Pontine gaze paresis due to traumatic craniocervical hyperextension

Report of two cases

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Pontine gaze paresis is frequently due to tumor of the brain stem. Occasionally it may be caused by inflammation or ischemia. Two cases are reported, each with pontine gaze paresis and other signs of lower brain-stem injury, basal skull fractures, and second cervical vertebra fractures. This pattern of injuries is believed to be the result of craniocervical hyperextension with stretch injury to the brain stem at the junction of the medulla and pons.

KEY WORDS • pontine gaze paresis • craniocervical hyperextension • brain-stem injury

CLINICAL correlations are rare in cases of trauma to the lower brain stem because of the high frequency of immediate fatality. However, there has been extensive pathoanatomical correlation of closed head injury and craniocervical hyperextension, with brain-stem damage. We have recently seen two patients with brain-stem injuries associated with traumatic hyperextension of the head; both patients had striking clinical signs of lower brain-stem dysfunction, including pontine gaze pareses. Because we are not aware of any previous reports of traumatic pontine gaze pareses and because of the rarity of clinical reports of lower brain-stem injury with survival, we are reporting these two cases.

Case Reports

Case 1

This 20-year-old woman was seen in our intensive care unit after transfer by air from another hospital 300 miles away. She had been on a road trip and was a passenger in a truck when it collided with a car. She sustained a basal skull fracture, with cerebrospinal fluid (CSF) leak, and a C-2 vertebral fracture without displacement. She was not unconscious for any significant period, but initially she did require ventilator support.

When first evaluated by us, approximately 2 weeks after the collision, she was awake and alert, but had some difficulty in speaking. She had abrasions and lacerations of the skin of the forehead. She had undergone a tracheostomy. She moved her extremities, but only with great effort, and had reduced strength, especially in the lower extremities. She could turn her head to the right, but not to the left. She had a complete left facial paresis, which appeared to be peripheral, but she had some involuntary, intermittent contractions of the right side of the face. The eye movements were noteworthy: there was no ptosis and the pupillary reactions were normal, but there was ocular bobbing, with the eyes intermittently driven down and floating up. She had a complete left pontine gaze paresis, with inability to move either eye to the left of center. There was a very prominent skew deviation, with the left eye somewhat lower than the right. Funduscoppy, visual acuity, and vertical eye movements were normal. No visual field defect was appreciated. Corneal sensations were normal. There was a reduction of hearing in the left ear. Movements of the tongue were normal. The palate was in a normal position and moved normally. There was some loss of function of the left sternocleidomastoid muscle. The patient’s respiration was very irregular with prolonged periods of apnea. She was wide awake and quite able to breathe on command. She did not require ventilator
support at this point. Blood was noted behind the left tympanic membrane. The C-2 vertebra was fractured but not displaced. A basal skull fracture was noted with an extension through the left petrous pyramid. Computerized tomography (CT) scanning with contrast was entirely normal, with good visualization of the posterior fossa structures. She was placed in traction for her vertebral fracture.

Over a period of several weeks, the patient recovered from the left pontine gaze paresis, but retained a skew deviation. The respiratory pattern became normal. Movement of the left side of the face was restored after several months, with aberrant regeneration, so that the orbicularis muscle contracted in a striking fashion with movement of the mouth. After she was out of traction, it was quite apparent that she had a striking cerebellar syndrome. There was gross finger-to-nose movement abnormality, with dysmetria. She regained good strength in the arms and legs, but had gross ataxia and required a walker for ambulation.

Case 2

This 22-year-old woman was a passenger in a car when it struck a roadside structure, and she was thrown free. There was no definite period of loss of consciousness. The patient was admitted to the hospital with evidence of head trauma and cervical injury. When first seen by us, she was in traction because of a C-2 vertebral fracture without displacement. She had multiple facial abrasions and lacerations. She was able to move all extremities and was quite awake. The eye movements were the most striking neurological findings. On attempted left gaze, she had a very rapid jerking of the left eye, but very little lateral motion; the right eye did not move laterally at all. On right gaze, each eye moved symmetrically, but only 10° to the right of midline. Vertical eye movements were normal and there was no vertical nystagmus at this point. Pupillary movements were normal, and there was no ptosis. Corneal sensations were normal. The patient had a very subtle left facial weakness, more prominently involving the upper face. She had no gag reflex, but the other cranial nerves appeared normal. There was no deficit in respiratory drive and no evidence of long tract corticospinal tract defects or of cerebellar damage. Fundi were normal. Visual fields were essentially normal. Cold water caloric tests produced only a vertical jerking movement with irrigation of either ear.

The patient was kept in traction because of her cervical fracture. Over a period of 2 weeks, her gaze pareses resolved to a fast jerking nystagmus, with a fast component inward on lateral gaze to either side. She developed a fast up-beating, fine irregular nystagmus on up gaze. There was no nystagmus on down gaze. Vertical eye movements were otherwise normal and visual acuity was normal. The pupillary movements remained normal. The prominent management problem was a complete absence of gag reflex and inability to swallow. There was no dysphonia. Otolaryngological examination confirmed the swallowing defect and demonstrated normal vocal cord movement. Taste sensation was normal.

Discussion

Lesions of the paramedian pontine reticular formation at the level of the abducens nuclei result in ipsilateral paralysis of lateral gaze. An extensive lesion of the reticular formation at this level may also interrupt spontaneous breathing. The respiratory center consists of a diffusely arranged group of reticular cells extending from the rostral part of the medulla to the level of the obex. Thus, the first patient (Case 1) had a lesion of the reticular formation at the level of the pontomedullary junction involving the left lateral gaze center and the respiratory center. The skew deviation with left hypotropia indicates a lesion of the caudal pons on the left side. Ocular bobbing indicates extensive damage of the lower brain stem — it is quite rare for a patient with this ocular sign to survive. Her bilateral cerebellar syndrome was probably caused by injury to the cerebellar peduncles. She had a corticospinal tract deficit bilaterally, due to damage to the pyramids. To summarize, we believe that the brain-stem injury in this patient was an extensive lesion at the pontomedullary junction, involving the pyramids, reticular formation, and cerebellar peduncles. In addition, she had a defect in the seventh and eleventh cranial nerves on the left side, probably due to peripheral nerve injuries.

The second patient (Case 2) represented a much less extensive injury, with symmetrical involvement of the paramedian pontine reticular formation, causing bilateral pontine gaze pareses. In addition, another slightly more caudal lesion was implicated by her selective swallowing defect. A defect in gag reflex, with inability to swallow but normal taste sensation and no dysphonia, can only be explained by bilateral lesions of the caudal portion of the nucleus ambiguus.

As in Case 1, she had a basal skull fracture and a C-2 vertebral fracture.

We believe that four basic mechanisms warrant consideration as the cause of the injuries in these patients. Primary ischemia has been invoked as a cause of brain-stem injury, associated with trauma. We reject this mechanism in these patients because we do not believe that the pattern of neurological deficit fits into any known vascular distribution in the vertebral basilar system. A second mechanism which can result in injury to the medulla is herniation of the cerebellar tonsils, associated with a shift in intracranial contents. We have excluded this mechanism because at no time in the course of either of these patients was there any evidence of an increase in intracranial pressure, and CT scans demonstrated no evidence of a shift in intracranial contents or hematoma. The posterior fossa structures were well
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visualized in each patient and appeared to be normal. Furthermore, we do not believe that a lesion in the pons causing gaze pareses could be caused by herniation of the cerebellar tonsils. The third mechanism to be considered is primary focal impact damage to the brain stem, associated with blunt head trauma. Mitchell and Adams, and Strich have demonstrated that brain-stem injury involving the cerebellar peduncles and other structures surrounding the fourth ventricle can occur in closed-head trauma in the absence of any elevation of intracranial pressure or shift of intracranial contents. However, they pointed out that focal injury to the lower brain stem is always associated with significant injuries elsewhere in the brain, as part of the picture of diffuse brain damage due to closed-head injury. This contrasts with the clinical findings, where, in our patients, neurological injury was limited to the lower brain stem.

Lindenberg and Freytag reported a study of 21 cases of fatal closed-head injury, in which there was clinical evidence of sudden hyperextension. In these patients, the lesions were tears and hemorrhages in the region of the pyramids at the junction of the medulla andpons, with hemorrhages extending dorsally and rostrally into the tegmentum of the medulla and pons. Lesions of the face, forehead, and neck were present in most of these cases, suggesting severe hyperextension injury. Eleven of the 21 cases had basal skull fractures, and eight had fractures and/or dislocation of the C-1 or C-2 vertebrae. It should be noted that all 21 patients were dead on arrival at the hospital. Lindenberg and Freytag pointed out that the pia and adjacent subarachnoid tissues are fused into a tough sleeve around the caudal medulla and adjacent spinal cord, so that stretching forces of hyperextension are transmitted to the rostral medulla adjacent to the rather immobile pons.

Gilbert and Deonna reported a case of an 8-year-old child who suffered head trauma upon being thrown from an automobile, and developed a "locked-in" syndrome from which she recovered. She required respirator support for 3 weeks, and could not swallow for 3 months. It is suggested, although not specifically stated, that she had bilateral pontine gaze pareses, which cleared later. The authors postulated that the child's brain-stem injury resulted from craniocevical hyperextension because of lacerations of the forehead and a hematoma of the neck. Britt, et al., reported a similar case in 1977, with autopsy findings. Their patient was a young man who was involved in an automobile accident and sustained a locked-in syndrome. He suffered bilateral sixth nerve pareses, was quadraparetic, and was unable to swallow or speak. His respirations were normal. After 14 weeks of support, he died due to tracheal hemorrhage from erosion of an innominate artery. The postmortem findings showed cavitary necrosis of the ventral pontomedullary junction, and bilateral disruption of the sixth cranial nerves. There were mild gliotic changes in the pontine and medullary tegmentum. It was concluded that his injury was caused by severe hyperextension of the head.

Our patients both had pontine gaze paresis and other signs of lower brain-stem injury, basal skull fracture, and fracture of the C-2 vertebra. The presence of "hangman's fracture" in each of these patients is very strong evidence of hyperextension of the head. We believe that traumatic hyperextension of the head causing tears and hemorrhages at the junction of the medulla and pons, as described by Lindenberg and Freytag, is the mechanism of brain-stem injury in these patients. We are not aware of any previous report of pontine gaze paresis due to trauma.

References


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