Persistent Upgaze Restriction after Orbital Floor Fracture Repair

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Abstract

Keywords

- orbital fracture
- trapdoor fracture
- inferior rectus entrapment

Here we present two cases of marked postoperative upgaze restriction after successful repair of orbital floor fracture and release of inferior rectus entrapment. In both cases, follow-up imaging showed enlargement of the inferior rectus, and gradual resolution of gaze limitation was observed over several months of conservative management. Thus, in patients with postoperative findings suggestive of residual inferior rectus entrapment, follow-up imaging is indicated prior to returning to the operating room. With a markedly swollen inferior rectus muscle but no radiographic evidence of residual muscle entrapment in the fracture, a trial of conservative management may be warranted.

Persistent diplopia has commonly been reported as a complication of orbital floor fracture repair in children.^{1,2} In patients with postoperative diplopia, restricted extraocular motility, and positive forced ductions, surgical exploration for residual inferior rectus entrapment is indicated. However, here we present two cases of marked upgaze limitation and positive forced ductions 1 week following floor fracture repair, but without remaining entrapment on imaging or surgical exploration. Postoperative imaging in both cases revealed a markedly swollen inferior rectus muscle, and both patients demonstrated gradual improvement of upgaze over several months.

Case 1

The first patient was a 16-year-old male patient who presented with a left orbital floor trapdoor fracture with entrapment of the inferior rectus (►Fig. 1a). At the time of surgical repair, which occurred within 24 hours of the initial presentation, the entrapped soft tissues were successfully released with subsequent normalization of forced ductions, and no implant was placed. At postoperative week 1, there was near-complete limitation of upgaze and downgaze with moderately restricted forced ductions (►Fig. 2a). A follow-up CT scan showed enlargement of the left inferior rectus but no evidence of entrapment (►Fig. 1b). The patient was followed

up conservatively and given a 1-week methylprednisolone oral steroid taper (Medrol Dosepack). At postoperative week 3, the first sign of improvement was noted. Over the subsequent weeks, motility continued to gradually improve. At postoperative month 3, there was only mild limitation in upgaze and otherwise full motility (►Fig. 2b).

Case 2

The second patient was a 5-year-old male patient presenting with persistent, marked limitation in upgaze and diplopia 1 week following repair of a left orbital trapdoor floor fracture by an outside provider with placement of an absorbable implant. The initial surgical intervention was performed within 24 hours of the injury. Based on the finding of dramatic limitation in elevation despite surgical repair, surgical exploration was indicated to evaluate the orbital implant and soft tissues. At the time of surgical exploration, the implant was removed and residual entrapped soft tissues were released. Forced ductions were negative at the end of the case. At postoperative week 1, there was complete restriction in upgaze with persistent diplopia (►Fig. 3a), and CT showed enlargement of the inferior rectus and possible soft-tissue incorporation into the fracture line (►Fig. 4). On subsequent repeat orbitotomy, forced ductions were mildly restricted, but

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Fig. 1 A 16-year-old male patient presented with orbital floor fracture and left inferior rectus entrapment appreciated on coronal CT imaging of the orbits (a). Coronal CT imaging of the same patient at postoperative week 1 with enlargement of the left inferior rectus but no evidence of muscle entrapment (b).

Fig. 2 The same 16-year-old male patient presented at postoperative week 1 after surgical release of the entrapped inferior rectus muscle with near-complete limitation of vertical gaze (a). After 3 months of observation, motility was nearly completely full (b).

there was no residual entrapment observed on exploration. At postoperative week 1, there was persistent moderate limitation to upgaze with associated diplopia. Gradual resolution of his symptoms occurred over the next several months with near-complete recovery of motility by postoperative month 3 (► Fig. 3b).

Fig. 3 A 5-year-old male patient presented at postoperative week 1 after release of entrapped soft tissues with persistent limitation of upgaze and positive forced ductions (a). On repeat surgical exploration, no residual entrapment was found, and after 3 months of conservative management, the restriction had resolved (b).

Fig. 4 Coronal CT imaging of the 5-year-old male patient with left orbital floor fracture at postoperative week 1 after surgical exploration, showing enlargement of the left inferior rectus muscle and possible residual entrapment of soft tissues in the fracture line. On subsequent repeat surgical exploration, no residual entrapment was found.

Comment

Orbital floor fractures often result from blunt trauma to the orbit. In children, they tend to be of the trapdoor type, which can cause inferior rectus entrapment presenting clinically with limitation of upgaze, nausea, bradycardia, and positive forced ductions.³ The intermuscular septum may also be entrapped resulting in restriction. They can be repaired without an implant in some cases, or with a variety of alloplastic, allogeneic, or autogenous implants.⁴ Following surgical repair, persistent diplopia has frequently been reported as a complication.^{1,2} This can be due to muscle or nerve damage, residual entrapment, edema, or hemorrhage.⁵

With muscle or nerve damage, depression of the eye is limited as inferior rectus function is decreased, but forced ductions remain negative.⁶ These patients often exhibit a hypertropia, which improves over time with resolution of inferior rectus paresis. In the case of persistent entrapment, forced ductions are positive and further surgical exploration is indicated.

In our two cases, both patients presented with marked upgaze restriction and positive forced ductions 1 week following surgery, imitating residual inferior rectus entrapment. In both cases, follow-up imaging showed enlargement of the inferior rectus. In the first case, there was no radiographic evidence of residual entrapment on follow-up imaging, and the patient was managed conservatively. In the second case, there was possible remaining soft-tissue entrapment on imaging. However, on surgical exploration, no entrapment was found, and the patient was managed with observation. A possible mechanism explaining these clinical and imaging findings is marked postoperative edema or contusion of the inferior rectus resulting in resistance of the muscle body to the elongation necessary to allow the globe to rotate superiorly. This may be iatrogenic from either mechanical or thermal trauma during surgery. As the swelling and contusion improved, this limitation of upgaze resolved in both patients.

In both cases, near-complete resolution was observed by 3 months after surgery. A short course of oral steroid treatment was used in the first case, but it is not known

whether this hastens recovery. Thus, in patients with postoperative findings suggestive of residual inferior rectus entrapment, follow-up imaging is indicated before returning to the operating room. With a markedly swollen inferior rectus muscle but no radiographic evidence of residual muscle entrapment in the fracture, a trial of conservative management with or without oral steroids may be warranted.

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