

Basal Cell Adenoma of the Parotid Gland: A Case Report with Literature Review

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Abstract

Case Report

Basal cell adenoma (BCA) is a rare benign epithelial tumor of the salivary glands, accounting for 1 to 3% of all parotid tumors. We report the case of a 65-year-old woman with no significant medical history, presenting with a slowly growing right parotid swelling that had been evolving for one year. Ultrasound revealed a well-defined hypoechoic homogeneous nodule measuring 10 x 15 mm with peripheral Doppler vascularization. MRI showed an encapsulated nodule of the superficial lobe of the right parotid gland, with intermediate heterogeneous T2 signal, progressive gadolinium enhancement of type A, and no diffusion restriction, measuring 10 x 12 x 8 mm. The patient underwent superficial parotidectomy with facial nerve preservation and clear resection margins. Histopathological examination revealed an encapsulated tubulo-trabecular proliferation with round to oval basaloid cells, eosinophilic cytoplasm, fine chromatin, rare mitoses, and no necrosis. Immunohistochemistry showed positivity for p63 and LEF1 in basal cells, CD117 in luminal cells, focal beta-catenin expression, and negativity for mammaglobin. The postoperative course was marked by a transient buccal branch paresis, which resolved within three months with physiotherapy. At two years of follow-up, no recurrence was observed.

Keywords: Basal cell adenoma; parotid gland; benign salivary tumor; parotidectomy; immunohistochemistry; LEF1.

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INTRODUCTION

Salivary gland tumors represent a heterogeneous neoplastic group with one of the most complex histological classifications in tumor pathology [1]. Among benign epithelial tumors, pleomorphic adenoma is by far the most frequent, followed by Warthin tumor [2]. Basal cell adenoma (BCA) is a distinct and rare entity, accounting for 1 to 3% of all salivary gland tumors and approximately 5 to 6% of benign parotid tumors [3].

Initially classified among monomorphic adenomas, BCA was individualized as an autonomous entity by Gardner and Daley in 1983 [6], and subsequently recognized by the WHO classification of salivary gland tumors in 1991 [7]. It is distinguished from pleomorphic adenoma by the absence of myxochondroid stroma and chondroid differentiation, and from basal cell carcinoma by the absence of malignancy criteria [1].

We report a case of basal cell adenoma of the parotid gland and discuss the epidemiological, clinical, radiological, histological, and therapeutic aspects of this rare entity in light of the current literature.

CASE REPORT

A 65-year-old woman with no significant medical history presented with a progressive and painless right parotid swelling that had been slowly growing for approximately one year. There was no history of cervicofacial trauma or exposure to known risk factors.

Physical examination revealed a mass in the right parotid region, approximately 2 cm in its greatest dimension, firm in consistency, painless, and mobile. The overlying skin was normal, without erythema or ulceration. There were no peripheral facial nerve palsy and no palpable cervical lymphadenopathy. The patient was in good general condition (Figure 1).



Figure 1: Frontal view: swelling of the right parotid region (arrow)

Parotid ultrasound demonstrated a well-defined hypoechoic homogeneous nodule measuring 10 x 15 mm, with regular contours and peripheral color Doppler vascularization without central flow, suggestive of a benign encapsulated lesion.

MRI of the salivary glands revealed a rounded, well-defined nodule of the superficial lobe of the right

parotid gland, surrounded by a hypointense T1 capsule. The lesion showed intermediate heterogeneous T2 signal, progressive gadolinium enhancement with a type A curve, and no diffusion restriction. It measured 10 x 12 x 8 mm (transverse x anteroposterior x height). There was no extension to the deep lobe and no suspicious relationship with the facial nerve (Figure 2).



Figure 2: MRI, axial T1 sequence: encapsulated nodule of the superficial lobe of the right parotid gland (arrow)

The patient underwent superficial parotidectomy under general anesthesia. Dissection and preservation of the facial nerve were performed without intraoperative neuromonitoring. The surgical specimen consisted of a tissue fragment measuring 2.4 x 2.5 cm containing a well-defined encapsulated nodule.

Histopathological examination revealed, within the parotid tissue, an encapsulated and well-defined tumor proliferation arranged in tubular structures occasionally containing secretions, and in nests. Tumor cells were round to oval, with fine chromatin, occasionally nucleolated, and with abundant

eosinophilic cytoplasm. Rare mitoses were noted. No tumor necrosis was identified.

Immunohistochemical study showed nuclear positivity for p63 and LEF1 in basal cells, positivity for CD117 in luminal cells, and focal expression of beta-catenin in basal cells. Mammaglobin was negative. This immunohistochemical profile was consistent with basal cell adenoma of tubulo-trabecular type.

Resection margins were clear. The postoperative course was marked by a transient paresis of the buccal branch of the facial nerve, which resolved

within three months under facial physiotherapy. No salivary fistula or Frey syndrome was observed.

The postoperative follow-up is now two years, with no evidence of local recurrence on clinical and ultrasound surveillance.

DISCUSSION

Basal cell adenoma is a rare benign epithelial tumor of the salivary glands, first individualized as an autonomous entity distinct from other monomorphic adenomas by Gardner and Daley in 1983 [6], and subsequently recognized by the WHO classification in 1991 [7]. It represents approximately 1 to 3% of all salivary gland tumors, with a predominance in the parotid gland [1]. It occurs most frequently in women, between the fifth and seventh decades of life [10], which is consistent with the characteristics of our patient.

Clinically, BCA typically presents as a slowly growing, painless, well-defined, and mobile parotid swelling [1]. The absence of facial palsy, cervical lymphadenopathy, and skin changes is characteristic of the benign nature of the lesion. In our case, the one-year duration of evolution prior to consultation illustrates the slow and insidious growth pattern of this tumor, as previously reported [10].

MRI of the salivary glands is a key examination for pre-operative assessment of parotid tumors. The MRI profile of BCA is characterized by well-defined margins with a hypointense capsule, intermediate T2 signal, and variable enhancement patterns on dynamic study [11]. In our case, the intermediate heterogeneous T2 signal and type A progressive enhancement are consistent with BCA and differ from the typically high and homogeneous T2 signal of pleomorphic adenoma, in which the myxoid component accounts for the marked T2 hyperintensity [11].

The definitive diagnosis relies on histopathological examination. Histologically, BCA is characterized by a proliferation of basaloid cells organized in tubular, trabecular, solid, or membranous patterns, within an encapsulated and well-defined architecture [1,4]. In our case, the tubulo-trabecular pattern, round to oval cells with fine chromatin, abundant eosinophilic cytoplasm, absence of necrosis, and rare mitoses are in accordance with the WHO diagnostic criteria [4].

Immunohistochemistry plays an important role in confirming the diagnosis and in the differential diagnosis. Positivity for p63 and LEF1 in basal cells is characteristic of BCA, and the negativity for mammaglobin helps exclude other salivary gland tumors [8,13]. LEF1, a transcription factor of the Wnt/beta-catenin signaling pathway, is now recognized as a highly specific marker of BCA among salivary gland tumors [13]. Positivity for CD117 in luminal cells and focal

beta-catenin expression in basal cells are also consistently reported [8,14].

The main differential diagnoses of BCA include pleomorphic adenoma, canalicular adenoma, and basal cell carcinoma. Canalicular adenoma, although morphologically similar, differs by its exclusively minor salivary gland location and specific architectural features [2,5]. Basal cell carcinoma, the malignant counterpart of BCA, is distinguished by the presence of perineural invasion, vascular invasion, and necrosis [4,14]. The distinction from pleomorphic adenoma relies on the absence of myxochondroid stroma and negativity for GFAP and S100 in BCA [1,6].

The standard treatment for BCA is superficial parotidectomy with facial nerve preservation when the tumor is located in the superficial lobe, as performed in our case [15]. Obtaining clear resection margins is critical to prevent recurrence, the rate of which is estimated between 25 and 37% after incomplete excision, compared to less than 2% after complete resection [16]. In our observation, margins were clear and no recurrence was observed at two years of follow-up.

The transient buccal branch paresis observed postoperatively is a known complication of parotidectomy, related to intraoperative perineural manipulation, and its spontaneous resolution within a few weeks to a few months, facilitated by physiotherapy, is usual [15]. The risk of malignant transformation of BCA into basal cell carcinoma is low but documented, justifying regular long-term clinical and ultrasound surveillance [9,16].

CONCLUSION

Basal cell adenoma of the parotid gland is a rare benign tumor whose diagnosis relies on a combination of clinical, radiological, and above all histological and immunohistochemical findings. The IHC profile associating positivity for LEF1, p63, and CD117 is characteristic and allows differentiation from its main differential diagnoses. Superficial parotidectomy with facial nerve preservation and clear margins is the standard of care, offering excellent prognosis and low recurrence risk. Long-term surveillance nonetheless remains necessary given the rare but documented risk of malignant transformation.

Conflicts of Interest

The authors declare no conflict of interest.

All authors have read and approved the final version of the manuscript.

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