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Cavernous Sinus Meningioma Causing Bilateral Cavernous Sinus Syndrome and Profound Visual Impairment: A Case Report

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ABSTRACT

Background: Cavernous sinus syndrome (CSS) is an uncommon but potentially life- and sight-threatening neurological condition, most often presenting as a combination of multiple cranial nerve palsies. One of its primary neoplastic causes, cavernous sinus meningioma (CSM), can lead to compressive optic neuropathy and profound visual loss.

Case Report: A 46-year-old female presented with a gradual bilateral decline in visual acuity, accompanied by intense headaches and left facial asymmetry. Clinical evaluation showed ophthalmoplegia, ptosis, decreased facial sensation in the V1–V2 regions, abnormal fundus findings, and significant color vision loss. MRI revealed bilateral cavernous sinus lesions encasing the internal carotid arteries, consistent with meningioma. The patient underwent subtotal surgical excision, with residual tumor remaining within the cavernous sinus. Postoperatively, visual function remained markedly reduced. The case was further complicated by high myopia with degenerative changes and multifocal toxoplasmic chorioretinitis.

Conclusion: This report highlights the complexity of diagnosing and managing CSS caused by bilateral CSM, particularly when coexisting with other ocular pathologies. Early recognition, detailed neuroimaging, and multidisciplinary management focused on preserving neurological and visual function are crucial to achieving optimal outcomes in such rare cases.

KEYWORDS: Cavernous sinus syndrome, cavernous sinus meningioma, cranial nerve palsy, toxoplasmic chorioretinitis, high myopia.

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INTRODUCTION

The cavernous sinus (CS) consists of a pair of valveless venous spaces located bilaterally beside the sella turcica at the skull base. It extends forward to the anterior clinoid process and superior orbital fissure, and backward to the petrous portion of the temporal bone. As part of an extensive cranial venous network, it maintains multiple interconnections with both intracranial and extracranial venous channels. The spheno-parietal sinus and the superior and inferior ophthalmic veins contribute to venous inflow, while outflow occurs through the superior and inferior petrosal sinuses. The cavernous segment of the internal carotid artery (ICA) traverses its medial aspect, surrounded by a sympathetic perivascular plexus. The oculomotor (III), trochlear (IV), ophthalmic (V1), and maxillary (V2) nerves course through the lateral dural wall in a superior-to-inferior arrangement, while the abducens nerve (VI) runs adjacent to the ICA within the venous lumen, making it the only cranial nerve to pass directly through the sinus cavity[1].

Cavernous sinus syndrome (CSS) represents a rare but clinically important condition that may compromise both vision and vital neurological function. It is characterized by the simultaneous involvement of several cranial nerves—commonly III, IV, V1, V2, and VI—and may manifest with proptosis, conjunctival congestion, periorbital pain, or sensory loss across the forehead and midface. Dysfunction of the oculomotor and trochlear nerves leads to ophthalmoplegia and diplopia, while trigeminal involvement produces facial numbness or paresthesia. The onset may be gradual or acute, depending on whether the etiology is neoplastic, vascular, infectious, inflammatory, or traumatic. Because of its broad differential and overlapping features, CSS often presents significant diagnostic difficulty[2].

Magnetic resonance imaging (MRI) is the preferred imaging modality for evaluating cavernous sinus pathology due to its superior soft-tissue resolution compared with computed tomography (CT). Neoplastic lesions are among the most frequent causes and include meningioma, pituitary adenoma, schwannoma, lymphoma, metastases, and perineural tumor extension, along with direct invasion from nasopharyngeal carcinoma. However, radiologic findings alone may not always distinguish inflammatory from infectious lesions, underscoring the need for careful clinical–radiologic correlation[1].

A meningioma involving the CS can exert compressive or infiltrative effects on the optic nerve, leading to compressive optic neuropathy (CON) manifested as visual loss or field defects due to mechanical or ischemic damage. Although surgical excision is generally recommended when vision is threatened, complete resection is technically difficult owing to the intimate neurovascular relationships in this region[3–4]. Thus, detailed understanding of the anatomy, pathophysiology, and clinical spectrum of CS lesions

is vital for accurate diagnosis and appropriate management.

CASE REPORT

A 46-year-old woman presented with a one-year history of progressively worsening headaches and bilateral visual decline, more severe in the left eye. She also reported left-sided facial asymmetry and paresthesia (Figure 1). Examination revealed left upper eyelid ptosis and mild dysarthria. She had a history of high myopia (-12.00 D bilaterally) since childhood and had used oral hormonal contraceptives for five years, discontinued in 2021.

Figure 1. Clinical features of left facial palsy in the patient

Ophthalmologic evaluation showed visual acuity of 1/60 in the right eye and 1/300 in the left. Color vision was markedly reduced in both eyes. Mild left proptosis (18–105–22 mm) and restricted ocular motility were observed: -2 limitation in superotemporal, temporal, and inferotemporal gaze, and -1 limitation in upward gaze bilaterally. The left eye exhibited ptosis but no relative afferent pupillary defect (Figure 2).

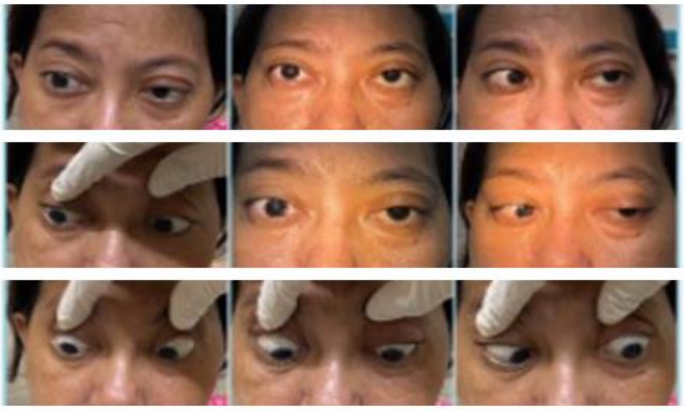

Figure 2. Nine gaze picture of the patient

Fundus examination revealed pale optic discs with indistinct margins. Multifocal chorioretinal atrophy, chorioretinal scarring, and choroidal thinning were seen in both eyes (Figure 3). Optical coherence tomography (OCT) showed significant RNFL thinning in the superior and inferior quadrants bilaterally, with localized macular thinning at 5–10 o'clock in the right eye and 2–4 o'clock in the left. Ganglion cell analysis confirmed thinning in the superotemporal and inferotemporal regions. Visual field testing (HFA) demonstrated bilateral central scotomas, more pronounced in the right eye (Figures 4–5).

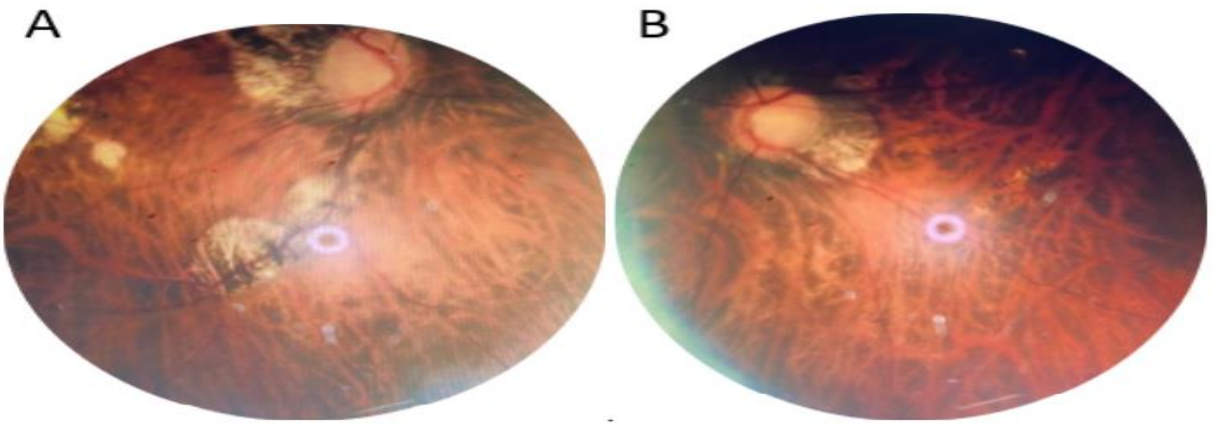

Figure 3. Posterior segment of (A) right eye and (B) left eye, showed fine border and slight pale colour in optic disc, multifocal chorioretinal atrophy, chorioretinal scar and choroidal thinning in retina of both eyes

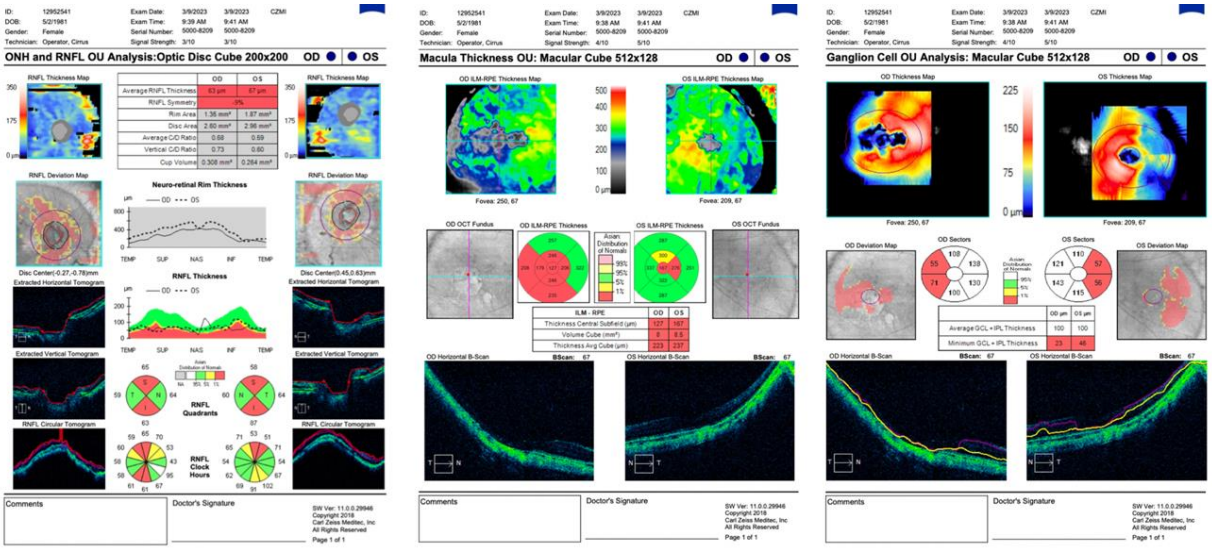

Figure 4. OCT examination in the revealed (A) significant thinning of the RNFL in the superior and inferior quadrants in both eyes, (B) Macula analysis found thinning in the area at 5-10 o'clock in the right eye and 2-4 o'clock in the left eye, (C) Ganglion cell analysis found thinning in the superotemporal and inferotemporal quadrant in both eyes.

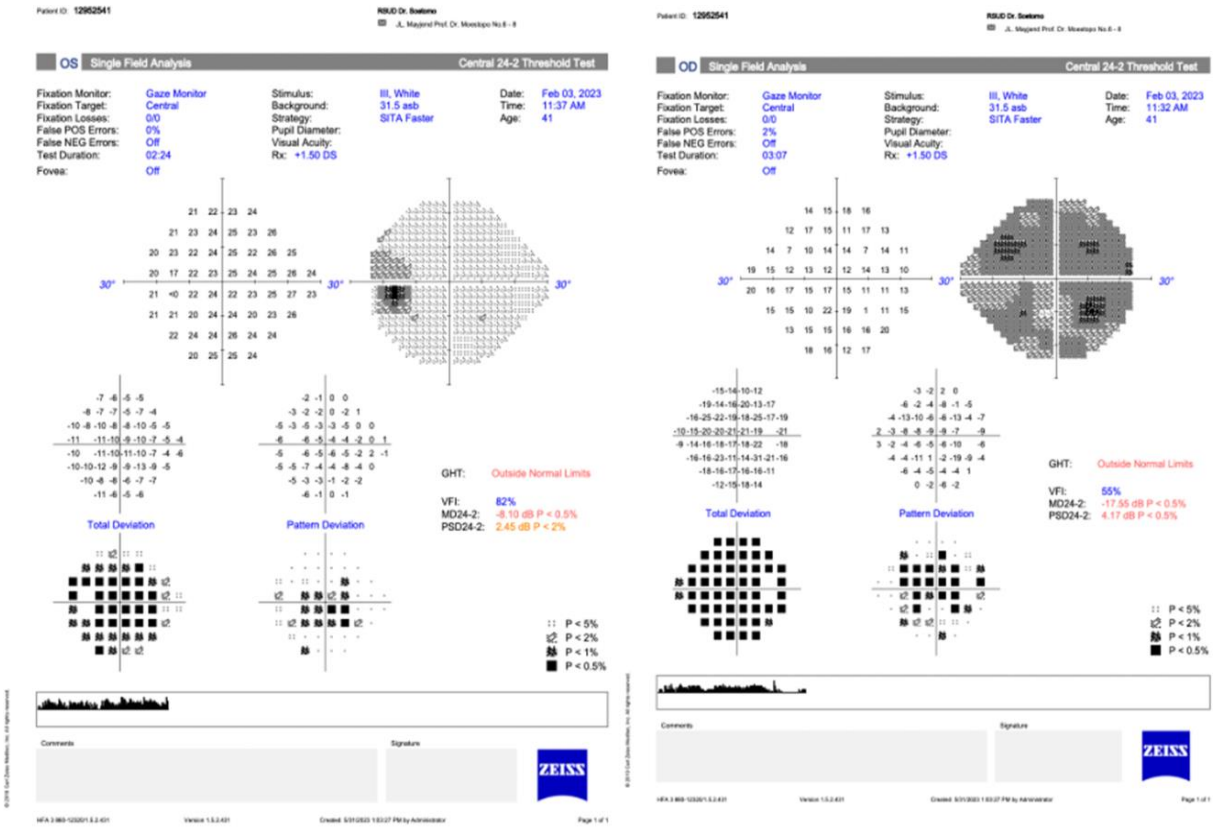

Figure 5. HFA revealed central scotoma in both eyes, larger in the right eye than in the left eye.

Neurological evaluation indicated involvement of ***CN II, III, IV, V, VI, and VII***, producing decreased vision, dyschromatopsia, central scotoma, ophthalmoplegia, hypoesthesia in the V1–V2 territories, and lower motor neuron facial palsy. Serologic testing revealed ***reactive IgG and IgM for Toxoplasma gondii*** and ***reactive IgG for rubella***. MRI of the brain and orbits several months prior showed a solid lesion (~2.3 × 4.6 × 1.3 cm) in the cavernous sinus extending into the clivus, consistent with meningioma, accompanied by paranasal sinusitis and mastoiditis (Figure 6). A follow-up MRI revealed bilateral CS masses with en plaque extension and ICA encasement, indicating progression (Figure 7).

A diagnosis of cavernous sinus syndrome secondary to cavernous sinus meningioma, with compressive optic neuropathy, pathologic myopia, and toxoplasmic chorioretinitis, was made. The patient underwent craniotomy with partial tumor excision and osteoplasty several weeks later. Residual tumor remained within the sinus. At postoperative review follow-up a few weeks later, visual acuity was 2/60 in the right eye and no light perception in the left. The patient reported complete left-eye blindness for three weeks. Ocular motility remained restricted, and therapy included citicoline 500 mg twice daily, neurotropic

supplementation, and folic acid once daily. Follow-up MRI and continued observation were planned by the neurosurgical team.

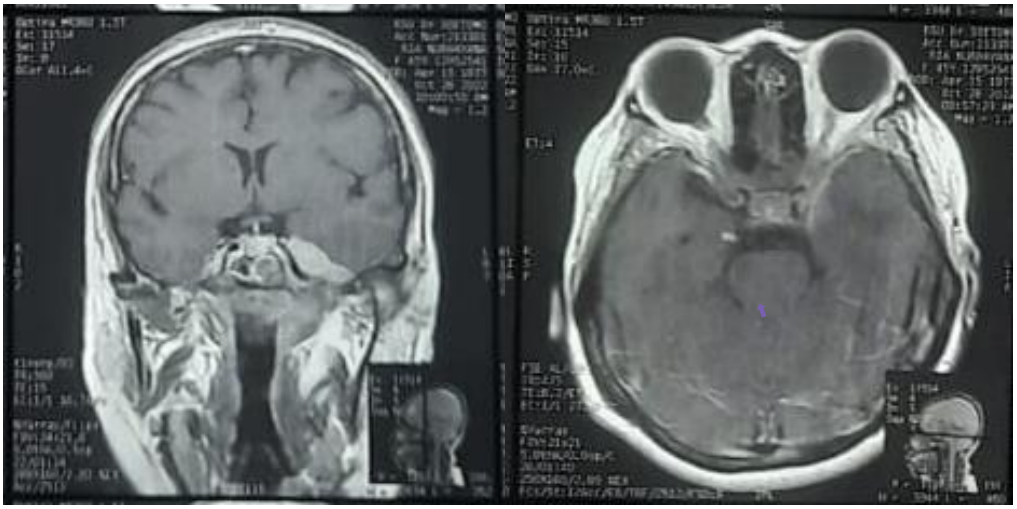

Figure 6. Brain MRI with and without contrast from the initial evaluation

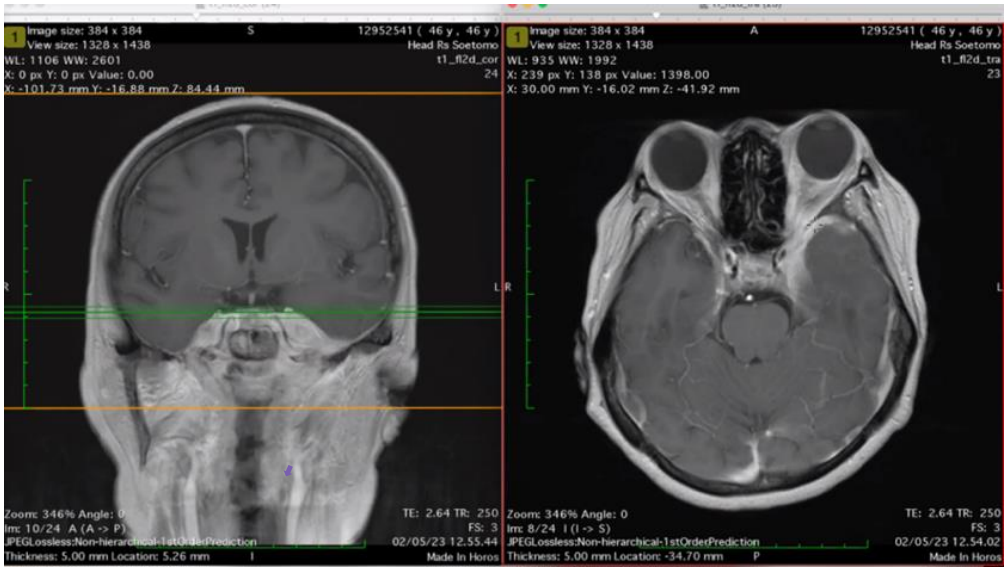

Figure 7. Follow-up brain MRI with contrast showing disease progression

DISCUSSION

The cavernous sinus is a complex venous structure located on both sides of the pituitary fossa, containing the ICA and several cranial nerves (III, IV, V1, V2, and VI). Lesions affecting this region may compromise these neurovascular structures, producing the characteristic constellation of deficits known as cavernous sinus syndrome (CSS)[5–6]. Patients typically present with ophthalmoplegia, ptosis, sensory loss over the face, and occasionally Horner's syndrome due to sympathetic fiber involvement. Combined abducens nerve palsy and Horner's syndrome constitute Parkinson's sign. Because of the confined anatomy, even small lesions can lead to pronounced neurological impairment[9].

CSS can produce variable symptoms including headache, diplopia, and facial numbness in the V1–V2 territories. Oculomotor, trochlear, and abducens involvement results in restricted eye movement, while trigeminal deficits lead to facial hypoesthesia. Disruption of sympathetic fibers causes ptosis and miosis, whereas parasympathetic impairment may cause pupil dilation[9–10]. Compressive optic neuropathy (CON) develops when the optic nerve is affected, manifesting as visual field defects, dyschromatopsia, and optic pallor[6,10–11]. Complete involvement of all cranial nerves, as observed in this case, is exceptionally rare; Fernández et al. reported total ophthalmoplegia in only 17% of CSS cases[11].

Cavernous sinus meningioma (CSM) is the predominant primary neoplasm of this region and a leading cause of CSS. Originating from arachnoid cap cells of the sinus dura or adjacent structures such as the sphenoid wing or orbit, it accounts for roughly 40% of CS tumors, with a female predominance likely linked to hormonal factors[7,14–15]. Although WHO grade I meningiomas are histologically benign, their proximity to the ICA and cranial nerves renders them clinically significant. Symptoms usually develop slowly, including headache, diplopia, and ptosis, with visual decline arising from optic nerve compression. The oculomotor nerve is most frequently affected, followed by abducens and trigeminal branches[2]. Tumor enlargement can result in ICA encasement, increasing the risk of ischemic complications[3].

MRI typically shows a homogeneously enhancing, dural-based mass that is isointense to gray matter on both T1- and T2-weighted images, often with a dural tail or ICA encasement—features that help differentiate it from pituitary adenoma[1]. CT imaging may demonstrate calcification or bony hyperostosis, further supporting the diagnosis[1,4]. The patient's imaging findings of bilateral, en plaque meningiomas encasing both ICAs fit this pattern. Histologically, meningiomas display considerable variability, including meningothelial, transitional, and fibroblastic subtypes. According to the World Health Organization (WHO), they are graded as benign (I), atypical (II), or anaplastic (III). This patient's tumor corresponded to a fibroblastic meningioma (grade I), composed of elongated spindle cells arranged in fascicles separated by collagen fibers, potentially mimicking schwannoma on frozen section[4].

Treatment depends on tumor size, extent, and neurological involvement. Complete resection is rarely feasible due to the high risk of neurovascular injury, so subtotal resection aimed at preserving function is usually performed[16]. Stereotactic radiosurgery (SRS) or fractionated radiotherapy is often used for postoperative control, achieving long-term stabilization in over 90% of cases[17]. Observation may suffice for small, asymptomatic lesions. The therapeutic objective is tumor control with preservation of vision and cranial nerve function, prioritizing quality of life over total resection[14–17].

Compressive optic neuropathy (CON) in CSM results from mechanical distortion or vascular compromise of the optic nerve. Persistent compression causes axonal loss and demyelination, leading to irreversible visual decline. The chance of visual recovery depends on duration and severity of compression—early intervention yields better outcomes, while chronic compression leads to permanent deficits[5,10]. Optimal management requires multidisciplinary collaboration among neurosurgeons, neurologists, and ophthalmologists. Continuous MRI surveillance is vital to monitor for recurrence or progression[15,18]. In this case, subtotal resection with preserved neurological function was the most appropriate choice given the bilateral tumor involvement.

Bilateral CSM are exceedingly rare, representing fewer than 2% of all meningioma cases. Their presence raises the possibility of multicentric origin or underlying hormonal or genetic predisposition, which further complicates both diagnosis and management.

CONCLUSION

Cavernous sinus meningioma can produce cavernous sinus syndrome through simultaneous involvement of multiple cranial nerves (III, IV, V1, V2, VI). Early and accurate diagnosis is essential given its potential to threaten both vision and life. Recognition of its characteristic clinical features, prompt neuroimaging, and multidisciplinary care are vital for improving prognosis and functional outcomes.

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