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Case Report

Meningioma of the cerebellar peduncle revealed by sudden deafness: A case report

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Abstract

Introduction: Meningiomas in adults represent the most frequent of all primary intracranial tumors in adults. Location and size of such tumors dictate their clinical characteristics. Multiple locations are extremely rare, resulting in atypical clinical presentation and complications, such as a cerebellar peduncle-based meningioma discussed in this case report, manifesting as sudden sensorineural hearing loss. We aim to discuss an exceptional etiology of sudden deafness, describing its clinical and therapeutic features in order to ensure a better understanding of meningioma of this location.

Case presentation: This was a 35-year-old woman with no medical history of pathology who was admitted to our department for a sudden sensorineural hearing loss (SSNHL) in the left ear. The physical examination revealed a normal, complete eardrum on both sides, as well as a neurovestibular examination without abnormalities. Audiometry showed left cochleosis. We completed a Magnetic Resonance Imaging (MRI) of the brain and the pontocerebellar angle, which showed a meningioma next to the left cerebellar peduncle associated with vestibular signal abnormalities related to a hemorrhagic rearrangement of the meningioma. We referred the patient to neurosurgery for additional management.

Clinical discussion: Sudden sensorineural hearing loss is a well-recognized clinical condition, typically idiopathic in origin. Meningiomas located outside the cerebellopontine angle are rarely implicated as a cause of hearing loss. Surgical resection of the meningioma can lead to significant restoration of auditory function. This underscores the importance of considering prompt surgical intervention in cases of SSNHL to potentially reverse hearing deficits.

Conclusion: Sudden deafness is rare, and its occurrence in association with cerebellopontine angle tumors is even rarer.

Keywords: Meningioma, cerebellar peduncle, sudden deafness, treatment

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1. Introduction

Meningiomas in adults represent the most frequent of all primary intracranial tumors, accounting for nearly one-third of all primary brain tumors in the United States. They arise from the arachnoid epithelium and are classified according to their location and histological WHO grade. The majority (90%) are WHO grade 1 (benign), while less than 10% are grade 2 (atypical) or grade 3 (malignant).

Meningiomas are more frequent in women, with an increasing incidence with age [1]. Although meningiomas can develop anywhere in the cranial cavity, about 10% occur in the posterior fossa, often in the convexity or pontocerebellar angle, with less common locations including the petroclival region and foramen magnum [2]. Location and size of such tumors dictate the clinical presentation.

Symptoms vary from cranial nerve lesions (such as hearing loss, facial weakness or numbness, and dysphagia), cerebellar symptoms (ataxia, dysarthria, dysmetria), and brainstem compression (hemiparesis).

Headaches are also common, and large tumors compressing the fourth ventricle can cause hydrocephalus with ataxic gait, dementia, and urinary incontinence [2]. A cerebellopontine angle (CPA) meningioma that involves the internal auditory canal can lead to sensorineural hearing loss. This can occur either through direct damage to the vestibulocochlear nerve or by disrupting blood flow to the nerve [3]. Advances in neuroimaging, including computed tomography (CT) and MRI, have facilitated the diagnosis of many meningiomas in the pre-symptomatic phase [4]. However, some cases are still diagnosed late, often after years of misattributed symptoms. In this report, we highlight a rare presentation of cerebellar peduncle meningioma manifesting as SSNHL.

Different locations can cause a wide variety of symptoms, which can explain the lack of knowledge regarding specific tumor location, and this case report aims to broaden our scope of knowledge regarding clinical symptoms associated with cerebellar peduncle meningioma.

2. Case report

This case involves a 35-year-old woman with no significant medical history who presented to our department

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with a SSNHL in her left ear. She reported no accompanying symptoms such as tinnitus, headache, balance problems, or dizziness. She also had no history of trauma, ear infection, or smoking.

On physical examination, both tympanic membranes appeared normal, and neurovestibular assessment revealed no abnormalities. Cranial nerve examination, including the trigeminal and facial nerves, was intact. Long tract signs, cerebellar function, and Romberg test were unremarkable. Coordination was preserved with a normal tandem walk, and there were no pathological signs such as gaze-evoked nystagmus, Bruns' nystagmus, abnormal head impulse test (HIT), or impaired vestibulo-ocular reflex (VOR).

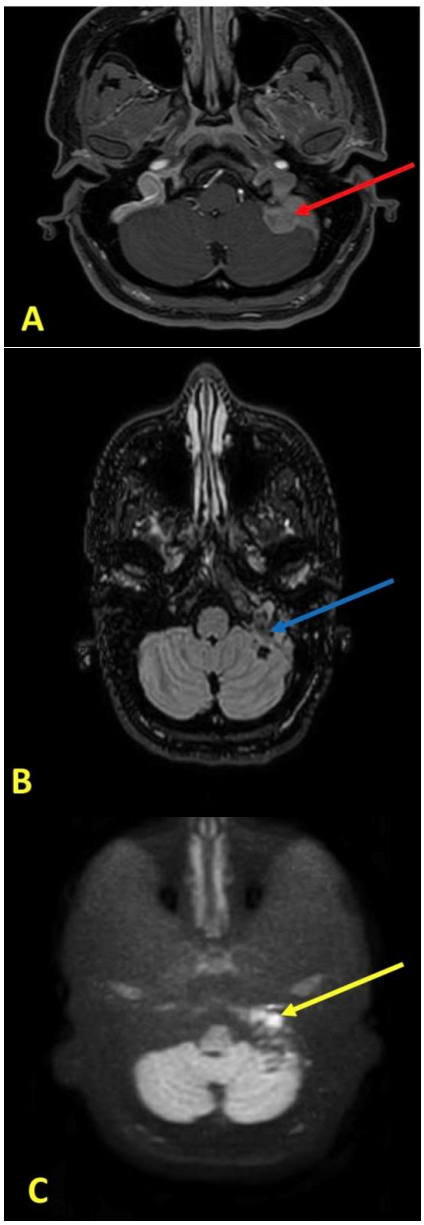

Fig. 1. MRI of the Cerebellopontine Angle.
(A) Fat-saturated contrast-enhanced T1-weighted MRI axial image (Fat-Sat C+ TI). (B) FLAIR axial image. (C) Diffusion-weighted imaging (DWI). Left cerebellopontine angle dural-based solid mass, having a vivid and homogeneous contrast enhancement (red arrow). It shows to be slightly hypointense on FLAIR compared to the cortex (blue arrow) and heterogeneous hyperintense on DWI image (yellow arrow). Mild compression of the left cerebellum is noted. It also projects onto the margin of the left jugular bulb.

Pure tone audiometry confirmed a left cochleosis. For further investigation, we performed an MRI of the brain and CPA that revealed a meningioma adjacent to the left cerebellar peduncle. The tumor was associated with vestibular sign abnormalities attributed to a local bleeding complication (Figure 1).

We referred the patient to neurosurgery for further management. The decision was to actively monitor the tumor with clinical follow-up and imaging. After 4 years of follow-up, the tumor remains stable.

3. Discussion

Meningiomas arise from arachnoid cap cells within the arachnoid villi and can develop in a multitude of locations, such as in the base of the skull, cranial vault, and in the spinal cord [5]. In the United States, meningiomas account for 36.6% of all primary central nervous system (CNS) tumors and 53.2% of non-malignant primary CNS tumors [6]. The overall incidence of meningioma was 8.3 per 100,000 persons between 2010 and 2014, an increase from 4.52 per 100,000 persons observed between 1998 and 2002 [6]. These increasing trends in the incidence of primary brain tumors, particularly meningiomas, have been observed in several countries over the last few decades. Some experts suggest that this increase may be influenced by factors such as population ageing, improved access to health care, advances in diagnostic techniques, changes in tumor classification systems, and higher rates of histological confirmation, even in older patients. Even though such factors can cause this increase, they do not fully account for the observed increase in incidence across the majority of age groups [5]. In addition to intrinsic risk factors, such as gender, ethnicity, allergic conditions, family and personal history, and genetic polymorphisms, several extrinsic risk factors are thought to influence the development of meningioma. Changes in these exogenous factors over time may influence incidence trends. While ionizing radiation is the only confirmed causal factor, other potential contributors have been proposed, including electromagnetic fields, diet, pesticides, and hormonal or reproductive factors [5].

The clinical presentation of meningioma varies according to its location. Meningioma can arise from the dura mater of the cranial and spinal nervous system.

Typically, these tumors are slow-growing and non-infiltrative, with symptoms developing insidiously over time, which explains the fact that numerous tumors are discovered incidentally on brain scans and MRIs.

Although no clinical presentation is pathognomonic or highly specific for meningioma, common symptoms include headache caused by the increase of intracranial pressure, focal neurological deficits (especially in cases of cranial nerve involvement), and seizures (either generalized or partial) due to focal mass effect [7].

Sudden hearing loss is an uncommon presentation, particularly in the setting of cerebellopontine angle tumors, where gradual hearing loss is more characteristic.

In a study conducted by Peraio, which included 50 women and 13 men with a median age of 55 years (range: 30–78 years), the average duration of symptoms before hospital admission and surgical intervention was 12 months. The most frequently reported presenting symptom was

hearing loss, noted in 37 patients (59%), and followed by dizziness in 27 patients (43%) and tinnitus in 16 patients (25%). Trigeminal nerve-related symptoms were observed in 15 patients (24%), with facial numbness reported in 12 cases and trigeminal neuralgia in 3 cases. Other symptoms included gait disturbance in 5 patients, dysphagia in 10 (16%), and diplopia in 3. Only one patient was diagnosed incidentally [8].

Ogasawara et al. found lower rates of hearing loss (29-31%) and higher rates of headache (33-37%) and dizziness (16%), but did not report on tinnitus or trigeminal neuralgia [9]. Meanwhile, Bu et al. reported hearing loss in 48% of cases, a strikingly high prevalence of headache at 96%, and significant rates of dizziness (28%), tinnitus (48%), and trigeminal neuralgia (29%) [10].

In fact, hearing represents a statistically significant symptom attributed to meningiomas, but it is rather gradual over a period of time.

In our case, our patient presented with a sudden decrease in hearing in her left ear. She did not report any accompanying symptoms such as tinnitus, headache, balance problems, or dizziness.

One of the least frequent symptoms is sudden deafness, with only two cases mentioning such a clinical presentation. In one case, there were no associated symptoms, while in the other, there was vertigo. The authors speculated that a pre-existing defect might have gone unnoticed, a possibility that may apply to other cases, although thorough patient questioning ruled out previous deficits.

CPA meningioma involving the internal auditory canal can cause sensorineural hearing loss through direct injury to the vestibulocochlear nerve or disruption of its blood supply [3].

If the facial nerve is affected, facial paralysis and dysfunction may occur. A larger CPA meningioma may compress the trigeminal nerve, resulting in symptoms such as decreased corneal reflex, facial hypoesthesia, or even facial or eye twitching [3].

If undiagnosed and untreated, CPA meningioma can cause significant morbidity and potentially life-threatening complications, including hydrocephalus, respiratory depression, and death [11].

MRI is the gold standard for diagnosing and monitoring meningiomas. For patients who are unable to undergo MRI, contrast-enhanced CT is an alternative [12,13].

MRI typically reveals a dural-based, well-defined lesion with homogeneous contrast enhancement. Benign meningiomas frequently exhibit a thickened, contrast-enhancing dural tail and demonstrate iso-intensity relative to gray matter on non-contrast sequences [3,9,12-14]. These extra-axial lesions may also show a cerebrospinal fluid gap adjacent to the tumor [8].

On CT, intralesional calcifications are frequently observed, along with bony changes such as hyperostosis or a "beaten brass" appearance in convexity tumors. While the majority of patients present with a solitary tumor, multiple meningiomas—referred to as "meningiomatosis"—can occur, particularly in syndromic conditions such as neurofibromatosis type 2 (NF-2). However, the presence of multiple extra-axial lesions should prompt consideration of metastasis [7].

The dural tail, although a classic finding, is not pathognomonic of meningioma. It can also be found in metastases and hemangio-pericytomas, but it can help in differentiating meningiomas from schwannomas, which lack this feature [15]. Peri-tumoral edema on T2 and T2-FLAIR on MRI is rare, but it may be seen nevertheless, particularly in secretory meningiomas or more aggressive subtypes with brain invasion. Central necrosis, which is hypo-intense and non-enhancing on T1, is not exclusive to malignant meningiomas, as benign tumors may also show this finding. While CT is better at resolving calcifications, susceptibility-weighted MRI can also detect them [7].

Involvement of cerebral vessels is frequently observed, especially in skull base meningiomas, which may be situated adjacent to or directly involve the carotid and basilar arteries or their branches. Although uncommon, cystic components may also be present within the lesion [15].

In our case, the MRI showed a left CPA dural-based solid mass with vivid and homogeneous enhancement, slightly hypointense on FLAIR compared with the cortex and heterogeneously hyperintense on DWI.

Newer techniques such as positron emission tomography (PET) are increasingly being used in clinical research. These methods are valuable for monitoring recurrence in previously irradiated meningiomas or guiding diagnosis when surgical biopsy is impossible [16,17]. In addition, 18-Fluoro-ethyl-tyrosine (18-FET) PET may provide improved visualization of skull base meningioma compared to conventional MRI [18].

Meningiomas are classified according to the WHO grading system, which serves as a framework for determining clinical prognosis and guiding treatment strategies. This system categorizes meningioma into three grades:

Grade 1 (Benign: 80%): These tumors are characterized by a low mitotic rate (<4 mitoses per 10 high-power fields [HPF]) and the absence of brain invasion. They are the most common and least aggressive type. Grade 2 (Atypical:18%): These meningiomas exhibit a mitotic rate of 4–19 per 10 HPF, demonstrate brain invasion, or display at least three of the following five histological features: spontaneous or geographic necrosis, prominent nucleoli, high cellularity, small cells with a high nucleus-to-cytoplasm ratio, and sheet-like growth without a distinct pattern. Grade 3 (Malignant: 2%): Defined by a mitotic rate exceeding 20 per 10 HPF or an overtly malignant histological appearance, these tumors are associated with a high risk of recurrence, aggressive clinical behavior, and poor overall survival [16-18].

The distribution of documented meningioma cases is as follows: approximately 80% are Grade 1, 18% are Grade 2, and 2% are Grade 3. This grading system underscores the importance of histopathological evaluation in predicting tumor behavior and guiding therapeutic decisions [9].

The management strategy for meningiomas should be individualized. For patients with incidentally discovered grade 1 tumors in low-risk areas, the initial approach often involves observation with periodic MRI [20]. Non-symptomatic lesions with imaging features consistent with meningioma typically have a slow growth rate on serial imaging, suggesting a benign nature [19,20]. Treatment decisions for a Grade 1 meningioma with non-invasive behavior on MRI control must take into account factors such as the patient's age, life expectancy given comorbid

conditions, and the likelihood of significant progression based on tumor location [19]. As most benign meningiomas have a mean volumetric growth rate of 5.82% annually, small lesions in benign anatomical locations have a low risk of progression, making watchful observation the preferred initial strategy [21].

Observation is not a viable long-term strategy for larger or rapidly developing tumors [12,22]. It is also not recommended for cases of symptomatic grade 1 tumors, tumors in symptomatic progression–prone locations, or grade 2 or 3 lesions due to their locally aggressive behavior and malignant potential [7,9].

Surgical resection is the gold standard for tumors unsuitable for observation, especially suspected grade 2 or 3 meningiomas [3,12,14,23]. Surgery provides immediate and long-term relief from the effects of the mass, while allowing a definitive histological diagnosis to be made [19].

The extent of such treatment is traditionally assessed using the Simpson grading system, which classifies the completeness of surgical removal based on intraoperative visual assessment, ranging from grade I (complete removal, including dura and bone) to grade V (biopsy only). Simpson grade I denotes macroscopically complete removal of the tumor, including resection of its dural attachment and any abnormal bone, with excision of the venous sinus if necessary [18,19]. At the other end of the spectrum, Simpson grade V means "simple decompression with or without biopsy". Simpson grades II and III involve gross total resection (GTR), with grade II including coagulation or resection of the dural attachment and grade III indicating gross removal of the tumor without dural resection. Grades I-III are generally categorized as GTR in the literature [19].

The Simpson grading system is correlated with the risk of symptomatic recurrence. At 10 years, the recurrence risk is 9% for Grade I tumors and 19% for Grade II tumors. In contrast, grades III-V, which are not suitable for radical surgical resection, are associated with a significantly higher likelihood of symptomatic recurrence [19].

For low-grade tumors that are unlikely to be completely resected, or in patients at high surgical risk, radiation monotherapy is an excellent alternative [9,10,19,20]. The decision to use adjuvant therapy is based on the risk and potential impact of local progression following surgery. Tumor grade provides a valuable framework for assessing the need for adjuvant radiotherapy [7,24].

Systemic approach has a limited role in the management of meningiomas and is generally reserved for salvage situations where local therapy is not feasible, as recommended by the National Comprehensive Cancer Network (NCCN) [7,10,19].

Evidence for the efficacy of systemic therapies remains sparse. NCCN guidelines recommend chemotherapy for advanced disease when further surgical resection or radiotherapy is not an option [19].

Meningiomas have a wide range of five-year recurrence rates, ranging from 7% for grade I tumors to 90% for grade III tumors. The risk of recurrence is significantly higher for incompletely resected tumors. Overall, the prognosis for CPA meningioma is favorable, with a ten-year survival rate of 81.4% for benign meningioma and approximately 57% for malignant meningioma [25].

Long-term follow-up with regular imaging is essential to detect and monitor recurrence [12].

In our case, the patient was placed on active tumor monitoring with clinical follow-up and imaging, and after 4 years of follow-up, the tumor remains stable.

4. Conclusion

Sudden deafness is rare, and its occurrence in association with CPA tumors is even rarer. Nevertheless, SSNHL, especially if unexplained or associated with other symptoms, should be considered as a possible manifestation of such CPA tumors. These tumors should be considered as a differential diagnosis of sudden SSNHL, especially in women. Meningiomas, which are primarily benign tumors arising from meningothelial cells, account for 37.6% of all primary CNS tumors. These tumors are more common in women, and the incidence increases with age.

Initial diagnosis is usually made by MRI or contrast-enhanced CT scan. The management approach is influenced by several patient- and tumor-specific factors, and due to the variable presentation of meningiomas, optimal treatment should be individualized. Early detection, especially early imaging and intervention, is crucial, as delayed diagnosis and treatment are associated with higher morbidity and mortality.

Ethics approval

Ethical approval was not required for this case report according to institutional policy.

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Declaration of Competing Interest

No conflict of interest to disclose.

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Authors' contributions

MEO: Data collection and analysis, and drafting paper. LM and SJ: Data collection and analysis. MB, HJ, MG, AM, and WK: Data acquisition and analysis.

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