

Recurrent meningioma mimicking cerebellopontine angle schwannoma: A case report of diagnostic and therapeutic challenges

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Abstract

Meningiomas of the cerebellopontine angle (CPA) are the second most common tumors in this region. However, their clinical and radiological presentations can occasionally mimic those of vestibular schwannomas, leading to significant diagnostic ambiguity. We present the case of a 61-year-old female who developed progressive right-sided hearing loss, intermittent vertigo, and facial twitching six years after the gross total resection of a CPA tumor initially diagnosed as a vestibular schwannoma. Current magnetic resonance imaging (MRI) revealed a recurrent enhancing mass extending into the internal auditory canal, with a subtle dural tail and adjacent hyperostosis.

Multidisciplinary review and subsequent stereotactic biopsy of the recurrent lesion, alongside re-evaluation of the original pathology, confirmed a World Health Organization (WHO) Grade I meningothelial meningioma. Immunohistochemistry (IHC) demonstrated strong positivity for somatostatin receptor 2A (SSTR2A) and epithelial membrane antigen (EMA), while next-generation sequencing identified a TRAF7 mutation in the absence of neurofibromatosis type 2 alterations. The patient was successfully managed with a subtotal resection to preserve facial nerve function, followed by stereotactic radiosurgery for residual disease, achieving stable tumor control at three years. This case underscores the critical importance of comprehensive IHC and molecular profiling in differentiating CPA tumors, highlighting how initial misdiagnosis can alter long-term surveillance and therapeutic strategies.

Keywords: Meningioma; Schwannoma; Cerebellopontine Angle Tumors; Recurrent; Misdiagnosis; Somatostatin Receptor 2A; Dural Tail Sign; TRAF7 Mutation

1. Introduction

Tumors of the cerebellopontine angle (CPA) pose unique diagnostic and surgical challenges due to their intimate proximity to critical neurovascular structures, including the brainstem and cranial nerves. Vestibular schwannomas are by far the most frequent neoplasms in this anatomical space, accounting for approximately 80% of cases. In contrast, meningiomas are the second most common entity, accounting for 5% to 10% of CPA lesions. [1,2] While typical imaging features, such as the dural tail sign and adjacent bony hyperostosis, often distinguish meningiomas from schwannomas, a subset of CPA meningiomas exhibits atypical presentations. Specifically, when a meningioma extends into or primarily occupies the internal auditory canal, it may closely mimic the clinical and radiological profile of a vestibular schwannoma. [3,4,5] This diagnostic overlap is particularly important because the surgical approaches, expected functional outcomes, and long-term recurrence risks differ substantially between the two pathologies. Reliance solely

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on histomorphology without robust IHC or molecular confirmation can occasionally lead to misdiagnosis, altering the trajectory of patient care. [5,6]

We report a rare case of a recurrent CPA meningioma that was initially misdiagnosed as vestibular schwannoma. This report aims to illustrate the diagnostic pitfalls associated with CPA tumors and to emphasize the indispensable role of modern molecular diagnostics, such as TRAF7 mutational analysis and somatostatin receptor 2A IHC, in ensuring accurate classification and optimal multidisciplinary management.

2. Case presentation

2.1. Clinical presentation

A 61-year-old female presented with a six-month history of progressive right-sided hearing loss, intermittent vertigo, and a dull retroauricular pressure sensation. She denied headaches or focal weakness. Six years earlier, she had undergone gross total resection of a right CPA tumor diagnosed at that time as a vestibular schwannoma based solely on histomorphology. Postoperatively, she had done well with only mild residual ipsilateral high-frequency hearing loss and no adjuvant therapy. She sought medical attention now because the hearing loss worsened to the point of functional disability, and she developed new, brief episodes of right facial twitching, which raised concern for tumor recurrence or an alternative diagnosis.

2.2. Physical examination, history, radiology, and differential diagnosis

Physical examination revealed absent right corneal reflex, mild right lower motor neuron facial weakness, and sensorineural hearing loss on the right with normal left-sided hearing. The Weber test was lateralized to the left, and the Rinne test was positive bilaterally. No nystagmus or gait ataxia was noted. Her past medical history was otherwise unremarkable, with no neurofibromatosis type 2 features or family history of brain tumors.

Laboratory studies, including complete blood count and basic metabolic panel, were within normal limits. MRI with contrast demonstrated a 2.2 cm, homogeneously enhancing, dural-based mass in the right cerebellopontine angle, extending into the internal auditory canal, without significant widening of the canal. Unlike a typical vestibular schwannoma, the lesion showed a broad dural attachment along the petrous ridge and a subtle "dural tail" sign. CT imaging revealed mild hyperostosis of the adjacent petrous bone.

The clinical and radiological differential diagnosis included recurrent vestibular schwannoma, meningothelial meningioma Grade I, and, less likely, an atypical meningioma or metastatic disease. The imaging features of dural tail and hyperostosis favored meningioma over schwannoma, though overlap remained significant given the prior schwannoma diagnosis.

2.3. Multidisciplinary tumor board discussion and management decisions

The tumor board reviewed the original surgical pathology slides from six years prior, which immediately raised suspicion for a prior misdiagnosis. Histopathologic examination revealed vague whorling and psammoma bodies, findings that are atypical of vestibular schwannoma and more suggestive of meningioma. The discussion centered on whether the original tumor was actually a meningioma initially misdiagnosed, now presenting with true recurrence, versus a second primary or radiation-induced tumor. Key radiology findings, dural tail and hyperostosis, were highlighted as atypical for schwannoma.

The team agreed that confirmatory IHC and molecular studies on both the original and recurrent tumors were necessary before proceeding with intervention. Given the patient's current symptoms, the board recommended a planned subtotal resection of the recurrent mass to preserve facial nerve function, followed by stereotactic radiosurgery for residual tumor, but only after pathological confirmation. The decision to defer immediate surgery was based on the indolent nature of a Grade I meningioma and the absence of rapid neurological decline.

2.4. Special studies, pathology, and final diagnosis

The tumor board recommended a stereotactic needle biopsy of the recurrent mass to obtain pathological confirmation before any definitive intervention. The biopsy was performed via a retrosigmoid approach under frameless stereotactic guidance, yielding small tissue cores. Histology showed meningothelial cells forming whorls with psammoma bodies,

and IHC revealed strong positivity for EMA, progesterone receptor, and SSTR2A, with negative S100 and SOX10, confirming meningothelial meningioma Grade I. The original paraffin block from six years earlier was also re-evaluated and showed identical whorling and psammoma bodies, with repeat immunohistochemistry confirming the same findings. The diagnosis of schwannoma of the prior tumor was corrected to meningioma. (Figure 1 A, B, C)

Following this confirmation, the patient underwent a planned subtotal resection via a retrosigmoid approach for symptom relief and tumor debulking.

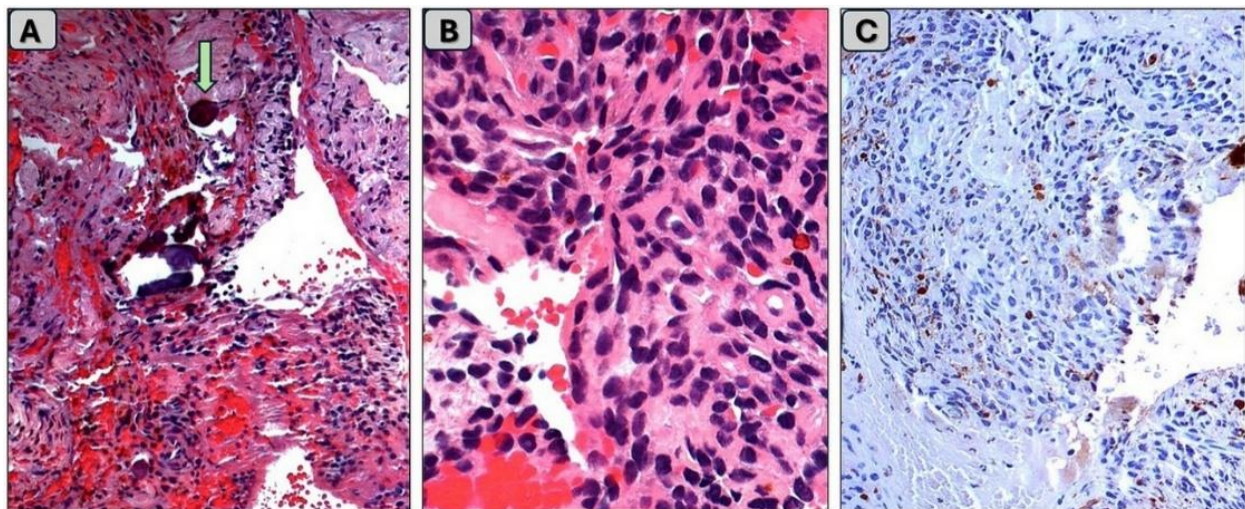
The resected specimen showed histomorphology similar to that of the original tumor, with uniform meningothelial whorls, scattered psammoma bodies, rare mitoses ($<1/10$ HPF), and no necrosis. Immunohistochemistry confirmed the same profile (EMA+, PR+, SSTR2A+, S100-, SOX10-), and the Ki-67 proliferation index was 2%. (Figure 2 A, B, C) Next-generation sequencing of both tumors identified the same mutations, including TRAF7 (c.1505G>A), with no NF2 alterations, consistent with WHO Grade I meningothelial meningioma. No TERT promoter mutation or CDKN2A deletion was detected. SSTR2A positivity was particularly useful in retrospectively confirming the original diagnosis. Comparative genomic hybridization showed no chromosomal copy-number differences between the two specimens, confirming the recurrence of the same tumor rather than a new primary lesion.

In the absence of any features suggestive of schwannoma, the diagnosis of mixed schwannoma/meningioma was not considered.

2.5. Management, outcome, and follow-up at three years

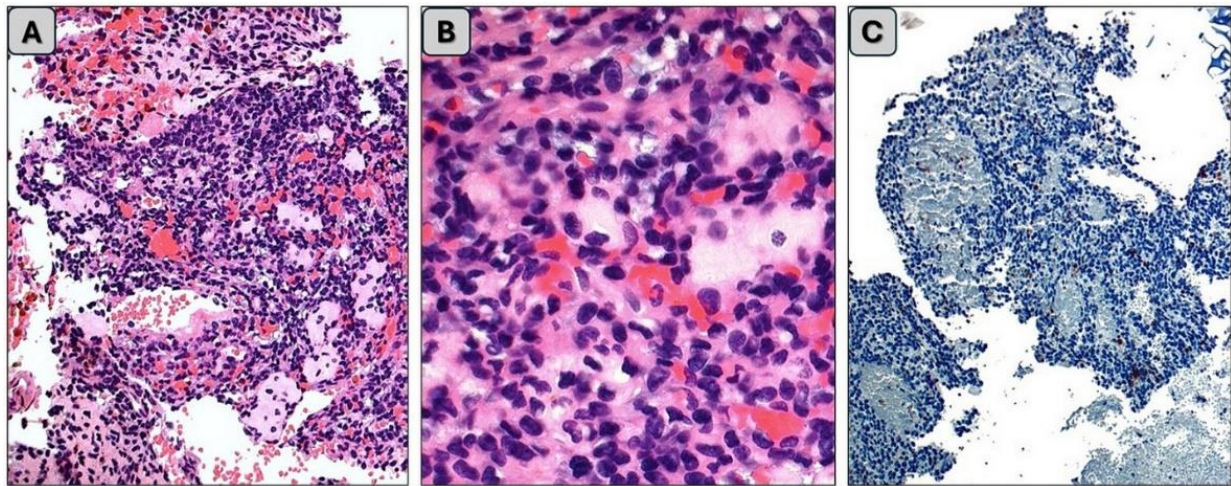
Following confirmation of recurrent meningothelial meningioma Grade I, the patient underwent subtotal resection of the recurrent mass with preservation of the facial nerve. Residual tumor along the cavernous sinus measured 8 mm.

Two months postoperatively, she received fractionated stereotactic radiosurgery (25 Gy in 5 fractions) for the residual disease. At three years, MRI showed a stable residual tumor without regrowth. Her facial weakness improved to House-Brackmann I, but the right hearing loss remained profound and was managed with a contralateral routing of sound hearing aid. She remained seizure-free and neurologically stable, with an annual surveillance MRI planned indefinitely.



1A: Low-power view showing meningothelial cells forming whorls with psammoma bodies (green arrow) and haemorrhage (H&E, X20).; 1B: High-power view showing whorl formation by meningothelial cells (H&E X40).; 1C: Tumor cells negative for S100

Figure 1 Microscopic features of the initial tumor six years earlier (Misdiagnosed as schwannoma)



2A: Low-power view showing features similar to the prior tumor (H&E X20); 2B: High-power view showing uniform meningothelial whorls, rare mitoses, and no necrosis (H&E X40); 2C: Tumor cells negative for S100

Figure 2 Microscopic features of the recurrent tumor (Diagnosed as Meningioma)

3. Discussion

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

Meningiomas are the most common primary intracranial tumors, traditionally thought to arise from arachnoid cap cells of the meninges. [7] They exhibit a strong female predominance and a peak incidence in the sixth and seventh decades of life. According to the 2021 WHO Classification of Tumors of the Central Nervous System, meningiomas are stratified into three grades based on histopathological criteria and, increasingly, molecular profiles. [9] While the majority are benign Grade I lesions, the recurrence rate remains a clinical concern, particularly for tumors in complex skull base locations like the CPA, where gross total resection is often precluded by the need to preserve cranial nerve function. [7,8]

Recent advances in molecular pathology have revolutionized the understanding of meningioma oncogenesis. While cerebral convexity meningiomas frequently harbor mutations in the neurofibromatosis type 2 (NF2) gene or chromosome 22q deletions, a distinct subset of skull base meningiomas, particularly those of the meningothelial subtype, is driven by non-NF2 mutations, most notably in TRAF7, KLF4, AKT1, and SMO. [9,10] The identification of a TRAF7 mutation not only aids in confirming the diagnosis of a skull base meningioma but also provides prognostic insights into its clinical behavior. Furthermore, IHC markers have evolved; SSTR2A has emerged as a highly sensitive and specific diagnostic marker for meningioma, reliably distinguishing it from histological mimics such as schwannoma, which typically express S100 and SOX10. [11,12]

Understanding these foundational epidemiological and molecular concepts is essential for navigating the complexities of CPA lesions.

3.2. Comparative Analysis of Our Case with Existing Literature

The phenomenon of a CPA meningioma mimicking a vestibular schwannoma is a recognized but relatively uncommon clinical entity. Previous literature has documented that up to 25% of CPA meningiomas can be initially misdiagnosed as schwannomas on neuroimaging, particularly when the tumor exhibits an intracanalicular component or lacks a prominent dural tail. [4,6,13]

In a study by Gao et al., meningiomas involving the internal auditory canal were predominantly meningothelial. They were associated with higher rates of postoperative residual tumor due to the surgical complexity of the region. [3] Our case aligns with these findings, as the tumor demonstrated extension into the internal auditory canal and was confirmed as a meningothelial subtype. However, our case diverges from typical reports by highlighting a delayed recognition of the misdiagnosis, occurring six years post-resection, only upon symptomatic recurrence.

The integration of modern molecular techniques in our case provides a stark contrast to older case series that relied solely on morphological assessment. The identification of the TRAF7 mutation and the absence of NF2 alterations in our patient are consistent with recent genomic landscapes of non-NF2-driven skull base meningiomas. [10]

Furthermore, the robust positivity for SSTR2A proved invaluable in retrospectively correcting the diagnosis, reinforcing the recommendations by Boulagnon-Rombi et al. that SSTR2A and epithelial membrane antigen should be routinely employed to differentiate meningiomas from S100-positive schwannomas. [12] This case adds to the literature by demonstrating the utility of comparative genomic hybridization to distinguish true recurrence from radiation-induced or de novo secondary primary tumors.

4. What have we learned from This Case?

This case underscores the importance of a multimodal diagnostic approach that integrates IHC, radiologic findings, and molecular studies rather than relying on a single modality.

Histomorphology allowed definitive characterization of the initial tumor. Upon recurrence years later, comparative evaluation demonstrated that the two lesions were identical. A key clinical lesson is the recognition of radiologic red flags; in this case, a broad dural attachment strongly favored meningioma over a CPA schwannoma. An accurate diagnosis required the correlation of all available data.

IHC analysis, including SSTR2A, EMA, PR, S100, and SOX10, played a critical role in tumor classification and distinction between entities with overlapping features.

Additionally, identification of shared molecular alterations, such as TRAF7 mutations detected by next-generation sequencing, provided strong evidence against a second primary neoplasm and in favor of tumor recurrence. This case highlights the importance of maintaining a low threshold for re-evaluation when a presumed recurrence demonstrates features that deviate from the original diagnosis.

Abbreviations

- Cerebellopontine angle (CPA);
- Immunohistochemistry (IHC);
- Somatostatin receptor 2A (SSTR2A)

5. Conclusion

This case highlights the profound diagnostic challenges encountered when a cerebellopontine angle meningioma clinically and radiologically mimics a vestibular schwannoma. The initial misdiagnosis, rooted in morphological ambiguity and the absence of advanced IHC profiling at the time of the first surgery, underscores a critical lesson for neuropathology and neurosurgery. The integration of highly specific markers, such as somatostatin receptor 2A, alongside molecular sequencing for mutations like TRAF7, is paramount for accurate tumor classification. This precision is not merely academic; it directly dictates the surgical strategy, the anticipated risk of recurrence, and the need for adjuvant therapies such as stereotactic radiosurgery.

By presenting this case, we emphasize to the medical community the necessity of a multidisciplinary approach and comprehensive molecular and IHC panels for the evaluation of CPA tumors, ensuring that patients receive the most appropriate, individualized, and effective long-term care.

Compliance with ethical standards

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All authors make the following declarations:

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- Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of ethical approval

This work did not involve any studies on human or animal subjects performed by the authors. It was a retrospective review of archival pathology material collected during routine clinical care, and all data were fully de-identified before analysis.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

Statement of informed consent

The patient was lost to follow-up, and all attempts to reach the patient were unsuccessful. Therefore, the paper has been sufficiently anonymized to maintain patient confidentiality.

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