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PD-L1 expression in oral squamous cell carcinoma: Clinicopathological correlation with histological grade using TPS and CPS scoring systems

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Abstract:
Oral squamous cell carcinoma (OSCC) is a common malignancy with high morbidity and mortality, and PD-L1 has emerged as a potential predictive biomarker for immunotherapy. In this prospective cross-sectional study, 84 histologically confirmed OSCC cases were evaluated for PD-L1 expression using immunohistochemistry and scored by Tumor Proportion Score (TPS) and Combined Positive Score (CPS), with tumor-infiltrating lymphocytes and tumor budding also assessed. PD-L1 positivity was observed in 68% of cases by TPS and 77% by CPS, with a significant association between PD-L1 expression and histological grade (TPS: $p = 0.022$; CPS: $p = 0.016$), while no significant association with survival was identified during follow-up. The highest PD-L1 expression was noted in moderately differentiated tumors, supporting a relationship between PD-L1 expression and tumor differentiation. Thus, we show that PD-L1 is frequently expressed in OSCC and may serve as a valuable predictive biomarker for immunotherapy, although further studies with larger cohorts and longer follow-up are needed to establish its prognostic significance.

Keywords: Oral squamous cell carcinoma, PD-L1, tumor proportion score (TPS), combined positive score (CPS), tumor budding, tumour infiltrating lymphocytes

Background:
Oral squamous cell carcinoma (OSCC) is the most common tumor of the oral cavity, with an increasing incidence worldwide, with up to 370,000 new cases per year **[1, 2].** Oral cavity cancer represents the 16th most common malignant neoplasm globally, with close to 188,000 cancer-related deaths annually **[3].** In India, oral cancer accounts for approximately 77,000 new cases and 52,000 deaths each year, representing nearly one-fourth of the global incidence **[4, 5].** The programmed-death 1 receptor (PD-1)/programmed-death ligand 1 (PD-L1, also called B7-H1, and CD274) pathway regulates T-cell responses and the balance between T-cell activation and tolerance, and acts as an immunologic checkpoint. The PD-1/PD-L1 pathway regulates T-cell responses and maintains the balance between activation and tolerance, acting as an important immunologic checkpoint **[6, 7].** PD-L1 is rarely expressed in normal tissues, but it is induced in tumor, which makes PD-L1 pathway different from other co-inhibitory pathways**.** It suggests that the selective expression of PD-L1 may be related to the clinical prognosis of cancer patients and can be used as a selective target for anti-tumor therapy **[8, 9].** In fact, antibodies blocking PD-1 and PD-L1 have demonstrated well responses in several different advanced malignancies **[10].** PD1/PDL1 expression can be determined by IHC**.** Tumor cells and inflammatory cells both can show PDL1 expression**.** Membranous staining in the tumor cells and membranous or cytoplasmic staining in the inflammatory cells considered positive. To score the PD1 expression, there are two scoring system widely used: Tumor proportion score and CPS (Combined Positive Score. CPS (Combined Positive Score) is defined as the number of PD-L1 positive cells TC (tumour cells) with membrane staining; IC (inflammatory cells) cells with membranous and/or cytoplasmic staining), divided by number of viable tumour cells and then multiplied by 100**.** TPS (Tumor Proportion Score) is the percentage of viable tumour cells showing partial or complete membranous staining relative to all viable tumour cells present in the sample**.** PDL1 expression can be used as predictive biomarker for avail immunotherapy. CPS score is widely used to access the PDL1 expression **[11].** A large cancer audit study including PD-L1 expression in oral cavity cancer was conducted by Peelay *et al.* **[12]** at tertiary cancer center of India. Therefore, it is of interest to evaluate PD-L1 expression in oral squamous cell carcinoma using both Tumor Proportion Score (TPS) and Combined Positive Score (CPS) systems and to correlate these scores with histological grade and other clinicopathological parameters.

Materials and Methods:
Study participants:
This prospective cross-sectional study was conducted after obtaining ethical approval from the Institutional Ethics Committee (IEC), AIIMS Rajkot. All patients with histologically confirmed oral squamous cell carcinoma (OSCC) presenting to the outpatient or inpatient departments during the study period were included. Cases with non-confirmatory or inconclusive

biopsies, squamous cell carcinomas from non-oral mucosal sites, and post-therapy OSCC were excluded.

Clinical and pathological data:
Detailed clinical history and demographic data were recorded for all confirmed OSCC cases. Histopathological evaluation and PD-L1 immunohistochemistry were performed and reviewed by at least two consultant histopathologists. Tumor budding and tumor-infiltrating lymphocytes (TILs) were also assessed for correlation with tumor grade and other clinicopathological parameters.

Immunohistochemistry:
PD-L1 immunostaining was performed manually using rabbit monoclonal antibody (Clone B7H1P) following standard protocol. A minimum of 100 viable tumor cells was required for adequacy. Tumor cells showing any visible membranous staining (partial or complete) were considered positive. Tumor-associated immune cells (inflammatory cells) in the tumor or surrounding stroma showing membranous or cytoplasmic staining were also counted. PD-L1 expression was scored using both Tumor Proportion Score (TPS) and Combined Positive Score (CPS) systems with a ≥1% cut-off for positivity. TPS was calculated as the percentage of viable tumor cells showing partial or complete membranous staining. CPS was determined as the number of PD-L1-positive tumor cells and immune cells divided by the number of viable tumor cells, multiplied by 100. PD-L1 expression (TPS) was categorized as negative (<1%), positive (1–19%), and high (≥20%).

Tumor-infiltrating lymphocytes (TILs) and tumor budding:
TILs were assessed within the tumor boundaries by quantifying only mononuclear cells (lymphocytes and plasma cells), excluding neutrophils, dendritic cells, and macrophages in necrotic areas. TIL levels were categorized as low (≤30%), moderate (30–70%), and high (>70%). Tumor budding was evaluated according to the 2016 International Tumor Budding Consensus Conference (ITBCC) guidelines. Tumor budding was defined as a single tumor cell or a cluster of ≤4 tumor cells infiltrating the stroma. Budding was graded as Bd1 (0–4 buds), Bd2 (5–9 buds), and Bd3 (≥10 buds).

Data analysis:
Data were compiled and analyzed using SPSS version 27 (IBM). Descriptive statistics were used for demographic and clinical variables. Associations between PD-L1 expression and clinicopathological parameters were evaluated using chi-square test, Fisher-Freeman-Halton exact test, and Pearson correlation. A two-sided p-value <0.05 was considered statistically significant.

Results:
A total of 84 cases of oral squamous cell carcinoma (SCC) were included in the study. The cohort showed a marked male predominance, with males accounting for 74/84 (88.09%) and females 10/84 (11.9%). The mean age of the patients was 49.27 ± 11.34 years, with ages ranging from 27 to 81 years, indicating that the disease predominantly affected middle-aged individuals. The distribution showed a fairly symmetrical spread, with a median age of 49 years. The most common tumor site was the buccal mucosa (51 cases, 60.71%), and followed by the tongue (20 cases, 23.80%). Other sites included the lip (4.76%), retromolar trigone (RMT) region (4.76%), tonsillar region (3.57%), and hard palate/floor of mouth (1.19% each). The majority of lesions were unifocal at presentation (75 cases, 89.28%), with multifocal involvement being uncommon. A significant proportion of patients had a positive history of tobacco consumption (67 cases, 79.76%), either in smoked or smokeless forms, reinforcing its strong etiological association. Clinically, the lesions most commonly presented as ulcerative growths (52 cases, 61.90%) followed by proliferative or exophytic masses (23 cases, 27.38%) while flat or mixed-type lesions (9 cases.10.71%) were relatively less frequent. Regional lymph node enlargement was noted in a subset of cases (18 cases, 21.42%), suggestive of locoregional spread, although many patients did not show clinically palpable lymphadenopathy at the time of presentation. Most of the tumors were moderately differentiated squamous cell carcinoma (MDSCC) in 59 cases (70.23%) followed by well differentiated SCC (WDSCC) in 20 cases (23.80%) and poorly differentiated SCC (PDSCC) in 05 cases (5.9%). TILs were common, with the majority of cases grouped at 25-40%, consistent with an intermediate immune response in most tumors. TILs presented a mean value of 25.3%, reflecting a moderate immune infiltration in most tumors, with a wide variability (from 5% to 50%). The most common values were 25% (n=26), 30% (n=24) and 40% (n=19). Tumor budding had a mean of 1.89, with most cases clustered between low to intermediate budding activity. The relatively wide standard deviations indicate heterogeneity in tumor biology among cases **(Table 1)**. PD-L1 expression was assessed using both Tumor Proportion Score (TPS) and Combined Positive Score (CPS) in 84 cases. Regarding TPS, 26 cases (30.9%) show <1% staining, 28 cases show 1- <20% staining and 13 cases show 20- <50% staining and 17 cases show ≥50% staining. Regarding CPS, 19 cases show <1% staining, 28 cases show 1- <20% staining and 19 cases show 20- <50% staining and 18 cases show ≥50% staining **(Figure 1 and Table 2)**. TPS scoring in moderately differentiated squamous cell carcinoma (MDSCC), demonstrated higher PD-L1 expression, with 26 out of 59 cases (44.1%) showing high expression (≥50%), followed by 20 cases (33.9%) with low expression and 13 cases (22.0%) being negative. On the other hand, WDSCC showed a predominantly negative expression of PD-L1 with 11 of 20 cases (55.0%) being negative, while only 3 cases (15.0%) showed a high expression. Poorly differentiated squamous cell carcinoma (PDSCC) was a small subset (n=5) with the majority showing negative expression (60.0%) and only one in both low and high expression group. CPS scoring in MDSCC, most of the cases expressed high CPS expression. A total of 32/59 (54.2%) cases had high CPS, 17 (28.8%) had low and 10 (16.9%) were negative. In WDSCC, most cases exhibited minimal expression of CPS (10/20; 50.0%), and only 4 cases

(20.0%) displayed increased expression. There is a statistically significant association between PD-L1 expression (TPS categories) and Tumor grade in Oral SCC (Fisher–Freeman–Halton exact test, p = 0.022). Higher PD-L1 expression was more frequently seen in moderately and poorly differentiated squamous cell carcinomas, whereas well-differentiated tumors predominantly showed low or negative expression. Statistical analysis demonstrated a significant association between CPS-based PD-L1 expression and tumor grade. Although the Pearson Chi-square test showed significance (p = 0.023), the Fisher–Freeman–Halton exact test was considered more appropriate and confirmed a statistically significant association (p = 0.016). Out of a total of 84 cases of Oral SCC, follow-up was conducted in 44 cases (52.3%). Remission was observed in 25 cases (29.8%), making it the most common documented clinical outcome in the dataset. These cases were distributed across different grades but were predominantly seen in moderately differentiated tumors. Recurrence was noted in only 3 cases (3.6%), representing the least frequent outcome. The small number of recurrence events suggests either effective initial management in most patients or limited follow-up data for recurrence assessment. Death was recorded in 16 cases (19.0%), indicating a significant proportion of adverse outcomes. These cases were more commonly associated with higher PD-L1 expression levels and were largely seen in moderately differentiated squamous cell carcinoma (MDSCC), suggesting a possible link with more aggressive disease behavior. **(Table 3)** death was observed across all PD-L1 expression categories for both TPS and CPS, without a consistent increasing trend with higher expression levels. A relatively higher proportion of deaths were noted in the TPS-negative group, while CPS categories showed a more even distribution. Overall, PD-L1 expression did not demonstrate a clear association with mortality in this cohort.

Table 1: Descriptive statistics of continuous variables

Variable	Mean ± SD	Median	Min	Max
Age (years)	49.27 ± 11.34	49	27	81
TILs (%)	25.30 ± 11.52	25	5	50
Tumor Budding (40×)	1.89 ± 1.40	2	0	8

Table 2: Association of tumour grade with TPS & CPS categories on IHC

Oral SCC Grade	TPS Negative (<1%)	TPS Low (1–19%)	TPS High (≥20%)	Total	CPS Negative (<1%)	CPS Low (1–19%)	CPS High (≥20%)	Total
MDSCC	13 (22.0%)	20 (33.9%)	26 (44.1%)	59	10 (16.9%)	17 (28.8%)	32 (54.2%)	59
PDSCC	3 (60.0%)	1 (20.0%)	1 (20.0%)	5	3 (60.0%)	1 (20.0%)	1 (20.0%)	5
WDSCC	11 (55.0%)	6 (30.0%)	3 (15.0%)	20	6 (30.0%)	10 (50.0%)	4 (20.0%)	20
Total	27	27	30	84	19	28	37	84

Table 3: Association of death with PD-L1 expression (TPS and CPS) in oral SCC

Outcome	TPS Negative (<1%)	TPS Low (1–49%)	TPS High (≥50%)	Total	CPS Negative	CPS Low	CPS High	Total
Death (n = 16)	8 (50.0%)	3 (18.8%)	5 (31.2%)	16	6 (37.5%)	5 (31.2%)	5 (31.2%)	16
Alive (n = 68)	19 (27.9%)	24 (35.3%)	25 (36.8%)	68	13 (19.1%)	23 (33.8%)	32 (47.1%)	68
Total (n = 84)	27	27	30	84	19	28	37	84

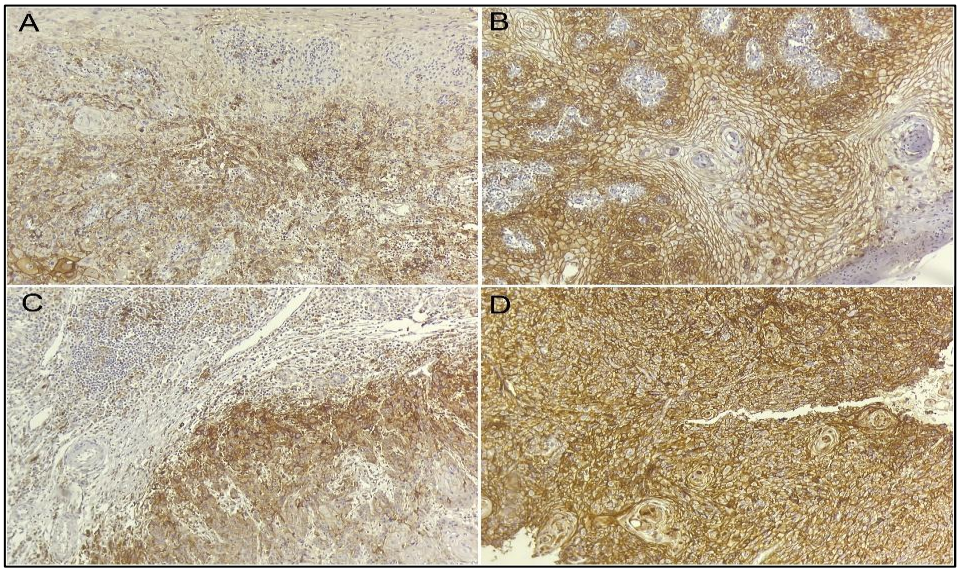


Figure 1: (A) PD-L1 IHC showing membranous staining with moderate intensity in invasive malignant squamous cells. Overlying benign squamous epithelium showing negative staining. (B) PD-L1 positivity in keratinizing squamous cell carcinoma with sparing of intervening stroma containing small blood vessels and inflammatory cells. (C) PD-L1 positivity involving both the malignant squamous cells (lower right) and immune cells (upper left). (D) Strong diffuse membranous positivity of tumour cells in a case of oral squamous cell carcinoma

Discussion:

Oral carcinoma is a major cause of cancer-related death worldwide. The Saurashtra region in Gujarat is notorious for widespread tobacco-chewing habits, including among youngsters. Immunotherapy has entered a new era with drugs targeting immune checkpoints such as programmed death receptor-1 (PD-1) and programmed death ligand-1 (PD-L1) **[13].** Inhibition of the PD-1/PD-L1 immune checkpoint pathway has emerged as a promising strategy for personalized cancer therapy. PD-1 is a key receptor involved in central and peripheral immune tolerance. Tumor-infiltrating lymphocytes (TILs) frequently express PD-1, providing a strong rationale for disrupting the PD-1/PD-L1 interaction to enhance anti-tumor responses. Consequently, pembrolizumab, a PD-1 inhibitor, is recommended as first-line therapy for unresectable, metastatic, recurrent, or treatment-resistant OSCC with PD-L1 overexpression **[14].** PD-L1 expression, evaluated by immunohistochemistry (IHC), is the most commonly used predictive biomarker for immunotherapy response in OSCC. High PD-L1 expression has been associated with clinical benefit in patients receiving anti-PD-1/PD-L1 therapy **[15, 16].** However, in untreated patients, high PD-L1 expression often correlates with poor prognosis due to immune evasion, aggressive tumor phenotype, and T-cell exhaustion **[17].** Previous studies have reported PD-L1 positivity rates in head and neck squamous cell carcinoma (HNSCC) ranging from 29.5% to 87% **[18-20].** Tumour budding is defined as isolated single cancer cells or small clusters (≤4 cells) in the tumor stroma, most prominent at the invasive front**.** It represents epithelial-mesenchymal transition and is an adverse prognostic feature in many solid tumors **[21, 22].** Although not yet included in the College of American Pathologists (CAP) protocol for oral carcinoma, the International Tumor Budding Consensus Conference (ITBCC) 2016 highlighted its prognostic value in OSCC and recommended its incorporation into reporting **[23].** The present study correlated tumor budding with histological grade and PD-L1 expression using TPS and CPS scoring systems. The tumor microenvironment, particularly TILs and tumor budding, plays a critical role in tumor progression and has led to major therapeutic advances **[24].** The International Immuno-Oncology Biomarkers Working Group has standardized TIL assessment in routine H&E sections for various malignancies **[25].** However, its application in oral malignancies remains limited. Tumor budding is associated with poor prognosis and aggressive behavior and has been studied in several cancers **[26],** yet no universally accepted protocol exists for reporting TILs and tumor budding in OSCC. The present study shows significant correlation of PD-L1 staining with histologic grade; similar to findings by Li *et al* **[27]**. study. Another study by Fiedler *et al.* **[28]** indicated association of Higher PDL1 staining in OSCC cases arising in non-smokers and non-drinkers. Study by Srivastava *et al.* **[29]** showed significant association of higher TPS and CPS scoring with perineural invasion**.**

Conclusion:

PD-L1 expression is variably present in oral squamous cell carcinoma and shows a statistically significant association with histological grade by both TPS and CPS, with higher expression in moderately and poorly differentiated tumors. It does not correlate consistently with clinical outcome or mortality and therefore is not a reliable independent prognostic marker for survival. Standardized reporting protocols and larger multi-center prospective studies are required to validate PD-L1 (TPS/CPS), together with tumor budding and TILs, as integrated tools for risk stratification and personalized management of OSCC.

Implications:

This study advances current knowledge by providing prospective data from a high-burden Indian cohort on the concurrent application of both TPS and CPS scoring systems for PD-L1 in OSCC, demonstrating a statistically significant association with histological grade (highest expression in moderately differentiated tumors). It further integrates assessment of tumor budding (per ITBCC 2016 guidelines) and tumor-infiltrating lymphocytes. These findings support refined risk stratification and patient selection for immune-checkpoint inhibitor therapy. The significant association between PD-L1 expression and higher histological grades supports its role as a biomarker of tumor aggressiveness and for risk stratification. TPS and CPS scoring can help select patients likely to benefit from immune checkpoint inhibitors such as pembrolizumab in advanced or recurrent OSCC. Although PD-L1 did not show a definitive link with survival in this cohort, its integration with other clinicopathological parameters and tumor microenvironment markers (TILs and tumor budding) may improve its predictive value. This study highlights the need for standardized protocols and larger prospective studies. In resource-limited, high-burden settings, these markers can aid personalized treatment and better therapeutic decision-making. The present work also proposes integrating TILs, tumor budding, demographic parameters, and tumor grading for improved prognostication in OSCC.

Limitations:

This study has several limitations. The sample size (n=84) is modest for subgroup analyses, especially poorly differentiated SCC (only 5 cases). Follow-up data were available for only 52.3% of patients, limiting survival analysis. Manual IHC assessment, though performed by experienced pathologists, carries inherent inter-observer variability.

Advancement to knowledge:

This study advances knowledge by providing prospective data from a high-burden Indian cohort on concurrent TPS and CPS scoring of PD-L1 in OSCC, demonstrating its significant association with histological grade (highest in moderately differentiated tumors).

References:

[1] Chen M *et al. Cancer Med*. 2025 **14**:e70957. [PMID: 40347073]
[2] Liu X *et al. BMC Oral Health*. 2025 **25**:1779. [PMID: 41239378]
[3] Bray F *et al. CA Cancer J Clin*. 2024 **74**:229. [PMID: 38572751]
[4] Akashanand *et al. Oral Oncology*. 2024 **159**:107063. [PMID: 39357385]
[5] Ray CS & Gupta PC. *Oral Dis*. 2025 **31**:1439. [PMID: 38950053]
[6] Ortega MA *et al. J Mol Med*. 2024 **102**:987. [PMID: 38935130]
[7] Gao M *et al. Cancer Lett*. 2024 **28**:588. [PMID: 38401888]
[8] Soltani M *et al. Clin Exp Med*. 2025 **25**:106. [PMID: 40180653]
[9] Strati A *et al. Int J Mol Sci*. 2025 **26**:1235. [PMID: 39941003]
[10] Javed SA *et al. Front Immunol*. 2024 **15**:1383456. [PMID: 38660299]
[11] Jeong JS *et al. Pathology*. 2024 **56**:969. [PMID: 39261273]
[12] Peelay Z *et al. Indian J Surg Oncol*. 2026 **17**:152. [PMID: 41641425]
[13] Liu Y *et al. Oral Oncol.* 2024 **151**:106730. [PMID: 38380036]
[14] Al-Azzawi HMA *et al. J Pers Med*. 2025 **15**:126. [PMID: 40278305]
[15] Sim Y *et al. Future Oncol*. 2024 **20**:2869. [PMID: 38861311]
[16] Yamashita M. *Microbiol Immunol*. 2025 **69**:350. [PMID: 40221937]
[17] Wang M *et al. J Immunother Cancer*. 2024 **12**:e009617. [PMID: 39357980]
[18] Gangadhar P *et al. J Oral Maxillofac Pathol*. 2024 **28**:29. [PMID: 38800420]
[19] Singh A *et al. Indian J Surg Oncol.* 2024 **15:**816. [PMID: 39555374]
[20] Nalwa A *et al. Indian J Surg Oncol*. 2026 **17**:764. [PMID: 42023198]
[21] Jakkulwar S *et al. Cureus*. 2024 **16**:e69624. [PMID: 39429322]
[22] Borde ND *et al. Indian J Pathol Microbiol*. 2025 *68*:310. [PMID: 39420848]
[23] Kopecka K *et al. Oral Dis*. 2025 **31**:2119. [PMID: 39617996]
[24] Jansari TR *et al. J Cancer Res Ther*. 2026 **22**:104. [PMID: 41910293]
[25] Weeda *et al. Histopathology*. 2026 **88**:1126. [PMID: 41640295]
[26] Verma L *et al. Sultan Qaboos Univ Med J*. 2024 **24**:52. [PMID: 38434449]
[27] Li Y *et al. Oral Dis*. 2025 **31**:815. [PMID: 39092614]
[28] Fiedler M *et al. Head Neck*. 2025 **47:**822. [PMID: 39462876]
[29] Srivastava P *et al. Cureus*. 2025 **17**:e85186. [PMID: 40600093]

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