

## Traumatic Orbital Emergencies

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### Introduction

Traumatic orbital emergencies require rapid diagnosis, accurate documentation of visual and orbital functions, and in certain cases, urgent intervention to prevent permanent loss of vision or intractable diplopia. Such patients frequently have other life or sight-threatening injuries. Consequently adnexal pathology is not infrequently over-looked. The purpose of this article is to highlight the chief adnexal emergencies that can occur in the context of head and ocular injury, and to outline their optimal management.

### 1. Orbital compartment syndrome due to retrobulbar haemorrhage

#### What is orbital compartment syndrome (OCS)?

The orbital soft tissues are contained within the rigid bony orbital walls, with the anterior opening bounded by the globe, orbital septum and the lateral and medial canthal tendons. Retrobulbar haemorrhage causes a rapid rise in intraorbital volume and pressure, reducing retinal and optic nerve blood flow. Unless urgently decompressed, ischaemia rapidly ensues, potentially leading to retinal infarction and permanent complete blindness<sup>1</sup>.

#### When should I suspect OCS?

Any patient with an acute orbital injury is at risk of OCS. Presenting features include a rapid reduction in vision (with or without a RAPD), tense, swollen lids, orbital congestion, and proptosis.

Other examination findings variably include periorbital haematoma, restricted eye movements (Fig 1), high intraocular pressure, (associated nausea or vomiting), chemosis and central retinal artery occlusion.

#### What should I do if I suspect OCS?

Immediate decompression is required, with a lateral canthotomy and cantholysis<sup>1</sup>. The following steps are a guide to management:

1. Inject subcutaneous local anesthesia at the lateral canthus.
2. Grasp the lateral lower lid with toothed forceps.
3. Divide the lateral canthal tendon by performing a long, horizontal full-thickness cut with blunt-ended straight scissors.
4. Divide the restricting bands of septum between the lower lid and the orbital rim, by cutting any tight bands within the first incision. Upon division, the lateral lid becomes freely mobile.
5. No surgical closure is desirable or required; it will heal spontaneously with an imperceptible scar.
6. Periodically reassess the following parameters:
  - a. Distance visual acuity
  - b. Colour appreciation
  - c. Pupil reactions and resolution of a RAPD
  - d. Orbital pressure (estimated by palpation)
  - e. Intraocular pressure

7. If there is no improvement in visual function, further disruption of the orbital septum can be achieved by performing step 4 on the upper lid.
8. If there is still no significant improvement, a senior opinion should be sought.

These steps can be performed with little prior experience, and ophthalmologists should be prepared to guide a general casualty physician through such steps by telephone to avoid unnecessary delay. A common error is to arrange imaging or medical treatment first,<sup>2</sup> this delaying the only intervention which reduces the intraorbital pressure.

#### When should I refer?

Urgently refer to an orbital or maxillofacial surgeon for surgical draining if perfusion does not return despite superior and inferior cantholysis.

### Education of lateral canthotomy and cantholysis

New ophthalmologists and emergency doctors should prepare for their first emergency lateral canthotomy and cantholysis by;

- Reviewing videos on the subject:
  - [www.youtube.com/watch?v=R8QuwrMHBLO](http://www.youtube.com/watch?v=R8QuwrMHBLO)
  - [www.youtube.com/watch?v=bUAagMd\\_Q8A](http://www.youtube.com/watch?v=bUAagMd_Q8A)
- Assisting a routine lateral tarsal strip operation or lower lid-approach orbitotomy:
  - Comparing the pre-operative fixed lid with the fully released lid.
  - Cutting the lateral canthal tendon and tight bands of septum.

### 2. Traumatic optic neuropathy (TON)

#### What is TON?

Damage to the optic nerve occurs in 2.3% of head injury patients<sup>3,7</sup> and is most commonly associated with road traffic accidents<sup>4</sup>. Direct TON occurs due to direct laceration/compression of the optic nerve from a projectile or fractured bony fragment (Fig 3), whereas indirect TON occurs from coup-contrecoup forces<sup>3</sup> from energy transmitted through the skull from distant trauma. Indirect TON typically occurs within the optic canal where the dura and periosteum adhere closely<sup>4</sup>. It also occurs where the nerve transitions from mobile to fixed sites; at the orbital apex and intracranially, close to the falciform dural fold<sup>3,4</sup>. Direct shearing of nerve axons and their pial blood vessels occurs<sup>3</sup>, followed by disrupted axonal transport and optic nerve swelling. Pressure then rises within the optic canal leading to a vicious cycle of further reduction in blood flow, with consequent ischaemia and death of retinal ganglion cells<sup>4</sup>. Poor presenting vision and lack of improvement within 48 hours are poor prognostic factors<sup>4</sup>.

#### When should I suspect TON?

If there is

- Trauma to the head
- Reduced vision and/or colour vision
- RAPD or fixed dilated pupil.

#### What should I do if I suspect TON?

1. Exclude an OCS clinically by palpating for a tense orbit.
2. Perform fundoscopy to exclude optic nerve avulsion and other causes of visual loss. A typical acute TON fundus appears normal, the exception being with an anterior optic nerve injury in which the optic disc appears swollen and haemorrhagic.
3. Perform a CT orbit to exclude an optic canal fracture and nerve impingement by displaced bone (Fig 3). Rule out a rare optic nerve sheath haemorrhage (refer for optic nerve sheath fenestration<sup>3</sup>) or carotid cavernous segment pseudoaneurysm (refer to interventional radiology<sup>3</sup>).
4. If the optic nerve is completely transected or avulsed, with NPL vision, all intervention is futile.

5. Cochrane reviews<sup>8,9</sup> and a Meta analysis<sup>4</sup> conclude that there is insufficient evidence to support any one treatment of TON when compared to observation<sup>4</sup>. Thus, current accepted options include:
  - a. Observation alone. (40-60% recovery rate<sup>4</sup>).
  - b. High dose steroids with a maximum of 1g methylprednisolone daily, commenced within 8 hours of injury, if there is no traumatic brain injury or steroid contraindication (44-62% rate of visual improvement<sup>4</sup>)
  - c. Optic nerve decompression at the optic canal. (27-82% improvement  $\pm$ steroids<sup>3</sup>.) Outcome improves if;
    - i. Vision is light perception or better and
    - ii. Optic canal fracture and nerve compression is present<sup>3</sup>.

(Further information in online article)

**When should I refer?**

If the CT orbit shows an optic canal fracture and nerve compression, consider urgent optic canal decompression either transcranially (neurosurgery) or endonasally (ENT)<sup>3,4</sup>.

**3. 'Trapdoor' orbital floor fracture with muscle entrapment**

**What is a 'trapdoor' fracture?**

Blunt injury to the orbital cavity can result in displacement of bone at the weakest part of the orbit; the inferior and medial walls. If the displaced fracture recoils back, soft tissues - including connective tissue, muscle sheath, and the muscle belly itself - can become entrapped. Generally, this is a concern in children and younger adults, whose bone retains flexibility. The orbital floor is the most common fracture site (typically along the medial aspect of the infra-orbital canal) with the inferior rectus being the most commonly involved muscle. The entrapped muscle sheath or peri-muscular connective tissue leads to constriction and secondary ischaemia of the muscle belly itself (whether or not the actual muscle belly is entrapped within the fracture). Left unaddressed, such ischaemia can lead to fibrosis and loss of function<sup>5</sup>, causing long-term diplopia despite subsequent surgical release.

**When should I suspect a trapdoor fracture?**

If a child/adolescent presents with;

- A history, or suspicion, of blunt orbital or head injury
- Double vision (or closing the affected eye to negate this).
- Reduced upgaze due to restriction by the trapped inferior rectus (Fig 2), and often reduced downgaze due to reduced inferior rectus function.
- Such patients may also present with:
  - An oculocardiac reflex, in which attempted upgaze causes bradycardia, nausea, vomiting, syncope, cardiac arrhythmias and asystole<sup>5</sup>.
  - Cheek numbness from infraorbital nerve damage by an orbital floor fracture.

Cases are frequently overlooked in the absence of ocular signs such as haemorrhage, termed 'white-eyed blowout fracture'<sup>5</sup>. Furthermore, general malaise due to the oculocardiac reflex can result in erroneous investigation for other aetiologies such as head injury, entailing significant delay in treatment.

**What should I do if I suspect a trapdoor fracture?**

1. Instruct the patient NOT to:
    - a. Blow their nose (reduce orbital emphysema)
    - b. Eat or drink (urgent surgery may be indicated)
  2. If unconscious, perform a forced duction test to identify restricted motility.
  3. Order an orbital CT with fine cuts and coronal reformatting.
  4. On the coronal views, examine for:
    - a. A 'teardrop' sign where soft tissue is seen below the orbital floor.
    - b. Asymmetry of the extraocular muscle shape and location (Fig 2).
    - c. Lack of the fat plane between the muscle and bone.
- N.B. The fracture may be hairline and undisplaced.

5. Commence oral antibiotics to reduce orbital infection risk.
6. Refer urgently to an orbital surgeon to release the entrapped soft tissues within 48 hours, or sooner if the oculocardiac reflex is present<sup>5</sup>. Conservative management may be appropriate if there are no clinical signs despite radiological evidence<sup>5</sup>.

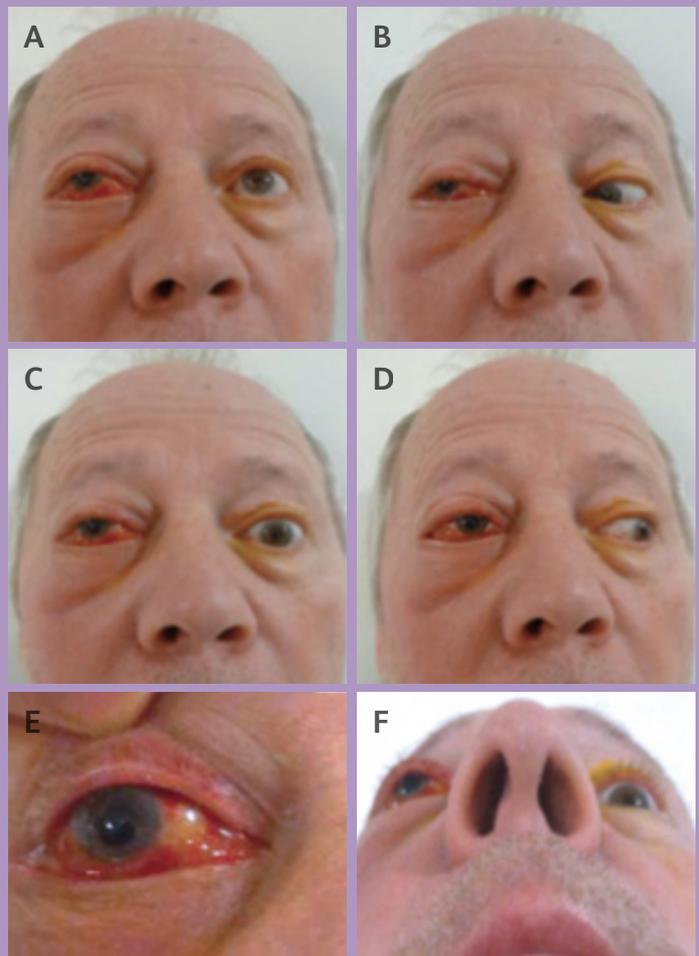
**When should I refer?**

Refer to an orthoptist for preoperative and postoperative documentation of ocular ductions.

If diplopia persists after stabilisation, refer to a strabismus surgeon.

**Conclusion**

Clinicians should have a high index of suspicion in all patients reporting visual symptoms following orbital or head injury. Retrobulbar haemorrhage is a visually devastating condition. Urgent decompression, a relatively straight-forward procedure, can rescue the patient from imminent and irreversible visual loss. Optimum management of traumatic optic neuropathy remains uncertain. Younger patients with head injury can suffer 'trapdoor' orbital fractures, which can present with few signs other than reduced upgaze.



**Figure 1** Images of a patient who developed a retrobulbar haemorrhage due to orbital trauma from a large piece of wood whilst chopping logs. Showing restricted eye movements in **A** up gaze, **B** right gaze, **C** primary position, **D** left gaze, **E** right dilated pupil, and **F** proptosis. Also seen here is periorbital swelling and conjunctival chemosis.

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