

Oral Cancer Risk in Patients with Oral Lichen Planus: A Narrative Review

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Abstract

Oral lichen planus (OLP) is a chronic immune-mediated inflammatory disorder of the oral mucosa and is widely regarded as an oral potentially malignant disorder. Although the association between OLP and oral squamous cell carcinoma (OSCC) has been investigated in numerous studies, the magnitude of malignant transformation risk remains variable and is influenced by diagnostic criteria, follow-up duration, lesion subtype, epithelial dysplasia, tobacco and alcohol exposure, and geographic differences. This narrative review summarizes current evidence on the relationship between OLP and oral cancer risk using a structured descriptive search of PubMed, Scopus, and Web of Science. Relevant English-language studies addressing OLP, malignant transformation, and OSCC risk were reviewed and synthesized descriptively. The available evidence suggests that patients with OLP may have an increased risk of developing OSCC compared with the general population; however, reported transformation rates differ considerably among studies. Erosive or atrophic lesions, persistent ulceration, tongue involvement, epithelial dysplasia, and conventional carcinogenic exposures may further increase clinical concern. In addition to early diagnosis, long-term surveillance and biopsy of suspicious or non-healing lesions are essential. Treatment should aim to control chronic inflammation while avoiding delayed recognition of dysplastic or malignant changes, particularly in patients receiving prolonged immunosuppressive or immunomodulatory therapy. Overall, OLP requires careful clinical monitoring because its malignant potential is context-dependent rather than uniform. Further well-designed studies with standardized diagnostic criteria and long-term follow-up are needed to clarify the biological mechanisms linking OLP to oral carcinogenesis and to improve risk stratification and prevention strategies.

Keywords: Oral lichen planus- oral squamous cell carcinoma- malignant transformation- oral potentially malignant disorder

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Introduction

Oral lichen planus is a chronic, relapsing, immune-mediated disease of the oral mucosa that is characterized by T-cell-dominant epithelial injury and fluctuating periods of activity and remission [1-7]. The disease most frequently affects middle-aged and older adults and is reported more often in women, although prevalence estimates vary by population, diagnostic criteria, and clinical setting [1, 8, 9]. Clinically, OLP may present as reticular white striae, papules, plaque-like lesions, atrophic erythema, erosions, ulcers, or bullous lesions, and the buccal mucosa, tongue, and gingiva are common sites of involvement [1, 8, 9]. OLP is included among oral potentially malignant disorders because a subset of patients may later develop oral squamous cell carcinoma at an involved or previously involved mucosal site [10-12]. This classification should be interpreted carefully because it means that the condition carries a statistically increased cancer risk, not that every lesion is destined to transform [10, 11]. Reported malignant transformation rates vary widely because studies differ in diagnostic definitions, duration of follow-up, inclusion of oral lichenoid lesions, histopathological thresholds, smoking and alcohol adjustment, and geographic background [13-20]. The long-standing controversy over OLP malignant potential is partly methodological rather than purely biological [16, 19, 21]. Earlier studies sometimes mixed classic OLP with oral lichenoid lesions, lichenoid drug reactions, contact reactions, and lesions with epithelial dysplasia already present at baseline [8, 22-23]. More recent analyses emphasize that strict clinicopathological criteria reduce apparent transformation rates, whereas broader inclusion criteria and longer follow-up often produce higher estimates [16, 18-19]. The clinical importance of this topic is that delayed recognition of dysplasia or early OSCC can occur when persistent red, erosive, ulcerative, or indurated lesions are repeatedly treated as inflammatory OLP without re-biopsy [17, 24, 25]. Tongue involvement, red-type morphology, tobacco use, alcohol consumption, HCV positivity in selected populations, and epithelial dysplasia have been repeatedly discussed as factors that increase clinical concern [16-19, 24-27]. Therefore, the central clinical question is not whether all OLP lesions are premalignant, but how to identify the smaller subgroup of patients who need closer surveillance and earlier biopsy [19, 21, 28]. This narrative review synthesizes evidence on the oral cancer risk of OLP, with emphasis on diagnostic uncertainty, malignant transformation rates, clinically meaningful risk indicators, biomarkers, treatment-related surveillance issues, and practical follow-up strategy [10, 13, 19]. It also distinguishes classic OLP from related lichenoid conditions and summarizes key transformation studies in a comparative table to clarify why reported rates differ across the literature [13-19, 21, 24-29].

Materials and Methods

This article was designed as a narrative review supported by a structured descriptive literature search.

The aim was to summarize and interpret current evidence regarding the association between OLP and the risk of oral cancer, particularly OSCC, rather than to conduct a formal systematic review or meta-analysis. PubMed, Scopus, and Web of Science were searched using combinations of the terms “oral lichen planus,” “oral cancer,” “oral squamous cell carcinoma,” “malignant transformation,” “oral potentially malignant disorder,” “oral epithelial dysplasia,” “risk factors,” “biomarkers,” “treatment,” and “follow-up.” Additional relevant articles were identified by screening reference lists of selected publications. Studies were considered relevant when they discussed OLP, oral lichenoid conditions, malignant transformation rates, clinical or histopathological risk factors, molecular mechanisms, diagnostic methods, surveillance, or treatment-related considerations. Priority was given to peer-reviewed journal articles, cohort studies, clinically relevant observational studies, systematic reviews, meta-analyses, consensus reports, and recent molecular studies directly related to OLP, OPMDs, OSCC, or oral cancer surveillance. Articles focusing primarily on unrelated cancers, nanoparticle-based therapies, non-oral malignancies, or topics not directly connected to OLP and oral cancer risk were excluded from the narrative synthesis.

Risk Factors Associated with Oral Cancer Development in Patients with Oral Lichen Planus

The malignant transformation of OLP into OSCC is best understood as a low-frequency but clinically meaningful event rather than an inevitable disease outcome [13-19]. Across meta-analyses and cohort studies, reported transformation rates generally cluster around 0.4% to 2.6% for classic OLP, but higher rates are seen when oral lichenoid lesions, lichenoid dysplasia, or OLP with epithelial dysplasia are included [13-19]. This variability explains why a single transformation percentage should not be used for all patients without considering diagnostic quality and clinical context [16, 18-19]. Diagnostic classification is one of the strongest determinants of apparent risk [8, 16, 18-19]. Classic OLP is usually defined by compatible bilateral clinical findings and histopathological features such as band-like lymphocytic infiltration and basal cell degeneration without epithelial dysplasia [8, 22]. Oral lichenoid lesions and lichenoid contact reactions may resemble OLP but can have different triggers, including dental materials, drugs, or localized contact factors [1, 8, 23]. Lichenoid dysplasia is especially important because it may be biologically closer to dysplastic OPMD than to classic inflammatory OLP [18-19, 30]. Clinical subtype influences surveillance intensity because erosive, atrophic, ulcerative, and erythematous lesions are more concerning than stable reticular lesions [15, 24-26]. Red-type lesions may indicate more severe epithelial injury and persistent mucosal breakdown, creating repeated cycles of inflammation and repair [17, 24, 26]. Stable reticular OLP can still require monitoring, but persistent erosion or ulceration should lower the threshold for biopsy or repeat biopsy [24-26]. Lesion location also matters because OSCC does not arise

with equal frequency at all oral subsites [10, 11, 31]. Tongue lesions, particularly those on the lateral tongue, have been repeatedly described among transformed cases and are commonly regarded as higher-risk clinical sites [15, 25-26]. Floor-of-mouth lesions and persistent red or mixed red-white lesions also require careful assessment because these sites and appearances are high-risk in the broader oral cancer literature [10, 11, 30]. Epithelial dysplasia is the most important histopathological warning sign, but it also creates diagnostic ambiguity in OLP [8, 19, 30, 32]. Some diagnostic frameworks exclude epithelial dysplasia from classic OLP, whereas other authors acknowledge that dysplasia may develop during follow-up in patients previously diagnosed with OLP [8, 22, 33]. In clinical practice, the presence of dysplasia should always increase surveillance intensity regardless of whether the final label is OLP with dysplasia, lichenoid dysplasia, or another OPMD [10, 19, 30]. Tobacco and alcohol are established oral cancer risk factors and should be evaluated in every patient with OLP [10, 11, 16, 18]. Their independent contribution to OLP-specific transformation is difficult to isolate, but they add carcinogenic exposure to a mucosa already affected by chronic inflammation [16, 18, 27]. Patients with OLP who smoke or drink heavily should receive cessation counseling and closer follow-up, especially when other risk indicators such as tongue involvement, red lesions, dysplasia, or persistent ulceration are present [16, 18, 26]. Hepatitis C virus has been associated with lichen planus and OLP in several regions, but the strength and clinical relevance of this association vary geographically [34-35]. Meta-analytic data support an epidemiological association between lichen planus and HCV infection, and some OLP malignant transformation studies identify HCV seropositivity as a potential risk modifier [16, 18, 34, 35]. HCV should therefore be discussed as a population-dependent cofactor rather than a universal driver of OLP-related oral cancer [34, 35]. Human papillomavirus has been investigated in OLP and oral carcinogenesis, but its role in OLP malignant transformation remains less certain than in oropharyngeal cancer [36]. HPV DNA detection in OLP does not by itself prove causality because viral presence, transcriptional activity, lesion persistence, and malignant progression are different biological questions [36]. For this reason, HPV should be framed as a possible cofactor under investigation, not as a confirmed cause of malignant transformation in classic OLP [36]. Age and sex should be interpreted cautiously because OLP is more common in women and middle-aged or older adults [1, 9, 25]. A greater number of transformed cases among women in some cohorts may reflect the higher prevalence of OLP among women rather than a higher individual transformation probability [17, 25]. Older age may increase clinical concern because malignant progression usually requires time, and older patients may have longer disease duration, accumulated carcinogen exposure, and comorbidities that complicate surveillance [17, 24-25]. Systemic diseases such as diabetes mellitus, chronic liver disease, chronic kidney disease, hypertension,

dyslipidemia, and cardiovascular disease are reported with OLP in some studies, but most are not established direct predictors of OSCC development in classic OLP [37-40]. Diabetes may influence wound healing, candidiasis risk, immune response, and corticosteroid safety, while liver disease may matter because of HCV, bleeding risk, or drug metabolism [35, 37-38]. These conditions should be handled as possible modifiers of clinical management rather than presented as proven causes of malignant transformation [37-40]. Allergic reactions to dental materials are more relevant to oral lichenoid contact lesions than to classic OLP [1, 8]. A unilateral lesion adjacent to an amalgam or metal restoration should prompt consideration of a contact-related lichenoid lesion, especially when clinical distribution is anatomically linked to the restoration [1, 8]. Patch testing or restoration replacement may be appropriate in selected cases, but dental material hypersensitivity should not be overstated as a direct malignant transformation factor for classic OLP [1, 8]. Treatment-related immunosuppression is clinically important because symptom control should not compromise cancer surveillance [41-44]. Topical corticosteroids and other immunomodulatory agents are used to control symptomatic inflammation, but prolonged treatment can theoretically mask persistent lesions or delay biopsy when follow-up is inadequate [41-44]. Current evidence does not prove that OLP therapy itself causes OSCC, yet clinical surveillance must continue during treatment because a non-healing or changing lesion requires histopathological assessment regardless of symptom control [41-44]. Inadequate follow-up is a modifiable factor that can delay OSCC diagnosis in patients with OLP [17, 24, 26, 28]. Cohort studies and recent surveillance discussions emphasize that malignant transformation can occur after several years, so follow-up should be long-term and sometimes lifelong [17, 24, 28]. Practical surveillance should include lesion mapping, photographic documentation when possible, palpation, risk-factor review, patient education, and biopsy of persistent or changing lesions [24-26, 28]. Table 1 summarizes the reported malignant transformation rates of oral lichen planus and related oral lichenoid conditions.

Pathophysiological Mechanisms Linking OLP to Oral Carcinogenesis

The biological pathway from OLP to OSCC is not fully established, but chronic immune-mediated epithelial injury provides a plausible framework for carcinogenic susceptibility [2, 3, 45-46]. In OLP, basal keratinocytes are repeatedly damaged by cytotoxic immune responses, and repeated injury may drive cycles of epithelial regeneration, oxidative stress, and inflammatory remodeling [2, 3, 6-7]. Such cycles can increase the probability that genetically altered epithelial clones will persist in a chronically inflamed mucosal field [45-47]. Oxidative stress is a recurring theme in OLP molecular studies because reactive oxygen and nitrogen species can damage DNA, lipids, and proteins in epithelial cells [45-46, 48]. When repair mechanisms are overwhelmed or inefficient, oxidative damage may contribute to genomic instability

and altered cell-cycle control [45-46, 49]. This concept is consistent with studies reporting altered expression of apoptosis, proliferation, and cancer-hallmark markers in OLP samples, although causal proof remains incomplete [45-46, 49]. The inflammatory microenvironment may also influence malignant transformation through cytokines, chemokines, angiogenic mediators, and immune-checkpoint-related pathways [3, 46-47, 50]. A persistent inflammatory infiltrate can support epithelial remodeling and tissue turnover, but it may also alter local immune surveillance in ways that allow dysplastic clones to escape elimination [3, 46-47, 50]. The same immune response that produces symptomatic mucosal disease can therefore be protective, injurious, or permissive depending on timing, intensity, and local epithelial vulnerability [2-3, 6]. Cancerization in OLP should also be considered within the broader concept of field change in the oral mucosa [10-11, 31]. Tobacco, alcohol, chronic inflammation, microbial shifts, and repeated mucosal injury may act together across a field rather than at a single isolated lesion [10-11, 47-48, 51-52]. This helps explain why same-site documentation is essential when claiming true malignant transformation from a prior OLP lesion [16, 23, 31, 53]. Microbiome and metabolomic studies add another layer to this model by showing that lesional mucosa and saliva may differ from adjacent tissue or healthy controls [48, 51, 52]. Dysbiosis could theoretically intensify inflammation, influence epithelial metabolism, or modify immune signaling, but current studies cannot determine whether microbial changes are causes or consequences of OLP activity [48, 51, 52]. Therefore, microbial findings should be treated as hypothesis-generating rather than as clinically validated predictors of OSCC [48, 51, 52]. A balanced mechanistic interpretation is essential because overstatement may weaken the manuscript scientifically [16, 19, 21]. Current evidence supports a context-dependent model in which chronic inflammation, epithelial damage, dysplasia, lesion site, carcinogen exposure, infection-related cofactors, and follow-up quality interact to shape cancer risk [13-19, 45-46]. This model is more defensible than presenting OLP as either a harmless inflammatory condition or a uniformly premalignant lesion [10, 19, 21].

Biomarkers and Molecular Factors

Molecular studies support the biological plausibility of malignant transformation in a subset of OLP lesions, but no single biomarker is yet validated for routine prediction of OSCC development [45, 46, 49]. Investigated markers include molecules related to apoptosis, cell-cycle regulation, epithelial proliferation, oxidative stress, extracellular matrix remodeling, intercellular adhesion, immune activation, and inflammatory signaling [45-46, 49]. These findings are promising but remain limited by small cohorts, heterogeneous diagnostic criteria, and inconsistent separation of classic OLP from lichenoid dysplasia [45, 46, 49]. Immune dysregulation is central to OLP pathogenesis and provides a plausible link between chronic inflammation and carcinogenic

susceptibility [2, 3, 50]. Cytotoxic T cells, basal keratinocyte apoptosis, cytokine networks, and antigen-driven epithelial injury may generate a persistent inflammatory microenvironment [2, 3, 6, 7, 50]. Such a microenvironment can increase oxidative stress, epithelial turnover, and local immune remodeling, but inflammation alone is not sufficient to diagnose dysplasia or predict cancer [45-47]. Transcriptomic and cancer-hallmark studies suggest that OLP and OSCC may share altered pathways involving proliferation, apoptosis resistance, immune evasion, invasion-related remodeling, and angiogenesis [46, 48, 49]. Reported candidate genes and molecular patterns include *BCL9L*, *PER2*, *TSPAN33*, *p53*-related pathways, *Ki-67*, *COX-2*, galectins, and DNA repair-related molecules, although these markers are not uniformly validated [45-46, 49]. Because inflammatory atypia can mimic dysplastic change, molecular findings should be interpreted together with clinical behavior and histopathology [8, 30, 45]. The oral microbiome and local metabolic environment have also been explored as possible contributors to OLP activity and malignant transformation [48, 51, 52]. Studies comparing lesional and adjacent mucosa suggest that microbial communities and metabolomic profiles may differ in OLP, but whether these changes are causes, consequences, or modifiers of inflammation is still unclear [48, 51, 52]. Microbiome-based risk prediction therefore remains investigational and should not replace biopsy-based evaluation of suspicious lesions [48, 51, 52]. Salivary biomarkers are attractive because saliva collection is non-invasive, inexpensive, repeatable, and directly relevant to oral mucosal disease [54-61]. Studies have investigated salivary miRNAs, inflammatory cytokines, oxidative stress markers, C-reactive protein, MMP-9, CYFRA 21-1, aldehyde dehydrogenase-related markers, IL-1beta, IL-8, SAT, S100P, and OAZ1 in OSCC, OPMDs, and OLP-related comparisons [54-61]. However, variation in sample collection, assay platform, analytical thresholds, and patient selection prevents salivary biomarkers from being used as stand-alone diagnostic tools at present [57-61]. The most defensible conclusion is that molecular and salivary biomarkers may eventually improve risk stratification, but current patient management must still rely on clinical examination, histopathology, and longitudinal change [8, 28, 45]. Future biomarker studies should use standardized OLP definitions, clearly exclude misclassified dysplastic lesions at baseline, and report long-term outcomes at the same anatomical site [8, 16, 46]. Biomarker panels will be most useful if they complement, rather than replace, expert clinical judgment and biopsy of high-risk lesions [28, 45, 57].

Diagnostic Methods and Clinical Surveillance

Accurate diagnosis is central to any discussion of oral cancer risk because misclassification can inflate or underestimate transformation rates [8, 16, 19]. OLP can resemble oral lichenoid lesions, lichenoid drug reactions, contact hypersensitivity reactions, lupus erythematosus, leukoplakia, erythroplakia, proliferative verrucous leukoplakia, and early OSCC [1, 8, 10].

Table 1. Reported Malignant Transformation Rates of Oral Lichen Planus and Related Oral Lichenoid Conditions

Study	Design / sample	Diagnostic category	Reported transformation rate	Key interpretation
Aghbari et al., 2017 [13]	Systematic review and meta-analysis: 57 studies; 19,676 OLP patients	OLP and oral lichenoid lesions	1.1% for OLP; 2.5% for oral lichenoid lesions	Supported low but measurable malignant potential and showed the impact of diagnostic criteria.
Gonzalez-Moles et al., 2019 [14]	Systematic review and meta-analysis: 82 studies; 26,742 patients	OLP, oral lichenoid lesions, lichenoid reactions	1.14% for OLP; 1.88% for oral lichenoid lesions; 1.71% for lichenoid reactions	Emphasized diagnostic classification and study quality as key causes of rate variation.
Giuliani et al., 2019 [15]	Systematic review: 21 studies; 6,559 patients	OLP and oral lichenoid lesions	Overall 1.40%; 1.37% for OLP; 2.43% for oral lichenoid lesions	Suggested higher concern in red forms, tongue lesions, and female-predominant cohorts.
Idrees et al., 2021 [16]	Systematic review and meta-analysis applying strict criteria: 33 studies; 12,838 OLP patients	Strictly verified OLP	0.44% after reassessment	Argued that transformation is often overestimated without strict clinical and histological criteria.
Arduino et al., 2021 [17]	33-year cohort study; Northern Italy; 3,173 histopathologically confirmed patients	Histopathologically confirmed OLP	2.58%; 82 OSCC cases	Older age, red form, and less regular follow-up increased clinical concern.
Li et al., 2023 [18]	Systematic review and meta-analysis: 54 studies; 24,277 oral lichenoid condition patients	OLP, oral lichenoid lesions, lichenoid mucositis dysplasia	0.94% for OLP; 1.95% for oral lichenoid lesions; 6.31% for lichenoid mucositis dysplasia	Shown that dysplasia and diagnostic category substantially influence reported risk.
Gonzalez-Moles and Ramos-Garcia, 2024 [19]	Updated systematic review and meta-analysis; 101 studies; 38,083 patients	OLP and related oral lichenoid conditions	1.43% for OLP; 5.13% for OLP with dysplasia	Highlighted epithelial dysplasia as a major risk-stratification variable.
Tsushima et al., 2021 [24]	Retrospective study; Japan; 565 OLP patients	Clinical and histopathological OLP	0.7%; 4 SCC cases	Supported low transformation risk and emphasized differentiation from dysplasia and OLL.
Radochova et al., 2021 [25]	Retrospective study; Czech Republic; 271 OLP patients	Clinically and histologically confirmed OLP	0.74%; 2 SCC cases	Both transformed cases occurred in erosive OLP and developed on the tongue.
Zotti et al., 2021 [26]	Ten-year retrospective study	OLP follow-up cohort	Low transformation frequency	Highlighted smoking, alcohol, erythematous lesions, and tongue site as practical risk factors.
Guan et al., 2020 [27]	14-year longitudinal retrospective cohort; 829 patients	OLP and oral lichenoid lesions	Varied by diagnostic category	Reinforced the need to distinguish OLP from lichenoid lesions in follow-up studies.
Warakulasingha et al., 2023 [21]	Umbrella study of systematic reviews	OLP malignant transformation evidence base	Most systematic reviews reported approximately 1.1-1.4%	Summarized limitations of prior reviews and argued for stricter classification.
Roberts et al., 2024 [29]	Retrospective study; large erosive OLP cohort; Br J Oral Maxillofac Surg, 2024	Erosive OLP and suspected OLP cohort	1.39% overall; 5.98% for erosive OLP	Identified substantially higher concern in erosive OLP and supported closer surveillance of persistent erosive disease.
Ludecke et al., 2025 [28]	Clinical surveillance study	OLP monitoring with close follow-up and brush biopsy	Focused on early detection rather than pooled rate	Suggested that close surveillance may support earlier recognition of suspicious change.

Therefore, diagnosis should combine clinical pattern, lesion distribution, histopathological features, risk-factor assessment, and follow-up behavior [8, 22-23]. Clinical examination should document lesion morphology, color, symmetry, distribution, site, size, symptoms, and duration [1, 8]. Classic OLP often has bilateral or multifocal reticular white striae, but high-risk appearances include persistent red areas, erosions, ulceration, induration, heterogeneous red-white change, progressive enlargement, or unexplained pain [1, 8, 25]. Palpation is important because induration or fixation may indicate invasive disease even when surface changes appear modest [10-11]. Biopsy is essential when diagnosis is uncertain or when lesions show suspicious behavior [8, 22, 28]. Histopathology can confirm lichenoid mucositis, identify epithelial dysplasia, detect invasive carcinoma, and help distinguish classic OLP from lichenoid dysplasia or other OPMDs [8, 22, 30]. Repeat biopsy should be performed if a previously diagnosed lesion becomes persistent, ulcerated, indurated, erythroplakic, enlarging, treatment-resistant, or clinically different from its earlier appearance [24, 25, 28]. Histopathological interpretation requires caution because inflammation-related epithelial atypia can be difficult to separate from true dysplasia [8, 22, 30, 62]. Experienced oral pathologists are especially important when clinical concern and histological findings do not align [8, 22, 30, 62]. Dysplasia grading systems have only imperfect reproducibility, but the presence of moderate or severe dysplasia should substantially increase surveillance intensity [30, 62]. Adjunctive diagnostic tools may support surveillance but cannot replace biopsy in suspicious lesions [28, 57-61]. Brush biopsy, cytology, optical imaging, fluorescence visualization, salivary assays, and molecular tests may improve triage in selected settings, but their diagnostic performance depends on population, sampling technique, threshold definition, and lesion type [28, 57-61]. A negative adjunctive test should not reassure the clinician when the lesion is clinically suspicious [28, 57, 60]. Patch testing has a narrow but useful role when an oral lichenoid contact reaction is suspected [1, 8]. This situation is most likely when lesions are unilateral, localized adjacent to restorations, or anatomically related to dental materials [1, 8]. Patch testing and replacement of the suspected restoration may help selected patients, but this approach should not be presented as a routine cancer-prevention method for classic OLP [1, 8]. Follow-up intervals should be individualized according to clinical risk [17, 24-26, 28]. Stable reticular disease may be reviewed periodically, whereas erosive, atrophic, ulcerative, dysplastic, tongue-based, or clinically changing lesions require closer review [24-26, 28]. Many authors recommend long-term and sometimes lifelong follow-up because transformation can occur years after initial diagnosis [17, 24, 28]. A practical surveillance visit should include inspection and palpation of all oral subsites, comparison with prior photographs or lesion maps, review of pain and functional symptoms, assessment of tobacco and alcohol exposure, screening for candidiasis, and reinforcement of self-monitoring instructions [24-26, 28]. Patients should be instructed to report persistent ulcers,

new red areas, bleeding, thickening, enlargement, persistent pain, or lesions that fail to heal within a short interval [24-26, 28]. This patient-centered approach may reduce delayed diagnosis and is more clinically useful than relying on a single pooled transformation estimate [19, 21, 28].

Clinical Risk Stratification and Practical Follow-up Framework

A practical risk-stratification framework can help clinicians translate heterogeneous evidence into patient care [17, 24-26, 28]. Low-concern patients generally include those with stable, bilateral, reticular OLP, no dysplasia, no tongue or floor-of-mouth involvement, and no tobacco or heavy alcohol exposure [1, 8, 24]. Higher-concern patients include those with erosive, atrophic, ulcerative, erythroplakic, indurated, tongue-based, dysplastic, or treatment-resistant lesions [17, 24-26, 28]. Risk status should be dynamic rather than fixed at the first visit [24-26, 28]. A patient initially classified as lower concern may move into a higher-concern category if a lesion changes color, becomes painful, ulcerates, enlarges, develops induration, or fails to respond to appropriate treatment [24-26, 28]. This dynamic approach is clinically safer than assuming that a previous benign biopsy permanently excludes future dysplasia or OSCC [24-25, 28]. Follow-up intervals should reflect this risk profile [17, 24-26, 28]. Stable reticular disease may be reviewed at longer intervals, whereas erosive disease, tongue lesions, dysplasia, or persistent ulceration may require review every few months until stability is established [24-26, 28]. Annual lifelong follow-up has been recommended by some long-term cohort authors because malignant transformation can occur after prolonged disease duration [17, 28]. At each visit, documentation should include lesion site, size, clinical subtype, symptoms, palpation findings, photographs when possible, and comparison with previous records [24-26, 28]. This documentation is important because subtle progression may be missed when clinicians rely only on memory or nonspecific descriptions [24-25, 28]. Standardized follow-up records can also improve future research by making transformation claims more reliable [16, 18-19]. Patient education is a critical part of surveillance because patients often notice symptoms between appointments [24-26, 28]. Patients should be advised to seek reassessment for persistent ulcers, new red patches, bleeding, thickening, unexplained pain, enlargement, or lesions that do not heal within two weeks [24-26, 28]. Education should be framed carefully so that patients understand the risk is low but not negligible [13-19]. Referral pathways should be clear for general dentists, dermatologists, oral medicine clinicians, and surgeons [1, 8, 28]. Persistent or suspicious lesions should be referred to clinicians experienced in oral mucosal disease and oral biopsy, particularly when dysplasia or OSCC is possible [8, 28, 30]. Multidisciplinary care is especially useful for medically complex patients, patients receiving systemic immunosuppression, or patients with recurrent diagnostic uncertainty [37-44].

Treatment and Clinical Management

The primary treatment goal in OLP is symptom control, but management must also preserve cancer surveillance [32, 41-44]. No current OLP therapy has been proven to prevent OSCC development, so anti-inflammatory response should never be interpreted as elimination of malignant potential [32, 41-42]. Treatment plans should be integrated with follow-up, risk-factor modification, and biopsy of persistent or changing lesions [41-44]. Topical corticosteroids remain the first-line treatment for symptomatic OLP because they reduce pain, erythema, erosions, and inflammatory activity while limiting systemic exposure [32, 41-42]. High-potency topical preparations are commonly used for erosive or ulcerative disease, whereas milder disease may be managed with topical therapy and local supportive measures [32, 41-42]. Clinicians should monitor for candidiasis, mucosal irritation, steroid-related adverse effects, and persistent lesions that do not respond as expected [32, 41-42]. Topical calcineurin inhibitors may be considered in selected patients with refractory disease, but their use should be cautious and monitored [42-44]. Meta-analytic evidence suggests that calcineurin inhibitors can improve symptoms, yet comparisons with corticosteroids and long-term safety questions remain clinically relevant [41, 43-44]. Because these agents modulate local immunity, treatment should be accompanied by careful examination and biopsy of lesions that persist or change [42-44]. Systemic corticosteroids and systemic immunomodulatory agents may be needed for severe, widespread, or refractory OLP, but long-term use is limited by adverse effects [32, 42, 44]. Retinoids, biologic pathway-directed therapies, dapsone, antimalarial agents, phototherapy, photodynamic therapy, hyaluronic acid, curcumin, aloe vera, and low-level laser therapy have been studied with varying evidence quality [41-44, 63-70]. These approaches may be reasonable for selected patients, but none should be described as a validated strategy for preventing malignant transformation [41-44, 64-70]. Photodynamic therapy and photobiomodulation have received attention because they may reduce symptoms with a relatively favorable tolerability profile [65-67]. Randomized trials and systematic reviews suggest potential benefit in symptomatic OLP, although protocols differ in photosensitizer, wavelength, exposure, session number, and outcome measures [65-67]. These therapies should be framed as symptom-management options rather than definitive cancer-prevention interventions [65-67]. Immunosuppression-related cancer surveillance should be discussed with balanced language [41-44]. There is limited direct evidence that topical corticosteroids or calcineurin inhibitors cause OSCC in OLP, but prolonged immunosuppression may theoretically alter local immune surveillance or mask lesions that require biopsy [42-44]. Therefore, clinicians should avoid repeatedly escalating anti-inflammatory therapy for a persistent ulcer, red lesion, or indurated area without histopathological reassessment [24, 28, 42]. Risk-factor modification is a core part of OLP management because tobacco and alcohol are established oral carcinogens [10-11, 16, 18]. Smoking cessation,

reduction of heavy alcohol consumption, management of local trauma, treatment of candidiasis, and optimization of oral hygiene should be included in routine care [10-11, 42]. When HCV infection is suspected or epidemiologically relevant, appropriate medical evaluation should be considered because HCV may modify OLP burden and possibly cancer risk in some populations [34-35]. Comorbidities should influence treatment safety even when they are not proven malignant transformation drivers [37-40]. Diabetes can increase candidiasis risk and impair healing, liver disease can influence drug metabolism and bleeding risk, and cardiovascular disease may shape systemic therapy choices [37-40]. This framing keeps the manuscript focused on oral cancer risk while still acknowledging clinically relevant systemic conditions [37-40]. The key clinical principle is that treatment and surveillance must proceed together [17, 24, 28]. A lesion that enlarges, hardens, ulcerates, becomes erythroplakic, bleeds, produces persistent pain, or fails to respond to appropriate therapy should be biopsied regardless of the current treatment regimen [24-26, 28]. This approach ensures that symptom control does not replace cancer surveillance, particularly when immunosuppressive or immunomodulatory therapy is used [41-44].

Limitations of Current Evidence and Future Directions

The evidence base is limited by diagnostic heterogeneity, retrospective design, inconsistent biopsy protocols, variable follow-up duration, and incomplete adjustment for tobacco, alcohol, HCV, epithelial dysplasia, and lesion subtype [13-19, 21]. These limitations explain why apparently similar studies can produce different malignant transformation estimates [16, 18-19]. A narrative review should therefore present the evidence descriptively and critically rather than implying systematic certainty that is not supported by the manuscript design [10, 19, 21]. Future studies should use standardized clinical and histopathological criteria that separate classic OLP, oral lichenoid lesions, lichenoid contact reactions, drug reactions, and lichenoid dysplasia [8, 16, 18-19]. Long-term prospective cohorts should report lesion site, lesion morphology, dysplasia grade, treatment exposure, follow-up interval, smoking, alcohol consumption, HCV status, diabetes, and reasons for repeat biopsy [16-19, 28]. The same-site relationship between baseline OLP and later OSCC should be documented because cancer arising elsewhere in the oral cavity may not represent true transformation of the original lesion [16, 23]. Molecular and salivary biomarker research should move from discovery studies to validated risk models [45-46, 49, 54-61]. Promising biomarkers need external validation, standardized sampling, reproducible thresholds, and clinically meaningful endpoints such as same-site dysplasia or OSCC development [45-46, 54-61]. Integrated models combining clinical subtype, tongue involvement, dysplasia, carcinogen exposure, HCV status, and biomarker panels may eventually guide personalized surveillance intervals [19, 28, 46]. This narrative review also has practical limitations because it is not a formal systematic review [10, 19, 21]. It does

not include PRISMA reporting, risk-of-bias scoring, study-level data extraction, or quantitative meta-analysis, and the Methods section states this explicitly to avoid methodological ambiguity [10, 19, 21]. The strength of this format is that it allows clinically oriented synthesis of risk, diagnosis, surveillance, biomarkers, and treatment, while the limitation is that it cannot provide a new pooled transformation estimate [13-19]. Future clinical protocols should define when biopsy is mandatory and when short-interval review is acceptable [24-26, 28]. Such protocols should consider the combined effect of lesion morphology, anatomical site, dysplasia history, tobacco and alcohol exposure, HCV status, treatment response, and patient ability to attend follow-up [16-19, 24-26, 28]. Developing practical risk categories may be more useful for clinicians than continuing to debate a single average malignant transformation rate [19, 21, 28]. Future biomarker studies should be designed around clinically meaningful decisions rather than isolated statistical differences [45-46, 49, 54-61]. A useful marker should help decide whether a patient needs biopsy, closer surveillance, referral, or reassurance beyond what clinical examination and histology already provide [28, 45, 57]. Until such evidence is available, biomarkers should remain complementary research tools rather than routine substitutes for clinicopathological assessment [45-46, 54-61].

In conclusion, OLP is a chronic immune-mediated inflammatory disorder of the oral mucosa and is recognized as an oral potentially malignant disorder. The overall risk of malignant transformation into OSCC appears to be low but clinically meaningful, and reported transformation rates vary because of differences in diagnostic criteria, inclusion of oral lichenoid lesions, follow-up duration, epithelial dysplasia, lesion subtype, lesion site, and carcinogenic exposures. The most clinically relevant indicators of increased concern include erosive, atrophic, ulcerative, or erythematous lesions; tongue localization; persistent non-healing ulceration; induration; epithelial dysplasia; tobacco use; alcohol consumption; possible HCV infection in selected populations; older age; and inadequate follow-up. Systemic conditions such as diabetes mellitus, chronic kidney disease, hypertension, and cardiovascular disease should be interpreted cautiously because they are not established direct predictors of malignant transformation in classic OLP. Clinical management should combine symptom control with long-term cancer surveillance. Topical corticosteroids remain the first-line therapy for symptomatic OLP, but prolonged immunosuppressive or immunomodulatory treatment should not delay biopsy of suspicious lesions. Regular follow-up, patient education, risk-factor modification, and histopathological evaluation of persistent or changing lesions are essential for reducing delayed diagnosis of OSCC. Further well-designed prospective studies with standardized diagnostic criteria and long-term follow-up are needed to clarify biological mechanisms, validate predictive biomarkers, and develop evidence-based surveillance strategies for patients at increased risk.

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Data availability

Not applicable because this narrative review used information from previously published articles.

Approved by any scientific body

Not applicable because the manuscript is not part of a student thesis or original clinical study.

Ethical issue and approval

Not applicable because no human participants, animals, or unpublished patient data were included.

Consent for publication

The author has given consent for publication.

Conflict of interest

The author declares no potential conflict of interest.

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