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Case Report

Traumatic Orbital Apex Syndrome: A Rare Entity

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ABSTRACT

Background: Orbital Apex Syndrome (OAS) is an extremely rare significant ocular complication affecting the multiple cranial nerves following craniomaxillofacial trauma. The syndrome's clinical features predominantly involve a combination of ophthalmoplegia, proptosis, ptosis, hypoesthesia of the forehead, and vision loss.

Case Presentation: We present the case of a young adult male who experienced traumatic OAS with facial bone fractures following a high energy injury from a road traffic accident. The patient exhibited ptosis, complete ophthalmoplegia in the left eye and visual acuity impairment. Diagnostic imaging revealed the left supraorbital rim, which was comminuted and depressed. Additionally, there were fractures of the orbital roof, medial orbital wall, floor and also right Zygomaticomaxillary complex fracture. Traumatic orbital apex syndrome was caused by direct compression due to a displaced fracture segment from the superior orbit. The patient was treated with a combination of emergent pharmacological and early surgical intervention and close monitoring thereafter. While there was some improvement in extraocular muscle movements, the visual impairment persisted.

Conclusion: The overall recovery of vision after orbital apex syndrome is generally poor, although steroid therapy or surgical decompression did appear to improve the remaining visual parameters especially if the patient presents to the emergency immediately after the injury. Additional standardized patient data is required to clarify the effectiveness of these therapies.

Key words: Maxillofacial Trauma, Orbital apex syndrome, Optic nerve, Opthalmoplegia, Visual acuity,

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Introduction

The human orbits are symmetrically paired structures divided by the paranasal cavity and nasal cavity. Seven bones of the craniofacial regions contribute to forming the orbital cavity, namely frontal, sphenoid, zygomatic, palatine, ethmoid, and lacrimal bones. The orbital apex is a tiny cone-shaped region that lies in between the optic canal, superior orbital fissure, and posterior ethmoidal foramen and.[1] It shares a posterior border with the cavernous sinus and an anterior boundary with the superior orbital fissure.[2] This area is home to a variety of neurovascular systems, including the carotid artery, periarterial sympathetic plexus, cavernous sinus, and cranial nerves like the optic, oculomotor, abducens, and ophthalmic branch of the trigeminal nerve.[3] The conditions affecting the orbital apex are superior orbital fissure syndrome, orbital apex syndrome, and cavernous sinus syndrome. Even though the anatomical location of involvement and clinical characteristics have led to various descriptions of these disorders, the diagnosis and treatment of these conditions are essentially the same.[4]

Orbital apex syndrome (OAS), also known as Jacod syndrome, is an uncommon condition related to various etiologies. The most frequent primary cause is trauma, which can be brought on by powerful blows that inadvertently harm the optic nerve and superior orbital fissure's anatomical features.[5] Other common causes of this syndrome include tumors of the head and neck, hematologic cancers, metastatic lesions, and dermoid and epidermoid cysts. Inflammatory disorders, infections, and parasitic infections can also cause OAS. Iatrogenic causes such as surgery of the orbit and sinuses can also cause OAS. Direct injury resulting from displaced bony fragments can cause anatomic compression of nerves and vessels, leading to OAS.[6]

Although the pathophysiology of OAS is still unclear there is evidence that different nerves including optic, lacrimal, frontal, trochlear, nasociliary, abducens, and oculomotor and ophthalmic artery are the structures that may be compressed or injured when they traverse the optic canal and SOF.[7] Its symptoms include ophthalmoplegia, proptosis, ptosis due to palsy of cranial nerves (CN) III, IV, and VI, hypoesthesia of the ipsilateral forehead, upper eyelid, and cornea due to the involvement of the trigeminal nerve's ophthalmic (V1) division, and finally, visual impairment due to optic neuropathy.[8]

As such, diagnosing orbital apex syndrome can be difficult, but it's important to catch it early to prevent irreversible harm to sensitive neurovascular structures. It is typically necessary to use imaging techniques like magnetic resonance imaging (MRI), and contrast-enhanced computed tomography (CT scan). A multidisciplinary strategy involving maxillofacial surgeons, neurosurgeons, and ophthalmologists in treatment and emphasis on addressing the underlying pathophysiology.[9]

Case Report

A 48-year-old male came to the emergency room (ER) more than 24 hours after suffering a blunt injury to his craniofacial region in a high-speed car accident in a far-fetched area. He was conscious and oriented with a GCS of 15/15. He complained of severe pain, and blurred and reduced vision in his left eye. He also reported numbness over his left upper face and forehead area immediately after the trauma.

On physical examination, the left pupil was mid-dilated, the iris was blood-stained, the direct light reflex was diminished, and the consensual light reflex was absent. As in figure 1 which is uploaded after consent from patient shows huge, irregular laceration measuring 8 cm covered the left upper eyebrow, lateral canthus area, upper and lower eyelid area, circumorbital ecchymosis, and subconjunctival bleeding, among other soft tissue injuries. On Ocular examination ophthalmoplegia with restricted left orbital ocular movement was observed, along with a left relative afferent pupillary defect that was particularly noticeable on left lateral gazing, indicating possible impingement of the optic nerve. The visual acuity of the patient was 6/18 and intraocular pressure of 25 mm Hg was measured in the affected eye. Regarding the left eye, there was also restricted superior, lateral, and medial vision, as well as limited extraocular movement. Additionally, the patient experienced hypoesthesia in the distribution of the ophthalmic and maxillary branches of the trigeminal nerve along the ipsilateral face.

A computed tomography scan in Figure 2 revealed that the patient had a fracture of the left supraorbital rim, which was comminuted and depressed. Additionally, there were fractures of the orbital roof, medial orbital wall, and floor. Fragments of the orbital roof bone bordering the optic nerve at the orbital apex were found to have been intra-orbitally displaced. A B-scan was also performed by the ophthalmology department. He also sustained a fracture of the right ZMC.

After being admitted to the surgical intensive care unit, a two-stage surgery was planned. First, the ophthalmology department did an emergency bedside washout and debridement of his wounds in addition to decompressing his lateral and posterior orbit. Soft tissue Lacerations sutured. The rebuilding of the facial frame by open reduction and internal fixation was carried out the next day following the patient's stabilization and proper clearance for maxillofacial surgery.

The patient received high-dose steroids in the form of 1 g of IV methylprednisolone daily for three days as an adjuvant treatment for their traumatic optic neuropathy. At 1 month follow-up, in figure 3 shows the patient still had decreased visual acuity in the left eye although it was improving according to the patient's own accord.

Figure 1. Pre op picture: Sutured laceration on Eyebrow, upper and lower eyelid, lateral canthus of eye

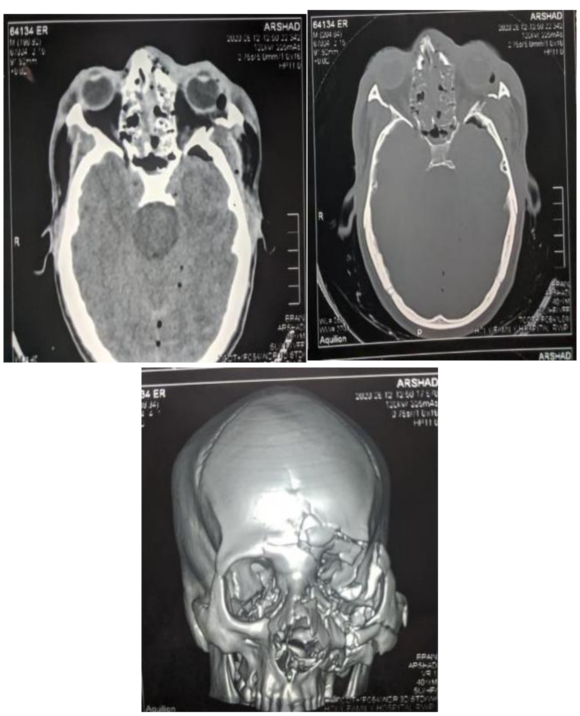

Figure 2. Investigations: CT scan showing Orbit zygomatic Complex Fracture

Figure 3. Post Op Pictures: Healed Laceration, Eye movement improve

Discussion

An intricate region where the vascular, bone, and neural systems converge is the orbital apex. The oculomotor, abducens, and Nasociliary nerves reach the orbit through the superior orbital fissure, whereas the optic nerve and the ophthalmic artery enter through the optic canal. Notably, meningeal coverings surround the optic nerve and are firmly attached to the periosteum. Understanding the relationships between these elements is essential for the diagnosis and treatment of a range of orbital and neuro-ophthalmological conditions.[10]

Visual impairment, ophthalmoplegia, ptosis, proptosis, dilated pupils, aberrant light reflex, and forehead hypoesthesia are the hallmarks of OAS. The ophthalmoplegia is a result of dysfunction in either CN III, CN IV, or CN VI ptosis is caused by weakness in the levator palpebral muscle (CN III) and the superior tarsal muscle (nasociliary nerve). Reduced tension in the extraocular muscles may be the cause of exophthalmos.[11] The anesthesia in the frontal and eyelid regions is caused by injury to CN V1. Furthermore, it is frequently discovered that the malfunctioning of the parasympathetic fibers in CN III is linked to the dilated pupil. The cause of the vision disturbance is identified as CN II optic neuropathy. The foregoing symptoms are together referred to as OAS.[12]

It is important to distinguish between the symptoms of the cavernous sinus, orbital apex, and superior orbital fissure nerve compression. Following a high-speed car collision, our patient was diagnosed with traumatic OAS. This condition resulted in visual loss, a fixed, dilated pupil, proptosis, ptosis, and anesthesia of the foreheads.[13] Inflammatory, viral, neoplastic, iatrogenic, traumatic, or vascular events can all lead to OAS. One common cause of OAS is trauma. Craniofacial fractures or hematomas in the orbital muscle cone and retrobulbar region could be the cause.[14] Traumatic nerve injury develops through both direct and indirect mechanisms. of them, the indirect influence mechanism is thought to be more prevalent. It is suggested that foreign substances or misplaced bone fragments cause direct nerve injury. According to the indirect effect mechanism, the nerve may be injured by impact shearing force that travels through the bones, by the globe's motion in relation to the optic nerve or its blood vessels, or by the compression force of hematoma or soft tissue edema.[15] The two most important ophthalmologic functions to be examined immediately if OAS is suspected in a patient with acute orbital trauma-related OAS are visual acuity and extraocular muscle motility, as vision loss, ophthalmoplegia are usually early symptoms of OAS.[16] For Diagnosis CT scan is the best method for examining bone involvement, particularly in the event of trauma, even though MRI is useful for diagnosing soft tissue lesions surrounding the OA region.[17] Possible findings on the CT scan include extraocular muscle impingement by bone fragment, optic nerve impingement, intrabulbar emphysema, ruptured globe, and optic nerve thickening due to edema. Furthermore, it has been established that 3D reconstructions work better for localizing complex fractures that involve several planes. When combined with the clinical results we diagnose OAS more accurately.[18]

Treatment option is chosen on the base of individual etiology and the health status of the patients. Prompt diagnosis and early management are paramount to lessen morbidity. Treatments for infectious causes include surgical debridement, antibiotics, for traumatic causes decompression surgery along with systemic corticosteroids. These corticosteroids are administered intravenously for three days at a dose of one gram of methylprednisolone and then slowly taper oral administration of one milligram per kilogram of body weight of prednisolone.[19] The patient in this case report presented late (more than 24 hours after the RTA) which could explain why the aggressive and early pharmacologic and surgical intervention still couldn’t lead to a significant improvement of the traumatic optic neuropathy.

Conclusion

Early presentation and accurate diagnosis, individualized treatment plans, and a multidisciplinary approach are essential for improving patient prognosis. Further research is necessary to establish a unified treatment protocol for managing this rare condition. In the present study, a high percentage of infectious and sharp waste was improperly disposed of in participating dental hospitals and clinics. However, the vast majority of them disposed of their solid waste in conventional ways.

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