

Synchronous CP angle meningioma and schwannoma in the absence of neurofibromatosis: a rare case report

Abstract

Background: Meningiomas represent the most common primary tumours of the central nervous system and are typically benign, slow-growing neoplasms originating from arachnoid cap cells. They are graded according to the World Health Organisation classification system into three categories based on histopathological features. Schwannomas are benign tumours arising from Schwann cells of the peripheral nerve sheaths and only rarely demonstrate malignant transformation. The simultaneous occurrence of meningioma and schwannoma in the same intracranial region, in the absence of underlying genetic syndromes or prior radiation exposure, is an uncommon clinical finding.

Case description: A 40-year-old female presented with progressive right-sided facial weakness and was diagnosed with Grade IV facial nerve palsy. Magnetic resonance imaging revealed a lesion located in the right cerebellopontine angle. Surgical excision was performed, and subsequent histopathological evaluation demonstrated the presence of both meningioma and schwannoma within the same anatomical site. There was no clinical or radiological evidence suggestive of an associated hereditary tumour syndrome, and the patient had no history of cranial irradiation.

Conclusion: The coexistence of meningioma and schwannoma within the cerebellopontine angle without identifiable predisposing factors is rare and may pose diagnostic and therapeutic challenges. Recognition of this entity is important for appropriate surgical management and accurate histopathological assessment.

Keywords: collision tumour, neurofibromatosis, cerebellopontine angle, schwannoma, meningioma

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Seema Acharya, Saqib Ahmed, Shreya Joshi
Pathology, Shri Guru Ram Rai Institute of Medical & Health Sciences, India

Correspondence: Saqib Ahmed, Department of Pathology, Shri Guru Ram Rai Institute of Medical & Health Sciences, Dehradun, India, Tel +91 941 1800648

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Abbreviations: CNS, central nervous system; NF2, neurofibromatosis type 2; CPA, cerebellopontine angle; WHO, world health organization

Introduction

Meningiomas are the most common primary tumors of the central nervous system (CNS), accounting for a substantial proportion of intracranial neoplasms.¹⁻³ They arise from arachnoid cap cells and are typically slow-growing with a predominantly benign course. Their clinical significance depends largely on anatomical location and associated mass effect.

The World Health Organization (WHO) classifies meningiomas into three grades based on histopathological and molecular features, reflecting biological behavior and prognosis.² Although most cases occur sporadically, neurofibromatosis type 2 (NF2) predisposes individuals to multiple intracranial tumors, including meningiomas and schwannomas.⁵

Schwannomas are benign tumors arising from Schwann cells of peripheral nerve sheaths and commonly involve cranial nerves, particularly the vestibular nerve.^{6,7} They are usually encapsulated and exhibit slow growth.

The coexistence of meningioma and schwannoma within the same anatomical location in the absence of NF2 or prior radiation exposure is exceedingly rare. We report a case of synchronous tumors in the cerebellopontine angle.

Case study

A 40-year-old woman presented with bilateral hearing loss, intermittent headache, and facial deviation of five days' duration. The symptoms were progressive and were accompanied by difficulty in facial movements. The patient had a history of prior right-sided retromastoid suboccipital craniotomy performed approximately 6 years earlier at an outside institution for a cerebellopontine angle lesion. However, detailed operative records and histopathological reports from the previous surgery were not available for review. Therefore, the exact indication for the prior procedure and the nature of the lesion excised could not be definitively established. In the absence of prior histopathological documentation, the possibility of tumour recurrence, residual disease, or previously unrecognised dual pathology cannot be completely excluded. There was no documented medical or allergic history. Although her father reportedly had similar nodular lesions over the body, there was no confirmed diagnosis of neurofibromatosis in the family. On examination, the patient was conscious, oriented, and hemodynamically stable, with an adequate Glasgow Coma Scale score. The systemic examination did not reveal any additional abnormalities. Routine laboratory investigations were within normal limits. Non-contrast computed tomography of the head revealed a relatively well-defined heterogeneous lesion in the right cerebellopontine angle region measuring approximately $1.8 \times 1.6 \times 2.6$ cm (Figure 1).

Intraoperative squash cytology from multiple extra-axial swellings revealed two morphologically distinct cellular populations. One population consisted of loosely cohesive clusters of round to oval to

spindle-shaped cells forming focal whorls with mild anisokaryosis in a relatively clear background, consistent with meningioma (Figure 2).

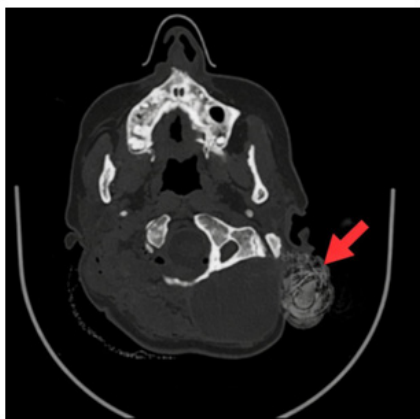


Figure 1 Non-contrast CT scan of the head showing a heterogeneous lesion in the right cerebellopontine angle with mass effect.

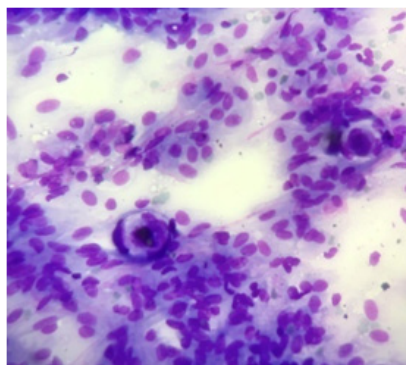


Figure 2 Intraoperative squash cytology demonstrating whorling of meningotheelial cells (MGG, X40).

The second population comprised cohesive spindle cells exhibiting mild nuclear pleomorphism, regular nuclear contours, and scant cytoplasm embedded within a fibrillary background, suggestive of schwannoma (Figure 3).

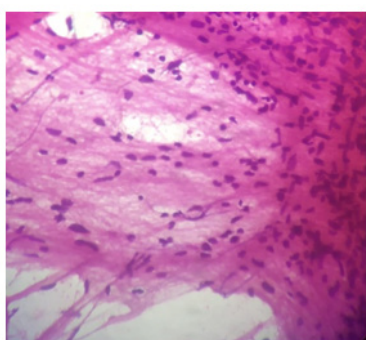


Figure 3 Cohesive spindle cells exhibiting mild nuclear pleomorphism, regular nuclear contours, and scant cytoplasm embedded within a fibrillary background (H&E×40).

These cytomorphological findings raised the possibility of a synchronous lesion. Subsequent histopathological examination confirmed the presence of meningioma admixed with schwannoma (Figure 4 & 5), consistent with a WHO Grade I lesion as per the 2021 WHO classification.⁴

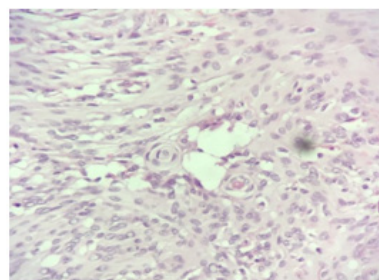


Figure 4 Histopathological section showing characteristic whorling of meningotheelial cells (H&E X40).

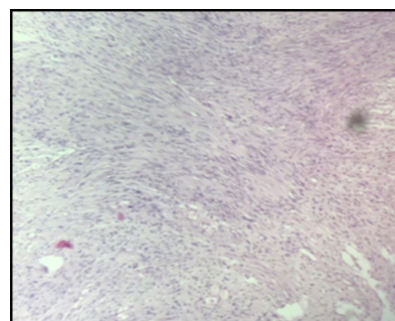


Figure 5 Alternating hypo- and hypercellular areas composed of spindle cells (H and E stain ×40).

Immunohistochemical analysis demonstrated epithelial membrane antigen (EMA) positivity in the meningioma component, there by substantiating the diagnosis (Figure 6).

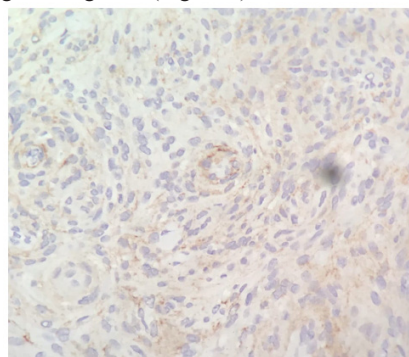


Figure 6 Immunohistochemistry showing dot-like membranous EMA positivity in the meningotheelial cells [X40].

The patient underwent complete surgical excision of the cerebellopontine angle lesion along with three additional posterior fossa lesions via a right retromastoid suboccipital approach. The postoperative course was uneventful, and she remained under neurosurgical follow-up.

Discussion

Meningiomas and vestibular schwannomas are among the most common benign intracranial tumours, collectively accounting for a significant proportion of extra-axial central nervous system neoplasms.¹ Meningiomas originate from arachnoid cap cells and are classified according to the 2021 World Health Organization (WHO) criteria based on histological and molecular features that guide prognostic stratification.⁴ Vestibular schwannomas arise from Schwann cells of the vestibular division of the eighth cranial nerve

and typically exhibit slow growth with progressive cranial nerve involvement.³

The synchronous occurrence of these tumours is most frequently associated with neurofibromatosis type 2 (NF2), a hereditary tumour predisposition syndrome caused by mutations in the NF2 gene on chromosome 22, leading to loss of function of the merlin tumour suppressor protein.⁵ Patients with NF2 commonly develop bilateral vestibular schwannomas in association with multiple meningiomas and other intracranial tumours. In addition to genetic predisposition, prior exposure to ionising radiation is a well-established independent risk factor for the development of multiple primary intracranial neoplasms, particularly meningiomas.² Epidemiological studies have further characterised the incidence and risk factors associated with meningiomas in the general population.³

In contrast, the coexistence of histologically distinct tumours such as meningioma and schwannoma within the same anatomical region, in the absence of NF2 or prior radiation exposure, is exceedingly rare.¹ Such cases are often described as “collision tumours,” particularly when occurring in the cerebellopontine angle (CPA), where two independent neoplasms develop in close proximity or become intermingled within a confined anatomical space.^{2,6}

The differential diagnosis of CPA lesions includes vestibular schwannoma (most common), meningioma, epidermoid cyst, arachnoid cyst, and metastasis. Vestibular schwannomas are typically associated with internal auditory canal widening, whereas meningiomas demonstrate a broad dural attachment and may exhibit a dural tail sign. Epidermoid cysts characteristically show restricted diffusion on MRI and lack contrast enhancement, distinguishing them from solid tumours. Metastases, although less common in this location, should be considered in patients with a known primary malignancy.⁷

A major diagnostic challenge in such cases is the preoperative identification of dual tumour pathology. Radiological imaging

often fails to distinguish closely apposed or intermixed tumours due to overlapping enhancement characteristics. Consequently, most cases are diagnosed intraoperatively or on histopathological examination. This underscores the importance of maintaining a high index of suspicion, particularly when intraoperative findings reveal heterogeneous tumour consistency or when squash cytology demonstrates distinct cellular populations.⁸

Several pathogenetic mechanisms have been proposed to explain this rare phenomenon, including coincidental independent tumorigenesis, shared oncogenic pathways, local microenvironmental alterations induced by one tumour facilitating the development of another, and differentiation from a common progenitor cell population.⁹ Molecular studies suggest that alterations in tumour suppressor pathways and growth factor signalling may contribute to meningioma development, potentially creating a permissive environment for adjacent neoplastic transformation.¹⁰

Although our patient reported a suggestive family history, there was no clinical or radiological evidence of NF2, and genetic testing was not available to establish a definitive molecular diagnosis.

From a surgical perspective, recognition of dual tumour pathology is critical for operative planning and optimal outcomes. Complete surgical excision remains the primary treatment modality for both meningiomas and vestibular schwannomas when safely achievable.¹¹ In the CPA, preservation of cranial nerve function, particularly the facial nerve, is of paramount importance. In the present case, intraoperative identification of differing tumour consistencies and planes of cleavage prompted careful pathological evaluation, ultimately confirming the coexistence of two distinct neoplasms. Immunohistochemical analysis played a crucial role in distinguishing meningotheial from schwannomatous components. A review of previously reported cases of synchronous cerebellopontine angle meningioma and schwannoma was performed to better understand the clinical profile, diagnostic challenges, and outcomes associated with this rare entity (Table 1).

Table 1 Summary of cases by clinical and pathological features

Study (Year)	Age/Sex	NF2 Status	Location	Clinical presentation	Pathology	Nature of lesion
Adib SD et al. ⁴	45/F	Positive	CPA	Hearing loss	Schwannoma	Collision tumor
Wiemels J et al. ⁵	NA	NA	Multiple	NA	NA	Epidemiological study
Evans DG et al. ⁶	NA	Positive	Multiple CNS sites	Multiple tumors	NF2 associated tumors	Syndromic
Hilton DA et al. ¹⁰	NA	NA	Peripheral nerve / CPA	NA	Schwannoma	Familial predisposition
Goldbrunner R et al. ¹¹	NA	NA	CNS	Variable	Meningioma	Guideline-based review
Present Case (2026)	40/F	Negative	CPA	Facial palsy	Meningioma + Schwannoma	Dual pathology(collision)

Comparison with previously reported cases highlights that synchronous meningioma and schwannoma most commonly occur in the setting of neurofibromatosis type 2 (NF2). In contrast, the present case is sporadic, with no clinical or radiological evidence of NF2, thereby adding to the limited number of such reports in the literature. Another distinguishing feature is the preoperative misdiagnosis as a single lesion, which is consistent with prior reports and underscores the limitations of imaging in identifying dual pathology.

Furthermore, unlike syndromic cases where multiple lesions are expected, the coexistence of histologically distinct tumours in a single anatomical compartment without genetic predisposition raises important questions regarding tumorigenesis, including

coincidental occurrence versus microenvironment-driven neoplastic transformation.

Conclusion

The synchronous occurrence of meningioma and schwannoma in the cerebellopontine angle without confirmed neurofibromatosis or prior radiation exposure is exceedingly rare. This case highlights the importance of maintaining a high index of suspicion when evaluating cerebellopontine angle lesions, particularly when cytological examination reveals dual morphological components. Comprehensive histopathological assessment supplemented with immunohistochemistry is critical for accurate diagnosis. Recognition

of such rare coexisting tumours not only aids in appropriate surgical management but also underscores the need for thorough clinical evaluation for underlying genetic predispositions. Further documentation of similar cases may help clarify the underlying pathogenetic mechanisms and improve understanding of this uncommon entity.

Acknowledgments

None.

Conflicts of interest

The authors declare that there are no conflicts of interest.

References

1. Ostrom QT, Cioffi G, Waite K, et al. CBTRUS statistical report: primary brain and other central nervous system tumors diagnosed in the United States in 2014-2018. *Neuro Oncol.* 2021;23:1–105.
2. Louis DN, Perry A, Wesseling P, et al. The 2021 WHO classification of tumors of the central nervous system: a summary. *Neuro Oncol.* 2021;23(8):1231–1251.
3. Rogers L, Barani I, Chamberlain M, et al. Meningiomas: knowledge base, treatment outcomes, and uncertainties: a RANO review. *J Neurosurg.* 2015;122(1):4–23.
4. Adib SD, Tatagiba M. Surgical management of collision tumors between vestibular schwannoma and meningioma in the cerebellopontine angle in patients with neurofibromatosis type 2. *Acta Neurochir (Wien).* 2019;161(6):1157–1163.
5. Wiemels J, Wrensch M, Claus EB. Epidemiology and aetiology of meningioma. *J Neurooncol.* 2010;99(3):307–314.
6. Evans DG. Neurofibromatosis type 2 (NF2): a clinical and molecular review. *Orphanet J Rare Dis.* 2009;4:16.
7. Perry A, Gutmann DH. Molecular pathogenesis of meningiomas. *J Neurooncol.* 2004;70(2):183–202.
8. Mawrin C, Perry A. Pathological classification and molecular genetics of meningiomas. *J Neurooncol.* 2010;99(3):379–391.
9. Rodriguez D, Young Poussaint T. Neuroimaging findings in neurofibromatosis type 1 and type 2. *Neuroimaging Clin N Am.* 2004;14(2):149–170.
10. Hilton DA, Hanemann CO. Schwannomas and their pathogenesis. *Brain Pathol.* 2014;24(3):205–220.
11. Goldbrunner R, et al. EANO guidelines for the diagnosis and treatment of meningiomas. *Lancet Oncol.* 2021;22(5):e281–e292