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Post Traumatic Intra Orbital Encephalocele, a Rare Cause of Pulsating Orbit: Case Report

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Abstract **Case Report**

Orbital trauma is a prevalent condition in clinical practice, particularly in situations of head trauma. Orbital roof fractures following a blunt trauma are an uncommon consequence of trauma. Traumatic encephaloceles in the orbital cavity are much more uncommon, with only a few examples reported to date. Early detection and therapy of raised intraorbital pressure can avoid irreparable optic nerve injury. In trauma patients with concomitant orbital injuries, orbital computed tomography (CT) with thin axial and coronal slices is beneficial. Decompression of the orbital roof is the most important stage in surgical therapy and should be done in all cases. We report the case of an adult with a post-traumatic orbital encephalocele. A few hours after a road traffic accident with head injury, the patient developed progressive pulsatile enophtalmosis. Computed tomography revealed multiple fractures of the orbital rim with herniation of the left frontal lobe into the orbit.

Keywords: Head injury, post-traumatic orbital encephalocele, proptosis.

BACKGROUND

Orbital roof fractures caused by physical trauma are uncommon, however the exact incidence of orbital trauma is unclear. The majority of the cases are related with concurrent head trauma. Even infrequent are traumatic encephaloceles in the orbital cavity [1].

The most common cause is a frontal or lateral blow to the orbital rim. It often appears as pulsing proptosis of the orbit. The two most prevalent causes for a delayed identification of orbital injuries are that ecchymosis and puffy eyelids are typical after head trauma, and the patient is generally obtunded after head trauma. All patients with ecchymosis and proptosis must have a full neuro-ophthalmological examination as well as a thorough palpation of the orbital rims.

Computed tomography (CT) scans of the skull, orbits, and optic foramina in both coronal and axial sections, provide enough information about the trauma and its complication and aid in the selection of an appropriate surgical method.

CASE REPORT

A 25-year-old man was admitted following a motor vehicle accident. Neurological examination revealed that he was unconscious with a glasgow coma scale (GCS) score of 7. He had bilateral periorbital hematoma more on the right side. Visual acuity and extraocular muscle motility of both eyes could not be evaluated completely due to periorbital hematoma, soft tissue swelling, and reduced level of consciousness of the patient. His pupils were bilaterally equal and reactive. He also had a pulsating enophtalmos on the left side.

Cranial CT axial sections of the head revealed bilateral hematoma in the frontal lobes and a left 6mm epidural hematoma causing a midline shift.

He had fracture all the walls of the left orbit, with entrapment of the medial and inferior rectus muscles and the extraconical fat, causing the eyeball to recede backwards. The CT scan also revealed the herniation of the brain parenchyma intra-orbitally through the roof fracture and coming into contact with the superior rectus muscle and displacing the left globe inferiorly.

The contralateral orbital rim suffered multiple fractures as well with detachment of bone fragments in the orbit

The patient suffered a cardiac arrest in the following hours and was declared deceased before undergoing surgery.

Figure 1: Head CT

Bilateral frontal, capsulo-lenticular and left thalamic oedemato-hemorrhagic contusions. Left fronto-temporal epidural hematoma 6mm of maximal thickness.

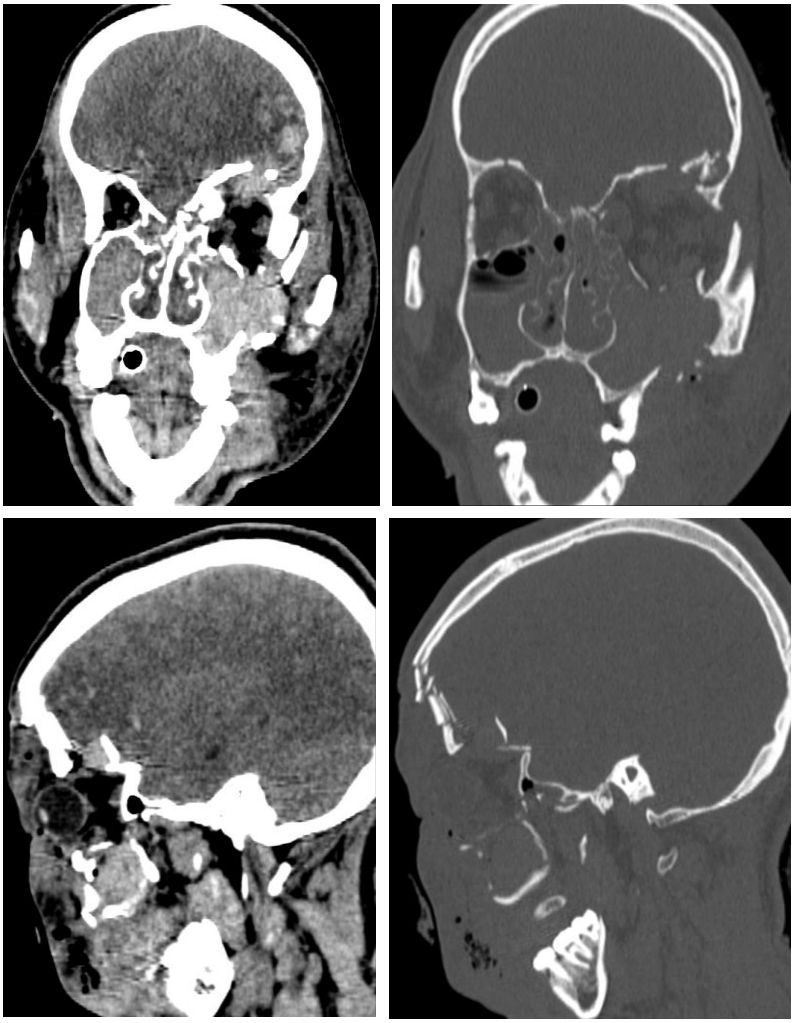

Figure 2: Head CT coronal and sagittal planes, on parenchymal et bone window

Fracture all the walls of the left orbit, with entrapment of the medial and inferior rectus muscles. Herniation of the brain parenchyma intra-orbitally through the roof fracture and coming into contact with the superior rectus muscle and displacing the left globe inferiorly.

Figure 3: Head CT 3D reformatting

Multiple and bilateral facial fracture

DISCUSSION

The classification of orbital roof fractures by Messinger [2] is based on the extent of displacement of bone fragments. The force applied to the orbital rim is transmitted to the adjacent thin bony roof structures, leading to their fracture [3].

Another mechanism involves an explosive rise in intraorbital pressure after injury, causing thin bone fragments to be blown out or in, into the brain or sinuses, respectively. The incidence of orbital roof fractures is higher in children due to the inability of the frontal sinus to dissipate the impact to the superior aspect of the orbital rim before age 7 [4]. Motor vehicle and motorcycle accidents are the leading causes of trauma, with direct assaults being rare [5].

Ocular symptoms of orbital roof fractures include periorbital edema and ecchymosis, restricted extraocular movements, pupillary defects, proptosis and ptosis, diplopia, and rarely ruptured globes [6]. Cranio-orbital fistulas with cerebrospinal fluid (CSF) leakage trapped within the orbit causing a fluid leak from the eye are exceedingly rare. Associated cranial lesions in supraorbital roof fractures include frontal contusions (43%), epidural hematomas (17%), traumatic subarachnoid hemorrhage (14%), and encephalocele (9%) [5].

The preoperative diagnosis of traumatic encephalocele into the orbital cavity was difficult before the introduction of CT scan. Direct skull x-ray can only identify large depressed fractures usually involving the frontal region. With the introduction of CT studies in coronal acquisition, an encephalocele could be suspected as a possible complication of orbital roof fractures when significant bone fragments were displaced upward or downwards [6].

Orbital roof fractures may enlarge over time due to gradual herniation of the arachnoid into the fracture, physiologic growth of the cranium and the brain (in children), continuous pulsation of the CSF, and absence of bony countercompression [7]. In most cases, the defect allows only the formation of intraorbital leptomeningeal cysts with rare subsequent oculorrhea. The mechanism of herniation of brain tissue into the orbit is an acute injury with the formation of a gradient between the pressure in the subarachnoid space (post-traumatic increase of intracranial pressure) and in the intraorbital compartment.

Whenever a diagnosis of orbital encephalocele is made, surgical intervention is necessary since the herniation enlarges and can cause progressive pulsatile exophthalmos with the risk of visual loss [1]. The herniated brain tissue is usually nonfunctioning and contused; therefore, there is no risk of functional damage associated with its evacuation. However, there is a risk of orbital infections and cellulitis [11].

Our patient was the victim of an acute traumatic encephalocele related to orbital roof fracture, which is unusual and rarely reported. For such cases, early diagnosis and treatment are vital as the raised intraorbital pressure may damage the optic nerve irreversibly.

CONCLUSION

In conclusion, in patients with acute brain injury and orbital roof fractures accompanied by frontal contusions, the possibility of orbital encephalocele should be considered. Our recommendation is that a CT scan of the orbit be used as the primary diagnostic tool in such cases. Upon confirmation of the encephalocele, surgical intervention via the subfrontal approach is necessary to remove the herniated contused brain tissue, close the dura, and reconstruct the orbital roof.

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