Traumatic Superior Orbital Fissure Syndrome: Case Report

Travmatik Superior Orbital Fissür Sendromu: Olgu sunumu

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Abstract: The superior orbital fissure syndrome (SOFS) is a very rare condition, especially due to trauma and it's therapeutic managements are still controversial. We present a case of SOFS and discus the timing and variety of surgical approaches. Although in most cases, a partial to complete spontaneous recovery of SOFS is to be expected, this case supports that decompressive surgery is the most effective method and must be performed as soon as possible, if there is a retrobulbar hematoma or clear radiological evidence of compression by bone displacement.

Key World: Superior orbital fissure syndrome, trauma, surgery.

INTRODUCTION

The superior orbital fissure syndrome, (SOFS) a very rare condition that may accompany facial fractures of the Le Fort types II and III, frontobasal skull fractures, zygomatic fractures and orbital fractures. Optalmoplegia, ptosis and proptosis of the eye, reflex dilation of the pupil, and anesthesia of the upper eyelid and forehead characterize SOFS.

This syndrome may be due to craniofacial fractures as well as neoplasms of the retrobulbar space, hematomas in the orbital muscle cone and retrobulbar space, hematoma and infection of the cavernous sinus (2,3,18) and uncertain etiology (14). Two pathogenetic mechanisms can be identified; the direct, which is when the nerves traversing the fissure are interrupted or compressed by displaced bone fragments (11). The second is indirect mechanism,
in which the orbital walls behave like a non-expandable box, so every increase in internal pressure caused at the moment of the injury by posterior displacement of the eyeball or later by edema and bleeding may compress the nerves against the rims of the fissure (1,3). Incomplete forms may also be seen like proptosis, which is due to loss of the tone of the extraocular muscles that normally exert a retracting force on the globe (7,11,27). Various degrees of ocular palsy may be noted, giving rise to "partial superior orbital fissure syndrome." This shows partial or complete involvement of the 3rd and 6th cranial nerves, together with the nasociliary nerve (11). The 4th cranial nerve is rarely involved alone (1). When a process in the orbital apex also involves the optic nerve and ophthalmic artery, may lead to retrobulbar neuritis, papilledema, and optical atrophy with impaired vision. It is called orbital apex syndrome (OAS) coined by Kjoer in 1945 (10).

This case supports that decompressive surgery seems to be the best choice instead of conservative approach, if there are fragments of bones in the superior orbital fissure.

**CASE REPORT**

A 25 year-old male had had multiple head and facial injuries following an automobile accident. He had been unconscious for a few minutes just after the accident. The patient had been in neurosurgery unit of another hospital for 28 days before he was transferred to our hospital with the diagnosis of complex facial fractures and paralysis of the third, fourth and sixth cranial nerves on the right side.

On admission, he was well oriented, stable and awake. There was swelling and ecchymosis on the right eyelids and laceration of the right upper eyelid. On ophtalmological examination, he had ptosis of the right upper eyelid with a fixed and dilated pupil, but normal visual acuity. He was unable to move his right eye. His right corneal reflex was absent and the findings on fundoscopic examination were within normal limits. There was hypoesthesia over the distribution of the right supratrochlear and supraorbital nerves. The remainder of the examination was within normal limits.

CT scan revealed displacement of the right zygomatic arch and zygoma, with fractures of the lateral walls of the right orbit and maxillary sinus. Displacement of the bone fragments in the region of the superior orbital fissure. The patient was then operated on under general anesthesia. Superior orbital fissure decompression was accomplished as follows; in supine position, the head was fixed in a similar technique as in frontotemporal orbitozygomatic approach. After craniotomy, dura was elevated from the orbital roof and bone fragments were removed so as to reach the superior and lateral borders of the superior orbital fissure. The part of clinoid process extending to the medial border of the optic nerve and to the lateral border of the dural entry into the superior orbital fissure was removed. The patient did well postoperatively and improved within the first two days of postoperative period. After 3 months, he exhibited a complete recovery (Fig.1- a, b, c, d).

**DISCUSSION**

The SOFS, well known condition caused by impairment of the nerves that cross the superior orbital fissure, first described in 1858 by Hirschfeld (8).

The superior orbital fissure is the narrow cleft through which the orbit communicates with the middle cranial fossa. It is a bony opening through which the most cranial nerves pass. All of the nerves course through the cavernous sinus or its walls converge on and pass through the fissure. The fissure has neural component formed by the oculomotor, trochlear, ophthalmic and abducens nerves and branches of the carotid sympatic plexus and venous component formed by the superior and inferior ophthalmic veins. Part of the annular tendon from which the rectus muscles arise is attached along the margin of the fissure. No major arteries pass through the fissure.

CT scan, especially the three-dimensional cuts of spiral CT, is essential for diagnosis of traumatic SOFS. In the literature, 2-mm slices (24) and coronal plane cuts are recommended because of the position of the fissure in the skull (2). Magnetic resonance imaging is of no use because of an identical signal produced by the intraconical and extraconical fat tissue and recent collections of blood (5). The differential diagnosis between SOFS and OAS can be extremely difficult and necessary in an unconscious patient in whom visual acuity cannot be checked. A CT scan can also show fractures involving the optic canal although the absence of a fracture of the canal does not rule out a lesion of the optic nerve in an unconscious patient (9). That's why evoked visual potentials can also be necessary in an
unconscious patient because if there is a compression of the optic nerve, there is a maximum latency of 6-8 hours to carry out surgical decompression with or without high dose corticotherapy in an OAS (23). This case was conscious and his optic nerve was intact so evoked visual potentials were not seemed essential.

It appears that complete or partial recovery of the sensory and motor functions usually occur without surgical exploration unless there is a retrobulbar hematoma or clear radiological evidence of bone displacement (12,21) and conservative treatment is recommended in traumatic cases (1,7,8,11,12,19,20,21,22,25,26).

Tendency towards conservative approach is due to several reasons; a late case eliminates an early decompression possibility (18). The medical condition of a patient may not be suitable for a surgical intervention such as poor respiratory status (18). Technical difficulties of the operation may complicate the case with further hemorrhage or injury of the nerves during surgical exploration of the orbital apex (12,15,17,21).

Mortada (14) suggested that spontaneous hemorrhages were the etiologic factors of SOFS and there was return of ocular movements after the hematoma evacuation in 5 of his cases. Hematomas in these area resolve within 3 weeks to 4 months but they may also organize to form an orbital floor cyst with no endothelial lining (27) That’s why it seems possible nevertheless that aspiration of a hematoma or surgical decompression may be of some help to spontaneous recovery (27). In our case, the nerves might be injured or compressed by displaced bone fragments. However, accompanying edema and bleeding might be the additive factors.

Exploration of the area is definitely indicated in early stage if there are sings of neoplasm or optic atrophy (27) but the optimum timing for surgery of traumatic SOFS surgery has not been mentioned in the literature so far. Although our patient was

Fig 1: a) Preoperatively, ptosis and gazing to the left. b) Postoperatively, gazing to the left. c-d) Axial and coronal CT scan demonstrating the fracture of the orbital lateral wall.
operated on in a rather late period, immediate beginning of resolving of symptoms and full recovery obtained in 3 months may imply the importance of the early surgical approach.

The transcranial surgical approach to the orbit may be arbitrarily performed in two ways; the first involves the removal of the frontotemporal bone with preservation of the supraorbital rim, as recommended initially by Dandy in 1922 (4). The second approach involves removal of the frontotemporal bones as well as the supraorbital arch, first described by Frazier for hypophyseal surgery (6). Most neurosurgeons use the frontotemporal approach with preservation of the supraciliary arch for all orbital tumours. The best possible visualisation routes of the orbital contents with minimal brain retraction are transcranial supraorbital, lateral microsurgical and anterior medial micro-orbitotomy approaches (13). In addition there are three intraorbital routes to the optic nerve and orbital apex, which can be accessed from a fronto-orbital craniotomy; medial, central and lateral approaches (16). Murakami (15) described 4 possible different routes of surgical decompression; extranasal intraorbital, modified extranasal intraorbital, extranasal transethmoidal and transtemporal routes. We have chosen modified frontotemporal orbitozigomatic approach as we have more experience in it.

The SOFS is a particularly rare condition, especially when associated with trauma and we believe that it usually requires exploration that should be performed as soon as possible.

REFERENCES