Petrotentorial Meningioma
A Case Report from Thammasat University Hospital

Group Members: Extern EP1, Academic Year 2021-2022

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Case Information:

Case: A 49 years old Female (Right hand dominance)

Domicile : Bangkok

Occupation: an office worker

Coverage: Social Security Scheme

Admission date : 7/3/65

Chief complaint: Left facial spasm 3 years PTA

Present illness :

3 years PTA: The patient developed left hemifacial spasm which involved both upper and lower eyelids as well as her left mouth corner. Her symptom lasted for 10-20 seconds each time and occured on and off for the whole day. Apart from left hemifacial spasm, she denied facial numbness, paresthesia, ptosis, facial palsy, weakening of arms and legs. She had no heachache, blurred vision, difficulty swallowing or speaking. She was able to hear normally. Furthermore, she denied any history of head trauma, facial injury, chronic otitis media and preceding URI symptoms including fever, runny nose and sore throat. Her symptoms persisted for the whole week, thus, she visited a private hospital and was prescribed Botulinum toxin injection to her left face, anticonvulsant drugs and vitamin B supplement pills. After regular follow-up, her left hemifacial spasm did not improve and became worse.

2 years PTA: the patient started to recognize an abnormal sound in her left ear. The sound was described as a clashing of diamonds and lasted for 30 -60 seconds, occuring on and off for the whole day. This abnormal sound was not associated with changing positions or pulsatile pattern. She has no hearing loss, nausea/vomiting , ear pain, ear discharge, blurred vision, ataxia or preceeding URI symptoms. Therefore, she decided to see the doctor and her hearing test was normal.

4 months PTA: she had progressive tinnitus of her left ear; the sound became louder and sounded like a broken speaker's voice. Also, she noticed that she sometimes had water leakage from the corner of her left mouth while drinking. However, she was able to close her eyes normally. In addition to progressive tinnitus of the left ear and left hemifacial spasm, she denied any fever, muscle weakness, numbness, anorexia or weight loss. As a consequence, she was introduced to initial investigations including MRI brain and referred to Thammasat university for proper management.

Past history :

- **Underlying diseases :** none
- **Current medication :** none
- **Food/drug allergy** : none
- **Past surgery /radiation exposure:** none
- **Herbal use/IVDU drug use :** none

Family & Social history:

- **Smoking/alcohol drinking :** none
- **Family history :** denies of TB or hematological diseases , family history of thyroid cancer(brother)

Physical examination :

- **Anthropometric** : BW 66 kg, HT 161 cm, BMI 25.5 kg/m2
- **Vital signs**: BP 123/80 mmHg, BT 36.6ºC, PR 96 bpm, RR 20 bpm
- **GA** : a middle age thai female, normosthenic built, alert, good consciousness
- **HEENT** : normal head contour, no red eyes, normal ear pinna, no pale conjunctiva, no icteric sclera, no palpable lymph nodes, no discharge from eyes and ears, no oral ulcer, no thyroid gland enlargement, no glossitis, no injected pharynx and tonsils
- **CVS**: no neck engorgement, no active precordium, no carotid bruit, regular pulse, capillary refill < 2 second, no heaves, no thrills, normal s1s2, no murmur
- **RS** :normal chest contour, no retraction, symmetrical chest movement, trachea in midline, resonance on percussion, clear and equal breath sound both lungs
- **Abdomen**: no superficial vein dilatation, normoactive bowel sound, no palpable liver and spleen, shifting dullness and fluid thrill are negative
- **Extremities** : no deformities, no bone or joint pain, no clubbing of fingers, no pitting edema, no rash

Neurological examination :

- Good consciousness; Oriented to time, place and person. E4V5M6.
- Cortical signs: No aphasia, No neglect, No alexia, No acalculia. Glabellar sign – negative, Palmomental sign – negative.
- Cranial Nerves Examination:
    - CN I : no anosmia
    - CN II : VA 20/20 BE , VF (confrontation test): No hemianopia, normal VF, RAPD negative, Pupils 3mm RTLBE. No papilledema.
    - CN III IV VI : Full EOM, no nystagmus, no ptosis
    - CN V : Corneal reflex – intact. Normal strength of masseter and temporalis muscles, intact and equal sensory V1-V3
    - CN VII : **LMN facial palsy. (Left hemifacial palsy House Brackmann gr. II),**
    - CN VIII : normal hearing both ears (rubbing test)
    - CN IX, X : no uvula deviation, normal gag reflex
    - CN XI : normal strength of SCM and trapezius muscles
    - CN XII : no tongue deviation nor fasciculation, no tongue atrophy

- Normal muscle tone, Motor Power V/V all extremities, DTR 2+ all extremities
- Sensory: normal pain, temperature and pinprick sensation all extremities.
- Long tract signs: Babinski's sign – Plantarflexion. Clonus test – negative
- Negative stiff neck
- Cerebellar sign: no dysdiadochokinesia, Finger to nose test – negative, No ataxia

Pertinents findings :

1. Left hemifacial spasm 3 years PTA
2. Tinnitus left ear 2 years PTA
3. Left Facial palsy House Brackmann grade II

Problem list :

1. Left hemifacial spasm and facial palsy (House-Brackmann grade II) with tinnitus of the left ear

Discussion & Differential diagnosis

Approach to the patient

The patient is presented with left hemifacial spasm, facial weakness without sparing of the left frontalis muscle, with tinnitus. This thereby signifies a lower motor neuron lesion of the Facial Nerve (CNVII) with possible involvement of the Vestibulocochlear Nerve (CNVIII). Hence, approaching the lesion location at the location where CNVII traverses with CNVIII seems the most appropriate for this case.

CNVII and CNVIII both arise from their respective nuclei at the caudal dorsal aspect of pons and both nuclei and nerves receive blood supply from the anterior inferior cerebellar artery (AICA) or its respective branches. Both CNVII and CNVIII depart the pons at the cerebellopontine angle - CNVII medially and CNVIII laterally, where the nerves simultaneously enter the internal acoustic canal. The nerves then part ways at the lateral end of the internal acoustic meatus - CNVII continuing into the fallopian canal in the petrous bone, and the cochlear part CNVIII innervates the cochlea. Making CNVII and CNVIII in close proximity at the caudal dorsal aspect of pons, the cerebellopontine angle, and the internal acoustic canal.

Cerebellopontine Angle Lesion

A cerebellopontine angle lesion likely in this patient as the motor division and sensory division (nervus intermedius of Wrisberg) along with CNVIII, explaining the patient's lower motor neuron lesion of the facial nerve and the patient's tinnitus. A lesion at this location should also cause a loss of taste at the ipsilateral anterior ⅔ of the tongue, yet is not reported in this patient, possibly due to an intact function of the contralateral facial nerve. Although lesions at cerebellopontine angle may also affect other adjacent structures such as the trigeminal nerve, and abducens nerve (*Brazis, 2011, p. 326*), the abnormalities of the mentioned structures is not seen in the patient. This could be explained possibly by the limited size of the lesion.

Internal Acoustic Canal Lesion

An internal acoustic canal lesion presents similarly to a cerebellopontine angle lesion (*Brazis, 2011, p. 326*) and is difficult to differentiate purely from physical examination. Hence a lesion at this location is also a likely differential diagnosis.

Pontine Lesion

The pons is a very compact space with multiple nuclei, nerves, and tracts located side-by-side to each other, making lesions to the facial nucleus (origin of the CNVII) or the superior olivary nucleus (origin of part of CNVIII) likely to affect adjacent structures such as the paramedian reticular formation, abducens fascicle, the trigeminal nerve nucleus, and the spinothalamic tract (*Brazis, 2011, p. 325*). As the patient lacks gaze abnormalities, or sensory abnormalities, a pontine lesion is less likely to explain the patient's current clinical manifestations.

Differential diagnosis

Since the lesion is likely to be localized either at the cerebellopontine angle or the internal acoustic canal, the likely etiology of the lesions are as follow:

- **Tumor -** is highly likely the cause of the patient's clinical presentation. The tumor is more likely to be benign due to gradual progression of symptoms. There are several benign tumors that can cause cranial nerve VII and VIII palsies. These are probably benign tumor in this patient
    - **Meningioma** - is one of the most common primary brain tumors in the adult population (Aruwaili et al., 2021), the tumor is generally slow-growing.
    - **Schwannoma -** is a benign tumor originating from nerve sheath of Schwann cells. Since, the patient presented with abnormal hearing and facial nerve palsy, these are the possible types of schwannoma.
        - **Vestibular schwannoma** is also known as acoustic neuroma. It is one of the most common tumors located at the cerebellopontine angle. Patients with vestibular schwannoma usually presented with ipsilateral sensorineural hearing loss, tinnitus and imbalance (U.S. Department of Health and Human Services, 2015). As the tumor progresses, it might compress the facial nerve and trigeminal nerve, leading to facial paralysis and facial numbness respectively.
        - **Facial nerve schwannoma** also causes facial palsy, tinnitus and hearing abnormalities.
    - **Epidermoid tumor** is the next differential diagnosis in this patient. Although it is possible, the prevalence of epidermoid tumors is lower than others. It accounts for about a percent of all the primary intracranial tumors, it is most commonly located at the cerebellopontine angle (Fernández and De Jesus, 2021). The cells are derived from the ectodermal tissue.

- **Vascular -** is unlikely, the onset of symptoms is generally sudden and abrupt. Nevertheless, there are possible vascular causes leading to progressive onset of symptoms such as aneurysms with thrombosis. Possible locations of aneurysm include anterior inferior cerebellar artery and superior tympanic artery. These arteries are located near the cerebellopontine angle, which is the outlet of cranial nerve VII and cranial nerve VIII. Other possible vascular causes are swelling of superior petrosal vein or anatomical variation such as normal loop of anterior inferior cerebellar artery, these lesions might compress facial nerve and vestibulocochlear nerves.

- **Infection** is improbable because of the patient's clinical presentation as the onset is gradually progressive. The patient also denied a history of previous infections or symptoms that suggest systemic inflammation such as fever. Nevertheless, possible infectious sources that might lead to facial paralysis and tinnitus are as follow:

- **Chronic otitis media (COM)** is a recurrent infection of the middle ears or mastoid air cells. One of the complications of this disease is facial nerve palsy (Kim et al., 2012). However, COM is unlikely because the patient did not have purulent ear discharge, otorrhea, aural fullness and otalgia. Physical examination also did not find tympanic membrane perforation.
- **Brain abscess located at the cerebellopontine angle** might compress the nearby cranial nerves causing palsies. However, it is less likely because the patient denied any previous head and neck infection. There are also no typical symptoms of brain abscess such as headache, mental status change or fever.
- **Atypical infection** might explain the gradual progression of disease and absence of systemic inflammation. However, it is one of the least differential diagnoses due to the low prevalence rate. There is a case report where a patient presented with facial paralysis and ipsilateral hearing loss, caused by tuberculous otitis media (Hwang et al., 2013)

In conclusion, tumour is the most likely etiology in this patient due to slow progression of symptoms. Possible tumours include schwannoma, meningioma and epidermoid tumour. The next likely cause could be vascular origins such as swelling of superior petrosal vein or anatomical variation such as normal loop of anterior inferior cerebellar artery.

Investigation

- **Complete Blood Count (CBC):** Hb 13.1g/dL, Hct 39.5%, WBC 7,068 cells/uL, Neutrophils 56.85%, Lymphocyte 33.8%, Monocyte 7.52%, Eosinophils 1.04%, Platelet 320,000 cells/uL
- **Blood Chemistry & Electrolytes:** BUN 7 mg/dL, Cr 0.55 mg/dL, Sodium 141 mmol/L, Potassium 4.1 mmol/L, Chloride 110 mmol/L, Bicarbonate 24 mmol/L
- **Audiogram**:
  - Right: Air/Bone conduction 5/5, Speech Reception Threshold 10, Phonetically balanced 92%.
  - Left: Air/Bone conduction 13/13, Speech Reception threshold 15, Phonetically balanced 80%
- **MRI Internal Auditory Canal (IAC with Inner Ear:** Well circumscribed T1 isointensity and T2 hyperintensity extra-axial lesion with vivid homogenous enhancement at left cerebellopontine angle (CP angle), left perimesencephalic and ambient cisterns, measured up to 4.5 x 3.7 x 4.2 cm. The lesion has broad-based attachment to left tentorium cerebelli, left petrous bone and posterior wall of left sphenoid sinus. Dural tail sign along the left petrous bone is seen. The mass has pressure effects towards the left cerebellum, left superior and middle cerebellar peduncles, as well as left side midbrain and pons with effacement of the fourth ventricles, suspected from brain edema and gliotic change. No internal auditory canal widening seen.

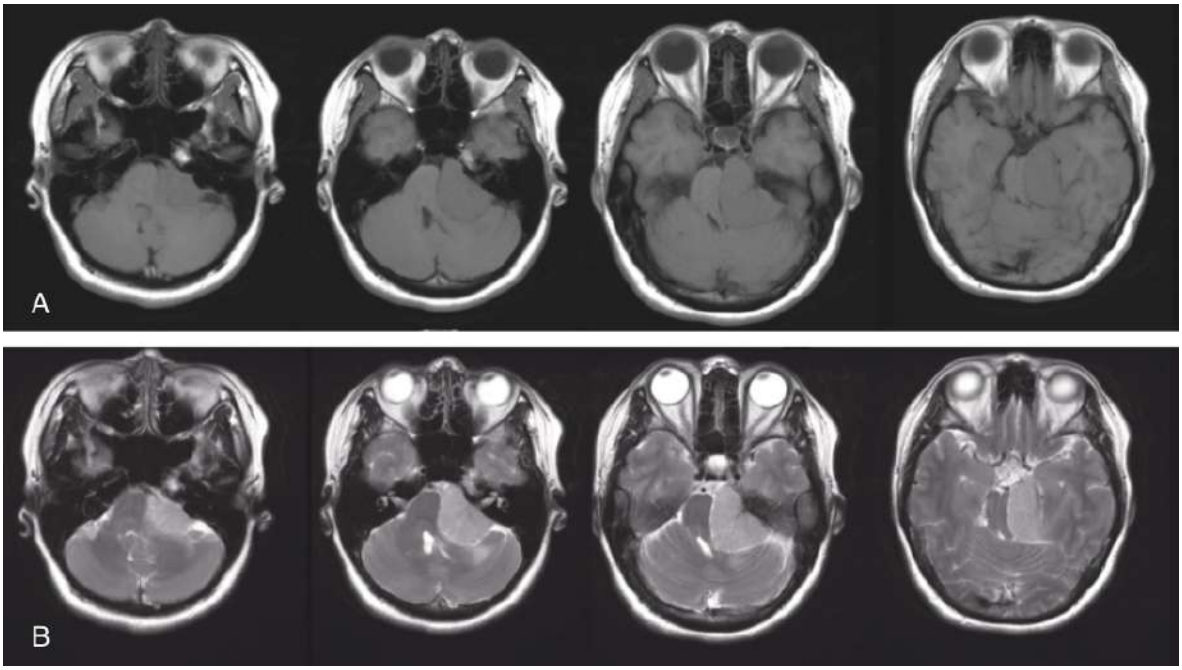


Figure 1 . MRI IAC with Inner Ear. *1A*: Axial view of the T1WI, *1B*: Axial view of T2WI.

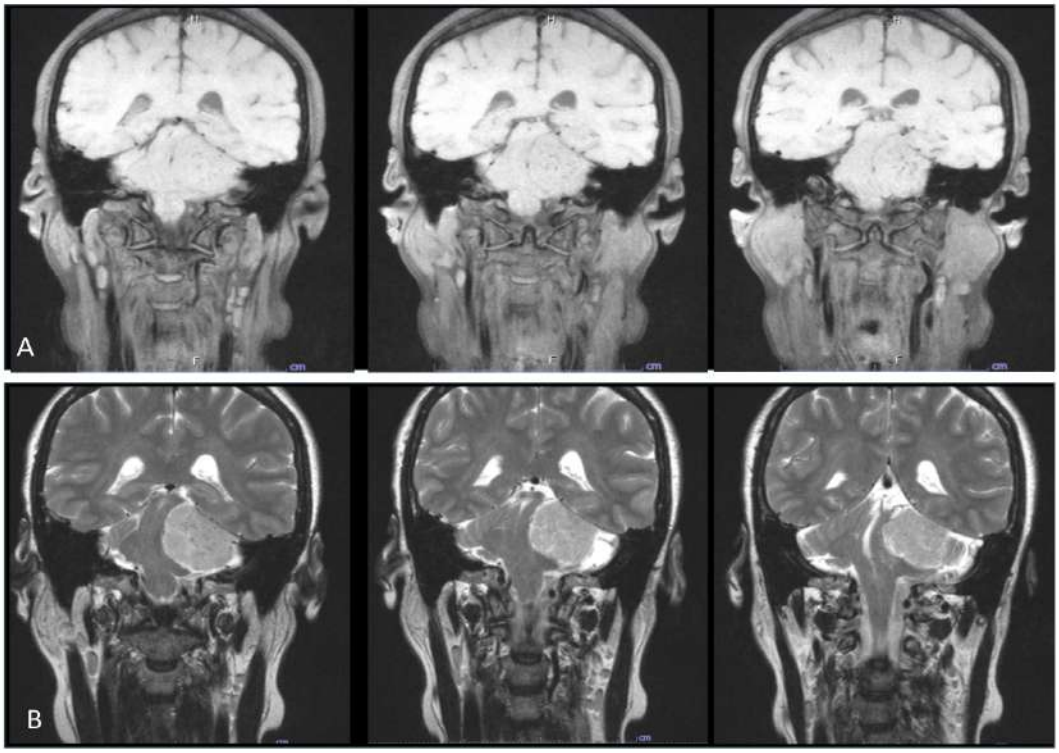


Figure 2. MRI IAC with Inner Ear. *2A*: Coronal view of the T1WI. *2B*: Coronal view of T2WI.

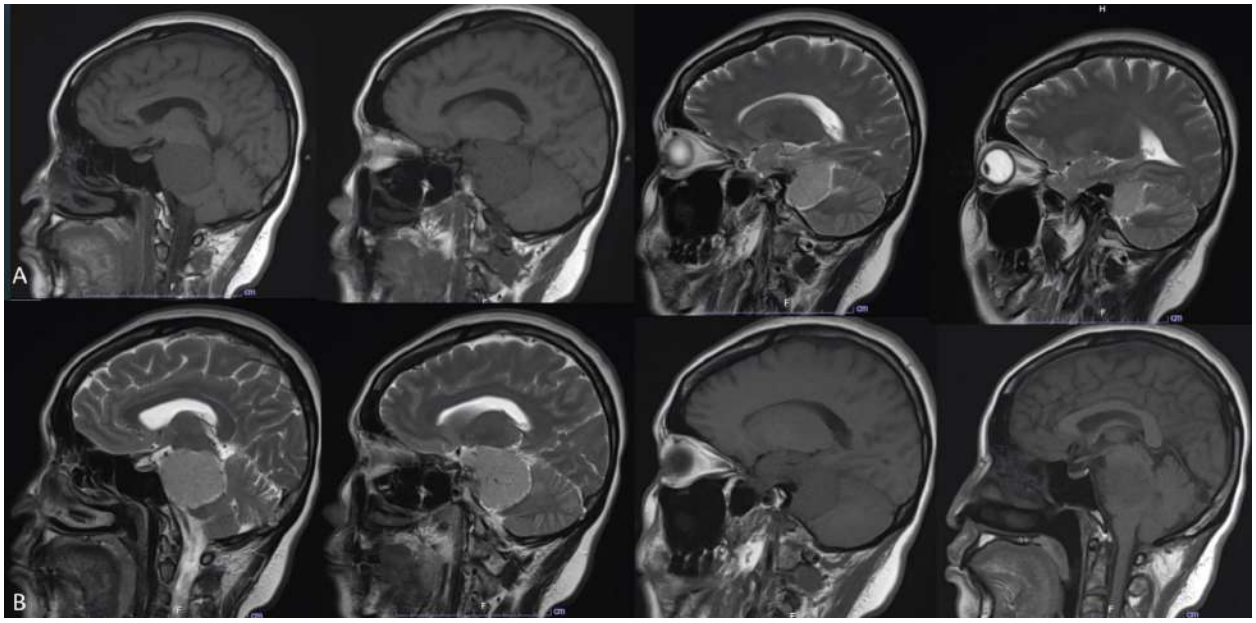


Figure 3. MRI IAC with Inner Ear. *3A*: Sagittal view of the T1WI. *3B*: Coronal view of T2WI.

Management

The patient proceeds into surgery for total removal of tumor by *Left combined transpetrosal approach technique for tumor removal with duraplasty.*

Intraoperative findings:

- Mild brain swelling
- Extra-axial grayish mass originating from a petrous ridge and tentorium with dural tail.
- Hypervascularized tentorium cerebelli
- Mass effects to the left temporal lobe, left cerebral peduncle and left cerebellum without arachnoid plane.
- Tumor lies inside the meckel cave and displaced CNIV medially and CNV inferiorly.
- Preserved all CN III, IV, V, VI, VII, VIII
- Superior petrosal vein and inferior petrosal vein was preserved.

Pathology Report:

- Gross description: multiple pieces of irregular soft light brown tissue measuring 4x3x1.5 cm in aggregate and weighing 9g.
- Diagnosis: *Angiomatous meningioma (WHO grade I)*

PetroTentorial Meningioma

Anatomy Review

Petrotentorial area

The tentorium cerebelli, a structure created from the inward folding of the dura, separates the cerebellum from the occipital lobes of the cerebral hemisphere, thus dividing the brain into supratentorial and infratentorial compartments. The structure attaches anteriorly to the clinoid processes of the sphenoid bone, laterally to the petrous ridge, posteriorly to the inner surface of the occipital bone and part of the parietal bone, and superiorly to the temporal bone (As demonstrated in **Figure 4**). The free dural edge, also known as the tentorial incisura, surrounds the brain stem - attaching anteriorly to the petrous apex and the clinoid processes, forming the anterior, posterior clinoid folds, and interclinoid fold (*Aziz et al., 2009; Moore et al., 2015).*

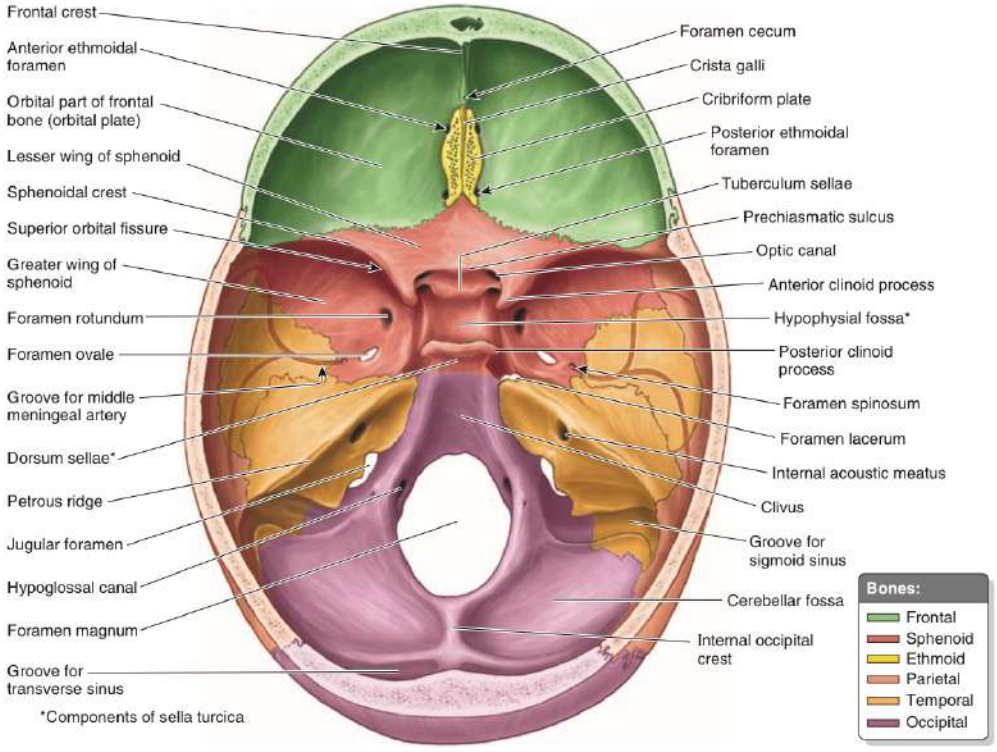


Figure 4: A superior view of the internal surface of the intracranial base - demonstrating the different sites of attachment of the tentorium cerebelli

Acquired from Moore et al., 2015

The tentorium cerebelli can be separated into 3 regions: anterior, middle, and posterior spaces as depicted in **Figure 5A**. The anterior space is located anteriorly to the midbrain and pons, extending beyond the optic chiasm to the interpeduncular fossa. The middle tentorial space is demarcated laterally by the temporal horn and inferiorly by the cerebellum and upper brainstem.

The posterior space is defined anteriorly by the midbrain and the tentorial edge. (*Aziz et al., 2009)*

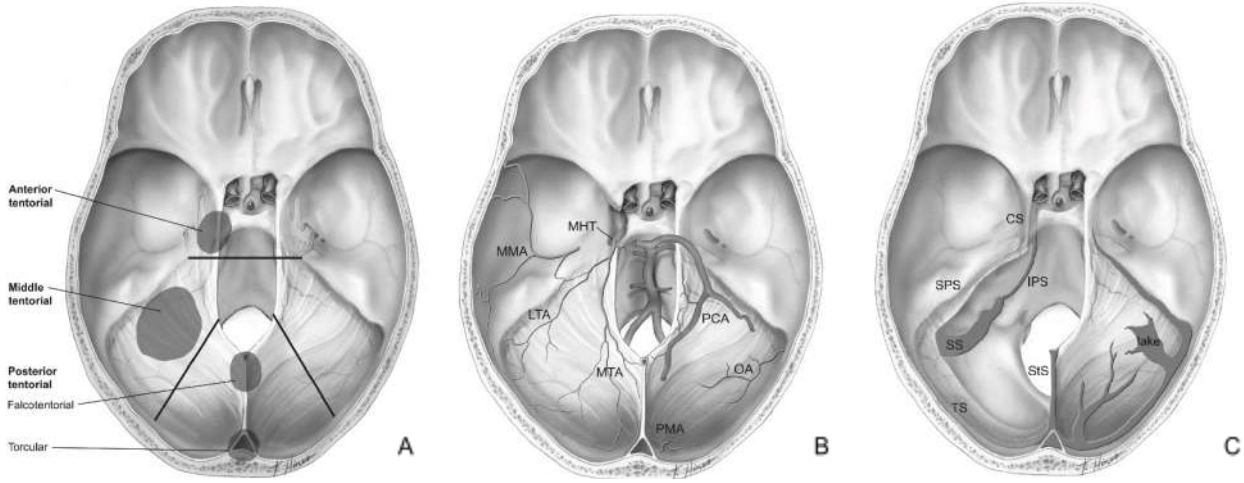


Figure 5: An alternative axial view of the cranium, *5A*: Depiction of the anatomical area of each tentorium cerebelli spaces, *5B*: Major arteries along the tentorium cerebelli, *5C*: Major veins and sinuses near the tentorium cerebelli

Adapted from Aziz et al., 2009

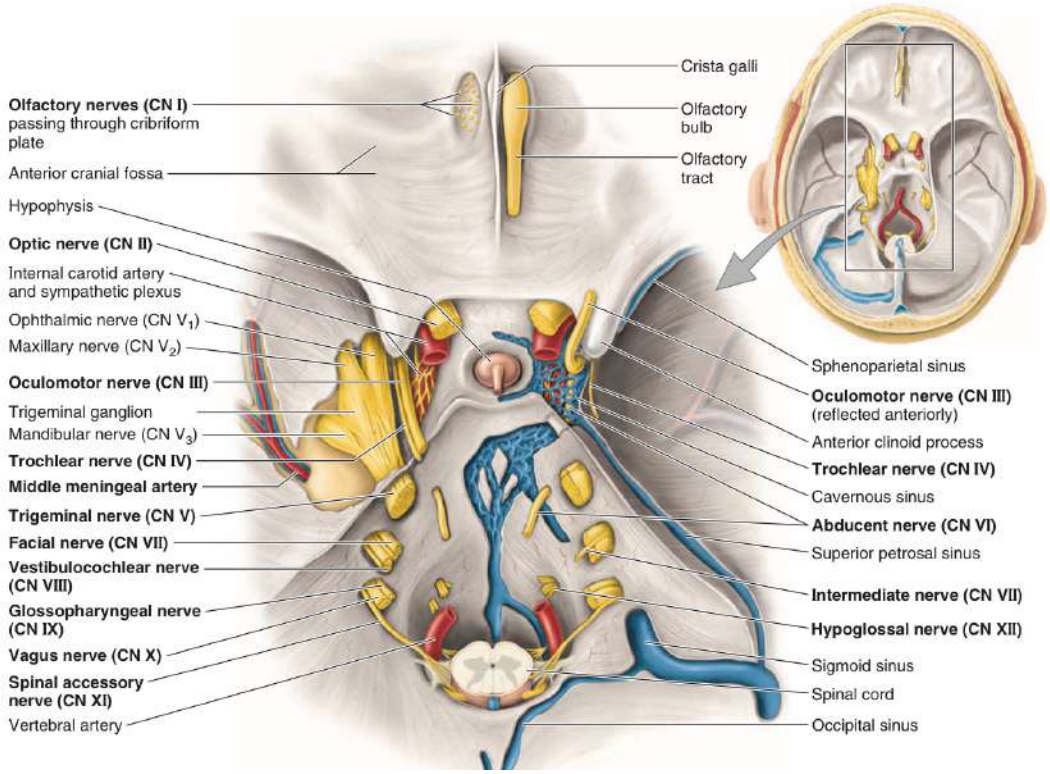


Figure 6: Another alternative view of the cranial base where the tentorium cerebelli is removed to unveil the underlying structures.

Acquired from Moore et al., 2015

The petrotentorial area is a region of the tentorial incisura where the tentorium cerebelli and the petrous bone are in close proximity, i.e. at the Middle Tentorial Space. This structure harbors many essential vessels and structures as summarized in **Table 1** and **Figure 5B, Figure 5C**, and imposes great surgical challenges due to its deep location (*Aziz et al., 2009)*. **Figure 6** demonstrates the major structures beneath the tentorium cerebelli together and the structures in close proximity to the petrous ridge.

Tentorial Incisura Region	**Vital Structures**
Anterior Space	● Arteries ○ Anterior Choroidal Artery ○ Basilar Bifurcation ○ Posterior Communicating Artery ○ Posterior Cerebral Artery ○ Superior Cerebellar Artery ● Veins ○ Basal vein of Rosenthal ● Other Structures ○ Optic Chiasm ○ Oculomotor Nerve ○ Trochlear Nerve ○ Medial Temporal Lobe ○ Uncus ○ Lateral Surface of Midbrain

Middle Space	● Arteries ○ Anterior Choroidal Artery ○ Middle Cerebral Artery ○ Posterior Cerebral Artery ○ Superior Cerebellar Artery ● Veins ○ Basal vein of Rosenthal ○ Transverse Sinus ○ Sigmoid Sinus ● Other Structures ○ Trochlear Nerve ○ Trigeminal Nerve ○ Facial Nerve ○ Vestibulocochlear Nerve ○ Crural Cistern ○ Ambient Cistern
Posterior Space	● Arteries ○ Posterior cerebral artery ○ Superior cerebellar artery ○ Medial posterior choroidal arteries ● Veins ○ Internal Cerebral Vein ○ Basal Vein ○ Vein of Galen ○ Basal vein of Rosenthal ○ Straight Sinus ● Other Structures ○ Pineal gland ○ Quadrigeminal Cistern

Table 1: A summary of vital structures at each area of tentorial incisura

Adapted from Aziz et al., 2009, Moore et al., 2015

Lesions at each tentorial incisura space may cause pathology in the mentioned structures in each respective space. The petrotentorial area is located in the middle tentorial incisura space, hence a lesion in this area may cause abnormalities in cranial nerve IV, V, VII, VIII (*Aziz et al., 2009*) - of which CNVII and CNVIII abnormalities were noted in this patient. Although rare, meningioma in this region may be presented with infarction of vessels in the area such as the middle cerebral artery (*Ko et al., 2014*).

Epidemiology:

Meningiomas are the most common primary central nervous system (CNS) tumors and account for approximately one-third of all primary brain and spinal tumors (Wiemels, J., et al., 2010). The incidence of meningioma increases progressively with age, with a median age at diagnosis of 65 years. Meningiomas are rare in children (Liu, Y. et al., 2008). Meningiomas are more common in women, with a female-to-male ratio of approximately two or three to one (Wiemels, J., et al., 2010).

Distribution of intracranial meningioma from most common to least common locations are as follows: Convexity (35%), Parasagittal 20%, Sphenoid ridge 20%, Intraventricular 5%, Tuberculum sellae 3%, Infratentorial 13% and others 4%. Hence, petrotentotial meningiomas are less common because they fall into the infratentorial type (Wiemels, J., et al., 2010).

Population-based studies estimate that 80 to 85 percent of meningiomas are World Health Organization (WHO) grade 1, approximately 15 to 18 percent are grade 2, and 1 to 3 percent are grade 3 (Wiemels, J., et al., 2010).

Pathogenesis & Etiology:

Meningioma is arised from the neoplastic changes of arachnoid cap cell or meningothelial cells which can occur in a various location of dura such as tentorium, falx cerebi, cerebral convexities and parasagittal area. Tentorial meningioma, a type of posterior fossa meningioma, is classified into a rare type of intracranial meningioma as it accounted for approximately 3% of meningiomas (Qin et al., 2021). The pathogenesis of meningioma is likely to be a cytogenetic abnormality of chromosome 22 where the tumor suppressor gene named NF2 gene located in. Clearly, many studies found that there is a positive correlation between mutation of chromosome 22 q12.2 and the development of meningiomas in up to 70 % of cases (Collins et al., 1990 & Ruttledge et al., 1994). NF2 gene is defined as a tumor suppressor gene that produces a merlin protein,hence if there is a mutation of NF2 gene, merlin protein is likely to be absent or deactivated leading to inability to suppress tumor cells. Numerous studies found that there is a negative relationship between incidence of meningiomas and merlin protein production (Histotsumatsu et al., 1997 & Evans et al., 2001). In addition to genetic alteration factors, the pathogenesis of meningioma is also related to extrinsic factors including radiation exposure and hormonal treatment therapy (Lamszus, 2004). The study reported that there is two fold increased incidence of meningioma in women than men as well as there is a high incidence in breast cancer patients and pregnant women (Lamszus, 2004). Therefore, it is clear that female sex hormne is one of the vital factors in developing meningiomas.

Signs and Symptoms:

From Granick et al. (1985); the most common symptoms of petro tentorial meningiomas were from the CN IV, V, VII, VIII abnormalities as already discussed above in **Table 1**; this can include:

- Abnormal eye movement
- Altered facial sensation/facial pain
- Paralysis of facial half (LMNL)
    - Can be graded according to House Brackmann grading system
- Loss of facial half sensory
- Facial spasm
- Unilateral reduced hearing or sensorineural hearing loss
- Vestibular symptoms
    - Vertigo
    - Dizziness
    - Headache
    - Imbalance/ Gait dysfunction
    - Tinnitus

As the petro tentorial meningioma can also be large in size, thus, other cranial nerve deficits, brainstem compression symptoms, and hydrocephalus can also be seen with larger tumors compressing these structures.

Other neurological physical examinations of notes that should always be checked also include the cerebellar sign such as the dysdiadochokinesia, the coordinated upper-extremity movement via the finger to nose, nystagmus, and ataxia.

All in all, the periphery physical examination must come up normal as well to definitely rule out other causes, this includes an intact tympanic membrane and intact external acoustic canal - structures which can interfere with hearing, vertigo, dizziness, nbalance and tinnitus.

Investigations:

Apart from clinical history taking and the neurological physical examination stated previously, other investigations can be sent to aid in the diagnosis.

According to Asawavichianginda et al. (1997); petro tentorial meningiomas commonly have an association with otologic symptoms, thus multimodality investigations should be sent in order to help rule out other peripheral conditions such an ENT disease/disorder concomitantly or before moving forward with the intracranial investigations.

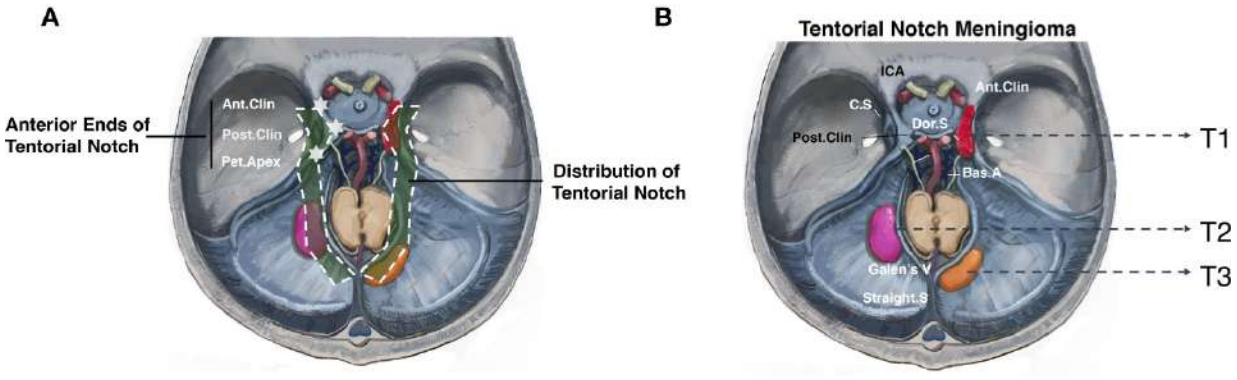
An audiogram is a useful tool to be done to identify whether there is a problem in the internal acoustic canal. Additionally, a Brainstem Evoked Response audiometry (BERA) can help as an objective and non-invasive method of hearing assessment which detects electrical activity from the inner ear to the inferior colliculus. The test can also extend its ability to follow

the auditory pathway from the peripheral endorgan through to the brain stem. Other simple vestibular function tests include the Weber and Rinne test which can help to differentiate between conductive and sensorineural hearing loss or mixed type by using the tuning fork.

Nevertheless, the gold standard of investigation is the radiological imaging of MRI brain with gadolinium (Simic, G. 2019). Classic signs on MRI for meningiomas are isointensity with the brain parenchyma on T1WI and T2WI, but most enhance with gadolinium, and also the "Dural tail sign" which occurs as a result of thickening and enhancement of the dura. Other radiologic diagnostic tools are CT brain with contrast that shows common characteristic of homogenous, densely enhancing mass with broad base of attachment along the dural border and the angiography whose mass may show up at an early arterial phase but persist beyond venous phase. Angiography can aid in preoperative embolisation that helps reduce vascularity and facilitates future surgical removal. However, sometimes; the only way to make a definitive diagnosis of the meningioma is through a biopsy. The neurosurgeon performs the biopsy, and the pathologist makes the final diagnosis, determining whether the tumor appears benign or malignant, and grading it accordingly.

As the specific picture of petro tentorial meningioma MRI are scarce; other MRIs of tentorial meningioma examples are shown in **Figure 7 - 11** in order to help aid readers for a clearer visualization in addition to the description provided.

According to Qin et al (2021), the meningioma located in relation to the tentorial, especially the *tentorial notch* can be classified into anterior (T1), middle (T2), and posterior notch (T3). According to the direction of tumor extension and correlation with the neurovascular structures, detailed subtypes of anterior TNMs were identified as the central (T1a), posterior (T1b), and medial type (T1c). The middle TNMs were divided into the infratentorial (T2a), supratentorial (T2b), and supra-infratentorial type (T2c). The posterior TNMs were divided into superior (T3a), inferior (T3b), lateral (T3c), and straight sinus type (T3d) in reference to Bassiouni's classification as shown in **Figure 7**.



C

Type	Tumor Base	Tumor Extension	No. Cases
T1	Anterior tentorial notch	Main body is located at the anterior tentorial space **Anteriorly extend to**: Oculomotor triangle to cavernous sinus **Laterally extend to:** Middle cranial fossa (along the lateral wall of cavernous sinus) **Inferiorly extend to:** Superior clivus **Superiorly extend to:** III IV VI nerves and posterior circulation (along the dorsum of sella and posterior clinoid process)	14
T2	Middle tentorial notch	Main body is located at the middle tentorial space **Anteriorly extend to:** Meckel's cavity **Inferiorly extend to:** IAC and posterior cranial fossa **Superiorly and laterally extend to:** Middle cranial fossa	22
T3	Posterior tentorial notch	Main body is located at the posterior tentorial space and falcotentorial junction **Extend to:** Deep venous system	17

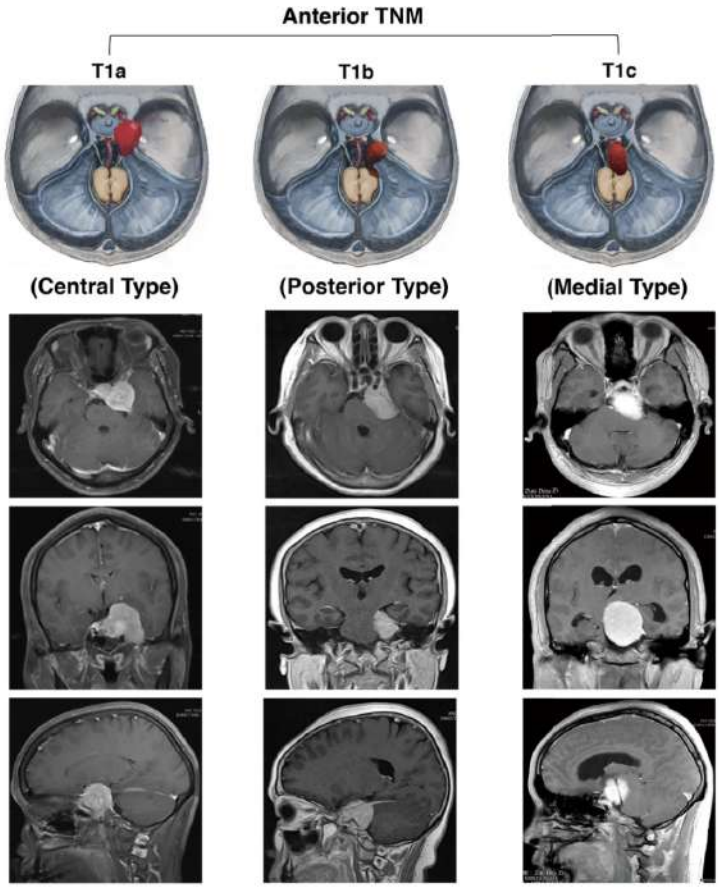
Figure 7 Primary classification of tentorial notch meningioma (TNM). **(A)** The schematic diagram of the distribution of the tentorial notch. **(B)** Schematic diagram of the primary classification of tentorial notch meningioma (TNM). T1: Anterior tentorial notch meningioma; T2: Middle tentorial notch meningioma. T3: Posterior tentorial notch meningioma. ICA, Internal carotid artery; Ant.Clin, Anterior clinoid process; C.S, Cavernous sinus; Dor.S, Dorsum Sellae; Bas.A, Basal artery; Galen's V, Galen's Vein; Straight S, Straight sinus; Post.Clin, Posterior clinoid process. **(C)** Interpretation of primary classification of tentorial notch meningioma (TNM).

Acquired from Chaoyin Qin et al., 2021

In which Qin et al. (2021) further divided the anterior TNM into three subtypes according to the growth direction, tumor involvement, and the origin and extension of the tumor base as Figure 8.

Subtype T1a: Central type
Subtype T1b: Inferior type
Subtype T1c: Medial type

A



B

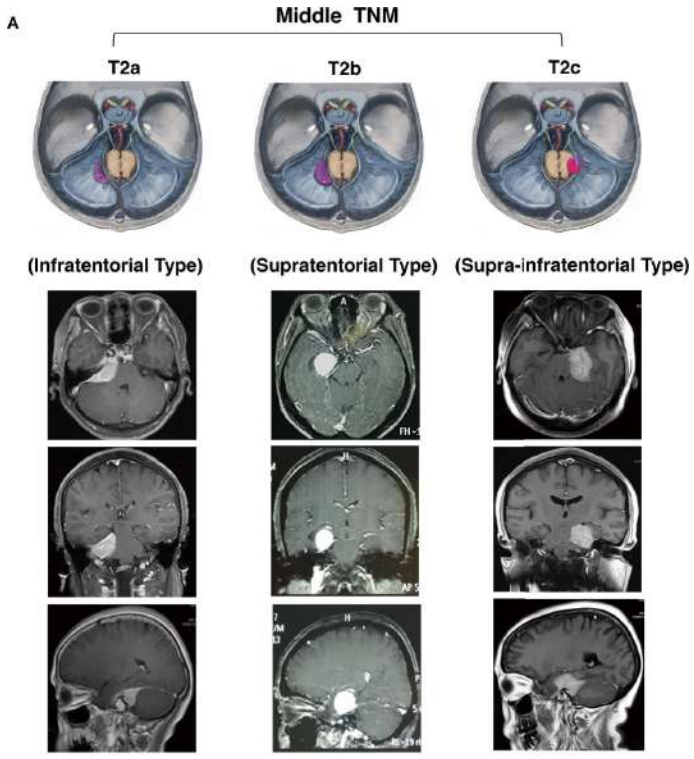
Type	Tumor Growth Direction	Tumor Involvement	Tumor Base Origin	Tumor Base Extension	No. Cases
T1a	Superior	Cavernous sinus Superior clivus Sella Middle cranial Fossa	Anterior/posterior clinoid process fold Inter-clinoid process fold Dura of oculomotor triangle	Widely extension: Lateral wall of CS Superior clivus Middle cranial fossa	5
T1b	Posterior (along the tentorium)	Meckel's cavity Middle cranial fossa	Tentorium around petrosal apex	Posteriorly along tentorium Doral petrosum	6
T1c	Medial	Brain stem (Compression) Basilar artery (displacement)	Posterior clinoid process fold Anterior tentorial notch	Displace the basilar artery Contralateral dorsum sella and superior clivus	3

Figure 8 Further classification of anterior tentorial notch meningioma (TNM). **(A)** Schematic diagram and axial, coronal, sagittal MRI T1-weighted images with gadolinium-based contrast of further classification of anterior tentorial notch meningioma (T1). T1a = central type, T1b = inferior type, T1c = medial type. **(B)** Interpretation of further classification of anterior tentorial notch meningioma (TNM).

Acquired from Qin et al., 2021

The inferior and medial types are easily diagnosed as petroclival meningioma through neuroimaging. The identification needs to be confirmed intraoperatively. Notably, the T1b and T1c TNMs originate from the cerebellum tentorium notch, not the dura of the petroclival junction medial to the trigeminal nerve, which is the true origin of petroclival meningiomas.

The middle notch is divided into the following subtypes: Infratentorial **(T2a)**, Supratentorial **(T2b)**, Supra-infratentorial **(T2c).** Anatomically, the main body of T2a TNM is below the tentorium, which can involve part of the dorsal dura of the petrosum. The T2b TNM grows superiorly on the tentorium without infratentorial extension. The T2c refers to tumors growing along both sides of the tentorium, and the middle and posterior skull base dura can both be involved (Figure 9).



B

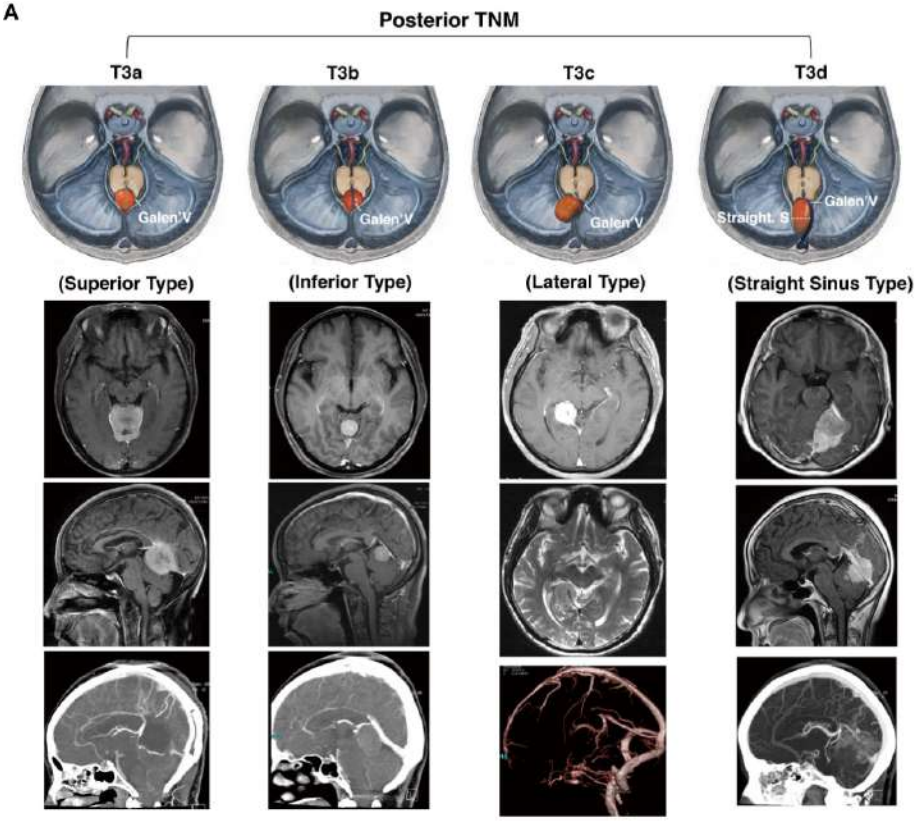
Type	Tumor Base	Tumor Extension	No. Cases
T2a	Middle tentorial notch	IAC and posterior cranial fossa	17
T2b	Middle tentorial notch	Meckel's cavity Middle cranial fossa	2
T2c	Middle tentorial notch	Meckel's cavity IAC and posterior cranial fossa Middle cranial fossa	3

Figure 9 Further classification of middle tentorial notch meningioma (TNM). **(A)** Schematic diagram and axial, coronal, sagittal MRI T1-weighted images with gadolinium-based contrast of further classification of middle tentorial notch meningioma (T2). T2a = infratentorial type, T2b = supratentorial type, T2c = supra-infratentorial type. **(B)** Interpretation of further classification of middle tentorial notch meningioma (TNM).

Acquired from Qin et al., 2021

The posterior notch TNM was defined in accordance with Bassiouni's classification in 2008 (Figure 10).

A



B

Type	Characteristic	No. Cases
T3a (Bassiouni I)	Originating between the leafs of the falx above the junction of the Galen's V with the straight sinus Displacing the Galenic venous system inferiorly	3
T3b (Bassiouni II)	Originating from underneath the tentorium near the junction of the Galen's V with the straight sinus Displacing the Galenic venous system superiorly	6
T3c (Bassiouni III)	Originating from the paramedian tentorial notch The Galen's V lies medial to the tumor (on the supero- or infero-medial aspect of the tumor)	3
T3d (Bassiouni IV)	Originating from the FT junction along the straight sinus Displacing the Galenic venous system contralaterally	5

Figure 10 Further classification of posterior tentorial notch meningioma (TNM). **(A)** Schematic diagram and axial, coronal, sagittal MRI T1-weighted images with gadolinium-based contrast of further classification of posterior tentorial notch meningioma (T3). T3a = superior type, T3b = inferior type, T3c = lateral type, T3d = straight sinus type. **(B)** Interpretation of further classification of posterior tentorial notch meningioma (TNM).

Acquired from Qin et al., 2021

Finally, to help aid in visualization; similarly to the petro tentorial meningioma - **Figure 11** below showed the petroclival meningioma - a more anterior meningioma but has similar characteristics to petro tentorial meningioma on the MRI.

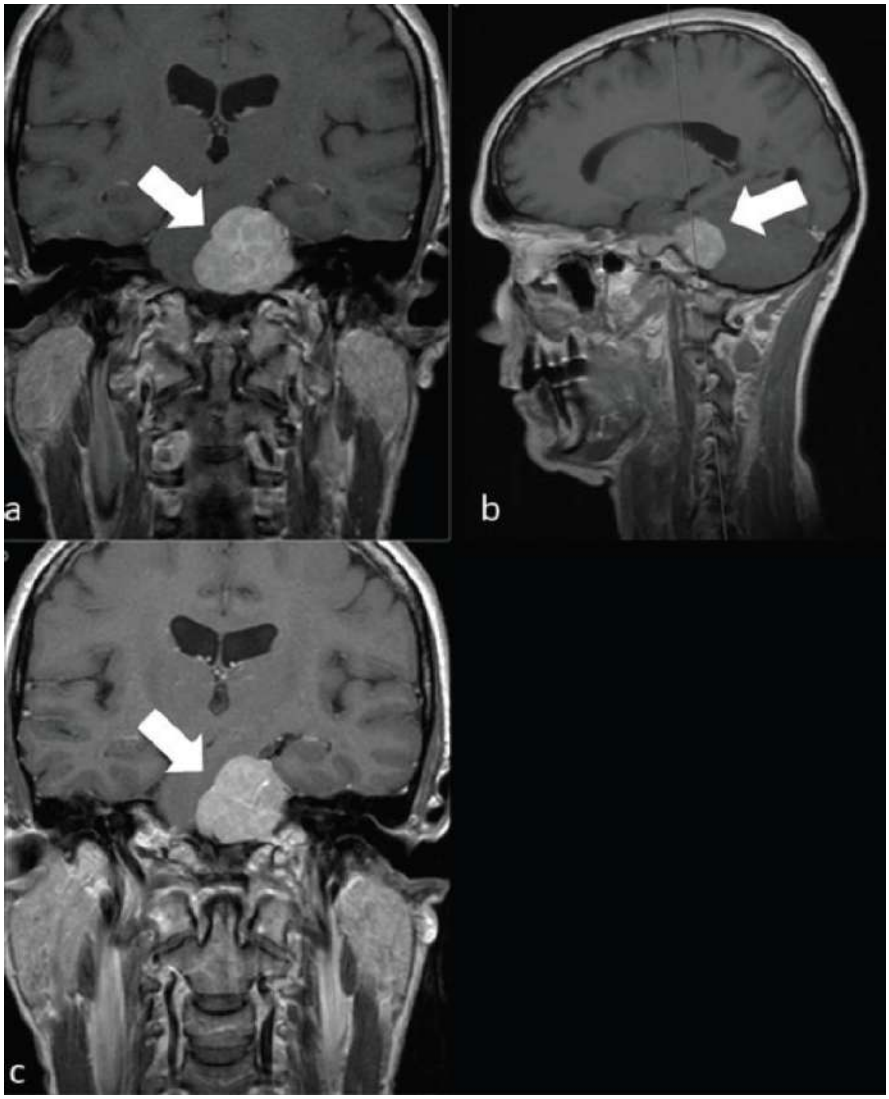


Figure 11 A 62-year-old man with a left petroclival meningioma (PCM). a) Left PCM on coronal T1 sequence. b) Left PCM on T1 sagittal sequence. c) Left PCM on T2 coronal sequence.

Acquired from Nicosia et al. 2019

Almost all of the examples pictures of the MRI brain of the meningiomas showed a similar characteristic of having isointensity with the brain parenchyma on T1WI and T2WI, but most enhance with gadolinium, and also the “Dural tail sign” which occurs as a result of thickening and enhancement of the dura.

Management:

Available definitive treatment options for petrotentorial meningioma are conservative management, stereotactic radiation and surgery. Conservative treatment is generally for asymptomatic patients, where MRI scans of the brain will be repeated every 6-12 months to observe the tumor's progression. For symptomatic patients or large, invasive tumors, surgical resection is generally recommended. Since the patient presented with symptoms, surgical resection is the most preferable treatment. Surgical resection offers immediate resolution of mass effect and complete tumor removal. Stereotactic radiosurgery is also one of the treatment options, but generally considered as an adjunct treatment or when surgical resection is not achievable.

Surgical resection is the most preferable treatment in meningiomas, and is accepted as the treatment with the most favorable long-term outcome. However, a number of considerations such as age, expected survival rate, and location of the tumor is usually taken into consideration before advancing into removal. Total removal of the tumor is usually preferred, however in locations that are difficult to access or large tumors, partial resection is done and is followed by stereotactic radiosurgery methods for size reduction (D'Ambrosio and Bruce, 2003).

Post-operative surveillance is recommended for monitoring recurrence and progression of residual tumor. The most common recurrence occurs 1-3 years after surgical resection. Surveillance usually consists of interval imaging methods such as MRIs for tumor visualization. A recommended interval for follow up includes 3, 6 and 12 months after surgery, then 6-12 months for the first five years, then every 1-3 years onwards for typical meningiomas. Atypical meningiomas follow up recommendations is at 3-6 months after surgery for 3-5 years, then every 6-12 months onwards (NCCN Head and Neck Cancer, 2022). Simpson classification could be used to determine recurrent rate of the meningioma after surgical resection.

Simpson grade	Definition	Recurrence (%)
I	Complete tumor resection including dural attachment and abnormal bone	9
II	Complete tumor resection, coagulation of dural attachment	19
III	Complete tumor resection w/o resection of dural attachment or bone	29
IV	Subtotal tumor resection	44
V	Decompression with biopsy	

(Muthukumar et al., 1998)

Conclusion

Meningiomas are the most common primary central nervous system tumors and account for approximately one-third of all primary brain and spinal tumors. It occurs from the neoplastic changes of arachnoid cap cell or meningothelial cells and is associated with various genetic abnormalities. Common clinical manifestations of petrotentorial meningiomas include CN IV, V, VII, VIII dysfunction and local mass effects to adjacent structures. Diagnosis can be made with imaging modalities such as MRI brain with gadolinium and from tissue pathology. Treatment options include watchful waiting with interval imaging surveillance, stereotactic radiation and surgery depending on various patient factors as well as tumor factors.

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