venous liposomal amphotericin B without local antifungal medication or further surgical debridement of tissue.

The patient was treated as an inpatient for 13 days with amphotericin B followed by a 2-week outpatient course of amphotericin B, as recommended by the Centers for Disease Control. The patient did not exhibit any signs of active Mucor infection at any time and has remained free of active Mucor infection in 6 months of close follow-up.

DISCUSSION

Although biopsy of the lacrimal sac for patients with primary acquired NLDO has been debated for many years, this case is an example of the potential to miss a fatal diagnosis.¹⁻⁶ It has been suggested that biopsies should only be performed in selected patients to contain costs. These include patients with clinical/radiographic suspicion of a lacrimal sac tumor, abnormal appearing tissue during DCR, or history of sarcoid, Wegener granulomatosis, lymphoma, and other infiltrative diseases.⁶ Our patient did not fit these criteria.

Orbital mucormycosis in an immunocompetent patient is exceedingly uncommon. Mucormycosis is typically a rapidly progressing, fatal, opportunistic infection that occurs in diabetic and immunocompromised patients. Sino-orbital mucormycosis has been reported in immunocompetent patients; however, these patients typically have a history of trauma, oral surgery, or chronic sinusitis.⁷ Our patient had no such history. The only reported cases of dacryocystitis from Mucorales species have been seen in immunocompromised patients with active signs of infection.

Other studies have incidentally noted fungal hyphae and elements in pathologically definite dacryoliths.⁸ However, no studies, to our knowledge, have noted zygomycete material in dacryoliths or on routine DCR pathologic specimen.

Although ordering histopathology on specimens taken during routine DCR, even with clinically benign appearing tissue, increases financial costs, there is a real risk of missing serious and potentially fatal diagnoses without routine biopsy. This rare and potentially fatal diagnosis would not have been diagnosed and treated successfully without a routine lacrimal sac biopsy.

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Oculocardiac Reflex Associated With a Large Orbital Floor Fracture

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Abstract: A 40-year-old man presented with bradycardia, left eye pain, and intermittent nausea 1 day after blunt trauma to the left orbit. Imaging revealed a large orbital floor fracture with significant herniation of orbital contents but no obvious extraocualar muscle entrapment. Oculocardiac reflex was suspected, and the fracture was repaired surgically within 24 hours of presentation. His bradycardia resolved immediately postoperatively. This case is a unique presentation of the oculocardiac reflex in a large orbital floor fracture with significant herniation of orbital contents but without extraocualar muscle entrapment.

he oculocardiac reflex is a well-known phenomenon that classically includes the triad of bradycardia, nausea, and syncope.¹ The afferent limb is the ophthalmic division of the trigeminal nerve. This signal travels through the reticular formation to the vagus nerve's visceral motor nuclei, which increases parasympathetic impulses carried by the vagus to the cardiovascular and gastrointestinal systems. In ophthalmology, the oculocardiac reflex has been reported secondary to pediatric trapdoor orbital floor fractures, strabismus surgery, and other less common orbital causes.¹⁻⁴ It is generally thought that the manipulation of the extraocular muscles stimulates the trigeminal nerve and initiates the reflex. Occasionally, in facial fractures that do not involve the globe or extraocular muscles, the trigeminal nucleus can be stimulated via the maxillary or mandibular divisions.⁵ We present a unique case of oculocardiac reflex due to a large orbital floor fracture that did not have evidence of extraocular muscle entrapment.

CASE REPORT

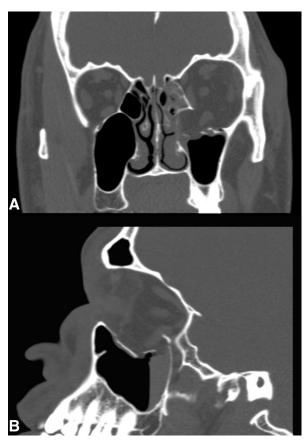
A 40-year-old man presented 1 day after sustaining blunt trauma to the left orbit during an assault. He complained of left eye pain and intermittent nausea but denied changes in vision, diplopia, lightheadedness, or syncopal episodes. His vital signs on presentation were significant for a heart rate of 38 beats per minute and blood pressure of 111/72 mm Hg. He demonstrated left-sided periorbital ecchymosis and decreased tactile sensation in the distribution of the maxillary division of the trigeminal nerve. He complained of pain on upgaze of the left eye but denied diplopia and did not demonstrate any restriction of extraocular muscles. His best-corrected visual acuity at distance was 20/30 OD and 20/40 OS. There was no afferent

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Coronal (A) and sagittal (B) CT show a large fracture of the left orbital floor with herniation of a large volume of orbital fat and inferior tenting and subsequent vertical elongation of the inferior rectus muscle.

pupillary defect, and intraocular pressure on applanation testing was 15 mm Hg OU. Examination of the globe and fundus revealed significant subconjunctival hemorrhage but no other abnormalities. CT of the orbits revealed a large, depressed left inferior orbital wall fracture (>80% floor defect) with herniation of orbital fat but no evidence of extraocular muscle entrapment. However, there was radiographic evidence of inferior tenting with vertical elongation of the inferior rectus muscle (Fig.).

On assessing the patient's electrocardiogram, he was noted to have a sinus bradycardia and intermittent conversion to a junctional rhythm when the heart rate dropped below 40 beats per minute.

The patient then underwent uneventful repair of left orbital floor fracture with a Medpor implant within 24 hours of presentation. There were no intraoperative or postoperative complications. One day postoperatively, the patient's heart rate was 60 beats per minute, and blood pressure was 154/80 mm Hg. His EKG demonstrated a normal sinus rhythm. Three weeks postoperatively, the ocular examination was within normal limits, and the heart rate was 62 beats per minute.

DISCUSSION

This case demonstrates an oculocardiac reflex from a large orbital floor fracture with significant herniation of orbital contents in the maxillary sinus but without obvious signs of extraocular muscle entrapment. The patient presented with bradycardia that appeared vagally mediated on EKG. In addition, the bradycardia resolved with surgical repair of the fracture and repositioning of the herniated orbital contents.

The oculocardiac reflex has been previously associated with "trapdoor-type" orbital floor fractures.¹ The orbital trapdoor fracture is rare and is found in children and young adults likely due to the elasticity of orbital bone in these younger patients. In a trapdoor fracture, the fracture fragment hinges open and allows herniation of orbital contents before the fragment snaps back in its original position. As a result, the orbital contents and often the inferior rectus muscle are entrapped in the fracture site and can result in an oculocardiac reflex.

Radiographic findings can often be helpful in evaluating patients with orbital fractures. Although there may not be clear evidence of extraocular muscle entrapment, elongation or rounding of the muscle can often indicate a persistent tractional force being placed on the muscle, which can often present in a similar fashion to entrapment.⁶ In our case, the fracture site was large and without clinical or radiographic evidence of entrapment. However, the large orbital floor defect and subsequent herniation of orbital contents created a tractional force on the inferior rectus similar to that found during entrapment or during manipulation during strabismus surgery.

The stimulus required to initiate the oculocardiac reflex has been shown to be variable, and it can be a graded response based on increasing traction.⁷ Continuation of this stimulation can cause escape from or fatigue of the reflex such that the heart rate does not remain maximally decreased.⁸ In our case, this phenomenon was difficult to appropriately evaluate because the patient's heart rate converted to a junctional rhythm (heart rate below 40 beats per minute), which was likely secondary to the vagal stimulus he received.

The oculocardiac reflex is a rare occurrence in orbital floor fractures. It causes a vagally mediated bradycardia that classically presents with nausea and syncope and can rarely prove fatal.⁹ Therefore, urgent repair is indicated in these cases. This case suggests the need to evaluate patients with orbital floor fractures carefully for signs and symptoms of the oculocardiac reflex.

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