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Traumatic Optic Neuropathy

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Introduction

Infantile Traumatic Optic Neuropathy (TON) is a condition in which acute injury to the optic nerve from direct or indirect trauma results in vision loss. The severity of optic nerve damage may range from simple contusion to complete avulsion of the optic nerve.

The most common reason for TON is indirect injury to the cranial nerve that is assumed to be the results of transmitted shock from associate orbital impact to the intracanalicular portion of cranial nerve. Direct TON may result from harm or from bony fragments within the optic canal or orbit piercing the cranial nerve. Orbital and cranial nerve sheath hematoma can even cause TON by direct compression

Risk Factors

A CT scan of a patient found to have traumatic optic pathology of the left eye. Note that this patient encompasses a left lupus Fort three fracture with a left superior orbital roof fracture. The medial and inferior wall of the left orbit also is broken.

There are not any glorious risk factors for TON. Within the International cranial nerve Trauma Study, eighty fifth of patients with indirect TON were male and therefore the average age of patients with TON was thirty four. The foremost common mechanisms of injury were automobile accident, bike accident, fall and assault. Whereas abusive injury could be a rare reason for TON, it's a vital concern in infants.

General Pathology

The exact pathology of indirect TON isn't well understood. The cranial nerve meninges is continuous with the orbital tissue layer, feat the cranial nerve liable to transmission of force from blunt head trauma, significantly that touching the superior orbital rim. Indirect TON has been hypothesized to result from cut injury to the intracanalicular portion of cranial nerve, which may cause nerve fiber injury or disturb the blood provide of the cranial nerve. It's additionally been urged that the cranial nerve could swell within the optic canal when trauma leading to accrued phenobarbitone pressure and secondary anaemia injury. Direct TON is plausible to be the results of tissue disruption secondary to foreign body or bony fragments impacting on the cranial nerve.

Diagnosis

The diagnosing of TON is created clinically supported history and ophthalmic signs. Like alternative optic neuropathies, patients with TON could have remittent central sight, remittent visual sense, associate sensory aperture defect and/or field of regard deficits. it's necessary to recollect that albeit rare, TON will be bilateral, thus associate sensory aperture defect might not be seen in patients with bilateral injury and vision loss. The cranial nerve head can seem traditional at the start, however optic atrophy will be seen 3-6 weeks when the initial traumatic event.

Physical examination

The initial external eye test could show signs of orbital trauma or fracture (soft tissue lump, hematoma, step-off on touching of orbital rim). Remittent sight associated an sensory aperture defect (in unilateral cases) also are seen. On funduscopic, the initial cranial nerve head assessment is traditional. Optic atrophy is also seen 3-6 weeks when trauma. Signs

- Decreased Vision
- Decreased visual sense
- Afferent aperture defect
- Visual field deficits

Clinical diagnosing

The clinical diagnosing of TON is created on the idea of a selected constellation of history and physical test findings. Patients have a history of trauma, and complain of or are found to own important visual loss, remittent visual sense, field of regard deficit, associate sensory outgrowth defect, and a expanded complex body part test negative to elucidate these signs.

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