

LETTERS TO THE EDITOR**TO THE EDITOR****Pure Dermatomal Sensory Deficits in Lateral Medullary Infarction**

Lateral medullary infarction (LMI) is a well-recognized vascular syndrome of the vertebrobasilar territory, which is typically characterized by the symptoms of vertigo, nystagmus, dysphagia, dysarthria, ipsilateral Horner's syndrome, ipsilateral ataxia and contralateral hypalgesia. Lateral medullary infarction is often incomplete, however, isolated and restricted sensory deficits along the somatotopic topography of the spinothalamic tract are less common in LMI¹⁻⁴. Isolated contralateral dermatomal pain and temperature sensory deficits below the T3 sensory level as a single manifestation of LMI has not been reported previously. Here we will describe a LMI patient who uniquely presented with infrequent symptoms of contralateral dermatomal pain and temperature sensory deficits.

CASE REPORT

A 60-year-old woman with hypertension and diabetes mellitus for five years noticed that, an isolated numbness appeared in the left-sided lower extremity, when preparing breakfast at about eight o'clock four days before admission. Clinical evaluations didn't show any abnormal neurological findings except hypesthesia in the left lower extremity. Brain computed tomography (CT) didn't reveal any abnormal findings either. Therefore, she was treated as lumbar disc herniation and prescribed for NSAIDs initially. However, her symptoms did not improve. She had no headache, tinnitus, facial numbness, or weakness in the extremities, neither diplopia nor dysphagia. She had no previous history of head or neck trauma, and denied history of recent cold. Four days later, she visited the outpatient clinic of our department and was admitted. A neurological examination on admission revealed that she was alert and oriented, and did not have neck stiffness, facial or limb weakness, diplopia or Horner's syndrome. There was no hoarseness or dysarthria. Elevation of the soft palate was intact on phonation. Her tongue did not deviate on protrusion. Pain and thermal sensations of the left trunk and lower limb decreased to below the T3 level. Touch, vibration and joint sensations, as well as two-point discrimination, were preserved. The deep tendon reflexes were unremarkable; the plantar reflexes were flexor. There were no dysmetria on finger-to-ear and heel-to-shin testing.

Results from laboratory studies, including complete blood cell and platelet counts, erythrocyte sedimentation rate, blood electrolytes, chemistry, liver enzymes, cholesterol, triglycerides, the prothrombin and partial thromboplastin time were all normal, except for a mild increase in homocysteine. Chest X-ray, echocardiogram, and electrocardiography were all normal as well.

Thoracic spine MRI and somatosensory evoked potential were initially performed to exclude spine lesions, and revealed no abnormal findings. Diffuse-, T2-weighted and FLAIR magnetic resonance imaging (MRI) of the brain then performed on the fourth day after onset showed a small lesion with a high

signal intensity in the right lower medulla oblongata, and moderate low signal intensity in T1-weighted image, consistent with acute infarction (Figure 1). The patient was treated with antiplatelet agents, atorvastatin, and edaravone for 15 days, and the paresthesia did not change.

DISCUSSION

Lateral medullary infarction is one of the most well-characterized vascular infarctions of the brainstem. The clinical features of LMI can have diverse neurological manifestations due to the anatomical characteristics of the medulla⁵. According to previous reports, ataxia is the most common neurological symptom in LMI, and was more common in patients with lesions located in the laterocaudal part of the medulla. Contralateral hypalgesia is the next most frequent neurological symptom of LMI. The most common pattern of sensory abnormality in LMI is loss of pain and heat sensations on the ipsilateral side of the face and the lower part of the body on the contralateral side, which is associated with other common manifestations such as vertigo, unsteadiness, Horner's syndrome, and dysphagia. However, pure sensory deficits as an isolated symptom are not a feature of LMI. Moreover, pure sensory deficits in a dermatomal distribution of this patient have rarely been reported¹⁻⁴.

In this case, left-sided pure pain and temperature sensory deficits below T3 sensory level presented as a single and isolated sign of LMI without other common neurological symptoms. We attributed this to the lesion restricted to the right mediolateral aspect of the medulla, posterior to the inferior olivary nucleus³.

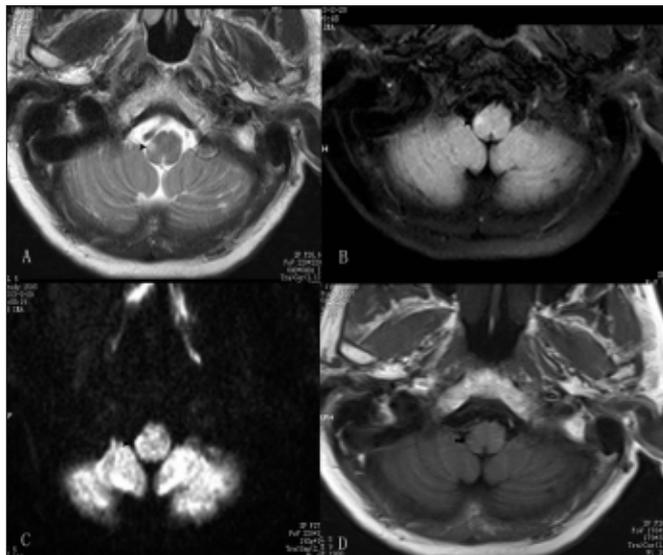


Figure 1: T2-weighted, FLAIR, and Diffusion-MRI of the brain revealed a small lesion with a high signal intensity in the right lateral medulla (arrow heads) (A,B,C), and moderate low signal intensity in T1-weighted image (arrow) (D) consistent with acute infarction.

Moreover, pain and temperature sensory deficits below the T3 level on the contralateral side of the body in our patient were due to the somatotopic organization of the spinothalamic tract, because of the sacral afferent fibers located in the lateral medullary part and the cervical afferent fibers ascended more medially. The lesion did not extend sufficiently far to the posterolateral medulla to affect the ipsilateral descending tract and the nucleus of the trigeminal nerve, thus sparing pain and temperature sensations in the ipsilateral face. The crossed ventral trigeminothalamic tract coursing to the medial part of the spinothalamic tract carries pain and temperature sensations from the contralateral side of the face. Thus, if the lesion extends more medially to involve ipsilateral cervical sensory fibers as well as the contralateral trigeminothalamic tract, this would lead to contralateral sensory deficits of the entire body without dermatomal representation and contralateral facial hypesthesia. However, if the lesion extends more posterolaterally to involve the ipsilateral spinocerebellar tract, this would lead to vertigo and ipsilateral ataxia (Figure 2).

The somatotopic topography is unclear in the medulla. Phan et al described a patient with pain and temperature sensory loss below the T9 sensory level associated with disequilibrium due to LMI following vertebral artery dissection¹. Iwasaki et al described a patient with hypoalgesia and hypothermesthesia in a lower extremity due to the compression of the medulla oblongata by an elongated vertebral artery (VA)². Shibata et al described a patient with an unusual manifestation of isolated limb pain and thermal sensory disturbance had sudden deafness due to medullary infarction caused by a vertebral artery dissection³. Song et al reported a patient with an isolated dermatomal sensory deficit at the T4 sensory level as single manifestation of LMI⁴. All of the four cases showed a small local infarction in the mediolateral aspect of lower part of the medulla oblongata, the same as this case. The observation reported here will help to improve the understanding of the somatotopic topography of the medulla.

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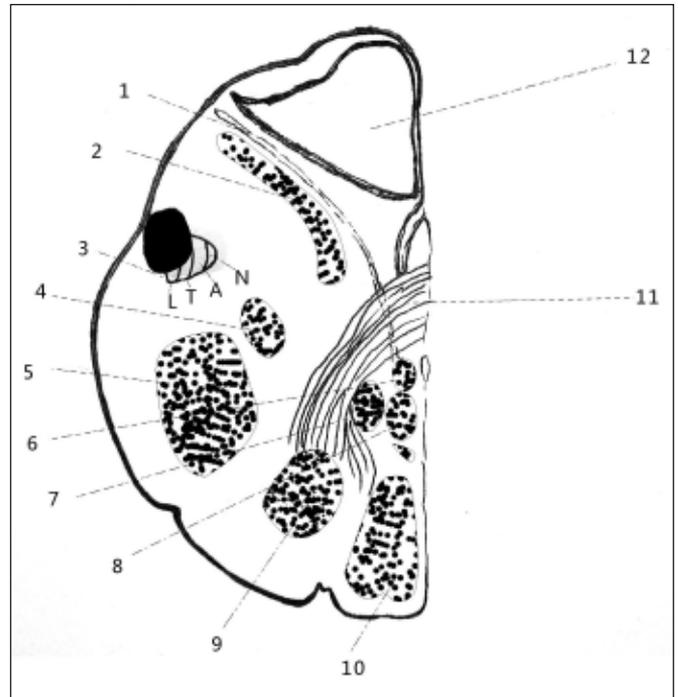


Figure 2: Sensory topography of the spinothalamic tract in the patient. The gray area indicates the presumed location of the lesion in the lower medulla oblongata. A, the area of arm dermatomal representation; L: leg; N: neck; T: trunk. 1. hypoglossal nerve intramedullary fiber; 2. inferior olivary nucleus; 3. spinothalamic tracts; 4. nucleus ambiguus; 5. spinal nucleus of trigeminal nerve; 6. hypoglossal nucleus; 7. nucleus of solitary tract; 8. dorsal nucleus of vagus nerve; 9. cuneate nucleus; 10. gracile nucleus; 11. decussation of medial lemniscus; 12. pyramid.

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