ORIGINAL ARTICLE

Expression of Desmoglein 3 (DSG 3) in different grades of oral squamous cell carcinoma and its correlation with lymph node metastasis

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ABSTRACT

Background/Objectives

Oral squamous cell carcinoma (OSCC) remains a major global health challenge, with lymph node metastasis representing the most critical prognostic determinant. Desmogleins, transmembrane adhesion proteins, have emerged as potential biomarkers in cancer progression, yet their precise roles in OSCC metastasis remain incompletely characterized. This study is aimed to evaluate the expression patterns of desmoglein 3 (DSG3) in OSCC and to investigate its association with clinicopathological parameters using immunohistochemical analysis.

Methods

This retrospective study included 80 surgically treated OSCC patients who underwent tumor resection with neck dissection. Formalin-fixed, paraffin-embedded tissue specimens were subjected to immunohistochemical staining for DSG3. Expression patterns were categorized based on staining intensity and correlated with clinicopathological variables.. Statistical associations were assessed using appropriate tests, with $p < 0.05$ considered significant.

Results

The cohort comprised predominantly male patients (80.3%) with a mean age of 48 ± 8 years. Advanced disease was prevalent, with 77.3% presenting as Stage IV and 84.8% demonstrating nodal involvement (N+). Buccal mucosa (34.8%) and tongue (30.3%) were the most common primary sites. Histological grades were evenly distributed. DSG3 expression demonstrated highly significant associations with nodal involvement ($p = 0.001$) and advanced pathological stage ($p = 0.001$).

Conclusions

DSG3 expression is significantly associated with lymph node metastasis and advanced pathological stage in OSCC, demonstrating superior predictive capacity compared to conventional histological grade. These findings support the potential utility of DSG3 as a molecular biomarker for risk stratification of occult nodal disease and treatment planning in OSCC patients

Keywords

Oral squamous cell carcinoma; Desmoglein 3, Lymph node metastasis; Immunohistochemistry; Biomarkers; Histological grade; Risk stratification

INTRODUCTION

Oral squamous cell carcinomas (OSCC), the most prevalent type of cancer in the mouth, account for 90–95% of all oral malignant lesions. An estimated 3,77,700 new cases and 1,77,800 deaths from oral cancer occur annually, making it a major worldwide health burden. 37.5 percent of the cases were found in Asia alone. [1-5]

Initially OSCC may be asymptomatic which later on become symptomatic and may create ulcers with irregular substrate edges which is

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firm to touch. OSCC occurs on posterior and lateral aspect of tongue most commonly, followed by other anatomical sites. OSCC primarily disseminates to ipsilateral cervical lymphatic nodes through lymphatic drainage, though it may also infiltrate contralateral or bilateral nodes. Common sites for OSCC metastases include the pulmonary system, osseous structures, and hepatic tissue. [6,7,8] About half of OSCC cases with advanced pathology (stage -III and -IV) experience recurrence. The high mortality linked to late-stage HNSCC can be attributed to the ability of neoplastic cells to infiltrate locoregionally, facilitated by a dense lymphatic network and a significant number of lymph nodes in the cervical area. Therefore, it is essential to identify metastatic pathology rapidly and precisely.[9]

The structural integrity of normal tissue relies on several factors that support cellular adhesion, such as intercellular adhesion structures and desmosomes. Reduction of desmosomal components correlates with tumour development. Decreased expression of desmosomal components, including desmoglein 1 (DSG1), desmoglein 3 (DSG3), desmocollins 2 and 3 (DSC2, DSC3), plakoglobin (PG), Plakophyllins 1 and 3 (PKP1-3), and desmopondin (DSP), is associated with poor prognosis in various malignancies. [10,11]

Desmoglein 3 (DSG3) is a 130 kDa glycoprotein in desmosomes that aids intercellular adhesion through calcium binding. Desmosomal cadherins, such as desmogleins and desmocollins, facilitate cell-cell adhesion by connecting to keratin filaments through adaptor proteins. DSG3, along with other desmosomal proteins, acts as a signalling hub. Elevated DSG2 or DSG3 levels have been noted in skin and head and neck cancers. Patel et al. (2008) demonstrated the importance of DSG3 as a target for detecting and treating HNSCC due to its increased expression in HNSCC lesions. Desmosome composition alterations influence signal transduction, facilitating transformation. Evidence shows that DSG3, an upstream regulator of Src, affects adherens junction formation. DSG3 overexpression results in filopodial protrusions and increased cell migration. [12,13,14]

Metastasis is a major contributor to cancer mortality, exceeding the effects of the primary tumour, particularly in head and neck carcinomas, which show a strong tendency for metastatic spread. The current medical landscape highlights the need for critical biomarkers that enable accurate diagnosis, prognostic evaluation, and assessment of therapeutic responses to neoplastic treatments. The potential of desmoglein 3 as a prognostic and diagnostic biomarker has been thoroughly investigated, and it is raised in head and neck squamous cell carcinoma (HNSCC). Cervical lymph node metastasis is becoming recognized as a critical element in the prognosis of oral squamous cell carcinoma (OSCC) and is crucial for clinical staging and therapy planning. However, individuals with clinically negative nodes frequently require elective neck dissection or treatment due to the difficulties in correctly detecting lymph node metastases, resulting in considerable morbidity and negative impacts on quality of life.

This study aimed to assess desmoglein 3 expression via immunohistochemistry (IHC) across various grades of OSCC and to determine its potential as a marker for occult lymph node micrometastasis.

MATERIALS AND METHODS

This retrospective observational study utilised archival formalin-fixed paraffin-embedded (FFPE) tissue blocks previously diagnosed as primary Oral Squamous Cell Carcinoma (OSCC). Samples were obtained from the Department of Oral Pathology and Histology at GITAM Dental College and Hospital, Visakhapatnam, and from a private cancer hospital's pathology department. Only cases with comprehensive clinical documentation, including TNM staging per American Joint Committee on Cancer (AJCC) guidelines, were included. Exclusion criteria included recurrent OSCC cases, blocks with inadequate or poorly preserved tissue, and cases missing complete data and information on tissue specimens and staging.

Sample size calculation utilised G*Power Software version 3.1.9.2, with an effect size of 0.40, α-error probability of 0.05, study power of 0.80, and three comparison groups. The minimum sample size recommended was 80. Immunohistochemical analysis was conducted for the Desmoglein-3 marker, resulting in the processing of 80 IHC sections. Sections 4–5 µm thick were obtained from each paraffin block using a rotary microtome and mounted on poly-L-lysine-coated slides. The slides were incubated at 60°C for one hour prior to the immunohistochemical staining procedure.

The IHC procedure involved deparaffinization in xylene and subsequent rehydration using graded alcohols. Heat-induced epitope retrieval utilised a citrate buffer at

pH 6.0. Endogenous peroxidase activity was inhibited by applying 3% hydrogen peroxide. Sections were incubated with primary monoclonal antibodies specific to DSG3 for 60 minutes at room temperature, followed by a horseradish peroxidase-linked secondary antibody detection system. The interaction between the antigen and antibody was clarified using 3,3′-diaminobenzidine (DAB) chromogen. The slides were counterstained with haematoxylin, then dehydrated, cleared, and mounted. Appropriate positive and negative controls were included during the staining process.

All stained sections were evaluated under a light microscopebytwoindependent,blindedoralpathologists. The assessment involved staining intensity, percentage of positively stained tumour cells, and patterns of cellular localisation. A semi-quantitative scoring system derived final expression scores. Demographic, clinical, histopathological, and immunohistochemical data were compiled and analysed statistically using SPSS software (version to be inserted). Descriptive statistics included mean, standard deviation, and percentages. Statistical analysis utilised the Fisher exact test, with a P value < 0.05 deemed statistically significant. Approval from the Institutional Ethical Committee was secured before the study began.(IEC/GDCH/2025/FR/01-03) Informed consent was waived due to the use of anonymised archival samples, following standard ethical guidelines.

RESULTS

Table 1 summarizes 80 patients (mean age 50 ± 9 years), predominantly male (78.8%). Buccal mucosa (33.8%) was the most common tumor site. Most tumors were pT3 (40.0%) or pT4 (36.2%), with pN2 nodal involvement in 55.0%, and 77.5% presenting in Stage IV. Histological grades were evenly distributed. The median lymph node yield was 13 (IQR: 9–18), with 3 (IQR: 1–5) positive nodes and median size of 1.15 cm. DSG3 expression was mainly moderate (45.0%), followed by low (33.8%).

Table 2 demonstrates that DSG3 reactivity showed a statistically significant association with pN-status ($p \leq 0.001$), pathological stage ($p \leq 0.001$), and number of positive lymph nodes ($p \leq 0.001$). Higher DSG3 expression was predominantly observed in patients with nodal metastasis (pN+), Stage IV disease, and increasing number of positive lymph nodes. However, no significant association was found between DSG3 expression and age ($p = 0.648$), gender ($p = 0.991$), histological grading ($p = 0.712$), pT-status ($p = 0.268$), or number of nodes dissected ($p = 0.452$).

Table 3 presents the association between clinic-pathological predictor variables and histopathological grade among 80 patients. No statistically significant association was observed between tumor grade and age ($p = 0.418$) or gender ($p = 0.296$). Similarly, histological differentiation did not show significant correlation with pT-status ($p = 0.781$), pN-status ($p = 0.447$), pathological stage ($p = 0.758$), number of nodes dissected ($p = 0.936$), or number of positive lymph nodes ($p = 0.252$).

Although not statistically significant ($p = 0.235$), tumours with three or more positive lymph nodes exhibited a higher likelihood of being poorly differentiated. [FIGURE 1] illustrates the expression levels of DSG3 in well-differentiated, moderately differentiated, and poorly differentiated tumours. Every grade has 33% cases devoid of DSG3. Low DSG3 expression is more prevalent in moderately differentiated tumours (41.7%) compared to well (20.8%) and poorly differentiated cancers (37.5%). Well-differentiated tumours exhibit high levels of DSG3 expression (60%), whereas moderately and poorly differentiated cancers show 20% expression.

DSG3 reactivity and nodal condition are illustrated in [FIGURE 2]. As DSG3 expression elevated, the proportion of node-positive instances augmented. All patients lacking DSG3 staining were negative for lymph nodes.

DSG-3 expression was minimal to absent in well-differentiated OSCC, suggesting a surface-type keratinising squamous phenotype rather than a mucosal pattern [FIGURE 3]. DSG3 immunostaining in moderately differentiated OSCC [FIGURE 4] exhibits patchy weak to moderate membranous staining, indicating partial retention of mucosal-type desmosomal adhesion. Poorly differentiated OSCC [FIGURE 5] has little to absent DSG-3 and sporadic moderate background staining, signifying high-grade malignancy and absence of mucosal-type adhesion. The low membranous reactivity and subtle staining of DSG-3 affirm its association with poor differentiation and aggressive biological behaviour.

DISCUSSION

OSCC is the sixth most frequent cancer in men and the twelveth most prevalent cancer in women, accounting for 3% of all cancer cases.[15] The tumor stage at diagnosis has a major impact on OSCC patients' five-year survival rate, which drops from 90% in the absence of lymph node metastasis to 50% with nodal metastasis. The prognostic relevance of nodal metastases may be significantly influenced by the size and classification of metastases. For thetreatment of patients, theidentification of neoplastic deposits in lymph nodes is essential. For the identification of hidden lymph node metastases, immunohistochemistry is a reasonably efficient and economical substitute for PCR immune arrays.[12] The current study population exhibited a mean age of 50 ± 9 years, significantly younger than other published OSCC series. Larsson et al. observed a mean age of 63.02 ± 17.5 years in their sample, indicating an approximate age disparity of 15 years. The younger demographic in our group may indicate regional disparities in risk factor exposure, especially the elevated prevalence of tobacco and betel quid consumption among younger individuals in specific geographic regions. The male preponderance noted in this study (78.8%) was significantly greater than that reported by Larsson et al. (56.8% male), indicating demographic and epidemiological disparities between the study communities.[16] The demographic variables are crucial factors in evaluating biomarker expression patterns and clinical relationships, since age and gender might affect tumour biology and treatment outcomes; however, neither exhibited significant associations with DSG3 expression in the current research. The prevalence of advanced-stage disease in our group, characterised by significant nodal involvement, underscores the ongoing issue of late-stage presentation in OSCC, aligning with international publications that highlight delayed detection as a primary factor in adverse outcomes.[13]

DSG3 Expression: Strong Associations with Nodal Metastasis and Advanced Stage

This study demonstrated significant correlations between DSG3 expression and nodal involvement ($p = 0.001$) and advanced pathological stage ($p = 0.001$). These findings align with numerous published studies identifying DSG3 as a marker of aggressive disease with metastatic potential. Larsson et al. found that DSG3 overexpression significantly correlated with cervical lymph node metastasis at diagnosis and early disease recurrence in their cohort, with DSG3 overexpression present in 51.1% (45/88) of specimens.[16]

Extensive functional studies support the mechanistic basis for DSG3's association with metastasis, highlighting its role in activating pro-metastatic signalling pathways. Kamekura et al. found that DSG3 promotes cancer cell migration and invasion by modulating activator protein 1 (AP-1) and activating Ezrin, which relies on protein kinase C. [17] Brennan et al. demonstrated that DSG3 promotes cancer cell growth, linking it to the DSG3-plakoglobin-TCF/LEF-Myc/cyclin D1/MMP signalling pathway, which connects DSG3 to transcriptional programs that drive proliferation and matrix degradation. [18] DSG3 modulates the YAP-Hippo pathway and suppresses p53 function, offering survival advantages to tumour cells under stress. [19]

Wang et al. reported significant correlations between decreased desmosomal components and lymph node metastasis in OSCC ($p < 0.05$). [20] Xin et al. reported reduced DSG3 expression and cytoplasmic internalisation linked to aggressive characteristics and increased nodal metastasis incidence. [21] This contradiction—where some studies indicate DSG3 overexpression and others report DSG3 loss, both linked to metastasis—likely reflects various aspects of desmosomal dysregulation identified through differing methodologies. Studies highlighting "overexpression" might identify absolute increases in DSG3 protein levels, whereas those indicating "loss" could be observing a loss of proper membranous localisation with cytoplasmic redistribution, or contrasting tumour tissue with adjacent normal epithelium instead of other tumour specimens. Both patterns indicate abnormal DSG3 biology that can promote metastasis via different mechanisms: overexpression may enhance pro-metastatic signalling, while loss of membranous localisation may weaken cell-cell adhesion, allowing detachment and dissemination. Numerous studies have explored DSG3 as an immunohistochemical marker for identifying occult lymph node metastases. Nagvekar et al. assessed DSG3 staining in 47 lymph node specimens from 10 OSCC patients, observing DSG3 positivity in histologically confirmed metastatic nodes, but did not detect additional occult micrometastases in histologically negative nodes. They observed DSG3-positive macrophages in lymph nodes, which raises concerns about the specificity of using DSG3 alone

as a marker for detecting nodal micrometastasis. [12] This finding indicates that although DSG3 is present in metastatic tumour deposits, its effectiveness for enhancing detection beyond standard histopathology may be constrained by background staining in reactive immune cells.

Histological Grade: Discordance Between Present Findings and Published Literature

This study revealed no significant correlation between DSG3 expression and histological grade ($p = 0.704$). This finding significantly contrasts with multiple published reports showing notable grade associations. Larsson et al. found that DSG3 overexpression correlated significantly with poor histologic differentiation. [16] Wang et al. noted that changes in desmosomal protein expression were linked to tumour grade, with lower expression correlating with worse differentiation ($p < 0.05$). These discrepancies necessitate careful interpretation and likely indicate various contributing factors.[20]

Differences in immunohistochemistry scoring systems are a key explanatory factor. The current study categorises DSG3 expression into four levels (negative, low, moderate, intense) and assesses staining intensities, potentially obscuring grade-specific patterns that emerge with binary classification comparing extremes. Moreover, variations in antibody clones, staining protocols, and threshold definitions for categorising expression levels can yield differing results when analysing the same biological phenomenon.

Tumour differentiation evaluated through traditional histological criteria reflects just one aspect of tumour biology, while molecular changes in adhesion molecules and signalling pathways may happen independently of morphological dedifferentiation. The study found significant associations of DSG3 with nodal status ($p = 0.001$) and pathological stage ($p = 0.001$), while histological grade showed no significant correlation ($p = 0.438$ and $p = 0.772$). This supports the notion that molecular markers may better reflect metastatic risk than traditional morphological grading, particularly in cohorts with advanced disease. The current study revealed that histological grade showed no significant associations with nodal involvement ($p = 0.438$), pathological stage ($p = 0.772$), pT status ($p = 0.796$), or the number of positive lymph nodes ($p = 0.235$). These findings differ from published reports indicating strong links between poor differentiation and nodal metastasis. The discordance likely reflects the predominance of advanced-stage disease in the cohort (77.3% Stage IV) and significant nodal involvement across all grades (84.8%), resulting in a ceiling effect where most patients display aggressive features irrespective of differentiation status.

Molecular Markers Versus Conventional Parameters: Implications for Risk Stratification

A central finding of the present study is the superior performance of molecular markers (DSG3) compared to conventional histopathological parameters (grade) in predicting nodal metastasis and advanced stage. DSG3 expression showed highly significant associations with nodal positivity and pathological stage compared to histological grade.

The observation of grade-independent nodal metastasis has important clinical implications for treatment planning. Current guidelines for OSCC management recommend consideration of elective neck dissection in clinically node-negative patients based on risk factors including tumor thickness, depth of invasion, and histological grade. Molecular marker assessment, particularly DSG3 expression, may provide more accurate identification of patients harboring occult nodal metastases who would benefit from elective neck treatment.

The mechanistic basis for grade-independent metastasis is well-supported by functional studies demonstrating that molecular alterations in cell adhesion and signaling can confer metastatic capacity independent of morphological dedifferentiation. DSG3 activates multiple pro-metastatic signaling cascades. [8-10] and these molecular changes can occur in tumors that retain relatively well-differentiated histological architecture. Wang et al. reported strong correlations between reduced desmosomal protein expression and nodal metastasis ($r = 0.734$), demonstrating that molecular changes in cell-cell adhesion predict metastatic behavior with greater precision than histological grade alone. [20] Xin et al. documented that altered subcellular localization of desmosomal proteins—such as cytoplasmic internalization rather than membranous expression—was associated with aggressive features and nodal metastasis, highlighting that protein distribution rather than overall differentiation state may be a more relevant determinant of metastatic potential.[21]

Clinical applications of the study indicate that assessing

DSG3 expression could improve risk stratification in OSCC specimens and that DSG3-related pathways may serve as therapeutic targets. [22-26] Preclinical studies show potential for DSG3-directed antibody therapies in squamous cell carcinoma. Future investigations would benefit from multicenter studies with standardized protocols and larger sample sizes to validate findings. However, limitations such as a single-institution design, modest sample size, and potential biases restrict generalizability and the ability to draw broader conclusions about DSG3 as a prognostic marker.

CONCLUSION

In conclusion, the present study demonstrates that DSG3 expression is significantly associated with lymph node metastasis and advanced pathological stage in OSCC. The superior performance of molecular markers (DSG3) compared to histological grade in predicting nodal disease supports an emerging paradigm in which molecular profiling may enhance risk stratification and treatment planning. These findings contribute to the evolving understanding of desmosomal dysregulation in OSCC pathogenesis and metastasis, though prospective validation with standardized methodologies and outcome data is required before routine clinical implementation. Future research integrating molecular markers with conventional parameters in multivariate prediction models and exploring DSG3-targeted therapeutic strategies may ultimately improve outcomes for OSCC patients through more precise risk stratification and personalized treatment approaches.

Table 1. *Baseline attributes of the study sample*

Variable	Number (%)
Age (mean ± SD)	50 ± 9
Gender	
Male	63 (78.8%)
Female	17 (21.2%)
Anatomic location	
Buccal mucosa	27 (33.8%)
Tongue	25 (31.3%)
Gingivo-buccal sulcus	15 (18.8%)
Lower alveolus	10 (12.5%)
Palate	3 (3.8%)
pT-status	
pT2	19 (23.8%)
pT3	32 (40.0%)
pT4	29 (36.2%)
pN-status	
pN0	13 (16.3%)
pN1	17 (21.2%)
pN2	44 (55.0%)
pN3	6 (7.5%)
Pathological stage	
Stage III	18 (22.5%)
Stage IV	62 (77.5%)
Histological grading	
Well differentiated	26 (32.5%)
Moderately differentiated	28 (35.0%)
Poorly differentiated	26 (32.5%)
Total number of nodes (median (IQR))	13 (9–18)
Nodes positive (median (IQR))	3 (1–5)
Lymph-node size (median (IQR))	1.15 (0.9–1.7)
DSG3 Reactivity	
No stain	8 (10.0%)
Low	27 (33.8%)
Moderate	36 (45.0%)
Intense	9 (11.2%)

Table 2. *Relationship between DSG3 reactivity and the predictive factors*

VARIABLE	DSG3 Reactivity	DSG3 Reactivity	DSG3 Reactivity	DSG3 Reactivity	p-value (Fisher exact test)
	No stain (n=8)	Low (n=27)	Moderate (n=36)	Intense (n=9)	
Age					
<55 years	6 (75.0%)	20 (74.1%)	24 (66.7%)	5 (55.6%)	0.648
≥55 years	2 (25.0%)	7 (25.9%)	12 (33.3%)	4 (44.4%)	0.648
Gender					
Male	6 (75.0%)	21 (77.8%)	29 (80.6%)	7 (77.8%)	0.991
Female	2 (25.0%)	6 (22.2%)	7 (19.4%)	2 (22.2%)	0.991
Histological grading					
Well differentiated	3 (37.5%)	7 (25.9%)	14 (38.9%)	4 (44.4%)	0.712
Moderately differentiated	2 (25.0%)	11 (40.7%)	11 (30.6%)	2 (22.2%)	0.712
Poorly differentiated	3 (37.5%)	9 (33.3%)	11 (30.6%)	3 (33.3%)	0.712
pT-status					
pT2	1 (12.5%)	7 (25.9%)	10 (27.8%)	1 (11.1%)	0.268
pT3	5 (62.5%)	11 (40.7%)	14 (38.9%)	2 (22.2%)	0.268
pT4	2 (25.0%)	9 (33.3%)	12 (33.3%)	6 (66.7%)	0.268
pN-status					
pN0	7 (87.5%)	4 (14.8%)	2 (5.6%)	0 (0.0%)	≤0.001*
pN+	1 (12.5%)	23 (85.2%)	34 (94.4%)	9 (100%)	≤0.001*
Pathological stage					
Stage III	6 (75.0%)	7 (25.9%)	4 (11.1%)	1 (11.1%)	≤0.001*
Stage IV	2 (25.0%)	20 (74.1%)	32 (88.9%)	8 (88.9%)	≤0.001*
Nodes dissected					
≥12	5 (62.5%)	14 (51.9%)	19 (52.8%)	4 (44.4%)	0.452
<12	3 (37.5%)	13 (48.1%)	17 (47.2%)	5 (55.6%)	0.452
Number of positive nodes					
0	6 (75.0%)	4 (14.8%)	2 (5.6%)	0 (0.0%)	≤0.001*
1	1 (12.5%)	12 (44.4%)	16 (44.4%)	2 (22.2%)	≤0.001*
2	1 (12.5%)	6 (22.2%)	9 (25.0%)	2 (22.2%)	≤0.001*
≥3	0 (0.0%)	5 (18.6%)	9 (25.0%)	5 (55.6%)	≤0.001*

Table 3. *Relationship between the predictor variables and OSCC grade*

Variable	Histopathological Grade			p-value
	Well differentiated (n=26)	Moderately differentiated (n=28)	Poorly differentiated (n=26)	
Age				
<55 years	17 (65.4%)	21 (75.0%)	20 (76.9%)	0.418
≥55 years	9 (34.6%)	7 (25.0%)	6 (23.1%)	0.418
Gender				
Male	20 (76.9%)	23 (82.1%)	20 (76.9%)	0.296
Female	6 (23.1%)	5 (17.9%)	6 (23.1%)	0.296
pT-status				
pT2	6 (23.1%)	8 (28.6%)	5 (19.2%)	0.781
pT3	11 (42.3%)	11 (39.3%)	10 (38.5%)	0.781
pT4	9 (34.6%)	9 (32.1%)	11 (42.3%)	0.781
pN-status				
pN0	4 (15.4%)	5 (17.9%)	4 (15.4%)	0.447
pN+	22 (84.6%)	23 (82.1%)	22 (84.6%)	0.447
Pathological stage				
Stage III	6 (23.1%)	7 (25.0%)	5 (19.2%)	0.758
Stage IV	20 (76.9%)	21 (75.0%)	21 (80.8%)	0.758
Nodes dissected				
<12	13 (50.0%)	14 (50.0%)	13 (50.0%)	0936
≥12	13 (50.0%)	14 (50.0%)	13 (50.0%)	0936
Number of positive nodes				
0	3 (11.5%)	4 (14.3%)	3 (11.5%)	0.252
1	12 (46.2%)	14 (50.0%)	10 (38.5%)	0.252
2	6 (23.1%)	5 (17.9%)	7 (26.9%)	0.252
≥3	5 (19.2%)	5 (17.8%)	6 (23.1%)	0.252

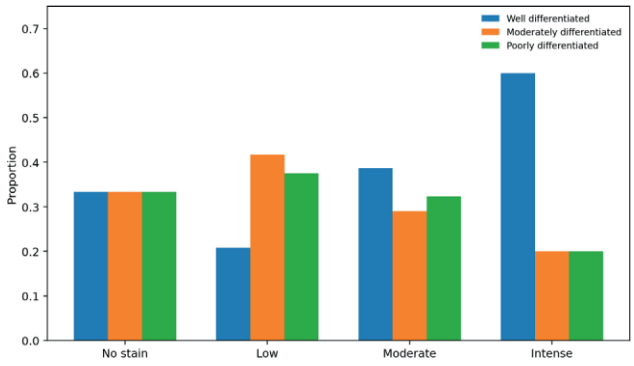

Figure 1. *Distribution of DSG3 expression in oral cancer based on differentiation levels.*

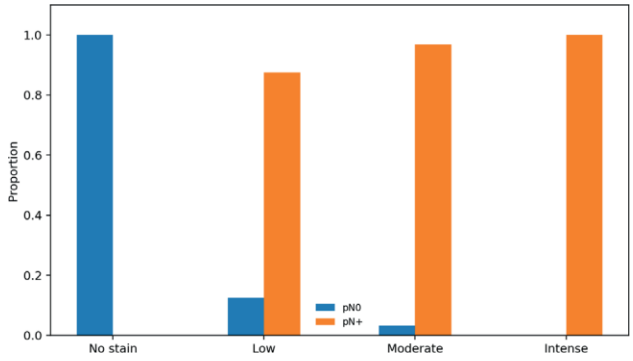

Figure 2. *Distribution of DSG3 expression in oral cancer based on lymph node metastasis.*

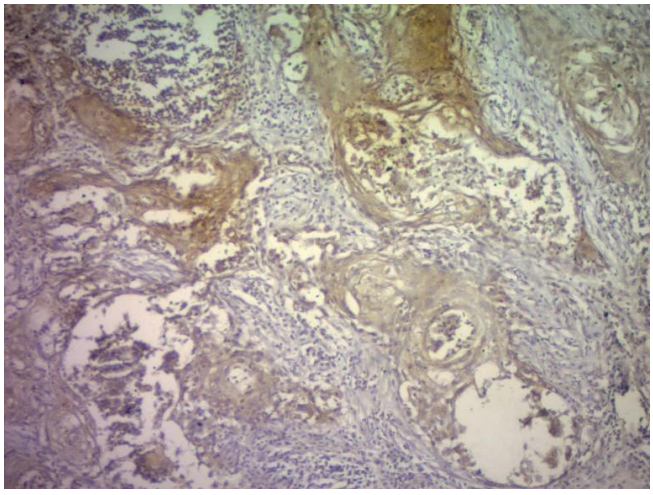

Figure 3. *Desmoglein 3 expression in well differentiated OSCC. (10×)*

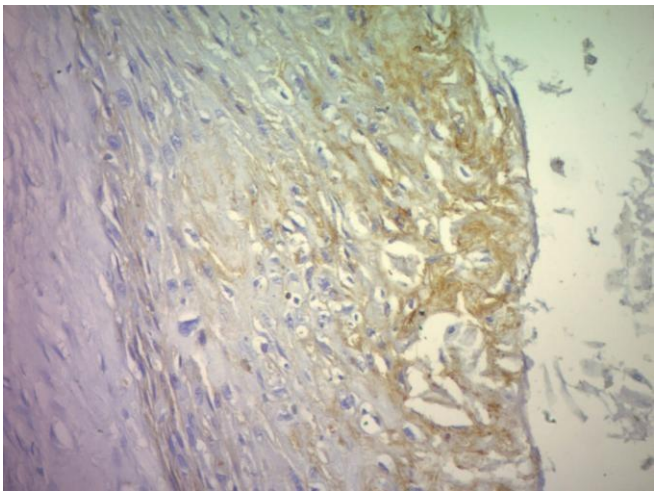

Figure 4. *Desmoglein 3 expression in moderately differentiated OSCC. (20×)*

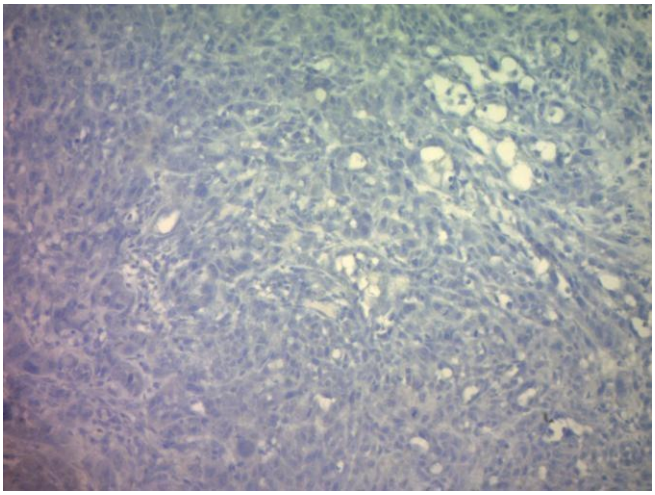

Figure 5. *Desmoglein 3 expression in poorly differentiated OSCC. (20×)*

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