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Traumatic superior orbital fissure syndrome: a rare case report

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ABSTRACT

Superior orbital fissure syndrome is a severe clinical entity characterized by injuries of neurovascular structures passing through the superior orbital fissure. A 38-year-old male patient admitted to outpatient clinic with left upper eyelid ptosis, ecchymosis, mydriasis, hypoesthesia in upper eyelid and frontal region, blepharoptosis, restriction of ocular movements in all directions, loss of accommodation reflex and absence of direct pupillary reflex. Three-dimensional computed tomography scans demonstrated left-sided orbital fractures resulting in obliteration of the superior orbital fissure. He underwent surgical decompression. The patient had intravenous methylprednisolone administration at preoperative and postoperative periods during hospitalization. He was discharged on the postoperative day 5. At the 9th postoperative month, the cranial nerve functions were almost completely recovered. We consider that the decompressive surgery in conjunction with perioperative steroid therapy may be effective for the traumatic superior orbital fissure syndrome.

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Keywords: Superior orbital fissure syndrome; cranial nerve; ophthalmoplegia; orbital trauma; steroid; surgical decompression

Introduction

Superior orbital fissure syndrome (SOFS) is a rare and severe clinical entity characterized by traumatic or non-traumatic injuries to the cranial nerves (CN III, IV, V1 and VI) along with the autonomic nerves and vascular structures passing through the superior orbital fissure [1, 2].

The etiological factors associated with the SOFS can be divided into three categories: traumas, tumors and infections [3]. Numerous etiological factors including carotid-cavernous sinus fistula [1], maxillofacial traumas [1, 3-5], pseudoaneurysm of the internal carotid artery [6], orbital metastatic hepatocellular carcinoma [7], aspergillus sinusitis [8], etc., were noted as the possible causes of the SOFS. Regardless of the underlying etiological factors, the clinical symptoms of the syndrome are primarily the results of the inflammation and the compression of the neurovascular structures in the superior orbital fissure [4].

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In this report, we aimed to share our clinical experience regarding the treatment and the follow-up of a patient with traumatic SOFS and to draw attention of the clinicians to the importance of its association with the maxillofacial traumas.

Case Presentation

A 38-year-old male patient, who had a motor vehicle accident 10 days previously, admitted to the outpatient clinic of plastic and reconstructive surgery with the complaint of being unable to open his left upper eyelid. As a result of blunt chest trauma, he had developed a left-sided pneumothorax, treated with chest tube drainage, and had been followed-up in another hospital's intensive care unit for 7 days. The patient stated that, although he had a concomitant head trauma. Neither physical examination nor imaging studies were performed during the follow-up period for this situation.

Physical examination of the left eye revealed ecchymosis, subconjunctival hemorrhage, mydriasis, hypoesthesia in the upper eyelid and the frontal region, blepharoptosis, restriction of the ocular movements in all directions and loss of accommodation reflex, loss of corneal reflex and absence of direct pupillary reflex. Due to the fractures of the left frontozygomatic region, the patient had tenderness on palpation and step deformities were palpated. Ophthalmological examination did not reveal any signs of optic neuropathy, bruit, chemosis or displacement of the globe (Figure 1).

Three-dimensional computed tomography scans demonstrated left-sided fracture lines involving the frontozygomatic region and greater wing of the sphenoid bone resulting in en-bloc displacement of the lateral orbital wall into the orbital cavity and obliteration of the superior orbital fissure (Figure 2). Magnetic resonance imaging of the left orbita did not reveal any pathological findings of the globe or the optic nerve.

For surgical decompression, the patient was operated under general anesthesia. A subgaleal bicoronal flap was raised, extending laterally to the supraauricular areas and anteriorly to the frontal

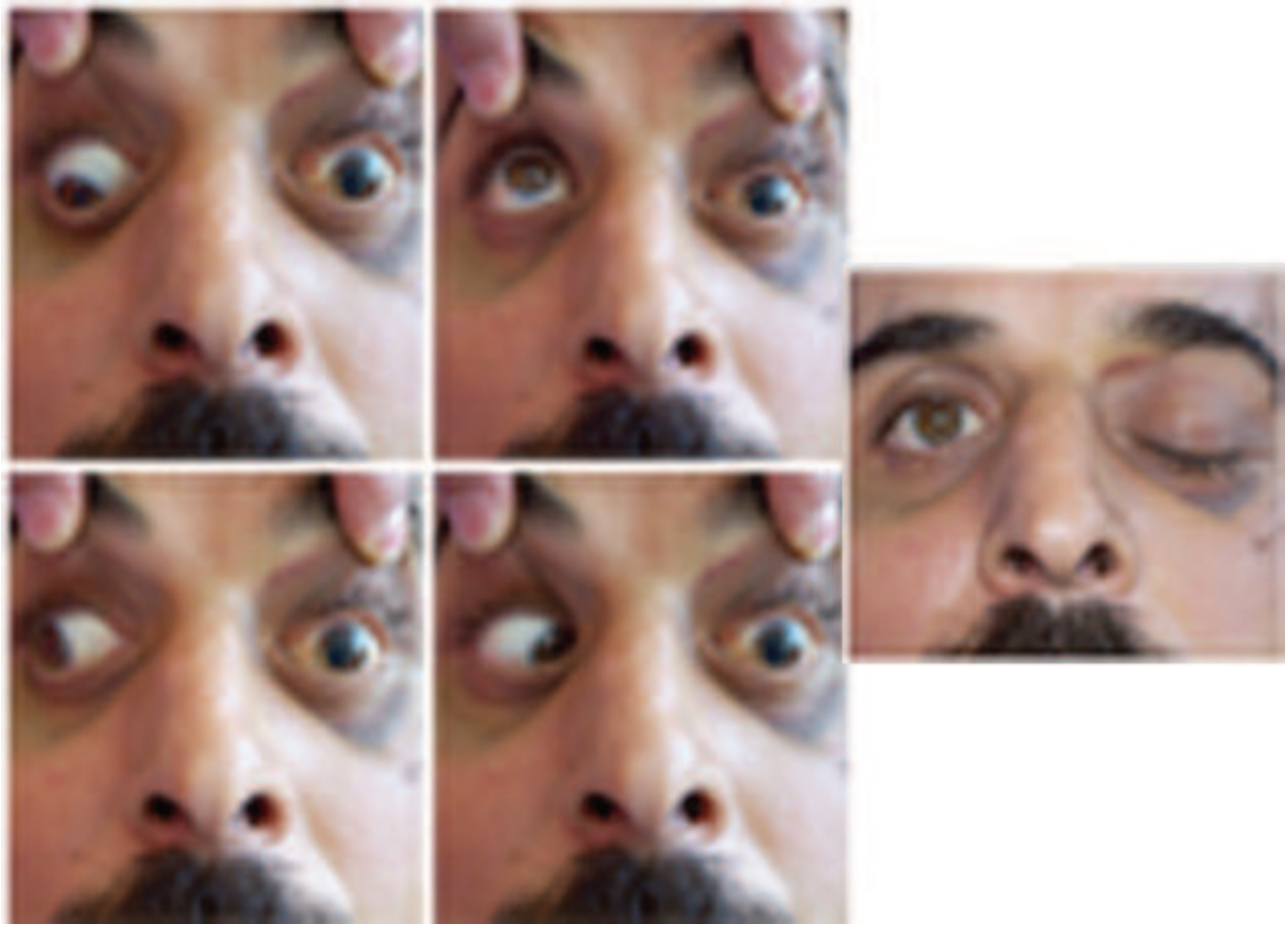

Figure 1. Due to total ophthalmoplegia of the left eye, the patients had blepharoptosis, mydriasis and restriction of the left-eye movements in all directions.

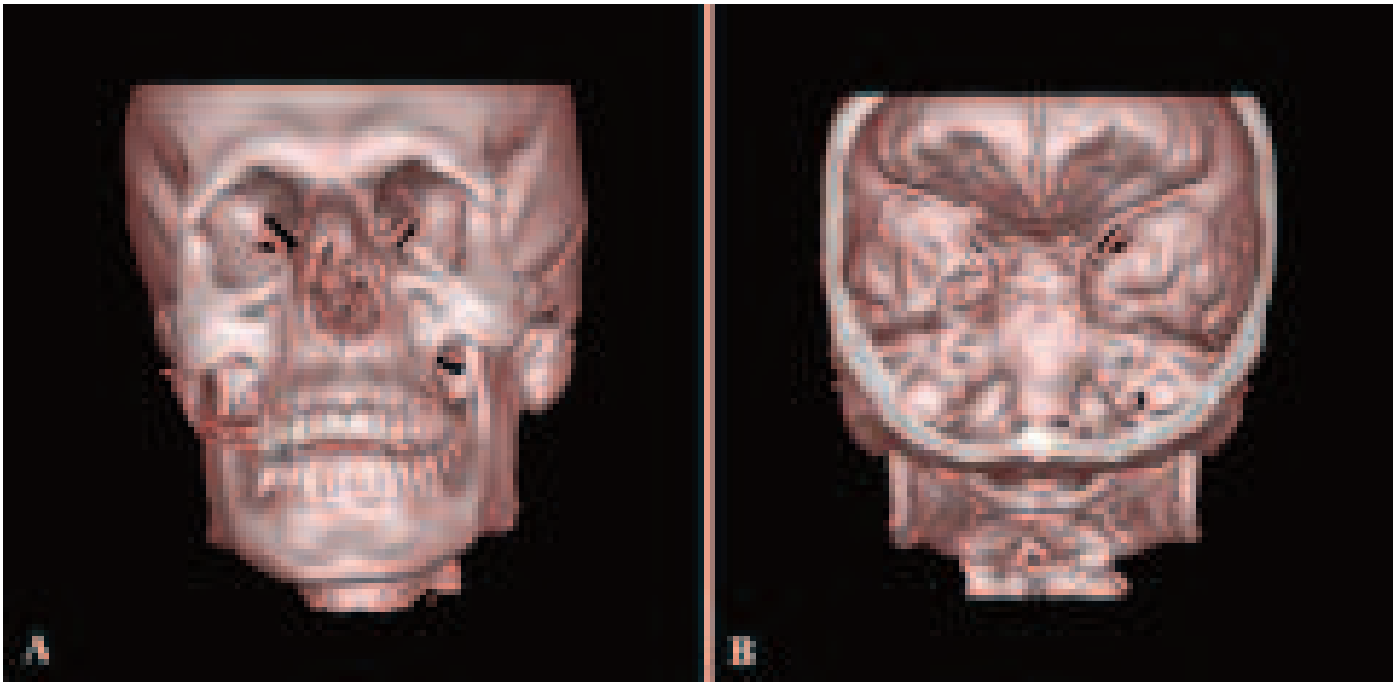

Figure 2. Preoperative 3D CT scans of the skull. (A) An anterior view demonstrates the obliterated superior orbital fissure (arrow) as a result of a complex fractures displacing the lateral orbital wall medially and (B) posterior view of the obliterated superior orbital fissure (arrow). 3D CT=three-dimensional computed tomography.

region. Through a periostal incision located superiorly to the left supraorbital bar, the subperiostal plane was reached and dissected till the lateral orbital wall and the zygomatic arc were exposed. A neurosurgeon then performed a left frontal craniotomy and lateralized the medially displaced lateral orbital wall to expose the orbital cavity. The medially impacted greater wing of the sphenoid bone was thereby lateralized and the superior orbital fissure was decompressed. The rigid fixation of the craniotomy flap was then subsequently performed, along with the rigid fixation of the fracture of the frontozygomatic suture line. After surgical hemostasis, a Jackson-Pratt drain was placed, the bicoronal flap was sutured and the procedure was successfully completed without any complication. The patient had intravenous methylprednisolone administration (1 mg/kg once daily) at pre-operative and post-operative periods till discharge from the

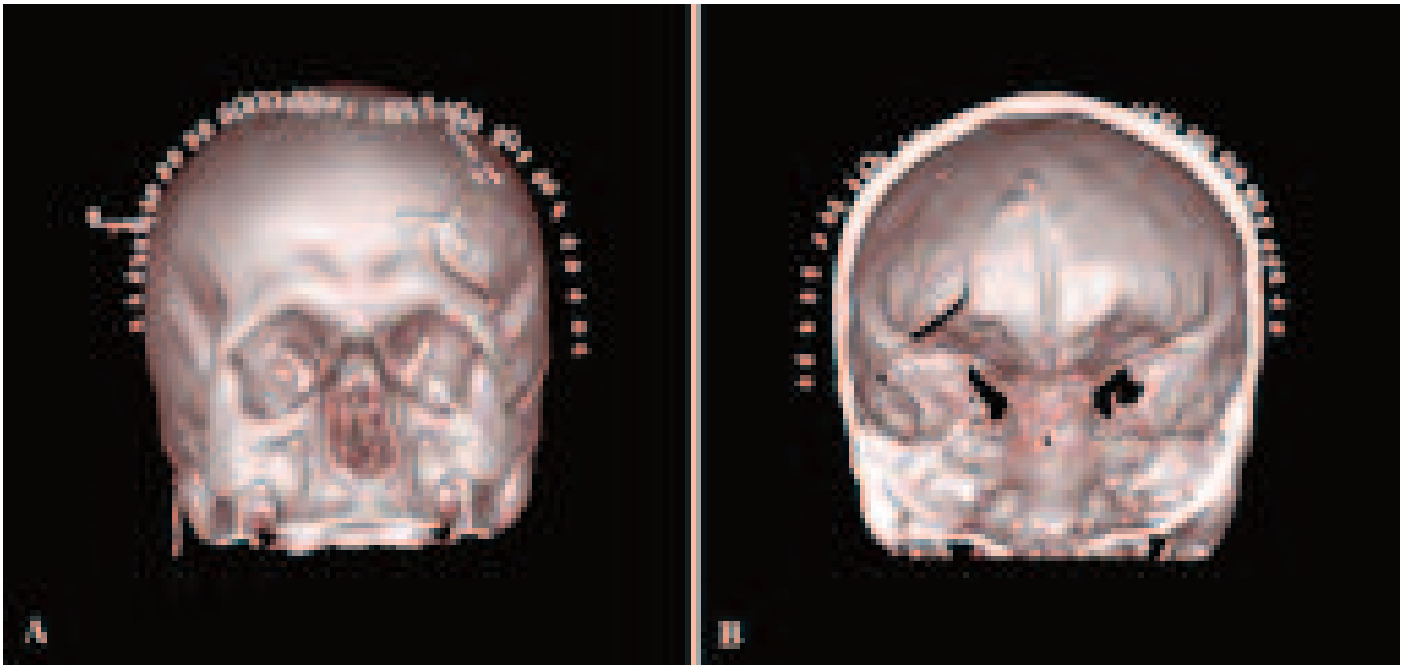

Figure 3. Postoperative 3D CT scans. A. Anterior (arrow) and B. posterior view of the reduced fracture lines and decompressed superior orbital fissure (arrow). 3D CT=three-dimensional computed tomography.

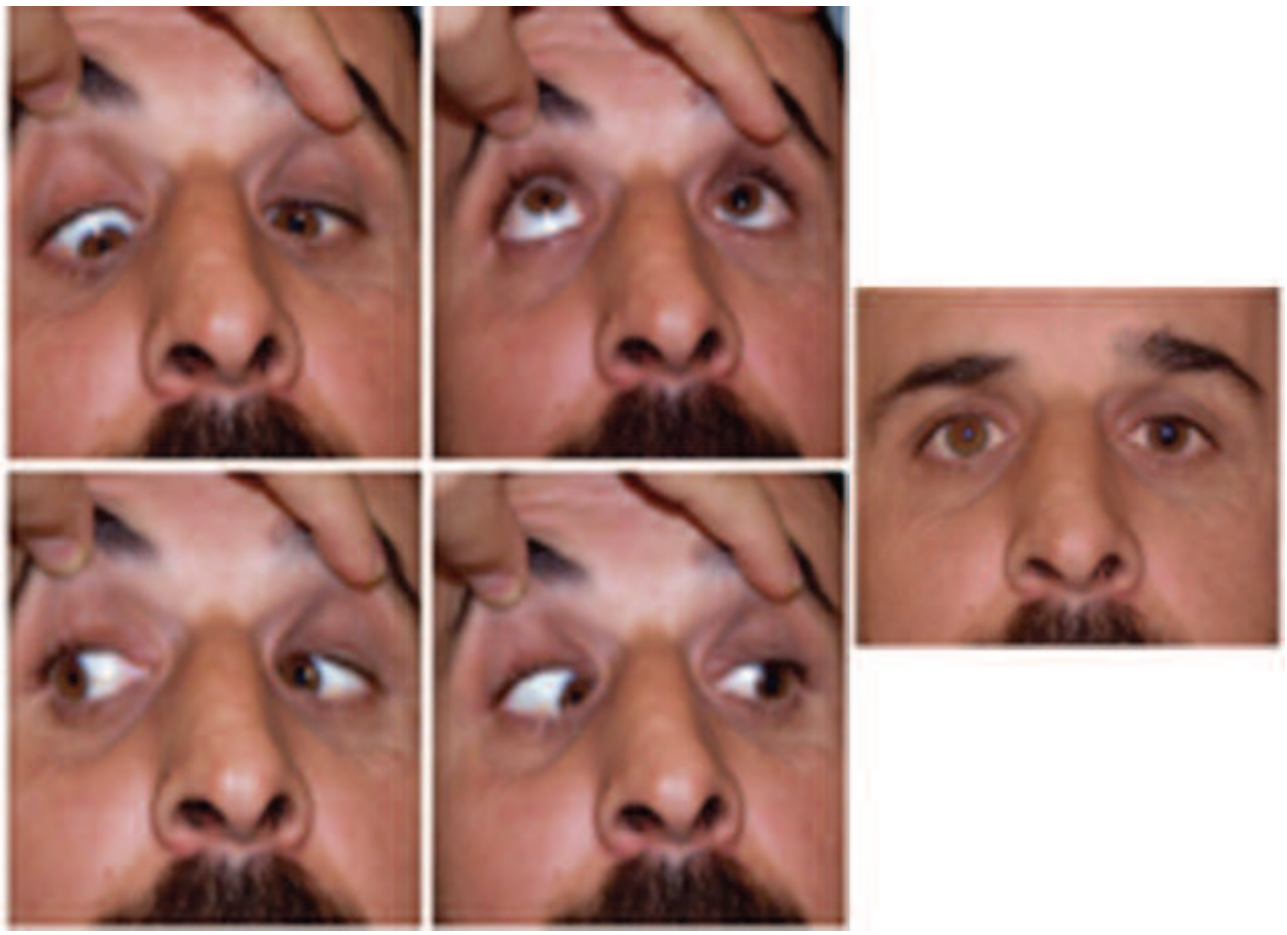

Figure 4. Postoperative examination on the 2nd month demonstrating minimal restriction in elevation and depression movements of the left eye with minimal mydriasis.

hospital. On the 1st post-operative day, three-dimensional computed tomography scans demonstrated that the former fracture lines were reduced and the superior orbital fissure was larger in size (Figure 3). The patient was discharged on the 5th post-operative day on medication regimen included artificial eye drop, analgesic, antibiotic and reduced dosage of oral methylprednisolone.

Physical examination performed at the 2nd postoperative month revealed partial recovery of the sensory and motor functions of the cranial nerves; elevation and depression movements of the left eye were minimally restricted and minimal mydriasis was still present (Figure 4).

At the 9th post-operative month, the cranial nerve functions were almost completely recovered with the only pathological findings being 2-mm restriction of the elevation movement of the left eye and persistent minimal mydriasis (Figure 5).

Discussion

Superior orbital fissure is an important anatomic structure that lies between the superior and lateral orbital walls and serves as a conduit between the orbital cavity and the middle cranial fossa. Oculomotor nerve (CN III), abducens nerve (CN VI), nasociliary branch of the ophthalmic nerve (CN V1) and sympathetic-parasympathetic nerve fibers together reach the orbital cavity by passing through the intra-conal compartment of the superior orbital fissure, which was firmly bounded by the annulus of Zinn. Also, lacrimal and frontal branches of the ophthalmic nerve (CN V1), trochlear nerve (CN IV) and superior ophthalmic vein are the main neurovascular structures of the extra-conal compartment of the superior orbital fissure (Figure 6) [9, 10].

SOFS is a rarely described symptom complex due to compression of the compartments of the superior orbital fissure. Clinical presentation may vary from

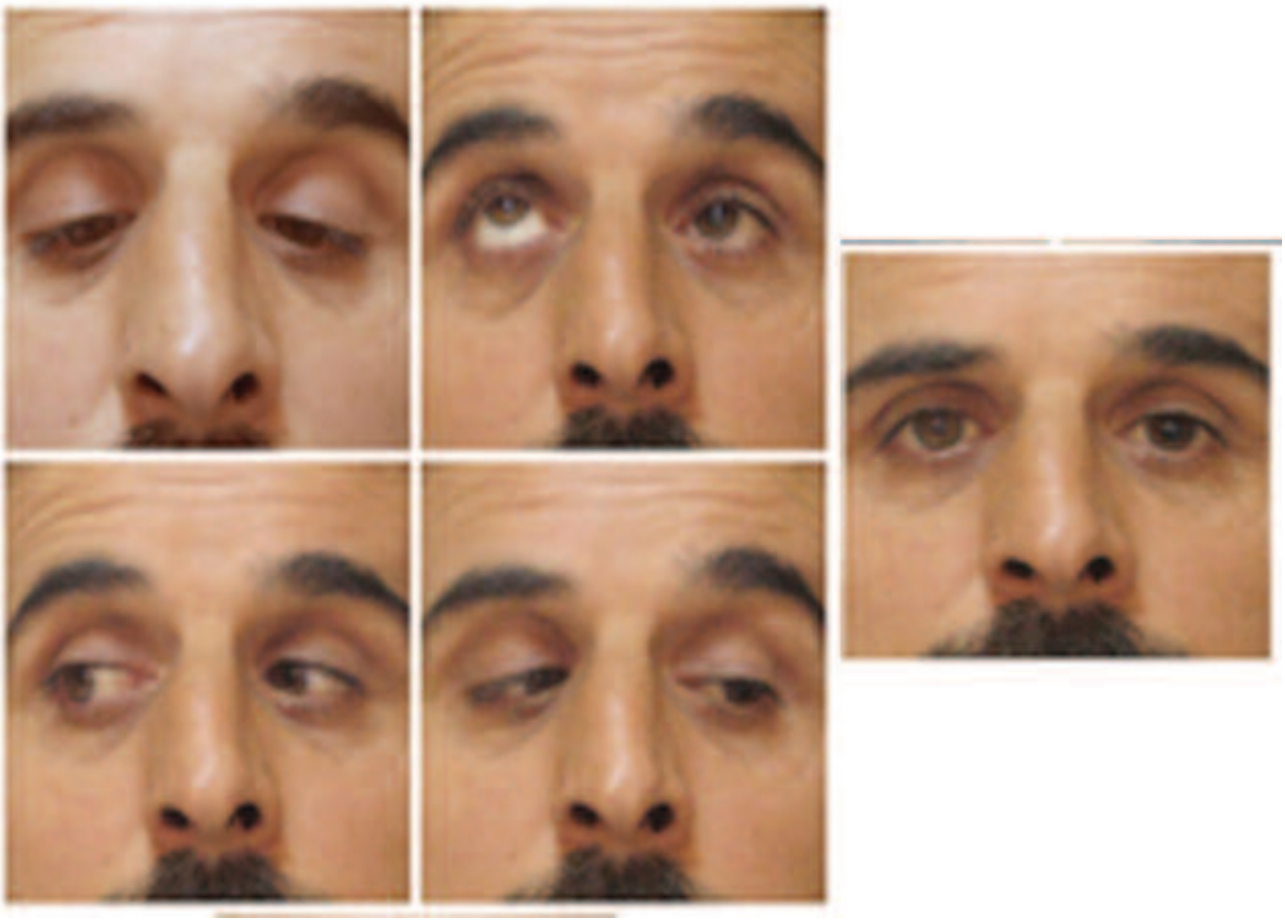

Figure 5. Postoperative examination on the 9th month demonstrating 2-mm restriction of the elevation movement of the left eye and persistent minimal mydriasis.

partial to complete neurologic sequelae depending on the degree of the injury. The clinical findings of the syndrome may include upper eyelid ptosis (due to sympathetic denervation of the Mueller muscle or involvement of the CN III resulting in paralysis of the levator palpebrae superioris muscle); ophthalmoplegia (due to paralysis of the CN III, IV and VI); mydriasis, loss of accomodation reflex, loss of direct pupillary reflex (due to involvement of the CN III and its accompanying parasympathetic fibers resulting in

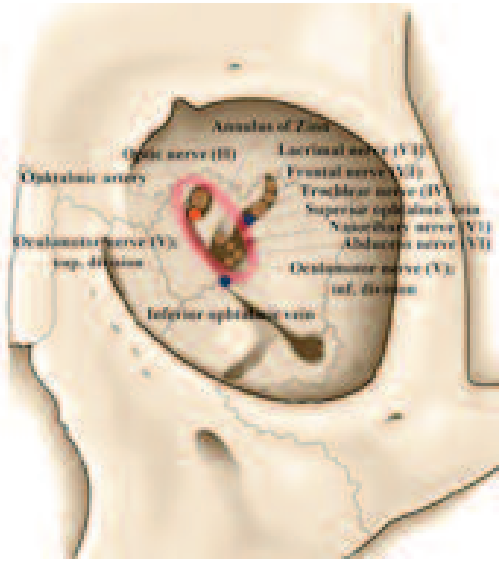

Figure 6. An illustration of the left superior orbital fissure, along with the anatomic structures passing through it.

ciliary muscle paralysis); loss of sensation in the region of the globe, upper eyelid and forehead region and loss of corneal reflex (due to CN V1 paralysis) and proptosis (due to loss of extraocular muscle tone) [1, 4, 9].

Approximately 0.3% of the maxillofacial fractures were reported to be associated with the traumatic SOFS and the most common fractures were described as Le Fort II-III, orbital and zygomaticomaxillary complex fractures [1, 3-5].

The most important clinical consideration in differential diagnosis of the SOFS is the orbital apex syndrome which presents with the findings of the SOFS and varying degrees of visual loss due to optic neuropathy [2]. Also, carotid-cavernous sinus fistula, an abnormal communication between the cavernous sinuses and the carotid arterial system, may mimic the presentation of the SOFS with additional signs and symptoms such as orbital or frontal bruit, chemosis, pulsating exophtalmus, loss of visual acuity, etc [11]. Therefore, in cases of suspected SOFS, it is crucial to perform a proper ophthalmologic examination to rule out the orbital apex syndrome and carotid-cavernous sinus fistula. In the presented case here, there was no evidence of optic involvement, orbital/frontal bruit, chemosis or exophtalmus, excluding orbital apex syndrome and carotid-cavernous sinus fistula.

In the traumatic SOFS, two mechanisms are responsible for the compression of the neurovascular structures of the superior orbital fissure; (1) an indirect mechanism, occurs due to presence of intraorbital edema and hemorrhage resulting in increased intraorbital pressure (indirect traumatic SOFS), (2) a direct mechanism, occurs due to displacement of the bone fragments resulting in narrowing of the superior orbital fissure (direct traumatic SOFS) [1]. Due to the previously reported cases of the traumatic SOFS, a consensus on the treatment algorithm does not currently exist. However, in our opinion, treatment may be effective if targeted to the mentioned pathophysiologic mechanisms of the syndrome.

In the literature, some authors suggest the possibility of recovery with observation or steroid therapy alone and some advocate the use of surgical decompression in cases with displaced sphenoid bone fragments obliterating the superior orbital fissure. In a clinical study of 33 cases [1], clinical observation (n=22) or mega-dose methylprednisolone administration (n=5) were the treatments for indirect traumatic SOFS. Of the cases that treated with steroid administration, 2 had complete recovery of the cranial nerve functions. Also, 6 cases, which were treated surgically for displaced sphenoid bone fragments, demonstrated similar recovery at the follow-ups. The authors stated that the role of the steroids is unclear for the treatment of the SOFS and surgical decompression may be beneficial to those cases with evidence of displaced sphenoid bone fragments.

In another clinical study by Acarturk *et al* [5], 11 cases with SOFS or orbital apex syndrome were completely recovered by intravenous administration of mega-dose methylprednisolone. None of these cases had displaced bone fragments in the superior orbital fissure and 8 had undergone surgery for the treatment of the concomitant maxillofacial fractures. Reduction of the fractures may have a positive effect regarding decompression of the superior orbital fissure and disappearance of the clinical signs and symptoms. Therefore, despite authors' suggestions, the effect of the mega-dose methylprednisolone treatment in the management of the SOFS does not seem obvious in this article.

Conclusion

SOFS is an important entity to recognize clinically and it is characterized by the presence of the signs and symptoms including ophthalmoplegia, ptosis, proptosis, mydriasis, hypoesthesia at the upper eyelid and forehead region, etc. In the presented case, the presence of severe craniofacial trauma had been over-looked during the case's follow-up period in another hospital; illustrating the importance of the careful evaluation of the multi-trauma cases for any concomitant maxillofacial traumas. In the light of our experience with this rare case, we consider that the decompressive surgery in conjunction with the perioperative steroid therapy may be the treatment modality for the direct traumatic SOFS. Future case reports and series of SOFS may aid in developing a consensus on the treatment of this syndrome and we wish that the case presented will contribute to this aim.

Informed consent

Written informed consent was obtained from the patient for the publication of this case report.

Conflict of interest

The authors declared that there are no potential

conflicts of interest with respect to the research, authorship, and/or publication of this article.

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