

Original Article

CK17 Expression in Oral Lichen Planus, Oral Epithelial Dysplasia and Oral Squamous Cell Carcinoma: An Immunohistochemical Study

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KEY WORDS

Lichen planus;
Squamous cell carcinoma;
CK17;
Oral cancer;
Epithelial dysplasia;

ABSTRACT

Background: Cytokeratin 17 (CK17) is a cytoskeletal protein that is absent in stratified epithelia but can be induced in activated keratinocytes. The precise mechanisms of CK17 in premalignant and malignant progression remain unclear. In addition, accurate histopathological differentiation between oral lichen planus (OLP) and epithelial dysplasia remains a diagnostic challenge.

Purpose: The present study was designed to evaluate CK17 expression using immunohistochemical analysis across seven diagnostic groups, including normal oral epithelium, OLP, low-grade dysplasia, high-grade dysplasia, conventional squamous cell carcinoma (SCC), papillary SCC, and verrucous carcinoma, to determine whether CK17 could serve as an adjunctive biomarker for assessing epithelial dysplasia in OLP.

Materials and Method: In this retrospective cross-sectional study, 69 formalin-fixed paraffin-embedded tissue samples were assessed. Some blocks contained more than one histopathologically distinct area, resulting in 88 evaluable tissue sections for immunohistochemical analysis. Immunohistochemical staining was performed using a monoclonal anti-CK17 antibody. CK17-positive epithelial cells were counted in at least three randomly selected fields at 200× magnification. Cases with <5% reactive cells were considered negative, whereas those with >5% reactivity were classified as positive. Among positive cases, sections showing >60% positive cells were defined as strong expression, and those with <60% as weak expression. Statistical analyses were conducted using the independent-samples Kruskal-Wallis test and the Bonferroni correction to address multiple testing in SPSS version 28.

Results: Strong CK17 expression (>60% positive cells) was observed in 67 of 88 evaluated samples (76.1% of the total cohort). A statistically significant difference in CK17 expression scores was observed among diagnostic groups (Kruskal-Wallis $H(6)=80.87$, p Value<0.001). However, pairwise comparisons with Bonferroni adjustment showed no significant difference between OLP and SCC (Adj. $p=1.000$) and between OLP and high-grade dysplasia (Adj. $p=1.000$).

Conclusion: CK17 expression was strongly positive in high-grade dysplasia and in all examined malignant lesions, including SCC, papillary SCC, and verrucous carcinoma, supporting its role as a marker of advanced epithelial alteration. However, its similarly high expression in OLP indicates that CK17 lacks sufficient specificity to reliably distinguish immune-mediated epithelial changes from dysplastic transformation.

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Introduction

Keratins are a large family of intermediate filament proteins predominantly expressed in epithelial cells, where they provide structural integrity to the cytoskeleton and maintain cellular stability [1]. Beyond their structural functions, keratins regulate cellular proliferation and apoptosis, facilitate molecular transport across epithelial cells, participate in immune responses, and their expression patterns serve as reliable immunohistochemical (IHC) markers for identifying epithelial differentiation and neoplastic transformation [2-4].

Among the cytokeratin family, cytokeratin 17 (CK17) has gained particular importance because its expression is increased at both the gene and protein levels in oral squamous cell carcinoma (OSCC) compared with normal oral mucosa [5]. CK17 is a low-molecular-weight (48.1 kDa) cytoskeletal protein that is normally absent in stratified epithelia but can be induced in activated keratinocytes [6]. It is considered a basal/myoepithelial cell-specific marker and is also involved in the formation and maintenance of epithelial structures [1]. Several studies have shown that CK17 is upregulated not only in OSCC but also in premalignant lesions such as leukoplakia with epithelial dysplasia, suggesting its potential role as a diagnostic marker for both premalignant and malignant oral lesions [2, 5, 7]. Although the precise mechanisms of CK17 in cancer progression remain unclear, it has been implicated in multiple carcinogenic pathways, including transcriptional regulation, subcellular localization, glycolysis, and enhancement of cancer stemness [8].

Oral leukoplakia is the most common oral potentially malignant disorder (OPMD) and is considered a precursor lesion for several cases of OSCC [2]. Dysplastic changes in the stratified squamous epithelium are characterized by cellular atypia and the loss of normal maturation and stratification [9-10]. It has been shown that CK17 expression is significantly higher in leukoplakia with epithelial dysplasia compared to lesions without dysplasia [7]. This finding suggests that CK17 upregulation may occur early during oral carcinogenesis and could serve as a useful marker for identifying premalignant lesions with higher malignancy potential. However, the exact pattern and diagnostic reliability of CK17 expression in leukoplakia remain ambiguous, highlighting the need for further studies to clarify its role in the

progression from dysplasia to carcinoma [7].

Oral lichen planus (OLP), another OPMD, is a chronic immune-mediated inflammatory disease of the oral mucosa with a global prevalence of approximately 1%. Its pathogenesis involves a complex interplay of environmental triggers, such as stress, trauma, infection, and genetic factors, leading to a T-cell-mediated cytotoxic response against basal keratinocytes [11]. OLP carries a reported malignant transformation risk of approximately 1.4% [12]. The distinction between OLP, oral lichenoid reactions (OLRs), and oral lichenoid dysplasia (OLD) remains a significant diagnostic challenge in oral pathology. OLP typically presents as bilateral and relatively symmetrical lesions of unknown etiology, whereas OLRs are usually associated with identifiable triggering factors such as dental restorative materials, medications, or systemic conditions and often demonstrate a more localized or asymmetrical distribution [13]. OLD has been described as a distinct entity in which lichenoid inflammatory features are accompanied by epithelial dysplasia, and it may closely mimic OLP both clinically and histopathologically. Given its dysplastic component, OLD carries a recognized malignant potential, making its distinction from OLP clinically essential [13]. Atypical presentations, including unilateral lesions, may further complicate the diagnostic process and necessitate careful clinicopathological correlation. In addition, histopathological evaluation of oral mucosal biopsies may occasionally be inconclusive, leading to diagnostic uncertainty and potential delay in appropriate management [14]. Other conditions, including OLR, lupus erythematosus, leukoplakia, erythroleukoplakia, and proliferative verrucous leukoplakia, may also present with overlapping clinical and histopathological features with OLP, further increasing the risk of diagnostic confusion [15-16]. Therefore, adjunctive IHC markers have been proposed to improve diagnostic accuracy beyond conventional histopathological assessment [14].

Altered keratin expression, particularly CK17, has been associated with epithelial barrier dysfunction and immune imbalance in OLP [12]. While the World Health Organization classifies OLP as an OPMD, the exact magnitude of its malignant potential remains controversial among researchers. Therefore, regular follow-up of patients with OLP and oral lichenoid lesions has

been recommended to allow early detection of epithelial dysplastic changes and to reduce the risk of malignant transformation [17-18]. Given these inconsistent findings and the limited understanding of how epithelial differentiation correlates with CK17 expression in oral mucosa, further studies are warranted to clarify its role in predicting the progression of premalignant lesions toward OSCC.

This study aimed to investigate the IHC expression of CK17 across a spectrum of oral epithelial lesions, including OLP, epithelial dysplasia, and OSCC variants. The objective was to assess whether CK17 expression patterns provide additional diagnostic value in distinguishing OLP from epithelial dysplasia and malignant transformation in oral epithelial lesions.

Materials and Method

Study Design and Sample Selection

The present study was approved by the Ethics Committee of Shahid Beheshti University of Medical Sciences (IR.SBMU.DRC.REC.1403.204). A total of 69 paraffin-embedded tissue blocks were retrospectively collected from the archives of the Department of Oral and Maxillofacial Pathology, School of Dentistry, Shahid Beheshti University of Medical Sciences, and a private oral pathology service. However, some blocks contained more than one histopathologically distinct area (e.g., adjacent normal or dysplastic epithelium), resulting in 88 evaluable tissue sections for IHC assessment. The samples included 43 females (62.3%) and 26 males (37.6%). The mean age of the participants was 57.25 ± 14.68 years (range: 18–94 years). The samples were located in the following sites: tongue (37.6%), buccal mucosa (33.3%), gingiva (11.6%), lip (7.2%), palate (5.8%), and floor of the mouth (4.3%). For statistical analysis, specimens were categorized into seven diagnostic groups: control epithelium including irritation fibroma and adjacent normal epithelium from OSCC lesions (n=18), OLP (n=19, 11 reticular, 8 erosive), low-grade dysplasia (n=4), high-grade dysplasia (n=15), conventional SCC (well-differentiated type) (n=23), papillary SCC (n=6), and verrucous carcinoma (n=3). These seven groups were used for all intergroup comparisons of CK17 expression. Papillary SCC and verrucous carcinoma are low-grade variants of conventional SCC and are associated with a better prognosis [19]. Dysplastic lesions

were further categorized using the binary grading system (low-risk and high-risk dysplasia) to facilitate comparative analysis. Control epithelium included both histologically normal epithelium adjacent to OSCC lesions and reactive soft tissue lesions (irritation fibroma). This approach was used to obtain non-dysplastic comparison tissues and to minimize the potential impact of field cancerization effects in tumor-adjacent mucosa, which may harbor molecular alterations despite normal histological appearance. As this investigation was designed as a retrospective archival study, all available and eligible cases within the defined study period (2021-2025) were consecutively included.

Inclusion and Exclusion Criteria

Eligible samples included those with a confirmed histopathological diagnosis of the aforementioned lesions. For OLP, cases that had a clinically bilateral presentation were included in the study. Samples were excluded if they had inadequate tissue, poor fixation quality, or incomplete records.

IHC

IHC staining was performed on 4- μ m-thick sections using the EnVision detection system. The sections were deparaffinized and rehydrated through graded ethanol, followed by blocking endogenous peroxidase activity for 5 minutes at room temperature.

Antigen retrieval was carried out in Tris-EDTA buffer (pH 9.0) at 100°C for 40 minutes using a pressure cooker. After rinsing with deionized water and washing with TBS, the sections were incubated for 30 minutes at ambient temperature with the primary antibody: rabbit monoclonal anti-human cytokeratin 17 (clone EP98, dilution 1:50, IgG isotype, Master Diagnostica, Spain).

Following another TBS wash, sections were treated with the secondary detection polymer for 30 minutes at room temperature and visualized using 3,3'-diaminobenzidine (DAB) as the chromogen for 5 minutes. Slides were then counterstained with hematoxylin, rinsed, and mounted.

A normal salivary gland served as the positive control, while the negative control was prepared by replacing the primary antibody with a non-immune serum. CK17 immunoreactivity was localized to the cytoplasm and cell membrane.

Evaluation of Immunostaining

All stained slides were independently examined by an

oral pathologist (S.A.M) under a light microscope in a blinded manner. Only epithelial cell staining was considered for CK17 evaluation. Stromal inflammatory cells and other non-epithelial elements were excluded from scoring. CK17-positive epithelial cells were quantified according to the method described by Tojyo *et al.* [20], which was based on the scoring system originally proposed by Kitamura *et al.* [7]. Positive cells were counted in at least three randomly selected high-power fields at $\times 200$ magnification. Sections with $<5\%$ reactive cells were considered negative, whereas those with $\geq 5\%$ reactive cells were classified as positive. Positive cases were further categorized as weak ($<60\%$ positive cells) or strong ($\geq 60\%$ positive cells). These thresholds were adopted a priori from previously published CK17 IHC studies to ensure methodological consistency and comparability with the existing literature.

Statistical Analysis

Given the ordinal nature of CK17 expression scores and non-normal distribution of data, nonparametric tests were applied. Intergroup comparisons were performed using the Kruskal–Wallis test, followed by pairwise comparisons with Bonferroni correction for multiple testing.

Results

In this study, about 4.5% of the lesions did not display CK17 expression. In OLP and high-grade dysplasia, the entire epithelium from basal cells to the superficial layer was stained. All reticular and erosive types of OLP strongly expressed the CK17 marker ($>60\%$). In OSCC groups, almost all nests, large islands, sheets, and indi-

vidual cells were positive with or without keratin pearl formation. Low-grade dysplasia and control groups revealed patchy expression (Figures 1-4). A statistically significant difference in score distribution was observed across diagnostic groups (Kruskal–Wallis $H(6)=80.87$, $p < 0.001$). The diagnosis-by-score cross tabulation (Table 1) summarizes the distribution of cases across three score categories ($<5\%$, $5\text{--}60\%$, and $>60\%$) within each diagnostic group. SCC (23/23), papillary SCC (6/6), high-grade dysplasia (15/15), OLP (19/19), and verrucous carcinoma (3/3) were entirely classified in the $>60\%$ category (strong expression), whereas all low-grade dysplasia cases (4/4) fell in the $5\text{--}60\%$ category (weak expression). In the control group, cases were distributed across all three categories ($<5\%$: 4/18; $5\text{--}60\%$: 13/18; $>60\%$: 1/18). Figures 1-4 show CK17 expression in various lesions. Overall, 67 of 88 evaluated samples (76.1% of the total cohort) were classified as showing strong CK17 expression. Strong CK17 expression was observed in OLP, high-grade dysplasia, and all malignant lesions, whereas low-grade dysplasia showed weak expression (Table 1). Furthermore, pairwise post-hoc comparisons with Bonferroni adjustment revealed several significant differences between diagnostic groups. After adjustment, low-grade dysplasia differed significantly from SCC (Adj. $p = 0.001$), papillary SCC (Adj. $p = 0.013$), high-grade dysplasia (Adj. $p = 0.002$), and OLP (Adj. $p = 0.001$). No significant difference was observed between low-grade dysplasia and the control group (Adj. $p = 1.000$).

Both the normal epithelium adjacent to the tumor and the epithelium associated with the irritation fibroma

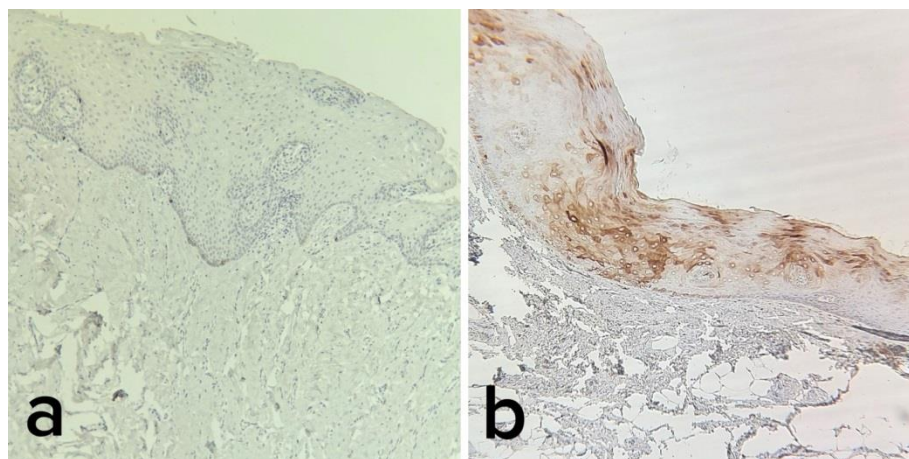


Figure 1: **a:** Control group showing negative expression of CK17 (IHC 100 $\times$). **b:** Low-grade dysplasia showing weak CK17 expression (IHC 100 $\times$)

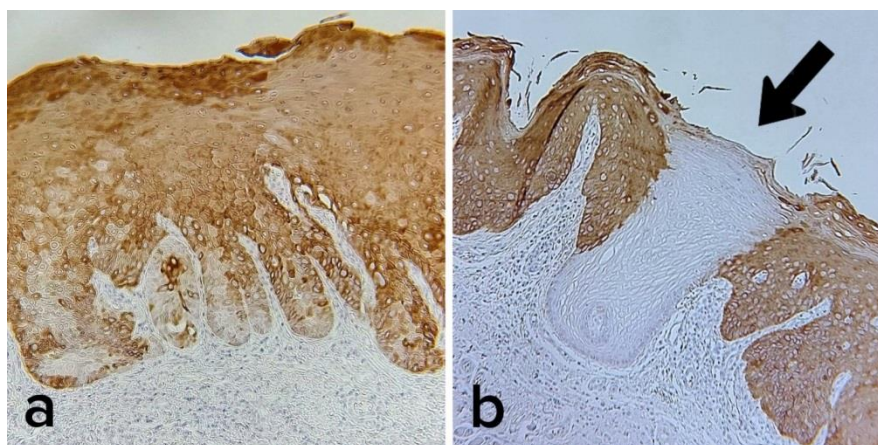


Figure 2: High-grade dysplasia, **a:** CK17-positive cells showing cytoplasmic brown staining extending from the basal layer to the superficial layer (IHC 100 $\times$), **b:** Adjacent non-dysplastic epithelium showing no expression of CK17 (arrow) (IHC 100 $\times$)

showed almost similar results. Irritation fibroma cases ($n=7$) showed the following distribution: score 2 ($n=1$), score 1 ($n=5$), and score 0 ($n=1$). Adjacent normal epithelium ($n=11$) showed score 0 ($n=3$) and score 1 ($n=8$).

The control group differed significantly from SCC (Adj. $p < 0.001$), papillary SCC (Adj. $p < 0.001$), high-grade dysplasia (Adj. $p < 0.001$), OLP (Adj. $p < 0.001$), and verrucous carcinoma (Adj. $p = 0.008$). After Bonferroni correction, no significant difference was observed between OLP and SCC (Adj. $p = 1.000$) or between OLP and high-grade dysplasia (Adj. $p = 1.000$).

Discussion

CK17 expression demonstrated a clear association with the severity of epithelial alteration in the present study. Strong CK17 positivity was observed in high-grade dysplasia and all malignant lesions, whereas low-grade dysplasia showed expression scores comparable to those

in the normal epithelium. Some authors have noted that normal epithelium adjacent to the tumor may not be reliable due to the field cancerization phenomenon [2]. To solve this problem, we used both the normal epithelium adjacent to the tumor and the epithelium of reactive soft tissue lesions (irritation fibroma). CK17 expression increases with the progression of epithelial changes and is more closely associated with advanced dysplastic and malignant transformation than with early epithelial alterations. Similar findings have been reported in oral and laryngeal epithelial lesions, where CK17 positivity is predominantly detected in high-grade dysplasia and carcinoma in situ but is rarely observed in low-grade lesions [2, 14]. These findings support the role of CK17 as a marker associated with higher grades of epithelial dysplasia and malignant transformation. In this study, CK17 expression was consistently observed in all malignant lesions, including conventional

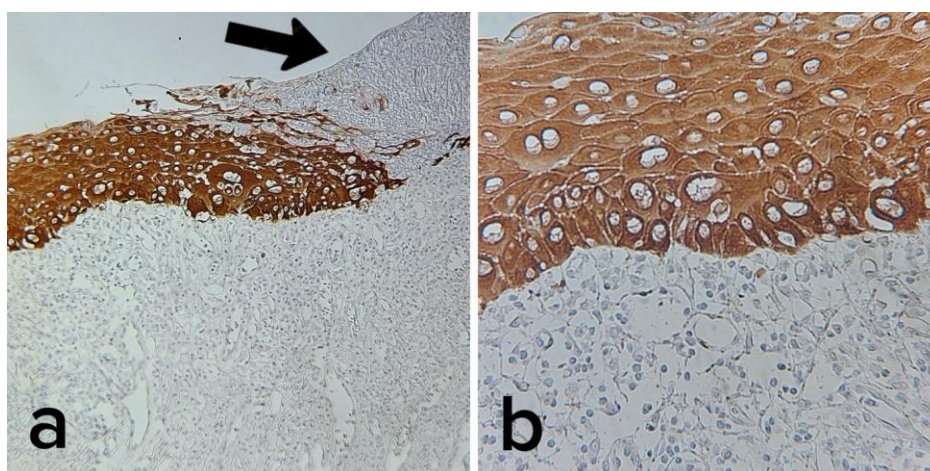


Figure 3: Oral lichen planus. **a:** Erosive form showing CK17 expression throughout the epithelium. Arrow indicates fibrinopurulent membrane (IHC 100 $\times$), **b:** Reticular form showing CK17 expression (IHC 400 $\times$)

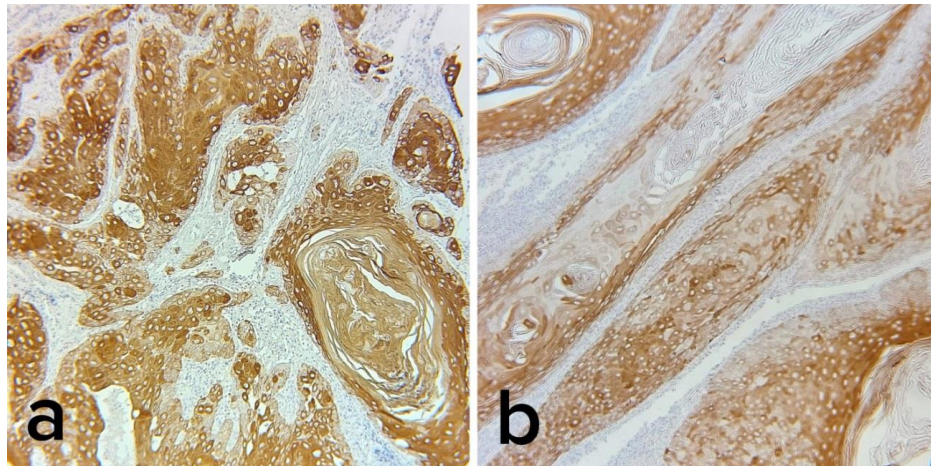


Figure 4: Squamous cell carcinoma. **a:** Conventional SCC showing CK17-positive cells with cytoplasmic brown staining in nests and islands (IHC 100×), **b:** Papillary SCC showing strong CK17 expression (IHC 100×)

Table 1: Statistical analysis of CK17 expression between all lesions and control groups

Diagnosis		Score			Total
		<5%	5%-60%	>60%	
Squamous cell carcinoma	Count	0	0	23	23
	% within diagnosis	0.0%	0.0%	100.0%	100.0%
Papillary squamous cell carcinoma	Count	0	0	6	6
	% within diagnosis	0.0%	0.0%	100.0%	100.0%
Low grade dysplasia	Count	0	4	0	4
	% within diagnosis	0.0%	100.0%	0.0%	100.0%
High grade dysplasia	Count	0	0	15	15
	% within diagnosis	0.0%	0.0%	100.0%	100.0%
Lichen planus	Count	0	0	19	19
	% within diagnosis	0.0%	0.0%	100.0%	100.0%
Control	Count	4	13	1	18
	% within diagnosis	22.2%	72.2%	5.6%	100.0%
Verrucous carcinoma	Count	0	0	3	3
	% within diagnosis	0.0%	0.0%	100.0%	100.0%
Total	Count	4	17	67	88
	% within diagnosis	4.5%	19.3%	76.1%	100.0%

well-differentiated SCC, papillary SCC, and verrucous carcinoma. The uniform positivity across different histopathological variants indicates that CK17 expression is maintained regardless of tumor subtype. Previous studies have similarly demonstrated increased CK17 expression in OSCC compared to normal mucosa, supporting its association with malignant epithelial transformation [2, 6-7]. Due to the absence of moderately and poorly differentiated SCC cases, the association between CK17 expression and tumor grade could not be

further evaluated in the present study.

Furthermore, higher CK17 expression has been reported as a potential predictor of resistance to immune checkpoint blockade in SCC, suggesting a role in tumor immune modulation [8]. Overall, these findings suggest that CK17 expression is associated with biologically active and clinically significant epithelial alterations.

OLP represents a diagnostically challenging lesion in oral pathology [14]. The histopathological distinction between OLP and epithelial dysplasia is not always straightforward [16]. In fact, some cases of epithelial dysplasia may have histopathological features similar to OLP, which is known as OLD. This disease can be easily confused with OLP and presents a real potential for malignancy [14, 16]. The highly chronic inflammatory process present in OLP may lead to cellular abnormalities similar to those seen in epithelial dysplasia, making the diagnosis more difficult [15]. In addition, lesions such as OLD may mimic OLP both clinically and histopathologically, further complicating accurate diagnosis [13, 16]. For this reason, adjunctive IHC markers have been proposed to improve diagnostic accuracy beyond conventional morphological assessment [14]. A major focus of the present study was evaluating CK17 expression in OLP. In our study, CK17 expression was strongly positive in all OLP cases, demonstrating diffuse positivity across the full thickness of the epithelium, consistent with previous reports [12]. Expression scores were comparable to those observed in high-grade dysplasia and malignant lesions. This overlapping expression pattern across immune-mediated, premalignant, and malignant lesions suggests that CK17 expression

lacks sufficient specificity to differentiate OLP from dysplastic or neoplastic changes. Han *et al.* [12] reported a relationship between CK17 expression and OLP severity. They found higher CK17 scores in erosive OLP than in non-erosive forms. In the present study, both forms of OLP (reticular and erosive) showed a high expression of this protein, suggesting that CK17 expression may not directly correlate with clinical subtype or severity in OLP.

Although CK17 showed a high expression in dysplastic lesions and SCC variants, its similar expression in OLP proposes that CK17 alone does not provide sufficient diagnostic specificity to differentiate OLP from dysplastic transformation. CK17 expression has been reported to be closely linked to stress responses, and individuals with OLP exhibit a higher prevalence of stress, suggesting that elevated CK17 expression in OLP may reflect stress-related epithelial activation rather than being a specific marker of malignant progression [12].

Likewise, previous reports indicate that CK17 expression in leukoplakia is variable and may not reliably differentiate these lesions from normal mucosa when used alone [7]. Similarly, in another study, high CK17 expression was reported in both oral SCC and benign pseudoepitheliomatous hyperplasia, indicating that increased CK17 expression is not exclusively restricted to malignant lesions [21]. Accordingly, the diagnostic value of CK17 may be enhanced when assessed with additional markers, such as cytokeratin 13 (CK13), particularly in diagnostically challenging lesions. In this context, the reciprocal switching between CK13 and CK17 may provide additional insight. It has been suggested that epithelial transformation is accompanied by a shift from CK13 positivity to CK17 expression; however, this transition appears only partially complete in OLP, whereas a complete switch is observed in malignant lesions [21]. Although the mechanism of CK17 involvement in the pathogenesis of OLP remains unknown [12], the transition from CK13 to CK17 in epithelial cells affected by OLP proposes that these cells were either activated or damaged by lymphocytic infiltration. However, they were not involved in a neoplastic process, as they lacked other IHC profile factors indicating malignancy [22]. Accordingly, the combined evaluation of CK13 and CK17 has been proposed to improve diagnostic discrimination in oral epithelial lesions [7, 23].

The present study has certain limitations, including the relatively small sample size in some diagnostic subgroups and the evaluation of a single IHC marker (CK17) without the inclusion of additional complementary biomarkers. Future studies with larger, prospectively designed cohorts are recommended to further validate the diagnostic and biological role of CK17 in oral epithelial lesions.

Conclusion

CK17 expression was strongly positive in high-grade dysplasia and in all examined malignant lesions, including SCC, papillary SCC, and verrucous carcinoma, supporting its role as a marker of advanced epithelial alteration. However, its similarly high expression in OLP indicates that CK17 lacks sufficient specificity to reliably distinguish immune-mediated epithelial alterations from dysplastic transformation when used alone. On the other hand, the similarity in the expression of this protein in OSCC and OLP suggests CK17 might actually be a marker of hyperproliferation and stress rather than malignant transformation. Therefore, CK17 may be useful in identifying lesions with higher malignant potential, but combined evaluation with additional biomarkers may be required to improve diagnostic accuracy.

Acknowledgments

None

Conflict of interest

None declared.

References

- [1] Gyanchandani A, Shukla S, Vagha S, Acharaya S, Kadu RP. Diagnostic Utility of Cytokeratin 17 Expression in Oral Squamous Cell Carcinoma: A Review. *Cureus*. 2022; 14: e27041.
- [2] Sanguansin S, Kosanwat T, Juengsomjit R, Poomsawat S. Diagnostic Value of Cytokeratin 17 during Oral Carcinogenesis: An Immunohistochemical Study. *Int J Dent*. 2021; 2021: 4089549.
- [3] Lalli A, Tilakaratne WM, Ariyawardana A, Fitchett C, Leigh IM, Hagi-Pavli E, *et al.* An altered keratinocyte phenotype in oral submucous fibrosis: correlation of keratin K17 expression with disease severity. *J Oral Pathol Med*. 2008; 37: 211-220.

- [4] Hassan SS, Attia MA, Attia AM, Nofal RA, Fathi A. Distribution of Cytokeratin 17 in the Parenchymal Elements of Rat's Submandibular Glands Subjected to Fractionated Radiotherapy. *Eur J Dent*. 2020; 14: 440-447.
- [5] Toyoshima T, Vairaktaris E, Nkenke E, Schlegel KA, Neukam FW, Ries J. Cytokeratin 17 mRNA expression has potential for diagnostic marker of oral squamous cell carcinoma. *J Cancer Res Clin Oncol*. 2008; 134: 515-521.
- [6] Wei KJ, Zhang L, Yang X, Zhong LP, Zhou XJ, Pan HY, et al. Overexpression of cytokeratin 17 protein in oral squamous cell carcinoma in vitro and in vivo. *Oral Dis*. 2009; 15: 111-117.
- [7] Kitamura R, Toyoshima T, Tanaka H, Kawano S, Kiyosue T, Matsubara R, et al. Association of cytokeratin 17 expression with differentiation in oral squamous cell carcinoma. *J Cancer Res Clin Oncol*. 2012; 138:1299-1310.
- [8] Lozar T, Laklouk I, Golfinos AE, Gavrielatou N, Xu J, Flynn C, et al. Stress Keratin 17 Is a Predictive Biomarker Inversely Associated with Response to Immune Check-Point Blockade in Head and Neck Squamous Cell Carcinomas and Beyond. *Cancers (Basel)*. 2023; 15: 4905.
- [9] Atarbashi-Moghadam S, Niazmand M, Vafadar S, Gholami Toghchi S. Comparison of Mast Cell Count in Oral Epithelial Dysplasia and Squamous Cell Carcinoma. *J Dent (Shiraz)*. 2024; 25: 369-373.
- [10] Eftekhari Moghadam P, Atarbashi-Moghadam S. CD1a Expression in Oral Dysplastic Lesions: An Immunohistochemical Analysis. *J Dent (Shiraz)*. 2026; 27: 276-282.
- [11] Alarcón I, Alarcón C, Arellano J. Overview of lichen planus pathogenesis and review of possible future therapies. *JAAD Reviews*. 2025; 3: 146-154.
- [12] Han X, Zhao R, Zhang Q, Shen X, Sun K. Increased expression of keratin 17 in oral lichen planus and its correlation with disease severity. *J Dent Sci*. 2024; 19: 1525-1532.
- [13] Bajpai M, Agarwal D, Bhalla A, VatchalaRani RM, Kumar M. Unilateral lichen planus: A rare case report. *J Nat Sci Biol Med*. 2014; 5: 453-455.
- [14] Wu CL, Huang CC, Wu SY, Jiang SS, Tsai FY, Hsiao JR. A new scoring system facilitating diagnosis of oral squamous malignancy on biopsy specimens. *BMC Oral Health*. 2022; 22: 165.
- [15] Sousa FA, Paradella TC, Brandão AA, Rosa LE. Oral lichen planus versus epithelial dysplasia: difficulties in diagnosis. *Braz J Otorhinolaryngol*. 2009; 75: 716-720.
- [16] Ono J, Okada Y, Kanri Y, Sano H, Hasegawa H. Immunohistochemical study of differential expressions of CAR, E-Cadherin, CK-13,-17, p53 and Ki-67 in oral lichen planus, lichenoid lesion and lichenoid epithelial dysplasia. *J Hard Tissue Biology*. 2021; 30: 355-362.
- [17] Tsushima F, Sakurai J, Uesugi A, Oikawa Y, Ohsako T, Mochizuki Y, et al. Malignant transformation of oral lichen planus: A retrospective study of 565 Japanese patients. *BMC Oral Health*. 2021; 21: 298.
- [18] Kakoei S, Torabi M, Rad M, Karbasi N, Mafi S. Retrospective Study of Oral Lichen Planus and Oral Lichenoid Lesions: Clinical Profile and Malignant Transformation. *J Dent*. 2022; 23: 452.
- [19] Atarbashi-Moghadam S, Lotfi A, Roghanizadeh L, Mirebeigi Jamasbi SS, Akbarzadeh Fathabadi Z. Infrequent Histopathologic Subtypes of Oral Squamous Cell Carcinoma: A Case Series with Emphasis on Histopathologic Characteristics. *J Dent (Shiraz)*. 2025; 26: 186-193.
- [20] Tojyo I, Shintani Y, Nakanishi T, Okamoto K, Hiraishi Y, Fujita S, et al. PD-L1 expression correlated with p53 expression in oral squamous cell carcinoma. *Maxillofac Plast Reconstr Surg*. 2019; 41: 56.
- [21] Al-Eryani K, Karasneh J, Sedghizadeh PP, Ram S, Sawair F. Lack of Utility of Cytokeratins in Differentiating Pseudocarcinomatous Hyperplasia of Granular Cell Tumors from Oral Squamous Cell Carcinoma. *Asian Pac J Cancer Prev*. 2016; 17: 1785-1787.
- [22] Abé T, Kitagawa N, Yoshimoto S, Maruyama S, Yamazaki M, Inai T, Hashimoto S, Saku T. Keratin 17-positive Civatte bodies in oral lichen planus-distribution variety, diagnostic significance and histopathogenesis. *Sci Rep*. 2020; 10: 14586.
- [23] Kanemoto H, Obata K, Umemori K, Hasegawa K, Ono S, Ono K, Yutori H, Ibaragi S. Multimodal Prediction of Cervical Lymph Node Metastasis and Recurrence in Oral Squamous Cell Carcinoma. *Anticancer Res*. 2023; 43: 4993-5001.