The shaking limb – a lacunar syndrome

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Summary: A 76 year old man with shaking movements of the fingers, weakness of the arm and lacunar infarctions on computed tomographic scan is described. The shaking limb should be included in the group of lacunar syndromes.

Introduction

Unilateral involuntary movements or limb shaking are known to occur with cerebral ischaemia as a manifestation of a carotid transient ischaemic attack (TIA). Many such patients have small infarcts on computed tomographic (CT) scan in the appropriate hemisphere.1,2

We wish to describe a patient with shaking movements of the hand and weakness of the arm lasting for three days. CT scan disclosed two small low density areas. Although precise anatomical localization was not possible it could well be that this patient had a lacune in a site which gave rise to involuntary movements.

Case report

A 76 year old man presented two hours after the sudden onset of uncontrollable rapid shaking movements of the right hand. Immediately before the symptoms, he had been cutting a hedge with a pair of shears for an hour. There was no accompanying impairment of consciousness or 'march' of symptoms. There was no history of similar events, convulsive phenomena or migrainous headache. He was known to be hypertensive with ischaemic heart disease, atrial fibrillation and cardiac failure.

Examination of the central nervous system revealed coarse, irregular movements of the little, ring and middle fingers of the right hand with flexion at the wrist. The movements were rapid at about 3-5 oscillations per second and were not increased on hyperextending the wrist and fingers. He had weakness of the right hand and arm. The reflexes were brisk on the right side. There was no cardiac decompensation or audible carotid bruits. He was in atrial fibrillation and was normotensive.

On investigations, haematological and biochemical profiles were normal. X-ray of the cervical spine showed narrowing of the C4-C5 and C6-C7 disc spaces. Computerized tomography the following day showed two small lacunar infarcts, one in the left posterior parietal region and another deep in the left frontal region. Duplex scan and ultrasonography revealed a calcified plaque at the bifurcation of the right common carotid artery extending into the right internal carotid artery. Haemodynamic changes were consistent with 40-60% stenosis of the latter.

An electroencephalogram 3 weeks after presentation showed an abnormal recording with generalized slowing and a suggestion of a left sided focal lesion.

A provisional diagnosis of partial epilepsy (epilepsia partialis continua) was made and he was treated with clonazepam and anticoagulated with intravenous heparin for a week. The movements decreased in intensity and ceased by the third day with complete return of power in the hand and arm. He had no further symptoms at 3-month follow-up.

Discussion

Our patient had continuous movements of his right hand with weakness lasting 3 days and the CT scan demonstrated two lacunar infarcts, one in the posterior parietal and the other in the frontal region on the contralateral side. Three of the eight patients described by Baquis et al.1 had small infarcts in the frontal, central semiovale and parietal regions, all on the contralateral side of the movements. Seven other patients with repetitive movements had small infarcts and in six of them these infarcts were in the appropriate hemisphere.2

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The recognition of lacunar syndromes has come from careful correlation of clinical and anatomical data and at least 20 different lacunar syndromes have been described. The clinical pattern is one of gradual or stuttering onset in about one third of the patients. Occasionally there is a prior transient ischaemic attack. This patient could be labelled as having had a protracted transient ischaemic attack (PTIA) or a reversible ischaemic neurological deficit (RIND).

The accepted concept is that in TIA focal cerebral ischaemia is not accompanied by infarction and this is supported by computerized tomography, although there is evidence to the contrary. Calandre et al. reported the incidence of focal ischaemic lesions on CT as 25% in TIA and RIND and 35% is stroke with minimum residuum (SMR). Lacunar infarctions have been found to occur with TIA and with RIND. Hemiballism has been included in this group of lacunar syndromes and has been related to lacunar infarcts in the subthalamic nucleus. Mas et al. described a patient with reversible hemiballism due to a lacunar infarct in the lateral part of the contralateral lenticular nucleus which was documented on the CT scan.

Lacunar infarctions are usually not considered to give rise to epilepsy. These lacunes are more indicative of a widespread cerebral vascular disease rather than that directly responsible for the epilepsy. The EEG in our patient showed an abnormal recording with suggestion of a focal lesion on the contralateral side. The movements however lacked the features of focal epilepsy and were very similar to those described by Baquis et al. In their cases the EEG showed slowed activity but no epileptiform patterns. However, a seizure mechanism, either primary or related to cerebral ischaemia, cannot be ruled out.

A lacunar infarct can result from a small embolic particle of thrombus from the heart or large artery. Small lacunar infarcts most often result from occlusion by lipohyalinosis. According to Hachinski et al., one third of the patients with ischaemic stroke have evidence of both a potential cardio-embolic source and atherosclerotic cerebrovascular disease. In about 50% of these patients this leads to diagnostic ambiguity. Limb shaking seems an unusual expression of cerebral ischaemia manifesting as TIA or RIND. Lacunar infarctions have been known to occur with TIA or RIND and limb shaking should be included in the group of lacunar syndromes.

References

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