Neuronavigation-Guided Endoscopic Decompression of Superior Orbital Fissure Fracture: Case Report and Literature Review

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ABSTRACT

Objective: We present a rare case of an isolated superior orbital fissure fracture resulting from blunt injury to the face and presenting with selective cranial nerve deficits surgically treated with a neuroendoscopic approach. The anatomy of the superior orbital fissure is detailed, and the peculiarities of the surgical approach described. **Method:** A review of the existing literature reveals this is the first reported case of a neuronavigation-assisted endoscopic approach used in the extraction of a superior orbital fracture fragment with good outcome. Current guidelines for an endoscopic approach in orbital trauma are reviewed, and pertinent literature is discussed. **Conclusion:** Neuronavigation-assisted decompression should be considered as an effective means of removing superior orbital fissure fractures.

KEYWORDS: Orbital trauma, superior orbital fissure, neuroendoscopy, neuronavigation

The superior orbital fissure is a narrow cleft connecting the middle cranial fossa and the orbit. It carries delicate structures including the oculomotor nerve (III), trochlear (IV), sensory ophthalmic branches of the trigeminal nerve (nasociliary, frontal, and lacrimal), abducens (VI), superior and inferior divisions of ophthalmic vein, and sympathetic fibers from the cavernous plexus.¹ (Fig. 1)

Orbital trauma causing compression and disruption of these structures may present clinically with a variety of symptoms, referred to as superior orbital fissure syndrome (SOFS). Traumatic SOFS is rare and may be apparent in ~0.5 to 1% of all maxillofacial injuries, being more common in males and adults.² Documented cases of traumatic SOFS include early reports in the 1970s from Nakagawa

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Figure 1 Anatomy of the superior orbital fissure, orbital view. Note the relative position of the cranial nerve branches in relation to the annulus of Zinn. L, lacrimal branch of trigeminal nerve; F, frontal branch of trigeminal nerve; IV, trochlear; SOV, superior ophthalmic vein; III-s, superior branch of oculomotor nerve; VI, abducens; NC, nasociliary branch of trigeminal nerve; ON, optic nerve; oa, ophthalmic artery; III-i, inferior branch of oculomotor nerve; PN, pterygoid nerve; V2, maxillary branch of trigeminal nerve; IOV, inferior ophthalmic vein.

and Shiga³ and from Kurzer and Patel.⁴ With this report, we intend to highlight the importance of the microsurgical anatomy of the superior orbital fissure to better understand its clinical implications, as well as to provide a first report on the use of an endoscopic approach for decompression and extraction of the superior orbital fissure bony fragment, in this case of a displaced sphenoid sinus wall fracture.

CASE REPORT

A 19-year-old man was admitted to the emergency department after sustaining high velocity blunt trauma from a baseball to right orbit and face. Initial complaints were headache, diplopia, and temporomandibular malocclusion. Ophthalmolog-

ical evaluation of the right eye revealed a supra-orbital laceration, a 3+ adduction deficit, and a 1+ limitation in supraduction and infraduction. There was no medial movement beyond midline, but a small elevation could be achieved. The eyelid elevated easily. No pupil dilation was present, and sensation to the face was grossly intact. No cerebrospinal fluid (CSF) leakage was noted by the patient or found on physical examination.

Computerized tomography (CT) and 64-slice CT angiogram (Lightspeed VCT; GE Healthcare, Milwaukee, WI) with three-dimensional reconstructions (GE Advantage, Windows 3D Workstation, Version 4.2; General Electric Medical Systems, Milwaukee, WI) revealed a fracture of the right lateral wall of the sphenoid sinus traversing the orbital fissure without evidence of vascular involvement (Fig. 2). In addition, the

Figure 2 Three-dimensional reconstructions from computerized tomography angiogram revealing the fracture fragment (circle) traversing the superior orbital fissure at the level of the inferior branch of the oculomotor nerve. Surgical trajectory is indicated by the arrow in part C. (A) Orbital view is shown; (B) sagittal view; (C) surgical/inferior oblique view; (D) axial view. (Scanner: Lightspeed VCT; GE Healthcare, Milwaukee, WI. Software: GE Advantage, Windows 3D Workstation, Version 4.2; General Electric Medical Systems, Milwaukee, WI.)

following fractures were seen: temporal bone, right Le Fort I, nasal septal, and hard palate, but none of these were significantly displaced. The patient was initially started on intravenous methylprednisolone (1 mg/kg/d) and topical scopolamine 0.25% three times per day. As conservative measures, the head of the bed was elevated 30 degrees, and the patient was instructed not to perform Valsalva maneuvers or forcefully blow through the nostrils.

Over the next 2 days, the oculomotor symptoms persisted, and the patient reported newly evident right facial numbness in the territories of the first and second divisions of the trigeminal nerve. The pattern of extraocular movement deficit suggested a partial right third nerve palsy (inferior

branch) with intact fourth and sixth nerve function and possible involvement of the second branch of the fifth cranial nerve (V2).

Due to the new symptoms and lack of clinical improvement with conservative management, it was decided to attempt surgical decompression of the superior orbital fissure by removing the offending sphenoid bone fragment using neuronavigation-assisted endoscopy. Scopolamine treatment was stopped 24 hours preoperatively.

In this case, to gain full access to the face of the sphenoid, an ipsilateral endoscopic anterior and posterior ethmoidectomy with resection of the middle turbinate was performed. Due to the potential for CSF leakage, a pedicled septal flap based on the

Figure 3 Comparison of preoperative and postoperative axial computerized tomography views. (A) Preoperative view of shows the fracture fragment (arrow). (B) Postoperative view reveals complete removal with absence of pneumocephalus (arrow).

posteronasal branches of the sphenopalatine artery was developed as described by Pinheiro-Neto et al.⁵ Next, a wide-access sphenoidotomy was created by drilling the rostrum of the sphenoid to the floor of the sphenoid cavity, to the medial extent of the pterygoid plate, and superiorly to the roof of the sphenoid. The location of the sphenoid wall breach was not immediately visible due to significant mucosal edema and clot, thus its location was confirmed with image guidance using the BrainLab neuronavigation system (BrainLab, Munich, Germany). After some mucosal resection, the bony fragment was found in transverse position wedged into the lateral wall. The fragment was removed without any CSF leak or hemorrhage. The patient was given positive pressure Valsalva with no resultant CSF leak. After cleaning, the septal flap was used to repair the defect. Postoperative CT showed no evidence of pneumocephalus and confirmed a complete bony fragment removal (Fig. 3).

Evaluation at 1 week postsurgery showed improvement to normal binocular vision in neutral position. Right eye adduction weakness was still present but greatly improved, and movement past midline could be observed. All other cranial nerves were intact and the facial paresthesias had resolved. Evaluation at 4 weeks showed greatly improved extraocular motion, with no obvious gaze divergence except on extreme left gaze deviation, at which point the patient had very mild subjective double vision. At last follow-up, 2 months after decompression,

the patient reported normal vision with no diplopia even on extreme gaze. Normalization of the examination was confirmed by an ophthalmologist.

DISCUSSION

The superior orbital fissure is a small, bone-limited space that connects the middle cranial fossa and the orbit. Its microsurgical anatomy has been described in detail elsewhere.^{1,6,7} The nine different shapes, with two major variations defined for the superior orbital fissure in the current literature, become extremely relevant when the surgical target is near or located in the superior orbital fissure.^{8,9} These variations in structure may affect not only the clinical expression but also any approach to therapy after injury. Bony fragments can disrupt the structures in this region directly, as in this case with displaced bone fracture fragment or by secondary effects resulting from causes such as edema, increased intraocular pressure, or hematoma.^{2,10,11} Traumatic SOFS can present clinically with internal and/or external ophthalmoplegia, hypoesthesia or anesthesia in the frontal periorbital region, corneal anesthesia, proptosis, ptosis, and lacrimal hyposecretion.^{2,11} The presence of exophthalmos should raise suspicion for traumatic carotid-cavernous fistula, in which case a CT angiogram and/or formal four-vessel angiogram should be performed.²

In the context of trauma, the highest available resolution CT with coronal reconstructions remains the first choice for diagnostic imaging. However, failure to identify a bony fracture does not exclude other pathology, and further tests may be necessary to understand the cause.

Even though the syndrome has been described in the literature since 1858, debate continues on proper management.^{12,13} The main management strategies to treat traumatic SOFS include watchful waiting, high-dose steroids (dexamethasone, 16 to 24 mg/d; methylprednisolone, 1 mg/kg/d), and surgical decompression. Treatment has best results when initiated early after injury, usually starting with high-dose steroids and proceeding to surgical decompression within several days if there is no improvement.^{12,14}

Surgery can also be considered when there is evidence of impinging bone fragments on the nerve or unresolved orbital hematoma.² Although it is generally agreed that some fractures of the orbit benefit from reduction of the rim and decompression exploration of the fissure, linear fractures that are not displaced are less likely to benefit and the surgical intervention itself may cause iatrogenic damage to the optic nerve.^{15,16} In our case, the treatment for a third nerve injury of this type is made difficult by the location of the bone fragment and the likelihood of aberrant third nerve regeneration with or without treatment. In review of the literature and because of the scarcity of cases of traumatic SOFS, there is little available on the surgical approach for decompression of isolated traumatic SOFS resulting from a bony fragment. External repair and decompression in orbital trauma was once the standard of care with improvements in ocular function after repair.¹⁷ The first reported case of transnasal decompression used for SOFS was described by Ukleja and Kazmierczak in 1982.¹⁸ Menard et al reported a transconjunctival approach for SOFS resulting from orbital trauma.¹⁹ Other possible approaches include the coronal, palpebral, and transantral/transorbital technique.² Endoscopic decompression in orbital trauma, especially when the optic

nerve is involved, is receiving more attention, but there is a lack of management guidelines.²⁰

Transnasal endoscopic repair is already well documented and widely accepted as a safe method for treatment and repair of orbital blowout fracture,^{21–24} however, the use of neuronavigation-assisted transnasal endoscopic decompression in cases of partial or complete SOFS secondary to trauma has not been reported to our knowledge.

CONCLUSION

Superior orbital fissure cranial nerve compression syndromes are relatively rare complications of craniofacial trauma. When caused by bony fragments accessible through the nasal sinuses, neuronavigation-assisted endoscopic decompression should be considered.

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