Letter to the Editor

Eur Neurol 2008;59:216–218
DOI: 10.1159/000114051

Received: January 3, 2007
Accepted: June 4, 2007
Published online: January 29, 2008

Isolated Spinothalamic Sensory Impairment of the Left Arm following Contralateral Medullary Infarction: A Case Report

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Cerebral MRI with thin brainstem slices (thickness 2 mm) detected a small ischemic lesion in the right dorsolateral and ventral medulla (fig. 1). MR angiography showed occlusion of the right vertebral artery (fig. 2), as did duplex sonography. Sensory evoked potentials from the median and tibial nerves were not delayed, providing evidence of normal lemniscal sensation.

The patient was discharged with the diagnosis of a lacunar brainstem stroke following occlusion of the right vertebral artery. Medication with aspirin and simvastatin was initiated for the purpose of secondary prevention.

Discussion

In our patient, impairment restricted to pain and temperature sensation were the only features of a lacunar medullary infarction affecting the spinothalamic tract. There were no other symptoms suggestive of brainstem stroke on examination 4 weeks later. Furthermore, the patient reported no other transient neurological deficit. Nevertheless, it cannot be ruled out that other brainstem symptoms, such as mild Horner’s syndrome, were initially present, but not noticed by the patient.

Ischemic stroke with pure sensory symptoms may result from infarction of the brainstem, the thalamus, the lenticulocapsular area/corona radiata or the sensory cortex [1, 2]. Lemniscal and spinothalamic sensation may be affected together or selectively. In brainstem lacunar stroke, paramedian pontine infarction is the most common cause of pure sensory impairment involving lemniscal sensation [3]. Lateral medullary infarction is a rare cause of isolated contralateral spinothalamic sensory impairment and has been described only twice in the literature [4, 5], with the suggestion that it be classified as type IV according to Stopford’s classification of sensory loss in lateral medullary infarction [4, 6]. Most commonly, lateral medullary infarction leads to a sensory deficit according to Wallenberg’s syndrome, with loss of temperature and pain sensation in the ipsilateral face and the contralateral hemibody [7].

To our knowledge, this is the first reported case of lateral medullary infarction with isolated pain and temperature impairment following vertebral artery occlusion. Vertebral artery occlusion may be either asymptomatic or may lead to poste-
Sensory Stroke in Medullary Infarction

Prior circulation stroke or transient ischemic attack [8]. Occlusive disease of the vertebral artery represents the most common cause of lateral medullary infarction [7, 9–11]. In this atypical presentation, an active search for a cardiac or arterial cause of the stroke is indicated. In our patient, occlusion of the distal right vertebral artery suggestive of atherosclerotic-thrombotic occlusion was found using MR angiography and duplex ultrasound examination. Atheromatous intima thickening was seen in both internal carotid arteries in duplex sonography, without significant stenosis. Patient cardiovascular risk factors included...
smoking, adiposity, hypercholesterolemia, and hypertension. Aside from atheroma, cardioembolism and dissection are frequent causes of vertebral artery occlusion [7] in patients with medullary infarction. Transthoracic echocardiography showed left ventricular hypertrophy with normal ejection fraction, but did not point to cardioembolic stroke etiology. ECG did not suggest atrial fibrillation.

Thin-slice (2 mm) MRI scan showed dorsolateral infarction of the right middle medulla oblongata. The lesion was visible on one sagittal and two axial slices only. Thus, it is not surprising that the infarct was not evident on the MRI scan (slice thickness 6 mm) performed 4 weeks before admission to our hospital. In lacunar brainstem lesions, thin-slice MRI is likely to demonstrate higher sensitivity and has been suggested in standard imaging protocols for brainstem ischemia [12].

The patient had contralateral spinothalamic sensory deficit, including the arm and trunk. Cerrato et al. [13] reported spinothalamic sensory impairment of the left arm and neck combined with lemniscal sensory impairment of the right side in a patient with infarction of the lower lateral medulla. The authors concluded that a lesion was present in the right medial fibers of the spinothalamic tract representing the arm and neck. In our patient, the lesion extended to more superficial spinothalamic fibers, including the representation of the left trunk. Figure 3 shows an enlarged view of the infarction in relation to an anatomical drawing of the medulla oblongata at this level. The lesion correlates well with the location of the lateral spinothalamic tract. Although clinically not affected in our patient, involvement of superficial fibers from the sacral region and the leg could be expected from the lesion’s location. However, it has been reported that sensory findings in brainstem lesions do not always correspond well with their anatomical site [14, 15].

Arterial blood is supplied to the medulla by perforating branches arising from the vertebral artery, anterior and posterior spinal artery, anterior inferior and posterior inferior cerebellar artery and the basilar artery [16]. Those perforators are divided into distinct subgroups (anteromedial, anterolateral, lateral and posterior). Infarct location in our patient suggests the involvement of branches of the lateral group arising from the vertebral or posterior inferior cerebellar artery.

In conclusion, our report shows the diversity of pure sensory stroke in the brainstem. Although lateral medullary infarction is rarely connected with restricted spinothalamic impairment, it should be considered an infrequent cause of pure sensory stroke.

References