

Magnetic Resonance Imaging of Medial Medullary Infarction

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Medial medullary infarction is characterized by ipsilateral hypoglossal nerve palsy, contralateral hemiparesis sparing the face, and contralateral disturbance of deep sensation. Although it is possible to make a clinical diagnosis in typical patients, diagnosis is difficult if hypoglossal nerve palsy is absent. We describe a patient with medial medullary infarction without hypoglossal nerve palsy. The patient suffered from left hemiplegia and homolateral disturbance of deep sensation. Magnetic resonance imaging revealed the site of the lesion to be in the medial portion of the upper medulla oblongata. The result of somatosensory evoked potential testing was compatible with disturbance of the medullary medial lemniscus. In a review of the literature, we examined the relation between clinical features and lesion location in 16 patients with medial medullary infarction and compared these to the present patient. Motor paresis was present in every patient, while disturbance of deep sensation was recorded in nine of 13 patients and hypoglossal nerve palsy in six of 14 patients. In atypical patients with medial medullary infarction (such as the present patient), magnetic resonance imaging is necessary to detect the lesion and to make a clinical diagnosis. (*Stroke* 1990;21:963–966)

In 1908 Spiller¹ described the clinical and pathologic features of medial medullary infarction, and in 1914 Déjérine² used the term “syndromes bulbaires antérieur ou interolivaire” to describe the triad of contralateral hemiplegia sparing the face, disturbance of deep sensation, and ipsilateral hypoglossal nerve palsy. We describe a patient with medial medullary infarction in whom hypoglossal nerve palsy was absent. Magnetic resonance imaging (MRI) revealed the site of infarction. We also discuss 16 patients from the literature who had medial medullary infarction.

Case Report

While walking home, a 67-year-old woman experienced an odd sensation in which the sky appeared to turn pink for a few moments. She also noticed a tingling sensation in her left upper limb. She arrived home several minutes later and noted that her left upper and lower limbs had become weak. The weakness became progressively worse until she was no longer able to move her left limbs at all; at this point she was transported to a hospital emergency department. On admission, she could not move her left

limbs and was unaware of their position. X-ray computed tomography of the head did not detect any abnormalities. Four months later, she was admitted to our hospital.

On examination she could protrude her tongue straight in the midline, and neither atrophy nor fasciculation were observed on the tongue. All of the other cranial nerves were normal on examination. Manual muscle testing showed that the strength in her left limbs was reduced (3/5 to 4/5); facial muscle strength, however, was normal. The muscle stretch reflexes were exaggerated and the muscle tonus was increased in her left limbs. Babinski's and Chaddock's signs were negative. Vibratory sensation was disturbed on the left radial condyle and great toe; however, thermal and tactile sensations were intact. There was no disturbance of coordination. The patient had no history of hypertension, smoking, or diabetes.

Results of routine laboratory blood examinations, including hematocrit, lipid analysis, uric acid concentration, and glucose tolerance, were normal. X-ray computed tomography of the head was normal. MRI of the brainstem with a 5-mm slice revealed an area of high signal intensity on T2-weighted images and an area of low signal intensity on T1-weighted images in the right medial medulla (Figures 1 and 2) with no other abnormal findings in the cerebrum, brainstem, or cerebellum. The infarct was located in the upper third of the medulla oblongata. Needle electromyog-

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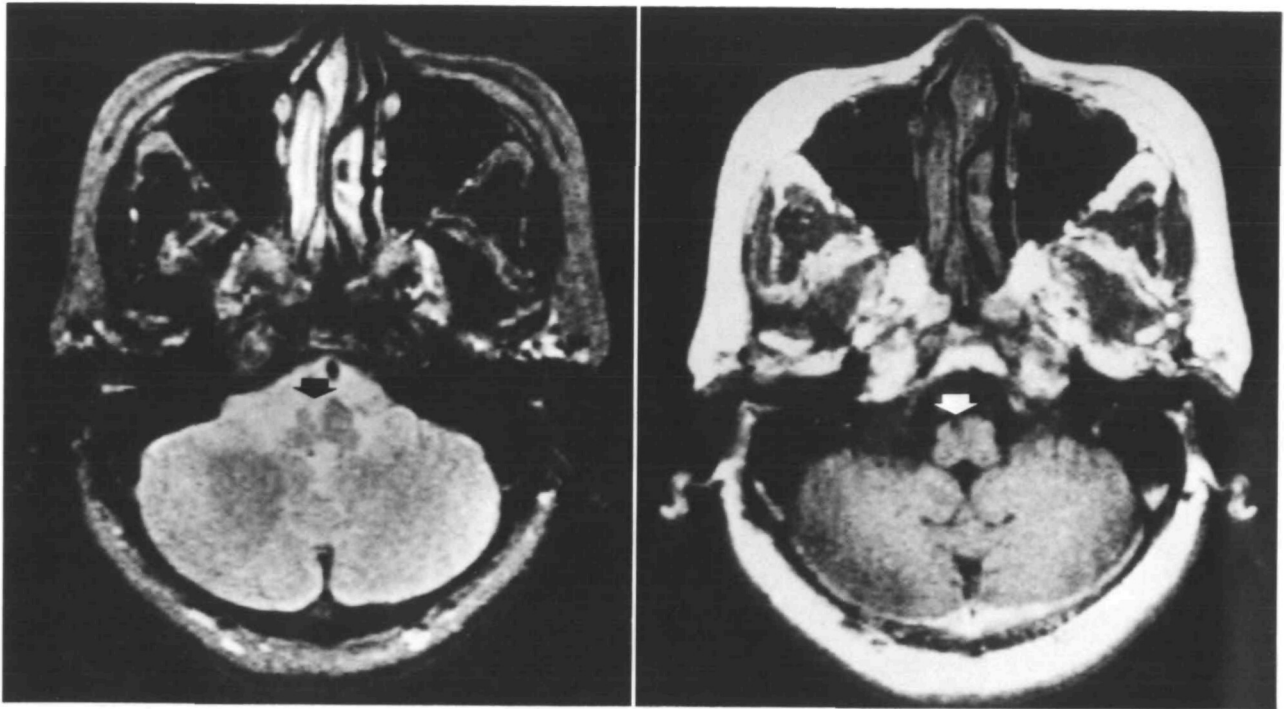
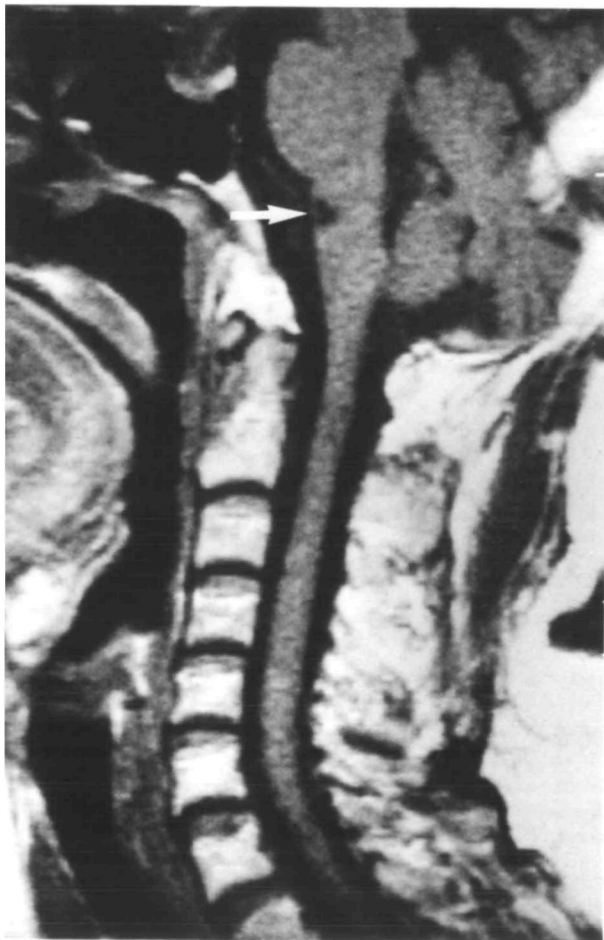


FIGURE 1. Magnetic resonance images of brainstem. Left: Resonance time (TR) 2,700 msec; echo time (TE) 80 msec; T2-weighted image. Right: TR 520 msec; TE 25 msec; T1-weighted image. Transaxial sections of upper medulla showing wedge-shaped infarct in right medial ventral portion (arrows).



raphy of the tongue showed no denervation. Digital subtraction angiography showed hypoperfusion of the right vertebral artery without focal stenosis or occlusion (Figure 3). Recordings of somatosensory evoked potentials revealed that the amplitudes of P₁₃₋₁₄ and N₁₅ were markedly decreased on stimulation of the left side (Figure 4), which was consistent with a disturbance of the medial lemniscus.

After several months the patient's spastic gait improved slightly, and she was able to walk without supports.

Discussion

Medial medullary infarction is very rare. In one series of 700 patients with cerebrovascular disorders, only four could be classified as having medial medullary infarction.³ In a review of the relevant literature, we found descriptions of 16 patients with medial medullary infarction³⁻¹⁵ and studied the clinical manifestations and sites of infarction in each patient. Every patient had motor paresis; disturbance of deep sensation was observed in eight of 12 patients; and hypoglossal nerve palsy was recorded in six of 13 patients. Only four of 11 patients had all three clinical features. We conclude that it is not easy to diagnose medial medullary infarction from clinical manifestations. The prognosis was poor in 12 of 15 patients. In the three patients with good prognosis,

FIGURE 2. Magnetic resonance image of brainstem; resonance time 520 msec; echo time 25 msec. Sagittal section showing infarct below pontomedullary junction (arrow).

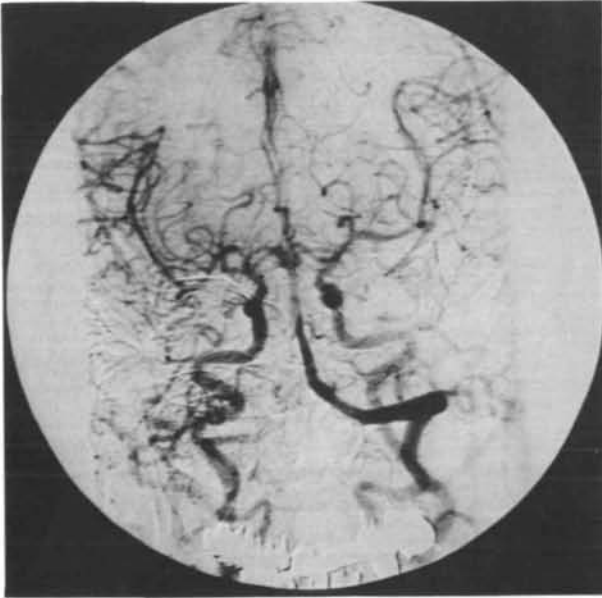


FIGURE 3. Digital subtraction angiogram of vertebral artery showing hypoperfusion on right. No focal stenosis or occlusion was revealed.

hypoglossal nerve palsy was absent and it was impossible to make a clinical diagnosis; pathologic examinations, which were carried out several decades after the stroke, resulted in the diagnosis. In the present patient the lesion was limited to the upper medulla, and hypoglossal nerve palsy was absent. Since the hypoglossal nucleus is a cell column of 18 mm in the medulla,¹⁶ infarction limited to a small portion does not cause hypoglossal nerve palsy.⁵ Therefore, it is necessary to detect the infarct to make a diagnosis in patients without hypoglossal nerve palsy.

In all the reported patients, the sites of the infarcts included the medullary pyramid. The upper third of the medullary pyramid is supplied by the vertebral arteries and their penetrating branches. In the present patient and in two other patients, the infarct was limited to the upper third of the medullary pyramid. The lower two thirds of the medullary pyramid are supplied by the anterior spinal artery and its branches.^{11,17,18} Seven patients had an infarct limited to the lower two thirds of the medullary pyramid. Among three patients with infarcts limited to the upper third of the medullary pyramid, prognosis was good except in one patient who was 78 years old and died of pneumonia. Motor paresis was unilateral and hypoglossal nerve palsy was absent from all three of these cases. In contrast to these three patients, the

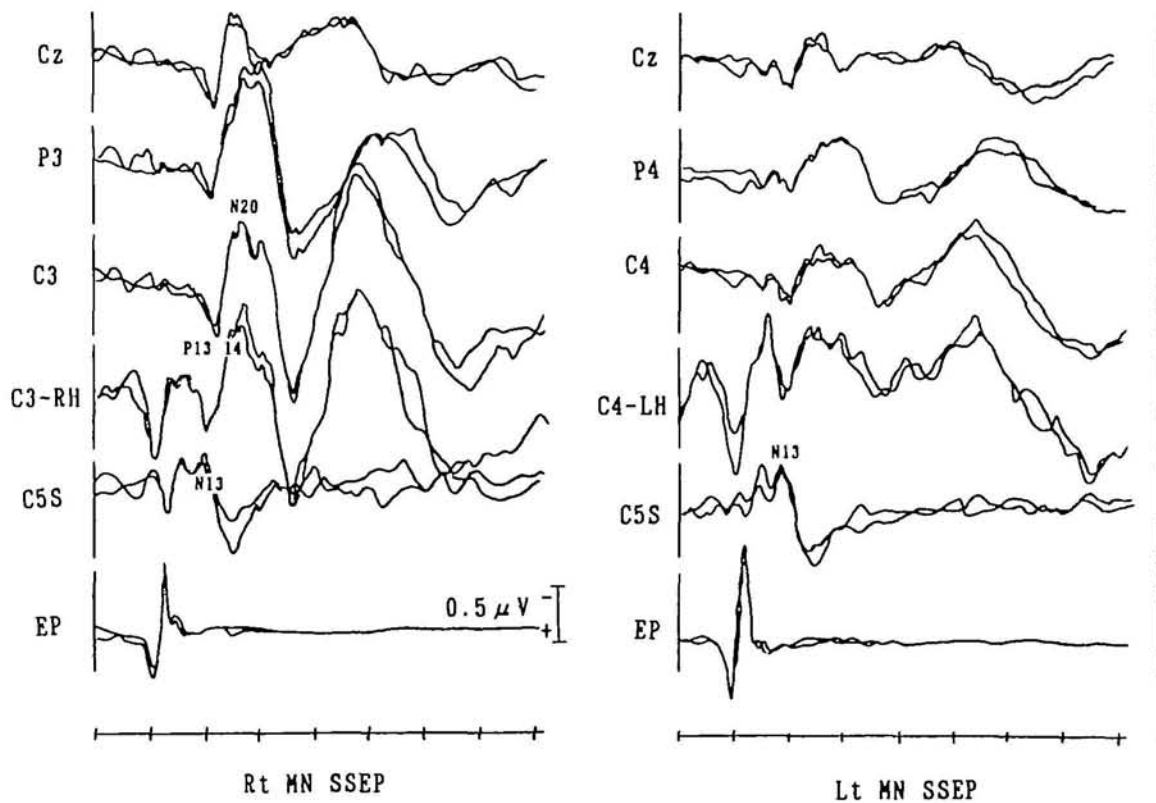


FIGURE 4. Recordings of somatosensory evoked potential (SSEP) stimulating median nerve (MN). EP (Erb potential) and N13 were evoked well on both sides. On C3 and C4, the amplitudes of P13-14 and N20 were reduced on left (Lt) MN SSEP. This finding is consistent with a lesion in the right medullary medial lemniscus. Cz, P3, P4, C3, P3, and C5S (5th cervical spine) were recorded with cephalic references. C3-RH (right hand) and C4-LH (left hand) were recorded with noncephalic references. (Negative upward in all records).

seven with a lesion limited to the lower two thirds of the medullary pyramid had a high incidence of respiratory paresis and the prognosis was poor, even for those with small lesions. Six of these seven patients died. In addition, three of these seven patients had tetraparesis due to the bilateral anterior spinal arteries joining together in the lower medulla so that a single anterior spinal artery supplied the lower medulla, resulting in lesions often extending bilaterally. The cause of death was respiratory or circulatory disturbance, which occurred ≤ 48 hours after the infarction.

Thus, detection of the site of lesions using MRI is important to determine the prognosis in patients with medial medullary infarction.

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KEY WORDS • cerebral infarction • magnetic resonance imaging • medulla oblongata