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LETTER TO THE EDITOR

Pseudo carpal tunnel syndrome due to incomplete pure sensory stroke

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Dear Editor,

Pure sensory stroke (PSS) is a clinical type of lacunar infarction. Thalamic lacunar infarction is the most frequent cause of PSS although brainstem lacunar infarction may also cause the condition [1–3]. In PSS cases, sensory symptoms are typically observed in half of the body including the face [1]. Localized distribution of sensory impairment, such as cheiro-oral syndrome, has also been described. In extreme cases, sensory symptoms are confined to the unilateral upper limb or hand [2]. Such cases may have a risk of misdiagnosis as peripheral neuropathy. We report a patient with lacunar infarction of the pons who presented with symptoms and signs closely resembling carpal tunnel syndrome (CTS). Median nerve somatosensory-evoked potentials (SEP) were useful for localization.

Case report

A 79-year-old right-handed man, who had been treated for hypertension and hyperuricemia, became aware of numbness in the left hand while watching television. He visited a general practitioner the next day because the symptom did not improve. There were no abnormal findings on a head CT. The general practitioner referred him to our neurology clinic on the same day and booked a head MRI examination several days later.

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Neurological examinations revealed decreased perception of pain and light touch from the left thumb to the radial side of the ring finger. The distribution of numbness and objective sensory disturbance closely resembled that of CTS (Fig. 1a). Careful interview and examination of perioral sensations did not reveal any abnormalities. There was no weakness, and tendon reflexes were normal. Limb or truncal ataxia was not observed. Tinel's sign was positive on the left side, and Phalen's test was negative on both sides. History taking did not reveal morning worsening, nocturnal awakening, exacerbation of symptoms by specific activities, or flick sign.

CTS was suspected from the neurological findings, and nerve conduction studies (NCS) were performed 1 week later. NCS revealed CTS findings on both sides that were worse on the right side, the asymptomatic side. Distal motor latency (DML) and amplitude of sensory nerve action potential (SNAP) of the median nerve were 3.9 ms and 18.3 μV on the right side and 3.7 ms and 20.8 μV on the left side, respectively (upper normal limit of our institution: DML, 3.8 ms; amplitude of SNAP, 9.2 μV). A comparison test, which compares the sensory conduction of the median and the radial nerves in the thumb, showed bilaterally abnormal results. These were again worse on the right side (difference in peak latency of SNAP between median and radial nerves were 1.06 ms on the right side and 0.48 ms on the left side; upper normal limit of our institution, 0.46 ms). The ring-finger method, which compares the sensory conduction of the median and the ulnar nerves in the ring finger, was not informative because of the cubital tunnel syndrome on both sides. These CTS-like findings were interpreted as subclinical and incidental because the side did not coincide with the clinical severity. Subsequently, median nerve SEP was performed for localizing the lesion, which revealed that the N20 amplitude of the left side was considerably lower than that of the contralateral side, despite normal far-field potentials up to P13/14 (Fig. 1b). Based on this finding, intracranial lesions were suspected. Vibration and position senses were examined at this point for the first time, and no abnormalities were observed.

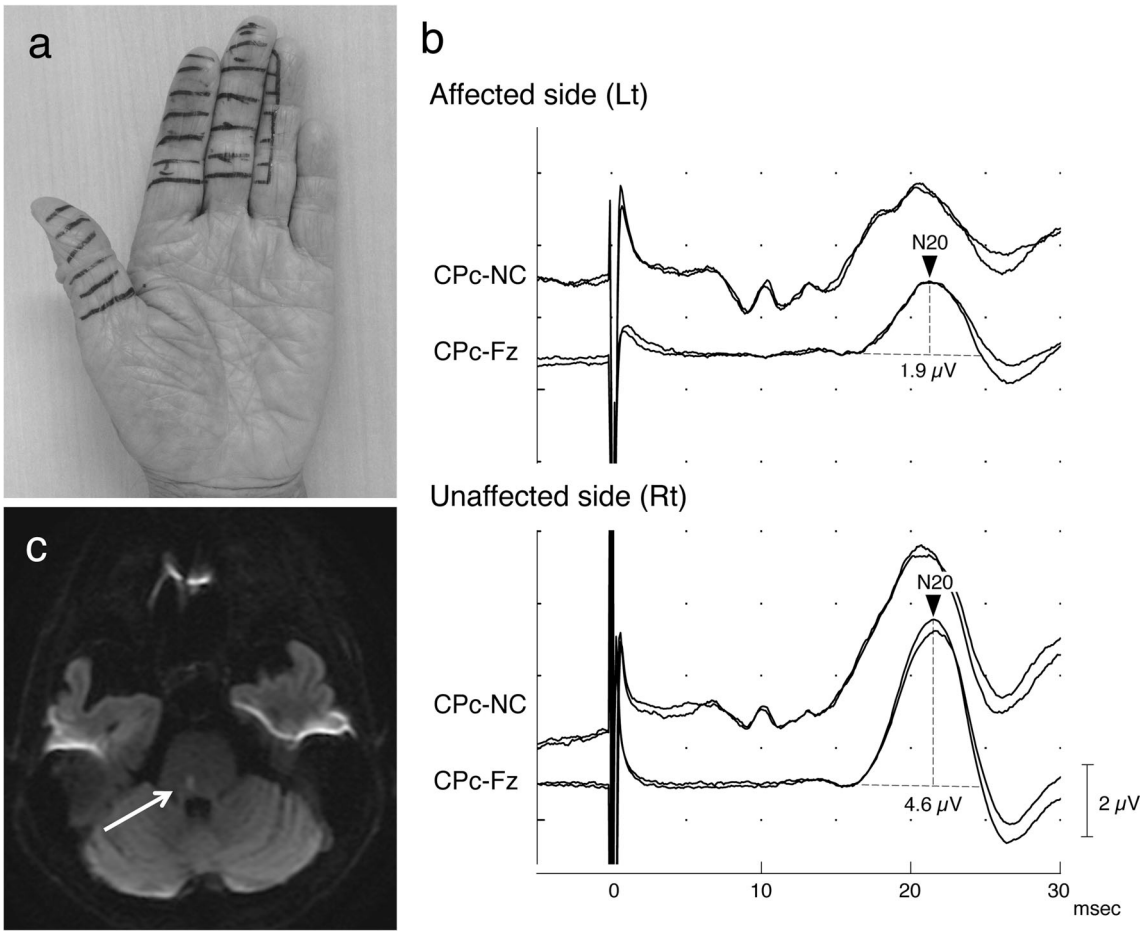

Fig. 1 Distribution of numbness and hypesthesia (**a**). Median nerve SEP findings in this patient. The amplitude of N20 on the affected side was less than half (41%) of that on the unaffected side (**b**). Axial diffusion-weighted imaging of MRI showing a high-intensity area on the medial side of the right pontine tegmentum (**c**)

After neurophysiological examinations and evaluations, we checked the head MRI ordered by his family doctor and found a lacunar infarction in the right pontine tegmentum in diffusion-weighted imaging (Fig. 1c). This was not recognized as the responsible lesion by either his family doctor or the radiologist. An antiplatelet drug was prescribed, and sensory impairment disappeared in a few months.

Discussion

In the present case, the distribution of sensory disturbance was very similar to that of CTS. Rare cases of stroke presenting with sensory impairment confined to the unilateral hand have been described as already mentioned [2]. However, there has been no previous report of stroke that presented with CTS-like sensory impairment alone. In such a case, the patient may be easily misdiagnosed as peripheral neuropathy. In fact, we initially suspected CTS, although the sudden onset seemed atypical. Furthermore, subclinical CTS findings were observed in NCS on both sides. If stroke is not suspected, head imaging tests may not have been conducted. The discordance of the side of the greater NCS abnormality and the clinical symptoms led us to further evaluation by median nerve SEP, which contributed to the correct localization.

Head MRI may not detect a small brainstem stroke in its earliest phase [4], and median nerve SEP may be useful for localization in such cases. There are a few previous reports that documented the reduction of the N20 amplitude in median nerve SEP on the affected side in PSS cases due to brainstem infarction [3, 5]. In the present case also, median nerve SEP findings, i.e., normal P13/14 and reduced N20 amplitude, localized the lesion at the intracranial portion, including medial lemniscus, and reevaluation of head MRI revealed a small lesion at the medial lemniscus, which had been overlooked until then. Reduced N20 amplitude and not prolonged latency may be due to the axonal nature of the lesion involved in the stroke.

Clinicians should consider the possibility of PSS in a patient presenting with acute-onset CTS-like symptoms and signs. Median nerve SEP may help localization in such patients.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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