



Cerebellopontine Angle Meningioma Presenting as Trigeminal Neuralgia in a Patient with Meningiomatosis: A Case Report

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ABSTRACT

Introduction: Trigeminal neuralgia (TN) is a paroxysmal neuropathic facial pain that is classified as classical, idiopathic or secondary. Space-occupying lesions account for approximately 4–6% of TN presentations, and meningioma is one of the most frequent culprits. The coexistence of TN with meningiomatosis—two or more spatially separate meningiomas—is uncommon, and in such patients the clinician must determine which lesion is responsible for the pain before rational treatment can be planned.

Case Illustration: A 51-year-old woman attended the neurology outpatient clinic of a regional general hospital in Gianyar, Bali, Indonesia, with a 2-year history of left-sided facial pain that had progressively worsened over the preceding year despite consultations at several hospitals. The pain was electric shock-like, predominantly periocular, lasted from seconds to 1 minute, and was triggered by light touch, wearing a face mask, chewing,

tooth-brushing and face-washing. She also reported rotatory dizziness aggravated by positional change with nausea and vomiting, which had improved at presentation. Examination revealed hyperaesthesia in the territories of the left ophthalmic (V1) and mandibular (V3) divisions with trigger points in the left periorbital and mandibular regions; cerebellar testing was normal. Contrast-enhanced magnetic resonance imaging with angiography (MRI-MRA) demonstrated two strongly enhancing extra-axial masses with dural tails: a left temporal/cerebellopontine angle (CPA) lesion measuring $4.14 \times 2.63 \times 4.40$ cm that encased the cavernous segment of the left internal carotid artery, extended to the posterior sphenoid sinus and compressed the left pons, and a right temporo-occipital lesion measuring $2.68 \times 3.86 \times 3.84$ cm, consistent with meningiomatosis. Laboratory investigations were not performed. The patient was diagnosed with secondary TN attributed to the left CPA meningioma and was treated with carbamazepine 200 mg three times daily, mecobalamin 500 µg twice daily and betahistine 12 mg twice daily.

Discussion: Strict clinicoradiological correlation identified the left CPA–petroclival component, not the contralateral temporo-occipital lesion, as the pain generator. Involvement of the porus trigeminus/Meckel’s cave and pontine root entry zone explains the paroxysmal V3 pain, whereas extension along the lateral wall of the cavernous sinus explains the unusual V1 predominance. Hyperaesthesia, V1 involvement and concomitant vestibular symptoms are red flags that should prompt MRI in every patient with TN. Sodium-channel blockers are appropriate first-line symptomatic therapy, but secondary TN is more often refractory; definitive control of tumour-related pain is achieved more reliably with microsurgical decompression than with tumour-targeted

radiosurgery, while the second, asymptomatic lesion may be kept under surveillance. HLA-B*15:02 carriage, which is prevalent in Indonesia, must be considered when carbamazepine is prescribed.

Conclusion: TN may be the index manifestation of meningiomatosis. Early contrast-enhanced MRI, lesion-by-lesion clinicoradiological correlation, pharmacogenomically safe symptomatic therapy and timely referral for multidisciplinary neurosurgical decision-making are essential to optimise outcomes, particularly in secondary-care settings.

Keywords: Secondary trigeminal neuralgia; Cerebellopontine angle; Petroclival meningioma; Multiple meningiomas (meningiomatosis); Carbamazepine

INTRODUCTION

Background

Trigeminal neuralgia (TN) is the prototypical neuropathic facial pain syndrome, characterised by recurrent, unilateral, brief, electric shock-like paroxysms confined to the distribution of one or more divisions of the trigeminal nerve, abrupt in onset and termination and typically provoked by innocuous stimuli. The contemporary nosology endorsed by the International Classification of Headache Disorders, 3rd edition (ICHD-3), and by the European Academy of Neurology (EAN) distinguishes classical TN (associated with neurovascular compression producing morphological changes of the trigeminal root), idiopathic TN (no identifiable cause) and secondary TN, which is attributed to an underlying neurological disease such as multiple sclerosis or a space-occupying lesion.¹⁻³

The disorder is more common than historically assumed. A recent systematic review and meta-regression encompassing more than 170 million individuals estimated a pooled global incidence of 25.33 per 100,000 person-years and a lifetime prevalence of 108.43 per 100,000 inhabitants, with a female predominance and preferential involvement of the maxillary and mandibular divisions over the ophthalmic division. Because the paroxysms may be triggered by speaking, eating or oral hygiene, TN exerts a disproportionate impact on nutrition, sleep, mood and occupational function.⁴⁻⁶

Demyelination of primary trigeminal afferents at, or close to, the root entry zone is regarded as the final common pathway for most forms of TN. In classical TN the insult is pulsatile vascular compression, whereas in secondary TN it may be produced by a demyelinating plaque or by direct compression, distortion or stretching of the nerve by a tumour. Importantly, no single clinical characteristic reliably excludes a secondary cause; for this reason, the EAN guideline recommends magnetic resonance imaging (MRI) as part of the routine work-up of every patient with TN.^{3,5,6}

Tumours account for a small but clinically decisive fraction of TN. In a single-centre series of 819 consecutive patients, 4.4% had a cerebellopontine angle (CPA) tumour, and in a larger cohort

of 3,242 patients presenting with TN symptoms, 5.1% had a tumour-related aetiology, with epidermoid cysts (31.7%), meningiomas (31.1%) and vestibular schwannomas (18.3%) being the leading causes. Notably, meningioma-related TN in that cohort showed a strong female predominance (86%) and a mean age at onset of 51.8 years.^{7,8}

Meningiomas are the most common primary intracranial neoplasm, representing 41.7% of all primary brain and other central nervous system tumours and 56.8% of non-malignant tumours in the most recent Central Brain Tumor Registry of the United States (CBTRUS) report, with a clear female preponderance. Under the fifth edition of the World Health Organization (WHO) classification of central nervous system tumours, meningioma is considered a single tumour type with multiple morphological subtypes and with grading criteria that increasingly integrate molecular information. The European Association of Neuro-Oncology (EANO) guideline emphasises that a provisional diagnosis is made by MRI, that asymptomatic lesions may be managed by a watch-and-scan strategy, and that symptomatic lesions are primarily treated surgically, with radiosurgery or fractionated radiotherapy as alternatives when resection is not feasible.^{9–11}

A subset of patients harbour two or more spatially separate synchronous or metachronous meningiomas, a condition termed multiple meningiomas or meningiomatosis. Reported frequencies range from 1% to 10% of meningioma patients; in a population-based Finnish series, 4% of patients with meningioma had meningiomatosis without neurofibromatosis type 2 (NF2), whereas 1% had multiple meningiomas associated with NF2. Meningiomatosis may be sporadic, familial or radiation-induced, and it poses particular diagnostic and therapeutic challenges because each lesion may have a different biology and natural history.^{12,13}

When TN occurs in a patient with multiple intracranial meningiomas, the clinician faces a specific question that does not arise with a solitary tumour: which lesion, if any, is responsible for the pain? This question is not academic. Microsurgical resection or decompression of a symptomatic petroclival or CPA meningioma relieves TN in approximately 82–92% of patients, whereas tumour-targeted stereotactic radiosurgery (SRS) achieves durable pain control in a

substantially smaller proportion, and an incorrect attribution of symptoms would expose the patient to the risks of skull base intervention without clinical benefit.^{14–16}

The present report describes a woman in whom left-sided TN involving the ophthalmic and mandibular divisions was the presenting manifestation of meningiomatosis comprising a left CPA–petroclival meningioma with cavernous sinus extension and a contralateral temporo-occipital meningioma. The case is reported in accordance with the CARE (CAse REport) guidelines.^{17,18}

Research Gap

Although tumour-related TN has been well characterised in surgical series and meta-analyses, the available evidence is dominated by solitary lesions treated in high-volume skull base centres, and the specific scenario of TN occurring in meningiomatosis is rarely addressed. In particular, the literature provides little guidance on (i) the lesion-by-lesion clinicoradiological reasoning required to identify the symptomatic tumour among multiple meningiomas; (ii) the anatomical explanation of ophthalmic-division involvement, which is distinctly uncommon in TN of any cause; (iii) the interpretation of concomitant vestibular symptoms in the same patient; and (iv) the initial management of such patients in secondary-care hospitals in low- and middle-income settings, where neurosurgical and radiosurgical resources are limited and where pharmacogenomic risks associated with carbamazepine, notably HLA-B*15:02 carriage, are highly relevant. Data from Indonesia and South-East Asia on this presentation are scarce.^{4,12,14,15}

Novelty

This report contributes (1) a documented instance of secondary TN as the index manifestation of bilateral, synchronous multiple meningiomas; (2) an explicit clinicoradiological correlation model that attributes the pain to the left CPA–petroclival lesion and explains the V1 plus V3 phenotype through combined involvement of the porus trigeminus/Meckel's cave, the lateral wall of the cavernous sinus and the pontine root entry zone; (3) an integrated interpretation of the accompanying positional vertigo; and (4) a practical, evidence-based management pathway adapted to an Indonesian regional hospital, incorporating pharmacogenomic safety, hormonal-exposure assessment and referral criteria.

Objectives

The general objective of this report is to describe a case of secondary TN caused by a CPA meningioma in a patient with meningiomatosis and to analyse the relationship between the neuralgic presentation and the imaging findings. The specific objectives are: (a) to present the clinical history, neurological findings, neuroimaging and initial management in detail; (b) to establish the diagnosis according to ICHD-3 and the Cruccu grading system; (c) to explain the anatomical and pathophysiological basis linking the left CPA–petroclival lesion to the left V1 and V3 neuralgia; and (d) to review the current evidence on the pharmacological and interventional management of tumour-related TN and multiple meningiomas.^{1,2}

Benefits

For clinical practice, this report reinforces the principle that MRI with contrast is indispensable in TN, highlights red flags that should raise suspicion of a secondary aetiology, and provides a structured approach to the patient with multiple meningiomas. For education, it offers a didactic example of neuroanatomical localisation in facial pain for general practitioners and residents. For health-service planning, it emphasises the need for referral pathways from regional hospitals to tertiary neurosurgical centres and for access to pharmacogenomic testing. For research, it adds to the limited regional literature and identifies questions suitable for multicentre registries.^{3,19}

Clinical Hypothesis

Because a single case cannot test a statistical hypothesis, the following working clinical hypothesis was formulated and examined through clinicoradiological correlation: the left-sided paroxysmal facial pain is secondary TN caused by direct compression and distortion of the left trigeminal nerve complex—at the level of the pontine root entry zone, the porus trigeminus/Meckel's cave and the lateral wall of the cavernous sinus—by the left CPA–petroclival component of meningiomatosis, rather than by the contralateral right temporo-occipital meningioma or by an incidental primary (classical or idiopathic) TN. The alternative explanation—coincident primary TN with an incidental tumour burden—was considered and is addressed in the Discussion.^{1,3}

CASE ILLUSTRATION

Patient Information

A 51-year-old woman presented to the neurology outpatient clinic of Sanjiwani Regional General Hospital (RSUD Sanjiwani), Gianyar, Bali, Indonesia. Personal identifiers have been removed in accordance with the CARE guidelines. The patient's age is recorded as 51 years in the clinical record and as 52 years on the MRI request.¹⁷

Chief Complaint and History of Present Illness

The chief complaint was pain on the left side of the face for 2 years. The pain was initially mild but had become progressively more severe during the preceding year. It was located predominantly in the region of the left eye and was described as an electric shock-like sensation. Each attack was brief, lasting from a few seconds up to approximately 1 minute. Attacks were provoked by light tactile stimuli, specifically wearing a face mask, chewing food, brushing the teeth and washing the face. The patient had consulted several hospitals for evaluation before presentation, but her complaints had not improved; details of the previous investigations and treatments were not available.

In addition, the patient reported rotatory dizziness (vertigo) that was aggravated by changes in position and was accompanied by nausea and vomiting. At the time of presentation, the vertigo had improved. There was no family history of similar complaints.

Physical and Neurological Examination

Blood pressure was 110/70 mmHg. Sensory examination demonstrated hyperaesthesia in the cutaneous territories of the left ophthalmic (V1) and mandibular (V3) divisions of the trigeminal nerve. Trigger points were identified in the left periorbital region and the mandibular area. Coordination was within normal limits: the finger-to-nose test showed no dysmetria, there was no dysdiadochokinesia, the heel-to-knee (heel-shin) test was normal, tandem gait was normal, and the rebound phenomenon was absent. The examination findings recorded at presentation are summarised in Table 1.

Table 1. Physical and neurological examination findings at presentation

Domain	Finding	Clinical interpretation
Blood pressure	110/70 mmHg	Normotensive
Trigeminal sensory function	Hyperaesthesia in the left ophthalmic (V1) and mandibular (V3) territories	Positive sensory sign; red flag for secondary TN
Trigger zones	Left periorbital region and mandibular area	Triggered paroxysms (clinically established TN)
Finger-to-nose test	No dysmetria	No appendicular ataxia of the upper limbs
Diadochokinesis	No dysdiadochokinesia	Normal
Heel-to-knee (heel–shin) test	Normal	No appendicular ataxia of the lower limbs
Tandem gait	Normal	No truncal/gait ataxia
Rebound phenomenon	Absent	Normal

TN, trigeminal neuralgia. Only findings documented in the clinical record are listed; corneal reflex, masseter strength, other cranial nerves, hearing and nystagmus were not recorded.

Neuroimaging

Brain MRI with MR angiography was performed using multiplanar T1-weighted, T2-weighted, fluid-attenuated inversion recovery (FLAIR), T2 fast-field-echo (T2FFE), diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) sequences before contrast administration, followed by three-dimensional time-of-flight (3D TOF) MRA and multiplanar fat-saturated T1-weighted sequences after intravenous gadolinium.

Two strongly enhancing extra-axial masses with a dural tail were demonstrated (Figures 1 and 2). The first, located in the left temporal region at the left CPA, measured $4.14 \times 2.63 \times 4.40$ cm; it covered (encased) the cavernous segment of the left internal carotid artery, extended to the posterior sphenoid sinus and compressed the left side of the pons. The second, located in the right temporo-

occipital region, measured $2.68 \times 3.86 \times 3.84$ cm. Using the ellipsoid (ABC/2) approximation, the estimated volumes are approximately 24 cm^3 and 20 cm^3 , respectively. The basal ganglia, thalami, internal capsules, ventricles, midbrain, medulla, cerebellum, sulci, basal cisterns, dural venous sinuses, sella, orbits, craniovertebral junction and bones were reported as normal.

The paranasal sinuses showed mild mucosal thickening of the bilateral maxillary, ethmoid and sphenoid sinuses, with hypertrophy of both inferior turbinates and deviation of the nasal septum to the right. MR angiography showed that the left internal carotid artery was encased as described, with a hypoplastic left vertebral artery interpreted as an anatomical variant; the remaining intracranial arteries were normal. The radiological impression was meningiomatosis. The complete findings are presented in Table 2.

Table 2. Contrast-enhanced brain MRI and MR angiography findings

Structure	Finding
Left temporal / left CPA	Strongly enhancing extra-axial mass with dural tail, $4.14 \times 2.63 \times 4.40$ cm ($\approx 24 \text{ cm}^3$, ABC/2); covers the cavernous segment of the left ICA; extends to the posterior sphenoid sinus; compresses the left pons
Right temporo-occipital	Strongly enhancing extra-axial mass with dural tail, $2.68 \times 3.86 \times 3.84$ cm ($\approx 20 \text{ cm}^3$, ABC/2)
Brain parenchyma	Basal ganglia, thalami, internal capsules, midbrain, medulla and cerebellum normal; ventricles, sulci and basal cisterns normal
Dural venous sinuses, sella, orbits	Normal
Paranasal sinuses and mastoid	Mild mucosal thickening of bilateral maxillary, ethmoid and sphenoid sinuses; hypertrophy of bilateral inferior turbinates; nasal septal deviation to the right
Craniovertebral junction and bones	Normal

MR angiography	Left ICA encased as above; right ICA, ACAs, AComA, MCAs, PCAs, PComAs and basilar artery normal; left vertebral artery hypoplastic (anatomical variant)
Radiological impression	All features suggestive of meningiomatosis

ABC/2, ellipsoid volume approximation calculated by the authors; ACA, anterior cerebral artery; AComA, anterior communicating artery; CPA, cerebellopontine angle; ICA, internal carotid artery; MCA, middle cerebral artery; PCA, posterior cerebral artery; PComA, posterior communicating artery.

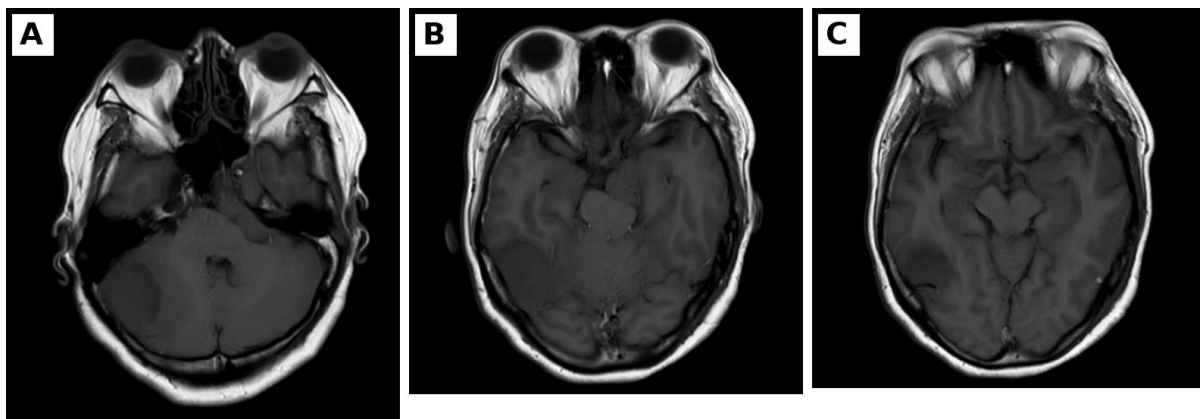


Figure 1. Axial pre-contrast T1-weighted images (radiological convention: the patient's left is on the image right). (A) Level of the posterior fossa: an isointense extra-axial mass occupies the left petroclival region/cerebellopontine angle adjacent to the left pons, and a second isointense extra-axial lesion is seen in the right posterior temporo-occipital region. (B) Suprasellar/midbrain level: the right temporo-occipital lesion and the parasellar extension of the left lesion. (C) Level of the lateral ventricles: the right temporo-occipital lesion.

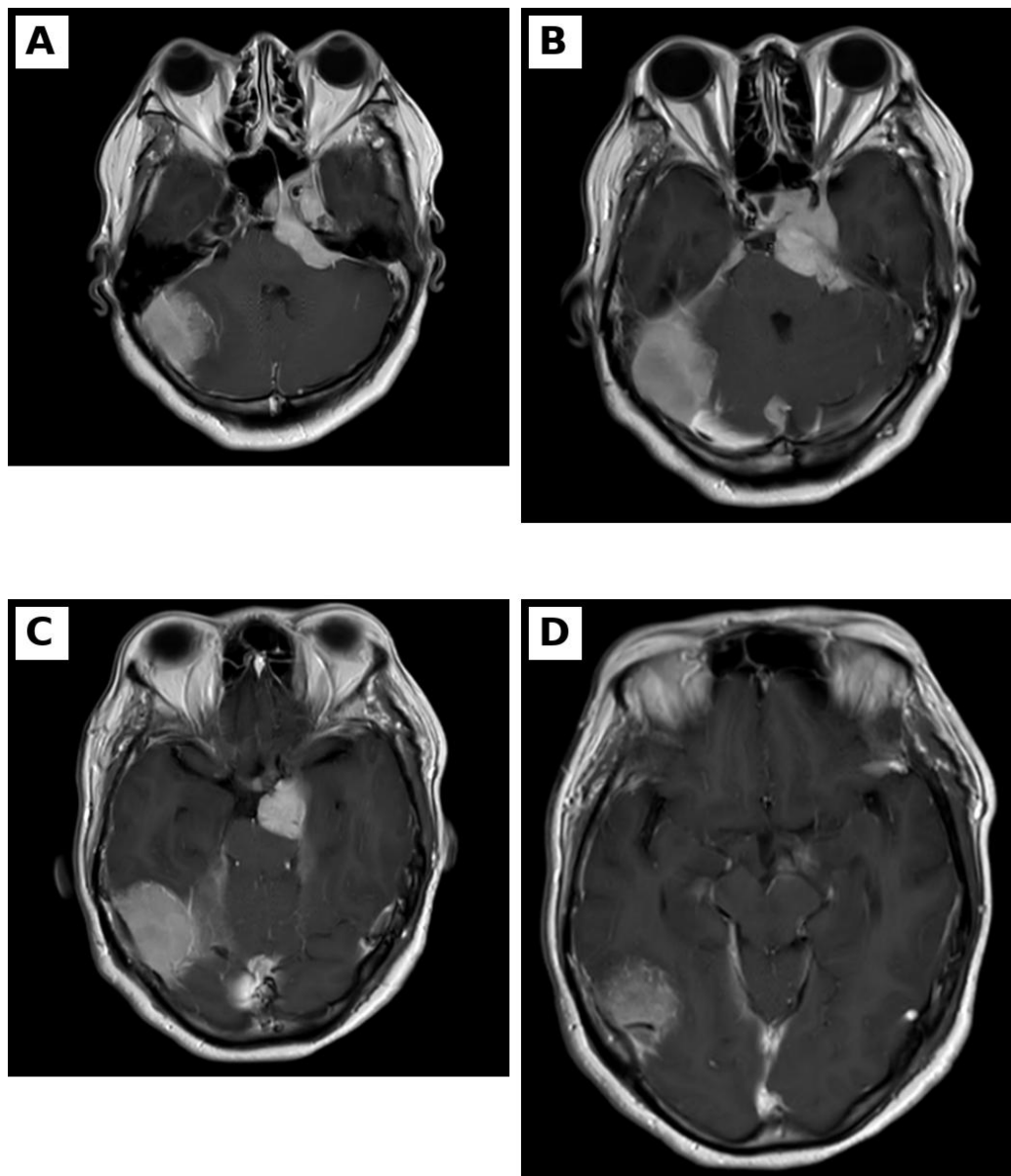


Figure 2. Axial gadolinium-enhanced fat-saturated T1-weighted images. (A, B) Strong, homogeneous enhancement of the left petroclival/cerebellopontine angle meningeioma extending anteriorly towards the left cavernous sinus and compressing the left side of the pons; the right posterior temporo-occipital meningeioma is also enhancing. (C) The left lesion at the parasellar level adjacent to the cavernous segment of the left internal carotid artery, and the right temporo-occipital

meningioma with dural attachment. (D) Superior extent of the right temporo-occipital meningioma. Both lesions showed a dural tail on the radiology report, consistent with meningiomatosis.

Diagnosis

The clinical features—recurrent, unilateral, paroxysmal, electric shock-like pain lasting seconds to 1 minute within trigeminal territories, precipitated by innocuous stimuli at defined trigger zones—fulfil the ICHD-3 criteria for TN. The demonstration of a space-occupying lesion in contact with the ipsilateral trigeminal pathway establishes the diagnosis of secondary TN attributed to a space-occupying lesion (ICHD-3 code 13.1.1.2.2) and, according to the grading system of Cruccu and colleagues, a definite neuropathic pain.^{1,2}

The working diagnoses were therefore: (1) secondary TN of the left ophthalmic and mandibular divisions, purely paroxysmal, caused by the left CPA (petroclival) meningioma; (2) meningiomatosis (multiple meningiomas) comprising the left CPA–petroclival lesion with cavernous sinus extension and pontine compression and a right temporo-occipital lesion, with presumptive diagnosis based on imaging and without histological confirmation; (3) positional vertigo with nausea and vomiting, improving; and (4) incidental findings of bilateral paranasal sinus mucosal thickening, bilateral inferior turbinate hypertrophy, rightward nasal septal deviation and left vertebral artery hypoplasia. Table 3 applies the diagnostic criteria to the present case.

Table 3. Application of diagnostic criteria to the present case^{1,2}

Criterion	Requirement	Present case
ICHD-3 13.1.1 A	Recurrent paroxysms of unilateral facial pain in the distribution(s) of one or more divisions of the trigeminal nerve, with no radiation beyond	Left-sided paroxysms in V1 (periocular) and V3 territories — fulfilled
ICHD-3 13.1.1 B	Pain lasting from a fraction of a second to 2 minutes; severe intensity; electric shock-like,	Seconds to 1 minute; electric shock-like; progressively severe — fulfilled

	shooting, stabbing or sharp	
ICHD-3 13.1.1 C	Precipitated by innocuous stimuli within the affected distribution	Triggered by light touch, mask, chewing, tooth-brushing, face-washing — fulfilled
ICHD-3 13.1.1.2.2	Space-occupying lesion demonstrated in contact with the affected trigeminal nerve	Left CPA–petroclival meningioma compressing the left pons and extending to the cavernous sinus — fulfilled
Cruccu grading	Possible → clinically established → definite (aetiology shown by imaging)	Definite secondary TN (definite neuropathic pain)

CPA, cerebellopontine angle; ICHD-3, International Classification of Headache Disorders, 3rd edition; TN, trigeminal neuralgia.

Therapeutic Intervention

The patient was treated with oral carbamazepine 200 mg three times daily (600 mg/day), oral mecobalamin 500 µg twice daily and oral betahistine 12 mg twice daily.

Timeline

The chronology of the illness and management is summarised in Table 4. Follow-up information after the index encounter (pain response, tolerability and the definitive management decision) was not available at the time this report was prepared.

Table 4. Timeline of the case

Time	Event
≈2 years before presentation	Onset of mild, paroxysmal left-sided facial pain
≈1 year before presentation	Progressive worsening of pain; consultations at several hospitals without improvement
Before presentation	Rotatory positional vertigo with nausea and vomiting

(date not recorded)	
Index visit, Neurology Outpatient Clinic, RSUD Sanjiwani	Clinical diagnosis of TN; hyperaesthesia in left V1 and V3; trigger points; normal cerebellar examination; vertigo improving
Work-up	Contrast-enhanced brain MRI-MRA: left CPA–petroclival and right temporo-occipital meningiomas (meningiomas); laboratory tests not performed
Treatment	Carbamazepine 200 mg three times daily, mecobalamin 500 µg twice daily, betahistine 12 mg twice daily
Follow-up	Not available at the time of writing

CPA, cerebellopontine angle; MRI-MRA, magnetic resonance imaging and angiography; TN, trigeminal neuralgia.

DISCUSSION

Overview

This case illustrates three clinically important principles. First, a TN phenotype that is otherwise typical may conceal a large skull base tumour, and MRI is therefore mandatory. Second, in a patient with multiple meningiomas, symptoms must be attributed lesion by lesion through careful neuroanatomical correlation. Third, symptomatic pharmacotherapy is only a bridge; definitive management of tumour-related TN is decided by the relationship between the tumour and the trigeminal nerve, the tumour's extension (particularly into the cavernous sinus) and the available expertise.^{3,5,14}

Diagnostic Reasoning: Secondary Rather Than Classical TN

The patient's attacks fulfilled every core criterion of TN: they were paroxysmal, unilateral, confined to trigeminal territories, electric in quality, lasted seconds to 1 minute and were triggered by innocuous stimuli. Such a typical phenotype does not exclude a secondary cause. The EAN panel concluded that no clinical characteristic can exclude secondary TN, and the Lancet Neurology

review similarly recommends MRI with dedicated sequences to detect secondary causes and to evaluate neurovascular contact.^{2,3,5}

Several features in this patient nevertheless function as red flags. The first is sensory abnormality: hyperaesthesia was documented in V1 and V3. In a prospective study of 158 patients with classical TN, sensory abnormalities were present in 29% of non-operated patients, whereas tumour-related TN has been associated with preoperative neuropathic deficits in 61.5% of patients compared with 24.2% in idiopathic TN. The second is ophthalmic-division predominance: in the same prospective series, isolated V1 involvement occurred in only 4% of patients, and population data confirm that V2 and V3 are affected far more often than V1. The third is the presence of concomitant non-trigeminal neurological symptoms, in this case positional vertigo with nausea and vomiting.^{4,20,21}

Age is less discriminating in meningioma-related TN than in epidermoid-related TN. Although patients with tumour-related TN are, on average, younger at onset than those with classical TN, the mean age at symptom onset in meningioma-related TN (51.8 years) overlaps considerably with that of classical TN (approximately 52–53 years). The patient's age at onset (approximately 49 years) and sex are therefore typical for meningioma-related TN, but would not by themselves have raised suspicion. This underscores the value of systematic imaging rather than selective imaging based on age.^{7,8,20}

Up to 10% of TN cases may be caused by a brain tumour, and TN may be the sole or dominant manifestation of an intracranial neoplasm. In a series of 718 patients admitted for brain tumours, 2.3% had TN, which was the main clinical symptom in 0.8%. The 2-year interval between symptom onset and diagnosis in the present patient, despite visits to several hospitals, is consistent with the diagnostic delay described in tumour-related TN and in CPA meningiomas generally.^{22–24}

The differential diagnosis considered is summarised in Table 5. Short-lasting unilateral neuralgiform headache attacks (SUNCT/SUNA) may involve V1 but are accompanied by prominent cranial autonomic features, which were not described. Rhinosinusitis-related facial pain, suggested by the mucosal thickening on MRI, typically produces continuous pressure-like pain rather than triggered, electric paroxysms. Painful post-traumatic or post-herpetic trigeminal

neuropathy lacked a compatible history. Multiple sclerosis-related TN was not supported by the MRI findings.^{2,6}

Table 5. Differential diagnosis

Diagnosis	Features for	Features against / status
Classical TN (neurovascular compression)	Typical paroxysmal, triggered phenotype ²	Large ipsilateral mass explains symptoms; high-resolution nerve sequences not available — coexisting conflict not excluded ^{25,26}
Idiopathic TN	Typical phenotype	Excluded: a causative lesion is present
TN secondary to multiple sclerosis	Secondary TN phenotype	No demyelinating lesions reported on MRI
Painful trigeminal neuropathy (post- traumatic, post- herpetic)	Sensory abnormality	Pain is paroxysmal and triggered, not continuous; no relevant history ²
SUNCT/SUNA	V1 involvement; brief attacks	No cranial autonomic features described; attacks triggered ²
Rhinosinusitis-related facial pain	Sinus mucosal thickening on MRI	Pain electric, triggered and paroxysmal rather than continuous pressure
Other CPA lesions (vestibular or trigeminal schwannoma, epidermoid cyst,	CPA location; TN presentation ⁸	Strong homogeneous enhancement, dural tail and multiplicity favour meningioma; histology required ^{27,28}

metastasis, lymphoma)		
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CPA, cerebellopontine angle; SUNCT/SUNA, short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing/with cranial autonomic symptoms; TN, trigeminal neuralgia.

Why the Left CPA–Petroclival Meningioma, and Not the Right Lesion, Causes the Pain

In meningiomas, lesion-by-lesion attribution is essential. The right temporo-occipital meningioma is contralateral to the painful side and supratentorial, remote from both trigeminal nerves; it has no plausible anatomical relationship with the left trigeminal pathway and is therefore regarded as clinically silent with respect to the facial pain. The left lesion, by contrast, occupies the left CPA, compresses the left pons and extends anteriorly to encase the cavernous segment of the left internal carotid artery and reach the posterior sphenoid sinus—a configuration characteristic of a petroclival meningioma with cavernous sinus extension. The proposed correlation is illustrated in Figure 3.^{11,12}

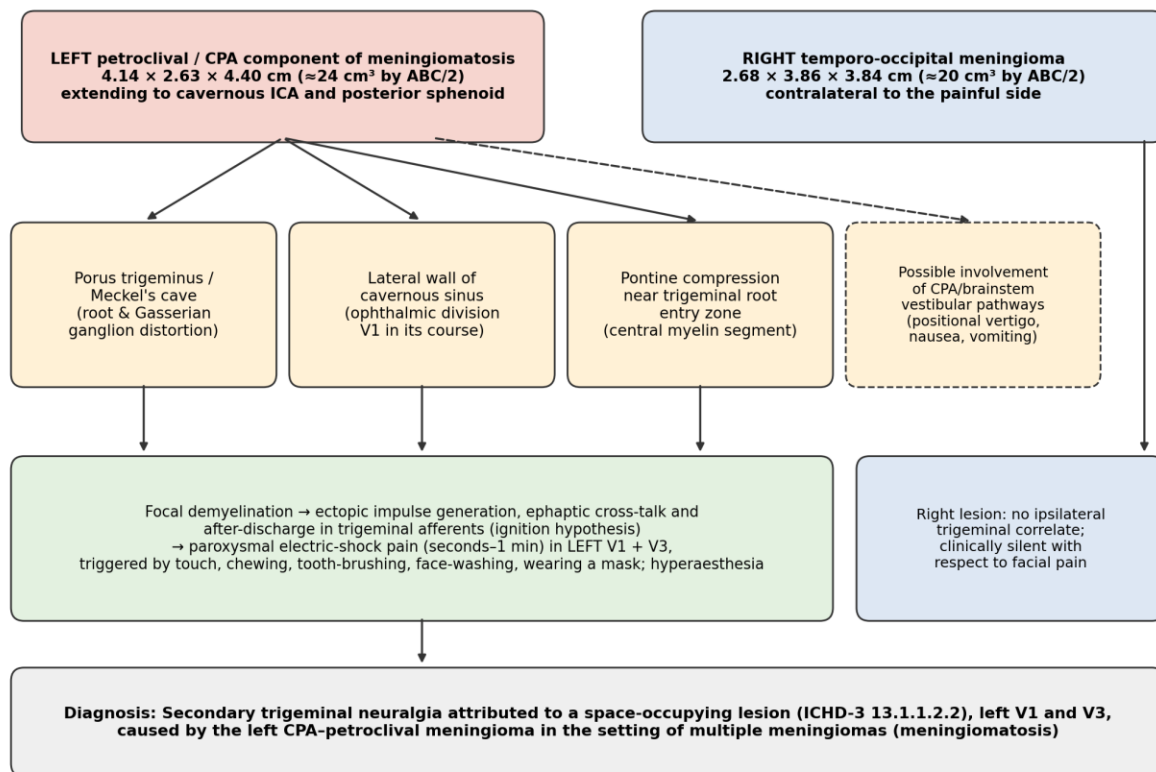


Figure 3. Proposed clinicoradiological correlation. The left cerebellopontine angle–petroclival meningioma is considered the pain generator through compression of the trigeminal nerve at the pontine root entry zone and porus trigeminus/Meckel’s cave (V3 component) and extension into the lateral wall of the cavernous sinus (V1 component). Involvement of vestibular pathways is a possible, unconfirmed explanation for the positional vertigo (dashed). The contralateral right temporo-occipital meningioma has no anatomical relationship with the left trigeminal pathway.

Meckel’s cave is a dural recess in the posteromedial middle cranial fossa that conducts the trigeminal nerve from the prepontine cistern to the cavernous sinus and houses the Gasserian ganglion and the proximal rootlets. Petroclival meningiomas frequently invade Meckel’s cave through the porus trigeminus, and tumours arising in or extending into this region produce TN in a high proportion of cases: in one surgical series of Meckel’s cave meningiomas, TN was the initial

symptom in 62.5% of patients, and in another, facial pain resolved after resection in all tumour types.²⁹⁻³²

The ophthalmic division is anatomically distinctive. Distal to the ganglion, V1 travels anteriorly within the lateral wall of the cavernous sinus before entering the orbit through the superior orbital fissure, whereas V2 and V3 leave the middle cranial fossa through the foramen rotundum and foramen ovale, respectively. The encasement of the cavernous internal carotid artery in this patient implies cavernous sinus involvement, providing a coherent anatomical explanation for the uncommon V1-predominant (periocular) pain, while compression of the ganglion and root at the porus trigeminus and along the pons explains the V3 component and trigger zone. Cavernous sinus invasion has been associated with poorer pain outcomes after microsurgery for petroclival meningioma-related TN, which is relevant to prognosis in the present case.^{29,30,32}

Surgical series have characterised the relationships between CPA tumours and the trigeminal root. The tumour may surround the nerve, compress and displace it, grow within it, compress it together with a vessel, or displace it towards a vessel; compression and displacement predominate in meningiomas. Additional arterial conflict, usually with the superior cerebellar artery, was found in 42.9% of patients in one series of tumour-related TN and in 16 of 52 patients in another, and meningioma-related TN tends more often than epidermoid-related TN to involve a responsible vessel. The absence of high-resolution heavily T2-weighted sequences in the present MRI means that a coexisting neurovascular conflict could not be assessed, a point of practical relevance for surgical planning.^{25,26,33}

Pathophysiology: From Compression to Paroxysmal Pain

Histopathological studies of trigeminal roots obtained at surgery have shown focal demyelination, dysmyelination, axonal loss and close apposition of demyelinated axons without intervening glial processes in the region of compression. This arrangement favours ectopic impulse generation and ephaptic (non-synaptic) transmission between adjacent fibres. Space-occupying masses in the posterior fossa are thought to act through the same mechanism, and relief after decompression is attributed to the separation of demyelinated axons and the reversal of conduction block.^{34,35}

The ignition hypothesis proposes that injured, hyperexcitable trigeminal afferents in the root or ganglion generate synchronised after-discharges in response to a trigger; activity spreads to neighbouring neurones and is then terminated by an intrinsic hyperpolarising process, producing a refractory period. This model accounts for the brief, explosive paroxysms, the triggering by light touch and the refractory intervals characteristic of TN, and it explains why sodium-channel blockers are effective. At the molecular level, altered expression and redistribution of voltage-gated sodium channels contribute to membrane hyperexcitability.^{36,37}

In this patient, the tumour could act at several points along the nerve simultaneously—the root entry zone (where central myelin predominates), the cisternal root, the porus trigeminus, the ganglion and the V1 segment in the cavernous sinus. A multisegmental compressive mechanism is consistent with the combination of paroxysmal pain and a positive sensory sign (hyperaesthesia) in two non-contiguous divisions. The attribution of the pain to direct compression rather than to chemical irritation is supported by surgical series in which relief followed tumour removal and nerve decompression.^{7,21,34}

Positional Vertigo in the Context of a CPA Meningioma

Audiovestibular symptoms are the most common manifestations of CPA meningiomas. In a multi-institutional cohort of 95 patients, hearing loss was present in 49.5%, ataxia in 42.1% and headache in 29.5%; in earlier series, vertigo or imbalance occurred in 19 of 32 patients, and trigeminal neuropathy in 16%. Vertigo may even be the sole presenting symptom of a CPA meningioma.^{24,38–40}

The positional character of the patient's vertigo raises two possibilities: a coincident peripheral positional vertigo, such as benign paroxysmal positional vertigo, or a central or retrocochlear vestibular disturbance related to the CPA mass with brainstem compression. Normal cerebellar testing does not exclude either possibility. Because nystagmus, positional manoeuvres, the head-impulse test and audiometry were not documented, the vertigo cannot be definitively attributed to the tumour; it is therefore presented as a possible association (Figure 3). Formal audiovestibular assessment is recommended both for aetiological clarification and as a baseline before any skull base intervention.^{38,39}

Betahistine was prescribed for symptomatic relief. A Cochrane review of 17 randomised trials found low-quality evidence that betahistine reduces vertigo symptoms compared with placebo (risk ratio 1.30, 95% confidence interval [CI] 1.05–1.60), with an adverse-event rate similar to placebo. Its benefit in vertigo of central or tumour-related origin has not been established, and treatment should be reviewed once the vertigo has resolved.⁴¹

Meningiomatosis: Aetiology, Genetics and Hormonal Considerations

Multiple meningiomas constitute a distinct clinical entity with sporadic, familial and radiation-induced forms. Two principal pathogenic theories have been proposed: independent origin of each tumour through separate genetic events and a monoclonal origin with subarachnoid spread. Molecular data support both. In a series of spatially separate sporadic meningiomas, individual tumours from the same patient harboured distinct driver mutations; conversely, whole-exome sequencing has shown monoclonal origin in five of six patients, with branching evolution that produced different WHO grades within the same individual. The practical implication is that the behaviour of one lesion does not reliably predict that of another, and each tumour should be managed on its own merits.^{12,42,43}

The possibility of an underlying tumour-predisposition syndrome must be addressed. Neurofibromatosis type 2 (NF2-related schwannomatosis) is the most important hereditary cause of multiple meningiomas; meningiomas occur in approximately half of affected patients, and loss of NF2 function is found in up to 60% of sporadic meningiomas. Somatic mosaicism may produce a mild phenotype such as isolated multiple meningiomas. Patients with apparently isolated multiple meningiomas should therefore undergo a careful family history, skin examination, slit-lamp examination of the lens, audiovestibular testing and craniospinal MRI. The negative family history in this patient is reassuring but does not exclude mosaicism.^{44,45}

The prognosis of sporadic multiple meningiomas is not necessarily worse than that of solitary tumours. In a matched case–control study of 34 patients with sporadic multiple meningiomas (91.2% women), intervention rates, recurrence-free survival and overall survival did not differ from those of patients with solitary meningiomas, and the authors recommended that management be directed towards the symptomatic or high-risk lesion. This principle supports focusing definitive

treatment on the left CPA–petroclival tumour while keeping the right lesion under surveillance.^{11,12,46}

Hormonal factors deserve attention in a woman of this age. Progesterone receptors are expressed in approximately 76% of meningiomas and are more frequent in women and in skull base tumours. A French national case–control study demonstrated an excess risk of meningioma with prolonged use of several progestogens, including injectable medroxyprogesterone acetate (odds ratio 5.55), and a 2026 meta-analysis reported a pooled odds ratio of 2.68 for depot medroxyprogesterone acetate, with tumours predominantly located at the anterior and middle skull base and regression reported after withdrawal of some agents. Because injectable progestogen contraception is widely used in Indonesia, a detailed contraceptive and hormone-therapy history should be obtained, and high-risk progestogens should be avoided where alternatives exist.^{47–49}

Neuroimaging Interpretation and Its Limitations

The diagnosis of meningioma in this case is radiological. Homogeneous strong enhancement of extra-axial dural-based masses with a dural tail is highly suggestive. A systematic review of 2,159 tumours found a dural tail sign in 74.2% of meningiomas, representing tumour infiltration in 72.2% of sampled tails, with a sensitivity of 93.0% but a specificity of only 29.0% for tumour cells. The dural tail also occurs in other lesions, including schwannomas, lymphoma, metastases and granulomatous disease, and meningioma mimics accounted for 7.2% of radiologically diagnosed meningiomas in one surgical series. Definitive diagnosis, grading and molecular characterisation therefore require histology.^{11,27,28,50}

The MRI protocol, while adequate to demonstrate the tumours, did not include high-resolution three-dimensional heavily T2-weighted sequences (for example, constructive interference in steady state or fast imaging employing steady-state acquisition) that best depict the cisternal trigeminal nerve and its relationship with adjacent vessels. Such sequences, preferably at 3 T, are recommended in TN work-up and would help to delineate the nerve, Meckel’s cave invasion and any coexisting neurovascular conflict before surgery.^{3,19,51}

The incidental findings merit brief comment. Left vertebral artery hypoplasia is a common anatomical variant and does not explain the symptoms. The paranasal sinus mucosal thickening,

turbinate hypertrophy and septal deviation warrant otorhinolaryngological review; however, the triggered, electric, paroxysmal pain pattern is not consistent with rhinosinusitis-related facial pain.²

Pharmacological Management

Carbamazepine and oxcarbazepine are the drugs of first choice for long-term treatment of TN, and the EAN recommends that secondary TN be treated according to the same principles as primary TN. Carbamazepine reduces neuronal hyperexcitability through use-dependent blockade of voltage-gated sodium channels. The dose used in this patient (600 mg/day) lies within the usual therapeutic range, and titration according to response and tolerability is appropriate. Lamotrigine, gabapentin, pregabalin, baclofen and botulinum toxin type A may be used alone or as add-on therapy, and intravenous fosphenytoin or lidocaine can be used for acute exacerbations; intravenous fosphenytoin has been reported to control a TN crisis due to a petroclival meningioma.^{3,52,53}

Secondary TN is more difficult to treat. In a real-world cohort of 354 patients, the initial response rates to carbamazepine and oxcarbazepine were 88.3% and 90.9%, but 27% of patients with secondary TN were refractory compared with 6% of patients with classical TN; side effects occurred more frequently with carbamazepine (43.6%) than with oxcarbazepine (30.3%), and discontinuation was more frequent in secondary and idiopathic forms. Close monitoring and early consideration of interventional options are therefore justified.⁵⁴

Pharmacogenomic safety is a specific concern in Indonesian patients. The HLA-B*15:02 allele is strongly associated with carbamazepine-induced Stevens–Johnson syndrome and toxic epidermal necrolysis (SJS/TEN), with a pooled odds ratio of 79.84 in a meta-analysis and an even higher estimate among Malaysians. The association has been confirmed in patients receiving carbamazepine for neuropathic pain, in whom SJS/TEN developed at a mean of approximately 11 days after initiation, and prospective screening in Taiwan eliminated carbamazepine-induced SJS/TEN among allele-negative patients. HLA-B*15:02 is the most frequent HLA-B allele in the Indonesian Malay population (15.5%). Although universal screening did not meet the Indonesian cost-effectiveness threshold in a model-based analysis, genotyping should be performed when available; where it is not, patients should be counselled to stop the drug and seek care immediately if rash, mucosal lesions or fever occur, particularly during the first 2 months.^{55–59}

Baseline and follow-up laboratory monitoring is recommended with carbamazepine, including complete blood count, serum sodium and liver function tests, because of the risks of haematological toxicity, hyponatraemia and hepatotoxicity. Laboratory tests were not performed in this patient; obtaining them early in follow-up is advisable.^{3,52}

Mecobalamin was prescribed as an adjunct. Systematic reviews suggest some benefit of vitamin B12 in post-herpetic neuralgia (level II evidence) and painful peripheral neuropathy (level III evidence), and a meta-analysis of 15 trials found improved global clinical efficacy with mecobalamin, particularly in combination therapy, without a significant effect on pain scores. There is no direct evidence for its efficacy in TN, and it should be regarded as a low-risk adjunct rather than a disease-modifying therapy.^{60,61}

Definitive Management of the Symptomatic Lesion

For tumour-related TN, the key evidence compares microsurgical resection and nerve decompression with tumour-targeted radiosurgery. A meta-analysis of 32 studies comprising 662 patients with CPA tumour-related TN reported pain relief in 92%, postoperative facial numbness in 18% and recurrence in 19%, with surgical resection superior to SRS for both pain relief and recurrence. A systematic review of 517 patients found improvement in 92.2% after surgery compared with 79.1% after tumour-targeted radiosurgery, with a higher recurrence rate after radiosurgery (26.5%). The principal evidence is summarised in Table 6.^{15,16}

Table 6. Selected evidence on tumour-related trigeminal neuralgia

Study	Design / population	Principal findings
Zhang et al., 2020 ⁷	Single centre; 819 TN patients	36 (4.4%) CPA tumour-related; earlier onset than classical TN; excellent outcome in 80.6% after surgery
Segura-Lozano et al., 2026 ⁸	Single centre; 3,242 TN patients	164 (5.1%) tumour-related; meningiomas 31.1%; 86% of meningioma cases in women; mean onset 51.8 years

Wei et al., 2017 ²¹	39 tumour-related vs 360 idiopathic TN	Neuropathic deficits 61.5% vs 24.2%; total removal predicted excellent long-term outcome
Penchev et al., 2025 ¹⁵	Meta-analysis; 32 studies, 662 patients (CPA tumours)	Pain relief 92%; numbness 18%; recurrence 19%; resection superior to SRS
Nugroho et al., 2023 ¹⁶	Systematic review; 517 patients	Improvement 92.2% after surgery vs 79.1% after tumour-targeted SRS; recurrence 26.5% after SRS
Hallak et al., 2023 ¹⁴	Meta-analysis; 138 petroclival meningioma patients	Pain resolution 82% after resection vs 31% after SRS
Byun et al., 2023 ⁶²	Meta-analysis; petroclival meningioma	Improvement 89% vs 37%; medication-free relief 90.7% vs 34.5% (microsurgery vs radiosurgery)
Park et al., 2020 ⁶³	70 small petroclival meningiomas with TN	BNI I–II in 91% after microsurgery vs 37% after GKRS
Byeon et al., 2024 ³⁰	28 petroclival meningiomas with TN	Relief without medication 82.1%; cavernous sinus invasion associated with poor pain outcome
Jiang et al., 2022 ⁶⁴	15 CPA tumour patients unfit for craniotomy	Percutaneous balloon compression: immediate relief in 14/15; facial numbness in 14

BNI, Barrow Neurological Institute pain score; CPA, cerebellopontine angle; GKRS, Gamma Knife radiosurgery; SRS, stereotactic radiosurgery; TN, trigeminal neuralgia.

The evidence is particularly consistent for petroclival meningiomas. A meta-analysis of 138 patients showed pain resolution in 82% after resection versus 31% after SRS; another meta-analysis

reported improvement in 89% versus 37% and medication-free pain relief in 90.7% versus 34.5%. In small petroclival meningiomas with TN, medication-free relief was achieved in 91% after microsurgery compared with 37% after Gamma Knife radiosurgery, and radiosurgery provided effective tumour control while TN persisted to varying degrees in most patients. Opening the porus trigeminus to remove tumour from Meckel's cave has been emphasised, with 82.1% of patients relieved without medication.^{14,30,62,63,65}

Several nuances are relevant to this patient. First, the tumour is large (approximately 24 cm³) and encases the cavernous internal carotid artery; cavernous sinus invasion limits radical resection and predicts poorer pain outcomes. Second, a coexisting arterial conflict should be sought and treated by microvascular decompression during tumour surgery, because inspection of the entire nerve root after tumour removal is considered key to operative success. Third, a strategy of resecting the tumour while deliberately sparing the portion involving the trigeminal ganglion has achieved pain freedom in 83.3% of patients with non-acoustic CPA tumours, which may reduce morbidity when complete resection is hazardous.^{25,30,32,66}

When craniotomy is unsuitable, alternatives exist. Radiosurgery provides durable tumour control: in a multicentre series of 4,565 patients with benign meningiomas, the control rate was 92.5% with 5- and 10-year progression-free survival of 95.2% and 88.6%, and a meta-analysis of cavernous sinus meningiomas reported 10-year progression-free survival of 84.9%. Simultaneous radiosurgery to the tumour and the trigeminal nerve has produced pain freedom in small series, and percutaneous balloon compression achieved immediate pain relief in 14 of 15 patients with CPA tumour-related TN in whom craniotomy was not feasible, at the cost of facial numbness in most.^{64,67–69}

For the right temporo-occipital meningioma, which is asymptomatic, the EANO guideline supports a watch-and-scan strategy with serial MRI, reserving treatment for growth, symptoms or high-risk features. For asymptomatic skull base meningiomas, radiosurgery has been associated with better local control than active surveillance, but the right lesion is supratentorial, and the decision should be individualised by a multidisciplinary team. International consensus emphasises

individualised, multidisciplinary decision-making that integrates tumour location, growth, symptoms, histology and patient preference.^{11,70,71}

Recommended Management Pathway for This Patient

On the basis of the evidence reviewed, the following pathway is proposed for patients presenting similarly in a regional hospital setting. These recommendations reflect the authors' interpretation of the literature and were not all implemented in the present case.^{3,11,71}

- Symptom control: continue carbamazepine with gradual titration; record pain intensity (numerical rating scale) and the Barrow Neurological Institute pain score at each visit; consider oxcarbazepine or add-on therapy if response or tolerability is inadequate.
- Safety: HLA-B*15:02 genotyping where available; counselling about cutaneous reactions; complete blood count, serum sodium and liver function tests at baseline and during follow-up.
- Completion of the neurological assessment: full cranial nerve examination including corneal reflex and masseter strength, otoscopy, pure-tone audiometry, examination for nystagmus, positional manoeuvres and the head-impulse test.
- Imaging refinement: high-resolution three-dimensional heavily T2-weighted sequences of the trigeminal nerve; craniospinal MRI and ophthalmological slit-lamp examination to screen for NF2-related schwannomatosis.
- Hormonal history: document contraceptive and hormone-therapy exposure, and avoid high-risk progestogens.
- Referral: early referral to a tertiary neurosurgical centre for multidisciplinary discussion of microsurgical decompression with or without microvascular decompression, or radiosurgery when surgery is unsuitable; histological confirmation when tissue is obtained.
- Surveillance: serial MRI of the asymptomatic right temporo-occipital meningioma; otorhinolaryngological review of the sinonasal findings.

Relevance to Secondary-Care Settings in Indonesia

In many regional hospitals, patients with facial pain are first managed empirically, and access to MRI, skull base neurosurgery and radiosurgery may be limited or delayed. This case, in which

the diagnosis was established by a regional neurology service after 2 years of symptoms, demonstrates that contrast-enhanced MRI at the secondary-care level can fundamentally change the diagnostic category and the management trajectory. The evidence that pain outcomes are better after microsurgery than after tumour-targeted radiosurgery, and that outcomes are poorer with cavernous sinus invasion, supports early referral rather than prolonged escalation of medication.^{3,15,30}

Strengths and Limitations

The strengths of this report are its detailed description of the pain phenotype and trigger zones, the availability of contrast-enhanced MRI with MRA, and the explicit clinico-radiological correlation in a patient with multiple lesions. Its limitations must be acknowledged. Histological confirmation was not available, and meningioma is a radiological diagnosis. Laboratory investigations, pain-intensity scores, a complete cranial nerve examination, audio-vestibular testing and high-resolution trigeminal sequences were not documented. Details of prior treatments, hormonal exposure and follow-up outcomes were unavailable. As a single case, the report cannot establish causality or generalisability; the conclusions are based on anatomical plausibility and consistency with published series.^{17,18}

CONCLUSION AND RECOMMENDATIONS

Conclusion

This report describes a woman with secondary TN of the left ophthalmic and mandibular divisions as the presenting manifestation of meningiomatosis. Clinico-radiological correlation attributes the pain to the left CPA–petroclival meningioma through compression of the trigeminal nerve at the pontine root entry zone and porus trigeminus/Meckel's cave, with V1 involvement explained by cavernous sinus extension. The contralateral temporo-occipital meningioma is clinically silent with respect to the facial pain. The working clinical hypothesis is therefore supported, although histological confirmation and follow-up are required.

Recommendations

- Contrast-enhanced MRI should be performed in every patient with TN, because a typical phenotype does not exclude a secondary cause; sensory signs, V1 involvement and additional cranial nerve or vestibular symptoms are red flags.

- In patients with multiple meningiomas, symptoms should be attributed lesion by lesion before treatment decisions are made; asymptomatic lesions can generally be kept under MRI surveillance.
- Carbamazepine or oxcarbazepine remains the first-line symptomatic therapy, but HLA-B*15:02 status, laboratory monitoring and the higher refractoriness of secondary TN should be considered.
- Patients with tumour-related TN should be referred early for multidisciplinary neurosurgical evaluation, because microsurgical decompression provides superior pain outcomes compared with tumour-targeted radiosurgery.
- Screening for NF2-related schwannomatosis and assessment of exposure to high-risk progestogens are recommended in women with multiple meningiomas.
- Regional referral pathways and registries for tumour-related TN in Indonesia should be developed to improve access to specialist care and to generate local outcome data.

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