SHORT COMMUNICATION

Medial rectus muscle incarceration in pediatric medial orbital wall trapdoor fractures

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PURPOSE. We describe two cases of orbital trapdoor fractures with medial rectus muscle incarceration.

METHODS. Small interventional case series.

RESULTS. This is a retrospective university based report of two healthy males (11 and 14 years old) who developed diplopia following blunt orbital trauma. Both patients had decreased horizontal ocular motility of the involved eye with minimal additional evidence of trauma. Computed tomography (CT) demonstrated no significant bony displacement; however, the left medial rectus muscle was located within the ethmoid sinus in the first and had an abnormal size and shape in the second case. In both cases, during urgent surgical repair, the incarcerated medial rectus muscle was gently released from linear non-displaced medial wall fractures and ocular motility normalized postoperatively.

CONCLUSIONS. In pediatric patients sustaining blunt orbital trauma, medial rectus incarceration should be considered and managed accordingly. (Eur J Ophthalmol 2004; 14: 330-3)

Key Words. Blowout fracture, Diplopia, Muscle entrapment, Ophthalmoplegia, Orbit fracture, Trapdoor fracture

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INTRODUCTION

Abnormal ocular motility following blunt orbital trauma can result from numerous abnormalities including muscle contusion, cranial nerve palsy, the mass effect of soft tissue edema and hemorrhage, globe malposition and muscle malposition or entrapment. The latter can be subdivided into muscle impingement by bone fragments of the adjacent orbital wall of a comminuted fracture, common in adults, and muscle incarceration in trapdoor-type fractures, seen almost exclusively in children. Trapdoor fractures result from a linear fracture with outward displacement of adjacent bone, which immediately returns to its original position, potentially incarcerating orbital soft tissue (1-6).

Commonly pediatric trapdoor fractures are characterized by little evidence of soft tissue trauma, a relative lack of enophthalmos, little to no bone displacement on imaging, and may be associated with an oculocardiac reflex on attempted ocular motility (4). The vast majority of published cases of trapdoor fractures with muscle incarceration have involved the orbital floor (1-5). Although, medial rectus muscle impinge-

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ment by comminuted fractures is well recognized, reports of medial rectus trapdoor fracture incarceration are vanishingly rare (5). In this report we describe two such cases and compare our findings to what has been published regarding inferior trapdoor fractures.

RESULTS

Case 1

A healthy eleven year-old male was evaluated for diplopia occurring immediately after being punched in the left eye two days before referral. There was minimal upper eyelid ecchymosis without additional signs of injury (Fig. 1a). Abduction and adduction of the left eye were limited. The remainder of the examination, including exophthalmometry measurements, was normal. Computed tomography (CT) demonstrated no orbital wall displacement with the medial rectus located within the ethmoid sinus (Fig. 1b). Under anesthesia, prior to urgent surgical repair the day of presentation, forced duction testing confirmed restriction. Via anterior orbitotomy a linear fracture of the medial wall, with minimal bone displacement, was found. Incarcerated soft tissue including the medial rectus muscle was gently released and returned to its normal position without bone fixation. Ocular motility normalized by the third post-operative month with symmetric globe positions.

Case 2

A healthy fourteen year-old male developed diplopia and nausea with ocular motility following blunt left orbital trauma during a boxing match three days prior to referral. There was no appreciable evidence of external injury including a lack of ecchymosis, chemosis, and subconjunctival hemorrhage. Left eye abduction and adduction were markedly reduced and the remainder of the examination was unremarkable. No significant bone displacement was seen on CT. The medial rectus muscle was in a normal location but had an abnormal shape and was circumferentially smaller than the contralateral muscle (Fig. 2). Surgical repair was performed the day of initial evaluation. Similar to case 1, forced duction testing under anesthesia demonstrated marked restriction to ab-

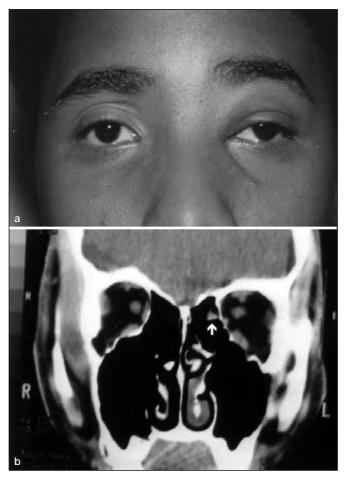


Fig. 1 - Patient 1. **a)** Clinical appearance with evidence of trauma limited to mild left upper eyelid ecchymosis. **b)** Computed tomography demonstrating the left medial rectus muscle located within the adjacent ethmoid sinus (arrow) with an absence of bone displacement.



Fig. 2 - Patient 2. Computed tomography demonstrating left medial rectus incarceration in a medial orbital wall trapdoor fracture (arrow).

duction. Intraoperatively, the medial rectus muscle was freed from a non-displaced medial orbital wall fracture and ocular motility gradually returned to normal during the first three post-operative months.

DISCUSSION

The majority of available information regarding pediatric trapdoor fractures and muscle entrapment refers to the orbit floor. As illustrated by this report, medial wall trapdoor fractures with medial rectus muscle entrapment may also occur. These cases demonstrated important similarities to floor fractures. Regarding management, as is commonly seen with inferior trapdoor fractures, both patients had complete return of extraocular motility with immediate intervention. Additionally, there were several important similarities in presentation: namely, the relative lack of external signs of trauma.

Jordan et al coined the term "white eyed blowout fracture" emphasizing the common lack of external signs of trauma, which may decrease clinicians' level of suspicion of serious injury (3). This along with additional characteristics of trapdoor fractures makes them likely to evade recognition. Radiologic evidence of injury may be minimal; attention should be directed at the location and appearance of the muscles themselves, as there may be no obvious bone abnormality. Lastly, the presence of an oculocardiac reflex with associated nausea may decrease cooperation making thorough ocular motility evaluation difficult in a child (4). As illustrated by the described cases, medial rectus muscle incarceration in a trapdoor fractures may present similarly. Both patients had minimal evidence of trauma and one had a mild oculocardiac reflex, evidenced by nausea with attempted ocular motility.

Several reports have suggested that superior outcome is achieved when surgical intervention of muscle entrapment in inferior wall trapdoor fractures is performed within days of injury (7). Egbert et al demonstrated more rapid resolution of diplopia with early intervention. Although they reported a trend towards a decrease in persistent long-term diplopia, this difference was not statistically significant (1). Bansagi and Meyer found that time to surgical intervention was critical, with earlier surgical repair yielding better clinical outcomes (5). Jordan et al similarly reported improved outcomes with early intervention (3). Altogether, improved short and long-term outcomes have been well established with early, within a few days of injury, surgical repair of inferior rectus entrapment in trapdoor fractures. Given established benefit in patients with inferior rectus involvement, it seems prudent to similarly manage medial rectus entrapment. Although admittedly anecdotal, this is supported by the cases described here. We observed favorable outcomes with early intervention of medial rectus incarceration; both patients underwent repair within three days of injury with complete recovery of ocular motility.

The exact mechanism of orbital wall fracture has been debated. There are two predominant proposed mechanisms: the hydraulic theory and the buckling theory (8-10). The hydraulic theory maintains that globe retropulsion results in elevated orbital pressure causing a fracture (8). The buckling theory suggests that fracture results from a force transmitted to the orbit rim resulting in folding of the adjacent wall (9). There is clinical and experimental evidence supporting each theory (8-10). Depending on the nature of injury, hydraulic and buckling mechanisms likely contribute to varying degrees, with some fractures being caused by a single and others by a combination of mechanisms.

Trapdoor fractures occur when the bone flaps return to their normal position. This is dependent on ample bone elasticity, characteristic of children. In contrast, the brittle nature of adult bone leads to the frequently seen comminuted fractures (6, 11). Soft tissue prolapse through a trapdoor fracture likely results from a rise in orbital pressure, although the fracture itself might result from either a hydraulic or buckling mechanism. In comminuted fractures soft tissue prolapse might result from more passive mechanisms, such as gravity or soft tissue edema. An elaborate connective tissue network surrounds and connects the extraocular muscles (12). Restriction of ocular motility may result from entrapment of either a rectus muscle itself or surrounding connective tissue (13). This is illustrated by the cases described in this report. In the first case the muscle itself was trapped within the fracture. This is contrasted by the second case where restricted motility was largely due to entrapment of the surrounding connective tissues. In such cases, the muscle may be in a relatively normal position; there-

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fore, radiographic evidence of entrapment is particularly lacking. This further illustrates that entrapment is a clinical diagnosis and stresses the importance of motility evaluation.

In closing, in pediatric patients with blunt orbital trauma, medial rectus incarceration should be considered despite a relative lack of clinical and radiographic evidence and managed accordingly. Early intervention will likely achieve resolution of an associated ophthalmoplegia. Reprint requests to: Timothy J. McCulley, MD Director of Ophthalmic Plastic and Reconstructive Surgery and Neuro-Ophthalmology Stanford University School of Medicine Department of Ophthalmology 900 Blake Wilbur Dr, Rm W-3074 Stanford, California 94304-5353 mcculley@stanford.edu

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