Ipsilateral Visual Loss Caused by Optic Nerve Compression between a Tuberculum Sellae Meningioma and the Internal Carotid Artery

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Abstract

We report a rare case of ipsilateral visual loss caused by compression of the optic nerve between a tuberculum sellae meningioma (TSM) and the internal carotid artery (ICA). The patient was a 70-year-old female who presented with a 2-year history of left visual disturbance with a TSM on magnetic resonance imaging. No tumor infiltration to the optic canal was identified in the preoperative images. Extended endoscopic transsphenoidal surgery was performed and showed no infiltration to the optic canal. The tumor was removed completely, and optic nerve compression was found between the TSM and atherosclerotic ICA. This report shows an atypical case in which compression of the optic nerve between a TSM and the ICA caused ipsilateral visual loss despite no infiltration to the optic canal.

Keywords
- compression
- internal carotid artery
- ipsilateral visual loss
- optic nerve
- tuberculum sellae meningioma

Introduction

Tuberculum sellae meningiomas (TSMs), which comprise approximately 4 to 10% of all intracranial meningiomas, arise from the dura of the tuberculum sellae, chiasmatic sulcus, planum sphenoid, and diaphragma sellae.[1–3] These tumors are anatomically closely related to the optic apparatus, such as the optic nerves and chiasma, and internal carotid arteries (ICAs); therefore, they frequently cause an insidious visual disturbance with optic nerve or tract compression. Several studies reveal that 67 to 84.6% of TSMs show involvement with one or both optic canals, leading to visual dysfunction.[1,2,4–6] The presence of significant asymmetric visual findings is highly indicative of optic canal involvement.[7] Larger tumors may exert a mass effect on the optic chiasma, resulting in bitemporal hemianopsia. They can also displace ICAs laterally or occasionally encase ICAs densely. The extent of the visual deficit is the single most important reason for surgical treatment, and the postoperative visual outcome is the primary concern after surgery. There has been much controversy over the surgical approaches to TSMs, whether they are transcranial or transsphenoidal.[8–11] Many reports show detailed visual outcomes after surgery,[2,3,12–14] and they assessed the cause of ipsilateral visual loss as optic canal invasion or optic nerve lateral compression.

We encountered a rare case of unilateral visual dysfunction due to optic nerve compression between the TSM and carotid artery, although the tumor did not infiltrate the optic canal.

Case Report

A 70-year-old woman with mild hypertension and diabetes mellitus presented with gradual progression of left visual loss, which was initially diagnosed as normal pressure

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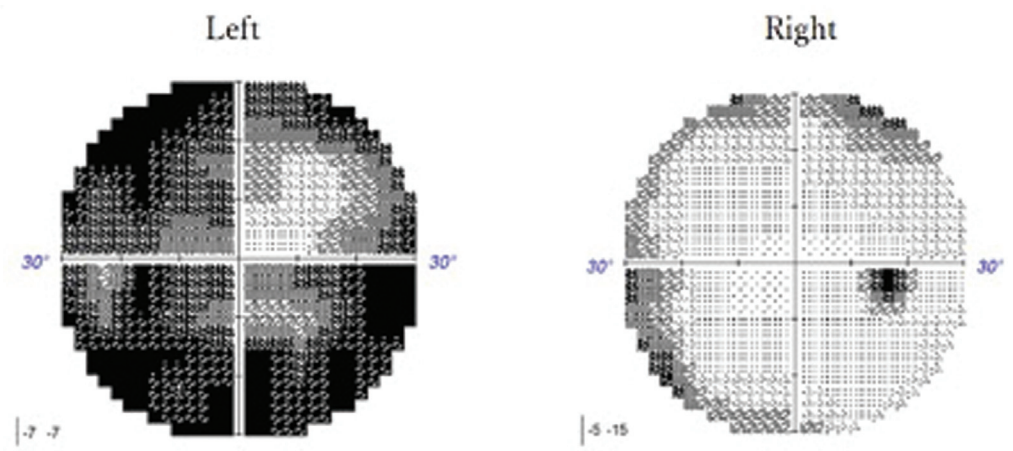

Fig. 1 Preoperative visual field: left (**A**), and right (**B**). The left visual field shows loss of three quadrants with only the superonasal quadrant.

glaucoma at the ophthalmology clinic. One year later, a TSM was incidentally found on magnetic resonance imaging (MRI) images taken to evaluate the patient's dizziness. During follow-up, the size of the TSM gradually increased, leading to further left visual dysfunction, and the patient was referred to us for further evaluation and treatment 2 years after the onset of her symptoms.

Serial visual field examination showed loss of three quadrants in the left eye, with only superonasal quadrant preservation (►**Fig. 1**). The left visual acuity was 0.3, and the right was 1.0, suggesting left optic neuropathy.

T1-weighted gadolinium-enhanced MRI revealed a round TSM measuring approximately 1.3 cm (►**Fig. 2A,B**). We confirmed that the TSM did not infiltrate the left optic canal (►**Fig. 2C**).

The patient underwent extended endoscopic transsphenoidal surgery. The dura was opened, and the tumor was removed. Inspection of the sellae region showed that the denatured left optic nerve was compressed by the left ICA (►**Fig. 3**). Carotid artery atherosclerosis was observed at the part of the compression. The tumor was completely removed, and the left optic nerve was decompressed without

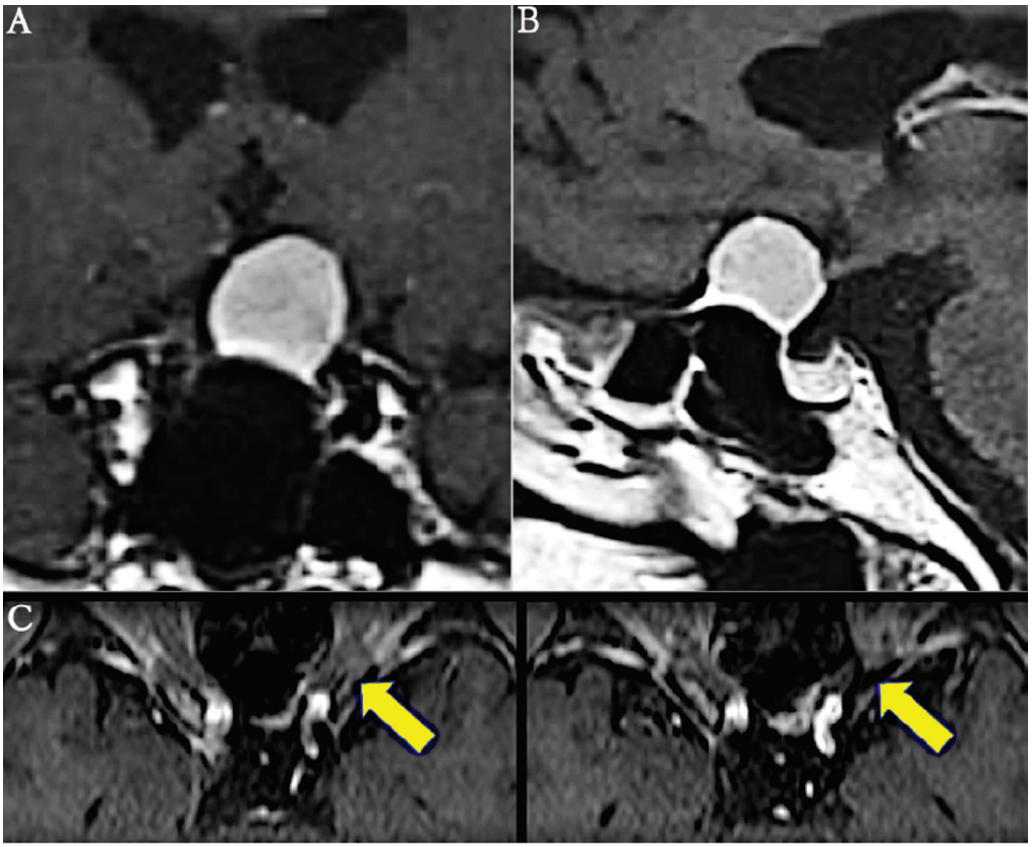

Fig. 2 Preoperative coronal (**A**), sagittal (**B**), and coronal (**C**) magnetic resonance imaging postcontrast enhancement evaluations. This mass with a dural tail was consistent with a tuberculum sellae meningioma. No invasion of the left optic canal by the tumor was identified (*arrow*).

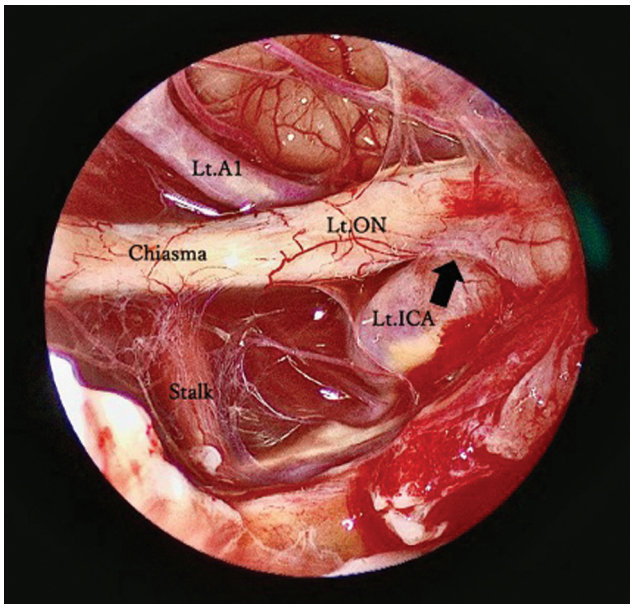

Fig. 3 The intraoperative left optic nerve compressed by the left internal carotid artery (ICA) was identified after endoscopic removal of the tuberculum sellae meningioma (*arrow*). Atherosclerotic changes in the ICA were identified. Although the optic canal was opened, there was no canal invasion of the tumor. After tumor resection, a distinct indentation was observed in the compressed optic nerve.

manipulating the optic pathways. Optic canal unroofing was performed, but the tumor did not infiltrate the optic canal in the intraoperative views. The tumor was pathologically diagnosed as meningioma. The patient tolerated the procedure well and was discharged 2 weeks later.

A neuro-ophthalmologic evaluation performed 3 months after surgery revealed improved visual acuity of 1.0 in the left eye with a smaller central scotoma. We experienced a rare case of unilateral visual dysfunction due to optic nerve compression between the TSM and carotid artery, although the tumor did not infiltrate the optic canal.

Discussion

We present a rare case of optic nerve compression between the TSM and ICA that caused ipsilateral visual loss. Once the tumor was debulked, compression of the lower part of the optic nerve was observed. Although optic canal unroofing was performed, no infiltration to the optic canal was identified in the surgery. The chiasma compression was not identified. There was a dramatic improvement and near-complete resolution in the patient's postoperative visual field at the 3-month follow-up.

In many studies, visual loss secondary to the mechanical compression of the optic nerve or chiasma by tumors, particularly TSMs, is well established.[1,2,4–6] Margalit et al[2] reported 50 cases of meningioma involving the optic nerve, describing the anatomical relationship with the tumor and the degree of ICA involvement. Most cases describe tumor infiltration to the optic canal or optic nerve lateral compression, causing ipsilateral visual field defects. Additionally, many studies have found that chiasma compression is the cause of bitemporal hemianopia. However, we did not find a case in which a TSM and the ICA compressed the optic nerve causing ipsilateral visual disturbance.

We found only two similar papers describing visual loss caused by compression of the optic nerve or vessels due to pressure exerted by a tumor. Bejjani et al[15] reported a suprasellar meningioma compressing the anterior cerebral artery complex with chiasma, which caused an inferior homonymous wedge defect. Mizrahi et al[16] presented a case of optic nerve strangulation by the A1 segment of the anterior cerebral artery with a TSM, which caused loss of three quadrants in the left eye.

In this case, although preoperative MRI and intraoperative views indicated that the TSM did not infiltrate the optic canal, the patient had an ipsilateral visual field deficit, which did not follow the classic pattern. We realized the anatomical relationship and the cause of atypical visual field loss during the surgery. Additionally, compression of part of the ICA revealed atherosclerotic changes, contributing to vascular compression of the optic nerve. We reviewed the preoperative MRI again, which revealed optic nerve compression between the tumor and ICA consistent with the intraoperative view (►**Fig. 4**). In conclusion, given that there was no infiltration to the optic canal on MRI and intraoperative views, the cause of ipsilateral visual loss in this patient was considered to be compression of the optic nerve between the atherosclerotic ICA and TSM.

In most studies, there is little discussion on the cause of ipsilateral visual loss cases without optic canal invasion. Tumor and vascular compressive neuropathy of the optic nerve, such as this case, is rare and cannot be diagnosed

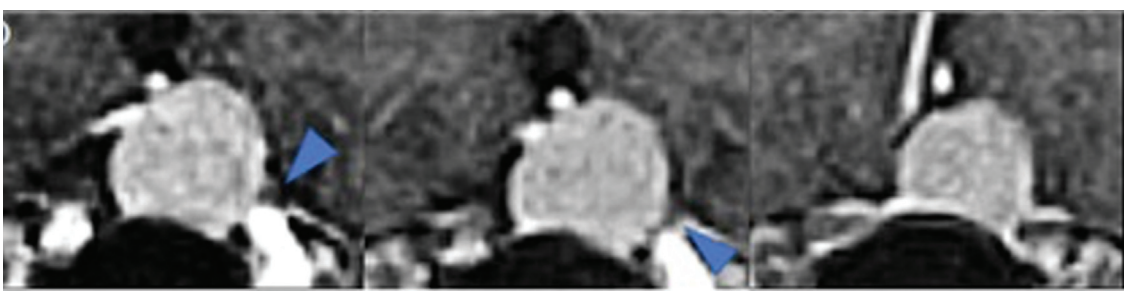

Fig. 4 Preoperative coronal magnetic resonance imagings revealed a compressed optic nerve between the tumor and internal carotid artery, which is consistent with the intraoperative compression of the optic nerve (*arrowhead*).

simply by the radiological finding of optic nerve dislocation. To the best of our knowledge, no endoscopic endonasal image such as this case has been provided in the pertinent literature. In atypical changes such as this case, clinicians should carefully consider the various possibilities of anatomical relationships or abnormalities.

Many studies have shown postoperative improvement in the visual field.[4,17–19] However, as Di Somma et al reported that postoperative visual acuity could not possibly improve due to optic nerve atrophy due to long-standing compression, although tumor removal was completed.[19,20] Improvement in the visual field disturbance in this patient may be accounted for by the short preoperative period, and the operation was performed before atrophy of the optic nerve occurred.

Conclusion

This case illustrates atypical visual field loss caused by TSM. The ipsilateral optic nerve was compressed between the TSM and ICA, leading to ipsilateral visual loss with no optic canal involvement.

Informed Consent

For the case report, informed consent was obtained from the patient for publication of this case report and accompanying images.

Conflict of Interest

None declared.

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