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IMAGES IN EMERGENCY MEDICINE

Pediatrics

Adolescent with headache and double vision

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1 | CASE REPORT- TRAUMATIC UNILATERAL INTERNUCLEAR OPHTHALMOPLEGIA

A 14-year-old male presents after falling backwards from the bed of a moving truck of unknown speed. He complains of a posterior headache and double vision. On exam, he is found to have a rightward dysconjugate gaze deviation with rotary nystagmus bilaterally (Figures 1-2). Visual acuity is OD 20/20, OS 20/20. Glasgow Coma Scale is 15. He has an ataxic gait, but the rest of his neurological exam is non-focal. Computed tomography (CT) brain and CT cervical spine are negative. Another study is done that reveals the diagnosis.

FIGURE 1 Leftward conjugate gaze deviation

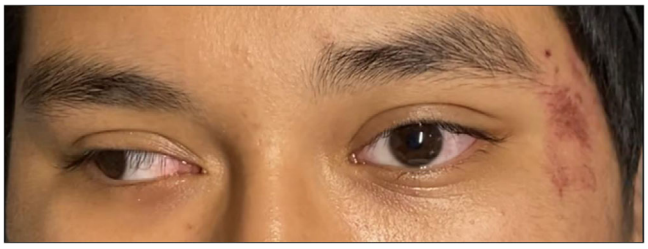

FIGURE 2 Rightward gaze deviation with adduction palsy of the left eye

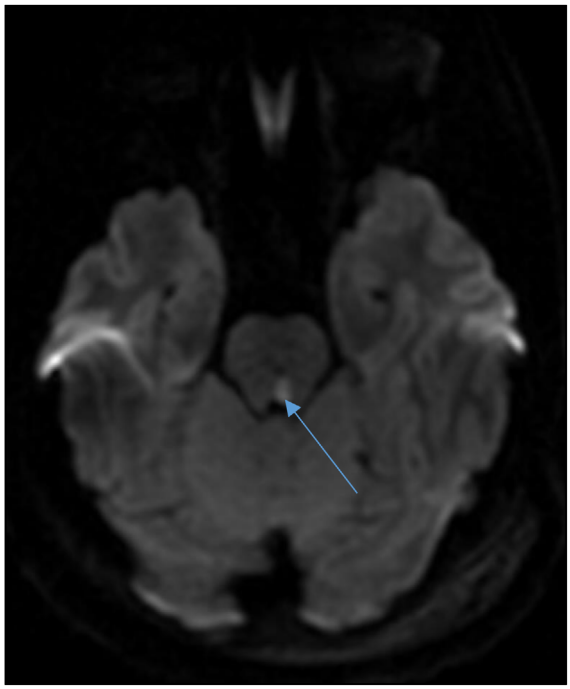

FIGURE 3 Traumatic unilateral internuclear ophthalmoplegia (INO). MRI brain w/wo contrast remarkable for hypointensity in the posterior aspect of pons in the region of the left facial colliculus

Traumatic unilateral internuclear ophthalmoplegia (INO). Magnetic resonance imaging (MRI) of the brain is remarkable for hyperintensity in the posterior aspect of pons in the region of the left facial colliculus suggestive of microhemorrhages in the midline brain and posterior pons (Figure 3). The patient is observed overnight and then discharged as he reported symptomatic improvement.

INO is caused by a disruption of the medial longitudinal fasciculus (MLF), the pathway between the contralateral cranial nerve (CN) VI and ipsilateral CN III nuclei. This causes impaired adduction in the ipsilateral eye with conjugate gaze movement. The most common cause of a traumatic unilateral INO is a small paramedian pontine infarction. The MLF is located in watershed areas that are prone to ischemia and/or hemorrhage as a result of the shearing forces of trauma.[1-3] Although a primary demyelinating process is possible, the temporal relationship to inciting trauma, the unilaterality of presentation, and MRI findings of acute microhemorrhage in the distribution of the MLF suggest a demyelinating process is unlikely. INO presentation in a demyelinating process tends to be bilateral.[4] Reports of traumatic brain injury cases requiring ophthalmology evaluation suggest that only 1.6%–2% of these patients were diagnosed with INO. In multiple case series on traumatic INO patients, 60% had full resolution within 6 months.[5,6] Recovery from INO is variable and dependent on the etiology and the presence of other neurological symptoms. INO with additional symptoms of vertigo, ataxia, dysarthria, facial palsy, and pyramidal tract signs correlate with a worse prognosis.[7-9] Recovery is typically gradual and likely varies (48 hours–2 years) with an individual's neuroplasticity—suggesting that younger patients fare better. Overall functional prognosis is excellent despite persistence of INO.[10] At a 2 week follow-up, the patient is no longer ataxic but reports intermittent dizziness. The patient still has persistence of his INO.

Teaching points:

1. Trauma is a rare but possible cause of INO.
2. INO is a very localizing finding on physical exam and should prompt the need for imaging.
3. MRI is the imaging method of choice to evaluate brainstem involvement in patients presenting with INO.
4. Overall prognosis is excellent; however, it is variable depending on the pathogenesis of presentation, age of the patient, and presence of additional neurological findings.

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