

Hemimedullary Syndrome with Ipsilateral Sensorimotor Deficits

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Dear Sir,

Hemimedullary syndrome has been introduced as Babinski-Nageotte syndrome or Reinhold syndrome with ischemic lesion in unilateral lateral and medial medulla oblongata [1–3]. The identity of hemimedullary stroke has remained controversial because both medial and lateral medullary infarctions rarely occur simultaneously and vascular supplies of the medial and lateral medulla usually differ [4, 5]. Rarely, the lower medullary lesion caused the symptoms of ipsilateral hemiparesis with sensory loss [6, 7]. Here we describe a patient with hemimedullary syndrome in conjunction with ipsilateral hemiparesis and sensory loss caused by ipsilateral, vertebral artery dissection. The ipsilateral motor weakness was evaluated by diffusion tensor imaging (DTI) and transcranial magnetic stimulation (TMS) study.

Case Report

A 50-year-old man with a 5-year history of hypertension and diabetes was admitted to our hospital on March 26, 2005, because of vertigo with nausea, vomiting and right-sided weakness. His symptoms had begun 2 days prior to admission. However, he did not experience headache, neck pain, or visual symptoms.

On admission, he complained of right-sided weakness, right facial palsy, dysarthria, and dysphagia. His blood pressure was 167/104 mm Hg, pulse rate was regular at 78/min and body temperature was 36.7°C. Physical examination showed no abnormality in the chest or abdomen. His mental state was alert. Neurologic examination demonstrated right upper, motor neuron-type facial palsy with dysarthria, dysphagia, and paralysis of the right tongue and palate. His right eye had left beating and upbeat nystagmus in horizontal and vertical planes, and he also showed right-sided head tilt, ocular tilt, lateropulsion and hypometric saccade to the right. In addition, he showed right hemiparesis (arm: MRC grade IV, leg: III), right-sided hyperalgesia and decreased perception of vibration sense, as well as increased deep tendon reflex in the right arm and leg. His right limbs were ataxic and he could not stand without assistance.

Brain magnetic resonance imaging with angiography was performed. Diffusion-weighted imaging revealed a hyperintense lesion in the right dorsolateral medulla oblongata and FLAIR imaging showed a hyperintense lesion in the right medial and lateral medulla oblongata with extension of the lesion in the cervicomedullary junction

(fig. 1A–C). Magnetic resonance angiography did not delineate the right vertebral artery and luminal widening of the left vertebral artery, which suggested dissection of the vertebral artery (fig. 1D). These neuro-radiological studies did not document any parenchymal or vascular lesions in the anterior circulation. Blood test showed no specific abnormality in the thrombotic profile. Echocardiography with contrast echo study showed a 0.13-cm-sized, patent foramen ovale with right-to-left shunt. We evaluated the pyramidal motor pathway by DTI and TMS study. DTI showed a decreased mean fractional anisotropy value in the right lower medullary region (right, 0.54 ± 0.08 ; left, 0.74 ± 0.09) as well as a decreased density of the right-sided pyramidal motor fibers (fig. 1E). TMS study suggested delayed conduction velocity in the right postdecussational corticospinal pathway (table 1).

Anticoagulation therapy had been discontinued because of massive duodenal ulcer bleeding. One month after admission, neurologic examination showed mild neurological deficits, which included right monoparesis in the leg (MRC grade IV), right facial palsy with tongue deviation to the right, and right-sided limb ataxia. Sensory deficits in his right side and the degree of nystagmus were markedly diminished.

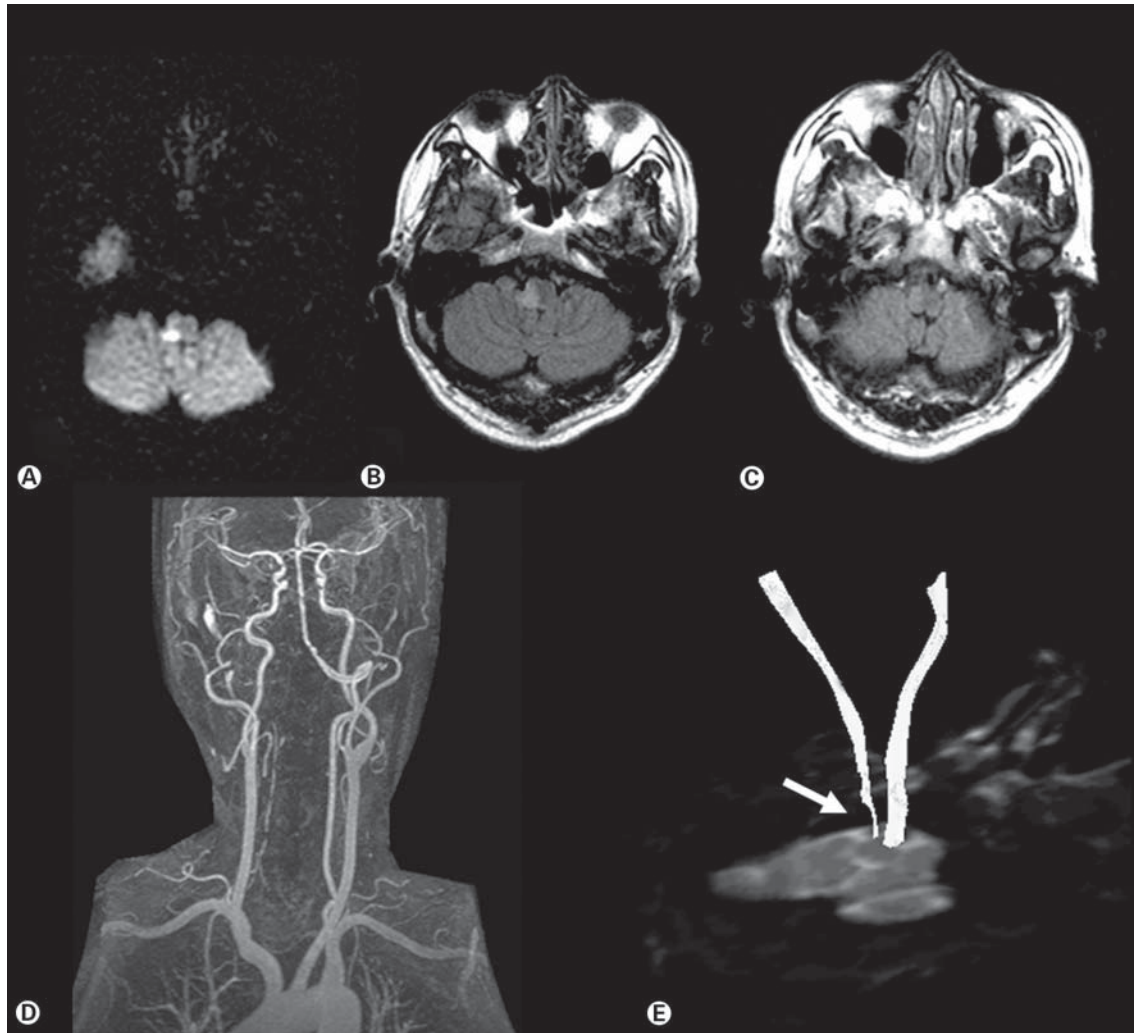


Fig. 1. Brain magnetic resonance imaging and angiography performed 3 days after symptom onset. **A** Diffusion-weighted imaging shows a hyperintense lesion in the right dorsolateral medulla. **B** FLAIR magnetic resonance imaging demonstrates hyperintensity in the right medial and lateral medullary oblongata. **C** High signal intensity in the right hemimedullary area extended to the cervicomedullary junction in FLAIR image. **D** Magnetic resonance angiography shows multiple narrowings in the course of the right vertebral artery and luminal widening of the right vertebral artery. **E** Diffusion tensor tractography shows decreased fiber density in the right pyramidal tract at the medullary level (fractional anisotropy value: right, 0.54 ± 0.08 ; left, 0.74 ± 0.09).

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Discussion

This is the first case report of hemimedullary infarction with ipsilateral hemiparesis documented by anatomical and functional studies. Our patient presented right hemiparesis due to right medial medullary involvement, which was an ipsilateral, postdecussational, pyramidal tract lesion. The condition was documented by decreased fiber density in the right postdecussated motor pathway on DTI study and delayed

cortical motor conduction latency recorded from the tibialis anterior muscle in the right postdecussated portion on TMS study [8]. The more profound leg weakness could be explained by the lateral involvement of the pyramidal tract at the medullary level on 3-dimensional tractography. These anatomical and functional studies revealed right medial medullary involvement, and our patient also showed right paralysis of the tongue and palate, as well as decreased

vibratory sense. In addition to clinical findings of medial medullary involvement, lateral medullary involvement was also present, as suggested by the patient's ocular findings, hyperalgesia to pinprick, hiccup, and right-sided ataxia. Grossly, he showed right-sided head tilt, and ophthalmoscopic examination revealed right upward deviation (17°) of the right eye visual axis. The nystagmus was composed of left beating and upbeat in vertical and horizontal planes,

Table 1. Results of transcranial magnetic stimulation

| Evoked site | Recording site | | | | | |
|--------------------|--------------------------------|-----------------|-------|-------------------------------|-----------------|-------|
| | right tibialis anterior muscle | | | left tibialis anterior muscle | | |
| | latency ms | amplitude mV | CML-F | latency ms | amplitude mV | CML-F |
| Right motor cortex | 38.0 | 0.20 | 16.9 | 30.8 | 1.98 | 7.8 |
| Left motor cortex | 39.0 | 0.25 | 14.6 | 35.3 | 0.15 | 11.2 |

The right-sided, postdecussated portion showed delayed conduction with increased CML-F compared with the left tibialis anterior recording site.

CML-F = cortical latency – (M latency + F latency – 1)/2; normal range <13.4.

along with the presence of right lateropulsion and ipsilateral hypometric saccade, which was consistent with lateral medullary involvement [9]. The upbeat nystagmus might be explained by dorsomedial extension of the ischemic lesion to the nucleus intercalatus [10]. Ipsilateral sensory symptoms in the limb and body are an uncommon but distinct variant caused by caudal lesions extending dorsomedially that probably involve the ipsilateral dorsal column or decussating lemniscal fibers [11]. Our patient showed ipsilateral sensory deficits caused by lesion extended dorsomedially in the cervicomedullary junction. Also, magnetic resonance angiography showed multiple tubular stenoses with adjacent fusiform dilatation of the right vertebral artery and segmental widening with septum in the left vertebral artery, which suggested arterial dissection of both vertebral arteries [12]. Unlike previously reported cases of hemimedullary syndrome like Babinski-Nageotte syndrome or Reinhold syndrome, our patient showed ip-

ilateral sensorimotor deficits with medial and lateral medullary lesion extended to the cervicomedullary junction, which was caused by extensive ischemic insult due to vertebral artery dissection.

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