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Multiple cranial nerve palsies with altitudinal hemianopsia following traumatic brain injury in remote area: A case report

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Abstract
Cranial nerve injury (CNI) is a potential complication of traumatic brain injury (TBI). Diagnosis of CNI in TBI can be challenging and there is no consensus on the diagnosis and treatment of CN injury post TBI to date. We report a 49 year-old man who was admitted with a history of motorcycle accident and presented with headache, dizziness, and visual loss. Examination of his right eye showed oculomotor and trochlear nerve palsies with visual impairment and altitudinal hemianopsia. His head CT scan showed multiple cranial fractures. There was no improvement after methylprednisolone and antibiotic administration. Head trauma patients must undergo a routine comprehensive neurological and eye examinations. Further investigations are needed to evaluate the progression and impact of the head trauma that occurred.

Keywords: Altitudinal hemianopsia, case report, multiple nerve cranial palsies, traumatic brain injury

Introduction
Cranial nerve injury (CNI) is a potential complication of traumatic brain injury (TBI) with most patients having a delayed presentation of CNI [1, 2]. In a prospective study by Basheer *et al*., the incidence of CNI in 256 TBI patients was 14.8% [2]. CNI was more common in men, age 30s to 50s, patients with severe head injury, associated base of skull fractures, and facial fractures [2, 3]. Most patients had single nerve palsy with the facial nerve being the most commonly affected cranial nerve (CN) [2, 3, 4]. CN II involvement following head trauma may cause visual loss with devastating consequences [5]. In multiple CN palsy following non-severe head trauma, the most frequent association was between CNs VII and VIII in a study by Coello *et al*. [4] and between CNs III, IV, VI in another study by Pathi *et al*. [6]. Diagnosis of CNI in TBI can be challenging and there is no consensus on the diagnosis and treatment of CN injury post TBI to date [1].

Case Report
A 49-year-old man was admitted to the emergency room with a history of motorcycle accident eleven hours prior admission and presented with headache, dizziness, and visual loss. The patient had no specific past medical history.
On his initial examination, his Glasgow coma scale (GCS) was 15, blood pressure was 142/102 mmHg, heart rate was 64/min, respiratory rate was 20/min, and temperature was 36, 3 degrees Celsius. There were multiple excoriations on his face, stomach, and lower extremities. Other physical examinations showed no abnormalities.
On neurological examinations, his right eye deviated outward in the primary gaze (exotropia). Extraocular movement examination showed oculomotor and trochlear nerve palsies on his right eye, but blepharoptosis could not be evaluated due to the presence of right eyelid hematoma (Fig. 1). The other cranial nerves were normal in the examination. No meningeal signs were present. The results of motor and sensory examinations of the upper and lower limbs were normal. Physiological reflexes were normal and no pathological reflexes were found.

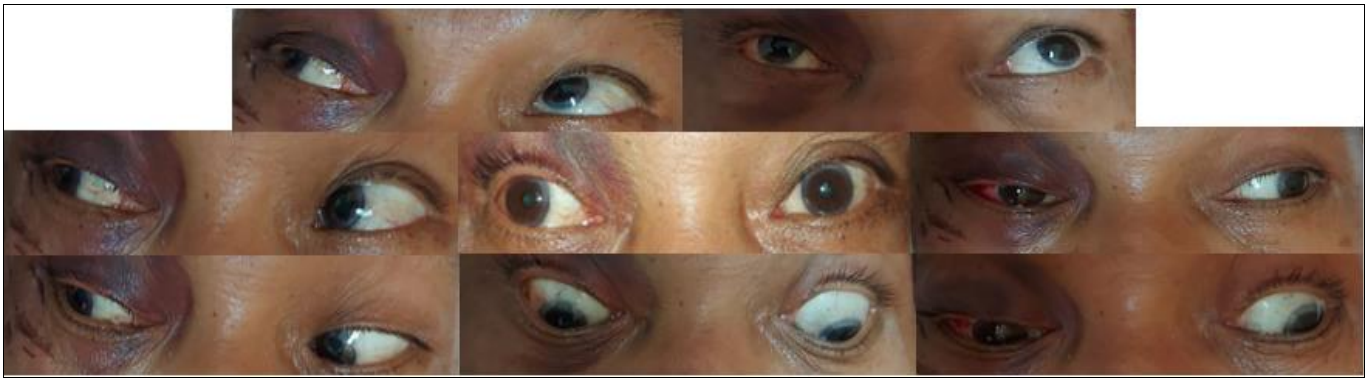

Fig 1: Oculomotor and trochlear nerve palsies.

On ophthalmic examinations, visual acuity of the right eye was counting finger. Anterior segment examination of the right eye showed periorbital swelling with hematoma, subconjunctival bleeding, and the pupil was dilated up to five millimeters with positive relative afferent pupillary defect (RAPD), compared with three millimeters dilation on the unaffected side. Confrontation visual field test showed that the visual field of the right eye was inferior altitudinal hemianopsia. Fundus examination of the right eye appeared normal.

When the patient was admitted, laboratory result showed a high white blood cell count 27.000/µL. Computerized tomography (CT) of the brain revealed minimal pneumocephaly in the right frontal lobe; superoinferior wall and septum of sphenoid sinus fracture; linear fracture of right frontal, parietal, and zygoma; bilateral sphenoid hematosinus; and soft tissue swelling at right temporal and left parietal (Fig 2, Fig 3, Fig 4).

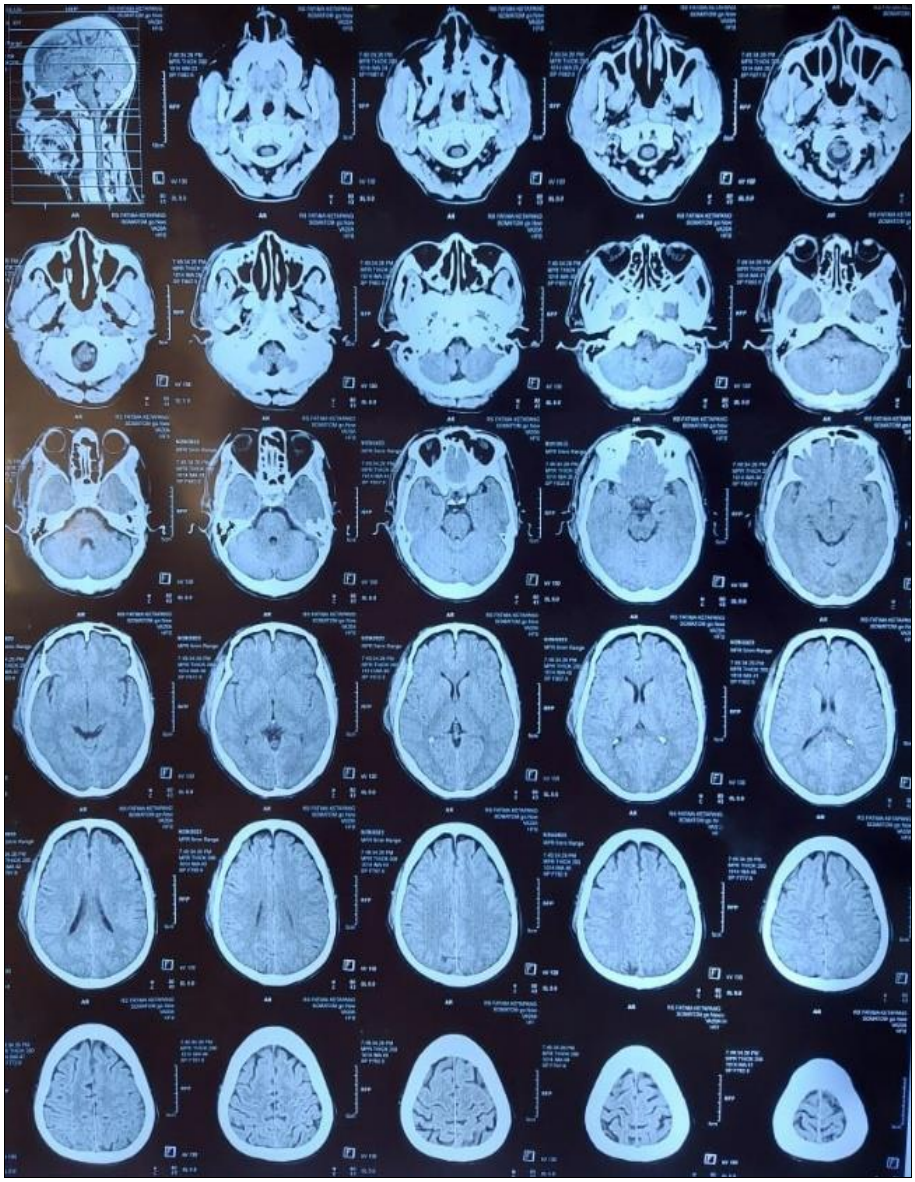

Fig 2: Head CT scan after traum.

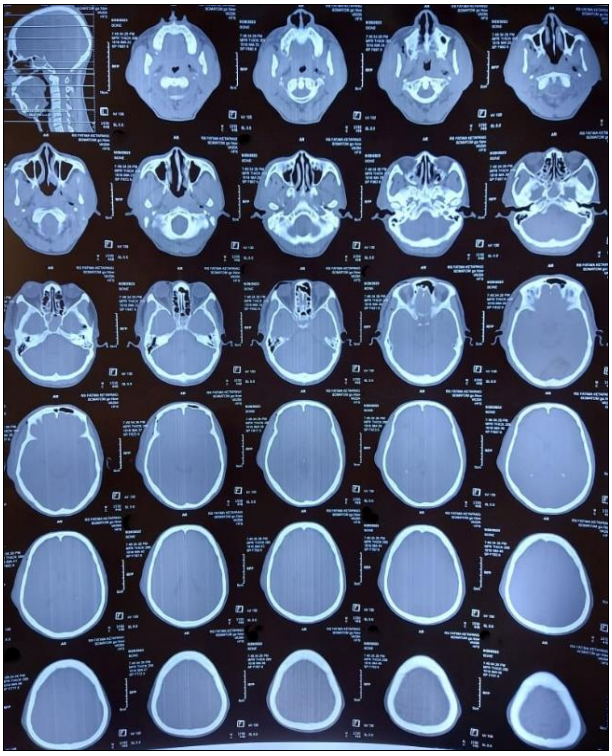

Fig 3: Bone window image of head CT scan after trauma

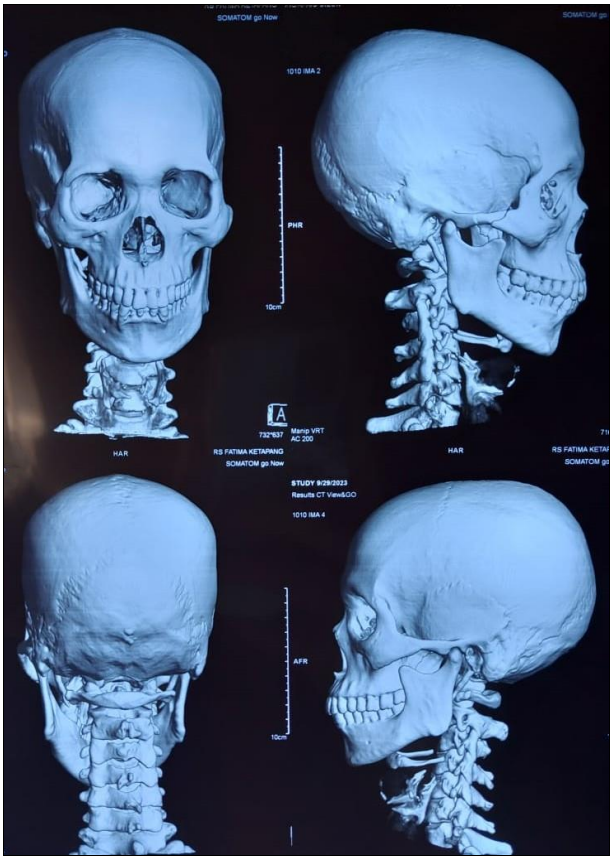

Fig 4: Three dimensional head CT scan after trauma

The patient was treated with intravenous methylprednisolone 1000 mg/day for two days and continued with oral methylprednisolone at a dose of 48 mg/day for three days, intravenous antibiotic, and pain-reliever medication. The patient was also given an eye patch on his right eye. There was no improvement in symptoms on the 5th day of hospitalization.

Discussion

Traumatic optic neuropathy (TON) is defined as any insult of the optic nerve secondary to trauma. According to the mechanism of injury, TON is caused by anatomical disruption to the optic nerve (direct TON) or transmission of forces to the optic nerve from a distant site (indirect TON). There is a two-stage model that explains the pathophysiology of TON such as an immediate shearing of retinal ganglion cell axons that results in neuronal loss and the ensuing compartment syndrome from the optic nerve swelling within the optic canal which impairs the blood supply of the retinal ganglion cells. Presence of a sphenoidal bone fracture is thought to be an indirect measure of the significant compressive forces involved at impact that occurs in about half of TON cases. The optic disc appearance depends on the anatomical site and timing of the injury. In the event of posterior injury relative to the entry point of the central retinal vessels, which is more common, the fundus can look entirely normal and optic disc pallor usually develops around 6 weeks following the initial injury. The main treatment options in TON are systemic steroids of varying doses, duration, and mode of administration; surgical decompression of the optic canal; a combination of steroids and surgery; and observation alone. Using steroid in TON is still controversial. A maximum daily dose of 1 gram intravenous methylprednisolone has been advocated in TON to minimize the risk of neurotoxicity. Recovery of visual impairment in TON depends on various factors with poor prognostic factors such as loss of consciousness, lack of visual recovery after 48 hours, absence of visual evoked responses, optic canal fracture, direct TON, and poor baseline visual acuity [5].

Traumatic injury to the vascular supply to the optic nerve frequently results in an altitudinal hemianopsia that mostly affects the inferior part. The vessels in the arachnoidal membrane which passes to the optic nerve through the subarachnoid space are easily damaged by edema, torsion, or other injuries. The ischemia produced by trauma to these vessels results in a unilateral altitudinal defect having a horizontal border, great density, and steep edges [7].

Oculomotor nerve palsy in adults can be caused by aneurysms, trauma, diabetes mellitus, and neoplasms. Signs of oculomotor nerve palsy include mydriasis, blepharoptosis, and extraocular muscle movement impairment. Lesions in the sphenocavernous region, the orbital apex, and the brain stem may affect the oculomotor nerve through direct injury or indirect compression. Some authors suggest waiting for at least six months before strabismus surgery to further evaluate the cause of oculomotor palsy and allows for possible spontaneous recovery. A case of an 8-year-old with traumatic oculomotor nerve palsy reported by Kook *et al*. showed that the levator and pupillary function almost recovered to the normal range a year after a car accident with early steroid therapy and occlusion therapy [8].

Most acquired trochlear nerve injury (TNI) is caused by trauma which accounts for approximately one third of cases with trochlear nerve palsy. The trochlear nerve is susceptible to injury due to its long intracranial course (75 mm) and thin structure. The rigid tentorium lies next to the trochlear nerve where it can be easily compressed. Most traumatic TNI are associated with midbrain parenchymal contusions and hematomas, as well as injury in the cisterns along the nerve's extra-axial course. Other mechanisms include orbital fractures or frontal or orbital impact causing

a contusion against the trochlear nerve's attachment at the medullary velum. Management of traumatic isolated trochlear nerve palsy includes measures to overcome diplopia such as the use of prisms and eye patch for symptomatic relief. Steroids use in terms of dosage, duration and efficacy for traumatic TNI has not been well studied. There is usually a 6-month period post injury where the patient's condition is observed for any spontaneous recovery before surgical intervention is considered. The trochlear nerve has the highest rate of recovery amongst the ocular motor nerves and at least 50% of patients recover with recovery time varying between one week to six months. Tay and Chua reported a 30-year-old man with isolated trochlear nerve palsy in TBI with improvement of diplopia five months following head injury with conservative management [1].

Presence of multiple cranial fractures presented in our case could be an indirect measure of significant mechanical forces that might cause injury to the blood vessels supplying the cranial nerves. Further investigations such as MRI and Visual Field Analyzer were not available in our hospital. There was no improvement after steroid administration which could be a poor prognostic factor in this case. There is still a possibility of spontaneous recovery in the next few months as in the cases mentioned previously.

Conclusion

Traumatic brain injury may cause multiple cranial nerve palsies. Comprehensive eye examination in head trauma patients is challenging especially in emergency setting. Head trauma patients who are stable and cooperative must undergo a routine comprehensive neurological and eye examinations since CNI may occur with delayed presentation. Further investigations are needed to evaluate the progression and impact of the head trauma that occurred.

Conflict of Interest

Not available

Financial Support

Not available

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