Successful Conservative Management of a Case of Traumatic Superior Orbital Fissure Syndrome

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Abstract

We report the case of a 22-year-old man who presented with complete ptosis, anisocoria, and total limitation of ocular motility of the right eye following blunt trauma. He was diagnosed to have traumatic superior orbital fissure syndrome based on clinical and radiologic evaluation. The patient achieved complete recovery of cranial nerve function. This unusual complication is reported in less than 1% of cases of craniomaxillofacial trauma. The clinical presentation with external and internal ophthalmoplegia but sparing of vision occurs as a result of the functional compromise of the neurovascular structures traversing the superior orbital fissure while sparing the optic nerve within the optic canal. Management may include observation, repair of associated fractures, megadose steroid therapy, and surgical decompression of the superior orbital fissure. A review of literature reveals that this condition with a unique ocular presentation is underreported in ophthalmic literature, perhaps as initial diagnosis may be obscured by coexisting craniofacial trauma.

Keywords
- trauma
- superior orbital fissure syndrome
- total ophthalmoplegia
- management

Introduction

Superior orbital fissure syndrome (SOFS) is a rare condition presenting with a combination of ophthalmoplegia, ptosis, proptosis, anesthesia along V1, and a fixed dilated pupil. Causes may be inflammatory, infectious, neoplastic, vascular, and traumatic.[1,2] We report a case of traumatic SOFS and review the literature on the pathogenesis and management of this unusual condition.

Case Report

A 22-year-old man was evaluated for closure of the right eyelid following a motorcycle accident.

Visual acuity was 6/6 in both eyes. The patient had right periorbital edema, ecchymosis, complete ptosis, and limited mandibular movement. Anisocoria was present with right pupil of 5 mm versus the left of 3 mm. Afferent pupillary response was normal. He had paresthesia of the right frontal region with mild proptosis and complete limitation of movement of the right globe in all directions (►**Fig. 1a**). The fundi were normal.

On palpation, there was tenderness over the zygomatic arch and frontozygomatic suture. Maxillofacial computed tomographic (CT) scan with three-dimensional (3D) reconstruction showed a right tripod fracture involving the zygomatic arch, orbital floor, lateral wall of orbit, fracture of the greater wing of sphenoid, and coronoid process of right mandibular ramus. Comminuted fractures of the anterior, posterolateral, and superior walls of the right maxillary sinus with air fluid levels were seen. Right optic canal and superior orbital fissure (SOF) were intact (►**Fig. 2**).

A diagnosis of SOFS associated with craniofacial fractures was made. Treatment options were discussed. The patient underwent open reduction and internal fixation of right

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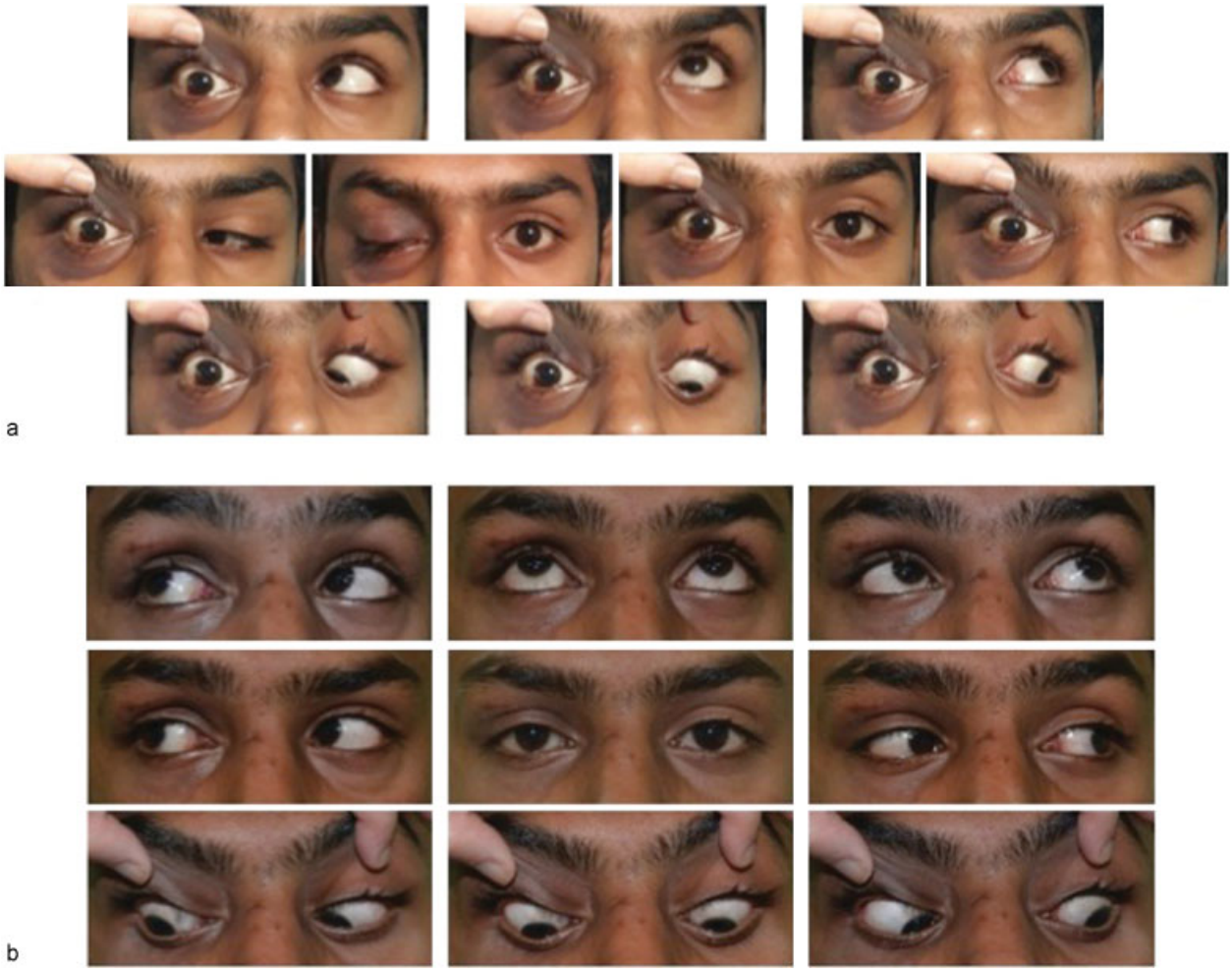

Fig. 1 (a) The patient at presentation with total ophthalmoplegia of the right eye and (b) complete recovery was observed after 4 months.

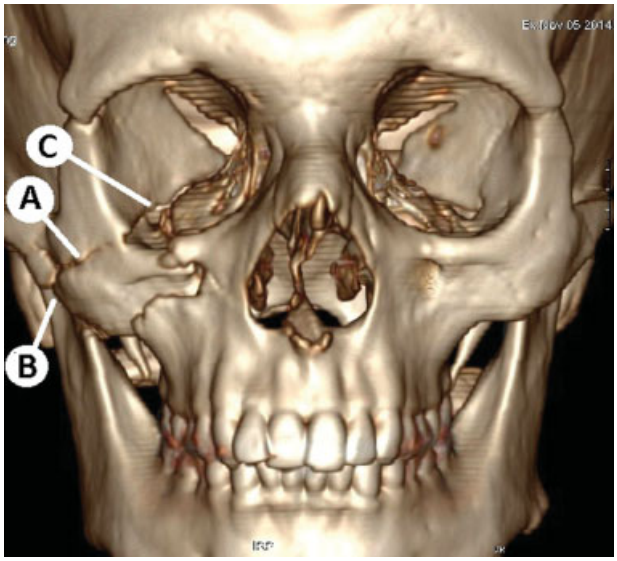

Fig. 2 3D CT reconstruction shows: (A) Right tripod fracture with fracture of the right zygomatic arch, orbital floor, and lateral wall of the orbit. (B) Fracture of the coronoid process of right mandibular ramus. (C) Fracture of greater wing of the right sphenoid is seen with fractured fragment causing impingement upon right inferior orbital fissure. (D) Right optic canal and superior orbital fissure is normal.

facial fractures. He did not receive systemic steroids or undergo any specific intervention to decompress the SOF.

He was kept under monthly observation to monitor recovery. After 4 months the patient's SOFS had resolved almost completely other than persistent paresthesia over right forehead, eyelid, and mild anisocoria (►**Fig. 1b**).

Discussion

The SOF is a narrow bony cleft (3 × 22 mm) transmitting CN III, IV, VI, frontal, lacrimal, and nasociliary branches of the Vth nerve, inferior and superior ophthalmic veins, and sympathetic filaments, from the middle cranial fossa to the orbit (►**Fig. 3**).

Traumatic SOFS is a very rare complication of craniofacial trauma, including skull, zygomaticomaxillary complex, orbital, Le Fort II, and Le Fort III fractures, with an incidence of 0.3 to 0.8%.[1,3]

SOFS can be caused by direct compression of the cranial bone by displaced bony fragments or indirectly by edema, bleeding or hematoma formation increasing the pressure and compressing the nerves against the bony margin of the fissure.[4,5]

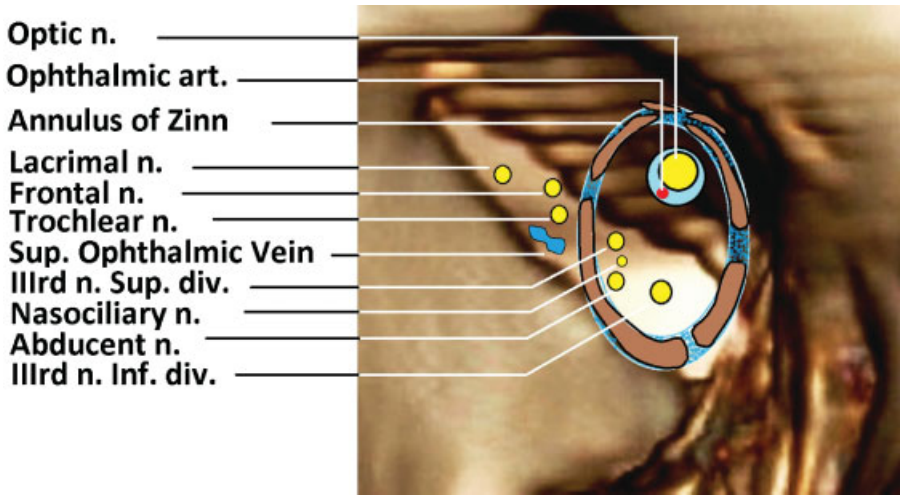

Fig. 3 Diagrammatic representation of right superior orbital fissure with its contents. art., artery; div., division; Inf, inferior; n., nerve; Sup., superior.

Traumatic aneurysm of the internal carotid artery, carotid-cavernous sinus fistula, and transmitted force causing a temporary neuropraxia are other indirect causes.[3,6]

Anatomically, the clinical signs of SOFS can be explained by the involvement of the respective nerve. External ophthalmoplegia and ptosis occur due to impairment of the III, IV, and VI nerves. Internal ophthalmoplegia arises from disruption of the parasympathetic fibers traveling with the III nerve. Injury to the lacrimal, frontal, and nasociliary nerves results in paresthesia of the forehead and upper eyelid, diminished corneal sensation, and lacrimal hyposecretion. Proptosis is caused by decreased tone of the extraocular muscles that normally act as globe retractors.[2] The III, VI, and nasociliary nerves are more susceptible to injury as they are confined within a tendinous ring.[4] VI CN is the most severely injured, whereas IV CN is the least injured and achieves the best functional recovery.[3]

Fractures in the orbital apex region are best imaged by high resolution CT scan with axial, coronal, and 3D reconstruction images. Coincident soft tissue pathology can be evaluated by magnetic resonance imaging (MRI) with contrast and fat suppression sequences. MR or CT angiography is useful in evaluating vascular lesions of the carotid sinus.[2,7]

Debate continues on proper management because of the small number of cases reported. Conservative treatment with observation alone has been proposed because nerve function may recover spontaneously. Routine surgical treatment of associated complex facial fractures can hasten the resolution of SOFS in selected cases.[8]

Steroid therapy may hasten functional recovery by reducing the swelling and edema.[5] Acartürk et al have reported excellent outcome in five patients with megadose steroid therapy.[9] The recommended regimen of steroid therapy is methylprednisolone 30 mg/kg loading dose followed by 15 mg/kg every 6 hourly for 3 days, followed by tapered dose of oral steroids.[5] Literature review shows that patients treated with steroids may have a better chance of neurologic recovery than those with observation alone (70 vs. 42.1%).[4] Treatment has best results when initiated early, starting with high-dose steroids and proceeding to surgical decompression within 10 to 14 days if there is no improvement.

Surgery should be considered when there is evidence of impinging bone fragments on the nerve or a nonresolving orbital hematoma. Several surgical approaches have been detailed to decompress the SOF, including the coronal, palpebral, and transantral/transorbital techniques. Recently, a case of neuronavigation-assisted transnasal endoscopic approach to extract a superior orbital fracture fragment has been described.[10]

Chen et al have done a retrospective review of 33 cases of traumatic SOFS over 14 years and found that functional recovery of all ocular motor nerves occurs mainly in the first 6 months after injury. Eight of 33 (24%) patients had complete functional recovery of all three cranial nerves, of whom 6 (21.4%) patients did not receive steroid therapy.[3] There are nearly 75 reported cases of traumatic SOFS in the literature but no consensus yet on the management of these patients. Steroids may give rise to a faster resolution, but it is uncertain whether it affects the extent of recovery.

Conclusion

SOFS is a rare complication of craniofacial injury with a dramatic ophthalmic manifestation. Clinical presentation and management may be delayed due to concomitant severe intracranial and facial injury. Diagnosis is based on clinical and radiologic examination. Any associated facial fracture should be repaired early. Surgical decompression is indicated if a displaced splenoid fracture with bony impingement of SOF is present. The role of megadose steroids continues to be controversial. Based on our case and the review of literature, we suggest that patients with

traumatic SOFS, without bony compression, can recover complete neural function without additional steroid therapy.

Note

The study was conducted at Venu Eye Institute and Research Centre, Sheikh Sarai, New Delhi, India.

Conflict of Interest

This study was not supported by any grant and none of the authors have any commercial interest to declare.

References

1 Zachariades N, Vairaktaris E, Papavassiliou D, Papademetriou I, Mezitis M, Triantafyllou D. The superior orbital fissure syndrome. J Maxillofac Surg 1985;13(3):125–128

2 Yeh S, Foroozan R. Orbital apex syndrome. Curr Opin Ophthalmol 2004;15(6):490–498

3 Chen CT, Wang TY, Tsay PK, Huang F, Lai JP, Chen YR. Traumatic superior orbital fissure syndrome: assessment of cranial nerve recovery in 33 cases. Plast Reconstr Surg 2010;126(1):205–212

4 Chen CT, Chen YR. Traumatic superior orbital fissure syndrome: current management. Craniomaxillofac Trauma Reconstr 2010; 3(1):9–16

5 Rai S, Rattan V. Traumatic superior orbital fissure syndrome: Review of literature and report of three cases. Natl J Maxillofac Surg 2012;3(2):222–225

6 McAvoy CE, Lacey B, Page AB. Traumatic superior orbital fissure syndrome. Eye (Lond) 2004;18(8):844–845

7 Ettl A, Zwrtek K, Daxer A, Salomonowitz E. Anatomy of the orbital apex and cavernous sinus on high-resolution magnetic resonance images. Surv Ophthalmol 2000;44(4):303–323

8 Evans HH, Wurth BA, Penna KJ. Superior orbital fissure syndrome: a case report. Craniomaxillofac Trauma Reconstr 2012;5(2):115–120

9 Acartürk S, Seküçoğlu T, Kesiktäs E. Mega dose corticosteroid treatment for traumatic superior orbital fissure and orbital apex syndromes. Ann Plast Surg 2004;53(1):60–64

10 Gasco J, Hooten K, Ridley RW, et al. Neuronavigation-guided endoscopic decompression of superior orbital fissure fracture: case report and literature review. Skull Base 2009;19(3):241–246