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Letters to the Editor

Digital acroparesthesia caused by pontine infarction

Keywords: Sensory stroke; Pontine infarction

Dear Sir,

Digital acroparesthesia, a sensory abnormality confined to distal fingers, has been rarely reported in association with lesions of thalamus and brainstem [1-4].

A hypertensive 43-year-old man developed sudden dizziness and diplopia followed by paresthesia restricted to the left fingers. He had a one-and-half syndrome and decreased position and vibration senses in the left whole fingers. Temperature and pinprick sensation, stereognosis and graphesthesia were normal. Brain MRI showed ischemic lesion in the right paramedian pontine tegmentum (Fig. 1a). A 42year-old heavy smoking female was admitted with sudden heaviness of her left limbs. On neurological examination, she had only a paresthesia restricted to the left fingers. The pinprick and temperature sensation were mildly decreased in the left hand. Position and vibration were normally perceived. Brain MRI showed right lateral tegmental pontine infarction (Fig. 1b).

In our patients, medial lemniscal and spinothalamic tract sensory deficits were caused by pontine paramedian and lateral tegmental infarction respectively. Most restricted sensory syndromes following stroke present a pattern of cheiro-oral, cheiro-pedal, cheiro-oral-pedal or hemisensory syndrome. Despite many case reports of restricted sensory syndrome, however, pure digital acroparesthesia caused by pontine infarction is extremely rare. Because somatotopic representation is compact in the long tracts, it is surprising that digital acroparesthesia would be attributable to stroke localized to the pons. In the pons, the medial lemniscus is located in the dorsal tegmentum, whereas the spinothalamic tract is localized in the lateral-posterior side of the medial lemniscus. The sensory pathways from the face, arm and leg are located from the medial to the lateral side in the medial lemniscus

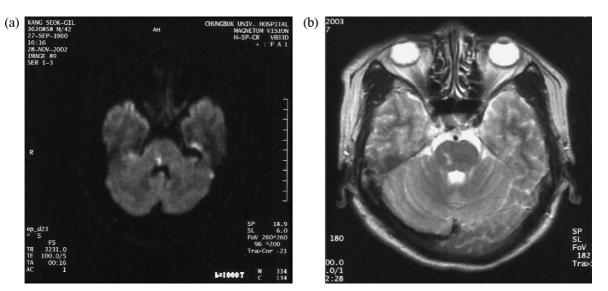


Fig. 1. Axial MRI scans of two patients. The diffusion-weighted imaging reveals a small area of high-signal intensity in the right paramedian pontine tegmentum (a) and the T2-weighted imaging reveals a high-signal intensity in the right lateral pontine tegmentum (b).

of the pons. Likewise, in the spinothalamic tract, the ascending fibers from the lower limbs are located laterally on the outside. The two sensory tracts approach one another as they ascend in the brainstem [5]. Digital paresthesia can be explained by the anatomic proximity of sensory fibers from acral parts of the body. Additionally, relatively small pontine lesions in our patients might be sufficient to explain the sparing of proximal limbs and even the face. However, the anatomic-proximity concept cannot always explain the sparing of proximal limbs and more frequent involvement of fingers. Other suggestions are the low-threshold concept based upon disproportionately large representation areas for acral parts and the presence of an uncrossed, polysynaptic ascending spinal cord sensory pathway from the trunk and proximal limbs [1]. Although, extremely rare, a very small lacune strategically located can make a pure digital acroparesthesia which may show tract-specific sensory abnormalities.

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Serum and cerebrospinal fluid uric acid levels in multiple sclerosis patients

Keywords: Multiple sclerosis; Uric acid; Cerebrospinal fluid

Dear Sir,

There is accumulating evidence that nitric oxide (NO) and its oxidizing congeners, particularly peroxynitrite (PN), contribute to the pathogenesis of MS [1,2]. Urate (UA), a

metabolic product present at relatively high levels in humans is a selective scavenger of radicals formed by PN [3]. This has led to the hypothesis that UA may offer natural protection against certain aspects of the pathophysiology of diseases like MS. To provide insight into this possibility a number of groups have studied serum UA levels in a variety of neurodegenerative diseases including MS. UA levels in the sera of MS patients are generally described as lower than age- and sex-matched controls [4–7] but this is not always the case [8] and may depend upon the stage of the disease [2,6]. In normal individuals UA does not readily cross the blood-brain barrier (BBB) and CSF UA levels are at least 10-fold lower than those of serum. However, studies in animal models of MS have demonstrated that UA levels in the CNS could become elevated as a consequence of the BBB disruption associated with the disease [9]. In prior studies, UA levels in the CSF of MS patients have been reported as either elevated [10] or equal to those of controls [11]. In the only study where serum and CSF UA levels were studied, both were noted as being equivalent to those of controls [11].

To examine this controversial issue further we have compared serum and CSF UA levels in 12 patients with MS and 10 age and gender-matched controls. The patients were diagnosed with MS according to the "McDonald" criteria. Control samples were obtained from patients as part of the initial clinical assessment for suspected neurological disorders which were subsequently ruled out. UA levels were determined with a commercially available enzymatic assay (Uricase-PAP) according to the manufacture's protocol (Diagnosticum Rt, Budapest, Hungary). The Students' t-test and the Mann-Whitney U-test were used to statistically assess the differences in UA levels between the groups. The MS patients were found to have significantly (p < 0.01) lower mean (M) and mean rank (MR) serum ($M = 3.32 \pm 0.31$ mg/dl, MR = 7.33) and CSF $(M = 0.15 \pm 0.03 \text{ mg/dl}, \text{MR} = 8.17)$ UA levels than those of control serum ($M = 5.52 \pm 0.27$ mg/dl, MR = 16.5 and CSF $(M = 0.24 \pm 0.03 \text{ mg/dl}, \text{ MR} = 15.5)$. These results indicate that when UA levels are low in the sera of MS patients they are also reduced in CSF if the BBB is intact. The implication is that there is reduced protection against peroxynitritedependent radicals both systemically and in the CNS of some MS patients.

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