Narrative Review

OLP vs OLL: Diagnostic Challenges and Malignancy Risk

Oral Lichen Planus VS Lichenoid Lesions: Diagnostic Challenges and Risk of Malignant Transformation – A Narrative Review

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ABSTRACT

Objective: The aim of the narrative review is to compare the incidence of the malignant transformation of oral lichen planus (OLP) and oral lichenoid lesions (OLL) in patients.

Place and Duration of Study: The literature review was undertaken at the Department of Oral and Maxillofacial Diagnostic Sciences, College of Dentistry, Qassim University, Saudi Arabia from January 2025 to May 2025.

Methods: The review of literature was undertaken with the help of electronic data bases, including Web of Science, PubMed, Google Scholar, Scopus, Medline, Embase, NCBI, Elsevier. The following keywords were used to search for the present study: oral lichen planus, oral lichenoid lesions, malignant transformation, and diagnostic challenges: 2014, May 2024.

Results: The reported rates of malignant transformation of OLP and OLL ranged from 0 to 5.4. The incidence of malignant transformation in OLP is uncertain due to overlap with OLL and with early-stage oral squamous cell carcinoma (OSCC) and lichenoid lesions.

Conclusion: Malignant transformation risk in OLP and OLL is variable with a low average risk. However, a combination of clinical and histological features complicates risks assessment and there is a need to put more emphasis on follow-up and consistent diagnosis.

Key Words: Oral lichen planus (OLP); Oral lichenoid lesions (OLL); Malignant transformation; Oral squamous cell carcinoma (OSCC)

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INTRODUCTION

Oral mucosal lesions are frequently seen in clinical practice, and oral lichen planus (OLP) and oral lichenoid lesions (OLL) can be challenging to diagnose and treat due to their similar clinical and histopathological appearances and unclear risk of malignant transformation[1]. Lichen planus (LP) or lichen ruber planus is autoimmune inflammatory skin and mucosal disease of the stratified squamous epithelium that is mainly associated with the damage caused by the cytolytic $CD8^+$ T-cells on the basal keratinocytes[2]. LP is also associated with a poor quality of life, both psychologically and socially, and hence better understanding of the disease pathophysiology is required for effective management. LP is clinically classified into cutaneous LP, mucosal LP and scalp LP, with variations in presentation but similar immunopathogenic process.

Overview of Oral Lichen Planus: Oral lichen planus (OLP), first described in 1869 by William James Erasmus Wilson, is an inflammatory, remitting and relapsing, chronic oral mucosal disease[3]. The disease typically presents as bilateral, white, reticular white lines (Wickham striae). OLP can be clinically classified into six types: reticular, erosive, atrophic, papular, plaque-like and bullous, and some also include additional types[4]. The cause and pathogenesis of OLP remain uncertain, but it is apparent that an immune dysfunction is present, with altered expression of cytokines (such as interleukins, transforming growth factor-β, interferon-γ and tumor necrosis factor-α) in lesional and peripheral samples.

Clinical Characteristics of Oral Lichen Planus: Oral lichen planus (OLP) may involve the oral and genital mucosa, oesophagus, scalp, nails and the eye, and is more common in middle aged females. The most frequent reticular form is usually asymptomatic with bilateral white lacy lines on the buccal mucosa whereas

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papular OLP is rare and resembles small white coalesced papules. The plaque-like variant is similar to leukoplakia and usually occurs on the buccal mucosa and tongue[5]. While reticular, papular and plaque-like OLP is usually asymptomatic and hyperkeratotic, the erosive, atrophic and bullous varieties are symptomatic and display erythema, ulceration and easily ruptured vesicles. And lastly, stress has been reported to induce or worsen the condition, possibly as a result of stress-induced immune responses with increased levels of cortisol and cytokines[6].

Oral Lichen Planus versus Oral Lichenoid Lesions: Oral lichen planus (OLP) and oral lichenoid lesions (OLL) are often confused due to similar appearances. Erosive OLP is often painful, with burning on eating spicy foods and xerostomia in nearly 45% of cases. OLP and OLL may be associated with a variety of systemic conditions such as Sjögren's syndrome, hepatitis C infection, type 1 diabetes mellitus and graft-versus-host disease[7]. OSCC, which accounts for 90% of oral cancers, has also been reported to undergo malignant transformation in OLP as early as 1910. But the WHO diagnostic criteria may not be specific enough to distinguish OLP from OLL. However, Van der Meij and Van der Waal proposed diagnostic criteria for OLP including clinical and histopathological features; lesions not fulfilling these criteria are defined as OLL and are often less homogeneous and may be lichenoid reactions or contact stomatitis[8].

Malignant Potential and Management: Oral lichen planus (OLP) occurs in 0.5% to 2.6% of the global population, most commonly on the buccal mucosa, followed by the gingiva, tongue and lips. The WHO classifies OLP as a potentially malignant condition, but the malignant potential of this condition is unclear; some studies have reported that malignant transformation described in OLP is due to misdiagnosis of an oral lichenoid lesion (OLL). Primary treatment goals of OLP are symptomatic relief and improved quality of life, with initial steps being education, stress management and elimination of potential local irritants, such as sharp tooth surfaces, ill-fitting dentures, candidiasis and occlusal trauma[9]. Topical corticosteroids, particularly 0.1% triamcinolone acetonide, are still the primary treatment and others including calcineurin inhibitors (tacrolimus) are used in non-responsive cases; systemic corticosteroids are reserved for severe cases. Recently, photodynamic therapy and ozone therapy have proven effective with few side effects[10].

METHODS

Search Strategy: A literature review was undertaken to determine the risk of malignant transformation of oral lichen planus (OLP) and oral lichenoid lesions (OLL). The study was conducted from January 2025 to May 2025, with data extracted, reviewed and analysed. Data extraction and literature review was undertaken at the Department of Oral and Maxillofacial Diagnostic Sciences, College of Dentistry, Qassim University, Saudi Arabia.

An electronic search was performed using various databases including Web of Science, PubMed, Google Scholar, Scopus, MEDLINE, Embase, NCBI and other Elsevier-indexed publications. The search was conducted for all publications between January 2014 and May 2024. Keywords searched included oral lichen planus, oral lichenoid lesions, malignant transformation, and diagnosis. These terms were searched individually and in conjunction with Boolean operators. The last step was to manually search the references of the selected studies to find more studies. A systematic screening process was used. The titles and abstracts of identified studies were reviewed to determine their level of relevance and then full-text screening was undertaken. Eligible studies were included.

Eligibility Criteria: This narrative review included original articles, review articles and case reports of patients with oral lichen planus and/or oral lichenoid lesions. The studies must be published in or after 2014, and the article must be in English. Articles were excluded if they had insufficient or unclear information on malignant transformation, focused on oral potentially malignant disorders other than OLP or OLL, or were published in any other language than English.

FINDINGS

Malignant Transformation Rates: The papers comprising this review are all consistent in their findings, showing that oral lichen planus (OLP) and oral lichenoid lesions (OLL) both have a quantifiable but comparatively small risk of developing into malignant lesions. The majority of the reported transformation rates vary between 0.5-3.9. Rates of large cohort studies are often below 2 and higher estimates more often so associated with OLL. Such variability is likely due to differences in the criteria of diagnosis, follow-up time, and population. The evidence, in general, confirms the assertion that OLL has a greater malignant potential than OLP, and correct diagnosis and classification needs to be performed.

3.2 Risk Factors and Predisposing Conditions: Not all patients run the risk of developing malignant transformation. Malignant change seems to have a specific vulnerability to lesions of the lateral margin of the tongue, as well as the lingual gingiva. Besides this, erosive and atrophic variations of OLP are also more risky than reticular or plaque-like OLP. The lifestyle determinants like smoking and alcoholism could also contribute to carcinogenesis. Systemic diseases such as thyroid disorders, diabetes mellitus, and hepatitis C infection are also often linked to OLP but there is no conclusive evidence that they cause it. These results

highlight the necessity to risk stratify patients and use individual follow-up guidelines.

Diagnostic Challenges: The high clinical and histopathological overlap between OLP and OLL (Fig. 1) is also one of the key barriers to estimating the malignant risk. Differentiating the two entities is frequently based on the minor diagnostic guidelines or exclusion of discernible precipitants like dental materials or drugs. This error in classification can explain the variation in the reported rates of malignant transformations in the studies. New diagnostic modalities, such as narrow-band imaging (NBI) have potential in improving the characterization of lesions and may be useful in the process of identifying regions that need to be biopsied or followed up.

Table No. 1: Description of studies selected for Narrative Review

Name and year of the study	Study design	Duration	Sample size	Purpose	Outcome
(Tsushima et al., 2021)[11]	Retrospective study		n=565	1- To evaluate the demographic and clinical characteristics of patients with OLP. 2- Rate of malignant transformation of OLP patients.	OLP is associated with a low rate of malignant transformation.
(Zotti et al., 2021)[12]	Retrospective study	5-10 years	N= 100	1- Evaluate the duration of OLP transformation in OSCC. 2- Risk factors of OLP diagnosed with OSCC. 3- Forms of OLP evolved in cancer.	4 out of 8 malignant transformations in 4 years. Odd ratios between malignant transformation and location.
(Guan et al., 2020)[13]	Longitudinal Retrospective Cohort study	14 years	Females= 548 Males=281	1- Evaluate the rate of malignant transformation in patients with OLP and OLL. 2- To assess the link between clinicopathologic aspects and malignant transformation.	Oral epithelial dysplasia occurs in <1% of patients with OLP/OLL. And OSCC was at 2.8% during the follow-up period.
(Ruokonen et al., 2017)[14]	Retrospective observational study		N=323 females:164 males: 159	1- Evaluate the OLP and OLL as precursor lesions in OSCC.	1- TN class 1 tumors-prevalent among patients with OLP or OLL (P=0.006). 2- Importance of active follow-up of patients with OLP and OLL.
(Cai et al., 2022)[15]	Retrospective cohort study		N= 3568	To evaluate the association between OLP and OSCC.	3 out of 10 primary cancers were distinct from the site of OLP. 2 were malignant of proliferative verrucous leukoplakia. No OSCCs are transformed from OLP.
(Arduino et al., 2021)[16]	Cohort Study	103.61 months	N= 3173	Histopathological diagnosis of oral cancer	32 men and 50 women developed OSCC. 21 patients died of oral cancer.
(Bandyopadhyay et al., 2017)[17]	Retrospective case series	3.5 Years	N= 143 cases	Evaluate the histopathologically diagnosed OLP and associated malignant transformation.	Two patients (1.4%) previously diagnosed with OLP developed OSCC.
(Gonzalez-Moles et al., 2017)[18]	Retrospective study	11 Years	N= 102 OLP= 21 OLL= 81	Evaluate the cancer incidence in case series of patients with OLP and OLL.	4 (3.9%) of patients developed cancer. One had OLL, three in OLP. Among three carcinomas (2 in the lower gingiva, 1 in the floor of the mouth).
(Kakoei et al., 2022)[19]	Retrospective study	23 years (1998-20)	N= 356 patients	Malignant transformation in OLP. OLL and associated risk factors.	Dysplastic changes occur in 6.2% of the patients.
(Saepoo et al., 2023)[20]	Retrospective cohort study	7 years	N= 117 OLP= 103(88%)	Prevalence of malignant transformation and incidence rate of OSCC	Prevalence of malignant transformation OLP/OLR (1.71%).

			OLR=14 (12%)		2 out of 117 patients. MT in OLP= 1.94% (2/103) The overall incidence rate of MT into OSCC= 0.0060
(Cozzani et al., 2019)[21]	Prospective study		N= 32 with OLP.	Evaluate value of Narrow-band imaging in selecting patients for biopsy, allowing early detection of malignancy from OLP.	Two patients marked positive through NBI and diagnosed with SCC after histological examination.
(Bindakhil et al., 2022)[22]	Retrospective study		N= 82 Women= 48 (58.25%) Men= 34 (41.5%) Mean age = 65.9yrs.	To examine if Topical Corticosteroid Therapy impacts the time of malignant transformation of OLP to OSCC.	The time duration between OLP and OSCC increased by 4 years in patients with TCT therapy. And decreased by 3 years in patients with candida overgrowth.
(Casparis et al., 2015)[23]	Retrospective study	10 years	N=542 patients 692 biopsies Men= 207 (38.2%) Women= 335(61.8%)	Incidence of malignant transformation of OLP.	Malignant transformation rate [MTR] = Higher in OLL (4.4%) in OLP (1.2%).

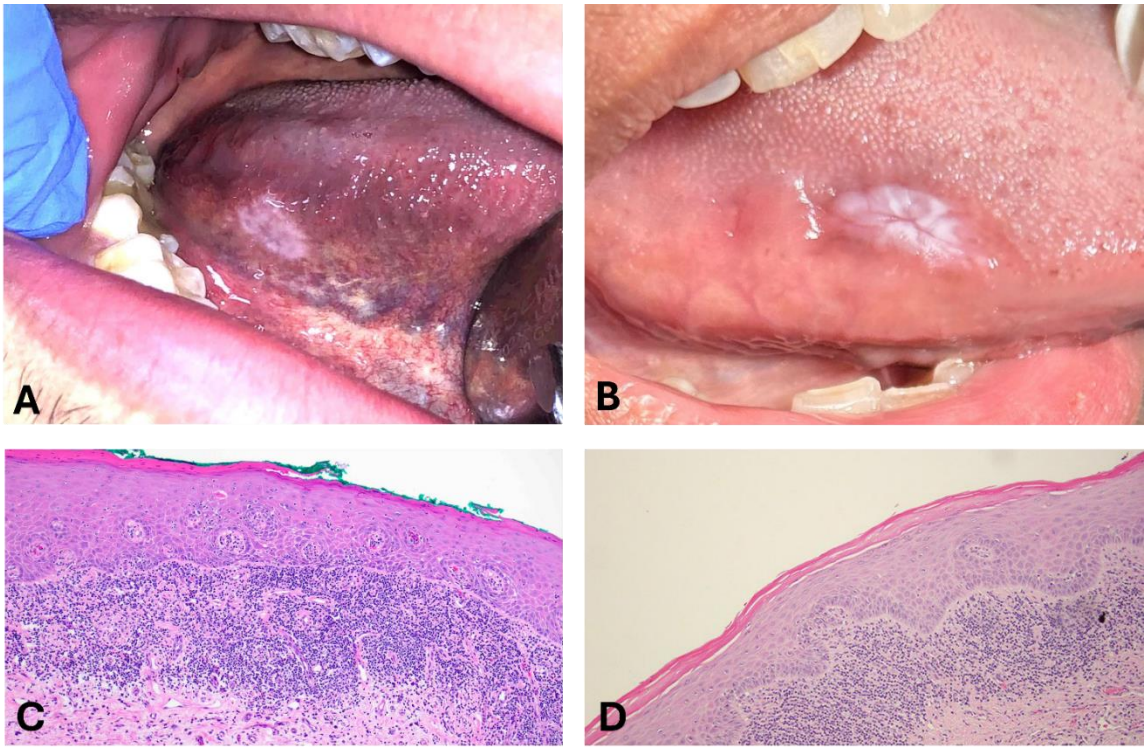

Figure No. 1: clinical and histological pictures of OLP and OLL. OLP (A) and OLL (B) Both have similar clinical features of Wickham stria within erythematous background. Histological features are also indistinguishable. (C) and (D) Show band like lymphocytic infiltrate subjacent to mucosal epithelium with basal layer degeneration and hyperkeratosis. (Clinical and histological photos are retrieved from the author's own archive)

Management and Future Directions: Treatment of OLP and OLL is mainly related to the control and reduction of symptoms and mitigation of malignant risk. Topical and systemic corticosteroids form the basis of treatment and there is some evidence to show that they can slow malignant transformation, though no established effect of protection has been recognised. Therapies adjunctive like photodynamic therapy, ozone

therapy and others have been researched and have shown benefits mostly due to symptomatic relief and not cancer prevention. Future studies must focus on the discovery of credible biomarkers and new imaging modalities to facilitate early distinguishing OLP and OLL and longitudinal studies on the effects of therapeutic interventions on the malignant outcomes in the long term.

DISCUSSION

This review suggests that the rate of malignant transformation of oral lichen planus (OLP) is low but clinically significant (0-5.4%), although risk assessment is made difficult by its association with oral lichenoid lesions (OLL) and its similarities to early oral squamous cell carcinoma (OSCC). It seems OLL may have a greater malignant potential, highlighting the need for accurate diagnosis[9]. The inflammatory process in OLP leads to oxidative stress, DNA damage and molecular changes, such as p53 overexpression, which supports its involvement in carcinogenesis, especially in erosive and erythematous forms and at high-risk sites, such as the lateral tongue. Moreover, systemic disease, lifestyle factors and misdiagnosis (e.g. leukoplakia and lichenoid drug reactions) add to the challenge[16]. This underscores the importance of clear diagnostic criteria, risk stratification based on lesion type, and regular surveillance, with early detection through clinical assessment being crucial for better outcomes.

CONCLUSION

This review highlights that while OLP/OLL carry a low but significant malignant potential, the diagnostic challenge due to overlapping clinical and histologic features of both conditions still persists. Advances in imaging and targeted therapy show promise, but long-term follow-up and precise diagnostic criteria are essential for reducing chances of malignant transformation.

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