Face-arm-trunk-leg sensory loss limited to the contralateral side in lateral medullary infarction: a new variant

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Abstract
Two patients are reported on who experienced loss of pain and temperature sensation in the entire contralateral hemibody but sparing the ipsilateral face (pure sensory stroke pattern) related to acute lateral medullary infarction. In both patients MRI showed a notch-like retro-olivary ischaemic lesion in the ventromedial tegmentum with preservation of the far lateral medulla. The mediolateral lesion involved the crossed lateral spinothalamic tract and the ventral trigeminothalamic tract, corresponding to sensory loss in the contralateral face, arm, and upper trunk. The ventrolateral extension of infarct damaged the far lateral part of the spinothalamic tract, corresponding to sensory loss in the contralateral lower trunk and leg. The findings suggest that hemisensory loss of the spinothalamic type involving—and limited to—the whole hemibody can occur in infarction in the lower brainstem. This form of pure sensory stroke may be classified as type IV of sensory loss in lateral medullary infarction.

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Loss of pain and temperature sensation due to lateral medullary infarction are well known.1–5 They classically involve the ipsilateral side of the face and the lower part of the body on the contralateral side, corresponding to far lateral medullary lesions (crossed type) or the type I sensory loss of Stopford’s classification.3 More medial lesions may lead to contralateral sensory loss limited to the upper part of the body (type II sensory loss), whereas large mediolateral lesions are a combination of crossed and unilateral pattern (bilaterial type or type III sensory loss). Sensory loss involving the face, trunk, arm, and leg on the side opposite to the lesion without affecting the ipsilateral side of the face—that is, a “pure sensory stroke” pattern—has never been reported.

Here we report on two patients with MRI evidence of acute lateral medullary infarction who experienced loss of the sensations of pain and heat in the entire contralateral hemibody, but sparing the ipsilateral side of the face.

Case report

PATIENT 1
A 30 year old man suddenly developed pulsatile frontal headache that became very intense within a few minutes, with nausea and photophobia. He immediately noticed paraesthesias and numbness affecting the entire left side of the body, especially the left hemiface and the left arm. He also complained of pain in the left trunk. Because of vertigo and severe unsteadiness, he was forced to walk on all fours. He did not notice diplopia or changes in the voice and in swallowing. The next morning, he was unable to walk without assistance and still complained of vertigo and vomiting and was therefore admitted to hospital.
On admission he was normotensive, and the general examination was normal. Neurological examination showed neither nystagmus nor ophthalmoparesis. The pupils were equal and reactive. The soft palate and tongue moved well, and there was no facial weakness. Pain and heat sensation was decreased over the entire left side. Vibratory and postural sensation was preserved. Touch sensation on the left side was reported to be different from that on the contralateral side of the body, but the patient was able to detect touch when his eyes were closed and to localise touch stimuli. Touch and pinprick discrimination were intact.

The deep tendon reflexes were brisk without Babinski’s sign. There was no weakness of the limbs. A minimal dysmetria was present on the right side.

Cerebral MRI disclosed a small left midlateral medulla infarct involving a notch-like band in the retro-olivary medullary tegmentum. The far lateral surface was intact and there was no cerebellar lesion (fig 2).

**Discussion**

The most common pattern of sensory abnormality in patients with lateral medullary ischaemia is loss of pain and heat sensation on the ipsilateral side of the face and the lower part of the body on the contralateral side. Sensory loss can also involve touch. This classic pattern (Wallenberg syndrome) is due to the involvement of the crossed lateral spinothalamic tract and the ipsilateral descending tract and nucleus of the trigeminal nerve. If the lesion extends more medially, the crossed trigeminothalamic tract (which carries pain and heat sensation from the contralateral side of the face) may be involved, leading to bilateral facial hypaesthesia (Stopford type III).

Less often, the hypalgesia is only contralateral, affecting the face, arm and trunk when the lesion is midlateral (Stopford type II). The preservation of the descending tract and nucleus of the trigeminal nerve explains the sparing of the ipsilateral side of the face. These different patterns have been clearly described by Matsumoto et al.

This partial sensory defect (upper or lower part of the body) on the contralateral side of the body is due to the somatotopical organisation of the spinothalamic tract. The sacral afferent fibres are located in the lateral medullary part and the cervical afferent fibres ascend more medially. The crossed ventral trigeminothalamic tract appends on to the medial part of the spinothalamic tract and carries pain and heat sensation from the contralateral side of the face.

In the series of nine cases reported by Matsumoto et al, in patients with the unilateral pattern of sensory defect the whole hemibody was never affected. This partial involvement was also seen in the study of Vuilleumier et al. In each case, the sparing of the leg was explained by the somatotopical disposition of the spinothalamic tract.

We are not aware of any report describing the “pure sensory stroke” pattern in addition to the common manifestations of lateral medullary infarction. In fact, in both our patients, MRI...
showed a notch-like retro-olivary ischaemic lesion in the ventromedial tegmentum, with preservation of the far lateral medulla. The topography of this lesion may explain this new clinical type of lateral medullary infarction, as it corresponds well with the anatomy of sensory pathways in the lower brainstem. Indeed, the mediolateral lesion involved both the crossed lateral spinothalamic tract and the ventral trigeminothalamic tract, corresponding to sensory loss in the contralateral face, arm, and upper trunk. The ventrolateral extension of the infarct damaged the far lateral part of the spinothalamic tract, corresponding to sensory loss in the contralateral lower trunk and leg.

Our findings show that hemisensory loss of the spinothalamic type, involving, and limited to, the whole hemibody, could be consistent with infarction in the lower brainstem. This previously unreported form of pure sensory stroke may be classified as type IV of sensory loss in lateral medullary infarction.

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