ORIGINAL RESEARCH PAPER

Ophthalmology

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STRABISMUS MANAGEMENT AFTER ORBITAL BLOWOUT FRACTURE

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ABSTRACT

Orbital floor fractures (OBF) account for 40% of mid-facial injuries and are therefore the most common of all trauma injuries in this region. The post-treatment complication that often follows orbital floor repair is residual diplopia or persistent diplopia and is seen in 86% of the OBF cases.[1]

The causes for persistent diplopia can be varied and is often related to the degree of inflammation, trauma to musculature, fat or nerves and surgical timing.[2] Some of the common causes of the same are - malpositioning of the globe, fibrosis of the inferior fibro fatty muscular complex following trauma, direct damage to an extraocular muscle (commonly inferior rectus muscle), local injury to a motor nerve, ischemia (or compartment syndrome), iatrogenic damage during reconstructive surgery or entrapment under improperly placed alloplastic material.

Our case report mentions a rare case of persistent vertical diplopia even after successful repair of orbital blowout fracture. A 15-year-old male patient following a road traffic accident presented with persistent headache and vertical diplopia. The patient was evaluated by a oral maxillofacial surgeon and a presumptive diagnosis of a case of large orbital floor fracture with entrapment of inferior rectus muscle was made which was confirmed on CT Scan. He was managed surgically by reduction of the fracture and fixation with a titanium mesh. 2 weeks post-surgery he reported to the squint clinic with complaints of persistent double vision. On comprehensive ocular examination, it was found that patient had vertical diplopia with limitation of infraduction in the left eye with negative FDT, on re-evaluation of MRI scans with 1 mm cuts, a partial left inferior rectus tear was seen and documented as the cause of persistent diplopia. Patient was treated conservatively by prescribing prismatic glasses with fusional exercises. After 6 months of follow up, the patient was relieved of diplopia in primary position but there was a residual hypotropia in downgaze for which he was prescribed prisms only for downgaze.

INTRODUCTION

Orbital fractures first described by Mackenzie (1884) account for 36.3% of all facial fracture presentations[3] and the prevalence of the same in children ranges from 3-45%.[4] The prevalence is higher in children above the age of 7 years because in children under this age group, the sinus walls are thicker, there is presence of more cheek fat pad, mid face is relatively smaller and there is greater bone elasticity all of which are thought to be protective of the orbital floor.[5]

The orbital fractures are classified into three main categories namely- orbital rim fractures, comminuted orbital wall fractures and trapdoor orbital wall fractures[6] of which the most common in children are orbital trapdoor fractures.

The orbital trapdoor fracture is anatomic subtype of orbital floor fractures and occurs as a result of transient increase in orbital pressure. As a result of this a linear orbital wall fracture is created which leads to outward displacement of flap of bone which then immediately results to its original position. The ample bone elasticity of children's bones owing to the high proportion of osteocytes and smaller amount of calcified osseous tissues is responsible for this mechanism.

These types of fractures may or may not lead to entrapment of soft tissues. Reduced duction in the direction of fracture occurs as a consequence of entrapment of an extraocular muscle or associated connective tissue in most of the cases. Other commonly reported signs and symptoms that occur as a result of entrapment of soft tissue and muscle within the defect includes- severe motility restriction, pain on extra-ocular movement, early tissue necrosis and ischemia. Oculo-cardiac reflex (nausea, vomiting and/or bradycardia) is induced due to eye movement and is often a common finding in trapdoor fractures.[7]

The clinical presentation of orbital floor fractures in children is different from that in adults and it commonly occurs as a result of low velocity but high force crush injuries.[8] Some of the common etiological factors include- sports, accidents during play, assault and motor vehicle accidents.[9]

In accordance to various studies, it has been reported that the most commonly reported consequence that follows orbital floor fractures in children are diplopia and extra-ocular motility limitations.[10,11] Diplopia occurs as a result of entrapment of extraocular muscles, swelling/hemorrhage, direct damage and injury to the nerve or damage to the muscles during initial injury.

In children, persistent diplopia is a more commonly reported complication because of the unique pediatric trapdoor fracture pattern that often leads to direct damage to extra ocular muscles and nerves.[13] It is also observed post-operatively following surgical repair of orbital floor fracture in children.[14] It has been reported that delayed repair can be responsible for high rates of extra-ocular motility and persistent diplopia in children. The causes of delayed repair in children are – tissue ischemia and subsequent inflammatory response that can lead to fibrosis and long term decreased motility[15]. Also, owing to rapid bone generation, callus formation takes place around the fracture within 1 week thus impeding surgical approximation of fractured edges and delaying repair.

Therefore, in order to prevent the occurrence of aforementioned serious complications, it is of utmost importance to diagnose and systematically plan the treatment of orbital floor fractures followed by persistent diplopia in children.

CASE STUDY

A 15-year-old healthy male patient met with a road traffic accident wherein he suffered blunt force injury to his left temple region following which he lost consciousness and suffered from nasal and oral bleeds. On regaining consciousness, he experienced constant headache and diplopia with nausea and dizziness. There was pain with eye movement and marked tenderness to palpation over inferior aspect of orbital rim. Infraorbital paresthesia was documented. The patient was a child and symptoms were suggestive of orbital blowout fracture with entrapment of muscle and was taken as an emergency and was evaluated by an oral maxillofacial surgeon and was shifted for further imaging. A CT scan with coronal and axial cuts was performed to evaluate for orbital floor fracture and showed an orbital floor fracture of left side with caudal dislocation of the bone fragment and herniation of the Inferior rectus muscle and orbital fat into the maxillary sinus. No ophthalmology opinion was taken at the time. The patient was prescribed a 10-day course of oral amoxicillin/ clavulanate and oral steroids 1mg/kg bodyweight and was advised not blow his nose and use nasal decongestants twice a day. A planned surgical repair of the orbital fracture using a titanium mesh was done under general anesthesia.

He was referred to our pediatric ophthalmology clinic 2 weeks later with complaints of persistent double vision and left eye swelling even after successful repair. On general examination, patient was conscious, cooperative and oriented to time place and person. His vitals were within normal limits.

On local examination, there was significant left periorbital swelling with associated ecchymosis. A sutured wound over left upper and lower lid. No evidence of hyphaema in the left eye was present. Visual acuity of 6/6 was recorded in right eye and 6/9 in left eye. Both pupils were equal in size and reactive to light. Mild enophthalmos (2mm) was present. On ocular motility examination, -2.5 limitation in depression and a limitation of infraduction in left eye. There was hypertropia in primary position and increasing in downgaze. Diplopia charting and hess charting showed underaction of inferior rectus muscle in left eye. Forced duction test was negative and a weak force generation test along with downward saccades. This detailed Ocular motility evaluation narrowed down the diagnosis to partial tear of inferior rectus, i.e. paresis of the inferior rectus muscle.

INVESTIGATIONS

Post-operative MRI revealed a partial tear of the inferior rectus muscle ruling out any need for ophthalmological surgical intervention. MRI also showed a well-positioned titanium mesh as well as repositioned herniated tissues.

DIFFERENTIAL DIAGNOSIS:

Many factors have been reported as possible causes of persistent post-operative diplopia. These include delayed repair, fracture volume, radiological evidence of extra ocular muscle swelling, patient age and fracture site. Other commonly reported reasons for development of diplopia post orbital floor repair are:[16]

Intraorbital Edema –

This is a common cause of diplopia following orbital blowout fractures and also after repair of the same. It exhibits mild non-specific limitation of motility along with mild FDT restriction. There is usually no deviation and normal saccades.

Entrapment Of Inferior Rectus Muscle-

A restrictive strabismus could result from entrapment of inferior rectus muscle either due to the trap door orbital floor fracture or following repair using an implant. This shows limitation in supraduction with a positive FDT and a normal FGT with hypotropia.

Orbital Adherence Syndrome

Fat Adherence Syndrome (FAS) –

A restrictive strabismus that might occur following trauma or corrective surgery. Corroboratory clinical evidence to confirm occurrence of permanent restriction of ocular motility is available.[17] Although the exact pathophysiology is not well understood but violation of tenons capsule and exposure of fat to EOM is said to play a significant role. Traumatized fat can develop into fibrous scar and could lead to tethering of the globe or muscle sleeve or sclera to the periosteum resulting in restrictive strabismus and persistent diplopia. The results of surgical of FAS are poor and usually don't alleviate the diplopia.[18]

Rupture Of The Inferior Rectus Muscle-

In cases of orbital blow out fracture, there is a sudden and enormous rise in the intra orbital pressure which pushes down the inferior rectus muscle which could lead to a tear in the inferior rectus muscle. It presents as a complete palsy of the inferior rectus muscle with no movement in downgaze beyond midline and a negative FDT and absent FGT along with a large angle hypertropia.

Inferior Oblique Muscle Adherence Syndrome (IOMAS) -

It is a complication of ocular surgery wherein the inferior oblique muscle gets scarred into the temporal corner of inferior rectus attachment. Its clinical presentation is similar to FAS, but it responds well to surgical correction.[19]

Extra Ocular Muscle Restriction And Resultant Diplopia Following Orbital Floor Fracture Repair Using Titanium Implants Is Also Reported.[20]**-**

This can be due to the fibrotic adherence between peri-orbital tissue and/or the extra ocular muscles and or the pores or gaps in the implants. The above fact is corroborated by the evidence of alleviation of the diplopia by removal of titanium graft and placement of allogenic or autogenous membrane graft.[21]

Despite proper anatomic reduction of orbital fractures, diplopia can still persist, and orbital surgery in itself poses a risk of injury to ocular motor nerves.[22] Also limited literature is available that focuses on neurogenic contribution of diplopia that often occurs as a result of orbital trauma.

Flap Tear Of Inferior Rectus Muscle-

Flap tear of rectus muscle is a common cause of persistent diplopia and is produced by sudden downward force exerted by orbital tissue displaced to the fractured site. This traction of connective tissue produces tearing of outer orbital layer away from inner global layer of the muscle. In our case report, patient reported with limited ocular motility (limited depression), a negative forced duction test (FDT), weak forced generation test (FGT) and presence of hypertropia. All of these pointed to the classical signs and symptoms observed in case of partial tear of IR muscle and hence ruling out the above stated differential diagnoses.

TREATMENT:

Two weeks post operatively upon referral to pediatric ophthalmology clinic, a detailed ocular examination and MRI Orbit was performed which helped us decide to manage the patient conservatively with a combination of orthoptics and prismatic glasses. The option of resurgery was also explored but the patient was reluctant. The patient was able to fuse with 12 Prism diopter in primary position. So, he was prescribed with 6PD base up in right eye and 6PD base down in left eye in spectacles and a separate pair for near vision.

OUTCOME AND FOLLOW UP

- Satisfactory functional recovery was achieved after 3 months without the need for any ophthalmological

surgical intervention.

- After 3 months, there was improvement in enophthalmos, hypertropia present only in downgaze and -1 limitation in infraduction.
- Complete functional recovery took around 6 months wherein there was no enophthahlmos. He was ortho with no diplopia in primary gaze, small residual <10PD L/R for downgaze and in laevoversion for which he was advised surgery.

DISCUSSION:

Persistent diplopia occurs as a common complication of post-surgical repair of orbital floor fractures (OBF), causes of which can be many, varying from missed diagnosis or incorrect reconstruction to muscle fibrosis, muscle edema, hemorrhage and nerve palsies.[22]It has been reported that resultant diplopia and ocular motor disbalance persists in about 10-30% of cases with blowout fractures post repair despite proper surgical management.[23] The presenting case report is thus a peculiar case of persistent diplopia following repair of orbital floor fracture.

Presence of diplopia is one of major indications that warrant surgical intervention in a case of blow out orbital fracture.[24]The timing of surgery plays an important role in determining the development of residual diplopia. Deferring the surgery until the periorbital edema subsides, increases the risk of the impinged orbital tissues to develop fibrosis. This can further lead to abnormal scar formation which can alter the postoperative alignment and limit ocular rotation.[25]

Proper management of blow-out fractures with ocular motility imbalance and residual diplopia necessitates a proper complete neurologic and ocular examination and also helps to rule out any other serious injuries. Compared to adults, children with orbital floor fractures are prone to entrapment of muscles. The most common presenting signs and symptoms can be- persistent nausea, vomiting, eye pain, diplopia or limited upward gaze, and is considered a surgical emergency due to potential ischemia of the muscle involved.

There is a well-documented oculovagal reflex associated with orbital injuries which can explain bradycardia and nausea or vomiting following a fracture. CT is the imaging modality of choice with thin cuts through the orbits, as plain radiographs have only a 50% sensitivity in detecting orbital fractures. Typically, non-operative management is recommended in the absence of entrapment, enophthalmos and vertical ocular dystopia.

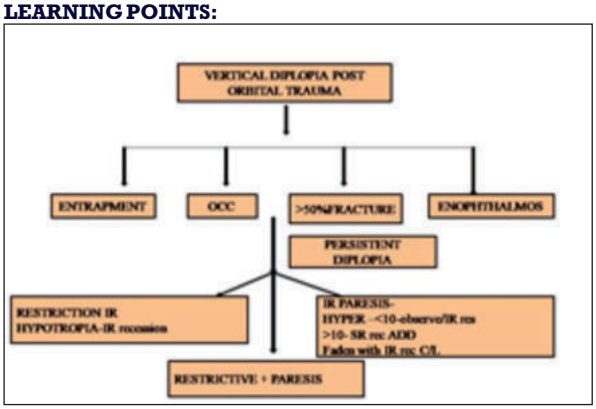
Rupture of the IR muscle is also a possible cause of persistent vertical diplopia at the time of presentation and can lead to persistent infraduction deficit after blow out fracture. In the presenting case report, patient reported with flap tear/partial tear of inferior rectus (IR) muscle which lead to persistent diplopia.

In the presenting case, concerning the management of orbital floor fracture followed by development of resultant residual diplopia, the patient was offered surgical correction for symptomatic relief for diplopia.

However, the patient denied surgical correction and wanted to continue with conservative management and thus was managed so with a combination of orthoptics and prismatic glasses.

From the presenting case report, the common concluding points of significance are that it can be proved that persistent diplopia after successful repair is a frequent complication following orbital floor fracture as observed and that it can occur as a result of damage to the muscles and its consequent fibrosis. Identification of the cause early and prompt treatment can significantly reduce the incidence of persistent diplopia.

LEARNING POINTS:



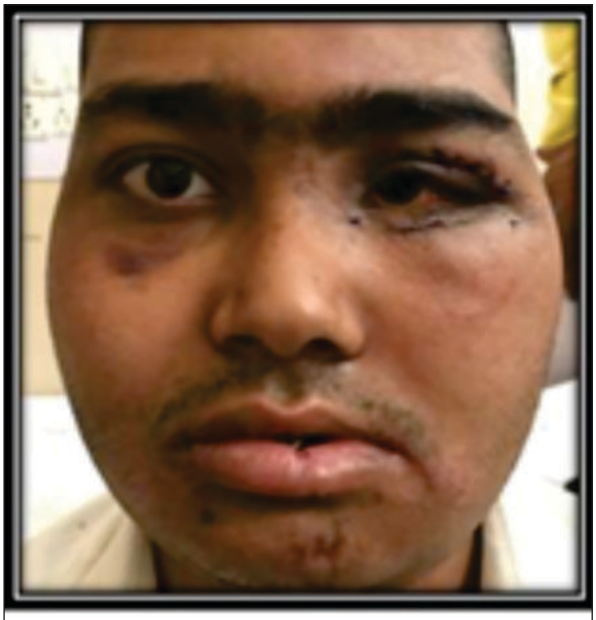
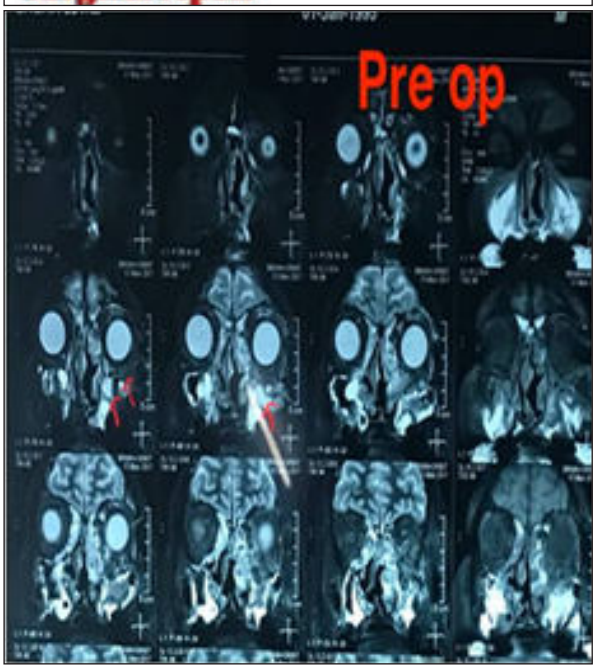


Fig 1-showing enophthalmos and sutured wounds 10 days post surgical repair



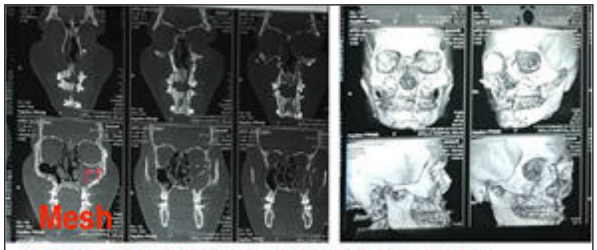


Fig-3 CT Scan 3D Face shows comminuted fracture of walls of maxillary sinuses.Metallic mesh in floor of left orbit. Multiple metallic fixator seen over maxillary sinus wall

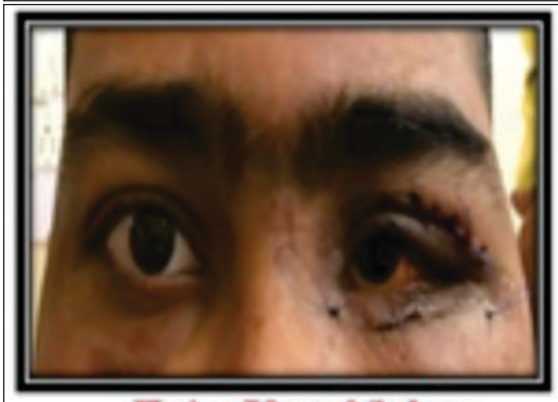


Fig 4- mild exophthalmos

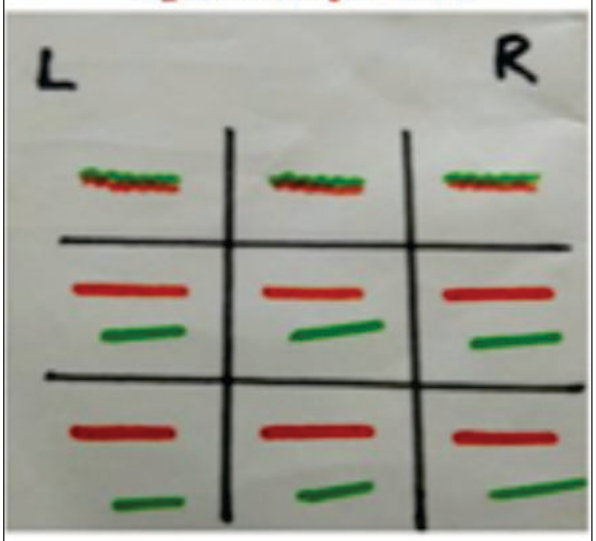
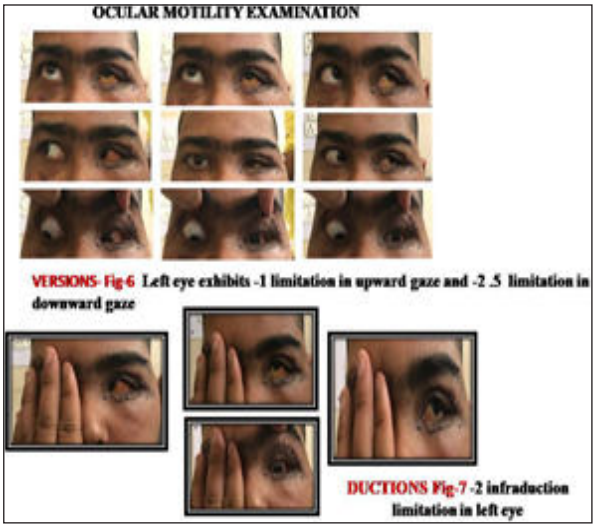


Fig-5 Diplopia charting at 1 m



VERSIONS- Fig 6 Left eye exhibits -1 limitation in upward gaze and -2 .5 limitation in downward gaze

DUCTIONS Fig-7 -2 infraduction limitation in left eye

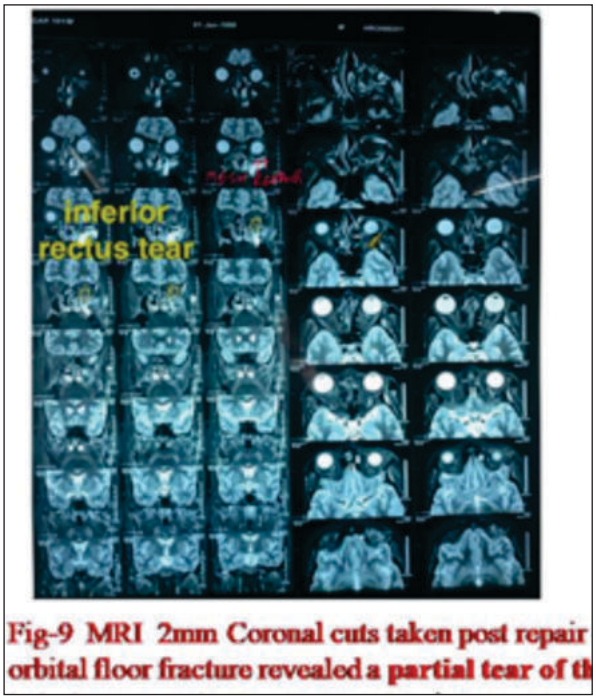
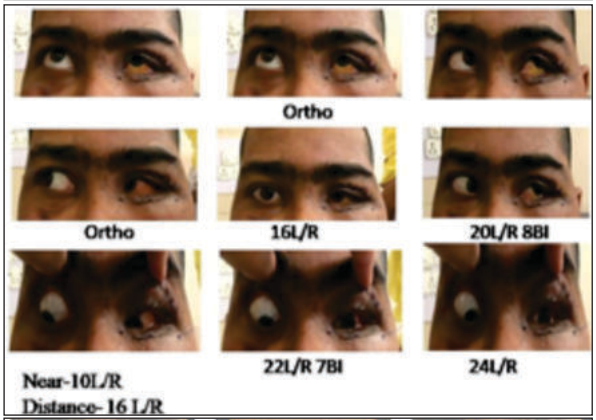


Fig-9 MRI 2mm Coronal cuts taken post repair orbital floor fracture revealed a partial tear of th



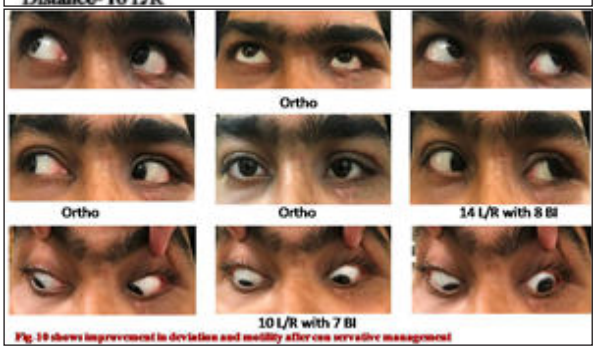


Fig. 10 shows improvement in deviation and motility after con servative management

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