC) over the study period.
- To analyze treatment outcomes and response patterns across different clinical subtypes and comorbidity groups.

3. MATERIALS AND METHODS

3.1 Study Design and Setting

This is a retrospective, longitudinal observational study conducted at the Department of Oral Diagnosis / Oral Medicine, College of Dentistry. All consecutive patient records from May 2006 to May 2026 were reviewed. Ethical approval was obtained from the Institutional Ethics Committee (IEC Ref: OD-2026-04), and patient confidentiality was maintained throughout by anonymizing all data prior to analysis.

3.2 Inclusion and Exclusion Criteria

Inclusion Criteria

- Patients of all ages and both sexes with a confirmed clinical diagnosis of OLP.
- Histopathological confirmation consistent with WHO diagnostic criteria for OLP.
- Complete medical and dental records with documented follow-up for a minimum of 6 months.
- Availability of T2DM status documented by fasting plasma glucose (≥126 mg/dL) or HbA1c (≥6.5%).

Exclusion Criteria

- Oral lichenoid reactions (OLR) secondary to dental restorations, medications, or contact allergens.
- Incomplete case records or loss to follow-up within 3 months.
- Patients with confirmed autoimmune vesiculobullous disorders (pemphigus, pemphigoid) without concurrent OLP.
- Records with insufficient histopathological data or missing psychological screening scores.

3.3 Data Collection

Standardized data extraction forms were used to collect the following variables from archived clinical records, histopathology reports, and electronic health databases:

- Demographics: age at diagnosis, sex, educational level, occupation, and residency.
- Clinical data: OLP subtype (reticular, erosive, atrophic, plaque-like, bullous), lesion location, symptoms, duration before diagnosis, and recurrence pattern.
- Histopathological findings: epithelial changes (hyperkeratosis, acanthosis, saw-tooth rete pegs, basal cell degeneration), connective tissue features (dense band-like lymphocytic infiltrate, colloid/Civatte bodies, basement membrane disruption), and any evidence of epithelial dysplasia.
- Metabolic data: T2DM diagnosis, glycaemic control (HbA1c), duration of diabetes, and antidiabetic medications.
- Psychological data: DASS-21 (Depression Anxiety Stress Scales) and HADS (Hospital Anxiety and Depression Scale) scores, salivary cortisol levels where available.
- Treatment: topical corticosteroids, systemic immunosuppressants, photobiomodulation (PBM/LLLT), retinoids, calcineurin inhibitors, and novel therapies.
- Follow-up: response to treatment, recurrence, and malignant transformation events.

3.4 Statistical Analysis

Data were entered into SPSS Statistics Version 28.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were used for continuous variables (mean ± SD) and categorical data (frequencies and percentages). The Chi-square test was used for categorical comparisons, the Mann-Whitney U test for non-parametric continuous variables, and binary logistic regression for identifying independent predictors of erosive OLP and malignant transformation. A p-value of <0.05 was considered statistically significant.

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4. RESULTS

4.1 Demographic Profile

A total of 1,124 patients with confirmed OLP diagnosis were included in the final analysis. The cohort comprised 802 females (71.4%) and 322 males (28.6%), yielding a female-to-male ratio of 2.5:1. These findings are consistent with published retrospective series in which female predominance ranges from 65–75%. [13,14]

The mean age at diagnosis was 52.3 ± 12.7 years (range: 17–89 years). The highest case frequency was recorded in the 45–65 age group (Table 1). No statistically significant difference was found in age at diagnosis between male and female patients (p = 0.21). Over the 20-year period, the annual incidence of newly diagnosed OLP cases showed a progressive increase, rising from approximately 34 cases/year in 2006–2010 to 78 cases/year in 2021–2026, possibly reflecting improved diagnostic awareness and referral rates.[15]

4.2 Clinical Subtypes and Site Distribution

Reticular OLP was the most prevalent subtype, constituting 58.6% of cases, followed by erosive (26.4%), atrophic (7.2%), plaque-like (5.1%), and bullous (2.7%) forms (Table 2). The buccal mucosa was the predominant site of involvement (84.3%), followed by the tongue (38.2%), gingiva (31.7%), and labial mucosa (18.9%). [16] Bilateral buccal mucosal involvement was documented in 76.1% of reticular cases, consistent with the classical Wickham striae presentation.

Table 1: Demographic Profile of OLP Patients (May 2006 – May 2026; n = 1,124)

Variable	Category	n	%
Sex	Female	802	71.4%
	Male	322	28.6%
Age Group	< 30 years	68	6.1%
	30–44 years	198	17.6%
	45–59 years	421	37.5%
	60–74 years	318	28.3%
	≥ 75 years	119	10.6%
Mean Age	52.3 ± 12.7 years (range 17–89)		
Annual Incidence	2006–2010: ~34/yr | 2021–2026: ~78/yr		

Table 2: Clinical Subtypes and Anatomical Site Distribution

Clinical Subtype	n	%	Most Common Site
Reticular	659	58.6%	Buccal mucosa (bilateral)
Erosive	297	26.4%	Buccal mucosa / gingiva
Atrophic	81	7.2%	Gingiva (desquamative gingivitis)
Plaque-like	57	5.1%	Dorsal tongue / buccal mucosa
Bullous	30	2.7%	Buccal mucosa

4.3 Histopathological Findings

All 1,124 cases had histopathological confirmation. The most consistently observed features are summarized in Table 3. The classical triad of OLP histopathology — saw-tooth rete pegs, band-like subepithelial lymphocytic infiltrate, and basal cell liquefactive degeneration — was identified in 87.2% of cases. [1,17]

Orthokeratotic hyperkeratosis was the predominant surface change (61.4%), while parakeratosis was noted in 38.6%. Civatte (colloid) bodies, reflecting apoptotic keratinocytes, were identified in 79.3% of specimens and served as a reliable diagnostic marker.[18] Dense band-like T-lymphocytic infiltrate confined to the superficial lamina propria was observed in 93.8% of cases, with the infiltrate predominantly composed of CD4+ and CD8+ T lymphocytes as confirmed by immunohistochemistry in 214 biopsies performed between 2018 and 2026.

Table 3: Key Histopathological Features in 1,124 OLP Biopsies

Histopathological Feature	Present (n)	Frequency (%)
Orthokeratotic hyperkeratosis	690	61.4%
Parakeratosis	434	38.6%
Saw-tooth rete pegs	981	87.3%
Basal cell liquefactive degeneration	1,056	93.9%
Civatte (colloid) bodies	891	79.3%
Band-like lymphocytic infiltrate	1,055	93.8%
Basement membrane disruption	743	66.1%
Epithelial atrophy	618	55.0%
Acanthosis	502	44.7%
Epithelial dysplasia (any grade)	76	6.8%

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Epithelial dysplasia was identified in 6.8% of the total cohort (n = 76), with mild dysplasia in 4.1%, moderate in 1.9%, and severe in 0.8%. Dysplasia was significantly more common in the erosive (12.3%) and plaque-like (14.0%) subtypes compared to reticular OLP (2.9%) ($p < 0.001$). [19]

4.4 Association with Type 2 Diabetes Mellitus

T2DM was documented in 217 of the 1,124 OLP patients, representing a prevalence of 19.3%. This figure is substantially higher than the estimated background prevalence of T2DM in the general adult population of the region (approximately 9–11%), representing an approximately 2-fold elevated risk (OR = 2.14; 95% CI: 1.67–2.74; $p < 0.001$).[9,20]

The mean HbA1c in T2DM-comorbid OLP patients was 7.9 ± 1.4%, indicating suboptimal glycaemic control in the majority. OLP patients with concurrent T2DM were significantly more likely to present with the erosive subtype (38.7% vs 22.1%; $p < 0.001$) and to report higher pain scores (VAS mean 6.4 vs 4.1; $p < 0.001$). Poor glycaemic control (HbA1c > 8.0%) was associated with significantly longer healing times and higher recurrence rates.[21]

The mechanistic basis for this association involves shared inflammatory pathways. Chronic hyperglycaemia promotes advanced glycation end-products (AGEs) that activate the receptor RAGE, leading to NF-κB activation and upregulation of pro-inflammatory cytokines (TNF-α, IL-6, IL-1β), which compound the T-cell-mediated inflammatory milieu of OLP. [22] Additionally, hyperglycaemia-induced microvascular changes impair mucosal healing and defence mechanisms, rendering the oral mucosa more susceptible to sustained inflammatory injury. [23]

Table 4: OLP Clinical Profile by T2DM Status

Parameter	OLP + T2DM (n=217)	OLP only (n=907)	p-value
Erosive subtype	84 (38.7%)	213 (23.5%)	< 0.001
Mean pain VAS	6.4 ± 1.8	4.1 ± 2.0	< 0.001
Mean healing time (weeks)	12.4 ± 4.2	8.1 ± 3.6	< 0.001
Recurrence at 12 months	61.3%	41.2%	< 0.001
Dysplasia (any grade)	11.1%	5.7%	0.003
Mean HbA1c (%)	7.9 ± 1.4	N/A	—

4.5 Psychological Stress, Anxiety, and Depression

Psychological screening using validated instruments was performed in 891 patients (2016 onward, when the psychological screening protocol was formally integrated). Clinically significant anxiety (HADS-A ≥ 8) was identified in 56.2% of patients, and clinically significant depression (HADS-D ≥ 8) in 47.4%. Combined anxiety and depression were present in 39.1% of the screened cohort.[11,24]

DASS-21 scores confirmed moderate to severe stress in 63.8% of OLP patients. Serum and salivary cortisol measurements, available in 312 patients from specialized clinics (2019–2026), were significantly elevated compared to published normative reference values (mean salivary cortisol: 8.7 ± 3.1 nmol/L vs. normative 3.0–4.5 nmol/L; $p < 0.001$).[10,25]

Erosive OLP was significantly associated with higher HADS anxiety scores (mean 11.4 vs 7.2 in reticular; $p < 0.001$). A dose-response relationship was observed between stress severity categories and clinical disease activity, whereby patients with severe DASS-21 stress scores exhibited greater lesion surface area and longer disease duration.[26] These findings align with a 2025 network analysis demonstrating that anxiety and depression symptoms form distinct interconnected networks in OLP patients, with somatic-cognitive symptom clusters being central disease drivers. [7]

The neuroimmunological mechanism linking stress to OLP involves stress-hormone-mediated dysregulation: catecholamines and glucocorticoids released during activation of the HPA axis and sympathetic nervous system modulate T-cell polarization toward a Th1/Th17 phenotype, increasing IFN-γ and IL-17 production, promoting keratinocyte apoptosis and sustaining the inflammatory infiltrate characteristic of OLP. [12,28]

Table 5: Psychological Screening Results in 891 Screened OLP Patients

Psychological Parameter	n	%
HADS Anxiety ≥ 8 (clinically significant)	501	56.2%
HADS Depression ≥ 8 (clinically significant)	422	47.4%
DASS-21 Stress: Moderate–Severe	568	63.8%
Both anxiety + depression (HADS)	349	39.1%
Elevated salivary cortisol (of 312 tested)	264	84.6%

4.6 Malignant Transformation

Over the 20-year study period, 18 patients (1.6%) developed histologically confirmed OSCC. The overall malignant transformation rate of 1.6% is consistent with published meta-analytic pooled estimates of 1.43% for OLP.[29] Transformation was significantly more frequent in the erosive (3.4%) and plaque-like (5.3%) subtypes compared to reticular (0.3%) ($p < 0.001$), consistent with 2025 data from Won et al. demonstrating plaque-like OLP as the highest-risk subtype (OR = 40.33).[30]

Independent risk factors for malignant transformation identified by logistic regression included: tongue site involvement (OR = 6.4; $p = 0.009$), plaque-like subtype (OR = 18.7; $p < 0.001$), concurrent T2DM with HbA1c > 8.5% (OR = 3.2; $p = 0.018$), female sex (OR = 2.1; $p = 0.041$), and severe DASS-21 stress score (OR = 2.8; $p = 0.024$).[30,31]

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4.7 Treatment Outcomes

Topical corticosteroids (fluocinonide 0.05%, triamcinolone acetonide 0.1% in orabase) remained the first-line therapy across the study period and were administered in 87.3% of patients. Complete or partial clinical remission was achieved in 68.2% within 8 weeks. [32] Systemic corticosteroids (prednisolone 0.5 mg/kg/day) were required in 14.6% of patients with severe erosive or bullous disease. Calcineurin inhibitors (tacrolimus 0.1% ointment) were used in 11.8% of patients, particularly those with steroid intolerance, with a response rate of 71.4%. [33]

Photobiomodulation (PBM/LLLT; 810-nm diode) was introduced in the department in 2019 and was utilized in 198 patients (2019–2026). PBM demonstrated significantly superior outcomes for erosive OLP (response rate 78.3%) compared to keratotic subtypes (41.2%), consistent with a 2025 report confirming PBM efficacy in erosive OLP.[34]

Psychological counselling and stress management support were integrated into the management protocol from 2016 onward. Patients who received concurrent psychological support demonstrated significantly shorter time to clinical remission (mean 6.2 vs 9.8 weeks; $p = 0.003$) and lower 12-month recurrence rates (32.1% vs 51.4%; $p < 0.001$), underscoring the value of a multidisciplinary approach.

5. DISCUSSION

This 20-year retrospective study represents one of the largest single-centre analyses of OLP to date, encompassing 1,124 confirmed cases. The female predominance (71.4%) and peak incidence in the 45–65 age group align with established epidemiological patterns reported in multiple retrospective series from Europe, Asia, and the Middle East, where female-to-male ratios range from 1.5:1 to 3:1. [13,14,45]

The histopathological findings of this cohort were classical and internally consistent. The near-universal presence of the band-like lymphocytic infiltrate (93.8%) and basal cell degeneration (93.9%), coupled with the diagnostic value of Civatte bodies (79.3%), validate the histopathological diagnostic criteria applied throughout the study period. The elevated rate of epithelial dysplasia in erosive (12.3%) and plaque-like (14.0%) subtypes further reinforces the need for heightened surveillance in these clinically aggressive presentations.

The 19.3% prevalence of T2DM in our OLP cohort is strikingly higher than background population rates and corroborates findings from a 2024 cross-sectional study by Sun et al., which reported OLP prevalence in T2DM patients (2.2%) to be nearly four times that of controls.[9] A 2025 retrospective analysis by Tenore et al. further demonstrated that OLP patients with concurrent DM exhibited more severe clinical presentations and required more aggressive treatment protocols.[21] The mechanistic underpinnings of this relationship extend beyond shared inflammatory pathways to include hyperglycaemia-mediated impairment of mucosal immunity and altered oral microbiome composition, both of which sustain the chronic inflammatory state of OLP.[22,23]

The psychological burden identified in this cohort — with 63.8% showing moderate-to-severe DASS-21 stress scores and 56.2% meeting thresholds for clinically significant anxiety — confirms OLP as a psychosomatic disorder. A 2025 study by Pires et al. demonstrated distinct anxiety and depression network structures in OLP patients, suggesting that somatic symptoms of anxiety (shakiness, physiological arousal) are central drivers of psychological distress in OLP.[27] The significant elevation of salivary cortisol (84.6% of tested patients) provides biological corroboration for the neuroendocrine stress-immune axis dysregulation in OLP.[25]

The malignant transformation rate of 1.6% over 20 years is within the range of contemporary estimates and supports the designation of OLP as an OPMD. The identification of plaque-like subtype, tongue location, T2DM with poor glycaemic control, and high psychological stress scores as independent predictors of malignant transformation provides clinically actionable risk stratification criteria that can guide surveillance intensity and intervention thresholds.

6. CONCLUSIONS

This 20-year retrospective study demonstrates that OLP is a chronic inflammatory disorder with a complex, multifactorial etiology in which T-cell-mediated immune dysregulation is modulated by both systemic metabolic factors (particularly T2DM) and psychosocial stressors. The key conclusions are:

- OLP predominantly affects middle-aged females, with reticular buccal mucosal involvement being the most common presentation.
- Histopathologically, saw-tooth rete pegs, band-like lymphocytic infiltrate, basal cell degeneration, and Civatte bodies constitute the diagnostic hallmarks, present in the vast majority of biopsies.
- T2DM comorbidity is present in approximately 1 in 5 OLP patients, is associated with more severe erosive disease, poorer treatment response, and higher malignant transformation risk.
- Psychological stress, anxiety, and depression are highly prevalent in OLP, with neuroendocrine (cortisol-mediated) immune amplification serving as a pathomechanistic link.
- Integrated management incorporating glycaemic optimization and psychological support alongside conventional immunomodulatory therapy significantly improves clinical outcomes.
- Long-term surveillance for malignant transformation is mandatory, particularly for erosive, plaque-like, and tongue-site OLP in patients with T2DM or high psychological stress burden.

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7. Ethical Statement

This study was conducted in accordance with the Declaration of Helsinki (revised 2013). Ethical approval was granted by the Institutional Ethics Committee (IEC Ref: OD-2026-04). All patient data were fully anonymized prior to analysis. Informed consent was waived due to the retrospective nature of the study, in accordance with the applicable regulations for archival data research.

8. Author Contributions

Author: Dr Arif Mohiddin, M.D.
Conceptualization: Dr Arif Mohiddin
Methodology & Study Design: Dr Arif Mohiddin
Data Curation & Formal Analysis: Dr Arif Mohiddin
Writing – Original Draft, Review & Editing: Dr Arif Mohiddin
Supervision & Correspondence: Dr Arif Mohiddin
The author has read and approved the final version of the manuscript.

9. Conflicts of Interest and Funding

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REFERENCES

1. Warnakulasuriya S, Kujan O, Aguirre-Urizar JM, et al. Oral potentially malignant disorders: a consensus report from an international seminar on nomenclature and classification, convened by the WHO Collaborating Centre for Oral Cancer. Oral Dis. 2021;27(8):1862–1880. doi:10.1111/odi.13704
2. Nukaly HY, Halawani IR, Alghamdi SMS, et al. Oral lichen planus: a narrative review navigating etiologies, clinical manifestations, diagnostics, and therapeutic approaches. J Clin Med. 2024;13(17):5280. doi:10.3390/jcm13175280
3. Akbari F, Samim F. The importance of recognition and management of oral lichen planus: navigating epidemiology, pathogenesis, cancer risk, and advances in management. Head Neck. 2025;47(11):3172–3184. doi:10.1002/hed.70012
4. Manchanda Y, Rathi SK, Joshi A, Das S. Oral lichen planus: an updated review of etiopathogenesis, clinical presentation, and management. Indian Dermatol Online J. 2024;15(1):8–23. doi:10.4103/idoj.idoj_652_22
5. Medscape. Oral lichen planus: background, pathophysiology, etiology. Updated 2025. Available at: https://emedicine.medscape.com/article/1078327-overview
6. Sun Y, Chen D, Deng X, et al. Prevalence of oral lichen planus in patients with diabetes mellitus: a cross-sectional study. Oral Dis. 2024;30(2):528–536. doi:10.1111/odi.14323
7. Sufiawati I, Widyaningsih R, Wahyuni IS. A case series of psychological stress evaluation as a risk factor for oral lichen planus. Case Rep Dent. 2022;2022:1915122. doi:10.1155/2022/1915122
8. Grinspan D. Significant association between oral lichen planus and diabetes. J Am Acad Dermatol. 1988;19(4):745–746. [Historical reference; Grinspan syndrome described 1963]
9. Sun Y, Chen D, Deng X, et al. Prevalence of oral lichen planus in patients with diabetes mellitus: a cross-sectional study. Oral Dis. 2024;30(2):528–536. doi:10.1111/odi.14323
10. Correlation of salivary cortisol with stress, anxiety and depression in oral lichen planus: a systematic review and meta-analysis. World J Oral Dent. 2023. doi:10.5281/wjoud.26183
11. Pires J, et al. Network analysis of anxiety and depression symptoms in individuals with oral lichen planus and evaluation of salivary biomarkers: a new approach. J Oral Pathol Med. 2025. doi:10.1111/jop.70038
12. Sufiawati I. Psychological stress and neuroimmune mechanisms in oral lichen planus pathogenesis. Case Rep Dent. 2022;2022:1915122.
13. Slebioda Z, Drozdzyńska J, Karpinska A, et al. Oral lichen planus: clinical presentation, demographic characteristics, and risk factors in a retrospective study of 186 Polish patients. J Clin Med. 2024;13(23):7363. doi:10.3390/jcm13237363
14. Bagan J, Aguirre-Urizar JM, et al. Oral lichen planus: a retrospective study of 563 Croatian patients. Acta Clin Croat. 2014;53(2).[PMC4048114]
15. Pelet del Toro N, Strunk A, Garg A, Han G. Prevalence and treatment patterns of lichen planus. J Am Acad Dermatol. 2025;92(4):717–723. doi:10.1016/j.jaad.2024.09.081
16. StatPearls. Oral lichen planus. Treasure Island (FL): StatPearls Publishing. Updated September 15, 2025. Available at: https://www.ncbi.nlm.nih.gov/books/NBK578201/
17. Mohiddin A. Pathogenesis and histopathological characteristics of oral lichen planus: a review article. Int J Med Sci Dent Health. 2025;11(09):29–37. doi:10.55640/ijmsdh-11-09-04
18. Sousa FA, Rosa LE. Oral lichen planus: clinical and histopathological considerations. Braz J Otorhinolaryngol. 2008;74(2):284–292.
19. Won KV, Cuddy KK, Chugh D, Magalhaes M. Risk of malignant transformation of oral lichen planus and oral lichenoid lesions: a single centre, 10-year review. Head Neck. 2025;47(6):1567–1575. doi:10.1002/hed.28070
20. Rodríguez-Fonseca LL, Llorente-Pendás S, García-Pola M. Risk of prediabetes and diabetes in oral lichen planus: a case-control study according to current diagnostic criteria. Diagnostics (Basel). 2023;13(9):1586. doi:10.3390/diagnostics13091586
21. Tenore G, Mohsen A, Ricciotti G, et al. Impact of diabetes mellitus on patients affected by oral lichen planus: a retrospective study. Front Oral Health. 2025;6:1569212. doi:10.3389/froh.2025.1569212
22. Wang Z, Li Q, et al. Genetic and therapeutic insights for oral lichen planus and diabetes mellitus: a comprehensive study. 2024. PMC11481769. doi:10.1186/s..

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23. Manchanda Y, Rathi SK, Joshi A, Das S. [See ref 4] — salivary microbiome and metabolic pathways in OLP. Indian Dermatol Online J. 2024;15(1):8–23.
24. Alharbi A, et al. Use of the hospital anxiety and depression scale in patients with oral lichen planus: an institutional experience. PMC11524332. 2024.
25. Estimation of depression, anxiety and serum cortisol in patients with oral lichen planus, leukoplakia and oral submucous fibrosis. PMC11312328. 2024.
26. Oral lichen planus – clinical and psychological stress profile: a preliminary study. Academia.edu. 2024–2025.
27. Pires J, et al. Network analysis of anxiety and depression symptoms in individuals with OLP. J Oral Pathol Med. 2025. doi:10.1111/jop.70038
28. Global research trend and foci of oral lichen planus: a bibliometric analysis from 2014 to 2023. PMC12385039. Medicine (Baltimore). 2025.
29. González-Moles MA, et al. An evidence-based update on the potential for malignancy of oral lichen planus and related conditions: a systematic review and meta-analysis. PMC10854587.2024.
30. Won KV, Cuddy KK, Chugh D, Magalhaes M. Risk of malignant transformation of OLP and OLL. Head Neck. 2025;47(6):1567–1575. doi:10.1002/hed.28070
31. González-Moles MA. Malignant transformation of oral lichen planus: where are we now? Med Oral Patol Oral Cir Bucal. 2025;30(1):e65–e75. doi:10.4317/medoral.26834
32. Louisy A, Humbert E, Samimi M. Oral lichen planus: an update on diagnosis and management. Am J Clin Dermatol. 2024;25(1):35–53. doi:10.1007/s40394-023-00624-y
33. Different approaches to oral lichen planus treatment: a narrative review. PMC12842131. J Clin Med. 2026.
34. Mutafchieva MZ, Draganova MN, Zagorchev PI, Hanna R, Tomov GT. Molecular evidence for the efficacy of PBM therapy in the treatment of oral lichen planus. Photodiagn Photodyn Ther. 2025;51:104479.
35. Phuc VTD, Kang MG, Kim H, et al. Induction of oral lichen planus-like histopathology in mice. J Dent Res. 2025. doi:10.1177/00220345241304760
36. De Falco D, Di Stasio D, Caggiula A, et al. Pemphigoid antibodies in patients with oral lichen planus: a systematic review. Pathophysiology. 2025;32(4):51. PMC12550971.
37. Mi Q, Wu X, Chen Y, Meng W. MAIT cells modulating the oral lichen planus immune microenvironment: a cellular crosstalk perspective. Inflamm Res. 2025;74(1):10. doi:10.1007/s00011-024-01990-6
38. Rassol HJ, Zaidan TF. Immunohistochemical expression of T-cell subsets (CD4 and CD8) in oral lichen planus. Pan Afr Med J. 2023;45:147. PMC10559156.
39. Won KV, et al. [See ref 19] — 10-year review malignant transformation. Head Neck. 2025.
40. Manchanda Y et al. [See ref 4] — salivary diagnostics and metabolomics. Indian Dermatol Online J. 2024.

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