

Sensory Dermatomal Representation in the Medial Lemniscus

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Background: Restricted sensory deficits along the somatotopic topography of the medial lemniscus rarely develop in medial medullary infarction. We describe a patient with medial medullary infarction who presented with dermatomal sensory deficits caused by a medial lemniscal lesion.

Case Description: A 58-year-old man presented with sudden right-sided hemiparesis and paresthesia. He had noticed the paresthesia below the level of the right L5 dermatome, where his vibration and position senses were mildly diminished. His paresthesia was more severe over the right calf and foot. Magnetic resonance images of the brain showed an acute small infarct in the medial-

ventral portion of the left rostral medulla oblongata. A nerve conduction study and electromyography showed no abnormalities. At follow-up, the patient's motor and sensory deficits had improved considerably.

Conclusions: The patient showed lemniscal sensory deficits below the right L5 dermatome that were caused by the partial involvement of the medial lemniscus. These findings suggest that lemniscal sensory dermatomal representation is preserved at least up to the level of the medulla oblongata.

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MEDIAL medullary infarction (MMI) is a rare occurrence. Although the detection rate of small brainstem lesions has increased since the advent of magnetic resonance imaging (MRI), MMI still accounts for fewer than 1% of the total number of strokes.¹ In MMI, sensory abnormality is the second most common manifestation to occur, preceded by motor weakness.^{1,2} However, sensory deficits that are restricted to certain parts of the patient's body have rarely been described.^{1,3} Prior studies have shown that these restricted acral sensory deficits develop after small infarctions in the brainstem,⁴⁻⁶ thalamus,⁶⁻⁸ or cortical-subcortical areas.^{9,10} To our knowledge, only 1 case of MMI has been previously reported with sensory deficits in a dermatomal distribution.¹ We describe a patient with MMI who presented with an unusual dermatomal sensory manifestation. The possible pathogenesis is discussed.

REPORT OF A CASE

A 58-year-old right-handed man presented with the sudden onset of right-sided hemiparesis and paresthesia. He had

been well until 10 hours before admission, when he suddenly noticed an uncomfortable sensation of tingling in his right side on walking. His sensory symptoms, which were restricted to his limbs, especially the right lower extremity, persisted without any change in intensity before admission. Dysarthria and right-sided hemiparesis followed 5 hours later. Because of the progressive worsening of his motor weakness, he was referred to our hospital. There were no changes in his mental status. He had a history of poorly controlled hypertension and diabetes mellitus. He worked as a taxi driver. He had never smoked and had no history of excessive alcohol intake.

On admission, his temperature was 36.1°C, his pulse rate was 76/min, and his blood pressure was 146/95 mm Hg. He was alert and oriented, with fluent speech. Funduscopic tests showed no hypertensive or diabetic retinopathy. He had slight dysarthria. The remaining cranial nerve functions were intact. Motor examination revealed right-sided hemiparesis (III/V). His vibration and position senses were mildly diminished below the level of the L5 dermatome over the right lower extremity, and he noticed paresthesia in the same areas

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Figure 1. Schematic drawing of lemniscal sensory deficit in the patient. The gradient of gray to black indicates the degree of sensory deficit below the level of the L5 dermatome.

(**Figure 1**). His pain and temperature senses were intact. The dermatomal paresthesia was more severe over the lateral surface of the calf and the dorsolateral aspect of the foot. The deep tendon reflexes were normal in all tested joints, and the results of a Babinski test were positive on the right sole.

The patient's preprandial and postprandial blood glucose concentrations were 7.1 mmol/L (128 mg/dL) and 13.5 mmol/L (244 mg/dL), respectively. His hemoglobin A_{1c} level was increased (0.08). Brain MRI scans obtained 1 day after the ictus demonstrated a small infarct in the medial-ventral portion of the left rostral medulla oblongata (**Figure 2**). Magnetic resonance angiography revealed no abnormalities in the basilar artery or in either distal vertebral artery, with the exception of mild stenosis in the stems of both middle cerebral arteries. The results of a nerve conduction study and electromyography to exclude the possibility of a simultaneous lumbosacral radiculopathy were normal. The patient was treated with a continuous infusion of heparin sodium for 5 days, followed by warfarin sodium therapy (4 mg/d), and showed a gradual improvement of motor and sensory deficits, without fluctuation. After 5 months of anticoagulant therapy, the patient's sensory symptoms had improved to nearly normal, and he was able to walk without assistance.

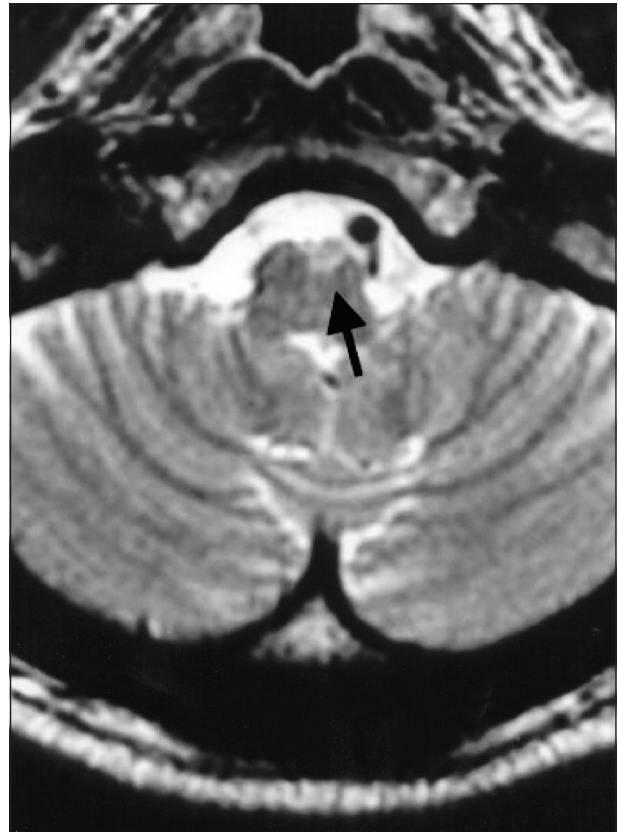


Figure 2. T2-weighted magnetic resonance imaging scan shows a small infarct in the left medial medulla oblongata (arrow).

COMMENT

The typical neurologic signs associated with MMI, or Dejerine syndrome, include (1) hemiparesis contralateral to the infarct, (2) hemisensory loss of the posterior column type contralateral to the infarct, and (3) weakness of the tongue ipsilateral to the infarct.^{11,12} Before the era of MRI, there were few case reports of MMI. Frequent manifestations of pure motor hemiparesis² or sensorimotor syndrome usually led clinical physicians to misclassify MMI as lacunar infarction. Moreover, pathological diagnosis by autopsy was extremely difficult and rare because of the relatively favorable prognosis of this disease.^{2,13} However, the introduction of high-resolution MRI has led to the clinical radiologic and etiologic correlation of brainstem ischemic strokes, especially strokes in the medulla and midbrain.^{1,2,13}

Numerous reports have provided evidence to show that restricted acral sensory deficits frequently occur after small strokes in the thalamic and cortical areas.^{4-10,14} Fisher¹⁵ stated that isolated paresthesia of the face, arms, and legs suggests thalamic involvement, whereas selective involvement of some fingers may indicate a cortical localization. Also, Kim¹⁴ observed that dominant sensory involvement of the upper lip, thumbs, and index fingers occurred with thalamic and thalamocortical strokes, and that cortical-subcortical strokes usually caused cheiro-oral or restricted finger involvement. Because of these findings, the somatotopic topography of the ventralis posterior nucleus of the thalamus is relatively well known. However, it is

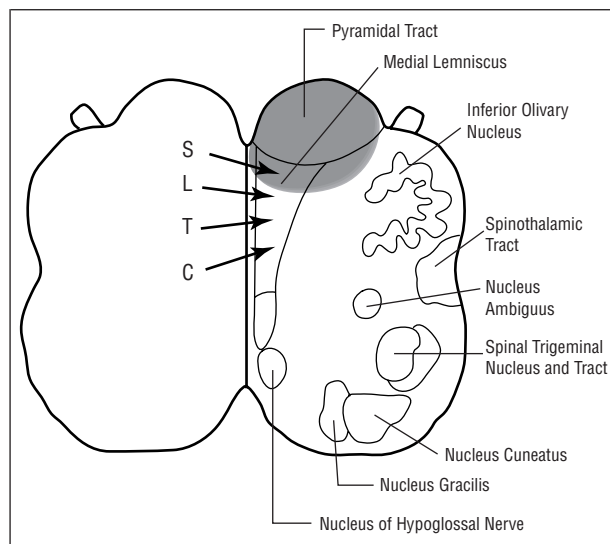


Figure 3. Sensory topography of the medial lemniscus in the patient. The gray area indicates the lesion; S, the area of sacral dermatomal representation; L, lumbar; T, thoracic; and C, cervical.

somewhat unclear in the brainstem. In the medulla oblongata, the medial lemniscus lies in a ventral-dorsal direction, in which the sensory topography is arranged so that the leg-representing area is located ventrally and the arm-representing area is located dorsally.¹⁶ On the level of the pons, most of the sacral segments are laterally located, and the cervical segments are medially located.¹⁷ However, the presence of dermatomal representation in these structures has not been previously reported. Recently, Kim et al³ described a patient with MMI whose paresthesia was confined to the lower part of the leg and was not distributed along the dermatomes. Although Bassetti et al¹ reported a case involving a patient with a pseudospinal sensory level over the trunk (T-11 level), they did not correlate it with functional neuroanatomy.

Our patient showed hemiparesis and a restricted lemniscal sensory deficit along the dermatomes. Both symptoms were caused by the involvement of the pyramid and medial lemniscus, respectively. Because there was no evidence of lumbosacral radiculopathy on electromyography or physical examination, we determined that the involvement of the medial lemniscus probably caused these peculiar manifestations. These findings suggest that sensory dermatomal representation may be preserved up to the level of the medulla oblongata, although, to our knowledge, there have been no corresponding reports with regard to the nuclei of the cuneatus or gracilis. Considering the established somatotopic topography of the medial lemniscus in the medulla oblongata, we speculated that most of the sacral dermatomes were ventrally located and that most of the cervical dermatomes were dorsally located (**Figure 3**). The fact that the patient had more severe paresthesia in the distal area of the lower extremity than in the proximal area might be explained by the “lowest threshold pattern.”^{18,19} A previous study on animals indicated a lower sensory threshold in the fingers and toes,¹⁹ and it is well understood that the acral parts of the human sensory pathway disproportionately represent the large areas of the cortex.^{20,21} These concepts can

be applied in the same way to the medial lemniscus in the human sensory pathway.

Our patient had no other vascular risk factors besides hypertension and diabetes mellitus, and neither transthoracic echocardiogram nor magnetic resonance angiography showed any remarkable abnormalities. Hence, thrombotic occlusion of the small perforating branches in the brainstem was responsible for the pathogenesis of the stroke in our patient.

In summary, we report the first case (to our knowledge) of MMI with dermatomal lemniscal sensory deficits caused by involvement of the medial lemniscus. Our findings suggest that sensory dermatomal representation may be preserved at least up to the level of the medulla oblongata. Although we were unable to determine the precise somatotopic arrangement of the lemniscal pathway in this case, it may provide some clues to elucidate the functional neuroanatomy of the medial lemniscus.

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