

Bilateral Sixth Nerve Palsy After Head Trauma

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Gaze deficits are not uncommon after head trauma and might be caused by injury to the central nervous system, the peripheral nerve, or the motor unit. Traumatic bilateral sixth cranial nerve palsies are a rare condition and are typically associated with additional intracranial, skull, and cervical spine injuries. We describe a case of a complete bilateral sixth nerve palsy in a 44-year-old male patient with trauma with no intracranial lesion, no associated skull or cervical spine fracture, and no altered level of consciousness. The emergency physician should be aware of the differential diagnosis, initial workup, and injuries associated with a traumatic gaze deficit.

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INTRODUCTION

Sixth cranial nerve palsy is a rare but known sequel of head trauma. Unilateral palsy of the sixth cranial nerve is reported to occur in 1% to 2.7% of all head traumas.^{1,2} Bilateral sixth nerve palsy after head trauma is rare and is highly associated with additional intracranial or cervical spine injury.¹⁻⁴ The differential diagnosis for traumatic lateral gaze palsy includes brain stem lesion, peripheral nerve injury with or without basilar skull fracture, and lateral rectus muscle injury or entrapment.⁵ Associated signs, symptoms, and imaging can differentiate central nervous system, peripheral nerve, and muscular injuries.⁵⁻⁷ The emergency physician should be aware of the differential diagnosis, initial workup, and injuries associated with a traumatic lateral gaze deficit.

CASE REPORT

A 44-year-old man was the belted driver in a single-car motor vehicle crash into a large tree. Emergency medical services found the patient awake and alert, with a Glasgow Coma Scale (GCS) score of 15. Out-of-hospital interventions included spinal stabilization and supplemental oxygen.

On arrival to our trauma room, the patient was awake, alert, and oriented. He complained of shortness of breath and chest pain on the right side. Initial vital signs were blood pressure 118/68 mm Hg, pulse rate 90 beats/min, respiratory rate 28 breaths/min, and oxygen saturation of 98% on a nonrebreather mask. His GCS score was 15. He had a 20-cm full-thickness laceration over the left side of the forehead. There was no evidence of trauma to the midface or orbits. Palpation of the chest revealed deformity and subcutaneous emphysema on the right side. Neurologic examination at the time of initial presentation was nonfocal. No gaze palsy was noted on initial assessment.

A supine anteroposterior chest film did not show pneumothorax but confirmed right subcutaneous air. A right-sided chest tube was placed without complication.

Completion of the secondary survey revealed that the patient was not able to abduct either eye past the midline (Figure 1), which is consistent with a complete bilateral sixth nerve palsy. The patient admitted to diplopia in the lateral gaze. All other extraocular movement was intact. The remainder of the cranial nerve and full neurologic examination remained normal.

Computed tomography (CT) of the head showed no intracranial or extracranial hemorrhage, no mass effect, and no edema. There were normal ventricles and no midline shift. The orbits, sinuses, skull base, and calvarium were normal. Plain films of the neck showed no fracture or dislocation. Neck CT demonstrated a C3-4 bulging disc and a C4-5 herniation without fracture or evidence of soft tissue swelling. A repeat head CT scan with thin cuts through the posterior fossa was obtained later on the day of admission, and results were negative. Magnetic resonance imaging (MRI) of the brain the next day was likewise unremarkable.

Recovery from the trauma was unremarkable; however, the bilateral sixth nerve palsy persisted, and the patient was discharged from the hospital. At 2 months after discharge, the palsy was persistent and complete. At 6 months after discharge, he showed marked improve-

Figure 1.

A, Looking straight; B, looking right; C, looking left.



ment in his ocular mobility and alignment, with some residual limitation of abduction of his right eye more than the left. At 11 months after the trauma, the patient underwent a right medial rectus recession and right lateral rectus resection for residual right esotropia.

DISCUSSION

Traumatic injuries to the brain stem, the peripheral sixth cranial nerve, or the orbits all have the potential to cause a lateral gaze deficit.⁵⁻⁷ Physical examination and appropriate imaging tests can identify the cause of a traumatic gaze palsy.

A traumatic lesion to the brain stem at the sixth nerve nucleus is usually accompanied by a seventh nerve palsy, as well as pyramidal tract signs, because of the close approximation of these structures.^{3,6} A central lesion might also be visualized on CT or MRI.

Injury to the medial orbital wall, causing muscular entrapment of the medial rectus, can simulate a sixth nerve palsy.^{6,7} Signs and symptoms of facial fracture are usually evident on physical examination, and orbital fractures can be seen on CT images.⁶

Isolated loss of lateral gaze with no other cranial nerve signs, no pyramidal tract signs, and no signs of muscular entrapment is thought to result from an injury to the peripheral nerve along its course from the brain stem to the lateral rectus.¹⁻⁷

The course of the sixth cranial nerve from the brain stem to the lateral rectus muscle is long and delicate. The nucleus of the sixth nerve sits in the pons and is closely associated with fibers of the seventh nerve. The nerve exits the pons anterior and medial to the seventh and eighth nerves and ascends vertically through the subarachnoid space. After approximately 15 mm, it penetrates the dura mater and continues to ascend vertically over the ridge of the petrous bone. The nerve reaches the apex of the petrous ridge in 2 to 3 mm and changes direction approximately 120° forward. At this point, it passes under the petroclinoidal (Gruber's) ligament, a thickening of the dura mater between the petrous bone and the posterior clinoidal process. The triangular space defined by the apex of the petrous bone,

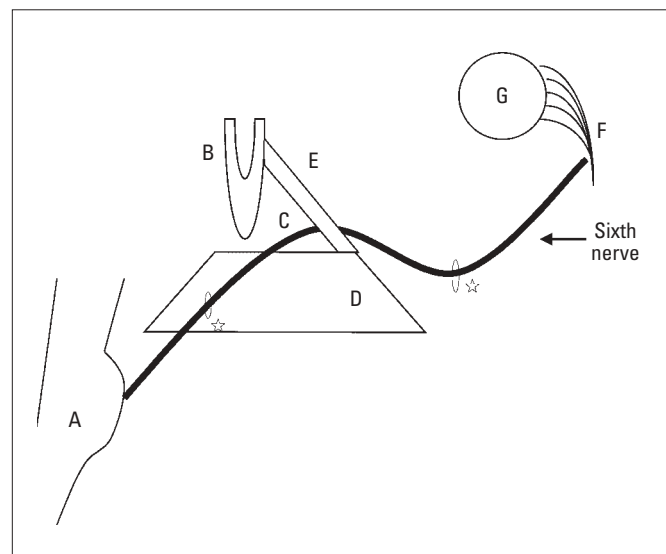
the posterior clinoidal process, and Gruber's ligament is known as Dorello's canal. After passing through Dorello's canal, the nerve passes through the cavernous sinus and the superior orbital fissure and innervates the lateral rectus muscle (Figure 2).^{1-3,5}

Because of their close anatomic association, fracture of the petrous bone often causes injury to the sixth nerve. This injury is well visualized on CT scanning and is often associated with fifth, seventh, and eighth cranial nerve palsies.³

In the absence of petrous fracture, Dorello's canal is the likely site for injury to the peripheral nerve because of its small area and the acute angle change of the nerve as it passes through the canal. The nerve is tethered by the dura before and after the canal. Currently, the most accepted theory proposes that head injury causes downward displacement of the sixth nerve in Dorello's canal, with contusion against the petrous ridge.⁸

Figure 2.

Schematic diagram of the course of the sixth cranial nerve from the pons (A) to the lateral rectus muscle (F). The nerve ascends over the petrous bone (D) and under Gruber's ligament (E) as it passes through Dorello's canal (C). The nerve is tethered by dura before and after the canal (stars). Injury to the peripheral nerve is thought to occur by contusion against the petrous ridge after midfrontal head impact. B, Posterior clinoidal process; G, globe.



Bilateral palsies might occur as a result of direct injury to both nerves independently or indirectly from increased intracranial pressure.^{1,5,7}

Traumatic sixth nerve palsies are highly associated with coma, intracranial injury, skull fracture, and/or cervical spine fracture and are indicative of very severe head trauma.^{1,4,6} In a series of 11 patients with traumatic bilateral sixth nerve palsy, 4 had cervical spine fracture, and 3 had skull fracture. Five of 6 patients in whom the data are complete had some period of coma.¹ In fact, bilateral sixth nerve palsy might be rare simply because the force needed to produce these lesions is generally incompatible with survival.^{1,6} This case is particularly interesting because there was no associated loss of consciousness, no intracranial lesion, no skull fracture, and no cervical spine fracture.

The spontaneous recovery rate of unilateral traumatic sixth nerve palsy has been estimated at anywhere from 12% to 73% at 6 months.^{9,10} Of those patients who will spontaneously recover, the median time to recovery is 90 days.¹⁰ Predictors of nonrecovery include complete, rather than partial, loss of abduction and bilateral palsy.¹¹

Acute management includes occlusion of one eye to alleviate the symptoms of diplopia.¹² Prism lenses can also be used to alleviate diplopia but are unsatisfactory for most patients in the long term because of degradation of image quality and persistent diplopia in certain gaze positions.¹²

Botulinum toxin causes flaccid paralysis by preventing release of acetylcholine at the neuromuscular junction.¹² Injection of botulinum toxin into the antagonist medial rectus muscle might eliminate its unopposed action and allow for single vision during the recovery period and might also prevent contracture of the unopposed medial rectus.¹³ Left untreated, contracture might prevent normal vision, even after return of function of the lateral rectus.¹²⁻¹⁴ In children specifically, botulinum toxin might allow binocularity and prevent amblyopia. In those patients who will not experience spontaneous recovery, botulinum toxin treatment might allow for a simpler surgical procedure.¹⁴

Surgery is currently indicated in those who have not recovered within 6 months to 1 year after injury. Several different eye muscle resections and transpositions are used to treat chronic sixth nerve palsy. The success rate of these surgeries ranges from 30% to 80%.^{7,12}

Our patient likely had independent bilateral contusions of the peripheral sixth nerve against the petrous ridge in Dorello's canal. Other possible causes include brain stem lesion, petrous bone fracture with sixth nerve injury, increased intracranial pressure, and orbital fracture with lateral rectus entrapment. Brain stem lesion was unlikely because there was no seventh nerve palsy and no pyramidal tract findings. CT and MRI confirmed no brain stem lesion. In addition, there was no petrous ridge fracture seen on CT images. There were no clinical signs or results of CT or MRI consistent with increased intracranial pressure. There were no clinical signs of medial rectus entrapment or orbital trauma, and no orbital fracture was seen on CT images.

Traumatic sixth nerve palsies are commonly associated with additional intracranial or cervical spine pathology. Our patient did not have loss of consciousness by history, and imaging ruled out intracranial lesion, skull fracture, and cervical spine fracture.

In the patient with traumatic lateral gaze deficit, all possible causes and associated injuries must be considered. Special consideration must be given to the neurologic examination, with particular attention to other cranial nerve palsies and pyramidal tract signs. The orbits should be carefully inspected and palpated for signs of trauma and evidence of entrapment. Head CT images should be obtained, with special attention to the pons, the petrous bone, and the orbits, as well as for associated intracranial injury. New gaze palsy in the patient with head trauma should alert the clinician to the possibility of associated cervical spine injury, and images of the cervical spine should routinely be obtained. The presence of a bilateral palsy is thought to indicate a severe trauma to the head and neck, and the appropriate high index of suspicion for associated injuries must be maintained. Referral to ophthalmology

is recommended, with close observation for 6 to 12 months.

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