

Case Report

Tonic Downward and Inward Ocular Deviation Ipsilateral to Pontine Tegmental Hemorrhage

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Key Words

Pontine hemorrhage · Eye movements

Abstract

A 61-year-old man presented with coma and left hemiparesis. He was found to have tonic downward and inward deviation of the right eye, and a right lateral gaze palsy. He also had occasional downward bobbing movements of the right eye, and a partial bilateral upgaze paresis. CT showed a right pontine tegmental hemorrhage extending to the ipsilateral midbrain. Tonic ocular downward and inward deviation associated with pontine tegmental hemorrhage may be due to irritation of mesencephalic downgaze and convergence centers by rostral extension of the hematoma. Although 'eyes seeming to peer at the tip of the nose' is characteristic of thalamic hemorrhage, it may also be seen secondary to pontine tegmental hemorrhage.

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His eyes were dysconjugate with the right eye tonically deviated down and inwards (fig. 2). There were occasional downward bobbing movements of the right eye. With the oculo-cephalic maneuver, outward movement and downward movement of the left eye were seen but the right eye moved minimally. On cold caloric testing of the right ear, both eyes moved to the right but neither eye moved past the midline. Cold caloric testing in the left ear elicited leftward deviation of both eyes, with further downward movement of the right eye. The right pupil was 2 mm and the left 3 mm, both reacted to light. Two weeks later, the patient responded to commands. He had a right lower motor neuron facial palsy, a left hemiparesis involving the arm more than the leg, and left-sided sensory loss to all modalities. His right eye remained slightly deviated inward and downward. He had a right lateral gaze palsy and reduced upgaze bilaterally (to both saccades and smooth pursuit), but left lateral gaze and bilateral downgaze were normal. Optokinetic nystagmus could not be elicited in either vertical or horizontal directions.

Discussion

Inward and downward ocular deviation ('seeming to peer at the tip of the nose') is characteristic of a thalamic hemorrhage [1, 2] and has only been described once previously in association with a pontine hemorrhage [3]. In Caplan and Goodwin's [3] series of lateral tegmental pontine hemorrhages, case 1 had a tonic downward and inward ocular deviation with ipsilateral ocular bobbing similar to our patient. Our patient and Caplan and Goodwin's patient establishes that lateral tegmental pontine hemorrhage may produce similar ocular findings to thalamic hemorrhage.

Case Report

A 61-year-old African-American man was found unresponsive. His blood pressure was 250/120 mm Hg and his Glasgow Coma Scale Score was 8. He had a right facial weakness and a left hemiparesis and required intubation for airway protection. A CT scan of the head showed a predominantly right-sided pontine tegmental hemorrhage, which extended up to the ponto-mesencephalic junction (fig. 1).

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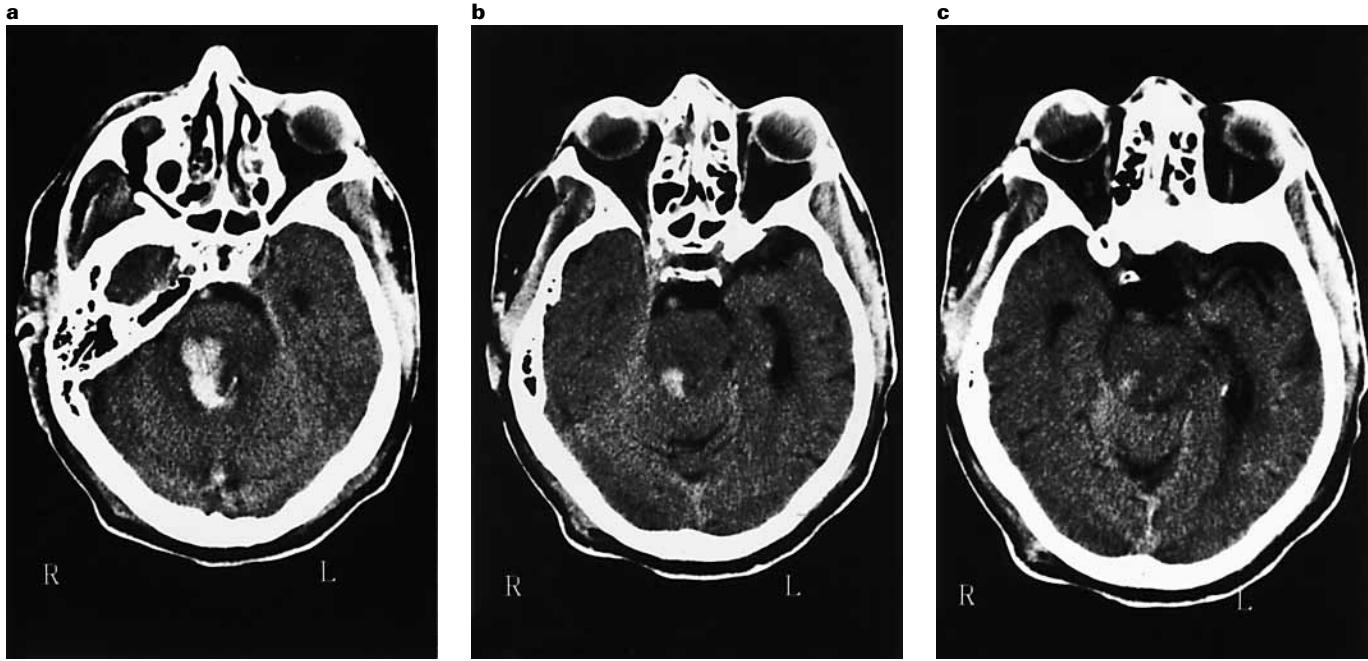


Fig. 1. CT scan showing hemorrhage in the right pontine tegmentum (**a, b**), extending up to the right ponto-mesencephalic junction (**c**).



Fig. 2. Photograph showing the downward and inward deviation of the right eye.

In thalamic hemorrhage, the downward component of the ocular deviation is thought to be due to extension of the hemorrhage into or compression of the midbrain. This is thought to cause irritation of structures concerned with downgaze or an imbalance due to an acute upgaze palsy [4]. The inward component of the ocular deviation in thalamic hemorrhage may be due to an irritation of the convergence neurons in the midbrain [5]. In Caplan and Goodwin's case, the pontine tegmental hemorrhage was found at autopsy to extend up to the midbrain, very similar to the extent of the hemorrhage on CT in our patient. This suggests that midbrain involvement may be im-

portant in the production of the ocular deviation seen in Caplan and Goodwin's and our patient, as well. In pontine tegmental hemorrhage, blood tracking up into the rostral midbrain may produce these eye findings, by a mechanism similar to that causing the ocular deviation associated with thalamic hemorrhage. However, the fact that the ocular deviation associated with pontine hemorrhage is unilateral and the lack of obvious involvement of the rostral midbrain in our case suggests that the mechanism may be different.

Vertical saccades are generated by burst neurons in the rostral interstitial nucleus of the medial longitudinal fasci-

culus (riMLF). Those responsible for downgaze project directly to the ipsilateral inferior rectus subnucleus and trochlear nucleus, while those for upgaze project through the posterior commissure to the contralateral oculomotor nucleus [6]. A unilateral downward deviation could be caused by irritation of downgaze neurons in the midbrain which are unilateral, and lie relatively caudally compared to the upgaze neurons [6, 7]. This would make them more liable to involvement by a hemorrhage extending up from the pons. It seems unlikely that the downward deviation of the eye in this patient is the ipsiversive hypotropia secondary to an ocular tilt reaction (OTR), as the other features of an OTR were not present and ipsiversive hypotropia is seen in medullary and lower pontine lesions not in ponto-mesencephalic lesions [8].

Inward deviation is more difficult to explain as the convergence pathways have not been clearly mapped out. The exact location of the convergence center is unclear, but convergence and divergence neurons are found in the midbrain reticular formation, and the convergence neurons project to the ipsilateral oculomotor nucleus [6]. The esotropia seen in pontine hemorrhage may be due to stimulation of these convergence pathways. The pathways of the divergence neurons is less well understood, but injury to them could also result in convergence. Divergence paralysis however is usually bilateral and does not pro-

duce convergence of the severity seen in our patient. Bogousslavsky and Regli [9] reported 2 patients with small pontine tegmental hemorrhages who had involuntary ocular convergence induced by lateral or downward voluntary gaze. They thought that the convergence seen was a central synkinetic phenomenon which can be unmasked secondary to injury to the pontine gaze centers if brainstem association pathways are intact. Our patient had presumed injury to the right pontine gaze center, but unlike these authors' case, the convergence of the right eye was not associated with an attempted voluntary eye movement. It is possible however that the inward deviation seen in our patient was the result of an unmasking of a direct cortico-mesencephalic mechanism as suggested in Bogousslavsky and Regli's case.

The incomplete upgaze palsy seen in our patient could be due to the pontine tegmental hematoma. There is experimental [10] and clinical [11-13] evidence that pontine lesions may cause impaired vertical eye movements. Burst neurons responsible for vertical saccades are found in the riMLF, in the rostral midbrain. Inputs to this nucleus include projections from the paramedian pontine reticular formation. Also, projections from the vestibular nuclei to the midbrain, ascending in the medial longitudinal fasciculus, are necessary for vertical smooth pursuit.

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