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Case Report

Traumatic superior orbital fissure syndrome: A case report and literature review

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Abstract

Superior orbital fissure syndrome (SOFS) is a complex of where the function of cranial nerve (III, IV, V, and VI) impaired, due to traumatic or non-traumatic cause, Patients can present with loss of sensation over the forehead, loss of tone of extraocular muscles and dilated pupil, and loss of accommodation reflex along with ptosis. Surgical intervention is only indicated if there is evidence of distorted SOF anatomy to restore the anatomy and size of SOF.

Keywords: Superior Orbital Fissure; Superior Orbital Fissure Syndrome; Trauma; Zygomaticomaxillary fractures

Abbreviations

SOFS: Superior orbital fissure syndrome; SOF: Superior orbital fissure; CT: Computed tomography; RTA: Road traffic accident; GCS Glasgow Coma Scale; EOM: Extraocular muscle; ICU: Intensive care unit; OA: Orbital apex

Introduction

The superior orbital fissure syndrome (SOFS) is a complication resulting from the narrowing of the superior orbital fissure (SOF) that can lead to an impaired function of the cranial nerves III, VI and VI [1,2]. Etiologies can be traumatic and non-traumatic [1]. Treatment of traumatic SOFS can include megadose of steroid if there is no evidence of anatomical distortion of the orbital fissure otherwise, surgical intervention is necessary to restore the SOF anatomy [3,4]. In this report, we are describing a case of traumatic SOFS. Computed tomography (CT) showed multiple maxillofacial fractures. The patient underwent steroid therapy with surgical correction to reestablish the distorted facial anatomy.

Case presentation

A 19-years-old male who suffered severe trauma due to a road traffic accident (RTA) reported to the emergency department at Sultan Qaboos University Hospital with a low Glasgow Coma Scale (GCS) of 8 where he was sedated and intubated. On maxillofacial examination, there were right face abrasion injuries with bilateral periorbital edema, ecchymosis, and subconjunctival hemorrhage. On ophthalmic examinations, extraocular muscle (EOM) could not be assessed as the patient was sedated. However, Pupil shows anisocoria as left pupil size 4mm and right pupil 2mm. The left eye shows severe eyelid swelling and ecchymosis, conjunctiva

chemosis temporally, with subconjunctival hemorrhage. The right pupil shows a sluggish reaction whereas the left shows no reaction. Initial computerized tomography (CT) scan showed multiple bony calvaria fractures along with multiple skull base fractures present involving anterior and middle cranial fossa. The fractures involved left squamous temporal, left zygoma and zygomatic arch, lateral wall of the orbit extending to the left greater wing and body of sphenoid bone showing displaced fracture causing a narrowing to the left SOF with a normal right side (Figure 1). Other fractures include anterior and lateral wall of left maxillary sinus with no retrobulbar hemorrhage.

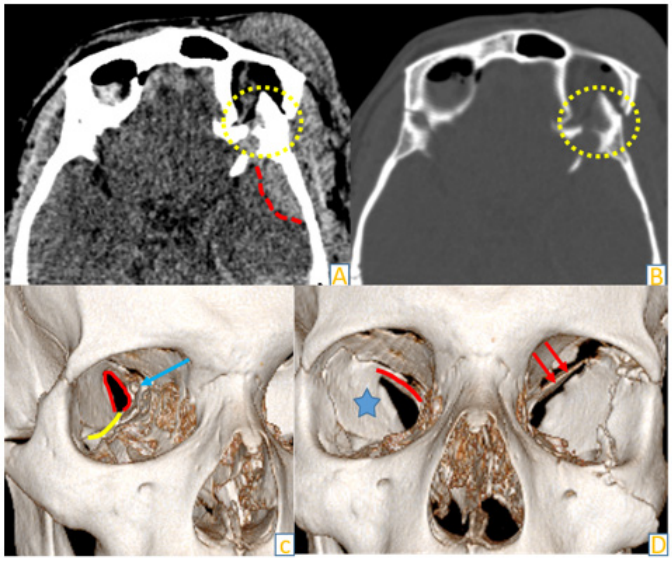

Figure 1: Axial Non-contrast CT scan brain soft tissue window (A) and bone window (B) show a large bony fragment of lesser wing of sphenoid bone within the superior orbital fissure (SOF) (yellow circle). Note is made of left anterior temporal region extradural hemorrhage (red broken line). 3D volume rendered image normal right orbit (C) demonstrating the normal anatomy of SOF which is continuous with inferior orbital fissure (red and yellow lines). Note is made of right optic nerve foramen (blue arrow). 3D volume rendered image of both orbits (D) demonstrating greater (blue star) and lesser wing of sphenoid (red line) in right orbit. Left orbit shows a large bony fragment occupying the left SOF (red arrows).

Multi-disciplinary teams were involved in managing this case including neurosurgery, general surgery, ophthalmology, intensive care unit (ICU) and oral maxillofacial surgery. The patient was kept on anticonvulsant (levetiracetam) and analgesic with full sedation with ventilatory support, brain protective measures, with pupil's size monitoring and charting. The patient was started on dexamethasone initially and then switched to IV methylprednisolone. Admission course complicated by left middle cerebral artery infarction which required decompressive craniectomy. After three weeks, he underwent open reduction and internal fixation (ORIF) of bilateral angle of mandible and left zygoma. Upon discharge, the patient was opening his eyes spontaneously with left partial ptosis with left pupil not reacting. On further follow ups, the patient developed blindness and the suspected causes are persistent increase intracranial pressure after the middle cerebral artery infraction and optic atrophy due to optic apex trauma.

Discussion and Anatomical considerations

The bony orbit is described as a conical structure that has an apex directed posteriorly and a base. The bony orbit is further divided into anterior, middle, and posterior thirds [5]. Moreover, the SOF and the optic foramen are two important communication channels that connect the orbit through the posterior third with the middle cranial fossa [6].

The SOF is a bear-shaped structure bounded laterally by the greater wing of the sphenoid, medially by the lesser wing of sphenoid and superiorly by the frontal bone [4]. The SOF separates the lateral wall and the roof of the orbit with an angulated narrow and upward lateral extension at 45 degrees sized 2-3 mm in width in the narrow part and 7-8 mm in the broader part, and 22 mm in length [7,8]. The middle cerebral fossa and the orbital apex are separated by the lesser and greater sphenoid wings, SOF forms the interconnection that allows structures to pass from the middle cerebral fossa to the orbital apex (OA) and vis versa. The size of SOF is approximately 2-3 mm in width and 22mm in length. Raymond, et al. studied 100 cadaveric orbits and found nine morphological forms of SOF, however, only two form shows significant morphological differences. The variant form of SOF shows no statistical significance when compared to sex and body size [8].

SOF is further divided into three sections: inferior, central, and lateral. The inferior part, located below the annular tendon and origin of the inferior rectus muscle, transmits the inferior ophthalmic vein, and is filled with a posterior extension of the orbital fat. The

central section, located behind and aligned with the lateral part of the annular tendon, transmits the superior and inferior division of the oculomotor nerve (III), the abducens nerve (VI) and nasociliary nerves (V), and the sensory and sympathetic roots of the ciliary ganglion, all of which pass through the oculomotor foramen located inside the annular tendon. The lateral section, which is the lateral narrow part, transmits the trochlear (IV), frontal and lacrimal branches of the ophthalmic division of the trigeminal nerve (V) and the superior branch of the ophthalmic vein, these structures are all located outside and lateral to the annular tendon [9]. When observing the posterior orbital wall, the most proximal structures are the optic nerve and ophthalmic artery which passes through the optic canal (Figure 2) [10].

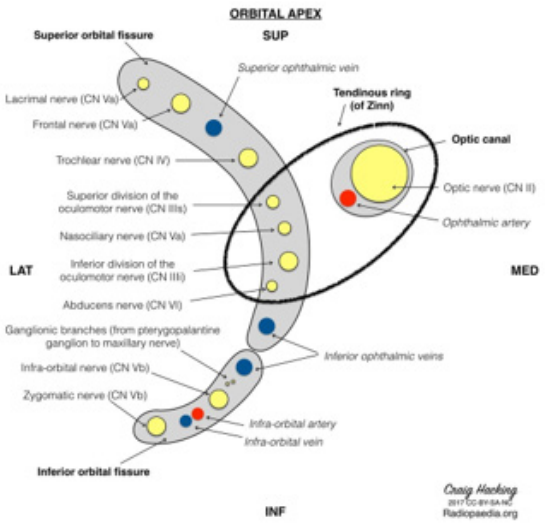

Figure 2: Diagrams of the contents of the superior orbital fissure, tendinous ring, inferior orbital fissure, and optic canal.

The oculomotor (III), trochlear (IV), abducent (VI) and branches from the ophthalmic division of the trigeminal nerve (V) are responsible for the motor innervation to the extraocular muscles and sensory innervation to the adjacent structures in the face and forehead [5,6]. Patients with SOFS can present with loss of sensation over the forehead due to the involvement of the supratrochlear and supraorbital nerves of the first division of the trigeminal nerve (V). Edema of the periorbital region can be explained by compression of the venous and lymphatic which affects the ophthalmic veins. Loss of tone of the extraocular muscle and the anterior prolapse of the globe can lead to proptosis. Block of the transmission through the oculomotor nerve can lead to dilatation of the pupil and loss of accommodation reflex due to the involvement of parasympathetic fibers, ptosis due to levator palpebrae superioris dysfunction, and loss of direct light reflex due to the block of the ipsilateral efferent. Patient can present with ophthalmoplegia due to the blockage of the oculomotor (III), trochlear (IV), and abducent (VI) nerve. lacrimal nerve involvement can lead to lacrimal hyposecretion [11]. In addition to the above symptoms, the optic nerve involvement will indicate orbital apex syndrome (OAS) and will not be labeled as SOFS [1]. The symptoms vary depending on the severity of the compression and the time of presentation as different healing stages can present with different clinical findings. Involvement of the sixth and third cranial nerves along with the nasolacrimal nerve can help determine whether it is partial or complete superior orbital fissure syndrome [4].

Causes of SOFS can be divided into traumatic causes and non-traumatic. Non-traumatic causes can be due to inflammation, tumors, and trauma [1]. Some literature reported inflammation of the central nervous system meninges, cavernous sinus, or retrobulbar space caused by syphilis and tuberculosis as a cause of SOFS [12–14]. However, traumatic SOFS in patient with skull fracture, zygomaticomaxillary complex, and orbital account as the major causes of SOFS as a direct cause or as a complication after facial bone fractures repair [4,5,11,15–20].

There are no clearly defined treatment in the literature. However, management of SOFS varies from conservative, steroid therapy, and surgical intervention. The Steroid has been used to reduce the edema caused by craniofacial fracture, Postma et al was the first to report the short-term use of dexamethasone (4 mg every 6 hours) in patient with traumatic SOFS caused by zygomaticomaxillary complex fracture and sphenoid fracture [21]. Acarturk, et al. had an excellent outcome when using megadose steroid of 30mg/kg every 6hrs in traumatic SOFS [3]. Methylprednisolone 30mg/kg loading dose followed by 15 mg/kg every 6 hrs. for 3 days is now for the treatment of SOFS (Table 1) [4].

Table 1: Literature analysis.

Author	Number of cases reported	Complete Recovery	Partial Recovery	Lost Follow-up	Skull fracture	Facial fracture	Sphenoid fracture	ORIF of Facial fracture	Surgical Decompression of SOF	Steroid therapy
Postma et al. [21]	1	1	0	0	0	1	1	0	0	1
Acartu¨rk et al. [3]	5	5	0	0	0	5	0	4	0	5
Chien-Tzu-ng, chen et al. [22]	33	8	25	0	7	22	6	22	6	5
K. H. Taylor [27]	1	0	1	0	0	0	1	0	0	0
Evan et al. [28]	1	1	0	0	0	1	1	1	1	1
Rai and Rattan [11]	3	1	2	2	0	2	0	1	0	3
Caldarelli et al. [16]	1	1	0	0	1	1	1	1	1	1
H. Jin et al. [23]	39	0	0	0	0		25	0	25	14
Özcan et al. [15]	1	1	0	0	0	0	0	0	0	1

Surgical intervention is indicated if there is distorted anatomy that causes narrowing of the SOF as in displaced fracture fragments [22]. One case was reported by Caldarelli, et al. developed SOFS 12 hours post trauma, with CT scan showing narrowing of the SOF. Initially, they started intravenous (IV) steroids with no improvement but had a full recovery after surgical decompression [16] In a review of 33 patient with SOFS, Chien-Tzung chen, et al. had similar cranial nerve recovery in patients who underwent surgical decompression despite initially worse functional level when compared with the non-sphenoid fracture group (Table 1) [22]. There was some consideration about early surgical intervention as it might increase swelling and ocular pressure which could worsen the cranial nerve palsy as T. Fujiwara et al reported one patient who underwent ORIF of the facial fracture on day 18 post trauma and developed SOFS on day 1 post operation [20]. However, Chen et al. reported no significant relationship between early surgical intervention and poor cranial nerve recovery [22]. Moreover, Jin, H et al. described the management and outcome in 39 patients diagnosed with SOFS. It was shown that the effective rate with surgical interventions within 3 days was 63.6%, whereases, it is shown to be 35.7% after 7 days with significant statistical differences between the two groups.

Superior orbital fissure syndrome can present with the following signs:

- Paresthesia along the ophthalmic division of the trigeminal nerve involving the supratrochlear and

supra orbital branches.

- External ophthalmoplegia due to paralysis of extra ocular muscles.
- Proptosis due to extraocular muscle tone loss or retrobulbar hematoma or ophthalmic vein Compression [23-25].
- Ptosis due to levator palpebrae and mullers muscle affection.

Clinical signs, symptoms and radiographical examinations are the most important in SOF diagnosis. In such patients Caldwell projections can be used to have a 20 to 25-degree tilt of the head that facilitates optimal projection of the superior orbital fissure, especially in severely traumatized patients. CT scan serves as an excellent tool for the diagnosis of SOFS. CT slices of 2mm thickness are usually recommended to visualize any compression around the fissure [26] in addition, spiral CT without the need for neck extension.

Conclusion

We presented a case of traumatic SOFS confirmed radiologically with zygomaticomaxillary and skull base fractures. SOFS can develop as a complication following trauma, either directly because of fractures causing narrowing of the SOF or edema and inflammatory process leading to compression of the structures passing through the fissure [5]. SOFS and OAS can have similar presentation; however, the involvement of optic nerve will indicate OAS [1]. Also the post traumatic carotid-cavernous fistula can be differentiated by pulsatile exophthalmos, SOFS treatment is not standardized as it's uncommon and less documented and depends on its etiology, it can be conservative, medical with steroid therapy or surgical decompression depending on the cause of the SOFS, presentation and CT scan finding. Early surgical intervention through orbital and

cranial extradural approach or endoscopic endonasal approach is crucial for patient recovery as they can prevent permanent damage to the structures passing through the SOF and hasten the resolution of SOFS [22,23]. In summary, SOFS is a rare complication of craniofacial trauma. Clinical symptoms and radiological investigations are the core in the diagnosis, conservative treatment with a megadose steroid is the first line of treatment in the absence of compression by a fracture fragment. Displaced bony fragments causing a compression would be considered as a bad prognosis and urge the need for surgical decompression. Reduction of the orbital blow-in fracture and reducing the infraorbital pressure would improve the cranial nerve function in maximum 6 months with proper treatment and good general condition.

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Conflict of Interest

We declare that, by doing this work, we do not have any financial interest nor conflict of interest.

References

1. Mortada A (1961) Superior Orbital Fissure Syndrome of Uncertain A etiology: Report of Ten Cases. British Journal of Ophthalmology 45(10): 662-671.
2. Banks P (1967) The superior orbital fissure syndrome. Oral Surgery, Oral Medicine, Oral Pathology 24(4): 455-458.
3. Acartürk S, Seküçoğlu T, Kesiktäs E (2004) Mega dose corticosteroid treatment for traumatic superior orbital fissure and orbital apex syndromes. Ann Plast Surg 53(1): 60-64.
4. Chen CT, Chen YR (2010) Traumatic Superior Orbital Fissure Syndrome: Current Management. Craniomaxillofac Trauma Reconstr 3(1): 9-16.
5. Ghosh SK, Narayan RK (2021) Fractures involving bony orbit: A comprehensive review of relevant clinical anatomy. Translational Research in Anatomy: 24.
6. Susan Standring (2022) Gray's Anatomy: 42nd edition | | ISBN: 9780702077050 | ANZ Elsevier Health Bookshop: Books. Elsevier, ed. Gray's Anatomy.
7. Morard M, Tcherekayev V, De Tribolet N (1994) The superior orbital fissure: a microanatomical study. Neurosurgery 35(6): 1087-1093.
8. Reymond J, Kwiatkowski J, Wysocki J (2008) Clinical anatomy of the superior orbital fissure and the orbital apex. Journal of Cranio-Maxillofacial Surgery 36(6): 346-353.
9. Natori Y, Rhoton AL (1995) Microsurgical anatomy of the superior orbital fissure. Neurosurgery 36(4): 762-775.
10. Ulutas M, Boyacı S, Akakın A, Kılıç T, Aksoy K (2016) Surgical anatomy of the cavernous sinus, superior orbital fissure, and orbital apex via a lateral orbitotomy approach: a cadaveric anatomical study. Acta Neurochir (Wien) 158(11): 2135-2148.
11. Rai S, Rattan V (2012) Traumatic superior orbital fissure syndrome: Review of literature and report of three cases. Natl J Maxillofac Surg 3(2): 222-225.
12. Chao JR, Khurana RN, Fawzi AA, Reddy HS, Rao NA (2006) Syphilis: Reemergence of an Old Adversary. Ophthalmology 113(11): 2074-2079.
13. Clarke H, Clarke V, Gill J, St John A, Lashley M (1992) Painful ophthalmoplegia secondary to a mucocele involving the sella turcica, superior orbital fissure, and sphenoid sinus. J Natl Med Assoc 84(3): 279-280.
14. Lakke JPWF (1962) Superior Orbital Fissure Syndrome: Report of a Case Caused by Local Pachymeningitis. Arch Neurol 7(4): 289-300.
15. Özcan SC, Önder F, Demir N, Özarslan Özcan D (2019) Traumatic superior orbital fissure syndrome. GMS Ophthalmol Cases 9: Doc10.
16. Caldarelli C, Benech R, Iaquinta C (2016) Superior Orbital Fissure Syndrome in Lateral Orbital Wall Fracture: Management and Classification Update. Craniomaxillofac Trauma Reconstr 9(4): 277-283.
17. Traumatic Superior Orbital Fissure Syndrome: 25.
18. Chen CT, Chen YR (2010) Traumatic Superior Orbital Fissure Syndrome: Current Management. Craniomaxillofac Trauma Reconstr 3(1): 9-16.
19. Gabikian P, Chowdhary AM, Kott B, Lazar DA, Britz GW (2012) Isolated Superior Orbital Fissure Syndrome Resulting from Gunshot Wound to the Head. Curr Probl Diagn Radiol 41(4): 112-113.
20. Fujiwara T, Matsuda K, Kubo T, Tomita K, Yano K, et al. (2009) Superior Orbital Fissure Syndrome After Repair of Maxillary and Naso-orbito-ethmoid Fractures: A Case Study. Journal of Plastic, Reconstructive and Aesthetic Surgery 62(12): e565-569.
21. Postma MP, Seldomridge GW, Vines FS (1990) Superior orbital fissure syndrome and bilateral internal carotid pseudoaneurysms. J Oral Maxillofac Surg 48(5): 503-508.
22. Chen CT, Wang TY, Tsay PK, Huang F, Lai JP, et al. (2010) Traumatic superior orbital fissure syndrome: assessment of cranial nerve recovery in 33 cases. Plast Reconstr Surg 126(1): 205-212.
23. Jin H, Gong S, Han K (2018) Clinical management of traumatic superior orbital fissure and orbital apex syndromes. Clin Neurol Neurosurg 165: 50-54.
24. Kjœr I (1945) A Case of Orbital Apex Syndrome in Collateral Pansinusitis. Acta Ophthalmol 23(4): 357-366.
25. Bowerman JE (1969) The superior orbital fissure syndrome complicating fractures of the facial skeleton. Br J Oral Surg 7(1): 1-6.
26. Balakrishnan A, Ebenezer V (2019) Superior Orbital Fissure Syndrome: A Review of Literature. Indian J Public Health Res Dev. 10(12): 1061-1063.
27. Taylor KH, Mizen KD, Spencer N (2010) Isolated fracture of the superior orbital fissure. British Journal of Oral and Maxillofacial Surgery 48(3): 178-179.
28. Evans HH, Wurth BA, Penna KJ (2012) Superior Orbital Fissure Syndrome: A Case Report. Craniomaxillofac Trauma Reconstr 5(2): 115-119.