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Original Article

An Immunohistochemical Study on Expression of β-catenin in Oral Squamous Cell Carcinomas - Comparative Analysis

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ABSTRACT

Background: β-catenin is a versatile protein that contributes to cell–cell adhesion and also acts in the regulation of gene expression. Dysregulation of the Wnt/β-catenin signaling pathway has been reported across various human malignancies.
Objective: This study aimed to assess the β-catenin expression in different grades of OSCC by assessing its qualitative and semi-quantitative immunohistochemical expression in comparison to normal oral epithelium.
Materials and Methods: A comparative immunohistochemical study was carried out on 45 tissue specimens, comprising two groups: Group A -30 cases of Oral Squamous Cell Carcinoma and Group B -15 samples of normal healthy oral mucosa serving as controls. Tissue sections were stained with haematoxylin and eosin to confirm the histopathological diagnosis of both cases and controls, followed by immunohistochemical staining for β-catenin expression. The stained sections were independently evaluated by two blinded observers using a trinocular research microscope. Assessment of β-catenin expression was performed based on its subcellular localization, including membranous, cytoplasmic, and nuclear compartments. A semi-quantitative immunoreactivity score (IRS) was calculated by considering the proportion of positive cells and staining intensity for each compartment and the observations were statistically analysed.
Results: β-catenin expression in normal mucosal epithelium exhibited predominantly membranous expression, whereas OSCC, with increasing histopathological grade, there was a progressive shift from membranous to cytoplasmic and nuclear localization, with poorly differentiated OSCC showing maximum nuclear accumulation and the difference in expression of β-catenin was statistically significant ($p < 0.001$).
Conclusion: The study concluded that β-catenin undergoes a redistribution from membrane-bound expression to cytoplasmic and nuclear localization during malignant transformation in OSCC. This shift reflects loss of cell adhesion and activation of oncogenic Wnt/β-catenin signaling. Therefore, β-catenin has potential to serve as a diagnostic biomarker and may help in assessing tumor progression and aggressiveness.

Keywords*: β-catenin, oral squamous cell carcinoma, membrane expression, nuclear expression, cytoplasmic expression.*

INTRODUCTION

β-catenin is a multifunctional protein that plays a crucial role in both cell–cell adhesion and intracellular signaling pathways. It is an integral component of the cadherin-mediated adhesion complex, linking E-cadherin to the actin cytoskeleton, thereby maintaining epithelial integrity and tissue architecture. [1] In addition to its structural role, β-catenin acts as a key regulator in the canonical Wnt signaling pathway, where it functions as a transcriptional co-activator influencing genes involved in cell proliferation, differentiation, and survival .[2,3] Under normal physiological conditions, β-catenin is predominantly localized at the cell membrane and is tightly regulated by a destruction complex consisting of APC, Axin, and GSK-3β, which promotes its degradation. However, dysregulation of the Wnt/β-catenin pathway leads to stabilization and accumulation of β-catenin in the cytoplasm, followed by its translocation into the nucleus, where it activates oncogenic target genes.[4,5]

Aberrant expression and altered subcellular localization of β-catenin have been implicated in the pathogenesis of various human malignancies, including oral squamous cell carcinoma (OSCC). Loss of membranous β-catenin expression is associated with reduced cell adhesion, increased invasiveness, and tumor progression, while increased cytoplasmic and nuclear localization correlates with enhanced proliferative activity and poor differentiation.[6,7]

In OSCC, progressive changes in β-catenin localization from membranous to cytoplasmic and nuclear have been linked to tumor aggressiveness and histopathological grading. These alterations reflect disruption of cell adhesion mechanisms and activation of oncogenic signaling pathways, thereby contributing to malignant transformation and progression.[6,7]
Therefore, evaluation of β-catenin expression through immunohistochemical analysis can provide valuable insights into the biological behavior of OSCC and its potential utility as a diagnostic biomarker.Based on these considerations, the present study aimed to assess the immunohistochemical expression of β-catenin in oral squamous cell carcinoma and compare it with normal oral epithelium by evaluating its qualitative and semi-quantitative expression in membranous, cytoplasmic, and nuclear compartments.

MATERIALS AND METHODS

This study was designed as a comparative immunohistochemical in vitro study done in the Department of Oral and Maxillofacial Pathology at Government Dental College and Hospital over a period of 6 months, from March 2025 to October 2025. Ethical clearance was obtained from the Institutional Ethics Committee No: 16/D001/IEC/GDC&H/2023-24.

The materials used in the study included formalin-fixed paraffin-embedded tissue specimens of oral squamous cell carcinoma and normal oral mucosa. Other materials comprised routine tissue processing reagents, a rotary microtome (Leica), primary antibody against β-catenin, secondary antibody (HRP-conjugated anti-rabbit IgG), haematoxylin and eosin stains, a Trinocular research microscope, glass slides, cover slips, staining jars, and standard laboratory equipment including a slide warming table and hot air oven.

The study protocol was carried out in the following steps:
a. Sample collection and preparation
b.Tissue sectioning and staining with Hematoxylin & Eosin and β-catenin (IHC marker)
c. Evaluation of stained sections
d. Statistical analysis

a. Sample Collection and Preparation

A total of 45 tissue specimens (N = 45) were study samples in this study. The sample size was calculated using Chi-square analysis with G*Power software (version 3.1.9.7). The samples were selected based on inclusion criteria, which included histopathologically confirmed cases of OSCC (graded according to WHO criteria) and normal oral mucosa obtained from healthy individuals with informed consent, preferably from third molar extraction sites. Specimens with any pathological alterations in the oral mucosa were excluded.

Thirty formalin-fixed, paraffin-embedded tissue specimens of diagnosed OSCC were retrieved from the Department of Oral Pathology archives. Fifteen normal oral mucosal tissue samples were obtained from the Department of Oral Surgery taken during procedures such as surgical impaction and management of jaw fractures. Normal tissue samples were processed routinely to prepare paraffin blocks.

Total 45 study samples (N) were categorized into two groups as follows:

- **Group A:** OSCC cases (n = 30)
- **Group B:** Normal oral mucosa (n = 15)

b. Tissue Sectioning and Staining
From each paraffin-embedded tissue block, two serial sections of approximately 3 µm thickness were obtained using a microtome.

- The first section was stained with haematoxylin and eosin (H&E) for confirmation of histopathological diagnosis.
- The second section was subjected to immunohistochemical staining for β-catenin expression using a primary antibody against β-catenin, followed by an appropriate HRP-conjugated secondary antibody.

c. Evaluation of H&E-Stained Sections and β-Catenin IHC-Stained Sections
The H&E-stained sections were independently evaluated by two blinded observers using a trinocular research microscope under 4x, 10x, and 40x magnifications for confirmatory histopathological diagnosis of Oral Squamous Cell Carcinoma.

The parameters such as membranous, cytoplasmic, and nuclear expression of β-catenin were evaluated both qualitatively and semi-quantitatively. Blinded evaluation was performed by two independent investigators. The pathologists assessed the immunohistochemical staining without prior knowledge of clinicopathological data. Any discrepancies were resolved using a multiheaded microscope.

Representative histological sections from each case were evaluated for β-catenin expression. For each section, five random high-power fields were selected and analyzed. Quantitative analysis was performed using Image software.

A semi-quantitative scoring system was employed to assess β-catenin expression separately in membranous, cytoplasmic, and nuclear compartments. The scoring was based on staining intensity and the percentage of positively stained cells.

Staining Intensity Score [8] :

Score	**Staining Intensity**
Score 0	No staining
Score 1	Weak staining
Score 2	Moderate staining
Score 3	Strong staining

Percentage of Positive Cells [9]:

Score	**Percentage of positive cells**
Score 0	No positive cells
Score 1	<10% positive cells
Score 2	10–50% positive cells
Score 3	50–80% positive cells
Score 4	>80% positive cells

The final immunoreactivity score was calculated by multiplying the intensity score and the percentage score:
Final Score = Intensity Score × Percentage Score
Based on the final score, β-catenin expression was interpreted as follows:[9]
0–1 → Negative expression
2–3 → Mild expression
4–8 → Moderate expression
9–12 → Strong expression

d. Statistical Analysis
All quantitative data were compiled and analysed using SPSS version 26. The results were expressed as mean ± standard deviation (SD) for continuous variables. The immunoreactive scores (IRS) of β-catenin expression in membranous, cytoplasmic, and nuclear compartments were assessed across different histopathological grades of oral squamous cell carcinoma (OSCC) and compared with the control group.

Data normality was confirmed using an Independent Samples t-test to compare mean IRS values between normal oral mucosa and OSCC cases. One-way analysis of variance (ANOVA) was used to compare β-catenin expression among different histopathological grades of OSCC (well-, moderately-, and poorly differentiated). Post-hoc analysis using Tukey's test was performed for intergroup comparisons.
Both p-values and mean differences were recorded to determine statistical significance. A p-value < 0.05 was considered statistically significant.

RESULTS

β-catenin expression was evaluated in a total of 45 specimens. The observations were as follows.

Table 1 showed predominantly strong membranous expression of β-catenin in normal oral mucosal epithelium, with minimal cytoplasmic and negligible nuclear expression.

Table 2 showed significant loss of membranous expression with significant increase in cytoplasmic and nuclear expression in OSCCs (cases) when compared to normal mucosal epithelium (controls) and the difference was highly statistically significant ($p < 0.001$).

Table 3 showed a significant decrease in membranous expression with a mild increase in cytoplasmic and nuclear expression in well-differentiated OSCCs (cases) compared to normal oral mucosal epithelium (controls) and the difference was highly statistically significant ($p < 0.05$).

Table 4 showed markedly elevated cytoplasmic and nuclear β-catenin expression with a corresponding reduction in membranous expression in moderately differentiated OSCCs (cases), compared to normal oral mucosal epithelium (controls) and the difference was highly statistically significant ($p < 0.001$) .

Table 5 showed the most pronounced decrease in membranous expression along with the highest cytoplasmic and nuclear expression in poorly differentiated OSCC (cases) compared to normal oral mucosal epithelium (controls) and the difference was highly statistically significant ($p < 0.001$).

Table 6 showed a significant difference in β-catenin expression among different histopathological grades of OSCCs and the difference was highly statistically significant ($p < 0.001$).

TABLES

Table 1: β-catenin Expression In Normal Oral Mucosal Epithelium

Localization	Mean ± SD	Interpretation
Membranous	7.12 ± 1.03	Strong
Cytoplasmic	1.24 ± 0.62	Mild
Nuclear	0.48 ± 0.29	Negative

Inference**:** Predominantly membranous expression with minimal cytoplasmic/nuclear localization.

Table 2: Comparison of β-catenin Expression in Normal Oral Mucosal Epithelium with OSCC

Localization	Normal (Mean ± SD)	OSCC (Mean ± SD)	p-value
Membranous	7.12 ± 1.03	3.84 ± 1.72	<0.001
Cytoplasmic	1.24 ± 0.62	3.96 ± 1.28	<0.001
Nuclear	0.48 ± 0.29	2.87 ± 1.05	<0.001

*Independent t-test ;Significance at $p < 0.05$

Inference:

- Significant loss of membranous expression
- Significant increase in cytoplasmic & nuclear expression
- Indicates β-catenin translocation (Wnt pathway activation)

Table 3: Comparison of β-catenin Expression In Well-Differentiated OSCC with Normal Oral Mucosal Epithelium

Localization	WDSCC	Normal	p-value
Membranous	5.92 ± 1.21	7.12 ± 1.03	<0.01
Cytoplasmic	2.14 ± 0.88	1.24 ± 0.62	<0.05
Nuclear	1.02 ± 0.45	0.48 ± 0.29	<0.05

*One-way ANOVA Significance at $p < 0.05$
Inference:
Early shift from membrane → cytoplasm

Table 4: Comparison of β-catenin Expression in Moderately Differentiated OSCC with Normal Oral Mucosal Epithelium

Localization	MDSCC	Normal	p-value
Membranous	3.74 ± 1.12	7.12 ± 1.03	<0.001
Cytoplasmic	4.12 ± 1.01	1.24 ± 0.62	<0.001
Nuclear	2.86 ± 0.88	0.48 ± 0.29	<0.001

*One-way ANOVA: Significance at $p < 0.05$
Inference:
Clear membrane dysregulation and intracellular accumulation

Table 5: Comparison of β-catenin Expression in Poorly Differentiated OSCC with Normal Oral Mucosal Epithelium

Localization	PDSCC	Normal	p-value
Membranous	1.96 ± 0.88	7.12 ± 1.03	<0.001
Cytoplasmic	5.12 ± 1.34	1.24 ± 0.62	<0.001
Nuclear	4.02 ± 1.02	0.48 ± 0.29	<0.001

*One-way ANOVA :Significance at $p < 0.05$
Inference:
Maximum nuclear accumulation → aggressive tumor biology

Table 6: Comparison of β-catenin Expression among OSCC Grades

Localization	WDSCC	MDSCC	PDSCC	p-value
Membranous	5.92	3.74	1.96	<0.001
Cytoplasmic	2.14	4.12	5.12	<0.001
Nuclear	1.02	2.86	4.02	<0.001

*Post-hoc (Tukey) test;Significance at $p < 0.05$
Inference:

1. Normal mucosa → Membrane-bound β-catenin (cell adhesion role)
2. OSCC → Loss of adhesion + nuclear signaling activation
3. Indicates:
    1. Epithelial–Mesenchymal Transition (EMT)
    2. Tumor progression
    3. Activation of Wnt/β-catenin pathway

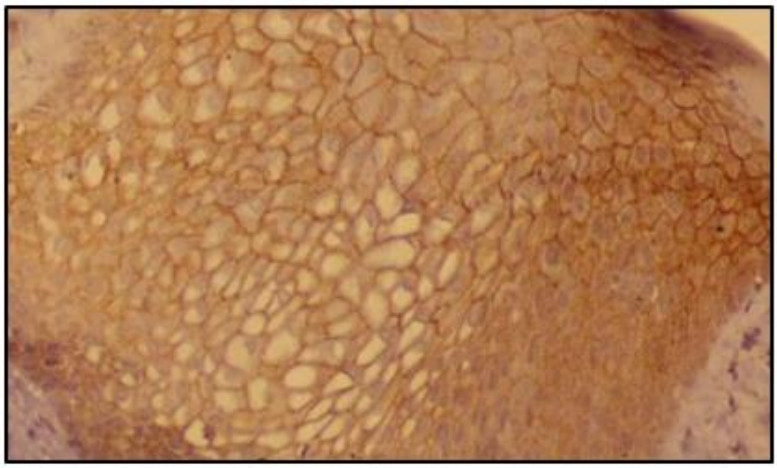

Figure 1- β-catenin in Normal Oral Mucosal Epithelium -only Membranous Expression with no Cytoplasm and Nuclear expression

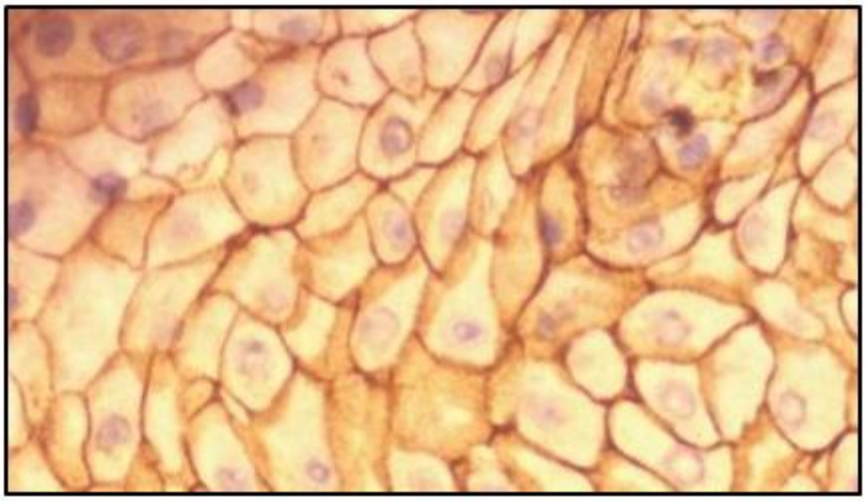

Figure 2- β-catenin in Well Differentiated OSCC- predominant Membranous,Cytoplasm expression and less Nuclear expression

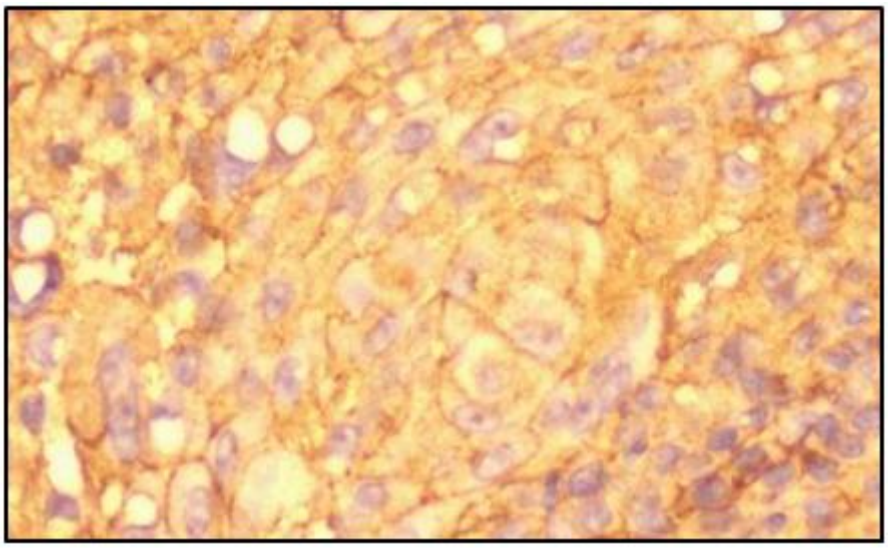

Figure 3- β-catenin in Moderately Differentiated OSCC- Membrane disruption with predominant Cytoplasm expression and less Nuclear expression

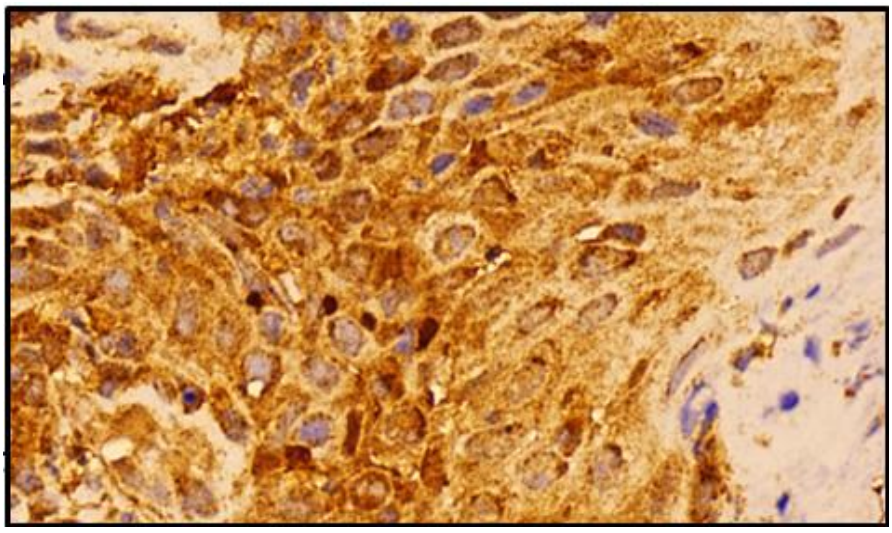

Figure 4- β-catenin in Poorly Differentiated OSCC- predominant Cytoplasm and Nuclear expression with least or no membranous expression

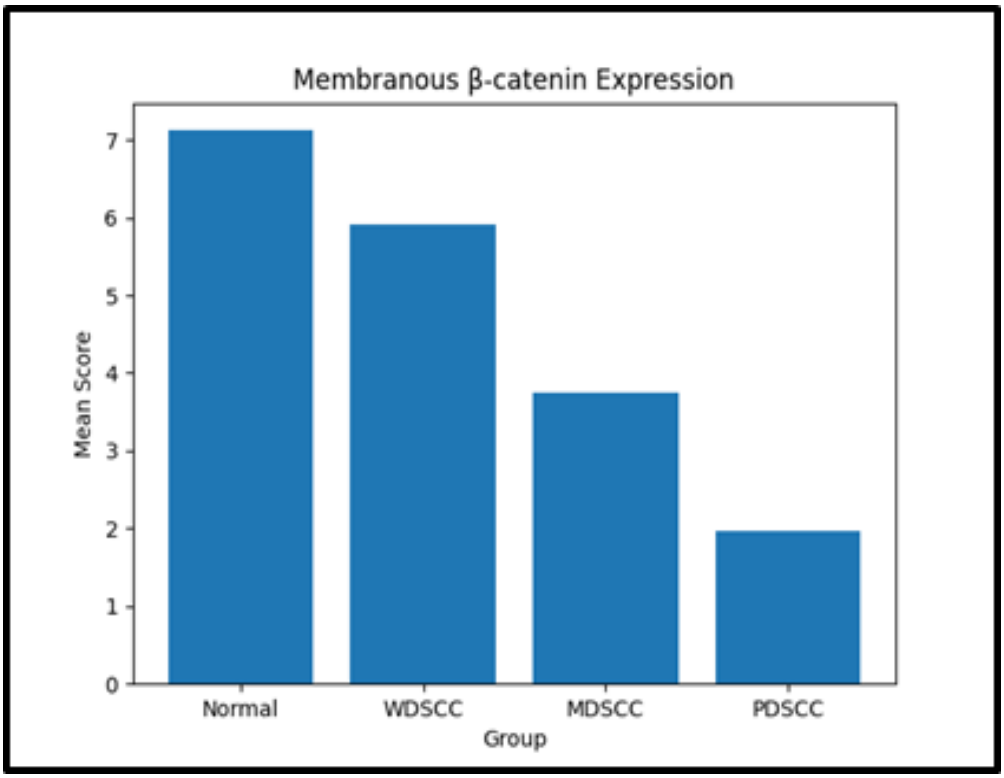

Figure 5- Comparison of Membranous expression of β-catenin in all groups (A and B)

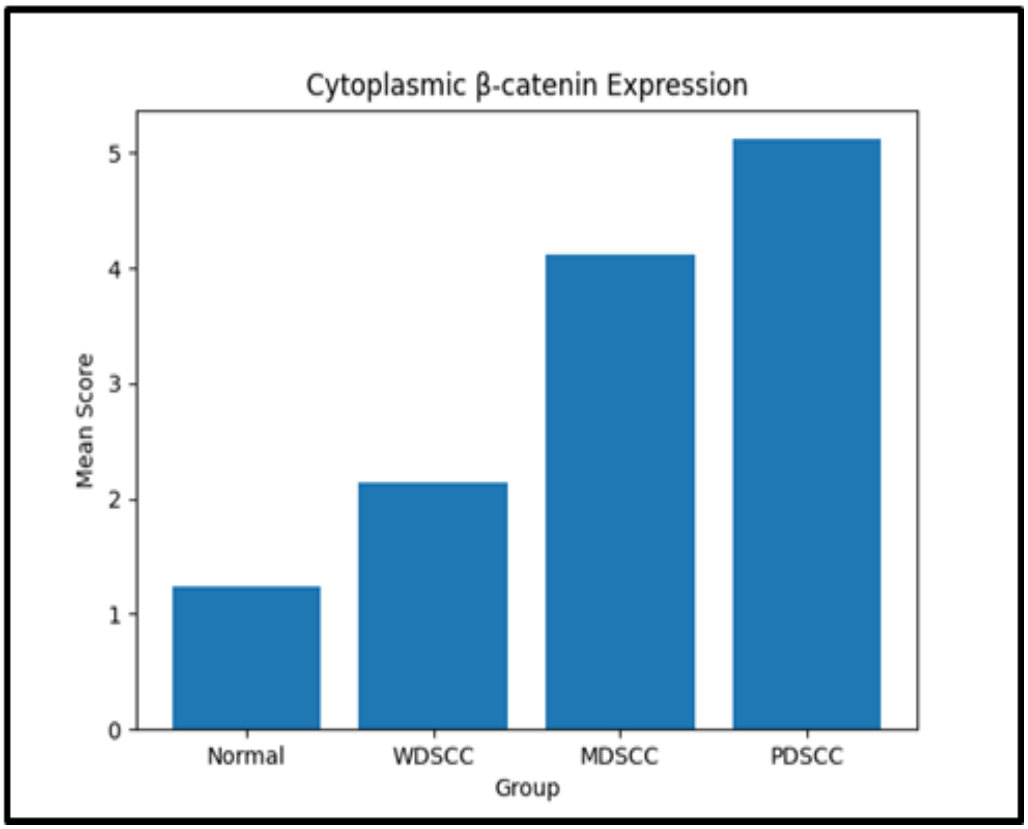

Figure 6- Comparison of Cytoplasmic expression of β-catenin in all groups (A and B)

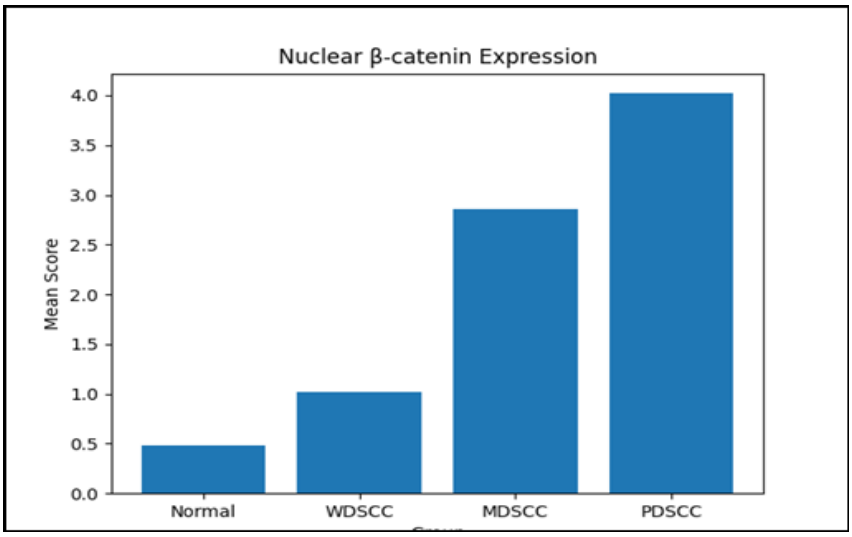

Figure 7- Comparison of Nuclear expression of β-catenin in all groups (A and B)

DISCUSSION

Normal oral mucosal epithelial cells predominantly showed membranous expression with minimal cytoplasmic/nuclear localization (Table 1).These findings were in consistent with study results of Silva et al.(2014) where in normal oral mucosal epithelium, β-catenin was detected only in the cytoplasmic membrane.[10]

The present study findings (Table 2) indicated that β-catenin expression was significantly altered in OSCC compared to normal oral mucosal epithelium, with a marked reduction in membranous expression and increased cytoplasmic and nuclear localization (Fig.1). Normal mucosal epithelial cells predominantly exhibited membranous staining, reflecting its role in maintaining cell to cell adhesion, whereas OSCC showed aberrant intracellular accumulation (Table 2). [11] These significant findings suggested a crucial role of β-catenin in the pathogenesis of OSCC and support its utility as a biomarker for malignant transformation.which is in accordance with study conducted by Zaid KW et al. (2014) .[2]

The present study observed a significant reduction in membranous β-catenin expression with a mild increase in cytoplasmic and nuclear expression in well-differentiated OSCC cases compared to normal mucosal epithelial cells ($p < 0.05$). Thus, an initial shift of β-catenin from the membrane to intracellular compartments is noticed in well differentiated OSCC cases (Table 3, Fig.2). This could be explained as an indication of early disruption of cell adhesion along with the beginning of activation of Wnt/β-catenin signaling.[12] This observation was in accordance with study results of Chaw et al.(2012) and Zaid KW et al.(2014) where similar patterns of altered β-catenin localization reported in OSCC and other epithelial malignancies, indicating early molecular changes associated with tumorogenesis.[1,2] Malignant cells showed β-catenin in membrane, cytoplasm or nucleus would suggest the well differentiated OSCC grade.

In moderately differentiated OSCC, β-catenin expression showed a marked increase in cytoplasmic and nuclear compartments along with a significant reduction in membranous expression (Table 4, Fig.3). Higher intracellular accumulation, particularly nuclear localization, suggested activation of transcriptional activity and correlates with increased tumor progression. This redistribution from membrane to cytoplasm and nucleus reflects progressive dysregulation of the Wnt/β-catenin pathway.[13] The moderate differentiation stage, therefore, represents a transitional phase where loss of cell adhesion becomes more evident and oncogenic signaling pathways are increasingly activated, contributing to tumor progression and poorer prognosis.[14] This shift also indicated the initiation of epithelial–mesenchymal transition (EMT), which plays a key role in tumor invasion and metastasis.[15] These observations were in accordance with the conclusion of Abbas R et al. (2024) that predominant malignant cells with β-catenin expression in cytoplasm, nucleus and less membranous expression could suggest moderately differentiated OSCC grade.[9]

Poorly differentiated OSCC cases demonstrated the most pronounced alterations, with a marked decrease in membranous expression and the highest cytoplasmic and nuclear β-catenin expression ($p < 0.001$), indicating significant intracellular accumulation. The increased nuclear localization reflects strong activation of the Wnt/β-catenin signaling pathway and is associated with aggressive tumor behavior and poor prognosis (Table 5, Fig.4). The higher the nuclear expression, the greater the transcriptional activation of oncogenic target genes, leading to enhanced proliferation, invasion, and malignant potential.[16]These observations correlated with the study results of Chen Z,et al. (2013) where his study showed that *β-catenin* overexpression in the nucleus, rather than in the cytoplasm, appeared to be associated with progress disease and a worse prognosis for CRC patients.[17]

The study observed a significant difference in pattern of expression of β-catenin as poorly differentiated OSCC displayed increased nuclear expression ,moderately differentiated OSCC exhibited nuclear and cytoplasm expression whereas membranous with cytoplasmic expression in well differentiated OSCC (Table 6). This changes could be explained as loss of adhesion with nuclear signaling activation and Wnt/catenin pathway and epithelial-mesenchymal interaction.This observation correlates with the previous studies, particularly Moles et al. (2016) which concluded that β-Catenin plays a major oncogenic role in OSCC, principally through increase in invasiveness due to loss of its membranous expression.[18]

In this study, it was observed that β-catenin expression undergoes a progressive shift from membranous localization in normal oral epithelium to increased cytoplasmic and nuclear accumulation in different grades of OSCC (Fig.5,6,7).

LIMITATIONS

The present study had certain limitations. The sample size was relatively small (45 cases), which may affect the statistical power and generalization of the results. Additionally, immunohistochemical findings were not validated using molecular techniques such as Western blotting or RT-PCR, which could have provided more definitive insights into β-catenin expression and its underlying molecular mechanisms.

CONCLUSION

The present study observed that well-differentiated oral SCC predominantly exhibited membranous β-catenin expression, whereas moderately and poorly differentiated carcinomas showed a progressive shift toward cytoplasmic and nuclear localization . This gradual redistribution with worsening tumor differentiation suggested loss of cell adhesion, activation of oncogenic signaling, and increasing tumor aggressiveness. Overall, these findings highlight the role of β-catenin as a key regulator in OSCC progression and emphasize its potential as a diagnostic biomarker.Further larger studies with huge samples are required for confirmatory results.

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