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Optic canal decompression: Crucial goal in SOM surgery for improved visual outcomes – A case series

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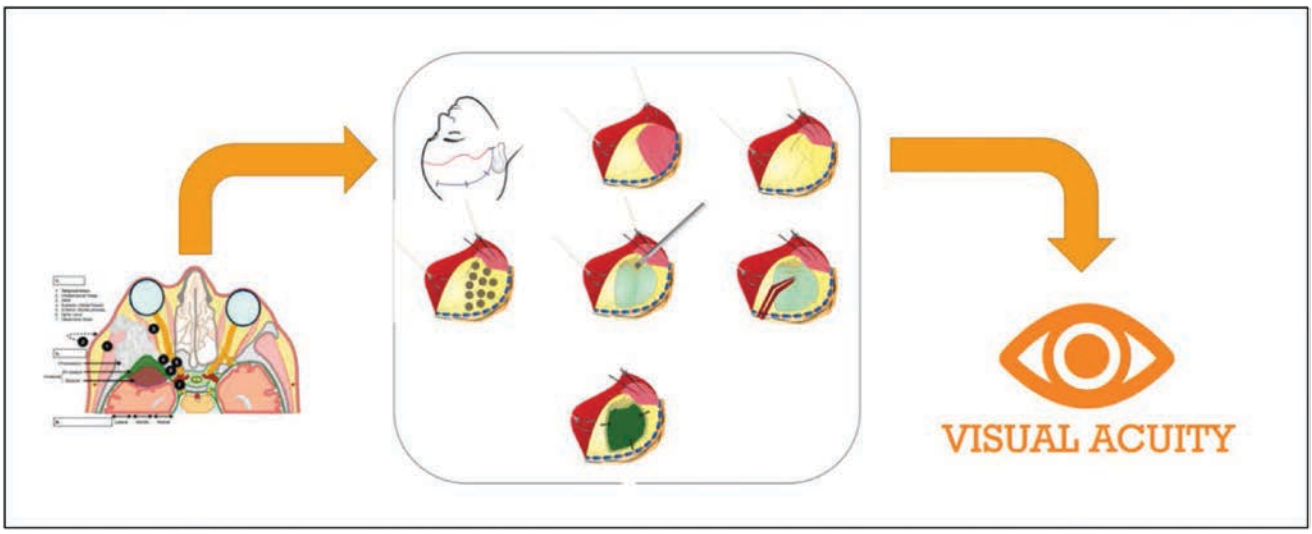
ABSTRACT

Background. Sphenoorbital meningioma (SOM) often presents with proptosis and vision impairment due to optic nerve compression within the optic canal, primarily caused by hyperostosis. This study aims to highlight the importance of optic canal decompression during SOM surgery. Fourteen patients with SOM and vision impairment underwent surgery focused on adequate optic canal decompression. Vision was evaluated preoperatively, at three months, and at six months postoperatively. Preoperative vision loss was confirmed to be due to optic nerve compression by surrounding hyperostotic bone. Six-month postoperative evaluations showed significant improvement in visual acuity in twelve of the fourteen patients. SOM frequently occurs in women in their 50s. Early surgical intervention is crucial for patients with visual symptoms to achieve better outcomes. While cosmetic correction of proptosis is important, the primary surgical goal should be decompression of the optic nerve.

Keywords: sphenoorbital meningioma, optic neuropathy, vision impairment, vision loss



Graphical abstract

INTRODUCTION

Optic canal decompression is a critical procedure in enhancing visual outcomes in surgeries related to skull base lesions, traumatic optic neuropathy, and other conditions affecting the optic nerve. The optic canal serves as a bony tunnel for the optic nerve, connecting the orbit to the middle cranial fossa. Studies

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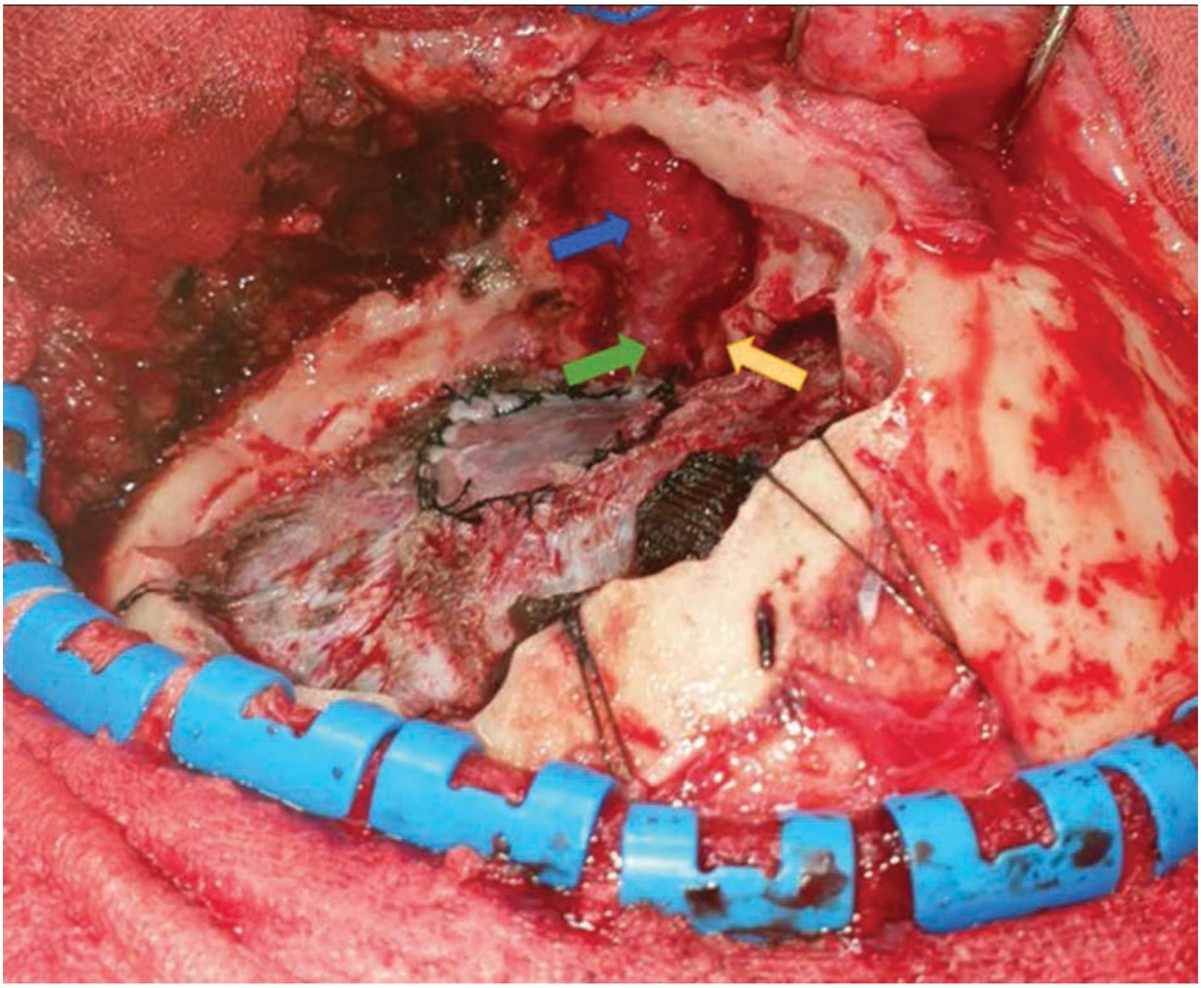


FIGURE 1. The surgical method used to decompress the left optic nerve started with a pterional skin incision on front othe left ear

Note: The yellow arrow indicates the optic canal; green arrow indicates the optic nerve; blue arrow indicates the eye's bulb

have shown the importance of optic canal decompression in improving visual function and reducing proptosis in patients with conditions such as suprasellar meningiomas [1], sphenoorbital meningiomas [2], and traumatic optic neuropathy [3]. Optic canal decompression works by alleviating pressure on the optic nerve, improving blood circulation, and preventing nerve injury [3]. This procedure is particularly crucial in cases of mechanical compression of the visual pathway due to factors like tumors or traumatic injuries.

The significance of optic canal deroofing and orbital wall decompression in optimizing visual outcomes has been highlighted in several studies, leading to visual improvement and reduced proptosis [2]. Similarly, studies emphasize the importance of effective optic canal decompression in creating more space for the optic nerve, contributing to positive visual outcomes [3,4]. Optic canal decompression is also essential in cases of traumatic optic neuropathy, with interventions such as endoscopic transnasal optic canal decompression showing promise in improving vision [5].

Advancements in surgical techniques, such as endoscopic approaches for optic canal decompression, have led to less invasive yet effective ways to address optic nerve compression [6,7]. These approaches, including endoscopic transethmoid-sphenoid optic canal decompression, offer a promising alternative to traditional methods. The increasing use of endoscopic trans-ethmosphenoid optic canal decompression has become an effective approach in recent years [8].

Studies on optic canal decompression in various conditions like dysthyroid optic neuropathy [9], traumatic optic neuropathy [10], and compressive optic neuropathy have shown the positive impact of surgical interventions aimed at relieving pressure on the optic nerve [11]. These interventions not only aid in improving visual acuity but also play a significant role in preventing further visual deterioration and promoting postoperative visual improvement.

Sphenoorbital meningioma (SOM) is a tumor arising from the sphenoid wing, often extending into the orbit and causing hyperostosis of the sphenoid bone [12-18]. Although rare, comprising only 2-9% of intracranial meningiomas, SOM constitutes about 20% of neurosurgical meningioma cases. Histologically benign and slow-growing, SOM can cause significant symptoms due to hyperostosis and optic canal compression, leading to proptosis, visual disturbances, and eye movement disorders. The hyperostosis typically spreads to the sphenoid wing, superior orbital fissure (SOF), optic canal (OC), and anterior clinoid process, with dura invasion extending to the temporal convexity, periorbita, SOF, OC, cavernous sinus, and sphenoid wing [15,19-21].

LITERATURE REVIEW

Optic canal decompression is a crucial intervention for improving visual outcomes in various surgical procedures, particularly in cases of traumatic optic neuropathy (TON) and suprasellar meningiomas. Studies have demonstrated that optic canal decompression surgery relieves mechanical compression on the visual pathway by enlarging the optic nerve space, enhancing local blood circulation, and preventing nerve injury [3]. Optic canal decompression has been identified as a significant factor influencing favorable changes in visual function following the resection of sphenoorbital meningiomas (SOM) [2]. Moreover, in cases of indirect traumatic optic neuropathy (ITON), endoscopic trans-ethmosphenoid optic canal decompression (ETOCD) has been linked to vision recovery and pathophysiological changes in retinal vasculature [8].

The efficacy of optic canal decompression has been further underscored in the context of traumatic optic neuropathy (TON). Studies have explored prognostic factors in TON patients treated with endoscopic transnasal optic canal decompression (ETOCD), using multimodal analysis based on imaging examinations such as optical coherence tomography angiography (OCTA) and CT scans [5]. Optic canal decompression has been established as a critical element in managing TON, with surgical techniques focusing on ensuring proper exposure of the optic canal and orbital apex for effective optic nerve decompression [22]. Additionally, research has explored the timing of optic canal decompression in TON cases, with delayed wider endoscopic optic decompression showing efficacy in improving visual outcomes [23].

In the surgical management of various optic nerve pathologies, including traumatic optic neuropathy and compressive optic neuropathy, optic canal decompression is a key intervention to enhance visual acuity and prevent further optic nerve damage [24]. Studies comparing different surgical procedures for optic nerve decompression highlight inferomedial decompression as a reliable procedure for preserving vision in dysthyroid optic neuropathy [9]. Furthermore, optic canal decompression via endoscopic endonasal approaches has proven effective in addressing traumatic orbital apex syndrome and traumatic oculomotor nerve palsy, underscoring the importance of precise surgical techniques in optic nerve decompression [25,26].

Optic canal decompression has also been explored in the context of indirect decompression for lumbar spinal stenosis and foraminal stenosis. While the efficacy of indirect decompression in treating foraminal stenosis has been acknowledged, its effectiveness in central canal and lateral recess stenosis remains an area of ongoing research (Park et al., 2020). Additionally, in cases of lumbar degenerative diseases, stand-alone percutaneous pedicle screw fixation has been recognized as a safe and effective technique for indirectly decompressing the spinal canal and neural foramina, emphasizing the importance of decompression in managing spinal stenosis [27].

MATERIALS AND METHODS

Fourteen patients with sphenoorbital meningioma (SOM) who underwent surgery at Soetomo General Hospital in 2023 were retrospectively analyzed. Data included patient records, radiological findings, surgical outcomes, and pre- and postoperative vision assessments at three and six months. Only cases with vision impairment due to optic nerve compression by hyperostotic bone were included. Factors analyzed included visual function, intraorbital tumor location, optic canal invasion, surgical approach, and observational analysis.

All patients had unilateral visual impairment and proptosis, with two (14.3%) also experiencing restricted eye movement. Fundoscopy revealed optic disc edema in 13 (92.8%) patients and atrophy in one (7.2%). MRI analysis categorized tumor location and optic canal compression. Tumor spread to the optic canal, superior orbital fissure (SOF), anterior clinoid process, and cavernous sinus is detailed in Table 1. Preoperative visual impairment distribution is summarized in Table 2. A pterional incision was used, with the head positioned 25° contralaterally. A supraorbital-pterional approach included opening the optic canal roof. In cases with diffuse tumor spread, an infratemporal approach with zygoma resection was added for better exposure. Extensive optic canal decompression and drilling of hyperostotic bone were performed. Intradural and intracranial tumors were resected, with the dura grafted using periosteum and bone reconstructed with acrylic. Visual acuity, eye movement, proptosis, and fundoscopy were evaluated at 1, 3, 6, and 12 months postoperatively. Follow-up imaging was conducted at one year due to the tumor's slow growth.

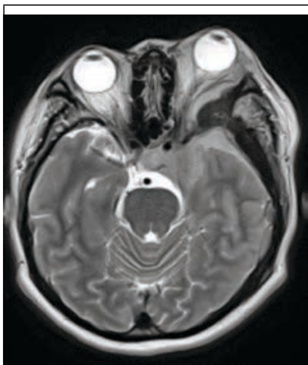
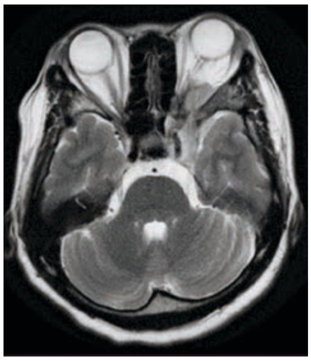
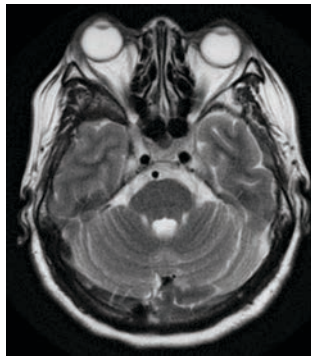
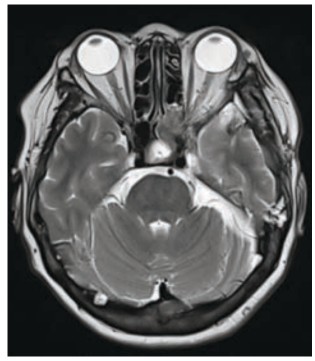
RESULTS

Achieving maximal safe resection to prevent recurrence and optimize outcomes was prioritized. Complete resection, classified as Simpson grades 1 or 2, was challenging due to the complex anatomy and intracranial and cavernous sinus invasion. Six months postoperatively, 12 of the 14 patients showed improved visual function, with no changes in 2 patients and no additional postoperative visual deterioration. Visual improvement correlated with intraorbital invasion type, optic canal invasion pattern, and optic disc status. Chronic optic nerve compression with atrophic papilla was less likely to recover, though proptosis correction improved cosmetic outcomes.

DISCUSSION

Dural invasion and hyperostosis are the primary components of sphenoorbital meningioma (SOM) extension. Extensive drilling of abnormal bone is essential [15,28]. According to Simpson's 1957 statement, aggressive resection, including removal of involved dura and abnormal bone, reduces recurrence [29]. Achieving total tumor removal in SOM is challenging due to complex anatomical structures and invasion of the superior orbital fissure (SOF), extraocular muscles, and cranial nerves [19,20,30]. Despite its benign nature, SOM recurrence occurs approximately five years post-resection. Proptosis and vision impairment are the primary reasons patients seek medical attention, emphasizing the need for optic nerve decompression [18,28,31].

TABLE 1. Tumor extension in sphenoorbital meningiomas

			
Difuse extension	**Infero-lateral extension**	**Lateral Extension**	**Lateral and infero medial extension**

Source: Primary Data, 2024

TABLE 2. Neuroradiological and clinical finding in 14 sphenoorbital meningiomas

Tumor location	Optic canal involvement	Superior orbital fissure	Anterior clinoid process	Cavernous sinus	Visual impairment	Proptosis
Diffuse	4 (100%)	3 (75%)	4 (100%)	4 (100%)	4 (100%)	4 (100%)
Lateral	3 (100%)	-	-	-	3 (100%)	2 (66,66%)
Infero-lateral	5 (100%)	-	-	-	5 (100%)	5 (100%)
Lateral and inferomedial	2 (100%)	-	-	2 (100%)	2 (100%)	2 (100%)

Source: Primary Data, 2024

TABLE 3. Correlation of tumor location, optic canal involvement and Simpson grade with the visual outcome

Tumor location	Number of cases	PVI	OCI	SA	Simpson grade				Visual outcome		
					1	2	3	4	Improved	Stable	Worsened
Diffuse	4	4 (100%)	4 (100%)	Supraorbital-pterional	-	2 (50%)	2 (50%)	-	2 (50%)	2 (50%)	-
Lateral	3	3 (100%)	3 (100%)	Lateral-orbitocranial	3 (100)	-	-	-	3 (100%)	-	-
Infero-lateral	5	5 (100%)	5 (100%)	Lateral-orbitocranial	3 (60%)	2 (40%)	-	-	5 (100%)	-	-
Lateral and inferomedial	2	2 (100%)	2 (100%)	Supraorbital-pterional	1 (50%)	1 (50%)	-	-	2 (100%)	-	-

Source: Primary Data, 2024

Surgical approaches for SOM vary, including pterional, supraorbital-pterional, fronto-temporo-orbitozygomatic, and fronto-orbito-malar, chosen based on the extent of hyperostosis and optic nerve decompression accessibility [30,32,33]. The pterional approach allows access to the sphenoid wing and anterior clinoid process but is limited for medial and inferior optic canal access. Tumors extending inferomedially benefit from a supraorbital-pterional approach, while lateral orbitocranial approaches are more suitable for lateral optic canal and orbit invasion [34-36].

In our series, 12 patients underwent pterional bone opening with extensive optic nerve decompression, while 2 required infratemporal and zygoma resection due to tumor extension. The pterional approach with maximal lateral orbital drilling corrected proptosis by restoring the ocular position within the orbit. Maintaining the anterolateral orbital cavity structure optimized cosmetic outcomes.

Maximal resection remains a topic of debate due to SOM's complexity. Early surgery for proptosis and vision impairment can stabilize and improve visual and cosmetic functions. Multidisciplinary collaboration among neurosurgeons, ophthalmologists, and radiologists is essential for optimal preoperative and postoperative management.

CONCLUSION

Early surgical intervention for sphenoorbital meningioma (SOM), with a focus on optic canal decompression, significantly improves visual outcomes and corrects proptosis. Our study of fourteen patients demonstrated that twelve experienced improved vision six months postoperatively. Effective management of SOM necessitates careful preoperative evaluation, strategic surgical approaches, and multidisciplinary collaboration.

Conflicts of interest: none declared
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