



Visual Field Recovery in Craniopharygioma: A Case of Reversible Hemianopia

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Abstract

Craniopharyngiomas are benign sellar and suprasellar tumors that frequently cause visual field defects due to compression of the optic chiasm, which are usually considered permanent. We report a rare case of reversible visual field loss in a 29-year-old woman with a partially calcified craniopharyngioma. The patient presented with left temporal hemianopia on Humphrey visual field testing, despite normal visual acuity in both eyes. Neuroimaging revealed an old, partially calcified sellar and suprasellar mass consistent with craniopharyngioma. Review of prior medical records showed a similar episode of right temporal hemianopia two years earlier, which had resolved spontaneously. This case demonstrates that visual field defects associated with craniopharyngioma may be reversible, possibly due to reduced mass effect from tumor regression or calcification. Awareness of this potential for recovery is important when evaluating visual prognosis and planning long-term follow-up in patients with craniopharyngioma.

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Introduction

Craniopharyngiomas are rare, benign epithelial tumors arising from remnants of Rathke's pouch and are typically located in the sellar and suprasellar region, accounting for approximately 2–5% of intracranial tumors [1]. Despite their benign histology, these tumors are associated with significant morbidity because of their proximity to critical neurovascular structures, particularly the optic nerves and optic chiasm [2].

Visual impairment is one of the most common presenting features of craniopharyngioma and may include reduced visual acuity, visual field defects, and optic atrophy, most frequently manifesting as temporal hemianopia due to chiasmal compression [3]. Visual field defects are usually considered permanent, especially in cases of chronic compression associated with optic disc pallor [4].

Visual recovery in patients with craniopharyngioma is uncommon and is most often reported following surgical decompression or treatment-related tumor regression [5]. Spontaneous improvement in visual fields is rarely described in the literature. Proposed mechanisms for visual recovery include reduction of mass effect, reversible conduction block of optic nerve fibers, and neuroplastic adaptation within the visual pathway [6].

Case Presentation

A 29-year-old woman presented with a complaint of defective vision in her left eye. She described a subjective reduction in the temporal visual field that had developed gradually. There was no associated eye pain, redness, headache, vomiting, or other neurological symptoms. Her medical history was unremarkable, and she was not on any long-term medi-

cations.

On examination, unaided visual acuity was 20/20 in both eyes. Pupillary reactions were normal, with no relative afferent pupillary defect. Anterior segment examination was unremarkable in both eyes. Fundus examination revealed temporal pallor of the optic discs bilaterally, while the macula and retinal vasculature appeared normal.

Humphrey Visual Field (HVF) test demonstrated a left temporal hemianopia (Figure 1). Given the presence of visual field loss with optic disc pallor, neuroimaging was arranged. CT (head) revealed an old, partially calcified mass in the sellar and suprasellar region, with radiological features consistent with a craniopharyngioma.

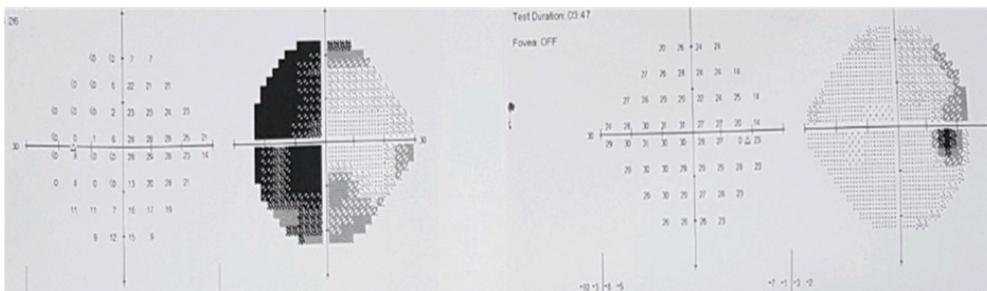


Figure 1: Currently Left Hemianopia

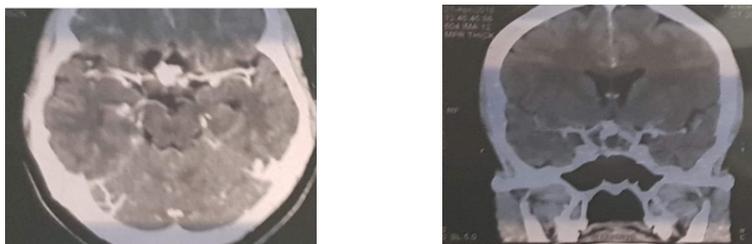


Figure 2: CT (Head) Shows Calcified Craniopharyngioma

Further review of the patient's past ocular history revealed a similar episode two years earlier, during which she had presented with defective vision in the right eye. Documentation from that time showed a right temporal hemianopia on Humphrey visual field testing (Figure 3). The visual field defect had subsequently improved without any surgical or medical intervention.

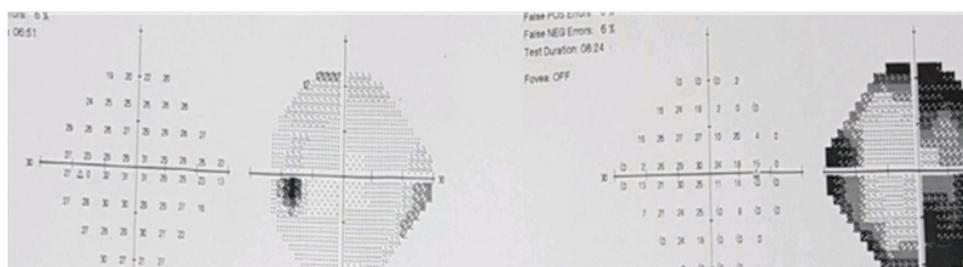


Figure 3: Previous Right Hemianopia

Based on the clinical findings, visual field defects, and neuroimaging results, a diagnosis of craniopharyngioma with sequential, reversible hemianopia was made. The patient was counseled regarding the condition and advised regular ophthalmic and neurological follow-up to monitor visual function and tumor status.

Discussion

Visual field defects are a common presenting feature of craniopharyngioma and are primarily caused by chronic compression of the optic nerves or optic chiasm. In most cases, prolonged compression leads to irreversible axonal damage, particularly when optic disc pallor is present, and visual recovery is therefore considered unlikely.

Although bitemporal hemianopia is the classical visual field defect associated with suprasellar tumors, asymmetric or unilateral temporal hemianopia may occur depending on the direction and extent of chiasmal involvement [7]. The sequential development of hemianopia in each eye observed in our patient suggests a dynamic pattern of compression rather than fixed structural damage to the visual pathways.

Visual recovery in craniopharyngioma has been reported most commonly following surgical decompression or treatment-related tumor regression. Spontaneous recovery without intervention, particularly in the presence of radiological evidence of a longstanding lesion, remains rare. One proposed mechanism for recovery is a reduction in mass effect due to tumor shrinkage or progressive calcification, which may relieve pressure on the optic chiasm and restore axonal conduction.

Another possible explanation is reversible conduction block of optic nerve fibers, in which functional impairment occurs without permanent axonal loss, allowing recovery once compressive forces are reduced [3]. In addition, neuroplasticity and cortical adaptation may contribute to visual improvement, especially in younger patients with preserved central visual acuity [6].

This case demonstrates a rare instance of reversible visual field loss in craniopharyngioma, with spontaneous recovery of a previously affected hemifield. It highlights that visual prognosis may not always be uniformly poor, even in cases with apparent chronic compression, and emphasizes the importance of careful longitudinal visual field assessment and cautious prognostication.

Conclusion

This case demonstrates a rare instance of reversible visual field loss in craniopharyngioma. Although defects are usually permanent due to chronic visual pathway compression, our patient developed sequential hemianopia with spontaneous recovery in the previously affected eye. This case highlights that visual function may recover when tumor regression or calcification reduces pressure on visual pathway structures.

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