Case Report

Restricted Dissociated Sensory Loss in a Patient With a Lateral Medullary Syndrome
A Clinical-MRI Study

Paolo Cerrato, MD; Daniele Imperiale, MD; Mauro Bergui, MD; Marco Giraudo, MD; Chiara Baima, MD; Maria Grasso, MD; Leonardo Lopiano, MD; Bruno Bergamasco, MD

Background—Various sensory syndromes in lateral medullary infarctions are described. A small variation in the location of a lesion may lead to very different clinical features, owing to the complex anatomy of the medulla oblongata. MRI may identify the location and extent of the ischemic lesions, allowing a clear clinical-anatomical correlation.

Case Description—We describe a man with an ischemic lesion in the right portion of the lower medulla that presented a contralateral impairment of spinothalamic sensory modalities and an ipsilateral impairment of lemniscal modalities with a restricted distribution (left forearm and hand, right hand and fingers, respectively). The restricted and dissociated sensory abnormalities represent the only permanent neurological consequence of that lesion.

Conclusions—The atypical sensory syndrome may be explained by the involvement of the medial portion of spinothalamic tract and the lateral portion of archiform fibers at the level of the lemniscal decussation. (Stroke. 2000;31:3064-3066.)

Key Words: cerebral infarction ■ lateral medullary syndrome ■ medulla oblongata

Lateral medullary syndrome (LMS) is a well-recognized vascular syndrome of the vertebrobasilar territory. Classically, sensory dysfunction in LMS is characterized by the dissociated involvement of the spinothalamic sensory modalities (impairment of pain and thermal sensation over the contralateral hemibody/limbs and over the ipsilateral face).1-4 Although sensory signs and symptoms may present various topographic distributions,4-10 a dissociated sensory pattern involving upper limbs has not yet been reported. Moreover, even if ipsilateral upper-extremity symptoms are occasionally reported, they have never been clearly explained.11 We describe a patient with a small ischemic area in the right part of the medulla oblongata, with an impairment of pain and thermal sensation in the left arm and forearm, and tactile discrimination and deep sensation in the right hand. MRI identified the location and extension of the small ischemic lesion, allowing a clear clinical-anatomical correlation.

Case Report

A 54-year-old man was admitted to our inpatient department because of the abrupt occurrence of gait and postural imbalance, dizziness, nausea, vomiting, and numbness of the right hand and fingers. Regarding vascular risk factors, mild hypertension and cigarette smoking were present. Upon admission, neurological examination revealed gait ataxia with falling to the right side; vertical nystagmus on downward gaze; decrease of vibration sense and 2-point discrimination over the right hand; and impairment of thermal and pain sense over the left hand, forearm, and, to a lesser degree, over the arm and neck. No other neurological signs were present.

A cranial CT obtained 24 hours from onset was normal. Extracranial vessel duplex ultrasonography showed a moderate atheromatosis of both internal carotid arteries without significant stenosis. Transthoracic echocardiography showed a mild left ventricle concentric hypertrophy, whereas transesophageal echocardiography was normal. MR angiography was normal, particularly concerning the vertebral arteries.

Routine blood analysis highlighted only a mild increase in hematocrit level and red blood cell count. Immunologic investigations, fasting lipid profile, and homocyst(e)ine plasma levels were all normal.

MRI showed a small, diagonal lesion in the dorsolateral portion of the right lower medulla, between the retro-olivary sulcus and the inferior cerebellar peduncle, sparing the most lateral portion (Figures 1 and 2). The lesion was consistent with an infarction in the territory of the perforating arteries arising from the vertebrobasilar junction.

The condition of the patient improved within a few days. Neurological examination at discharge revealed only an impairment of the temperature sense involving the left hand and forearm. He continued to complain of numbness and paresthesia of the right fingers (a described symptom of the proprioceptive pathways involvement),12 even though the abnormalities of 2-point discrimination, vibration, and joint...
position/movement sense were no longer evident. Although sensory symptoms and signs were unchanged at a 2-month follow-up examination, the patient was fully independent and able to work.

Discussion

The case here reported broads the spectrum of the LMS. Sensory abnormalities in our patient may represent an undescribed variant of LMS. The dissociated sensory impairment involving upper limbs (pain and thermal sense in the contralateral hand and forearm, tactile discrimination and deep sense in the ipsilateral hand and fingers) is explained by the site of the lesion (Figures 1 and 2) and by the anatomy of the lower medulla at the level of the lemniscal decussation (Figure 3). In the spinal cord and lower medulla, the proprioceptive pathway is located in posterior columns and is somatotopically organized, being the fibers from the cranial districts of the body and terminating in the nucleus cuneatus (NC). From the NC, located in the lateral position, the secondary proprioceptive fibers (also named “archiform”) cross the midline (so-called lemniscal decussation) and reach the opposite side of the medulla constituting the medial lemniscus (Figure 3). The archiform fibers are also somatotopically organized, being fibers from the upper limb located in lateral position.

Fibers in the spinothalamic tract in spinal cord and medulla are arranged in a concentric manner, with the most superficial coming from caudal districts and the most inner from cranial districts. Therefore, a lesion located between the medial lemniscus and spinothalamic tract in the lower medulla may explain the restricted dissociated sensory syndrome of our patient if it involves the lateral portion of archiform fibers and the medial portion of the spinothalamic tract (Figure 3). An isolated lesion located between the upper medulla and the parietal cortex cannot determine a dissociated sensory syndrome because the spinothalamic tract and medial lemniscus run on the same side. Brain stem ischemic lesions usually involve the 2 ascending sensory pathways in a separate fashion: the medial lemniscus is involved in paramedian medullary infarction and the spinothalamic tract in lateral medullary infarction because of the different vascular territories. A dissociated impairment of sensory modalities may be present in spinal cord lesions (the so-called Brown-Sequard syndrome), but sensory symptoms are almost always invariably associated with motor deficit.

Ipsilateral impairment of tactile discrimination and deep sensation may result from lesions of the medial portion of the medulla below the lemniscal decussation,8,10 but it is rarely reported in patients with LMS11,14; the Babinski-Nageotte syndrome, in which the medial lemniscus is occasionally involved, may represent a medially extended LMS.

In our patient, the sparing of pain and thermal sense over the left side of the trunk and leg may be related to the lack of involvement of the superficial layers of the spinothalamic tract, where the fibers from the caudal districts are located (Figure 3). Moreover, the incomplete impairment of lemniscal sensory modalities may be explained by the limited involvement of the proprioceptive fibers and is in agreement with experimental and clinical observations that lesions of the
dorsal column may be followed by only limited sensory losses.15,16

In addition, preservation of the nucleus ambiguus, usually involved in upper medullary infarction,9 may explain the absence of hoarseness and dysphagia. The initial occurrence of ataxia, vertical nystagmus on downward gaze, vertigo, and dizziness suggest the involvement of vestibular nuclei or vestibulo-cerebellar connection at the onset. Gait ataxia may also be related to the initial involvement of the inferior cerebellar peduncle or the spinocerebellar tracts in the medulla.9 Disappearance of those signs during the course may be explained by the sparing of the most posterolateral portion of the medulla in the definitive lesion (Figure 2). Finally, the absence of sensory impairment in ipsilateral trigeminal districts suggests that the lesion did not involve the most dorsal portion of medulla, where the descending tract and nucleus of the fifth nerve are located (Figure 2 and 3).

In conclusion, in this report we described a patient with a restricted (upper limbs) and dissociated (pain and thermal hypesthesia in the contralateral left hand and forearm; numbness and decrease of deep sensation in the ipsilateral right hand and fingers) sensory syndrome related to a small ischemic lesion in the right portion of the lower medulla, a location that may explain the dissociated sensory deficits because it presumably involves the medial portion of the spinothalamic tract and the lateral part of the archiform fibers at the level of the lemniscal decussation.

References
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