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Evidence of β-Catenin Immunohistochemistry in Oral Squamous Cell Carcinoma: Association with Clinicopathological Characteristics

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ABSTRACT

Oral squamous cell carcinoma (OSCC) is the most common type of cancer in the upper aerodigestive tract, while head and neck squamous cell carcinoma is the eighth most common cancer in men and the thirteenth most common cancer in women worldwide. The objective of the study was to assess the β-catenin immunohistochemistry expression and relate it to variables including age, gender, tumor location, clinical manifestation, and histopathological grade of OSCC. In all, 45 blocks of completely excised OSCC that had been previously formalin-fixed and embedded in paraffin were considered for the investigation. The anti-β-catenin antibody was used for the immunohistochemical staining. Forty-five patients who underwent excisional removal for OSCC were analyzed retrospectively through Hematoxylin, eosin, and immunohistochemical staining to evaluate clinical and pathological parameters. The majority of occurrences (31.11%) happened in the 70s, according to the results, with males being more affected than females, showing a male-to-female ratio of 1.5:1. The most common site of the tumor was the tongue (48.89%), and the majority of cases presented clinically as a mass (53.33%). Immunohistochemically, the β-catenin positive immunoreaction was most commonly observed in score 2 (51.11%). Statistically, β-catenin expression showed a significant association with tumor grade. The study found that β-catenin, a signaling molecule, reduces membrane localization and increases cytoplasmic and nuclear staining. Its expression is strongly associated with tumor grade, suggesting that β-catenin may promote tumor progression by enhancing cell proliferation and weakening cell adhesion.

Keywords: Immunohistochemical, Oral Squamous, β-Catenin

1 Introduction

ORAL squamous cell carcinomas (OSCC) make up the majority of upper aerodigestive tract malignancies, while head and neck squamous cell carcinomas (HNSCC) are the eighth most common malignancy in men and the thirteenth most common malignancy in women worldwide [1]. On a yearly basis, there are about half a million new cases globally [2], with more than 63,000 occurrences in Europe [3]. The term oral cancer (OC) is sometimes used synonymously with oral squamous cell carcinoma (OSCC), which is the most prevalent kind of oral tumor. Over 90% of all oral neoplasms are estimated to be oral squamous cell carcinoma [4]. Cancers constitute intricate tissues. They comprise tumor cells and adjacent stroma, which are formed by diverse mesenchymal cell types and the extracellular matrix (ECM). This tissue is collectively known as the tumor microenvironment (TME). Consequently, the tumor cell-centric perspective on cancer

fails to consider the environment in which dangerous cells exist. The adjacent microenvironment becomes active as cancer advances due to ongoing tumor-stromal interactions [5]. EMT causes cell-cell adherence (CCA) to be disrupted, apicobasal polarity to be lost, matrix remodeling to occur, and increased motility and invasiveness to occur [6, 7].

β-catenin, a 92-kDa protein, acts as both a structural component at cell-cell adhesion junctions and a transcriptional activator that facilitates the propagation of Wnt signals throughout the cell [8]. β-catenin is encoded by the CTNNB1 gene [9]. In order to bind to the actin cytoskeleton, β-catenin forms complexes with E-cadherin and α-catenin. Cellular chaos and the loss of CCA are outcomes of any disruption to β-catenin binding or gene deletion [10]. In humans, the CTNNB1 gene encodes the protein β-catenin, more often known as β-cat [9]. The role of β-catenin in CCA is to regulate cell adhesion at the plasma membrane mediated by cadherin and facilitate the interaction between adherens junction molecules and the actin cytoskeleton [11]. Some have suggested that the beginning and progression of HNSCC are linked to the reduced cell adhesion caused by β-cat/E-cad complexes [12]. This section of β-catenin is crucial for cell adhesion because it links E-cadherin (E-cad) to α-catenin (α-cat) and then to the actin microfilament structure of the cytoskeleton. Several cancers show dysregulation of the cadherin-catenin complex and reduced expression of β-catenin, which has been confirmed by multiple investigations [13]. In normal epithelial cells, β-catenin is found on the plasma membrane, where it forms a physical bond with cytoskeletal proteins (such as β-catenin and actinin-4) and cell-to-cell junctional proteins (like E-cadherin, for example) [14]. Evidence from earlier research suggests that aberrant β-catenin signaling has a role in the development of numerous malignancies and could potentially lead to benign tumor transformation. Ultimately, it appears that mutations in the β-catenin gene are unrelated to the observation of altered β-catenin expression or location in numerous OSCC cells [15].

1.1 The Aim

The study aims to evaluate the immunohistochemical expression of β-catenin and correlate its expression with age, gender, tumor site, clinical presentation, and histopathological grade of oral squamous cell carcinoma (OSCC).

2 Materials and Methods

From the Oral and Maxillofacial Pathology Department's archives at the University of Baghdad's College of Dentistry, 45 OSCC excisional blocks that were preserved with formalin and embedded in paraffin were retrieved for this study, as well as the Pathology Departments of Al-Shaheed Ghazi and Al-Yarmouk hospitals (2018–2024).

Control Samples

- Positive Tissue Control: Positive control slides, obtained per the antibody manufacturer's data sheet, ensured the accuracy of the staining technique, with one control involved in all test paths. Tissue blocks of colon adenocarcinoma were used for β-catenin monoclonal antibody.
- Negative Tissue Control: All reagents were applied except for the primary antibodies to confirm specificity.

2.1 Tissue Preparation and Staining

The specimens were serially sectioned after being fixed in formalin and embedded in paraffin.

- 4 µm sections were stained with H&E for histopathological re-evaluation.
- Five additional 4 µm sections were mounted on positively charged slides (Fisher Scientific, USA) for immunohistochemical (IHC) staining using three monoclonal and two polyclonal antibodies. IHC staining was performed for β-catenin. Two experienced pathologists confirmed diagnoses based on H&E sections. Demographic and clinical data, including patient age, sex, tumor site, and staging/grading [15], were obtained from case records.

2.2 Detection System

The Abcam anti-mouse and rabbit HPR/DAB IHC detection kit (Catalog No. ab80436) was used. This kit is a biotin-free immunoenzymatic antigen detection system. This technique involves the sequential incubation of the specimen with an unconjugated rabbit or mouse primary Ab specific to the target antigen, a rabbit anti-mouse secondary Ab, a secondary Ab-HRP conjugate, and substrate-chromogen (DAB).

2.3 Reagents within the Kit Include

- Hydrogen peroxide block (15 ml ready to use)
- Protein block (15 ml ready to use)
- Mouse-specifying reagent (rabbit anti-mouse Ab, unconjugated, ready to use).
- Goat anti-rabbit HRP conjugate (ready to use).
- DAB plus chromogen 50x.
- DAB plus substrate (15 ml).

2.4 Materials and Reagents Used but Not Supplied with the Kit

- Antigen Retrieval Solutions: Citrate buffer pH 6.0.
- Common Ab diluent (Riedel-deHaen Buffer solution pH 7.0 with anti-fungicide, Germany)
- Phosphate buffer pH 7.0 as a washing buffer (Syrbio)

- Hematoxylin solution as a counterstain (Syrbio)
- Absolute ethanol (Scharlau, Spain)
- Xylene (Scharlau, Spain).
- Distilled water.
- Mounting medium: DPX (Syrbio).

3 Results

In Table 1 and Figure 4, we can see the age distribution of the study sample. With an age range of 22 to 82 years (mean ± SD = 55.67 + 15.45), the seventh decade had the highest incidence rate, with 14 cases, or 31.11 percent.

TABLE 1. Age distribution of 45 OSCC according to 10-year age intervals.

Age interval (years)	No.	%
20-29	4	8.89
30-39	3	6.67
40-49	9	20.00
50-59	6	13.33
60-69	14	31.11
70-79	71	15.56
> 80	2	4.44
Total	45	100
Mean age	55.67±15.45	
Median	60	
Range	22-82	

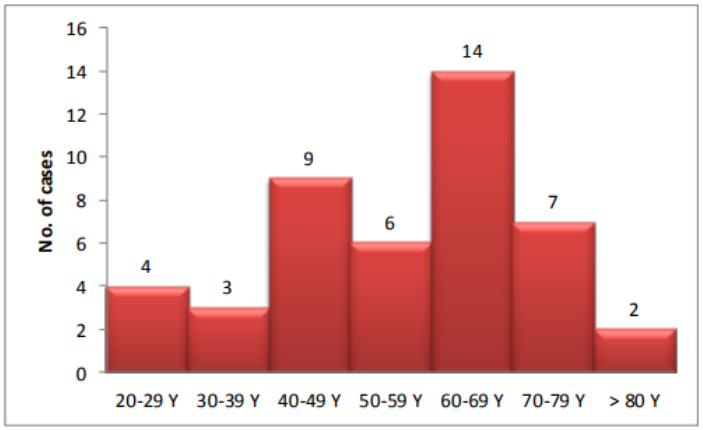

Fig. 4. Distribution of age in OSCC according to 10-year intervals.

There were twenty-seven male patients and eighteen female patients, for a ratio of 1.5:1 (Figure 5).

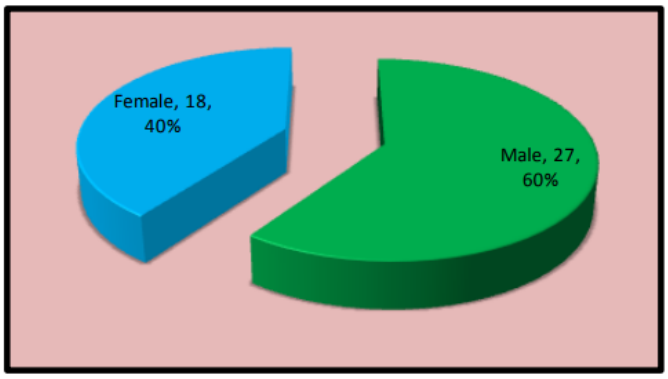

Fig. 5. Distribution of 45 OSCC cases according to gender.

Out of the total instances examined, twenty-four (53.33%) manifested as masses, while the remaining twenty-one (46.67%) showed signs of ulceration (Figure 6).

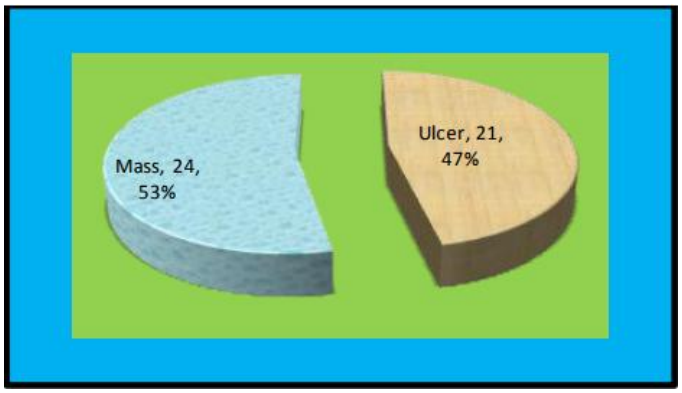

Fig. 6. Clinical presentation of 45 OSCC cases.

Most of the primary sites were found on the tongue (22 cases = 48.89%), next on the lip (8 cases = 17.78%), and finally on the floor of the mouth (1 case = 2.22%). Data in Table 2 and Figures 7 and 8.

TABLE 2. Site distribution of 45 OSCC cases.

Site	No.	%
Buccal mucosa	6	13.33
Tongue	22	48.89
Lip	8	17.78
Gingiva	3	6.67
Palate	2	4.44
Floor of month	1	2.22
Maxilla	3	6.67
Total	45	100

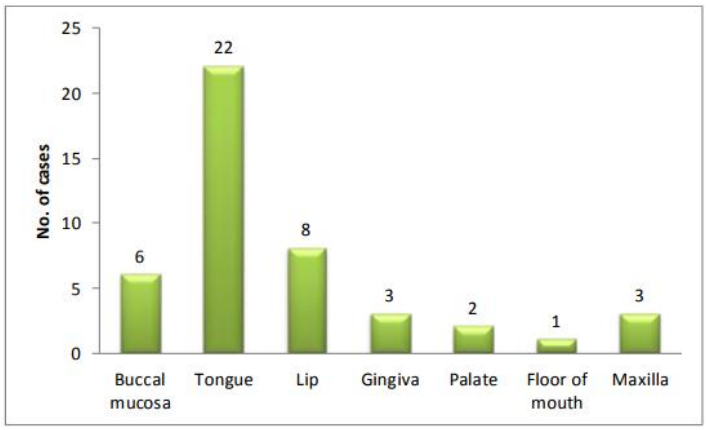

Fig. 7. Distribution of 45 OSCC cases according to site.

According to TNM classification, the majority of the patients had primary lesions with size T2 (18 patients = 40%); the remaining were as follows: T3 (12 patients = 26.67%), T1 (9 patients = 20%), and T4 (6 patients = 13.33%). Pathologically, 12 out of 45 patients (26.66%) exhibited lymph node involvement. Among these, six patients (13.33%) were classified as N1, and six patients (13.33%) as N2. There were no distant metastases in all 45 cases. Most of the identified OSCCs were stage II (15 cases = 33.33%), followed by stage III (12 cases = 26.67%), while stages I and IV represented 9 cases (20%) for each (Table 3).

TABLE 3. TNM and staging of 45 OSCC cases.

Criteria	T		N		M	
	No.	%	No.	%	No.	%
0			33	73.33	45	100
1	9	20	6	13.33		
2	18	40	6	13.33		
3	12	26.67				
4	6	13.33				
Stage	No.		%			
I	9		20.00			
II	15		33.33			
III	12		26.67			
IV	9		20.00			
Total	45		100			

3.1 Evaluation of β-Catenin Immunohistochemistry

Immunohistochemical staining of β-cat was detected as brown staining in the cytoplasm and/or nucleus of target antigen cells. Positive IHC expression was found in all 45 cases (100%). About half of the cases were reported as score 2 (23 cases = 51.11%), followed by score 3 (15 cases = 33.33%). The rest, 7 cases (15.56%), showed a score of 1 (Table 4, Figures 8-10).

TABLE 4. Immunohistochemical expression of the study biomarkers.

Score	ß-catenin	
	No.	%
1	7	15.56
2	23	51.11
3	15	33.33
4	---	---
Total	45	100

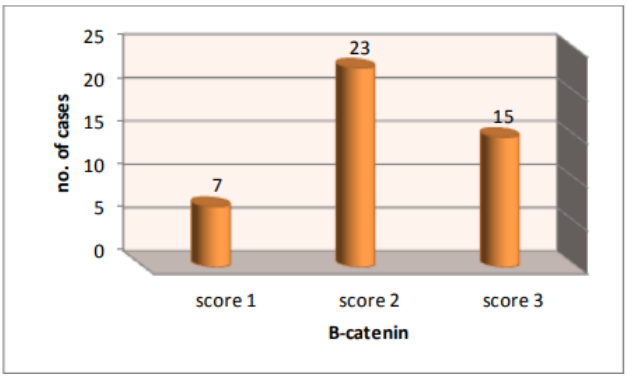

Fig. 8. Frequency distribution of β-cat expression in 45 cases OSCC.

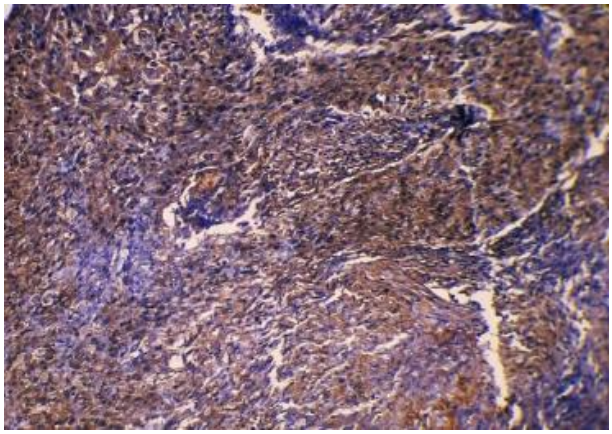

Fig. 9. Positive cytoplasm and/or nuclear immunostaining of β-cat in OSCC (10X).

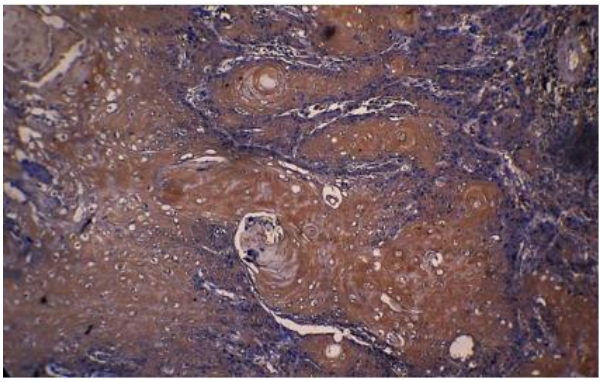

Fig. 10. Positive cytoplasm and/or nuclear immunostaining of β-cat in OSCC (40X).

3.2 Correlation of β-Cat with Age, Gender, Site and Clinical Presentation of OSCC

There was no statistically significant link found between β-cat expression and age (P-value=0.095), gender (P-value = 0.313), or site (P-value = 0.456) in the present investigation, as determined by the Chi-Square test. Tables 5-8 show the clinical presentation with a p-value of 0.366.

TABLE 5. Correlation of β-cat with the age.

Correlation parameters*	β-catenin
R	0.252
P	0.095
*r: Correlation coefficient; p: level significant	

TABLE 6. Correlation of β-cat with the gender in OSCC.

Gender	β-catenin IHC					
	1		2		3	
	No.	%	No.	%	No.	%
Male	6	13.33	13	28.89	8	17.78
Female	1	2.22	10	22.22	7	15.56
Total	7	15.56	23	51.11	15	33.33
P = 0.313 No significant						

TABLE 7. Correlation of β-cat with the site in OSCC.

Site		Buccal mucosa	Tongue	Lip	Gingiva	Palate	Floor of mouth	Maxilla
β-catenin	1	2	1	2	1	0	1	0
	2	3	13	3	1	1	0	2
	3	1	8	3	1	1	0	1
Total		6	22	8	3	2	1	3
= 0.456 No significant								

TABLE 8. Correlation of β-cat with the clinical presentation in OSCC.

β-catenin Score	Clinical presentation			
	Ulcer		Mass	
	No.	%	No.	%
1	3	6.67	4	8.89
2	13	28.89	10	22.22
3	5	11.11	10	22.22
Total	21	46.67	24	53.33
P = 0.366 No significant				

Regarding tumor staging and grading, the present study showed a statistically non-significant association between β-cat and stage of the tumor (P = 248), while a highly significant association was found with tumor grade (P-value = 0.001), as shown in Table 9.

TABLE 9. Association between-cat with tumor stage and grade.

β-catenin		
Stage	r	0.152
	p	0.248
Grade	r	0.633
	p	0.001

4 Discussion

The prediction of OSCC performance differs when utilizing standard histological and clinical indicators. Consequently, investigations into molecular biomarkers, surrounding cell adhesion, and matrix degradation indicators have been conducted as promising instruments for prognostic prediction in patients with OSCC. Immunohistochemistry (IHC) helps as a valuable instrument for elucidating predictive tumor markers correlated with the clinical outcomes of OSCC [17, 18].

Ozawa et al. in 1989 originally described β-catenin as one of several structural proteins that connect E-cadherin to the cytoskeleton in mouse cells. According to some reports, oral squamous cell carcinoma (OSCC) and other human cancer subtypes may share a link to tumorigenesis and deregulated β-catenin expression. This link may be directly related to the activation of the Wnt/β-catenin signaling pathway, which in turn triggers the transcription of specific genes that regulate important biological processes like proliferation, differentiation, and epithelial-mesenchymal transition [19, 20].

The current investigation found diminished membrane localization and pronounced cytoplasmic and/or nuclear staining, with β-cat overexpression detected in 51.11% and 33.33%, corresponding to scores 2 and 3, respectively. The results are in line with those of Ravindran and Devaraj in 2012 and Laxmidevi et al. in 2010, who found that β-catenin was stabilized and mostly located in the cytoplasm, with reduced expression on the cell membrane. This suggests that β-catenin is also functioning as a signaling molecule [21, 22]. Additionally, Kypta and Waxman (2012) concluded that the overexpression of β-catenin is attributed to excessive Wnt signaling [23]. A Wnt-independent signaling route may be involved in the transition from a normal to a neoplastic state since not all reported cases have shown the presence of Wnt signaling molecules. Because of this, there is conflicting evidence about whether OSCC samples exhibited increased or decreased expression of Wnt protein members [24].

However, not all documented instances have demonstrated the presence of Wnt signaling molecules, which implies that a Wnt-independent signaling pathway may be implicated in the change from a normal to a neoplastic state. Hence, some research has shown that OSCC samples had higher expression of Wnt protein members, while others have failed to do so [25].

The findings of the current investigation revealed a statistically non-significant association between β-cat expression and age, gender, location, and clinical presentation. This finding corroborated the investigations conducted by Gao et al. (2005) and Mahomed et al. (2007) [26, 27].

This study's results show that β-catenin expression was not significantly correlated with tumor stage (P = 0.248), but it was correlated with tumor grade (P = 0.001). Moderately and poorly differentiated tumors mostly exhibited cytoplasmic and/or nuclear expression, indicating that β-catenin plays a pivotal role in tumor facilitation. Progression by enhancing tumor cell proliferation and diminishing the efficacy of cell adhesion mechanisms. This aligns with the work conducted by Ravindran on differentiated tumors, emphasizing the fact that active Wnt signaling facilitates the growth of OSCC. The favorable correlation of β-catenin expression between well-differentiated and poorly differentiated OSCC, as well as between moderately differentiated and poorly differentiated OSCC, was found by Laxmidevi et al. (2010) [28]. Similarly, Lo Muzio et al. (1999) established an inverse correlation between the cellular localization of β-catenin expression and the level of differentiation in OSCC, indicating that diminished membrane expression correlated with lower differentiation [29]. In support of this finding, Zhi-gang et al. (2008) observed that poorly differentiated carcinoma primarily exhibited nuclear and cytoplasmic β-cat immunoreactivity. This further supports the idea that the OSCC is more likely to progress to advanced stages and metastasize if the Wnt signaling pathway is activated [30].

5 Conclusion

The study found that β-catenin, a signaling molecule, reduces membrane localization and increases cytoplasmic and nuclear staining. Its expression is strongly associated with tumor grade, suggesting it promotes tumor progression by enhancing proliferation and weakening cell adhesion.

Conflict of Interest: The authors declare no conflict of interest.

Financing: The study was performed without external funding.

Ethical consideration: The study was approved by University of Diyala, Diyala, Iraq.

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