Unravelling the Frozen Gaze: A rare occurrence of superior orbital fissure syndrome (SOFS) post craniofacial trauma

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SUMMARY

Superior Orbital Fissure Syndrome (SOFS), part of the orbital apex disorders, is characterized by ophthalmoplegia, ptosis, mydriasis and neurosensory disturbance over the ophthalmic branch of trigeminal nerve. We describe an uncommon case of SOFS in a 27-year-old fit and healthy Malay gentleman involved in an industrial injury with severe blunt trauma to his face. He sustained mild traumatic brain injury which was treated conservatively; and multiple facial bone fractures including i) displaced right zygomaticomaxillary complex (ZMC), ii) right frontal bone and superior orbital rim, iii) Lefort I, iv) nasal bone fractures and v) comminuted right orbital walls fracture; with compressed SOF by a displaced lateral wall fracture segment. The patient presented with all the classical features of SOFS with preserved optic nerve (CN II) function. There were restriction of extraocular muscle movements and binocular diplopia in all gazes. Reverse relative afferent pupillary defect (RAPD) was negative. Open reduction internal fixation (ORIF) via bicoronal flap, subciliary and intraoral upper vestibular approach was done. SOFS symptoms of diplopia and ophthalmoplegia showed improvement starting sixth week post-operatively. SOFS may prove challenging to manage due to variable and unpredictable outcomes. With timely surgical decompression and reduction of displaced fracture segments, the prognosis for SOFS may prove favourable.

INTRODUCTION

Superior Orbital Fissure Syndrome (SOFS), also known as Rochon–Duvigneaud syndrome, is part of a larger group of disorders known as orbital apex disorders. It is rather rare with a reported incidence of 0.3-0.8%.[1,2] SOFS is caused by compression of contents within the superior orbital fissure causing impairment of cranial nerve (CN) III, IV, V, VI. It consists of a constellation of classical symptoms including i) ptosis, ii) numbness over the forehead and upper eyelid, and iii) ophthalmoplegia. SOFS does not involve the optic nerve (CN II) and vision is usually preserved, differentiating it from orbital apex syndrome. The etiologies vary and include trauma, most commonly in zygomaticomaxillary complex (ZMC) and orbital wall fractures[3,4]; tumours; infection such as herpes zoster ; and vascular diseases for example, carotico-cavernous fistula or carotid aneurys.[5] It may also occur as a complication of open reduction internal fixation of midface fracture.[6] Trauma-related SOFS usually presents within 48 hours of a craniofacial injury[2], but delayed presentation has also been reported.[7]

To better understand the pathophysiology and mechanism of SOFS, it is crucial to be cognizant of the anatomy. The superior orbital fissure (SOF) is bound laterally by the greater wing of the sphenoid, medially by the lesser wing of the sphenoid, and superiorly by the frontal bone. It serves as a pathway between the orbit and the middle cranial fossa. According to Reymond et al. (2008)[8], the size of the SOF in an adult is approximately 2–3 mm in width at the apex, 7–8 mm at the base and 22 mm in length. The tendons of the lateral rectus muscle divide the fissure into two parts: the superior part containing the trochlear, frontal and lacrimal nerves (branches of CN V), and the superior ophthalmic vein; and the inferior part containing the superior and inferior branches of the oculomotor, abducens, nasociliary branch of ophthalmic nerve, and the inferior ophthalmic vein. Compression of structures that course through the SOF can be attributed to osseous fragment or mass effect from hematoma or oedema in the retrobulbar space or orbital muscle cone. Signs and symptoms of the syndrome may be either complete or partial depending upon the degree of compression of its related anatomical structure. Partial SOFS has been reported where involvement is strictly confined to the central sector, associated with isolated oculomotor, abducens and nasociliary dysfunction[9]; or without complete involvement of the ophthalmic division of the trigeminal nerve and without ptosis.

This report aims to describe an uncommon case of traumatic SOFS and its mechanism; discuss our experience in the management and treatment considerations and outcomes; and review existing literature.

CASE PRESENTATION

A 27-year-old fit and healthy Malay gentleman sustained severe blunt injury to his face caused by an industrial machinery. He was brought to the Emergency Department conscious and alert with a Glasgow Coma Scale of 15. He sustained traumatic brain injury with left frontal contusional bleed which was treated conservatively with close monitoring under the neurosurgical team. He was found to have multiple facial bone fractures including i) right zygomaticomaxillary

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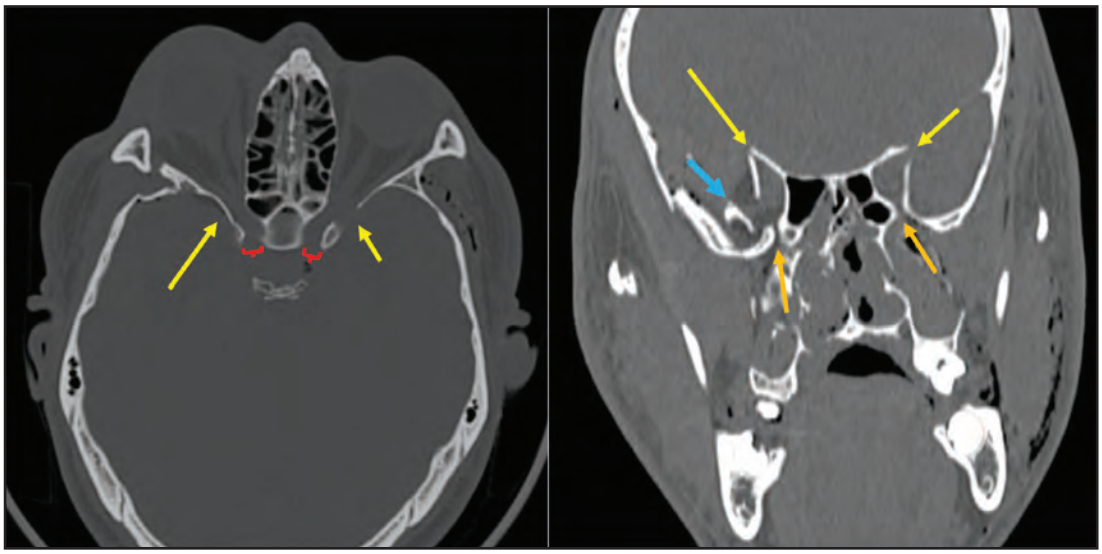

Fig. 1: Axial view of computed tomography showing compressed right superior orbital fissure (long yellow arrow) when compared with normal left superior orbital fissure (short yellow arrow). Optic canals appeared intact (in red). Coronal view showing displaced greater wing of sphenoid bone (blue arrow), compressed right superior orbital fissure (long yellow arrow) while inferior orbital fissures (orange arrows) appeared intact

complex with displaced right zygomatic arch, ii) right orbital walls, iii) Lefort I and II with palatal split, iv) nasal bone and septum, v) right frontal bone and right supraorbital rim fractures.

On examination, the right zygoma, cheek and superior orbital rim regions were depressed with palpable step deformity. Complete anaesthesia was present over the right forehead with an impairment of two-point discrimination. There was hypoesthesia over the right cheek, likely attributed to the Lefort fractures with right infraorbital nerve involvement. Mouth opening was satisfactory at 35mm. Occlusion was deranged with a mobile maxilla but firm mandible.

Comprehensive assessment of the right eye revealed periorbital haematoma, ptosis, mild proptosis, subconjunctival hemorrhage, ophthalmoplegia with complete absence of eye movements in all gazes and a positive forced duction test. Dystopia present was likely due to downward displacement of zygomaticomaxillary complex with right orbital floor blowout fracture and herniation of orbital contents. He also complained of binocular diplopia in all gazes. The right pupil was fixed and dilated at 7mm but with intact vision. Further ophthalmic examination showed loss of corneal reflex and relative afferent pupillary defect was negative. Fundoscopic examination by ophthalmology team was unremarkable and there was no raise in intraocular pressure.

Computed tomography revealed fractures of all walls of the right orbit with constriction of the right superior orbital fissure by displaced osseous fragment of the lateral wall. Bilateral optic canals were patent and intact. There was also evidence of impingement of the medial and inferior rectus muscles by fractured bony fragments. Both globes were intact and no intraconal or extraconal hematoma was noted. A diagnosis of multiple facial bone fractures complicated with superior orbital fissure syndrome (SOFS) was made.

One-week post trauma, open reduction internal fixation (ORIF) was performed for the right superior orbital rim and right zygomatic arch via bicoronal approach; right inferior orbital rim and right orbital floor reconstruction via subciliary approach; maxillary buttress and pyriform rims via intraoral upper vestibular approach. Post-operative forced duction test was negative. Dystopia was corrected and the depressed right malar and supraorbital rim projection were restored. A postoperative occipitomental view radiograph was done to evaluate the reduction.

At the sixth week post-surgery, there was an improvement of ptosis and ophthalmoplegia. Six months after surgery, there was almost complete recovery. Based on literature, a recovery period of up to 12 weeks has been reported and reaches its plateau by the end of 6 months. The patient is still being monitored closely for progression and recovery under the oral maxillofacial surgery and ophthalmology department.

DISCUSSION

The optimal management of traumatic SOFS remains poorly defined and is usually related to treating the cause. Management reported includes conservative treatment with close observation alone, medical management involving steroid administration with or without surgical intervention. In cases of obvious constriction of the SOF by displaced bone fragment, usually as evidenced on computed tomography, decompression surgery is warranted. Variable outcomes have been reported after surgery, including no improvement of SOFS symptoms and gradual recovery up to 12 weeks post-operatively.[2]

ORIF was performed one-week post-trauma to allow resolution of soft tissue oedema. The aims of surgery were to i) anatomically reduce and fix the fractures and restore the malar and superior orbital rim projection, ii) restore the orbital volume and correct the dystopia via orbital floor reconstruction, iii) mechanical decompression and release of

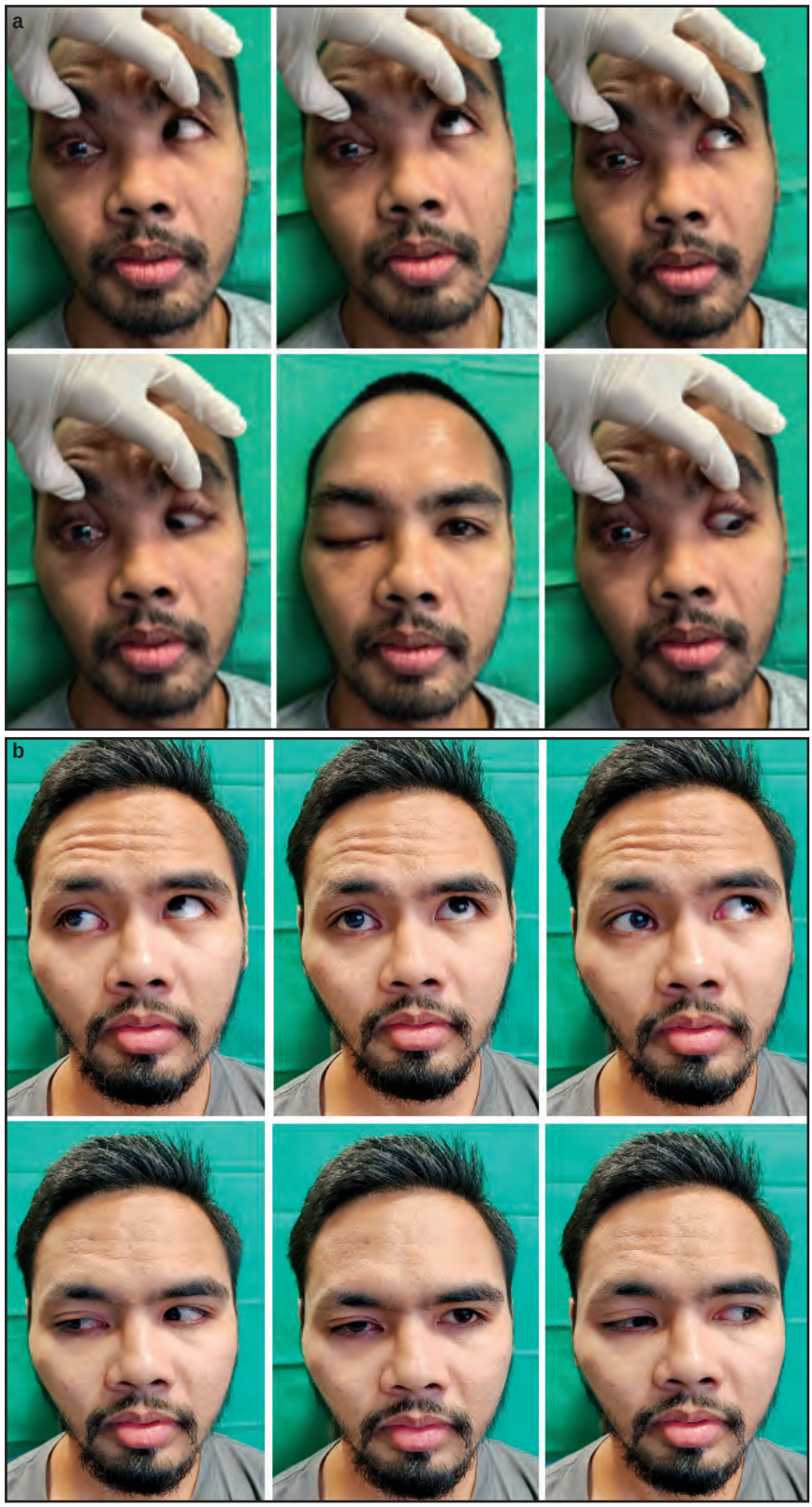

Fig. 2: (a) Post-trauma, right eye ptosis and ophthalmoplegia with complete absence of movement in all gazes showed significant improvement at (b) six months post-ORIF

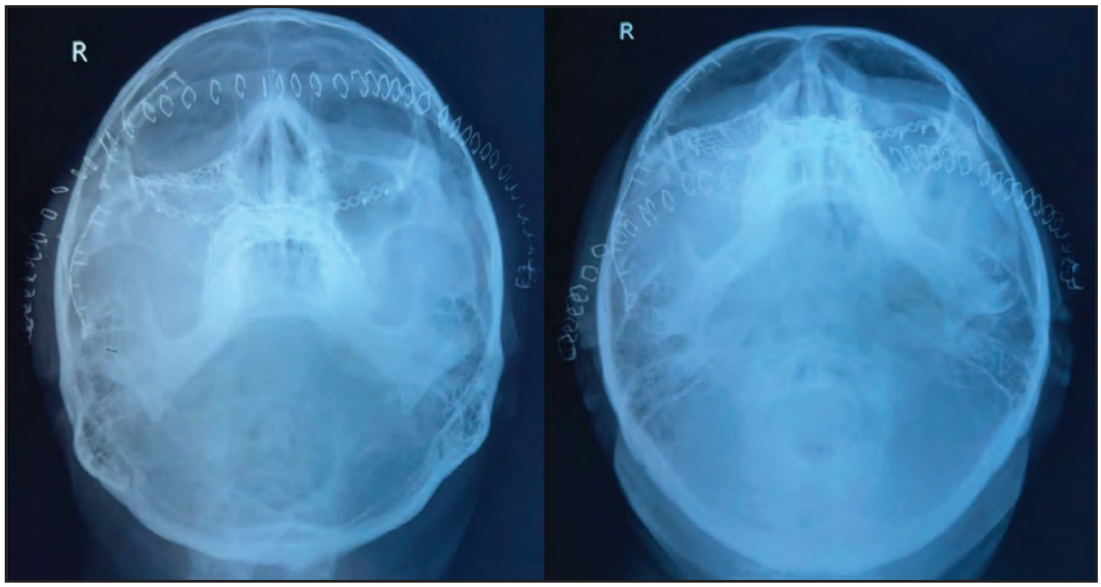

Fig. 3: Occipitomental 30 degrees view and Water's view of skull radiograph showing satisfactory reduction and fixation of facial bone fractures

neuronal contents of superior orbital fissure. According to literature, an initial observation period of 10–14 days before any surgical intervention was carried out is generally advocated due to concerns of raised ocular pressure and swelling.[9] Oedema developed immediately post-trauma may directly compress on the nerve or push the nerve against the bony margin of the fissure, which may attribute to nerve palsy and its subsequent transient dysfunction. Naturally narrower superior orbital fissure has been reported as a risk factor for SOFS.[10]

The pathophysiology or mechanism of injury can be broadly divided into neurological and/or muscular injury. Any entrapped extraocular muscles should be mechanically released and can be confirmed with a negative forced duction test. If decompressive surgery is delayed, the muscles may undergo compressive ischaemic injury and become irreversibly damaged from fibrosis. Studies have shown that early intervention in cases of muscle entrapment resulted in less postoperative diplopia.[11]

If ophthalmoplegia persists despite release of entrapped muscles, the cause is likely neurological in origin due to cranial nerve palsies. Neurological damage may be due to direct blunt trauma to cranial nerves III,IV,VI or indirect compression of nerves by osseous fragment or hematoma or oedema within the limited intraconal space.[12,13] Transmitted traumatic forces on the cranial nerves along its course through the superior orbital fissure may lead to axonal injury of varying severities, either neuropraxia, axonotmesis or neurotmesis. Tolerance of the nerve to injury and its reversibility depend on the type, diameter, location and direction of nerve course. Spontaneous improvement with just conservative management and careful observation has been documented in cases of ophthalmoplegia due to oedema without evidence of displaced bony fragment and compressed SOF.[2]

Varying doses of systemic corticosteroids have been advocated as treatment alone, or in conjunction with surgical decompression of facial bone fractures. Initial steroid therapy post-trauma has been shown to have variable results, generally with a favourable prognosis.[2,9,14] Different regimes of steroid administration, usually involving 'megadose', have been reported.[5,14] The mechanism appears to be from the ability of high doses steroid in rapidly reducing oedema in the limited intraconal space of the orbit and subsequent ischemia of delicate neuronal structures. Specific to this case, a routine peri-operative dose of IV Dexamethasone 8mg 8 hourly was administered intraoperatively and up to 3 days post-operatively in addition to eye drops of 0.1% dexamethasone 6 hourly. Corticosteroid, associated with morbidity in patients with traumatic brain injury, as reported by the Corticosteroid Randomisation After Significant Head injury (CRASH) trial protocol; was not administered in the acute setting pre-operatively in this case.[15]

In conclusion, superior orbital fissure syndrome may prove challenging to manage due to variable and unpredictable outcomes. With timely surgical decompression and reduction of displaced fracture segments, the prognosis for SOFS is generally favourable. Randomized clinical trial is recommended to further evaluate the efficacy of high dose steroids for SOFS in the acute setting . This case report may add value to the existing literature, owning to its scarcity.

CONFLICT OF INTEREST
None.

FUNDING
None.

ETHICAL APPROVAL
None.

PATIENT CONSENT

Written consent was obtained from the patient including the use of anonymized medical data and images for the publication of this case report, ensuring compliance with ethical standards.

STATEMENT TO CONFIRM

All authors have viewed and agreed to the submission.

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