Orbital Floor Fracture with Atypical Extraocular Muscle Entrapment Pattern and Intraoperative Asystole in an Adult

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Abstract

Keywords

- orbital fracture
- extraocular muscle entrapment
- adult trapdoor fracture

Extraocular muscle entrapment in a nondisplaced orbital fracture, although a well-known entity in pediatric trauma, is atypical in adults. It can present with a triad of bradycardia, nausea, and in rare cases, syncope, and result in severe fibrosis of damaged and incarcerated muscle. We present a case of muscle entrapment in a partially nondisplaced two-wall orbital fracture with accompanying preoperative bradycardia and intraoperative asystole in an adult.

Extraocular muscle entrapment in the setting of orbital floor fractures, when precisely defined as muscle incarcerated in a nondisplaced fracture line, is rarely seen in adults. This is in contrast to herniation of extraocular muscles through a fracture defect, which is common. The oculocardiac reflex (OCR) is a sign that can be elicited in patients with entrapped muscle tissue and is typically defined as the triad of bradycardia, nausea, and syncope. We present an interesting case of muscle entrapment in a partially nondisplaced two-wall orbital fracture with accompanying preoperative bradycardia and intraoperative asystole in an adult.

Case Report

A 27-year-old man was struck with a fist in the left periorbital area, and onset of binocular vertical diplopia and left orbital pain with eye movements was immediately noted. He was otherwise asymptomatic. Past medical history was notable only for asthma.

On examination, best corrected visual acuity and intraocular pressures were within normal limits. There was no relative afferent pupillary defect. Extraocular movement of

the right eye was normal, but demonstrated an 80% reduction of upgaze of the left eye (► **Fig. 1**). There was an elicited OCR with the patient's heart rate consistently dropping 33%, from ~75 to 50 beats per minute during attempted upgaze with associated nausea and presyncopal symptoms. Horizontal motility was normal. There was 2 mm of enophthalmos of the left globe. Anterior and posterior segment exams of both eyes were within normal limits; no associated injuries were identified on the ED screening examination.

Noncontrast orbital CT scan demonstrated a comminuted left orbital floor fracture and an associated minimally displaced fracture of the left lamina papyracea and nasal bones. The shape of the inferior rectus muscle was distorted vertically and it appeared to be partially entrapped in the orbital floor fracture (► **Fig. 2**).

Surgical exploration and fracture repair were conducted as soon as possible and within 24 hours of presentation. Forced duction testing performed intraoperatively confirmed restrictive strabismus. Upon exploration, the orbital floor fracture was identified and entrapped inferior rectus muscle tissue at the orbital floor fracture site identified. During release of the anterior portion of entrapped inferior rectus muscle, gentle

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Fig. 1 Left-sided restriction enophthalmos in primary gaze (above) with restriction in upgaze (below) in a patient acutely after left sided orbital trauma.

Fig. 2 Computed tomography of a left partially displaced orbital floor fracture and nondisplaced medial wall fracture with prominent herniation of orbital fat through the defect centrally and posterior entrapment of the inferior rectus muscle in nondisplaced fracture elements (red arrows).

traction reliably resulted in self-limited bradycardia. Once released, while exposing the posterior segment of entrapped muscle, suctioning of blood in the maxillary sinus was performed through the fracture defect in the orbital floor. This maneuver likely led to the creation of a vacuum in the sinus with resultant tension on the orbital fat and inferior rectus muscle. Immediate hypotropia and worsening of the enophthalmos was observed, and the patient sustained 15 seconds of asystole (►Fig. 3). The muscle was released and the anesthesia team administered 0.5 mg of atropine intravenously and regular sinus rhythm resumed. Ultimately, the fractures of the medial orbital wall and orbital floor were repaired with a prefabricated titanium orbital plate, which was secured to the orbital rim internally with a single titanium screw.

Forced duction testing performed immediately following fracture reduction showed free movement in all directions. Postoperative CT scan showed an appropriately positioned implant and release of the entrapped inferior rectus muscle (►Fig. 4). At 1-day, 1-week, and 3-month follow-up visits, diplopia and restriction of upgaze had completely resolved (►Fig. 5).

Discussion

Extraocular muscle entrapment in a nondisplaced orbital fracture is seen near exclusively in the pediatric population.^{1,2} Compared with adults, the bones of children contain a higher proportion of osteocytes relative to osteoblasts, and decreased mineralization, leading to increased compliance.³ Consequently, pediatric orbital fractures may not have the characteristic orbital fracture bone fragmentation pattern seen in adults with displacement of orbital bones and orbital contents often into adjacent sinuses. Instead, after blunt periorbital trauma in a child, the orbital floor bones may become temporarily displaced in a hinge-like fashion before snapping back into anatomic, nondisplaced position. In adult and pediatric cases of orbital trauma, simultaneous pressure on the globe at the time of facial impact pressurizes and displaces the orbital contents through the fracture. In children, muscle entrapment can occur if the contents do not escape before the compliant bone fragment snaps back into place (as seen in ►Fig. 6). This fracture pattern was first described in 1965 by Soll and Poley, who coined the term “trapdoor fracture.”⁴ Although a case of inferior rectus muscle sheath entrapment in an adult patient occurring in a non-displaced fracture has been reported,⁵ review of the literature suggests that extraocular muscle entrapment is extremely rare in the adult subgroups.^{2,6} Similarly, a case of medial rectus entrapment in a nondisplaced medial wall fracture has been reported,⁷ but review of all reported cases of medial rectus entrapment appears to be near exclusive to children.⁸

In this patient, a large orbital floor and medial wall fracture was present. Although the anterior portion was classically displaced, the posterior portion of the floor was atypically nondisplaced. Consequently, posterior portions of the left inferior rectus muscle and sheath became caught between two nondisplaced bone fragments (►Fig. 2, red arrows) and

Fig. 3 Intraoperative vital sign tracing demonstrating bradycardia to ~50 beats per minute, followed by frank asystole. After atropine administration, resultant tachycardia is noted.

clinically evident extraocular muscle entrapment was seen. While *entrapment* through a nondisplaced fracture is rare in adults, this should be differentiated from *herniation* of orbital tissue in a displaced fracture, which is common. In herniation, there is no strangulation of muscle tissue and the risks of muscle damage, necrosis, or the OCR are lower. Occasionally when muscle or perimuscular tissue is herniated, limitation of gaze can be manifest clinically and a distortion of the shape

of the inferior rectus can be observed radiographically⁶ (►Fig. 7).

On occasion, forced duction testing can differentiate between a gaze palsy related to muscle dysfunction or secondary to intramuscular hemorrhage. As the long-term sequelae differ, so does the management of these three mechanisms of gaze palsy—muscle bruising is observed, muscle/fascial entrapment in a displaced fracture is repaired nonemergently, and muscle incarceration through a nondisplaced fracture is repaired urgently to prevent secondary muscle necrosis and fibrosis. The radiographic and clinical terminology to describe entrapped, herniated extraocular muscle through a displaced fracture should be specific to differentiate this scenario from a nondisplaced fracture with muscle incarceration given the significant difference in management.

Similarly, the OCR is a feature found most commonly in pediatric patients with nondisplaced floor fractures.⁹ While there has been report of a trapdoor medial orbital fracture in an adult with nausea¹⁰ and another with nausea on presentation and delayed intraoperative bradycardia,⁹ we could not find report of the full triad of the OCR on presentation caused by extraocular muscle entrapment in a partially displaced orbital fracture in an adult. In addition, the patient's anatomy and injury morphology, different than most pediatric cases

Fig. 4 Postoperative computed tomography demonstrating repair of the orbital floor and medial wall fracture with titanium implant, highlighted in pink. The green overlay represents a mirror image of the right orbital bone segments.

Fig. 5 Postoperative photograph 3 months after surgery demonstrating orthotropia in primary gaze (above) and intact upgaze (below).

Fig. 6 CT scan of a 9-year-old girl with a severe up-gaze deficit from a left-sided nondisplaced orbital floor fracture with entrapment of the inferior rectus muscle.

with a similar entrapped muscle, led to an episode of intraoperative asystole.

The OCR, though rare, is a sign that may accompany entrapped muscle tissue, and it is one indication that immediate orbital fracture repair is warranted.¹¹ Comprising a triad of bradycardia, nausea, and syncope, the ophthalmic division of the trigeminal nerve serves as the afferent limb of the reflex. The impulse passes by way of the reticular formation to the vagus nerve's visceral motor nuclei, and the efferent message is carried by the vagus nerve to the heart and stomach.⁹ The cardiac end-organ effect includes decreased activity at the sinoatrial node.¹² The risk of fatal cardiac arrhythmia with the OCR is not trivial. Heart block, junctional rhythms, asystole, and death have been reported.¹³

In our case, the OCR noted preoperatively with attempted gaze and intraoperatively with gentle manipulation was moderate and consistent. During exposure of the posterior aspect of the fracture, the tension on the muscle was exacerbated when the orbital fat prolapsed through and then occluded the bone defect while suction was in the maxillary sinus. The initial surgical response when bradycardia develops during orbital surgery is to remove instrumentation from the orbit. In this case, when this was done, inspection of the eye revealed worsening of enophthalmos and new hypotropia. As this was recognized and released, the anesthesiologist administered atropine. Excellent communication between

Fig. 7 CT scan of 28-year-old man with a moderate up-gaze deficit from a right-sided displaced orbital floor fracture with herniation and distortion of the inferior rectus muscle.

the anesthesiologist and surgeon also helped to manage the problem quickly, and avoid significant sequela. This particular surgical scenario consisting of unscarred, mobile, and excessive orbital fat, muscle entrapment in the nondisplaced portion of the fracture, and a defect anteriorly through which suction instrumentation could be introduced into the sinus, highlights the etiology and magnitude of the OCR.

This case illustrates the importance of communication and partnership between surgeon and anesthesiologist for prompt restoration of normal sinus rhythm in such a scenario, and prevention of further episodes. In particular, not only should the surgeon remain attentive to the anesthesiologist, but it also has been recommended that the surgeon inform the anesthesiologist prior to any portion of the procedure that may predispose to the development of the OCR (e.g., placing pressure on the globe or manipulation of extraocular muscles) so that the anesthesiologist is prepared accordingly.¹⁴

It is also helpful to keep in mind other modifiable factors that have been reported to increase the risk of OCR to assess patient risk. Cardiac disease, hypoxia, and hypercarbia have been shown to increase patients' risks for developing OCR.¹⁴ Ophthalmic irrigation with cold water (0–20°C), potent narcotic agents (such as sufentanil and alfentanil), β -blockers, and calcium channel blockers have all been reported to exacerbate OCR.^{14–16} Perhaps most applicable to entrapped muscle repair in the pediatric population, anesthetic choice during strabismus surgery has been suggested to play a role in limiting the OCR. In a study comparing sevoflurane to halothane in such cases, Allison et al demonstrated fewer dysrhythmias and less pronounced stimulation induced bradycardia with sevoflurane.¹⁷

The treatment of OCR is rapid discontinuation of the presumptive stimulus. Surgical release of the entrapped muscle should be performed promptly in any unstable patient. In addition, in cases of true muscle entrapment in a nondisplaced fracture segment, long-term diplopia as a result of presumed necrosis of muscle tissue and fibrosis of the muscle sheath can occur as early as 8 hours from injury.¹⁸ Surgical release within 24 to 48 hours has been recommended as diplopia occurs with increased frequency in repairs performed further from injury.^{18,19} If an OCR is noted, medical therapy with antimuscarinic drugs such as atropine or glycopyrrolate can be used preoperatively or intraoperatively to aid in the temporary restoration of physiologic heart rate and prevent asystole. In refractory cases with an inability to reestablish cardiac output, Advanced Cardiac Life Support protocols should be followed.¹²

Though rarely elicited in the adult population, it is important that surgeons treating patients with orbital trauma be aware of the OCR, including its anatomic basis, potential causes, mitigating factors, and management. This case demonstrates that adult extraocular muscle entrapment can occur and lead to the OCR. Open communication with the anesthesiologist, immediate discontinuation of the presumptive stimulus, and administration of atropine led to prompt resumption of normal sinus rhythm, without further episodes and the patient had an excellent outcome.

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