



A rare case of post operative traumatic optic neuropathy in orbital floor fracture

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Purpose

An orbital blowout fracture is a commonly occurring facial trauma disorder that accounts for 7.6% of isolated facial fractures, and post operative complications include diplopia, enophthalmos, hypoesthesia and optic neuropathy. Among these complications, optic neuropathy is a potential blinding complication of an orbital blow out fracture. The most common form of traumatic optic neuropathy is an indirect injury to the optic nerve due to intraorbital hemorrhage, vascular insufficiency or a nerve sheath injury whereas direct damage to the optic nerve during dissection and insertion of implant materials is also possible.

We report a case of a patient with post operative traumatic optic neuropathy due to indirect nerve damage after traction forced was applied to release a severe adhesion during a dissection procedure.

Method

A 35-year-old female patient was referred from the ophthalmology department complaining of right eye enophthalmos and presented for surgical correction of a right-sided upward gaze limitation (Fig. 1A & B). Visual acuity was within the normal range on the right 0.9 and left sides 0.7. Computed tomography (CT) images of facial area, showed a focal bony dehiscence accompanying the inferior rectus muscle and fat entrapped in the medial aspect of the inferior wall of the right orbit (Fig. 2). An orbital tissue hernia including the inferior rectus muscle within the bone defect was released, and the orbital floor was reconstructed using a 2.0 × 3.0 cm synthetic resorbable implant. Careful dissection was difficult because of the severe adhesion surrounding the inferior rectus muscle.



Fig. 1. Patient with post-traumatic enophthalmos and hypoglobus of the right eye

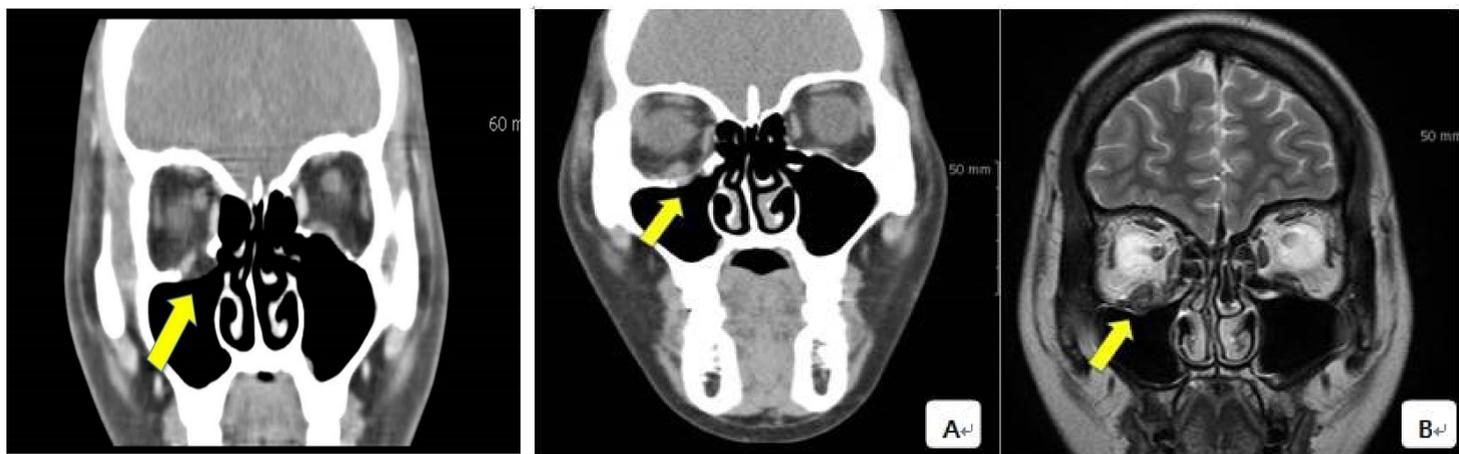


Figure 2. Coronal images showed an orbital floor fracture with inferior rectus muscle herniation.

Figure 3. (A) Postoperative computed tomography scan showed orbital floor reconstruction with synthetic resorbable implant and improvement of the herniation of inferior rectus muscle. (B) Postoperative coronal magnetic resonance image revealed no compression on the optic nerve but thickening of the inferior rectus muscle was noted (arrow).

Result

On post operative day 2, a physical examination, revealed that vertical eye movement remained restricted and that there was a superior visual defect with decreased visual acuity from 0.9 to 0.15. No evidence of optic nerve compression was detected on the immediate postoperative CT images but demonstrated improvement in the right inferior rectus muscle entrapment was observed (Fig. 3A). The patient was treated with intravenous and oral corticosteroid and the synthetic resorbable implant was removed under local anesthesia on day 2 after the operation. Mild fibrosis and thickening around the inferior rectus muscle was confirmed on magnetic resonance imaging 3 weeks after surgery without optic nerve injury (Fig. 3B).

Conclusion

In our case, the fibrous adhesion was severe and indirect injury due to traction force applied during massive dissection to the optic nerve and its surrounding orbital structures appear to be the most common cause of optic neuropathy. The possibility of direct injury to the optic nerve was low as we performed dissection < 3.0 cm from the margin of the inferior orbit where the depth was 3.5–4.5 cm from the margin of inferior orbit to the optic foramen.