TRAUMATIC SUPERIOR ORBITAL FISSURE SYNDROME - A CASE REPORT

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CASE REPORT

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Abstract

The superior orbital apex syndrome is a relatively uncommon complication of midface maxillofacial trauma. The clinical symptoms consist in ophthalmoplegia, palpebral ptosis, exophthalmia, fixed mydriasis, retrobulbar pain and supraorbital nerve hypoesthesia by involvement of the third (oculomotor nerve), fourth (trochlear), fifth (trigeminal) and sixth nerve (abducens). If there is involvement of the optical nerve, the syndrome is termed - orbital apex syndrome. In this article, we will present the case of a 33-years old male, victim of human aggression with traumatic superior orbital apex syndrome. We discuss details of diagnosis and surgical treatment. We will make, also, a review of literature on this subject. Even if the actual therapeutic algorithm is currently a matter of controversy, the generally accepted therapy plane initiated with a high dose of corticosteroids. Fine slice CT scan examination is mandatory for the correct planning. If the CT scan reveals a highly displaced maxillo-zygomatic complex fracture with or without orbital blow-out fracture, we recommend early surgical intervention after the resolving of the periorbital hematoma within 5 to 10 days ideally if concomitant intracranial injury or other conditions permit it. The early restoration of the orbital anatomy and volume will create the basis for cranial nerve decompression and function at the level of superior orbital fissure.

Keywords: trauma, superior orbital fissure, surgery

Introduction

The superior orbital apex syndrome is a relatively uncommon complication of midface maxillofacial trauma (1 in 130 cases according to the literature). The clinical symptoms were first described by JP Lake in 1962 and they consist in – ophthalmoplegia, palpebral ptosis, exophthalmia, fixed mydriasis, retrobulbar pain and supraorbital nerve hypoesthesia by involvement of the third (oculomotor nerve), fourth (trochlear), fifth (trigeminal) and sixth nerve (abducens). If there is involvement of the optical nerve,

the syndrome is termed - orbital apex syndrome (1). From an anatomical point of view, the superior orbital fissure is a passage of communication between the orbit and the medial cranial fossa. The bones that are involved in the anatomical delimitation are: laterally the greater wing of the sphenoid bone, medially the lesser wing of the sphenoid bone and the superior wall is part of the frontal bone. It has a triangular shape, rotated at an angle of 45 degrees with the base at the nasal side (Figure 1). The optical foramen is located medially on the lesser wing of the sphenoid bone. It is quite narrow – at approximately 2 mm in the superior narrow aspect and about 8 mm at the basal broader part, with a length of nearly 22 mm [1,2].

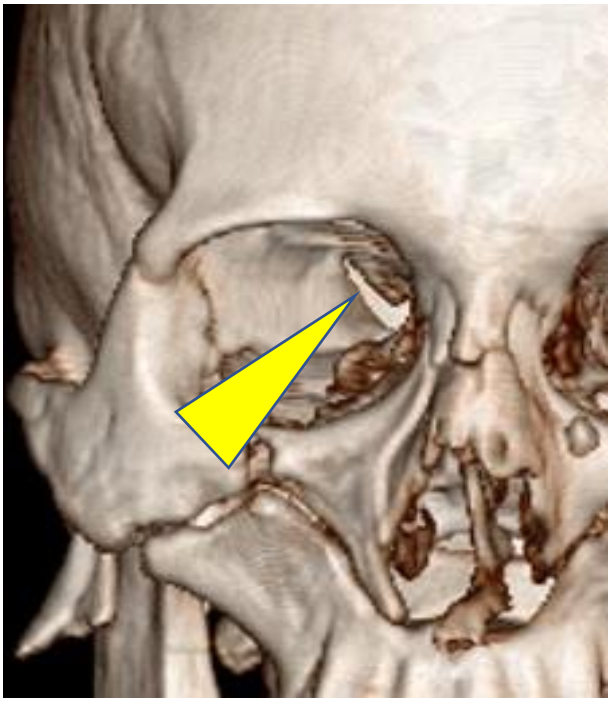

Figure 1 - Orbital superior fissure

As mentioned earlier, the anatomical contents of the orbital fissure explain the symptoms associated with orbital trauma at this level. The symptoms can vary depending on the intensity of the kinetic force involved in the trauma – and the severity of the fracture. The symptoms can be partial or complete (Figure 2, Figure 3). Ophthalmoplegia can be explained by lesions involving the oculomotor, trochlear and abducens nerve. The palpebral ptosis is due to impairment of the superior branch of the oculomotor nerve. Mydriasis is secondary to trauma of the parasympathetic fibres which course along the oculomotor nerve [3]. Exophthalmia is secondary to loosening of the extraocular muscle tone, which normally retract the globe in anatomical position [4]. The frontal area and superior eye lid hypoesthesia, retro-orbital pain is secondary to the impairment of the lacrimal and ophthalmic branches of the trigeminal nerve V1 branch [5].

Case presentation

A 33-years old male victim of human aggression, reported to our emergency unit department with swelling over the left zygomatic arch, inability to move the left eye, numbness of the infraorbital and frontal area. The ophthalmic examination revealed mydriasis of the left pupil, complete absence of eyeball abduction movement, partial absence of movement in the superior, inferior and medial gaze, partial palpebral ptosis. The patient was without impairment of the visual acuity, with loss of accommodation reflex, but with normal fundoscopic findings. Also, the patient presented with palpebral ptosis and proptosis.

The computerized tomography (CT) scan revealed left eye exophthalmia without retrobulbar hematoma. There was comminute fracture of the orbito-zygomatic-maxillary complex, associated with orbital floor blow-out fracture with herniation of the orbital contents into the maxillary sinus. Also, the CT scan showed a fracture line intersecting the spheno-zygomatic suture and rectus lateralis incarceration. Also, there was some haemorrhagic areas in the frontal lobe.

The patient was managed initially with systemic corticoids – dexamethasone 8

mg/12 h, B1and B12 vitamin therapy, Ceftriaxone antibiotic therapy. There was partial improvement in the palpebral ptosis and exophthalmia after 7 days of treatment, and almost complete haematoma resorption (Figure 4).

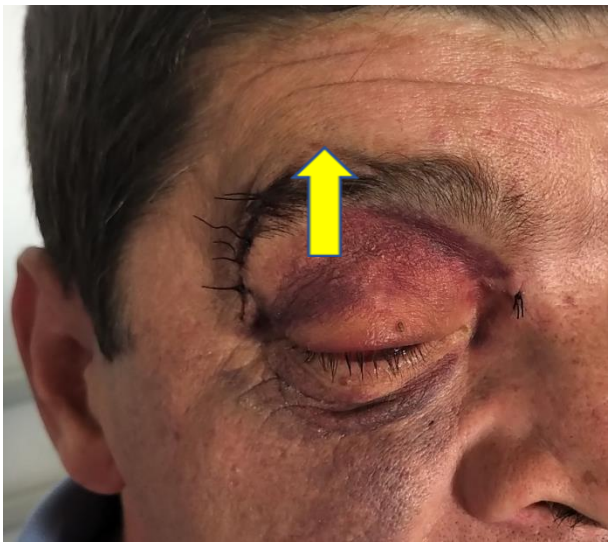

Figure 2 - Complete Superior Orbital Fissure Syndrome – palpebral ptosis

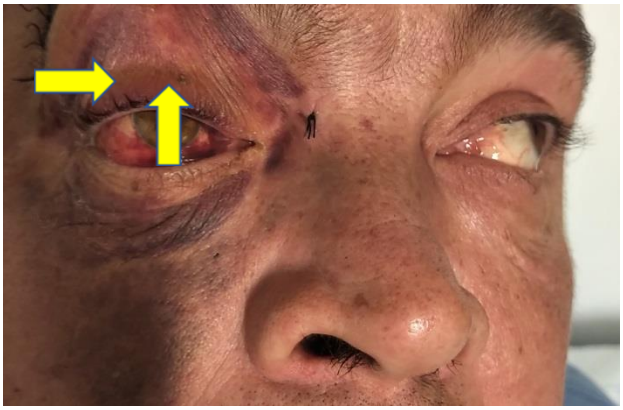

Figure 3 - Complete ophthalmoplegia, proptosis

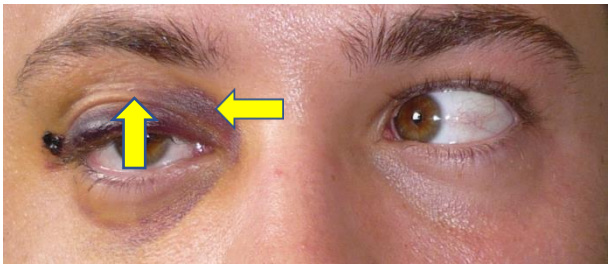

Figure 4 - Partial SFOS – partial left palpebral ptosis, ophthalmoplegia

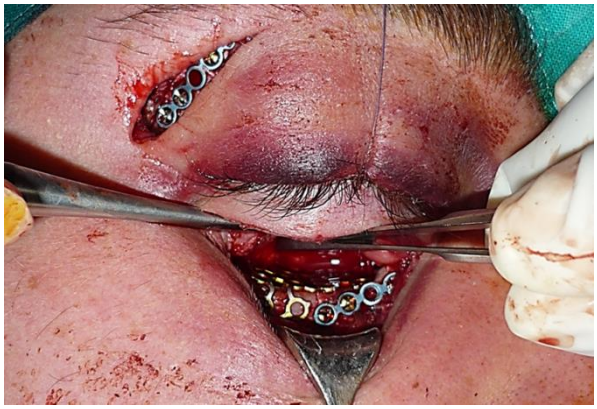

Figure 5 - Intraoperative aspect - orbital reconstruction with titanium plates

The surgical intervention was done on the 8th day after the admission. We performed the intervention under general anaesthesia and the access was combined - external orbital and oral approach. The first approach was lower eyelid sub tarsal, in a natural skin crease, with sectioning of the orbicularis muscle fibres. The dissection was carried superficial to the orbital septum down to the inferior orbital rim which presented a severe displacement and comminution of the bony fragments. The subperiosteal dissection was extended along the orbital floor which revealed a large bony dehiscence at this level – with gross herniation of the orbital fat into the maxillary sinus. The second incision was made at the medial aspect of the frontozygomatic suture, with preservation of the facial nerve. The dissection was carried deep to the fronto-zygomatic suture – which presented the inward and downward displacement of the zygomatic bone component of the suture. The last incision was made intraorally in the maxillary vestibulum, a mucoperiosteal flap was reflected so that the Dingman forceps can be used to disimpact the fragments of the fractured body of the zygoma.

After the satisfactory reduction of the fragments, the rigid fixation was obtained using titanium screws and plates placed first at the fronto-zygomatic suture and secondly, at the infraorbital margin. The orbital floor

reconstruction was performed after the repositioning of the herniated orbital contents in the orbital cavity and fixation using a titanium orbital preformed mesh (Figure 5). After the normal anatomy and orbital volume was obtained – the continuity of the sectioned orbicularis muscle was meticulously sutured in place, and perfect haemostasis was obtained to decrease the risk of hazard postoperative haemorrhage.

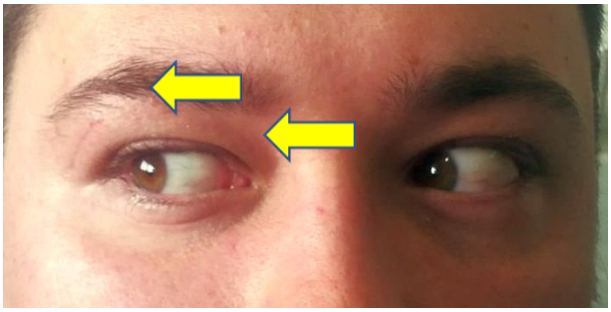

Figure 6 - Postoperative aspect with normal eye movement

Postoperatively, the patient received 4 mg/Dexamethasone twice per day and antibiotics (twice daily) with significant improvement of the symptoms after 72 hours, notably regarding the almost complete recovery of the ocular mobility. Also, the patient reported an improvement of the infraorbital and supraorbital hypoesthesia at approximately 7 days postop (Figure 6).

Discussions

Diagnosis of posttraumatic superior orbital apex fissure aside the clinical symptoms includes fine cut CT scanning, which can visualize the line of fractures, the displaced bony fragments, the retrobulbar compressive hematoma [2,5].

There is still controversy regarding the best treatment algorithm, due to the low incidence of this complication. In literature the incidence is reported at approximately 1 case in every 130 maxillofacial trauma case. Some authors speculate that a surgical intervention can aggravate the nerve damage by bony displacement and suggests only high dose corticosteroid therapy [6]. Other trauma centres favour open reduction and fixation of the orbital fracture [7]. Open reduction of orbital fractures associated with SFOS syndrome in conjunction with corticoids may increase the chance of decompression of the nervous and vascular structures located at the apex of the orbit, with favourable resolution of the associated symptoms [8].

The average timing for the orbital reconstruction varies from 1 to a maximum of 14 days according to the data in the literature [2,4,9]. In our centre, we prefer to perform surgery after 5 to 8 days after the traumatic moment. after a high dose cure of corticosteroids and the subsequent resolution of the orbital hematoma. We didn't experience any aggravation of the neurological symptoms after the open surgery.

In case of compressive retrobulbar hematoma associated with progressive visual acuity loss or worsening symptoms of SOFS after cortisone megadose, the decompression should be done as soon as possible, ideally under 24 hours from the onset of the neurological symptoms, either by open surgery (sub tarsal or sub ciliary access or superior lateral orbitotomy) or via endonasal endoscopic approach [8].

In our case report, the symptoms of SFOS were present in various degrees and solved partially after a megadose of corticosteroids. The surgical treatment offered in this case a rapid resolution of some of the associated symptoms by reconstructing the normal volume and anatomy of the orbit and progressive improvement of the symptoms of eye lid ptosis, facial hypoesthesia and diplopia within a few months after the surgical and corticosteroid treatment.

Conclusion

SOFS is an extremely rare, but debilitating complication of orbito-maxillo-zygomatic fractures. Even if the actual therapeutic algorithm is currently a matter of controversy, the generally accepted therapy plane initiated with a high dose of corticosteroids. Fine slice CT scan examination is mandatory for the correct planning. If the CT scan reveals a highly displaced maxillo-zygomatic complex fracture with or without orbital blow-out fracture, we recommend early surgical intervention after the resolving of the periorbital hematoma within 5 to 10 days ideally if concomitant intracranial injury or other conditions permit it. The early restoration of the orbital anatomy and volume will create the basis for cranial nerve decompression and function at the level of superior orbital fissure.

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