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Immunohistochemical Expression and Subcellular Localization of β-cateninin in Salivary Gland Malignancies

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Abstract

Objective(s): β-catenin is involved in cell-cell adhesion and Wnt signaling pathway, and alterations in its expression or subcellular localization may reflect biological changes in salivary gland malignancies. This study aimed to evaluate the immunohistochemical expression and subcellular localization of β-catenin in selected salivary gland malignancies. **Methods:** This retrospective cross-sectional study included 37 archived cases of salivary gland malignancies including mucoepidermoid carcinoma (N=16), adenoid cystic carcinoma (N=14), polymorphous adenocarcinoma (N=4), adenocarcinoma not otherwise specified (N=2), and secretory carcinoma (N=1). β-catenin immunohistochemistry was performed on formalin-fixed paraffin-embedded tissue sections. Staining extent was scored from 0 to 3, and localization was recorded as membranous, membranous/cytoplasmic and nuclear. Data were analyzed using Fisher exact, Kruskal-Wallis, Mann-Whitney U tests, and Bonferroni pairwise test at $p<0.05$. **Results:** β-catenin was positive in 35 cases (94.6%), and strong expression was observed in 26 cases (70.3%). Expression scores differed significantly among diagnostic groups ($p<0.05$); however, after Bonferroni pairwise test, only the difference between mucoepidermoid carcinoma and adenocarcinoma not otherwise specified remained significant ($p<0.05$). Combined membranous/cytoplasmic staining was the most frequent localization pattern (75.7%), followed by membranous staining (18.9%). No nuclear staining was detected. Cellular site of staining was significantly associated with histopathologic diagnosis ($p<0.001$). **Conclusion:** β-catenin may contribute to the pathogenesis and biological behavior of salivary gland malignancies, mainly through changes in its subcellular localization rather than simple positive or negative expression. Cytoplasmic expression may reflect altered adhesion-related function, although the absence of nuclear staining did not support overt canonical Wnt/β-catenin activation in this series.

Keywords: β-Catenin; Salivary Gland Neoplasms; Immunohistochemistry; Mucoepidermoid Carcinoma; Adenoid Cystic Carcinoma; Wnt Signaling Pathway

Introduction

Salivary gland malignancies are uncommon, heterogeneous head and neck neoplasms with broad histopathologic diversity and variable clinical behaviors. The most common malignant salivary gland tumors include mucoepidermoid carcinoma, adenoid cystic carcinoma, and polymorphous adenocarcinoma. Their diagnosis and risk assessment depend mainly on conventional pathologic criteria including the tumor type, grade, growth pattern, perineural invasion, and other invasive features. However, these criteria may not fully explain the biologic behavior of all cases, and additional immunohistochemical markers may help clarify tumor biology [1-5].

β-catenin is a multifunctional protein involved in both cell-cell adhesion and intracellular signaling [6-9]. At cell membrane, it interacts with E-cadherin as part of the cadherin-catenin complex and contributes to epithelial integrity [6, 7]. In canonical Wnt pathway, β-catenin may accumulate in the cytoplasm and translocate to the nucleus, where it regulates the transcription of genes involved in proliferation, survival, invasion, and tumor progression [8-10].

For this reason, β-catenin immunohistochemistry should be interpreted according to both expression and subcellular localization [6, 8-10]. Membranous staining is generally related to cell adhesion, whereas reduced membranous expression and cytoplasmic or nuclear accumulation may reflect altered adhesion and possible Wnt/ β-catenin pathway dysregulation [6, 8-10, 12, 13, 17-20].

Previous studies on β-catenin in salivary gland tumors have reported variable findings [11-20]. Some studies have described cytoplasmic redistribution or reduced membranous expression in malignant tumors, while others have demonstrated predominantly membranous or non-nuclear patterns [11-20]. Data are also limited for less common entities such as polymorphous adenocarcinoma, adenocarcinoma not otherwise specified, and secretory carcinoma [11, 12, 20, 22-24].

The aim of the present study was to evaluate the immunohistochemical expression and subcellular localization of β-catenin in selected salivary gland malignancies and to

assess its association with histopathologic diagnosis and clinicopathologic variables.

Methods

This retrospective cross-sectional study was approved by the Ethics Committee of Shahid Beheshti University of Medical Sciences, Tehran, Iran (IR.SBMU.DRC.REC.1403.167). Archives of the Department of Oral and Maxillofacial Pathology, School of Dentistry, Shahid Beheshti University of Medical Sciences, Tehran, Iran served as the data source. Archived hematoxylin and eosin-stained slides and corresponding formalin-fixed paraffin-embedded blocks of selected salivary gland malignancies diagnosed from March 2001 to September 2025 were reviewed.

Initially, 52 archived cases diagnosed as mucoepidermoid carcinoma (MEC), adenoid cystic carcinoma (AdCC), polymorphous adenocarcinoma (PAC), adenocarcinoma not otherwise specified (NOS), or secretory carcinoma were identified. Because of the rarity of these tumors, census sampling was used. Cases were included when the diagnosis was confirmed on microscopic review, the paraffin block and hematoxylin and eosin-stained slide were available, tissue was adequate for immunohistochemistry, fixation quality was acceptable, and clinicopathologic data were complete.

Cases with unavailable blocks, insufficient tissue, incomplete clinical records, consultation-only status, uncertain diagnosis, inadequate evaluable tumor tissue, or poor staining quality were excluded. After applying these criteria, 37 cases were finally included in the study. Age, sex, anatomic location, major or minor salivary gland origin, and final histopathologic diagnosis were recorded. Immunohistochemistry for β-catenin was performed on 2-3 μm sections. Slides were deparaffinized in xylene, rehydrated through graded alcohols, and treated with 3% hydrogen peroxide for 15 minutes. Antigen retrieval was performed in citrate buffer (pH=6.0) for 20 minutes in a microwave oven. Sections were incubated with mouse monoclonal anti-β-catenin antibody, clone 14, dilution 1:100 (Cat. No. 610153, BD Transduction Laboratories, San Jose, CA, USA), for one hour at room temperature. A secondary antibody system from Master Diagnostica was applied, followed by horseradish peroxidase and 3,3-diaminobenzidine visualization. Sections were counterstained with hematoxylin. Normal human testis was used as positive control, and negative controls were prepared by omitting the primary antibody.

The extent of β-catenin staining was scored as follows: 0, for absence of staining; 1, for weak focal staining in less than 10% of tumor cells; 2, for moderate staining in 10%-80% of tumor cells; and 3, for strong staining in more than 80% of tumor cells. This scoring system was adapted from Schneider et al [14]. The dominant localization was recorded as membranous, membranous/cytoplasmic, nuclear, or absence of staining [6, 8, 12, 13].

Data were analyzed using SPSS version 27.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were reported as means, standard deviations, ranges, frequencies, and percentages. The Kruskal-Wallis test was used to compare expression scores among diagnostic and anatomic groups, and Bonferroni test was used for pairwise comparisons. The Mann-Whitney U test was used for comparisons according to sex and major/minor salivary gland origin. Fisher exact test was used for categorical variables. The level of significance was set at 0.05.

Results

Table 1 shows demographic information of selected salivary gland malignancies.

Table 1- Demographics of samples

Diagnosis	N	Mean age ± SD (range)	Male	Female	Major/Minor gland
Mucoepidermoid carcinoma	16	48.00±18.15 (22-77)	6	10	1/15
Adenoid cystic carcinoma	14	54.64±8.73 (36-70)	7	7	2/12
Polymorphous adenocarcinoma	4	39.75±7.89 (29-48)	2	2	1/3
Adenocarcinoma NOS	2	57.50±3.54 (55-60)	0	2	2/0
Secretory carcinoma	1	50.00 (50)	0	1	1/0
Total	37	50.19±13.97 (22-77)	15	22	7/30

β-catenin immunoreactivity was detected in 35 of 37 cases (94.6%). Overall, strong expression was the most common score and was observed in 26 cases (70.3%), followed by moderate expression in seven cases (18.9%), weak expression in two cases (5.4%), and absence of expression in two cases (5.4%).

β-catenin expression score differed significantly among the histopathologic groups (Kruskal-Wallis test, H=11.051, df=4, $p<0.05$). Strong expression was most frequent in MEC, where 14 of 16 cases (87.5%) showed strong staining. In AdCC, eight out of 14 cases (57.1%) showed strong expression and six cases (42.9%) showed moderate expression. In PAC, three out of four cases (75.0%) showed strong expression and one case (25.0%) showed weak expression. Both adenocarcinoma NOS cases showed the absence of β-catenin expression, whereas the single secretory carcinoma case showed strong expression. The distribution of β-catenin expression scores according to histopathologic diagnosis is shown in Table 2.

Table 2- β-catenin expression score according to histopathologic diagnosis

Diagnosis	Absence	Weak (<10%)	Moderate (10%-80%)	Strong (>80%)	Total
Mucoepidermoid carcinoma	0 (0.0%)	1 (6.3%)	1 (6.3%)	14 (87.5%)	16 (100.0%)
Adenoid cystic carcinoma	0 (0.0%)	0 (0.0%)	6 (42.9%)	8 (57.1%)	14 (100.0%)
Polymorphous adenocarcinoma	0 (0.0%)	1 (25.0%)	0 (0.0%)	3 (75.0%)	4 (100.0%)
Adenocarcinoma NOS	2 (100.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	2 (100.0%)
Secretory carcinoma	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (100.0%)	1 (100.0%)
Total	2 (5.4%)	2 (5.4%)	7 (18.9%)	26 (70.3%)	37 (100.0%)

Pairwise comparisons between diagnostic groups showed significant differences in the unadjusted analyses between adenocarcinoma NOS and AdCC ($p<0.05$), PAC ($p<0.05$), MEC ($p<0.05$), and secretory carcinoma ($p<0.05$). However, after Bonferroni pairwise test, only the difference between adenocarcinoma NOS and MEC demonstrated statistically significant difference (adjusted $p<0.05$).

β-catenin expression score was also evaluated after categorizing the diagnoses into three groups: MEC, AdCC, and a combined group including PAC, adenocarcinoma NOS, and secretory carcinoma. In this grouped analysis, the difference in β-catenin expression score was not statistically significant (Kruskal-Wallis test, H=4.093, df=2, p=0.129). The mean ranks were 22.16 for MEC, 17.43 for AdCC, and 14.93 for the combined group.

The subcellular localization of β-catenin differed significantly among histopathologic groups (Fisher's exact test, $p<0.001$). The most common overall pattern was combined membranous and cytoplasmic staining, observed in 28 cases (75.7%), followed by exclusively membranous staining in seven cases (18.9%). Nuclear expression was not observed in any case, and the two negative cases corresponded to adenocarcinoma NOS.

In MEC, β-catenin staining was observed in epidermoid, goblet, and intermediate cells, whereas mucous cells showed only membranous staining. In AdCC, staining was observed in both ductal epithelial and myoepithelial cells and was present in cribriform, solid, and tubular patterns (Figures1-4). The subcellular localization patterns of β-catenin according to histopathologic diagnosis are summarized in Table 3.

No significant association was found between β-catenin expression score and sex (Mann-Whitney U test, U=127.5, p=0.249), anatomic location (Kruskal-Wallis test, H=7.433, df=6, p=0.272), or major versus minor salivary gland origin (Mann-Whitney U test, U=97.5, p=0.776). Age distribution also did not differ significantly among the diagnostic groups (Kruskal-Wallis test, H=7.240, df=4, p=0.124).

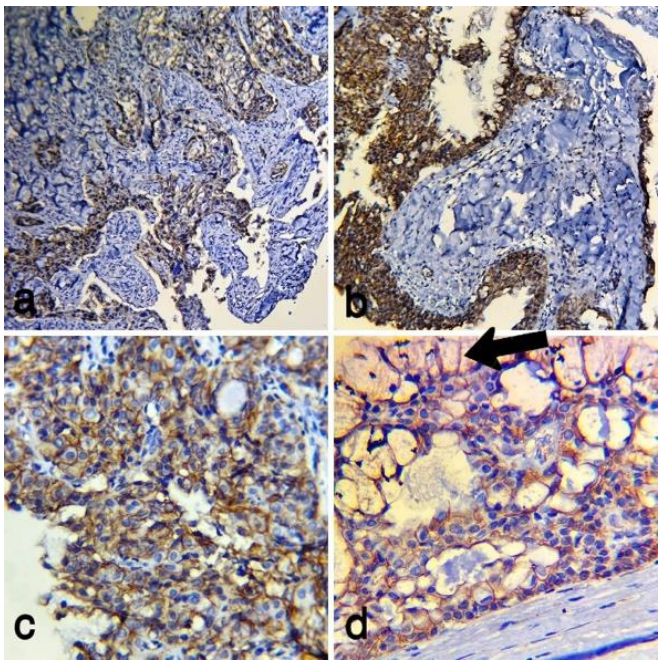

Figure 1: Mucoepidermoid carcinoma. a, b) strong β-catenin expression in nests and islands (IHC, ×100). C, d) β-catenin-positive cells with membranous and cytoplasmic brown staining in nests and islands of neoplastic cells. Note that the mucous cells show only membranous positivity (IHC, ×400).

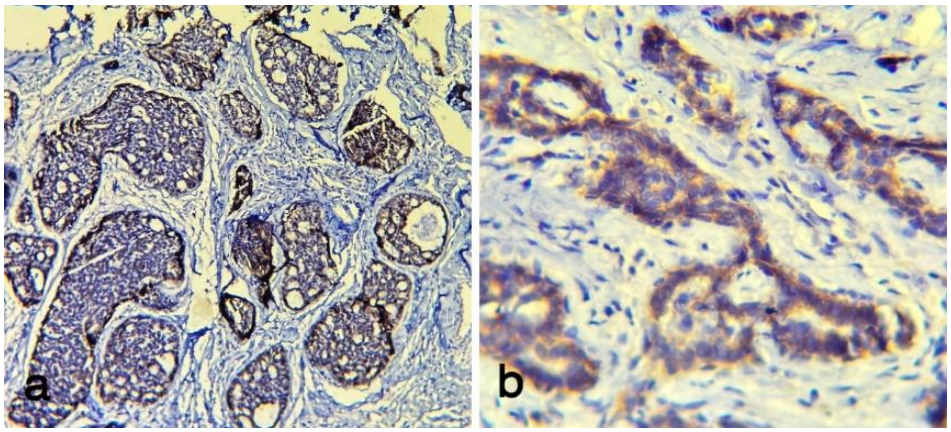

Figure2: Adenoid cystic carcinoma. a, b) β-catenin-positive cells with membranous and cytoplasmic brown staining in nests and islands of tumoral cells (IHC, 100×, 400×).

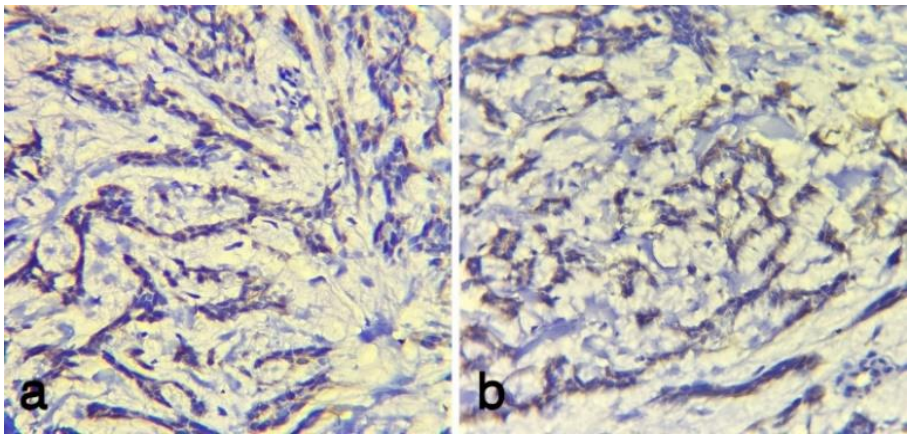

Figure3: Polymorphous adenocarcinoma. a, b) β-catenin positive cells with membranous and cytoplasmic brown staining in nests and single file pattern (IHC, 400×).

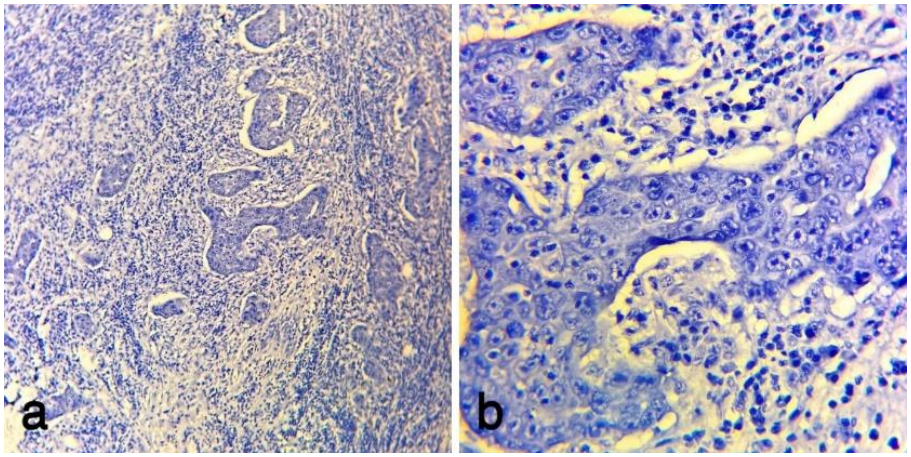

Figure 4: Adenocarcinoma NOS. a, b) tumoral cells are negative for β-catenin marker (IHC, 100×, 400×).

Table 3- Subcellular localization of β-catenin according to histopathologic diagnosis

Diagnosis	Absent	Membranous	Membranous/Cytoplasmic	Total
Mucoepidermoid carcinoma	0 (0.0%)	2 (12.5%)	14 (87.5%)	16 (100.0%)
Adenoid cystic carcinoma	0 (0.0%)	2 (14.3%)	12 (85.7%)	14 (100.0%)
Polymorphous adenocarcinoma	0 (0.0%)	3 (75.0%)	1 (25.0%)	4 (100.0%)
Adenocarcinoma NOS	2 (100.0%)	0 (0.0%)	0 (0.0%)	2 (100.0%)
Secretory carcinoma	0 (0.0%)	0 (0.0%)	1 (100.0%)	1 (100.0%)
Total	2 (5.4%)	7 (18.9%)	28 (75.7%)	37 (100.0%)

Discussion

The present study aimed to evaluate the immunohistochemical expression and subcellular localization of beta-catenin in salivary gland malignancies. The main findings of the current study were that β-catenin was expressed in most cases, the expression score differed significantly among histopathologic groups, and the localization pattern was significantly associated with tumor type. This pattern suggests that β-catenin was largely preserved in tumor cells, but in many cases, it was not restricted to its physiologic membranous compartment [6, 7]. The predominance of combined membranous and cytoplasmic staining may indicate partial redistribution of β-catenin from the adhesion complex, whereas the absence of nuclear staining argues against overt nuclear activation of the canonical Wnt/beta-catenin pathway detectable by IHC in this series [6-10].

In MEC, β-catenin showed a predominantly membranous/cytoplasmic pattern without nuclear expression. This finding suggests that β-catenin was generally preserved in MEC in the present series, but its distribution was not limited to the cell membrane [6, 7]. The cytoplasmic component may indicate partial redistribution of β-catenin from the cadherin-catenin adhesion complex into the intracellular compartment [6, 7, 12, 13]. However, because nuclear expression was absent, the findings do not provide direct immunohistochemical evidence of marked canonical Wnt/β-catenin pathway activation in these cases [8-10, 17-19].

Previous studies have reported variable β-catenin patterns in MEC [12, 13, 17-19]. Chandrashekar et al. observed membranous, cytoplasmic, and combined membranous/cytoplasmic staining patterns in MEC and suggested that reduced membranous expression and cytoplasmic localization may be related to decreased differentiation and invasive potential [12]. Batool et al. also reported β-catenin positivity in MEC, mainly with cytoplasmic or combined membranous/cytoplasmic expression, without nuclear staining [13]. In contrast, some studies have reported non-membranous or cytoplasmic/nuclear β-catenin expression in subsets of MEC and interpreted these findings as possible evidence of Wnt pathway dysregulation or tumor progression [17-19]. Therefore, the predominance of membranous/cytoplasmic staining without nuclear expression in the present study is partly consistent with previous reports, but does not support a prominent nuclear β-catenin pattern in the evaluated MEC cases [12, 13, 17-19].

In AdCC, all cases were positive for β-catenin, with moderate or strong expression. Similar to MEC, the most common localization pattern was combined membranous and cytoplasmic staining, and no nuclear expression was observed. This pattern suggests that β-catenin remains partly associated with membranous adhesion structures, while cytoplasmic redistribution is also present [6, 7, 12, 13]. The absence of nuclear staining indicates that, in this series, β-catenin did not show a clear nuclear translocation pattern in AdCC [8-10, 15, 16].

The findings in AdCC are comparable to some previous studies, but differ from the others [11-16, 21]. Furuse et al. reported mainly membranous β-catenin expression in AdCC, although reduced expression could be seen in less differentiated areas [11]. Some studies have reported cytoplasmic or aberrant non-nuclear patterns in malignant salivary gland tumors, including AdCC, and suggested that such changes may be related to an aggressive behavior [12, 13]. On the other hand, Zhou and Gao reported cytoplasmic/nuclear β-catenin staining in a subset of AdCC cases and observed reduced membranous expression in metastatic cases [16]. Wang et al. also suggested that aberrant Wnt/β-catenin signaling may contribute to invasion and metastasis in salivary AdCC [15]. Since the present study did not evaluate long-term clinical outcomes, recurrence, metastasis, or survival, the prognostic significance of β-catenin localization could not be determined.

In PAC, β-catenin showed predominantly membranous expression, although one case also demonstrated combined membranous/cytoplasmic staining. This pattern may still suggest relative preservation of the adhesion-related role of β-catenin in these tumors; however, the presence of a cytoplasmic component in one case may reflect partial dissociation of β-catenin from the membranous cadherin-catenin complex and its redistribution into the cytoplasm [6, 7, 12, 13]. This finding should be interpreted cautiously because of the limited number of cases. Furuse et al. reported strong membrane-associated β-catenin expression in PAC, which is in line with the membranous pattern observed in our cases [11]. Chandrashekar et al. also reported membranous staining in one PAC case and combined membranous/cytoplasmic staining in another [12]. In contrast, Ferrazzo et al. described cytoplasmic/nuclear beta-catenin expression with loss of membranous staining in many PAC cases [20]. These differences suggest that β-catenin localization in PAC may be variable across the studies and may be influenced by small sample sizes, diagnostic criteria, antibody protocols, and scoring methods [11, 12, 20, 22].

Both cases of adenocarcinoma NOS in the present study were negative for β-catenin. This finding was notable because adenocarcinoma NOS was the only diagnostic group in which a complete absence of staining was observed. Negative IHC staining may be influenced by technical factors such as fixation quality, antigen retrieval, tissue preservation, or intratumoral heterogeneity [11-14, 19].

Previous reports have not consistently shown complete absence of β-catenin in adenocarcinoma-related salivary gland malignancies [13, 14]. Schneider et al. reported β-catenin expression in most minor salivary gland carcinomas, although the intensity varied among the tumor types [14]. Batool et al. also reported β-catenin positivity in malignant salivary gland tumors, including carcinoma ex pleomorphic adenoma [13]. Therefore, the absence of staining in the two adenocarcinoma NOS cases in the present study should be considered an interesting finding that requires confirmation in larger case series.

In the single secretory carcinoma case, β-catenin showed strong membranous/cytoplasmic expression without nuclear staining, which is in line with the limited previous reports describing β-catenin positivity without nuclear expression in this entity [13].

Overall, β-catenin expression was detected in most cases, suggesting that simple positive/negative interpretation alone has limited discriminatory value in this series. However, the significant difference in expression score among histopathologic groups indicates that the extent of β-catenin expression was not uniform across tumor types. The only significant difference after Bonferroni pairwise test was observed between MEC and adenocarcinoma NOS, mainly reflecting strong expression in most MEC cases and absence of staining in both adenocarcinoma NOS cases; nevertheless, this finding should be interpreted cautiously because of the small number of adenocarcinoma NOS cases. In the grouped diagnostic analysis, expression score was no longer significant, probably because the combined group included biologically heterogeneous tumors with different staining behaviors, which may have diluted subtype-specific differences. In contrast, β-catenin localization remained significantly associated with tumor type however, this finding should be interpreted with caution due to the small number of cases in certain diagnostic categories, particularly the adenocarcinoma NOS group, which included only two cases. These results suggest that interpretation of β-catenin immunostaining in salivary gland malignancies should not be based only on the presence or absence of staining, but should also include the dominant cellular compartment of expression [6, 8, 9, 12, 13]. When assessed together with expression score, subcellular localization may provide a more informative interpretation of β-catenin immunostaining [6, 12, 13]. In this context, membranous staining may reflect preservation of the adhesion-related role of β-catenin, whereas cytoplasmic staining may represent redistribution from the cadherin–catenin complex [6, 7, 12, 13]. The predominance of membranous/cytoplasmic staining, together with the absence of nuclear expression, supports cytoplasmic redistribution rather than overt nuclear activation of the canonical Wnt/β-catenin pathway detectable by immunohistochemistry [8-10, 11-13, 15-20].

No significant association was found between β-catenin expression score and sex, anatomic location, or major versus minor salivary gland origin. This finding suggests that, in the present series, β-catenin expression patterns were more closely related to histopathologic subtype, than to demographic or anatomic variables.

This study had several limitations. First, the sample size was relatively small, especially for polymorphous adenocarcinoma, adenocarcinoma NOS, and secretory carcinoma. Second, the retrospective design had limited our access to complete clinical follow-up data, including the recurrence, metastasis, disease-free survival, and overall survival. Third, some important clinicopathologic parameters, such as the tumor grade, perineural invasion, lymphovascular invasion, surgical margin status, and treatment history were not included in the final statistical analysis. Fourth, immunohistochemistry alone cannot confirm activation of the Wnt/β-catenin pathway [8, 9]. Molecular analysis of pathway-related genes and regulators, such as CTNNB1, APC, AXIN, SFRP1, and WIF1, would be necessary to better clarify the biologic mechanisms underlying β-catenin redistribution [15, 17, 25].

Conclusion

The findings of this study suggested that β-catenin may be involved in the biological behavior and pathogenesis of salivary gland malignancies. However, its diagnostic value appeared to depend less on simple positivity or negativity and more on its subcellular localization. Cytoplasmic expression may reflect altered cell adhesion and possible involvement of β-catenin-related signaling in tumor progression, while the absence of nuclear expression suggests that overt activation of the canonical Wnt/β-catenin pathway was not evident in this series. Therefore, assessment of β-catenin localization may provide more meaningful biological insight than the evaluation of expression alone.

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Informed Consent Statement: The requirement for informed consent was waived due to the retrospective nature of the study and the anonymous use of archived tissue samples and clinicopathologic data.

Data Availability Statement: The datasets generated during and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Using AI: Artificial intelligence–based language editing tools (ChatGPT, OpenAI) were used to assist in improving the grammatical accuracy and clarity of the manuscript.

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

References

1. Moghadam SA, Abadi AM, Mokhtari S. Immunohistochemical analysis of CD34 expression in salivary gland tumors. J Oral Maxillofac Pathol. 2015;19(1):30-3. doi:10.4103/0973-029X.157197

2. Neville BW, Damm DD, Allen CM, Chi AC. Oral and Maxillofacial Pathology. 5th ed. St. Louis: Elsevier; 2024. ISBN: 9780323789813.

3. WHO Classification of Tumours Editorial Board. Head and Neck Tumours. 5th ed. Lyon: International Agency for Research on Cancer; 2024. ISBN: 978-92-832-4514-8.

4. Skálová A, Hyrcza MD, Leivo I. Update from the 5th Edition of the World Health Organization Classification of Head and Neck Tumors: Salivary Glands. Head Neck Pathol. 2022;16(1):40-53. doi:10.1007/s12105-022-01420-1

5. Rito M, Fonseca I. Salivary gland neoplasms: does morphological diversity reflect tumor heterogeneity? Pathobiology. 2018;85(1-2):85-95. doi:10.1159/000479070

6. Valenta T, Hausmann G, Basler K. The many faces and functions of beta-catenin. EMBO J. 2012;31(12):2714-36. doi:10.1038/emboj.2012.150

7. Ozawa M, Baribault H, Kemler R. The cytoplasmic domain of the cell adhesion molecule uvomorulin associates with three independent proteins structurally related in different species. EMBO J. 1989;8(6):1711-7. doi:10.1002/j.1460-2075.1989.tb03563.x

8. Clevers H. Wnt/beta-catenin signaling in development and disease. Cell. 2006;127(3):469-80. doi:10.1016/j.cell.2006.10.018

9. Clevers H, Nusse R. Wnt/beta-catenin signaling and disease. Cell. 2012;149(6):1192-205. doi:10.1016/j.cell.2012.05.012

10. Cadigan KM, Waterman ML. TCF/LEFs and Wnt signaling in the nucleus. Cold Spring Harb Perspect Biol. 2012;4(11):a007906. doi:10.1101/cshperspect.a007906

11. Furuse C, Cury PR, Altemani A, Pinto DS Jr, de Araujo NS, de Araujo VC. Beta-catenin and E-cadherin expression in salivary gland tumors. Int J Surg Pathol. 2006;14(3):212-7. doi:10.1177/1066896906290652

12. Chandrashekar C, Angadi PV, Krishnapillai R. Beta-catenin expression in benign and malignant salivary gland tumors. Int J Surg Pathol. 2011;19(4):433-40. doi:10.1177/1066896909346366

13. Batool H, Khan FW, Bashir A, Rafique Z, Mustafa BE, Babar K, et al. Expression of beta-catenin in salivary gland tumors. Cureus. 2024;16(10):e72249. doi:10.7759/cureus.72249

14. Schneider S, Thurnher D, Seemann R, Brunner M, Kadletz L, Ghanim B, et al. The prognostic significance of beta-catenin, cyclin D1 and PIN1 in minor salivary gland carcinoma: beta-catenin predicts overall survival. Eur Arch Otorhinolaryngol. 2016;273(5):1283-92. doi:10.1007/s00405-015-3609-6

15. Wang R, Geng N, Zhou Y, Zhang D, Li L, Li J, et al. Aberrant Wnt-1/beta-catenin signaling and WIF-1 deficiency are important events which promote tumor cell invasion and metastasis in salivary gland adenoid cystic carcinoma. Biomed Mater Eng. 2015;26(Suppl 1):S2145-53. doi:10.3233/BME-151520

16. Zhou CX, Gao Y. Aberrant expression of beta-catenin, Pin1 and cyclin D1 in salivary adenoid cystic carcinoma: relation to tumor proliferation and metastasis. Oncol Rep. 2006;16(3):505-11. doi:10.3892/or.16.3.505

17. Lee CH, Hung YJ, Lin CY, Hung PH, Hung HW, Shieh YS. Loss of SFRP1 expression is associated with aberrant beta-catenin distribution and tumor progression in mucoepidermoid carcinoma of salivary glands. Ann Surg Oncol. 2010;17(8):2237-46. doi:10.1245/s10434-010-0961-z

18. da Costa Miguel MC, Oliveira MC, Seabra FRG, Queiroz LMG, de Almeida Freitas R, de Souza LB. Beta-catenin and cyclin D1 in mucoepidermoid carcinoma of variable histologic grades. Arch Otolaryngol Head Neck Surg. 2005;131(8):701-6. doi:10.1001/archotol.131.8.701

19. Hakata Y, Fukui H, Sekikawa A, Yamagishi H, Ichikawa K, Tomita S, et al. Expression of beta-catenin and REG Ialpha in relation to cell proliferative ability in salivary gland tumors. Exp Ther Med. 2010;1(3):437-43. doi:10.3892/etm_00000068

20. Ferrazzo KL, Neto MM, dos Santos E, dos Santos Pinto D, de Sousa SOM. Differential expression of galectin-3, beta-catenin, and cyclin D1 in adenoid cystic carcinoma and polymorphous low-grade adenocarcinoma of salivary glands. J Oral Pathol Med. 2009;38(9):701-7. doi:10.1111/j.1600-0714.2009.00776.x

21. Park S, Vora M, van Zante A, Humtsoe J, Kim HS, Yom S, et al. Clinicopathologic implications of Myb and beta-catenin expression in adenoid cystic carcinoma. J Otolaryngol Head Neck Surg. 2020;49(1):48. doi:10.1186/s40463-020-00446-1

22. Castle JT, Thompson LDR, Frommelt RA, Wenig BM, Kessler HP. Polymorphous low grade adenocarcinoma: a clinicopathologic study of 164 cases. Cancer. 1999;86(2):207-19. doi:10.1002/(SICI)1097-0142(19990715)86:2<207::AID-CNCR4>3.0.CO;2-Q

23. Skalova A, Vanecek T, Sima R, Laco J, Weinreb I, Perez-Ordonez B, et al. Mammary analogue secretory carcinoma of salivary glands, containing the ETV6-NTRK3 fusion gene. Am J Surg Pathol. 2010;34(5):599-608. doi:10.1097/PAS.0b013e3181d9efcc

24. Chiosea SI, Griffith C, Assaad A, Seethala RR. Clinicopathological characterization of mammary analogue secretory carcinoma of salivary glands. Histopathology. 2012;61(3):387-94. doi:10.1111/j.1365-2559.2012.04232.x

25. Sato M, Yamamoto H, Hatanaka Y, Nishijima T, Jiromaru R, Yasumatsu R, et al. Wnt/beta-catenin signal alteration and its diagnostic utility in basal cell adenoma and histologically similar tumors of the salivary gland. Pathol Res Pract. 2018;214(4):586-92. doi:10.1016/j.prp.2017.12.016