Cerebrovasc Dis 2010;30:200–201
DOI: 10.1159/000317109

Infarction of Primary Sensorimotor Cortex Impairs Pain Perception

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Despite lesion studies and neurophysiological investigations it remains still doubtful if the primary somatosensory cortex (S1) contributes to human pain perception [1, 2]. Even functional imaging studies do not consistently show pain-related activation of S1 [3–5].

In this case report, we performed quantitative sensory testing (QST; i.e. determination of detection thresholds for warm, cold, heat pain, cold pain, touch, pin-prick pain) in a 75-year-old man with an acute lacunar infarction of the right somatosensory cortex (fig. 1a). MRI scans of the brain detected no previous stroke, MR angiography of head and neck revealed no stenosis. The patient had atrial fibrillation and an embolic stroke was suspected. Thermal QST was performed using a Peltier-driven device (TSA 2001-II, MEDOC, Israel). Furthermore, thresholds were determined for touch (von Frey hairs) and mechanical pain (pin-prick needle stimulators). The QST procedure was based on the protocol established by the German Research Network 'Neuropathic Pain' [6]. On the hand contralateral to the stroke, the patient showed impaired perception of innocuous warm ($\Delta 20^\circ\text{C}$), cold ($\Delta 32^\circ\text{C}$) and tactile stimuli ($>512\text{ mN}$; fig. 1b, black dots). Furthermore, the patient did not feel cold pain ($\Delta 32^\circ\text{C}$), heat pain ($\Delta 20^\circ\text{C}$) and pin-prick pain ($>512\text{ mN}$). Vibration detection threshold (VDT) and pressure pain threshold (PPT) were increased (VDT: 3.3/8, mean of three tests using a tuning fork; PPT: 1,009 kPa). There were no other neurological signs except mild paresis of the left hand. Three years and five months after the stroke we performed a follow-up study of the patient. The QST profile was now normal and no alterations of sensory thresholds could be detected (fig. 1b, light grey dots).

This case illustrates that infarction of S1 may indeed lead to impaired pain perception consistent with a role for S1 in pain processing. Basically, based on findings from functional imaging studies, the neuronal matrix underlying the human pain experience is a complex cortical network mainly consisting of S1, secondary somatosensory cortices, insula, anterior cingulate cortex and prefrontal cortices [3–5]. These cortical areas process different aspects of pain, like sensory-discriminative or affective motivational dimensions [5]. Nevertheless, the role of S1 in pain processing per se is under extensive debate, as there are several imaging studies that did not show S1 activation to painful stimulation. A meta-analysis of fMRI and PET studies reported pain-induced

S1 activation in only 36 of 65 studies which investigated normal subjects [3]. It can be speculated that some of these S1 activations in fMRI and PET studies are a result of tactile afferents simultaneously activated by the applied noxious stimulus [3, 4]. On the other hand, in animals, nociceptive projections from thalamus to S1 and specific nociceptive neurons in S1 have been demonstrated [7]. Also studies using magnetoencephalography (MEG) demonstrated S1 activations [8]. MEG studies suggest that processing of noxious stimuli occurs in more medial parts of S1, presumably area 1 [8]. This is in line with the more medially located S1 infarction in our patient (fig. 1a), suggesting that the lacunar infarction includes area 1 of S1. Nevertheless, exact fine-mapping is not possible due to the resolution of diffusion-weighted MR images. One advantage of the present case report is that the time window between the onset of the stroke and the first QST investigation was very small ($<1\text{ day}$). This very acute stage might explain why the psychophysical findings were so pronounced. Post-lesional plasticity might have prevented such findings in earlier studies where other parts of the cortical pain network might have adopted functions of S1 [1, 2, 9]. This assumption would be in line with our study, as the QST profile showed no alterations in the follow-up investigation. In summary, the present case report argues in favour of a role of S1 in pain processing.

Acknowledgement

Supported by the German Research Network 'Neuropathic Pain' (German Federal Ministry of Education and Research, BMBF) and the German Research Foundation (DFG; KFO 130).

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Fig. 1. a Diffusion-weighted MR image on day 1 showing the acute ischaemic infarct, which was restricted to the hand region of the right sensorimotor cortex. The central sulcus is indicated by the dotted line. **b** Z-sensory profile of the patient based on normative data of the quantitative sensory database of the German Research Network 'Neuropathic Pain' [5]. In the first investigation (acute stroke; black dots) the profile showed a predominant loss of sensory functions at the affected hand for cold detection thresholds (CDT), warm detection thresholds (WDT), cold pain threshold (CPT), heat pain threshold (HPT), mechanical detection thresh-

old (MDT), mechanical pain threshold (MPT), VDT and PPT. These thresholds were well outside the 95% confidence interval of the distribution of healthy subjects (grey zone). The patient had no dynamic mechanical allodynia (All). Three years and five months after the stroke we performed a follow-up study of the patient. The QST profile was normal and no alterations of sensory thresholds could be detected (light grey dots). Z score: number of standard deviations between patient data and group-specific mean values of a normative data base obtained from QST examinations in healthy subjects [6].