Spontaneous hemiballism and disappearance of parkinsonism following contralateral lenticular lacunar infarct.

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Hemiballism was observed in a 77-year-old woman with Parkinson disease after a contralateral lenticular infarct without apparent involvement of the subthalamic nucleus. Parkinsonian signs ipsilateral to the hemiballism remained abolished despite subsequent nearly complete recovery from the hyperkinesias. It is argued that clinical events were due to a single ischemic lesion of the putamen-pallidum complex.

Key-Words: Hemiballism — Parkinson disease — lacunar infarct — basal ganglia

Introduction

Spontaneous hemiballism has been described following pathologically or radiologically documented lesions of the caudate nucleus, putamen, globus pallidus and thalamus without involvement of the subthalamic nucleus (SThN) [6, 8, 9, 11].

In parkinsonian patients hemiballism has been reported as a postoperative complication following stereotaxic surgery [10]. We now describe a patient with Parkinson’s disease (PD) who developed left hemiballism associated with a CT scan documented ischemic lesion of the contralateral lenticular nucleus. In this patient the parkinsonian signs were abolished concurrently with the onset of the hemiballism, and did not reappear despite nearly complete recovery from the hyperkinesias.

Case report

A 77-year-old righthanded diabetic hypertensive woman with PD, was admitted to the Institute of Neurology because of involuntary movements of the face and left limbs of sudden onset. At the age of 76, she developed bilateral resting tremor in the upper limbs, associated with decrease of fine motility in both hands. At that time examination revealed additional bilateral loss of armswing, hypomimia, rigidity and gait difficulty. PD was diagnosed and L-Dopa (300 mg/day plus benserazide) and trihexyphenidyl (6 mg/day) treatment was started with benefit. On admission, she was alert and speech was normal. There were violent uncontrollable rapid and large amplitude proximal movements of the left limbs, with occasional distal choreiform movements and grimacing. Mild weakness and hyperreflexia were present in the left extremities but both plantar responses were flexor. A fine resting tremor and slight rigidity were present in the right limbs. Sensation was normal. The carotid pulses were symmetric, without bruits. General examination was unremarkable, apart from a blood pressure of 200/120 mmHg. Doppler recording with echotomography of the arteries of the neck, and blood test results...
were normal. ECG showed left bundle branch block. CT scan with contrast showed a small, hypodense nonenhancing lesion involving the anterior part of the pallidum, putamen and the anterior limb of the internal capsule. No abnormalities were found in lower sections through the subthalamic area. Diffuse cortical atrophy and ventricular enlargement were also shown (Fig. 1). Antiparkinsonian treatment was stopped and she was treated with (2 mg/day) without benefit; the association of clobazam (20 mg/day) produced a sustained improvement of the hyperkinesias. A few months later, neurological examination revealed slight distal choreiform movements of the left lower extremity occasionally involving the proximal muscles of the same limb, absence of resting tremor and rigidity. Tendon reflexes were symmetric, both plantar responses were flexor. Repeat CT scan remained unchanged. The residual hyperkinesias did not change when drug treatment was stopped. Two years later, the patient presented only with slight distal choreiform movements of the left lower limb, resting tremor, bradykinesia and rigidity of the right side.

Discussion

In this patient the clinical features and CT scan findings are indicative for a lacunar infarct in the territory supplied by perforating branches of the middle cerebral artery involving the basal ganglia contralateral to the hyperkinesias [1, 3]. The peculiar features of this patient are:
- the hemiballism developed contralateral to the lacunar infarct in the right basal ganglia without apparent involvement of the SThN;
- the parkinsonian signs ipsilateral to the hemiballism were suppressed and did not reappear despite partial recovery from the hyperkinesias. Spontaneous hemiballism following contralateral lesions of the neostriatum and globus pallidus without involvement of the SThN, has been documented by anatomical and recently radiological studies [6, 9, 11]. Although it is possible that a small lacunar infarction in the SThN could have escaped detection, in this patient the concordance between the unilateral dyskinesia and the contralateral CT scan abnormalities in the lenticular nucleus suggests a casual relationship. However, it is difficult to localize hemiballism to a particular area of the basal ganglia because of involvement of more than one structure i.e. putamen and globus pallidus.

The exact pathophysiological mechanism of spontaneous hemiballism remains uncertain. Common interpretation is based on disinhibition of SThN or of other deep structures, as putamen, caudate nucleus and thalamus, upon the globus pallidus, following lesion of the above mentioned structures or interruption of their neuronal circuits [4, 8, 11]. To our knowledge, spontaneous hemiballism has not previously been reported in patients with PD. In this patient the diffuse anatomical and biochemical abnormalities which are present in PD may well have constituted a background predisposing to the development of the hyperkinesias, by analogy with the mechanism hypothesized for hemiballism complicating stereotaxic surgery for PD [5, 10].

The second interesting feature of this patient is the disappearance of the parkinsonian signs contralateral to the deep hypodense lesion seen on CT scan. Apart from a recent report, in which disappearance of the parkinsonian signs has been described following spontaneous vascular "thalamotomy" [2], the suppression of parkinsonian features, mainly tremor, had been observed following hemiparesis and stereotaxic surgery. In the latter the main targets are usually the ventrolateral nucleus of the thalamus and less often the subthalamic white matter below the ventrolateral nucleus, the pallidothalamic pathways in Forel's field and the medial part of the pallidum, origin of the pallidofugal fibers [7]. In our patient unilateral improvement of parkinsonian signs definitely did