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New-onset cervical pain followed by ophthalmoplegia revealing cavernous sinus meningioma

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ABSTRACT

Background: Headache and facial pain may occur in patients with meningioma, but their diagnostic significance is often underestimated when pain resembles a primary headache phenotype or occurs in a patient with a long-standing history of tension-type headache. Cavernous sinus meningiomas are particularly relevant in headache medicine because of their proximity to the oculomotor nerves, trigeminal branches, internal carotid artery, and parasellar structures.

Case Report: A 62-year-old woman with a 30-year history of tension-type headache presented with a 1-month history of severe cervical pain, initially interpreted in the context of her previous headache disorder and treated with amitriptyline at a dose of 10 mg/day, administered at bedtime for 2 weeks, without clinical benefit. She subsequently developed acute vertical diplopia. Neurological examination revealed mild left ptosis, left ocular deviation, and impaired elevation of the left eye, consistent with partial left oculomotor nerve palsy. She also reported a transient cold sensation in the left maxillary territory. Contrast-enhanced brain MRI showed a homogeneously enhancing extra-axial lesion in the left cavernous sinus, extending toward the superior orbital fissure and lying close to the intracavernous segment of the internal carotid artery, compatible with cavernous sinus meningioma. No previous brain MRI or CT was available for comparison.

Discussion: The association of a new, refractory cervical pain with acute ocular motor dysfunction and ipsilateral trigeminal sensory symptoms supported a secondary pain disorder rather than a simple fluctuation of the patient's previous tension-type headache. Cavernous sinus meningioma should be considered in the differential diagnosis of new-onset cervical pain followed by ophthalmoplegia, particularly when oculomotor nerve palsy is accompanied by trigeminal sensory symptoms.

Conclusions: This case highlights a clinically relevant diagnostic pitfall in headache practice: a new or changed pain phenotype in a patient with a known primary headache disorder should prompt reassessment for secondary causes, especially when cranial nerve signs emerge.

Key words: secondary headache; ophthalmoplegia; cavernous sinus; meningioma; oculomotor nerve palsy.

Introduction

Headache may occur in patients with meningioma and may resemble a primary headache disorder. In a prospective series of 58 patients with meningioma, Schankin *et al.* identified meningioma-associated headache in 40% of cases; the phenotype was tension-type-like in more than half and migraine-like in approximately one fifth, underlining that tumor-related headache may resemble primary headache disorders. (1) This overlap is clinically relevant because a pre-existing headache diagnosis may delay recognition of a secondary pain syndrome.

Cavernous sinus meningiomas represent a distinct skull-base subgroup because of their anatomical relationship with cranial nerves III, IV, V1, V2, and VI, the internal carotid artery, and the peri-carotid sympathetic plexus. (2-4) Their presentation may include visual impairment, ocular motor dysfunction, diplopia, ptosis, facial sensory changes, retro-orbital pain, headache, or facial pain. (2-5) Cavernous sinus syndrome is diagnostically challenging because inflammatory, vascular, infectious, traumatic, and neoplastic causes may produce overlapping clinical and radiological features. (2)

We report a patient with a long-standing tension-type headache who developed a new severe cervical pain followed by acute partial oculomotor nerve palsy and transient ipsilateral V2 dysesthesia, leading to the diagnosis of cavernous sinus meningioma. The case is presented as a secondary headache, with emphasis on the red flags relevant to headache practice.

Case Report

A 62-year-old woman with a 30-year history of tension-type headache presented with a 1-month history of severe cervical pain. Her usual headache was bilateral, predominantly located in the temporal region, with a tightening quality. The episodes were not accompanied by nausea, vomiting, photophobia, phonophobia, cranial autonomic symptoms, or focal neurological deficits. The new cervical pain had initially been interpreted in the context of her previous headache history and treated with 10 mg of amitriptyline for 2 weeks, without clinical benefit. The pain was predominantly left/posterior cervical, continuous, rated 7/10, and not associated with fever, trauma, photophobia, nausea, or autonomic cranial symptoms. Neck movements were limited, and the pain was worsened by flexion, extension, or rotation of the neck. Cutaneous allodynia was absent. No cervical trauma, fever, radicular symptoms, or limb weakness was reported.

It was clinically relevant because of its recent onset, severity, persistence, and refractoriness to a treatment prescribed for the presumed primary headache background.

After approximately 1 month, the patient developed acute vertical diplopia. Neurological examination showed mild left pto-

sis associated with lateral deviation of the left eye and impaired elevation of the left eye, consistent with partial left oculomotor nerve palsy (**Video**). The patient also reported a transient cold sensation in the left maxillary territory, suggesting involvement of trigeminal sensory pathways. Pupils were equal and reactive to light. Visual acuity and visual fields were normal. No additional deficits involving cranial nerves were identified. Facial sensation was normal on examination. Facial strength, hearing, palatal elevation, and tongue movements were normal. Strength, muscle tone, deep tendon reflexes, plantar responses, coordination, gait, and sensory examination of the limbs were unremarkable. No meningeal signs were present. Because of the acute onset of partial third cranial nerve palsy, vascular imaging was performed to exclude an aneurysmal or other vascular cause. CT angiography showed no intracranial aneurysm, carotid-cavernous fistula, or other relevant vascular abnormality.

Contrast-enhanced brain MRI demonstrated a homogeneously enhancing extra-axial lesion in the left cavernous sinus, extending toward the superior orbital fissure and in proximity to the intracavernous segment of the internal carotid artery. The imaging appearance was compatible with cavernous sinus meningioma (**Figure 1**). A concise clinical timeline summarizing the patient's previous headache history, onset and evolution of the cervical pain, treatment with amitriptyline, onset of diplopia, neurological examination, and imaging findings is presented in **Figure 2**.

The combination of a new refractory cervical pain phenotype, acute partial oculomotor palsy, ipsilateral trigeminal sensory symptoms, and the identification of a cavernous sinus lesion raised concern for a secondary pain disorder rather than a simple exacerbation of the patient's previous tension-type headache.

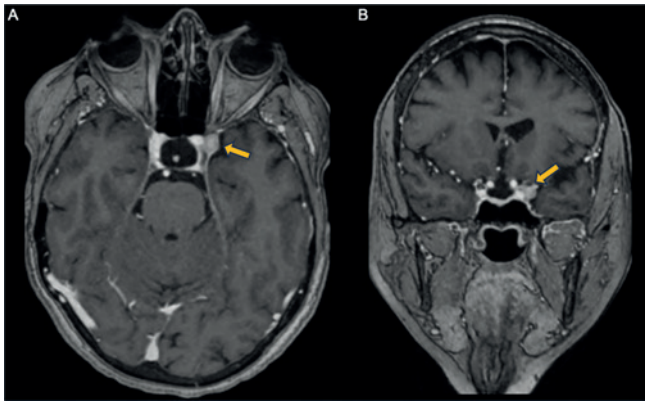

Figure 1. Brain MRI. Axial (A) and coronal (B) post-contrast T1-weighted images show an extra-axial lesion with homogeneous enhancement in the left cavernous sinus (arrows), extending toward the superior orbital fissure and in close proximity to the intracavernous segment of the internal carotid artery.

Discussion

This case illustrates a diagnostic anchoring error that is common in headache practice: attributing a new pain phenotype to a pre-existing primary headache disorder. The patient had a long-standing history of tension-type headache, but the clinical scenario changed when she developed a new, severe, persistent cervical pain followed by acute ocular motor dysfunction. According to the International Classification of Headache Disorders (3rd edition) principles, a new headache or substantially changed headache occurring in temporal association with a disorder capable of causing headache should be evaluated as a possible secondary headache, even if its phenotype resembles a primary headache disorder. (6)

The most relevant clinical issue in this case was the change from the patient's long-standing headache pattern to a new, severe, persistent, and treatment-refractory cervical pain. Several SNNOOP10[1] warning signs were present: i) the onset of a new pain phenotype after age 50 years, ii) a marked change

[1] SNNOOP10 refers to a mnemonic/list of red and orange flags suggestive of secondary headache, including systemic symptoms, neoplasm, neurologic deficit, sudden or abrupt onset, older age at onset, pattern change, positional headache, precipitated by sneezing/coughing or exercise, papilledema, progressive headache, pregnancy or puerperium, painful eye with autonomic features, post-traumatic onset, pathology of the immune system, and painkiller overuse or new headache.

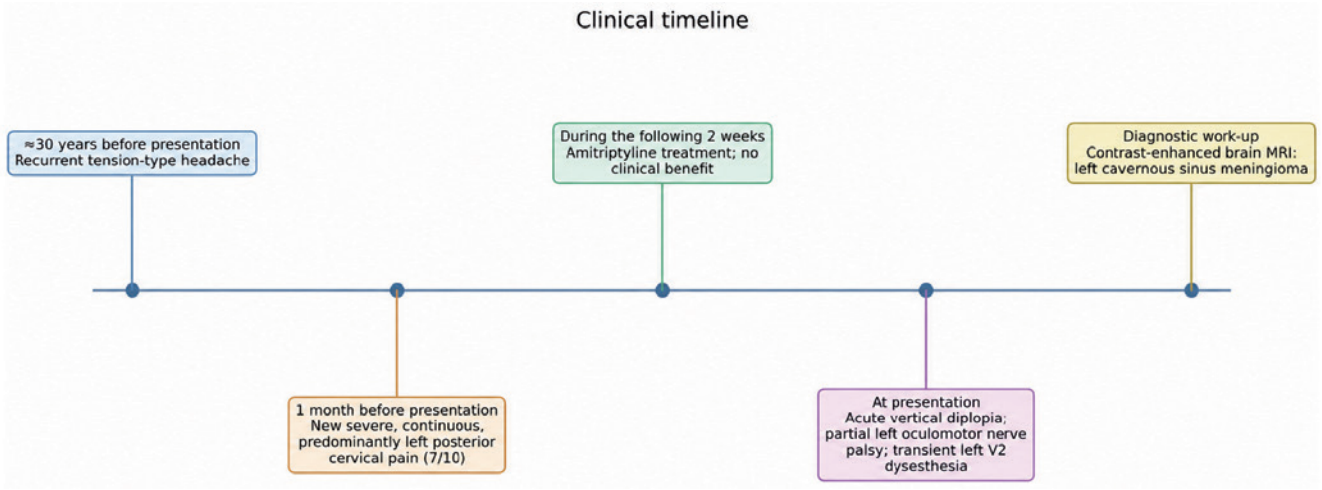

Figure 2. Clinical timeline of the patient's headache history, onset of cervical pain, treatment, development of ophthalmoplegia, and diagnostic workup.

Table 1. Main differential diagnoses of ophthalmoplegia associated with cranial, facial, orbital, or cervical pain.

Diagnostic category	Examples	Distinguishing clinical or imaging features
Neoplastic	Cavernous sinus meningioma, schwannoma, metastasis, pituitary region lesion	Progressive cranial neuropathies; enhancing cavernous sinus or parasellar mass
Vascular	Posterior communicating artery aneurysm, intracavernous carotid aneurysm, carotid-cavernous fistula	Acute onset; possible pupillary involvement; vascular abnormality on CTA/MRA
Thrombotic/infectious	Cavernous sinus thrombosis, invasive sinusitis, basal meningitis	Fever or systemic inflammation; orbital signs; venous thrombosis or meningeal enhancement
Inflammatory	Tolosa-Hunt syndrome, idiopathic orbital inflammation, granulomatous disease, vasculitis	Painful ophthalmoplegia; inflammatory enhancement; exclusion of structural, vascular, and infectious causes
Microvascular	Diabetic or hypertensive ocular motor neuropathy	Usually isolated ocular motor palsy; often pupil-sparing; vascular risk factors
Neuropathic	Recurrent painful ophthalmoplegic neuropathy	Recurrent attacks; typically with headache preceding ophthalmoplegia; diagnosis of exclusion

CTA, computed tomography angiography; MRA, magnetic resonance angiography.

from the previous headache pattern, iii) progressive persistence over approximately 1 month, and iv) the subsequent emergence of focal neurological signs. In particular, acute diplopia, partial oculomotor nerve palsy, and an ipsilateral trigeminal sensory symptom represented major indications for urgent neuroimaging. (7)

The case therefore illustrates the importance of reassessing a presumed primary headache diagnosis when the phenotype changes or focal neurological abnormalities emerge.

The anatomical localization explains the clinical picture. The cavernous sinus contains or is closely related to cranial nerves III, IV, V1, V2, and VI, as well as the internal carotid artery and its sympathetic plexus. (2-4) Cavernous sinus syndrome commonly includes ophthalmoplegia, ptosis, trigeminal sensory loss, ocular signs, Horner syndrome, headache, retro-orbital pain, and facial pain. (2) In this case, involvement of the left oculomotor nerve likely accounted for ptosis, ocular deviation, impaired elevation, and vertical diplopia. The transient cold sensation in the left maxillary territory was anatomically consistent with irritation or compression of trigeminal sensory pathways, particularly V2.

The differential diagnosis of ophthalmoplegia in patients presenting with a new pain syndrome is broad and should remain open until appropriate imaging and laboratory work-up are completed. Causes include cavernous sinus meningioma, schwannoma, metastatic disease, pituitary-region lesions, aneurysm of the intracavernous or posterior communicating artery, carotid-cavernous fistula, cavernous sinus thrombosis, Tolosa-Hunt syndrome, inflammatory pseudotumor, vasculitis, basal meningitis, diabetic ischemic ocular motor neuropathy, and recurrent painful ophthalmoplegic neuropathy (**Table 1**). (2,3,8) Contrast-enhanced MRI is central in the evaluation of cavernous sinus lesions and may show the typical features of meningioma, including an extra-axial lesion with intense enhancement and possible relationship with the cavernous internal carotid artery. (2,8,9)

The cervical predominance of pain is the most unusual element of the case. We performed a focused literature search for reports of cavernous sinus meningiomas presenting with cervical, occipital, or posterior cranio-cervical pain. Previous reports predominantly describe headache, retro-orbital pain, facial pain, or trigeminal symptoms as manifestations of cavernous sinus meningiomas. (5,10-12) This pain may persist even after tumor-directed treatment. (4,12) However, cervical pain as an initial or leading symptom is less typical. (3) In our patient, the cervical pain may represent referred cranio-cervical nociception in the setting of a parasellar/cavernous sinus lesion, although causality cannot be definitively proven without longitudinal pain data after treatment or radiological follow-up.

Management of cavernous sinus meningiomas is individualized and should involve a multidisciplinary team. The European Association of Neurosurgical Societies skull base consensus recommends initial clinical, ophthalmological, endocrinological, and radiological assessment, with management involving skull-base neurosurgeons or neuro-radiosurgeons, radiation oncologists, radiologists, ophthalmologists, and endocrinologists. (3)

A limitation of this report is that the causal relationship between the cervical pain and the cavernous sinus lesion cannot be definitively established in the absence of longitudinal pain assessment after tumor-directed treatment. Nevertheless, the temporal association between a new refractory pain phenotype, acute ocular motor dysfunction, ipsilateral trigeminal sensory symptoms, and the identification of a cavernous sinus lesion supports the clinical relevance of considering secondary causes in similar presentations.

Conclusions

A new severe cervical pain in a patient with long-standing tension-type headache should not automatically be interpreted as part of the previous headache disorder. The emergence of diplopia, ptosis, ocular motor deficit, or trigeminal sensory symptoms should prompt urgent evaluation for secondary causes, including cavernous sinus lesions. Cavernous sinus meningioma is an uncommon but important cause of ophthalmoplegia associated with a new pain syndrome and may enter the differential diagnosis of secondary headache or craniofacial pain.

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Online supplementary material:

Video. *Mild left ptosis with leftward deviation and impaired elevation of the left eye, consistent with partial left oculomotor nerve palsy. The patient is instructed to look to the right and then to the left and is asked whether she experiences diplopia. She is subsequently asked to look downward and upward, reporting diplopia on upward gaze. Ocular convergence is then assessed.*

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Conflict of interest: the authors have no conflict of interest to declare.

Ethics approval and consent to participate: no ethical committee approval was required for this case report by the Department, because this article does not contain any studies with human participants or animals. Informed consent was obtained from the patient included in this study.

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