## SHORT COMMUNICATION

# Restricted pain and thermal sensory loss in a patient with pontine lacunar infarction: a clinical MRI study

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Received 15 July 2004 Accepted 4 August 2004 Pure sensory syndrome (PSS) is characterized by hemisensory symptoms without other major neurological signs. It was initially attributed to thalamic lacunar infarction, but several reports have shown the PSS can be due to small infarcts involving the posterior part of the internal capsula, the cerebral cortex and the brainstem. Paramedian and lateral pontine infarctions are associated respectively with lemniscal and spinothalmic (ST) sensory impairment. We describe a patient with an isolated impairment of the ST modalities caused by a segmental paramedian pontine infarction.

## **Case report**

A 50-year-old man developed vertigo, dizziness and imbalance followed by tingling and burning sensation involving his left neck that rapidly spread to the arm, trunk and leg on the left side. Vertigo and dizziness disappeared in a few minutes while sensory complaints partially improved in the following days. On admission, neurological examination revealed an impairment of thermal and pain sense over the left trunk, leg and, to a lesser degree, over the arm and neck. Four days later, the thermal and pain hypoestesia were limited to the left leg below the hip including genital and perineal areas. Touch, vibration and position sense as well as twopoint discrimination, graphestesia and sterognosia were normal. The patient complained a burning sensation worsened by touch, rubbing and hot water. No other neurological signs were present.

Routine blood analysis, duplex ultrasonography and angio-magnetic resonance imaging (MRI) of cervical arteries, as well as transthoracic and transoesophageal echocardiography were normal. Two cranial computed tomography (CT) performed 5 and 48 h after the onset were unrevealing. The study of sensory-evoked potentials from legs revealed normal values. A brain MRI performed 5 days from the onset showed a small ischemic lesion in the right upper pontine tegmentum (Fig. 1a–c). Four months later, the sensory impairment over the leg and genital areas was still present.

### Discussion

In our patient, a restricted pain and thermal sensory impairment was the predominant feature of a small lacunar pontine infarction suggesting the exact location of the spinothalamic tract. Only the vertigo at the onset suggested a brainstem involvement. In pontine infarction, the sensory abnormalities may resemble those of a thalamic or cortical stroke (Derouesne *et al.*, 1984; Kim, 1991; Wolk *et al.*, 2002). Either deep or superficial sensibility may be impaired and the sensory abnormalities are usually associated with other neurological findings (Hommel *et al.*, 1989; Helgason and Wilbur, 1991; Shintani *et al.*, 1994; Kim and Bae, 1997; Shintani, 1998) that suggest the exact topography of the lesion.

Interestingly, in our patient, the sensory impairment involved the spinothalamic (ST) modalities only. The clinical features of our patient raise some considerations regarding the anatomic site of the infarct. In the pons the medial lemniscus is located in the paramedian dorsal tegmentum while the ST tract runs in a more lateral position (Fig. 1d) (Kim and Bae, 1997). These two sensory tracts run separately even if they approach one another as they ascend in the brainstem. Thus, paramedian and lateral pontine infarctions are associated respectively with lemniscal and ST sensory impairment (Helgason and Wilbur, 1991; Shintani et al., 1994; Kim and Bae, 1997; Shintani, 1998). Nevertheless, the small ischemic lesion (Fig. 1a-c) corresponded to the medial lemniscus rather than to the ST tract and we would have expected an impairment of lemniscal sensory modalities rather than a pain and thermal sensory loss. Besides, in the ST tract, the ascending fibers from the lower limb are located laterally on the outside while those from the upper limb are located on the inside and temperature fibers are segregated in a more lateral position than pain fibers. In our opinion, the restricted pain and thermal sensory loss and the small extension of the infarct suggest the exact location of the ST tract in the brainstem. In addition, we can take into consideration other hypotheses to

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Figure 1 MR scan, (a) sagittal, T2-weighted (3800/120/2 TR/TE/) excitations; (b) coronal, T2-weighted (3800/120/2); (c) axial, proton densityweighted (2800/20/2). A round-shaped hyperintense lesion involves the rostral pons, in a paramedial location, ventral to the ML (a). The lesion corresponds to a lacunar infarction in the territory of a perforating branch of the basilar artery. Notice the tortuous basilar artery in front of the pons (a). The exact location is shown in the artist drawing (d): the lateral portion of the ML and the medial aspect of the STT are involved.

explain the presence of a crural ST sensory impairment in a paramedian pontine infarction. First, it has been reported that, in brainstem lesions, sensory findings do not always correlate well with the anatomic site of the infarct (Helgason and Wilbur, 1991). Secondly, we hypothesize that pain and temperature fibers may be involved outside the ST tract: spinothalamic tract (STT) projection is rather diffuse not only in the thalamus but also in the brainstem and there is evidence that pain and temperature fibers are also carried by medial lemniscus as well as by spinoreticular or spinomesencephalic tracts (Brodal, 1981) projecting into the reticular formation of lower medulla and periacqueductal gray matter.

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