

Lateral Medullary Infarction Presenting as Brown-Séquard Syndrome-like Manifestation: A Case Report and Literature Review

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Abstract-

Purpose: Lateral medullary infarction is not uncommon in clinical practice of neurology. This report describes a patient who initially presented with Brown-Séquard syndrome-like manifestation but was later diagnosed with acute infarction in the left lower lateral medulla.

Case report: A 65-year-old woman presented with acute onset of unsteadiness, left side hemiparesis, left limb dysmetria, left side partial Horner syndrome, and paresthesia in the right lower limb and trunk with a sensory level at T5 on the right. No bulbar symptoms nor facial paresthesia was noted. Brown-Séquard syndrome was suspected initially, but cervical spine magnetic resonance imaging showed only mild spinal stenosis. Brain magnetic resonance imaging revealed acute infarction in the left lower lateral medulla. The mechanism of this unusual presentation is discussed.

Conclusion: Brown-Séquard syndrome-like manifestation can be a rare presentation of lower lateral medullary infarction.

Key Words: Brown-Séquard syndrome, cerebrovascular disease, lateral medullary infarction.

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INTRODUCTION

Lateral medullary infarction (LMI), also known as Wallenberg syndrome or Vieusseux-Wallenberg syndrome, was first described in 1808 by the Genevan physician, Gaspard Vieusseux, who precisely described

the disease that he was personally suffering at a meeting of the Société médicochirurgicale de Genève. Wallenberg later described the syndrome in 1895 (clinical) and 1901 (autopsy findings)⁽¹⁾. The typical neurological deficits of LMI include crossed sensory deficit (loss of pain and temperature sensation on the ipsilateral face and con-

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tralateral limb/body), vertigo or dizziness, ipsilateral Horner syndrome, cerebellar ataxia, diminished gag reflex, dysarthria, and dysphagia. Brown-Séquard syndrome, which was first described by the famous British neurologist Charles-Édouard Brown-Séquard in 1850, is characterized by contralateral loss of pain and temperature sensation, as well as ipsilateral weakness and loss of proprioception. We report a patient presenting with Brown-Séquard syndrome-like manifestation, but acute infarction in the left lower lateral medulla was confirmed later.

CASE REPORT

The patient is a 65-year-old woman with histories of diabetes mellitus (DM) for 15 years and hypertension for 17 years on regular medication. She presented with acute onset of non-spinning dizziness, unsteadiness, a tendency to lean to the left side while walking or sitting and paresthesia in the right lower limb and trunk upon awakening since 4 days ago before admission. She also reported poor appetite and mild nausea, but she denied headache, neck pain, radicular pain, double vision, hiccups, incontinence, slurred speech or difficulty in swallowing. The unsteadiness persisted, and the paresthesia progressed upward to the right lower chest region subsequently. She was sent to our emergency department and then was admitted to our ward.

On physical examination, no knocking pain in the spine nor carotid bruit was detected. Neurological examination revealed left side hemiparesis, equivocal Babinski sign on the left side, symmetric generalized hyporeflexia, left side partial Horner syndrome (miosis and ptosis), left limb dysmetria, left side deviation during sitting and walking, and decreased pinprick sensation from the right lower limb to the trunk without sacral sparing. A clear sensory level at T5 on the right side was detected. Her eye movements, facial expressions and sensation, gag reflex, vibration sense, joint position sense (JPS), and sphincter functions were all intact. No nystagmus, dysarthria, dysphagia, or tongue deviation was detected. Brown-Séquard syndrome and a lesion of the left cervical spinal cord were suspected initially.

However, cervical spine MRI showed only mild spinal stenosis. Due to multiple stroke risk factors (old age, DM, hypertension), dysmetria and unsteadiness, we ordered a brain magnetic resonance imaging (MRI) immediately to exclude a brainstem lesion. Brain MRI revealed acute infarction in the left lower lateral medulla (Fig. 1). No obvious arterial dissection nor severe basilar or vertebral artery stenosis was detected by brain magnetic resonance angiography (MRA). Serum electrolyte and blood cell counts were within normal limits except for hyperglycemia (208 mg/dl). Daily oral aspirin 100 mg was prescribed. Sensory level slightly progressed upward after admission. Intravenous heparin was given. Rehabilitation was arranged. After treatment, she could ambulate slowly with a walker. She was discharged two weeks after admission with a prescription of daily oral aspirin 100 mg. Her neurological deficits recovered gradually, and no sequelae were detected after 2 years of follow-up.

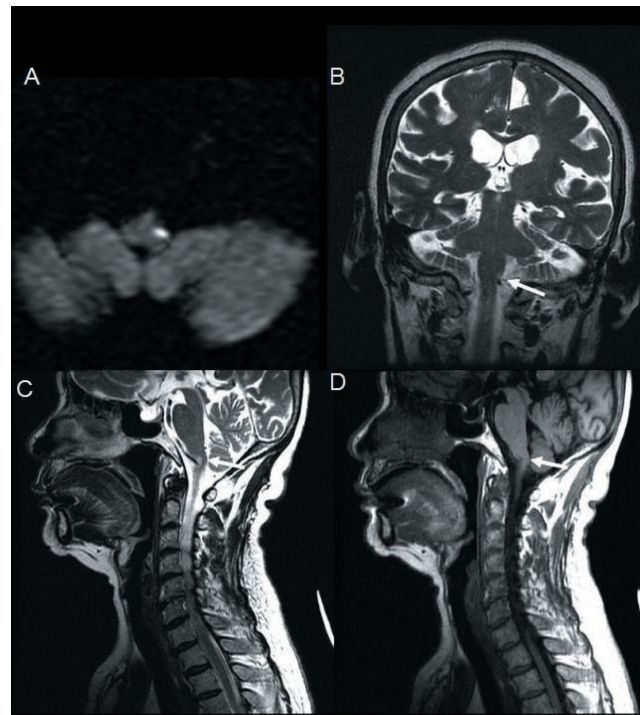


Figure 1. Brain MRI: diffusion weighted image (A), T2 (B, C) and T1 (D) weighted image showing left lower lateral medullary acute infarction (arrow).

DISCUSSION

Lateral medullary infarction, or Wallenberg syndrome, is the most common encountered medullary syndrome in the clinical practice of neurology. Medial medullary infarction (MMI) is relatively rare; the incidence of LMI is reportedly three to four times higher than that of MMI⁽²⁾.

Dissociated and crossed sensory loss is typical in LMI, and the classic sensory abnormality associated with MMI is contralateral lemniscal sensory loss of the entire hemibody, with or without facial involvement⁽³⁾. However, the incidence of this classic ipsilateral trigeminal and contralateral limb/body sensory pattern of LMI is only approximately 26%^(4,5). A sensory level on the trunk is an uncommon sensory deficit in both LMI and MMI. Kim collected data of 130 patients with pure LMI⁽⁵⁾. Limb/body sensory involvement without trigeminal involvement (isolated limb/body pattern) was noted in only 27 patients (21%). This sensory pattern was more common in caudal than in rostral medullary infarction. In the present case, the lesion is in the lower medulla. No facial sensory deficit in our case is compatible with their findings. Sensory gradient or level was observed in 28 patients (22%) in the series study of LMI by Kim. In MMI, lemniscal sensory loss with a pseudospinal level over the trunk has also been reported⁽³⁾. Possible explanations for this phenomenon are somatotopical organization of the spinothalamic tract and medial lemniscus. As in spinal cord, somatotopical organization also exists in spinothalamic tract at the level of medulla. Sacral afferent fibers are located in the lateral part, and cervical afferent fibers ascend more medially^(4,6). Somatotopical organization is also preserved in medial lemniscus at the level of medulla⁽⁷⁾. Sacral fibers are ventrally located and cervical fibers tend to ascend more dorsally. Besides, segmental arrangement of sensory fibers in the descending and ascending trigeminal tract and nuclei was also proposed by Kim⁽⁴⁾ et al. Due to the somatotopical organization mentioned above, sensory level may be reasonable in MMI and LMI. Figure 2 summarizes the proposed sensory lamination in rostral medulla. In the present case, a T5 sensory level at the contralateral side

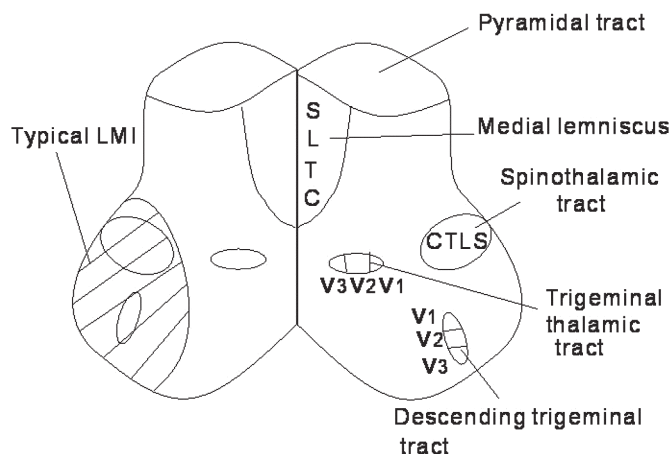


Figure 2. Proposed sensory lamination in rostral medulla. (LMI: lateral medullary infarction. C: cervical. T: thoracic. L: lumbar. S: sacral)

may be due to involvement of lateral part of the spinothalamic tract.

Contralateral hemiparesis is the hallmark of MMI, and muscle power is usually preserved in LMI. Ipsilateral weakness is a rare manifestation of both LMI and MMI. According to a case series of 167 LMI patients (concomitant cerebellar infarction not excluded) by Kameda et al., 7% of patients had ipsilateral spastic hemiplegia, which is compatible with Opalski syndrome⁽²⁾. The syndrome has been attributed to the occlusion of vertebral artery⁽⁸⁻⁹⁾. Ipsilateral weakness also occurs in MMI. A series of MMI patients reported by Bassetti et al. revealed ipsilateral weakness in two of seven patients⁽³⁾. The lesion causing ipsilateral weakness in MMI and LMI is thought to be at the level of cervicomedullary junction after pyramidal decussation. Kim mentioned several possible causes of ipsilateral limb weakness in LMI: (1) partial involvement of the already decussated pyramidal tract or uncrossed lateral corticospinal tract; (2) involvement of aberrant pyramidal fibers in the medial part of the tegmentum of the medulla oblongata; (3) lemniscal fibers and cerebellar tract involvement inducing abnormal sensorimotor feedback, resulting in mild weakness or loss of movement control⁽¹⁰⁾. Pyramidal tract involvement on brain MRI is not obvious in the

present case. No hyperreflexia has been detected after more than 2 years of follow-up, but DM may be a contributing factor. Therefore, we consider the involvement of aberrant pyramidal fibers after pyramidal decussation may be the cause of ipsilateral weakness. Cerebellar tract involvement may also contribute to the weakness to some extent. Vibration sensation and JPS is preserved in this patient, therefore lemniscal fibers involvement is less likely.

This case and our review indicate that (1) although dissociated and crossed sensory loss is typical in LMI, this sensory presentation is uncommon; (2) Sensory lamination exists in spinothalamic tract and medial lemniscus at the level of medulla, so sensory level may be a reasonable presentation of both MMI and LMI; (3) ipsilateral limb weakness can be a manifestation of both LMI and MMI if the lesion is located at the level of cervicomedullary junction after pyramidal decussation; (4) Brown-Séquard syndrome-like manifestations can be a rare presentation of LMI. If no spinal cord lesion is found, brain MRI should be performed to exclude possible medullary lesions.

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