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Isolated Dorsal Midbrain Infarct: An Uncommon Cause of Pure Sensory Stroke

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Introduction

Occlusion of branches of the thalamogeniculate arteries supplying the thalamic somatosensory nuclei is responsible for the vast majority of strokes presenting with the clinical syndrome termed by Fisher as ‘pure sensory stroke’ (isolated hemihypaesthesia or paraesthesia involving the face, trunk, arm and leg) [1]. Occasionally, pure sensory stroke can be caused by a lateral tegmental pontine or medullary infarct [2].

Brain ischaemia limited to the mesencephalon is uncommon, accounting for 0.2–2.3% of total admitted ischaemic strokes [2–5]. The anteromedial territory, supplied by the direct perforators of the basilar artery, is most frequently affected in patients with isolated midbrain infarct, whose neurological picture is dominated by oculomotor disturbances [2–5]. We report the uncommon case of a patient presenting with the clinical syndrome of pure sensory stroke due to an infarction limited to the dorsal midbrain territory.

Case Description

A 60-year-old man, with a history of hypercholesterolaemia and smoking, awakened with numbness on the right part of the inner mouth and the right arm and leg. Symptoms persisted over 3 days, when he consulted the family physician, who suspected stroke and referred him to our hospital for further evaluation. On examination the patient was alert, with normal and symmetrical tendon reflexes, while both plantar responses were flexor. A right hemihypaesthesia involving the face, trunk, arm and leg was manifest. No ataxia, dysarthria, limb weakness, or ocular motor manifestations were present.

MRI of the brain was then performed on the fourth day after ictus. A hyperintense lesion of ischaemic origin located in the left dorsal portion of the midbrain was demonstrated on fluid attenuated inversion recovery (FLAIR, fig. 1a), T₂-, as well as diffusion-weighted images. The infarct was obviously affecting structures localised in the dorsal (superior colliculus, periaqueductal grey matter) and the dorsolateral (medial lemniscus and lateral spinothalamic tract) midbrain territory, supplied by the superior cerebellar artery. MR angiographic images in the time-of-flight technique and ultrasound of the carotid and vertebral arteries were normal. Transthoracic echocardiography, 24-hour blood pressure monitoring and Holter electrocardiographic monitoring revealed no abnormalities. Visual- and auditory-evoked potentials as well as extensive blood coagulation studies were unremarkable.

The patient was diagnosed as having a brainstem lacunar stroke and was discharged on aspirin and lipid-lowering medication. A slight improvement of his sensory symptoms was reported at the follow-up evaluation 6 months later, while a new MRI revealed a substantial decrease in the extent of the hyperintense lesion on the FLAIR images (fig. 1b).

Fig. 1. A hyperintense lesion in the dorsal territory of the left midbrain is visible on the FLAIR MR images (**a**) performed on the fourth day after ictus. Six months later the same lesion is smaller on FLAIR (**b**) images as expected in the chronic stadium of an ischaemic lesion.

Fig. 2. Axial schematic diagram of midbrain depicting the anatomical structures and the different locations (A, anteromedial, B, anterolateral, C, lateral, D, dorsal) of midbrain infarctions.

Discussion

To our knowledge, no case of isolated dorsal midbrain infarction has been reported previously. The investigators of the Lausanne [3] and the Ege Stroke Registry [4] described 22 and 9 patients with infarct limited to the mesencephalon respectively. More recently, the clinical, radiological and pathophysiological findings of 40 patients with pure midbrain infarction were reported [5]. However, the authors did not observe any subject with lesion restricted to the dorsal midbrain. This territory is supplied by different arteries arising from the collicular artery, which gives rise to a network of small arteries also supplied by branches of the posterior cerebral artery. Sometimes the superior cerebellar artery also participates in the supply of the inferior colliculus. Furthermore, the same authors indicated that according to their experience, infarcts in this area were invariably associated with the concomitant involvement of the cerebellum [5].

In our case, the clinical features were characterised by sensory deficits caused by the involvement of the dorsolaterally located lemniscal and spinothalamic sensory fibres (fig. 2). Although the infarct affected the superior colliculus and the periaqueductal grey matter unilaterally as demonstrated on MRI, these lesions did not correspond to any clinically overt neurological deficit. Pure sensory stroke is most frequently associated with thalamic lacunes [1], or occasionally with lesions located in the lateral pontine tegmentum, involving the medial lemniscus and the lateral spinothalamic tracts in the rostral pons [2]. Two cases of lateral tegmental midbrain haemorrhages limited to the spinothalamic pathways have been described as extremely rare causes of pure sensory stroke [6, 7]. In addition, Kim and Kim [5] identified 2 patients with ischaemic lesions restricted to the lateral midbrain presenting with isolated sensory symptoms. Small vessel disease was the pathogenic mechanism of infarction in 1 case, while atherothrombotic large vessel disease was categorized as the pathogenic aetiology in the other. In the absence of any evidence of stenosis or occlusion of the large vessels and of any emboligenic heart disease, we considered small vessel disease as the most plausible cause of stroke in our patient.

In conclusion, the present report highlights the diversity of the topography of the underlying ischaemic lesions in patients presenting with pure sensory stroke. Therefore, although its incidence is extremely low, isolated posterior midbrain infarction should be considered as an infrequent cause of pure sensory stroke.

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Beauty Parlor Stroke Syndrome

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A 63-year-old woman visited her beauty parlor to have her hair cut. During shampooing with her head hanging backwards into a hair washbasin she developed sudden dizziness, nausea and started vomiting. The alarmed paramedics assumed a gastrointestinal disorder, and she was initially admitted to the department of gastroenterology. As symptoms persisted for 2 days, neurological advice was sought. The neurological examination revealed nystagmus at forced lateral view bilaterally, slight left-sided ataxia of both limbs and she was prone to fall to the left side in the Romberg test. The vascular risk factors were sufficiently treated diabetes type II and arterial hypertension. In our routine stroke workup which is based on the EUSI guidelines [1], no further pathological findings were detected, in particular no signs of cardioembolism. MRI of the brain showed an ischemic infarction in the territory of the left posterior inferior cerebellar artery (fig. 1a). MR angiography (fig. 1b) showed a smaller lumen of the left vertebral artery compared to the right but without signs of arterial dissection or major arteriosclerosis.

In our patient we diagnosed beauty parlor stroke syndrome, a term proposed by Weintraub in 1993 [2]. Pathophysiologically, acute arterial dissection is considered to be a major cause [3, 4]. In these cases, patients often reported about pain in the neck and a