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# "Eyes Tell the Tale". Ophthalmic manifestations of traumatic midbrain contusion in a conscious patient: A case report

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## ABSTRACT

Midbrain contusions are uncommon in civilian trauma and rarely present with isolated cranial nerve deficits. We present a rare case of a conscious 40-year-old male who developed bilateral ptosis, right medial rectus palsy, and left upgaze palsy following a motor vehicle accident. NCCT brain revealed multiple contusions in the midbrain, bilateral frontal, and right parietal lobes. The patient was managed conservatively. The constellation of neuro-ophthalmic signs correlated with damage to the midbrain tegmentum and dorsal structures. This case underlines the importance of careful neuro-ophthalmic examination in head injury and highlights rare presentations of traumatic midbrain injury.

Keywords
ophthalmic,
traumatic midbrain contusion

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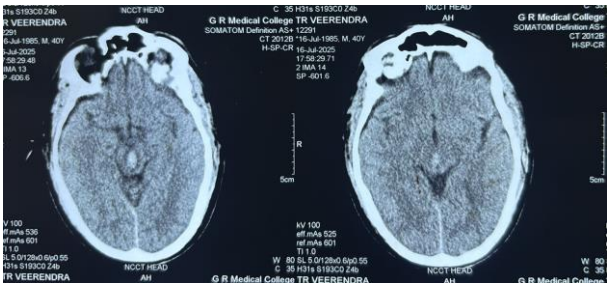
## INTRODUCTION

Traumatic midbrain hemorrhagic contusions are rare, especially when presenting with focal neuro-ophthalmic signs without significant loss of consciousness. Lower motor neuron (LMN) injuries involving the third, fourth, sixth, and seventh cranial nerves are relatively frequent following head trauma, yet they may be overlooked during the initial clinical evaluation.[1,2] The reported incidence of cranial nerve involvement in head injuries ranges from approximately 5% to 23%.[2,3] Such injuries, particularly when occurring in closed head trauma, are often linked to more severe neurological damage.[4] Trauma remains a significant cause of multiple cranial nerve palsies.[5] While isolated cranial nerve injuries are more common, simultaneous bilateral involvement of the third, fourth, or sixth cranial nerves is considered rare.[6]

The midbrain houses critical structures involved in vertical gaze, eyelid elevation, and extraocular movements. Injury to these structures, particularly the dorsal midbrain and oculomotor fascicles, can produce distinctive syndromes such as Parinaud's syndrome, oculomotor palsy, or ptosis. Few cases have been reported in literature where such findings occur in a patient with preserved sensorium.

## Case Presentation

A 40-year-old male was admitted with slurred speech, drooping of both eyelids and diplopia following a road traffic accident (RTA) on July 9, 2025. He was a passenger in a four-wheeler that lost balance and overturned moving at a high speed. The patient immediately lost consciousness and had two episodes of vomiting. There was no history of ENT bleed, seizures or cerebrospinal fluid leak. There was no abdomen, thorax or long bone injury. The vitals, hematological and biochemical parameters were within normal limits. Non contrast computed tomography of patient was performed after a detailed clinical examination. The patient was managed conservatively and was discharged after 7 days with an uneventful intraoperative course.




On Examination (O/E):

- Glasgow Coma Scale (GCS): E4V5M6
- Pupils: Right: Mid-dilated, non-reactive to both direct and consensual light.

  Left: Normal size, sluggishly reactive to both direct and consensual light.
- Cranial Nerve Examination:
- Right medial rectus palsy
- Left upgaze palsy
- Bilateral Grade 3 ptosis
- Motor Examination: No motor weakness in limbs.
- Reflexes and tone: Normal
- Other systemic examination: Unremarkable

Neuroimaging:

- Non-Contrast CT (NCCT) Brain: Revealed hemorrhagic contusions in: Midbrain (central and dorsal areas), Bilateral frontal lobes and Right parietal lobe

## Discussion

The constellation of neuro-ophthalmologic findings in this patient—bilateral ptosis, right medial rectus palsy, and left upgaze palsy—suggests selective involvement of the midbrain tegmentum and dorsal structures, particularly those involved in vertical gaze and oculomotor control.

The oculomotor nerve arises from the oculomotor and Edinger–Westphal nuclei in the midbrain. Its fascicles traverse the tegmentum, red nucleus, and cerebral peduncles. Bilateral ptosis suggests involvement of the central caudal subnucleus, while the right medial rectus palsy and left upgaze palsy indicate fascicular damage and disruption of the dorsal vertical gaze centers (riMLF, interstitial nucleus of Cajal, posterior commissure).

Traumatic midbrain contusions result from high-velocity rotational or deceleration forces. The midbrain's location between the tentorium and clivus makes it susceptible to shearing injuries. In this case, the dorsal midbrain contusions were responsible for the observed signs despite preserved consciousness.

This clinical scenario mimics a variant of Parinaud's syndrome, although classical features like light-near dissociation were absent. Bilateral cranial nerve findings in trauma can lead to diagnostic confusion with other conditions like myasthenia gravis or brainstem infarction, making imaging crucial.

Similar presentations have been reported in literature. Yokota et al. (1991) and Karibe et al. (2001) documented midbrain injuries presenting with isolated oculomotor palsies in conscious patients. Rucker (1958) described traumatic third nerve palsies linked to fascicular damage rather than peripheral disruption. Additionally, studies have shown that dorsal midbrain syndromes are more often due to vascular or neoplastic causes; traumatic causes are rarely reported, making this case particularly noteworthy.

Prognosis in such cases depends on the extent of midbrain damage and associated injuries. While some deficits may resolve, persistent symptoms may

require rehabilitation or surgical correction. This case highlights the need for detailed cranial nerve examination in all head injury patients, regardless of GCS.

## Conclusion

This case highlights a rare constellation of cranial nerve findings following a midbrain contusion due to RTA. Despite a good sensorium, the patient had striking neuro-ophthalmologic deficits, underscoring the importance of thorough cranial nerve evaluation in head injury. Early imaging and recognition of such patterns are vital for accurate diagnosis and prognostication. Head Injury with multiple cranial nerve palsies may not carry a bad prognosis as considered previously and may be reversible with early recognition and active management.

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