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(CASE REPORT)

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Parotid basal cell adenoma with atypia: Navigating the diagnostic gray zone, case report and a brief review of the literature

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Abstract

Basal cell adenoma (BCA) is an uncommon benign salivary gland neoplasm that most often arises in the parotid gland and is usually managed successfully by complete surgical excision. Its diagnostic importance lies in its morphologic overlap with malignant basaloid tumors, particularly basal cell adenocarcinoma and adenoid cystic carcinoma, in which invasion and perineural spread carry major prognostic and therapeutic implications.

We present a 58-year-old female with a slowly enlarging, painless right preauricular mass and no facial nerve dysfunction. Imaging demonstrated a sharply circumscribed superficial parotid lesion, and fine needle aspiration favored a benign basaloid neoplasm. She underwent right superficial parotidectomy with facial nerve preservation. Permanent sections showed a well-circumscribed, partially encapsulated solid and trabecular basaloid tumor with focal peripheral cribriform architecture. The tumor showed rare mitoses, clear margins, preserved myoepithelial differentiation, and no unequivocal perineural, lymphovascular, or infiltrative growth. The Ki-67 index was approximately 8%, supporting a benign process despite architectural atypia. After multidisciplinary review, the final diagnosis was BCA with atypia.

This case illustrates the diagnostic gray zone created by focal cribriform change and reinforces the notion that invasion, rather than architecture alone, remains the decisive criterion guiding conservative postoperative management.

Keywords: Basal cell adenoma; Architectural atypia; Fine needle aspiration; Immunohistochemistry; Basaloid neoplasm; Benign salivary gland tumor; β-catenin.

1. Introduction

BCA is a rare benign epithelial neoplasm of the salivary glands, accounting for approximately 1-3% of all salivary gland tumors, most commonly arising in the parotid gland. [1] It is characterized by a well-circumscribed, often encapsulated, proliferation of basaloid cells [1,2] Clinically, the tumors are slow-growing, painless masses, and their prognosis following surgical excision has been favorable. [3] Although benign, BCA can be challenging to diagnose when its histopathologic features resemble those of malignant basaloid neoplasms, such as basal cell adenocarcinoma and adenoid cystic carcinoma. [4] Features such as a focal cribriform pattern or overlapping immunohistochemical (IHC)

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profiles can raise the concern of malignancy. However, without definitive invasive manifestations such as perineural invasion, lymphovascular invasion, or infiltrative growth, proper classification is difficult. [1]

This borderline presentation falls within a diagnostic gray area not clearly defined by current classifications, potentially impacting clinical decisions. We report this case to illustrate the diagnostic hurdles involved in identifying a benign basal cell adenoma with atypical features and to underscore the need for a multidisciplinary approach to distinguishing benign from malignant tumors of basaloid salivary glands.

2. Case Presentation

2.1. Clinical Presentation and Initial Investigations

A 58-year-old woman with no significant past medical history presented to the head and neck surgery clinic with a six-month history of a slowly enlarging, painless swelling in the right preauricular region. She denied any facial weakness, trismus, skin changes, dysphagia, or constitutional symptoms such as fever, night sweats, or unintentional weight loss. She had no prior history of salivary gland disease, radiation exposure to the head and neck, or family history of salivary gland neoplasms.

On physical examination, she appeared well and in no acute distress. Vital signs were within normal limits. Inspection of the face revealed a subtle fullness in the right preauricular area without overlying skin changes, erythema, or fixation to the skin. Palpation demonstrated a firm, well-circumscribed, non-tender, predominantly superficial, and mostly mobile mass measuring approximately 2.5 × 2.0 cm in the right parotid region. There was no palpable regional lymphadenopathy in the cervical or submandibular chains. Cranial nerve VII was intact with symmetric facial movement bilaterally. Oral cavity and oropharyngeal examinations were unremarkable.

Laboratory investigations were obtained and returned within normal limits, including a complete blood count, comprehensive metabolic panel, and thyroid function studies. Inflammatory markers were not elevated.

2.2. Radiological Studies and Fine Needle Aspiration Cytology Findings

Ultrasound of the right parotid gland demonstrated a well-defined, hypoechoic, homogeneous ovoid lesion within the superficial lobe, measuring 2.4 × 1.9 × 1.8 cm, with no internal vascularity on Doppler imaging, no posterior acoustic shadowing, and no evidence of invasion into surrounding structures. Magnetic resonance imaging (MRI) with contrast confirmed a sharply circumscribed lesion with intermediate T1 and high T2 signal intensity, demonstrating avid but homogeneous enhancement without perineural spread, extraglandular extension, or suspicious lymph nodes. The imaging characteristics were most consistent with a benign, mostly encapsulated parotid neoplasm.

Fine needle aspiration (FNA) cytology was performed. The smears were cellular and demonstrated cohesive clusters and sheets of uniform basaloid cells with scant cytoplasm, round to oval nuclei with finely granular chromatin, and inconspicuous nucleoli. Myoepithelial cells with spindled morphology were noted in the background alongside stromal fragments. Mitoses were absent. Although the smears were highly cellular, with focal groups of overlapping cells, only mild cytological atypia was noted, and no evidence of malignancy was found. **(**Figure 1 A, B**)** The cytologic differential diagnosis favored a benign basaloid neoplasm, most consistent with basal cell adenoma, with pleomorphic adenoma as a secondary consideration.

2.3. Multidisciplinary Tumor Board Discussion and Management

Given clinical, radiologic, and cytologic concordance, the case was discussed at the multidisciplinary head and neck tumor board. Attendees included otolaryngology, head and neck surgery, pathology, radiology, medical oncology, and radiation oncology. The consensus recommendation was to proceed with right superficial parotidectomy with facial nerve preservation, with final definitive diagnosis deferred to permanent histopathology.

The patient underwent an uncomplicated right superficial parotidectomy. The excised mass was a well-encapsulated, tan-gray nodule with a smooth external surface and a glistening cut surface, devoid of necrosis or hemorrhage. Frozen section consultation was requested intraoperatively and reported a basaloid neoplasm with equivocal features; definitive subtyping was deferred to permanent sections.

Histopathologic examination of permanent sections revealed a well-circumscribed, partially encapsulated basaloid neoplasm with a predominantly solid and trabecular growth pattern. The tumor cells were uniform with scant cytoplasm and oval nuclei; however, focal areas at the periphery of the tumor demonstrated a cribriform architectural

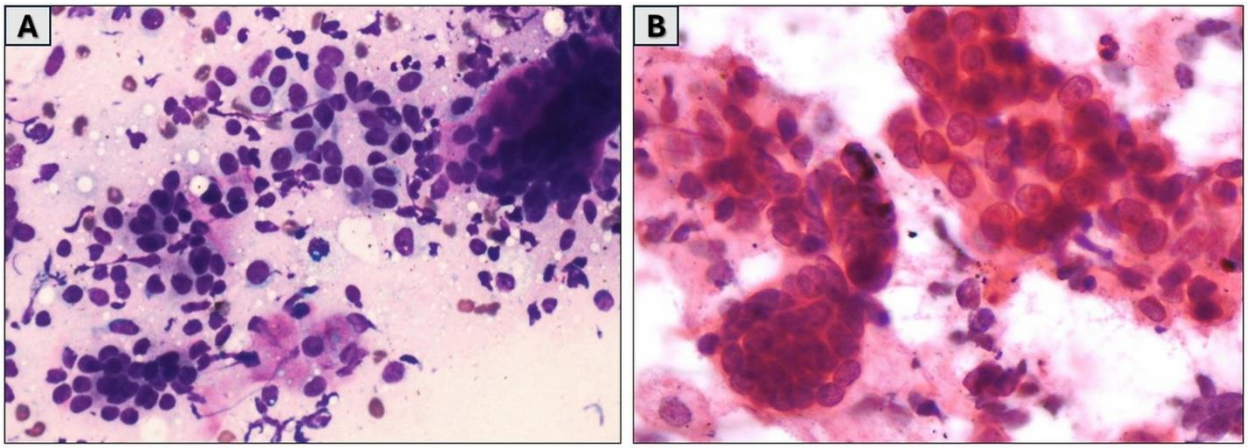
pattern with pseudocystic spaces reminiscent of adenoid cystic carcinoma. Mitotic figures were rare, and no atypical mitoses were identified. Critically, no unequivocal perineural invasion, lymphovascular invasion, or infiltrative growth into the surrounding parotid parenchyma was identified, and the surgical margins were clear. (Figure 2 A, B, C) An IHC panel was performed to characterize the lesion further. The tumor cells were diffusely positive for cytokeratin 7, p63, and CD117 (c-Kit). The Ki-67 proliferative index was approximately 8%. Calponin and smooth muscle action highlighted the peripheral myoepithelial layer. Notably, the Ki-67 index and absence of invasion favored a benign process despite the focal cribriform architecture.

The case was again presented to the tumor board for discussion after the postoperative pathology evaluation. The pathology team acknowledged the diagnostic ambiguity introduced by the peripheral cribriform architecture, which prompted consideration of basal cell adenocarcinoma or, less likely, early adenoid cystic carcinoma. However, given the complete absence of invasion, a low proliferative index, an intact peripheral myoepithelial layer, and the well-circumscribed nature of the mass, a consensus diagnosis of parotid basal cell adenoma with atypia was reached. This designation was recorded as a non-WHO-classified diagnostic category reflecting an atypical benign neoplasm with focal architectural complexity, but without the defining criterion of invasion required for a diagnosis of basal cell adenocarcinoma. The final pathologic staging was therefore consistent with a benign neoplasm.

Management following the tumor board consensus was conservative. Given the negative surgical margins and absence of invasive features, no adjuvant radiation or systemic therapy was indicated. The patient was counseled extensively regarding the atypical histologic findings and the theoretical, though unquantified, risk of local recurrence given the borderline architectural features.

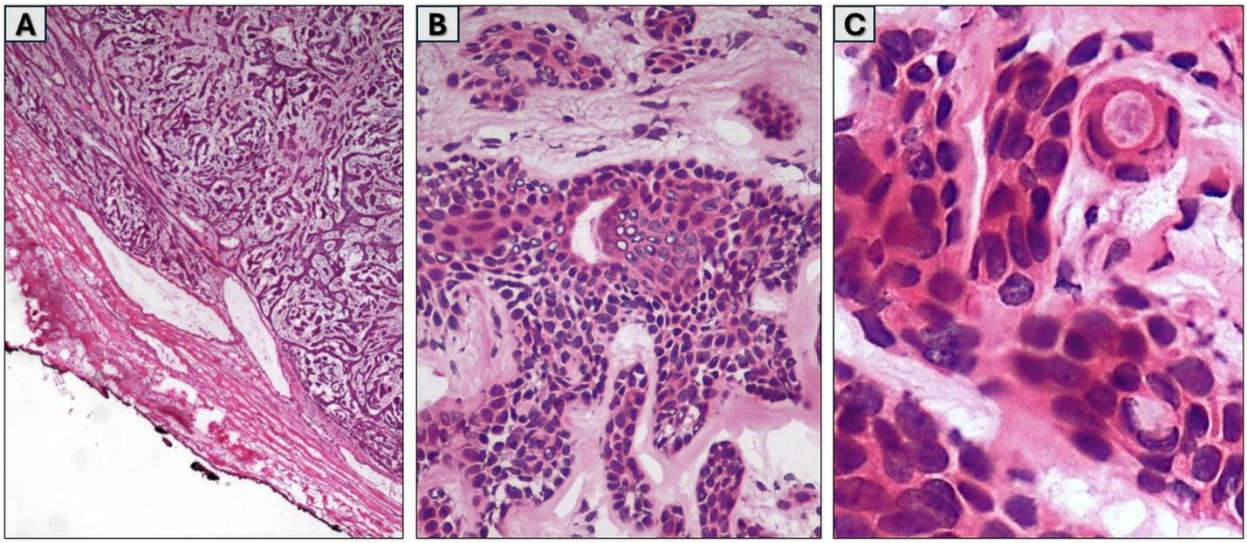
2.4. Outcome and Follow-Up

At her three-month postoperative visit, the patient had healed well without facial nerve deficit, Frey's syndrome, or palpable recurrence. She was enrolled in a structured surveillance program consisting of clinical examination every six months for the first two years, followed by annual follow-up visits for a minimum of five years, with cross-sectional imaging reserved for any clinical suspicion of recurrence. She was advised to promptly report any new swelling, pain, or facial nerve symptoms.



1A: Intermediate power view showing cohesive clusters and sheets of uniform basaloid cells with scant cytoplasm and scattered myoepithelial cells with spindled morphology (DQ stain X40).; **1B:** High power view showing cohesive clusters of overlapping uniform basaloid cells with scant cytoplasm, round to oval nuclei with fine granular chromatin, and inconspicuous nucleoli. Only mild cytological atypia was noted, and there is no evidence of malignancy (H&E stain X60).

Figure 1 FNA Cytology microscopic features of the parotid basal cell adenoma



2A: Low power view showing a well-circumscribed, partially encapsulated basaloid neoplasm (H&E stain X20).; **2B:** Intermediate power view showing solid and trabecular patterns (H&E stain X40).; **2C:** High power view showing uniform cells with scant cytoplasm, oval nuclei, and mild nuclear atypia (H&E stain X60).

Figure 2 Microscopic findings of the excised atypical parotid basal cell adenoma

3. Discussion

3.1. Background (History, epidemiology, risk factors, and WHO classification):

BCA develops from glandular epithelial cells and has a particular predilection for the parotid gland, especially its superficial lobe. This tumor was ultimately recognized as a distinct clinicopathologic entity following the advancement of histological classification by Kleinsasser and Klein in 1967. [5] Based on definitions from the latest World Health Organization (WHO) Classification of Head and Neck Tumors, 5th edition, BCA is a monomorphic basaloid tumor with a uniform cellular structure, well-circumscribed growth characteristics, and no invasive features, with no myxochondroid stromal structures, as seen in pleomorphic adenomas. [6]

Contrary to its low incidence, BCA is the third most common benign salivary gland tumor after pleomorphic adenoma and Warthin's tumor. Epidemiologically, BCA predominantly occurs in adults aged 50–70 years, with modest female predominance. [7] Most cases are sporadic, and associations with syndromes are rare; we still cannot identify the precise environmental or occupational risk factors. [8] Molecular studies have shown alterations in the Wnt/β-catenin signaling pathway, particularly CTNNB1, suggesting a possible role in tumorigenesis. [9] Even though it is benign, certain variants, such as those displaying architectural complexity or a characteristic abnormality, can be very difficult to diagnose. The convergent morphological features of malignant basaloid neoplasms, such as basal cell adenocarcinoma and adenoid cystic carcinoma, can make accurate identification difficult. A thorough analysis of histopathology, IHC, and detailed clinical information is necessary. [10] Such borderline presentations emphasize the importance of an exact diagnosis, as management and prognosis vary widely between benign and malignant salivary gland tumors. [6]

BCA most commonly occurs in the parotid gland or minor salivary glands, but can develop in atypical sites, including the lips, buccal mucosa, palate, and nasal septum. [11] Clinically, BCA presents as an unremarkable, slow-growing soft mass, often mobile, with no associated facial nerve damage or regional lymphadenopathy. [3] The histological hallmark of BCA is monomorphic basaloid epithelial cell proliferation with intact basal cell layers, but also peripheral myoepithelial differentiation. [4] Importantly, BCA lacks infiltrative growth patterns, perineural invasion, and significant cytological atypia, all features that indicate malignant transformation and thus can be distinguished from basal cell adenocarcinoma. [3,10]

Because the outcomes of surgical excision for BCA cases are generally good post-surgery, with remarkably low recurrence rates, compared to basal cell adenocarcinoma or adenoid cystic carcinoma, it is therefore crucial that a well-defined differential diagnosis be performed. Membranous subtypes have a 24% recurrence rate and are more likely to be multicentric than other forms. Although very rare overall, the membranous variant is associated with a higher rate of malignancy transformation than other subtypes. [8]

3.2. Pathogenesis, Pathophysiology

Tumor growth of BCA can occur in four patterns: solid, trabecular, tubular, and membranous. [12] Solid growth is similar to skin BCC, with nests of basaloid cells surrounded by epithelial cells. Trabecular is cord-like, while small duct lumens lined by eosinophiles characterize tubular. The membranous type is unique among the others in that it is unencapsulated with multiple nests of basaloid cells. [8] It is important to note that a tubular/trabecular combination variant is often seen and relatively common. [12]

The pathogenesis of BCA is poorly understood due to its rarity; BCA accounts for 1-3% of salivary tumors. Sato et al. have investigated the role of beta-catenin in tumorigenesis and pathogenesis. One important protein in the intricate signaling network known as the Wnt signaling pathway is β-catenin. β-catenin levels rise when the Wnt pathway is active, which raises transcription factor activity and is essential for healthy tissue formation and growth. However, when pathologically elevated β-catenin, caused by mutations in CTNNB1 (β-catenin), APC, or Axin, occurs, this growth pathway can become oncogenic. Aberrations in the Wnt pathway have been identified as a major mutational contributor to BCA. In BCAs, CTNNB1 mutations account for the majority of cases, with APC and AXIN mutations representing a minority. The authors concluded that β-catenin mutations can be used to differentiate BCA from other morphologically similar benign tumors because this mutation is more unique to BCA. [13]

Wilson et al. used next-generation sequencing (NGS) to characterize additional mutations in BCA and identified an ATM mutation in one case. The ATM gene encodes a protein that functions in DNA strand repair, which is most widely known for the mutation associated with ataxia telangiectasia. [14]

Yagai et al. found that nuclear β-catenin expression differed significantly between BCAC and BCA, but CTNNB1 expression was similar. This leads the authors to believe that both tumors share a common growth pathway. This can help support that BCAC is a malignant counterpart of BCA. This also shows that CTNNB1 wild-type tumors with β-catenin expression are characteristic of BCA rather than BCAC, indicating that their pathology is distinct from BCAC and may reflect a more benign or noninvasive disease course. [15] An example of such a mutation is the CYLD mutation in some BCA tumors, which is believed to stabilize β-catenin expression in the Wnt pathway. This mutation is characteristic of Brooke-Speigler syndrome, which includes multiple cylindromas, spiradenomas, and membranous BCA of the parotid gland. They also found that nuclear β- catenin-positive tumors were more likely to exhibit features associated with BCA/BCAC, such as peripheral palisading and S-100-positive nuclei, than β-catenin-negative tumors. This led the authors to suspect that B-catenin-negative tumors may be morphological mimics of BCA and other salivary tumors, as similarities between tumor types make differentiation difficult. [15]

3.3. Comparative Analysis of Our Case with Existing Literature. (Clinical, radiology, pathology, Lab, diagnosis, management, and outcome)

The clinical profile of this case closely paralleled the usual presentation of basal cell adenoma described in the literature. The patient was a 58-year-old female with a firm, mobile, painless parotid mass, matching the reported predilection for older adults, female patients, and superficial parotid involvement. [8] The radiologic impression of a well-circumscribed benign parotid neoplasm and the cytologic finding of cohesive basaloid cells were also consistent with previously described basal cell adenomas, which often appear as slow-growing encapsulated tumors and may be difficult to subtype preoperatively. [8] The main difference was the focal peripheral cribriform architecture and mild atypia.

The existing literature emphasizes that basal cell adenocarcinoma may resemble basal cell adenoma but is characterized by infiltrative growth into adjacent parotid tissue, fat, muscle, skin, or neurovascular spaces. [16,18] Similarly, adenoid cystic carcinoma may show basaloid cells, cribriform spaces, CD117 expression, and myoepithelial markers, but it is characteristically malignant and frequently demonstrates perineural invasion. [17] In contrast, the present tumor lacked infiltrative margins, perineural invasion, lymphovascular invasion, necrosis, and high proliferative activity. This distinction aligned with studies showing that cribriform patterns and limited capsular complexity may occur in basal cell neoplasms without necessarily indicating aggressive behavior. [18] Therefore, the case reinforced the literature while extending it by documenting a practical multidisciplinary approach to an atypical but noninvasive basal cell adenoma.

4. What Have We Learned from This Case?

This case demonstrates that a parotid basaloid neoplasm should not be classified as malignant solely because it contained focal cribriform architecture or focal cytologic atypia. The most important diagnostic lesson was that the pathologist and treating team had to search deliberately for invasion, perineural spread, lymphovascular invasion,

necrosis, atypical mitoses, and a high proliferative index before escalating the diagnosis to basal cell adenocarcinoma or adenoid cystic carcinoma. [16,17,18]

For clinicians, the principal red flags remained rapid growth, pain, facial nerve weakness, fixation to skin or deep tissues, suspicious lymphadenopathy, and radiologic evidence of extra-parenchymal or perineural extension. Their absence in this patient supported a planned superficial parotidectomy rather than radical treatment. However, the atypical histology justified careful counseling and structured surveillance. In future practice, this case reinforces the value of correlating clinical behavior, imaging, cytology, permanent histology, IHC, and multidisciplinary review before assigning malignant significance to borderline architectural features.

Abbreviations

- Basal cell adenoma (BCA);
- Basal cell adenocarcinoma (BCAC);
- Fine needle aspiration (FNA);
- Immunohistochemistry (IHC)

5. Conclusion

We report this case as it captures a diagnostically important but underrecognized gray zone in salivary gland pathology: a parotid basal cell adenoma with focal atypical cribriform architecture but without the defining features of malignancy. The case added value by showing that focal architectural complexity, CD117 positivity, and basaloid cytology could mimic adenoid cystic carcinoma or basal cell adenocarcinoma, yet still represent a benign neoplasm when invasion, perineural involvement, lymphovascular invasion, necrosis, and high proliferative activity were absent.

For the medical community, the practical message is that conservative management after complete excision may be appropriate when an atypical basal cell adenoma is rigorously evaluated, and margins are clear. At the same time, the case supports transparent patient counseling and longer clinical follow-up, because borderline histologic features may create uncertainty even when formal malignant criteria are not met. This report, therefore, provides a clinically useful example of restraint, precision, and multidisciplinary decision-making in parotid basaloid tumors.

Compliance with ethical standards

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All authors make the following declarations:

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Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

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