# Traumatic optic neuropathy

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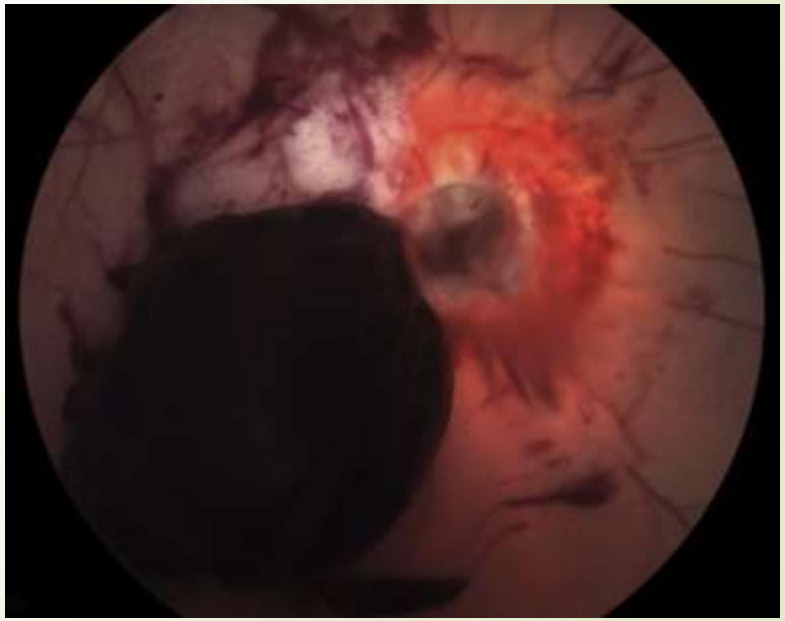


- **Traumatic optic neuropathy (TON)** refers to any insult to the optic nerve secondary to trauma.

- It can be classified depending on the site of injury **(optic nerve head, intraorbital, intracanalicular,or intracranial)** or according to the mode of injury (direct or indirect).

- In direct TON, there is significant anatomical disruption to the optic nerve, for example, from a projectile penetrating the orbit at high velocity (Fig. 1), or as a result of optic nerve avulsion (Fig. 2).

- **Indirect TON** is caused by the transmission of forces to the optic nerve from a distant site, without any overt damage to the surrounding tissue structures.

- The skull from **blunt trauma** is concentrated in the region of the optic canal.

- The ***intracanalicular segment*** of the optic nerve is particularly susceptible to this form of injury, because the Dural sheath is tightly adherent to the periosteum at this specific location.

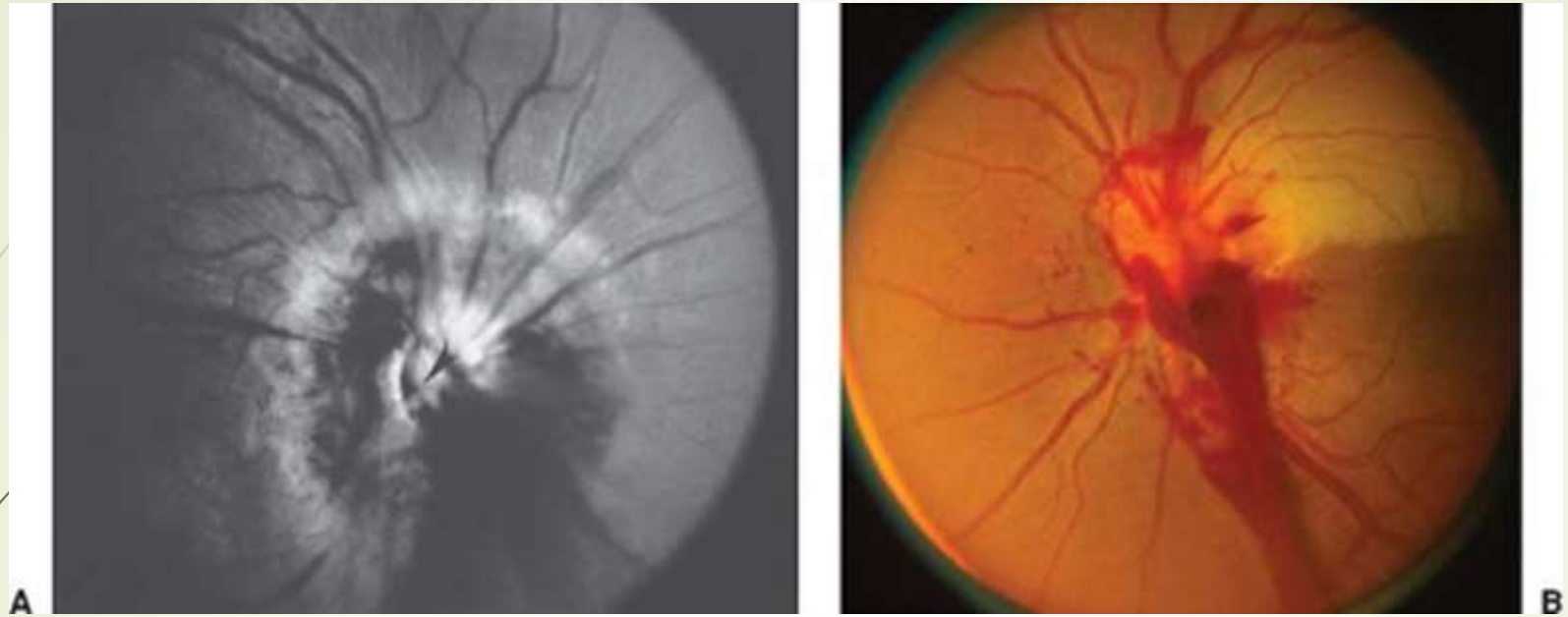
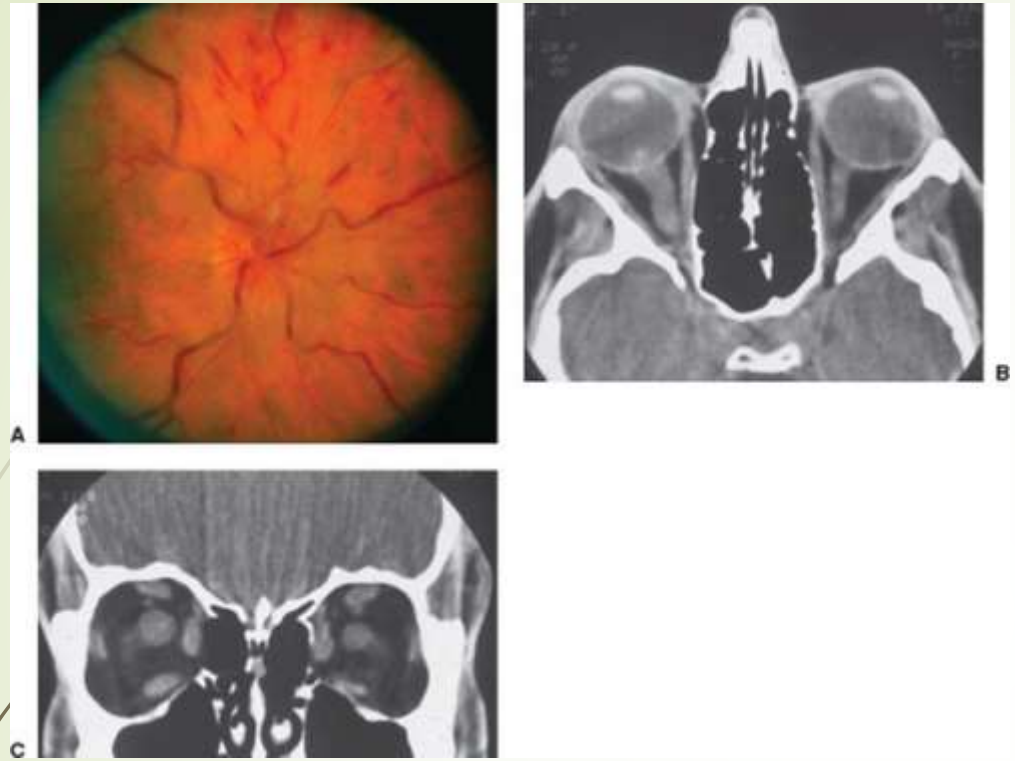
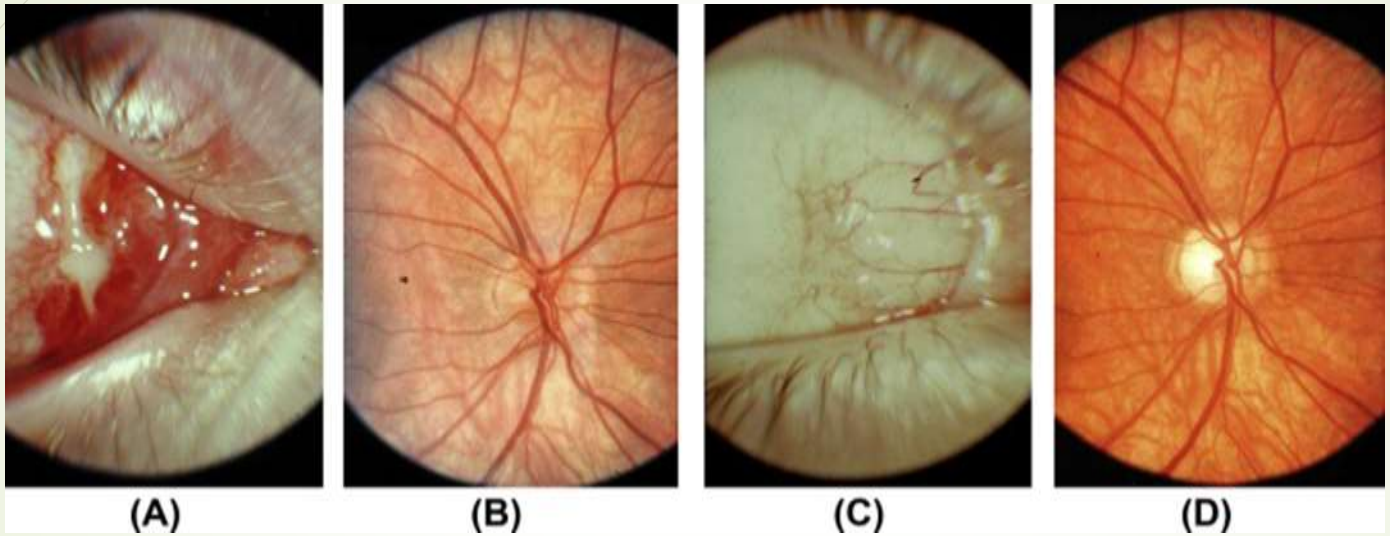



**Figure 1 A:Traumatic avulsion of the optic disc**. Note ring of hemorrhage around the optic disc. The site of avulsion is clearly visible as a crescentic dark area at the temporal portion of the disc (arrowhead).

**B:** Traumatic avulsion of left optic nerve with **superior branch retinal artery occlusion.**




- **Traumatic (proximal) anterior optic neuropathy and central retinal vein occlusion.** The patient was a 24-year-old man who was struck in the left eye while playing basketball and who immediately noted loss of vision in the eye. Visual acuity was light perception OD and 20/15 OS.
- A: **Ophthalmoscopy appearance** of the right ocular fundus reveals moderate hyperemic swelling of the optic disc. The retinal veins are moderately dilated, and there are flame-shaped hemorrhages in the peripapillary region.
- B: **CT scan, axial view,** shows moderate enlargement of the orbital portion of the right optic nerve.
- C: **CT scan, coronal view,** shows enlargement of right optic nerve compared with left nerve. Note small areas of increased density, consistent with hemorrhage, within the enlarged nerve




- Direct traumatic optic neuropathy.
- (A) Entry site of a projectile in the medial canthal region of the right eye.
- (B) The patient's posterior pole was normal when he was assessed shortly after the accident. His visual acuity at that time was no perception of light.
- (C) Conjunctival scar over the entry site.
- (D) Optic disc pallor, more marked temporally, was apparent 6 weeks later. The patient's visual acuity had not improved and he was subsequently lost to follow-up.

- The intracranial portion of the optic nerve in close proximity to the **falciform dural fold** is the next most common site at risk of injury.

- In one report using computerized tomography (CT) imaging, about half of all TON cases were found to have an associated sphenoidal bone fracture, an indirect measure of the significant compressive forces involved at impact.

- However, both **direct and indirect mechanisms** can contribute to optic nerve damage, and a clear distinction is not always possible.

# pathophysiology

- The pathophysiology of indirect TON is likely to be ***multifactorial***, and the concept of primary and secondary injury has been proposed.

- Following trauma, there is an immediate shearing of a proportion of retinal ganglion cell axons, an irreversible process that results in neuronal loss.

- There is then a degree of optic nerve swelling within the tight confines of the optic canal secondary to **direct mechanical trauma and vascular ischemia.**

- The ensuing ***compartment syndrome*** further impairs the already compromised blood supply to surviving retinal ganglion cells, setting up a downward spiral toward apoptotic cell death.

- This two-stage model of TON forms the basis for optic nerve decompression by medical or surgical means, in order to break this vicious cycle and to preserve the remaining retinal ganglion cells that survived the initial insult.

# Epidemiology

- TON is an **uncommon cause** of visual loss following blunt or penetrating head trauma with a reported incidence of 0.7e2.5% in published case series.
- The vast majority of affected patients are **young adult males** (79-85%) in their early 30s.
- The most common causes of TON in this patient group are **motor vehicle and bicycle accidents (49%),** falls (27%), and assaults (13%).

# Clinical assessment

- TON is a clinical diagnosis supported by a history of **direct or indirect trauma** to the head or face.
- The injury can sometimes be trivial, and a careful history of the incident must be elicited from the patient and any other witnesses that might have been present especially when dealing with children or unconscious patients.
- A detailed record should also be kept as cases of TON are not infrequently the subject of future **medico legal proceedings.**

- ***The following features are consistent with a diagnosis of TON:***

- Unilateral or bilateral ocular involvement.

- A relative afferent pupillary defect except in bilateral symmetric cases. A relative afferent pupillary defect is an important clinical sign.

- in patients with mild TON, it can be the only objective evidence of optic nerve dysfunction prior to the development of overt optic atrophy.

- Variable loss of visual acuity ranging from normal to no light perception. Between **40% and 60% of patients** present with severe visual loss of light perception or worse at baseline.

- Impairment of color vision.

- Variable visual field defects.

- The optic disc appearance will depend on the anatomical site and the timing of injury.

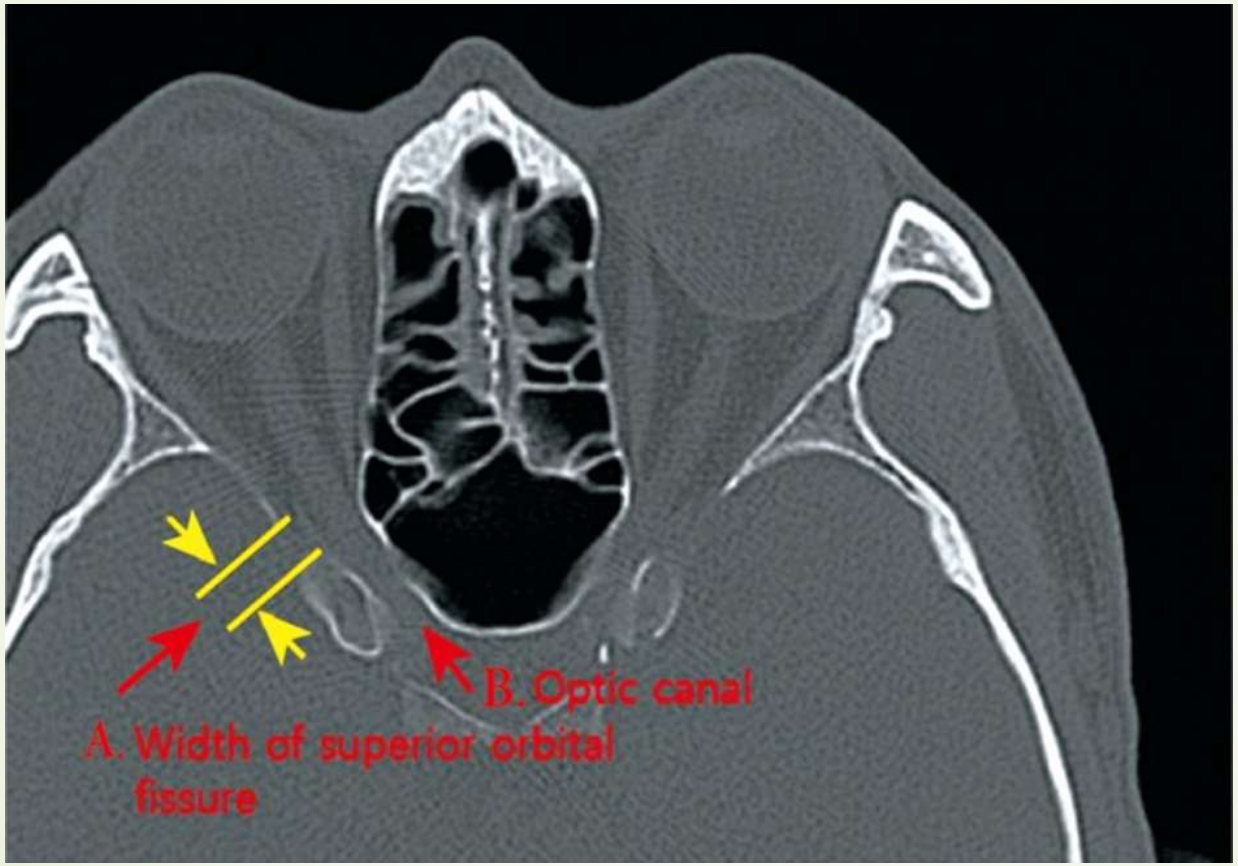
- With injuries to the optic nerve ***anterior*** to the entry point of the central retinal vessels, there is **optic disc swelling** with associated retinal hemorrhages.

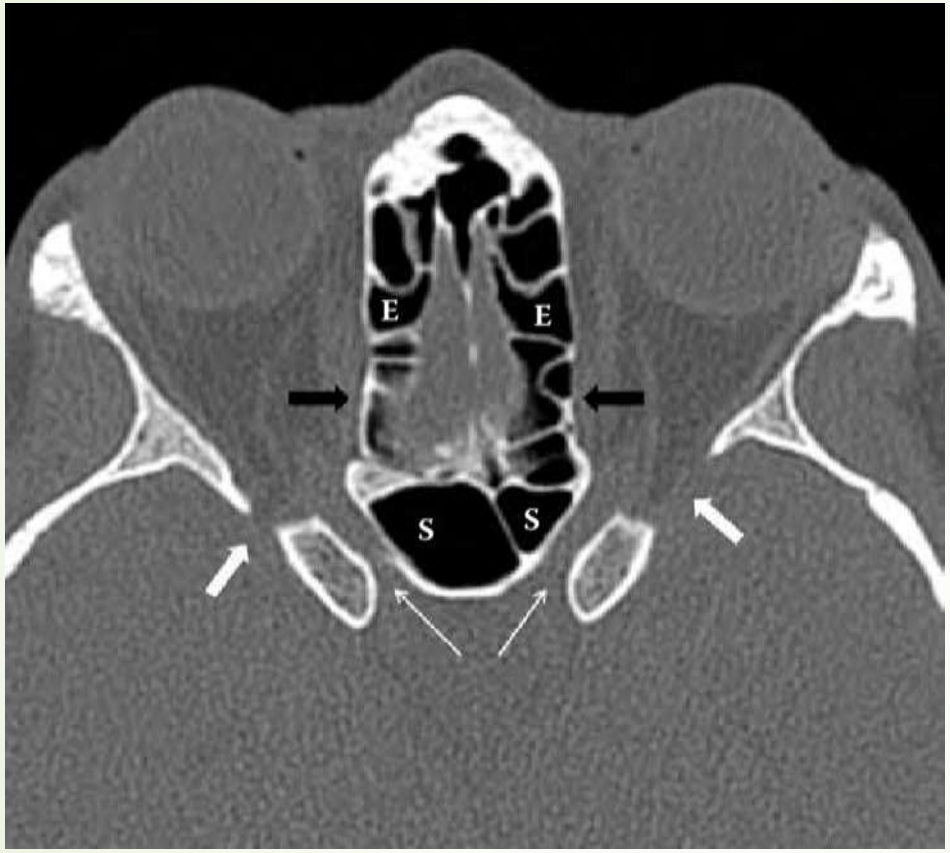
- With more posterior injuries, which are more common, the fundus can look entirely ***normal***.

- Optic disc pallor usually develops ***about 6 weeks*** following the initial injury (Fig. 1)

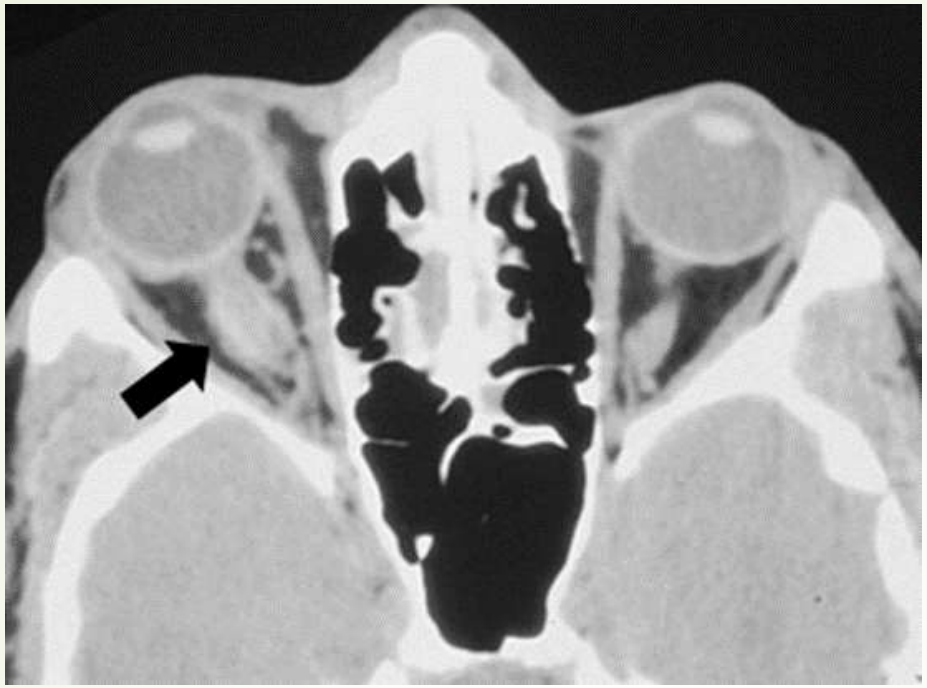
# Neuroimaging

- There is a wide variation in practice worldwide regarding the use of neuroimaging in TON.
- Some clinicians request CT or magnetic resonance imaging or both for all cases,
- whereas others limit these investigations to patients with progressive visual deterioration or when therapeutic interventions are being considered.

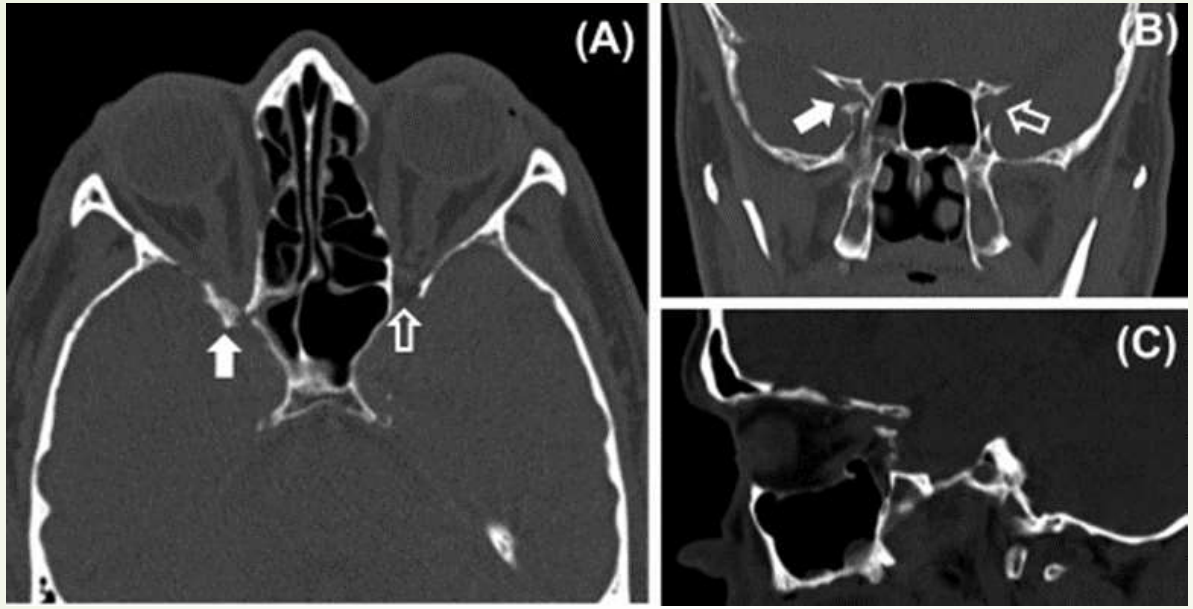

B. Optic canal
A. Width of superior orbital fissure




- **Normal axial computed tomography scan** through the optic canal (arrows); superior orbital fissure (thick white arrows); lamina papricea (thick black arrows); S, sphenoid sinus; E, ethmoid sinuses



Right optic nerve sheath hematoma




**Optic canal fracture and optic nerve compression.**

(A) Axial computerized tomography (CT) scan through the optic nerves showing a **fracture in the posterior part of the lateral wall** of the right orbit. The bone fragment is causing compression of the right optic nerve within the optic canal (white arrow). A normal wide optic canal can be seen on the left side (clear arrow);

(B) Coronal CT scan showing narrowing of the right optic foramen (white arrow) compared with the left side (clear arrow);

- ***CT SCAN*** is the best imaging modality for delineating optic canal fractures and their full extent in preparation for possible surgical intervention (Fig. 4).

- However, the clinical usefulness of universal neuroimaging in TON remains debatable as there is ***no consistent correlation*** between the finding of an optic canal fracture, the severity of visual loss, and the prognosis for visual recovery

# Prognostic factors

- A visual recovery rate of 40-60% has been reported for indirect TON cases managed conservatively, with ***baseline visual acuity*** being the most important predictor of final outcome.

- Other ***poor prognostic factors*** include loss of consciousness, lack of visual recovery after 48 hours, and absence of visual evoked responses.

- The presence of an ***optic canal fracture*** was found to predict a poor visual outcome in some, but not all, case series.

- ***Direct TON*** is a distinct category that results in severe, irreversible visual loss with little likelihood for recovery, and no intervention is of proven benefit.

# Management

Despite persisting uncertainties, the ***main treatment options*** in current use for TON are as follows:

(1) systemic steroids of varying doses, duration, and mode of administration;

(2) surgical decompression of the optic canal.

(3) combination of steroids and surgery.

(4) observation alone (i.e., conservative management).

# Steroids:

- The observed **neuroprotective effect** has been ascribed to the antioxidant properties of steroids and to the inhibition of free radical-induced lipid peroxidation.

- This hypothesis was further reinforced following the clinical introduction of steroids to the treatment of **traumatic spinal cord** injuries.

- The second National Acute Spinal Cord Injury Study**(NASCIS-II)** was a multicenter, randomized, double-blind, placebo controlled trial set up to assess the benefits of megadose steroids in patients with acute spinal cord injury.

- The treatment regimen consisted of an initial bolus dose of 30 mg/kg, followed by an infusion at 5.4 mg/kg/h for a total duration of 23 hours.

- Patients who received steroids within 8 hours of their injury had significantly ***better improvement*** in neurological functions compared to those in the placebo group or those who were treated after 8 hours.

- In the ***third NASCIS (NASCIS-III),*** patients who received steroids hours after their injury experienced greater motor and functional recovery when this regimen was maintained for 48 hours instead of 24 hours.

# International Optic Nerve Trauma Study

- The **International Optic Nerve Trauma Study (IONTS**) is the largest, prospective, multicenter study of TON published to date.

- It was intended to be a randomized controlled trial, but it had to be converted to an observational study after 2 years owing to recruitment failure.

- The majority of patients in ***the steroid group*** had either a mega dose (40%) or very high-dose regimen (18%), and all the participants in the surgical group, except for one, also received steroids.

- Follow-up data were available for 104 cases at 1 month and for 40 cases at 6 months.

- After adjustment for baseline visual acuity, ***no significant differences*** were found between the three treatment groups.

- A three-line increase in visual acuity or more occurred in 57% of the untreated group, 52% of the steroid group, and 32% of the surgery group.
- Interestingly, there was no trend suggesting an increased probability of **visual recovery** with higher doses of steroids or with earlier initiation of treatment.
- Although some case series have reported **higher improvement rates** with steroids, most published figures (44-62%) are comparable with IONTS.
- Crucially, none of these studies have demonstrated any convincing functional visual benefit following treatment with steroids.

# Surgery

- wide range of **intra- and extracranial surgical techniques** have been used to achieve optic nerve decompression in TON.

- Although the favored intervention is largely dictated by the expertise available locally and the surgeon's preference, there has been a shift toward minimally invasive extracranial approaches employing the transethmoidal, endonasal, or sublabial routes.

# Timing of surgery

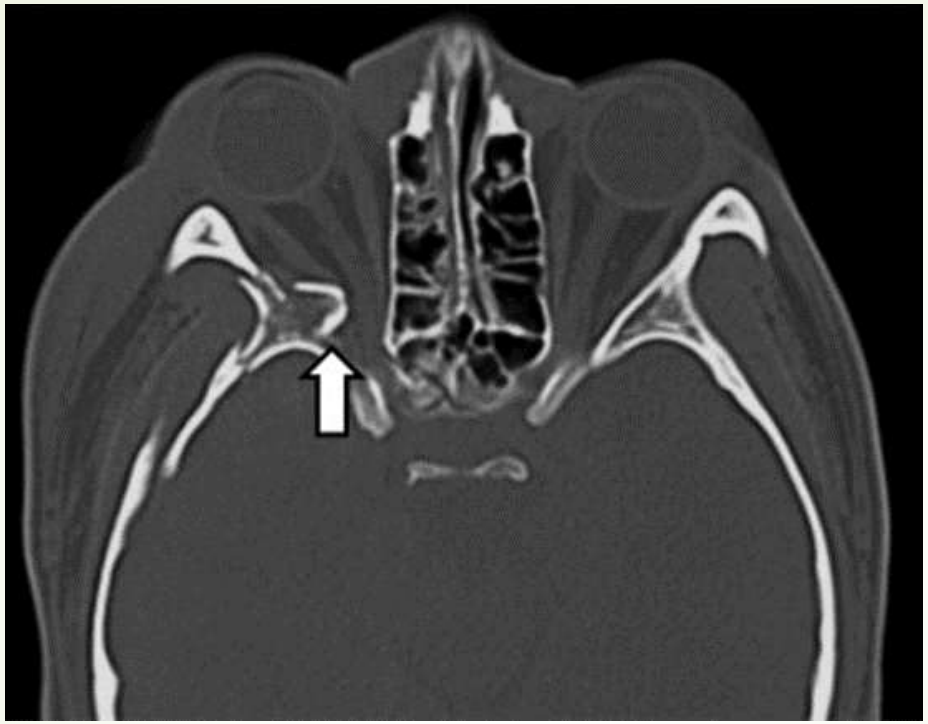
- The **timing of surgery** is a relevant issue in the context of trauma where life-threatening injuries often lead to unavoidable delays before a formal ophthalmological assessment can be carried out.

- Intuitively, **the longer the delay**, the less likely optic canal decompression would be expected to salvage compromised retinal ganglion cells and restore visual function.

# Optic canal fracture

- Some authorities argue that the optic canal should be imaged in all TON cases, and if a **fracture is identified with a bone fragment impinging on the optic nerve** (Fig. 5), prompt surgical intervention should be advocated.

- The counterargument is that some studies have actually identified the presence of an optic canal fracture as a **poor prognostic factor** for visual recovery, irrespective of the treatment modality used.



- Axial computerized tomography scan showing **bone fragments compressing** the right optic nerve in the posterior orbital region.
- **Multiple fractures** of the lateral orbital wall and of the greater wing of the sphenoid can also be noted on the right side.

- This makes biological sense, because a bone fragment is likely to transect a large proportion of retinal ganglion cell axons resulting in immediate irreversible injury and decompressing the optic canal in this situation is **unlikely to restore significant visual function.**

- There are no **randomized controlled trials** on the effectiveness of surgical optic nerve decompression in TON.

- As part of the IONTS cohort, three out of 33 patients (10%) who underwent external surgical decompression suffered **postoperative cerebrospinal fluid leak**, with one patient developing meningitis.

- Another case series reported accidental dural exposure in **5%** of patients who underwent endoscopic optic canal decompression.

- Given the **relatively high rate of spontaneous visual improvement** in indirect TON, the decision to subject a patient to a surgical intervention with potentially serious complications must be even more circumspect.