Stroke and restricted sensory syndromes

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Abstract. There have been sporadic case reports of a restricted sensory syndrome caused by stroke, most often as a cheiro-oral syndrome. We describe 14 patients with stroke who showed various restricted sensory syndromes and correlated their symptoms with the radiological findings. Twelve had small infarcts and two had haemorrhages; six had lesions in the posterolateral thalamus, five in the pontine tegmentum, one in the lenticulocapsular area, one in the frontoparietal subcortex, and one in the frontal white matter. Sensory abnormalities were in the perioral area, hands, fingers, feet and toes, in various combinations. Two patients with pontine stroke had bilateral sensory abnormalities associated with paramedian lesions. Thus, strokes in the sensory pathways can cause various restricted sensory syndromes of which 'cheiro-oral syndrome' is only one variant. Magnetic resonance imaging is of interest in the clinicoradiological correlation, and helps elucidate the somatotopic pattern of the human sensory pathways.

Key words: Stroke – Restricted – Sensory syndromes – Cheiro-oral syndrome

Sensory symptoms secondary to stroke can be focal and discrete. A well-known restricted sensory syndrome is the so-called 'cheiro-oral' syndrome with sensory changes in the contralateral hand and perioral area. Although it was first reported in a patient with a lesion in the parietal lobe [1], minor strokes involving the thalamus [2-9], brain stem [8, 10-14], and corona radiata [15, 16] have subsequently been reported as causing this syndrome. However, the 'cheiro-oral' syndrome is not the only restricted sensory syndrome caused by stroke. We recently reported three patients with midbrain lesions producing restricted trigeminal sensory changes probably due to focal involvement of the trigeminothalamic sensory tract [17]. In the present study, we describe 14 patients with stroke who showed various restricted sensory syndromes, correlating clinical features with imaging.

Subjects and methods

We studied 14 patients who had suffered a stroke with restricted sensory changes in whom computed tomography (CT) or magnetic resonance imaging (MRI) of the head demonstrated lesions considered appropriate to the clinical findings; all underwent neurological examination. Four were studied by CT and 10 by MRI at 1.5 T: axial T2-(TR/TE 2500/80) and T1-(TR-TE 600/20) weighted and sagittal T1-weighted images were generated with a slice thickness of 5 or 7 mm.

Case reports (Table 1)

Case 1

A 55-year-old diabetic man developed a tingling sensation on the left side of his body, and soon complained of uncomfortable paraesthesiae in the left perioral area, hand and foot. CT two weeks later showed a small infarct in the right posterolateral thalamus. About a month later, he developed choreoathetoid movements of his left hand and fingers. Two years later, neurological examination was normal except for painful paraesthesiae in the areas mentioned, and CT showed the same lesion as before (Fig. 1).

Case 2

A 49-year-old hypertensive women suddenly felt numbness over the left face and forearm. Neurological examination two days later was normal except for the paraesthesiae over the left perioral area and the tips of the left thumb and index finger. The paraesthesiae continued until seven days later when T2-weighted MRI showed a small infarct in the right posterolateral thalamus, more clearly identified with gadolinium enhancement.
Case 3

A 57-year-old hypertensive woman noted a lack of cold sensation in her left arm during bathing. When seen four months later she complained of burning para- and dysaesthesiae in the left perioral area, hard palate, left hand and fingers, and all the left toes except the fifth. The painful dysaesthesiae was more severe in the first three fingers of the hand, and in the first and second toes. MRI showed a small lacune in the posterolateral part of the right thalamus.

Case 6

A 54-year-old diabetic man developed chest pain followed by confusion and visual hallucinations. He became alert two days later, but had intermittent visual hallucinations in the right visual field. Neurological examination showed decreased recent memory, right homonymous hemianopia, mild left hemiparesis, and mildly decreased pinprick sensation on the left lower face. He complained of numbness in the left perioral area and distally in his left fingers, worse in the first three. CT showed multiple infarcts in both occipital areas and a small lacune in the right thalamocapsular region.

Case 7

A 88-year-old hypertensive man developed dizziness, dysarthria and diplopia. On right lateral gaze, there was limited adduction of the left eye with horizontal nystagmus in the right. Motor and sensory examination was normal except for the mild hypoesthesia of the right face. He was numb over a small area on the right upper lip and the distal volar surface of the left 5th finger. Pinprick and vibration sense was decreased in the tip of that finger, but position sense was normal. MRI showed a small infarct in the pons (Fig. 2). Four months later the paraesthesiae in the finger had disappeared, but his ocular motor abnormality and numb right upper lip remained.

Case 8

A 56-year-old hypertensive man suddenly felt dizzy and then collapsed, after which he complained of diplopia, left sided weakness and tingling sensation. On admission three months later, examination showed extropia of the left eye on forward gaze and marked limitation of horizontal gaze of both eyes except for abduction of the left, with which nystagmus of the left eye occurred. He had a mild left central facial palsy and hemiparesis. He complained of a continuous tingling sensation around his mouth, on the hard palate, and over the left palm, the tips of the right fingers and the left foot. Decreased pinprick sensation was found over the lower face, hand and foot while vibration and position sense was decreased only on the dorsum of the left foot. MRI demonstrated an old haemorrhage in the pontine tegmentum (Fig. 3).

Case 9

A 39-year-old hypertensive woman developed dysarthria, left hemiparesis and hemihypoesthesiae. She had an episode of heart failure three years previously. MRI showed pontine infarcts, most prominent in the right pontine tegmentum (Fig. 4a). Her neurological deficit gradually improved, but a mild left hemiparesis remained. Six months later she developed gait disturbance and paraesthesiae on the right palm and lower leg. She showed a mild quadriparesis worse on the left, hyperactive reflexes in all limbs, and bilateral extensor plantar responses, with decreased pinprick sensation on both palms, fingers and feet. In the right hand, the sensory abnormality was more severe in the fingers on ulnar side. Vibration and position sense was decreased in both feet. MRI showed an old infarct in the right pontine tegmentum and a new one in the left upper pons (Fig. 4b). Laboratory tests showed positive antinuclear antibody at 1:160, and positive lupus anticoagulant. Four vessel angiography was normal.