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INTRODUCTION

A comprehensive understanding of orbital fractures is necessary for the treating physician due to the functional and aesthetic deformities that often result. Studies have estimated that orbital fractures account for roughly 10 to 25% of all cases of facial fractures, and similar to all facial traumas, they are most commonly seen in conjunction with assaults and motor vehicle accidents [1]. Motor vehicle accidents related orbital fractures, in particular, tend to be more destructive and are associated with more concomitant organ injuries, zygoma fractures and multiple orbital wall fractures than the other common causes of assault, sports-related and falls etc [1]. Additional damage to the globe, optic nerve, extra ocular muscles is almost always an accompaniment to these fractures. About 11 to 15% of orbital fractures are associated with ophthalmological emergencies [2]. A retrospective chart review undertaken by Ansari in 2005 revealed 30 cases of blindness following facial trauma and the other subsequent facial fractures required operative intervention in 2503 patients. [3] Their analysis revealed the vast majority of cases were secondary to zygoma or zygomaxillary complex fractures with retrobulbar hemorrhage or severe damage to the eye. Therefore, in this case, an early evacuation of an infected subperiosteal and intra-orbital hematoma had prevented blindness and also death due to intra cranial infection.

REPORT

A 29 year old male had met with a road traffic accident while riding in a two wheeler in an inebriated condition. He had sustained head injuries and right facial injuries. He had a history of loss of consciousness for about twenty minutes and bleeding through nose. There was no history of seizures or vomiting. On receiving in the casualty, the patient was found to be drowsy and disoriented. However he obeyed to oral commands and moved
all four limbs to painful stimuli. His Glasgow coma scale score was 12/15, E4V4M4. His pulse rate was 98/minute, Respiratory rate was 20/minute, and Blood pressure was 140/80mmhg. He was found to have partial ptosis, severe proptosis of right eye with sub-conjunctival hemorrhage and restricted ocular movements. Right eye bed side visual acuity was 6/12 and left eye was 6/6. Right eye pupil was 4mm in size sluggishly reacting to direct light reflex. Left eye pupillary size and reflex was normal.

CT scan of the brain showed multiple frontal and temporal lobe 0.5 to 1 mm sized contusions. There was linear fracture of basifrontal, temporal, zygomatic and all the right orbital bones. CT orbits also revealed right haemomaxillary sinus and right orbital emphysema.

Patient was admitted in the emergency ward with room air ventilation. He was started on anti-cerebral edema measures, antibiotics, anti-convulsants, IV fluids and general supportive measures. Patient was advised topical lubricating eye drops and topical non-steroidal eye drops for the right eye. Patient regained consciousness and became oriented from the day of admission. His Glasgow coma scale score improved to 15/15 E4V5M6. Patient developed dryness of right eye cornea four days following the head injury and was advised tapping of lids along with lubricating eye ointments. Patient had developed severe exposure keratopathy and his vision dropped to 6/36 in right eye with corneal opacification in the inferior interpalpalbral part of cornea. Patient was also complaining of restricted ocular movements and double vision. On diplopia charting, it was found to be a crossed diplopia with right hypotropia. Colour vision was defective in right eye and normal in left eye indicating right optic neuropathy.

Surgical evacuation of the retro-bulbar and sub-periosteal haematoma was decided as the right eye proptosis was not resolving with medical management even after 10 days. With a curved right sub brow incision, periosteum of the superior orbital wall was incised and about 20 ml of altered blood and pus was aspirated from the retrobulbar and superior sub-periosteal space.
Proptosis was resolved from the first post operative period. Patient was able to close his eyes and the exposure keratopathy gradually improved. Post operative CT scan of orbit showed resolved intra orbital and sub periosteal haematoma.

**DISCUSSION**

Orbital hematomas can be caused by trauma and can be classified as intraorbital or subperiosteal by Landa[4]. Intraorbital hematomas are more common and show findings of subconjunctival hemorrhage, lid edema and bruising and diminished ocular movement. Subperiosteal hematomas, which occur secondary to rupture of subperiosteal blood vessels, are not as common and will present with proptosis, lid ecchymosis, exposure keratopathy, defective pupillary reflex and impairment of eye movement. The management of the hematoma depends on how impaired the vision is. Without visual disturbance, the hemorrhage can be observed without specific treatment. When vision is affected, the hematoma should be evacuated. Small retrobulbar hematomas generally get resorbed spontaneously within 3 weeks to 4 months. [5] However, hematoma may also organize to form an orbital blood cyst with no endothelial lining. Mortada [6] recommended an exploration of the orbital apex that is indicated through a lateral transconjunctival orbitotomy to evacuate the blood if medical treatment is unsuccessful in approximately 4 months. However, in this case, not only did the hematoma did not resolve with antibiotics and anti-inflammatories for 10 days, but also the collection behind the eye have turned to be purulent causing proptosis and severe exposure keratopathy compromising the vision. Therefore early evacuation of the subperiosteal haematoma is imperative in this case for prevention of the vision loss from exposure keratopathy and secondary corneal infection. Also the spread of infection to the brain causing meningitis and encephalitis had been prevented. In this case the pupillary reflex had been sluggish from the time of injury indicating optic neuropathy due to nerve compression from extrinsic.
(hematoma) or intrinsic (haematomata or edema) sources. Traumatic optic neuropathy is seen in about 0.5-5% of patients with orbital fractures [7]. This is confirmed by the fact that the patient had defective colour vision both during the pre operative and post operative period even though the vision improved to 6/9. There is a high association of orbital roof fractures with neurologic injury (57 to 90%), [8] ocular damage (14 to 38%), and additional orbital (76%) and facial fractures (33%). Certain orbital injuries require emergent surgical intervention [9]. The timing of surgery for non emergent injuries is often in a semi-delayed fashion around 7 to 14 days, before soft tissue scarring develops, but after resolution of posttraumatic edema. After 2 weeks, soft tissue fibrosis can become significant, especially in young males, and require sharp dissection for reduction of orbital tissues [10]. In this case, patient had been operated 10 days following injury, thus avoiding the complications of delayed intervention.

CONCLUSION

- Early surgical evacuation of a subperiosteal and an intra orbital haematoma reduced the proptosis favouring the patient to close the eyes completely resolving the exposure keropathy and the consequent vision threatening infectious keratitis
- Subperiosteal hematoma had turned purulent and if left undrained, would have spread to the brain through the roof fracture
- Antibiotics do not penetrate through pus, therefore conservative management with antibiotics would have proved ineffective
- A team approach to surgical management of craniofacial and zygomatico orbital fracture along with orbital, faciomaxillary and neurosurgeons can save the life of an individual apart from achieving good functional and cosmetic effects.

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