

CASE REPORTS

Unilateral oculomotor nerve palsy, ataxia and Parinaud's syndrome caused by ventral midbrain hemorrhage

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Abstract

We report a patient with unilateral midbrain hemorrhage which caused ipsilateral complete oculomotor nerve palsy with pupillary involvement, contralateral upgaze paresis, contralateral limb ataxia and Parinaud's syndrome. CT scan and MRI brain demonstrated a hemorrhage in the left paramedian midbrain probably involving the oculomotor fascicles; extension of the hemorrhage to the most rostral midbrain may have involved the pupillary fibers. It was previously thought that a lesion in the superior colliculus, surrounding nuclei (Darkschewitsch and Cajal nuclei), and the posterior commissure (i.e. dorsal midbrain) were responsible for clinical findings similar to those found in our patient, but our patient showed a hemorrhagic lesion in the left ventral midbrain which did not extend to dorsal midbrain. We propose that the responsible lesion in our patient might involve the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF).

INTRODUCTION

Brainstem hemorrhage accounts for less than 5% of cerebral hemorrhages.¹ The vast majority of brainstem hemorrhages are pontine hemorrhages; extremely few occur in the midbrain. There are many different locations of midbrain hemorrhage, producing interesting symptoms in many cases. We report the case of a patient with unilateral midbrain hemorrhage which caused ipsilateral oculomotor nerve palsy, contralateral upgaze paresis, ipsilateral limb ataxia and Parinaud's syndrome.

CASE REPORT

The patient was a 70-year-old right-handed man with a history of hypertension. He had no prior history of neurological problems. On November 30, 2008, when he left a concert hall, he had sudden onset of nausea and diplopia and was transferred to our hospital.

On admission, he was alert and cooperative. Visual fields were normal in both eyes on confrontation testing and bilateral simultaneous stimulation. His optic fundi were normal. His

pupils were round and unequal (right 2.5 mm, left 5.0 mm), and sluggish to both direct and indirect light reflex on the left side due to the left oculomotor nerve palsy. He showed partial ptosis and was unable to converge. On primary gaze, his left eye was mildly deviated downward and outward. There was upward gaze limitation both eyes. There were no ptosis and abnormality in the light reflex and movement on the right eye except for upward gaze limitation. The remaining cranial nerves were normal. No motor paralysis was present, and tendon reflexes were normal. Sensation was intact. There was ataxia of the right upper and lower limbs and trunk. On walking, he tended to veer to the right. He could not walk without assistance.

Brain CT scan showed a small hemorrhage in the left paramedian midbrain probably involving the oculomotor fascicles. Magnetic resonance imaging demonstrated a high intensity lesion in the tegmentum of the left ventral midbrain (Figure 1).

The ataxia of the right upper and lower limbs and trunk resolved within 2 weeks. He could walk without any assistance and was discharged home after 2 weeks. The disturbance of ocular

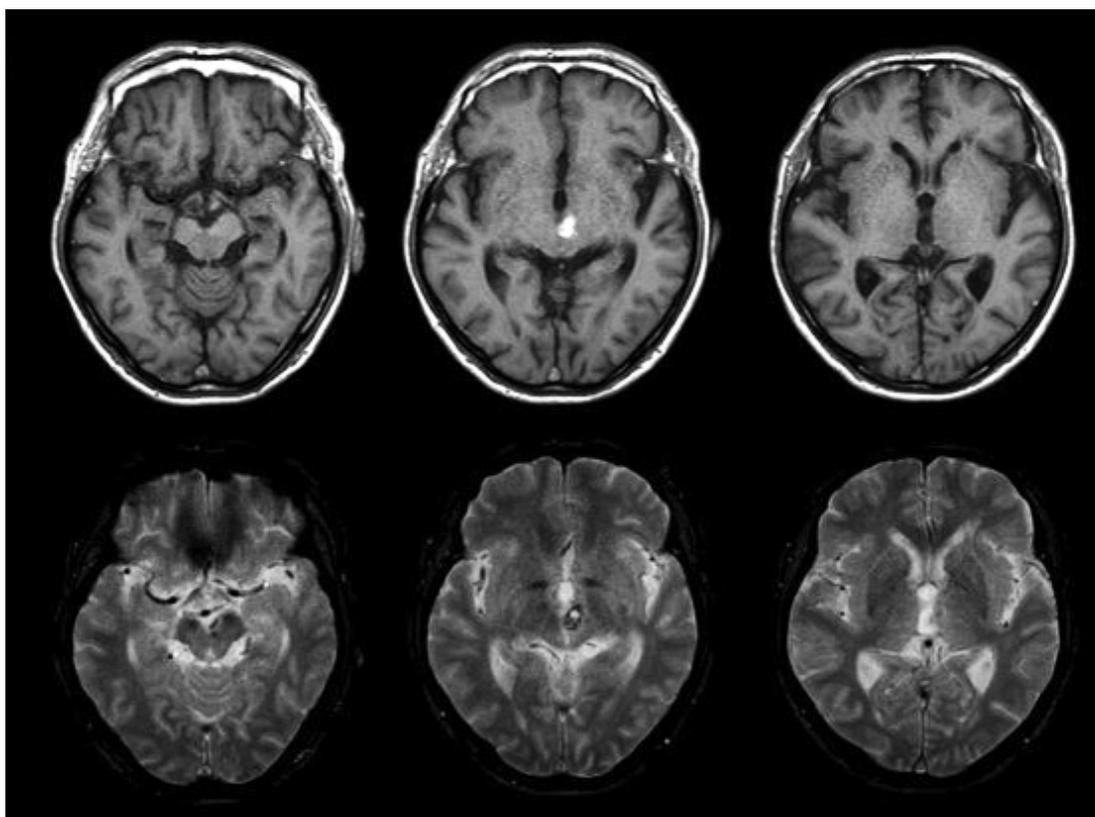


Figure 1. Magnetic resonance imaging demonstrated high intensity lesion in the tegmentum of the left midbrain.

movement gradually improved, but he still had diplopia 3 months after the onset of illness.

DISCUSSION

Cerebrovascular disease in the midbrain simultaneously damages the oculomotor nerve nucleus and other neurofibers. It gives rise to a wide range of neurological symptoms, many of which are already well-known.²⁻⁴ In addition to unilateral oculomotor nerve palsy and contralateral limb ataxia, our patient exhibited bilateral upward gaze palsy and convergence palsy. Claude's syndrome would be suspected if the only symptoms were oculomotor nerve palsy on the side of the lesion and contralateral limb ataxia, and it would be inferred that damage was to fibers from the oculomotor nerve nucleus and the cerebellothalamic connection fibers passing through the red nucleus. However, the presence of contralateral oculomotor disturbance indicates the possibility of Nothnagel's syndrome or Claude's syndrome, with Parinaud's syndrome. The classical Nothnagel's syndrome comprises ipsilateral oculomotor palsy with contra-

ipsilateral ataxia. Pierrot-Deseilligny *et al.*⁵ reported contralateral upward gaze palsy with ipsilateral oculomotor palsy, and Hino *et al.*⁶ referred to the possibility of Nothnagel's syndrome with contralateral upward gaze palsy. However, since classical Nothnagel's syndrome is caused by bilateral lesions extending from the superior colliculus to the inferior colliculus in the midbrain, as stated by Liu *et al.*³, it would seem that this is different from the foci of this patient.

Our patient exhibited Parinaud's syndrome from a unilateral lesion. The neurological mechanism of Parinaud's syndrome has yet to be elucidated. It was previously thought that the superior colliculus, surrounding nuclei (Darkschewitsch and Cajal nuclei), and the posterior commissure were responsible for the syndrome. However, in a monkey study in which the superior colliculus was destroyed, oculomotor disturbance was not permanent.⁷ Rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF) has also been implicated in vertebral gaze.⁸

This patient exhibited hemorrhagic lesions in the left ventral midbrain, which did not extend to the dorsal midbrain. However, brain edema

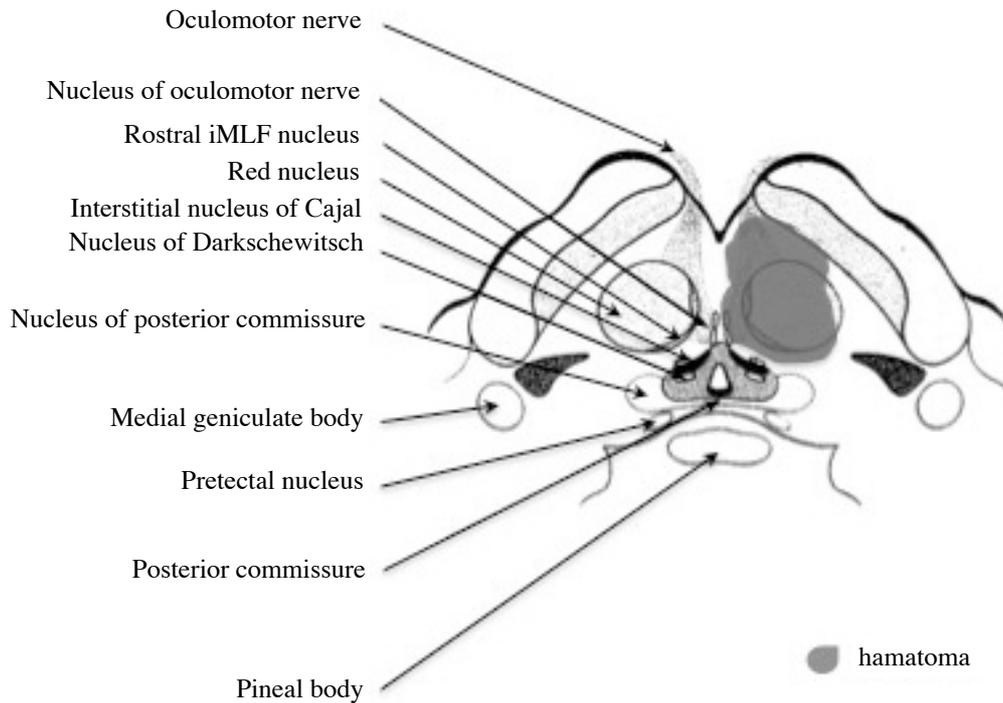


Figure 2. Anatomic localization of the hematoma.

could affect riMLF temporarily. We believe that his Parinaud's syndrome was a result of damage to the left oculomotor nerve nucleus, red nucleus, and riMLF (Figure 2), from the hemorrhage in the ventral midbrain.

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