case report

# Ballismus as a sign of transitional ischemic attack

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A 70-year-old woman presented to our emergency center with a complaint of jerking and twisting movements in her left upper limb and left ankle with deviation of her mouth toward the left. The movements had lasted two minutes and the deviation resolved spontaneously after 30 minutes. She had a history of similar movements five days earlier. During her stay in the emergency center, she experienced the same movements three times. A CT scan without contrast showed a small lesion in the left putamen. Four vessel color Doppler sonography showed a small atheroma plaque in the proximal part of the left internal carotid artery with stenosis less than ten percent. The repeated CT scans revealed progression of the hypodense lesion and the patient developed hemiparesis. In this case, ballismus movements were a cardinal sign for a future stroke and her problem can be considered a recurrent transient ischemic attack or a stroke in evolution.

Chorea or ballismus can be due to cerebral vessel disease, infections, drugs, metabolic abnormalities, neurodegenerative diseases, immunologic diseases, and tumors. It can also occur in nonketotic hyperglycemia in primary diabetes.[1,2] These involuntary movements are explainable with lesions in the subthalamic nucleuses, cerebral cortex, corpus striatum, thalamus and brain stem.[1,3] This case report shows the ballismus movement as a transient ischemic attack.

## CASE

A 70-year-old woman presented to our emergency center with a complaint of jerking and twisting movements, which had occurred about 30 minutes before presentation. The movements had lasted about two minutes. She described movements as jerking and twisting of high amplitude affecting the elbow, shoulder and hand wrist of her left upper limb and also her left ankle. Furthermore, her mouth deviated toward the left and this resolved spontaneously after 30 minutes. She had experienced similar movements without deviation of her mouth and without involvement of her left ankle five days earlier, which had occurred only once. During her stay in the emergency center she experienced three similar attacks, observed by medical personnel. Movements started slowly from her left shoulder and extended to her left hand wrist. Movements then became more rigorous and her left ankle was also involved. Subsequently, the amplitude and rate of movements decreased and finally, movements stopped spontaneously.

The patient had no past medical problems, and she did not use any drugs. On physical examination, she was alert and oriented to time and place. Her lungs were clear to auscultation bilaterally and cardiac examination was normal. Her abdomen was soft to touch without any abnormal findings. Cranial nerves were intact. Muscle strength of the upper and lower extremities was 5/5 and the Babinski sign was absent.

Her vital signs on admission included a blood pressure of 210/140 mm Hg, a pulse rate of 56/minute, a respiratory rate of 16/minute, a body temperature of 36.5 °C and $O_2$ saturation of 95% in room air. Blood glucose was 98 mg/dL by glucometry. Routine laboratory tests, done in the emergency center, were within normal limits (**Table 1**). An electrocardiogram showed a normal sinus rhythm with a heart rate of 52/minute, a normal heart axis and no ST-T changes (**Figure 1**). A brain CT without contrast showed a small hypodense lesion in the left putamen (**Figure 2**). In further evaluations in the neurology ward an electroencephalogram (EEG) there was slowing (7Hz), with

**Table 1.** Laboratory tests of patient in emergency department.

| Test | Result |
|---|---|
| White blood cell count (×1000/mm$^3$) | 9.0 |
| Hemoglobin (mg/dL) | 13.7 |
| Platelets (×1000/mm$^3$) | 218 |
| Blood glucose (mg/dL) | 78 |
| Urea (mg/dL) | 62 |
| Creatinine (mg/dL) | 1.1 |
| Serum sodium (mEq/L) | 144 |
| Serum potassium (mEq/L) | 4.2 |

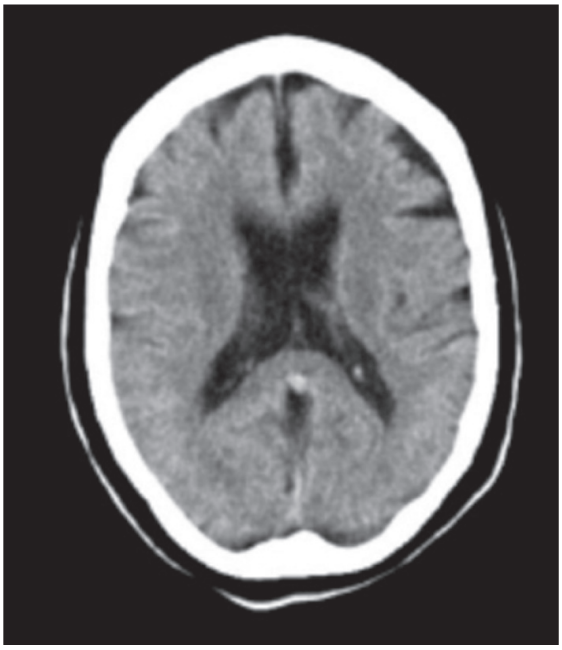

**Figure 1.** Brain CT scan of patient in emergency department.

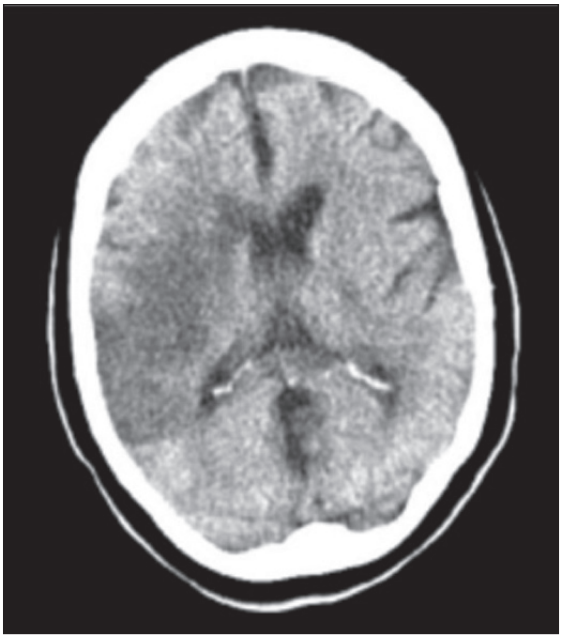

**Figure 2.** Brain CT scan of patient in neurology ward.

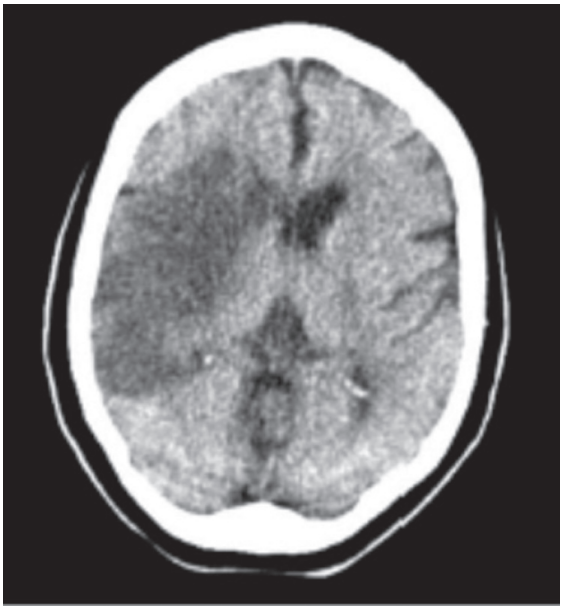

**Figure 3.** Brain CT scan of patient on third day of admission in neurology ward.

no discharge that was epileptic form-like sharp and spike. The patient was oriented and there was no sign of seizure. A four-vessel color Doppler sonography showed a small atheroma plaque in the proximal part of the left internal carotid artery with stenosis less than 10%. The repeated CT scans revealed progression of the hypodense lesion and the patient developed hemiparesis.

## DISCUSSION

Chorea or ballismus can be due to cerebral vessel disease, infections, drugs, metabolic abnormalities, neurodegenerative diseases, immunologic diseases, and tumors. It can also occur in nonketotic hyperglycemia in primary diabetes.[1,2] These involuntary movements are explained by lesions in the subthalamic nuclei, cerebral cortex, corpus striatum, thalamus and brain stem.[1,3] Involvement of cerebral vessels, especially those that affect the subthalamus nuclei, thalamus, corpus striatum and cerebral cortex can generate ballismus manifestations.[1] In this

case, ballismus acted rarely as a cardinal sign of stroke. The case presented by Das et al[4] indicated that the contralateral occlusion of the carotid artery can cause hemiballismus symptoms. Another case was reported due to bilateral carotid artery occlusion.[5] In the case report of Das et al the patient had proximally dominant involuntary movements of the left, upper and lower limbs and in evaluations, MRI showed a very small hemorrhage in the left basal ganglion, and the patient recovered without any neurologic sequel. Our patient developed left hemiparesis after experiencing these movements for several times during five days and also a lacuna in the basal ganglion was evident on her CT.

In the case reported by Gamez et al, the patient presented with ballismus movements and a history of familial amyotrophic lateral sclerosis (ALS) and movements occurred due to involvement of the subthalamus with ALS.[6] In contrast, our patient has no history of neurologic disease. In the case reported by Chaudhary et al the patient was an 11-year-old boy who presented with abnormal facial movements, and evaluations showed the elevated serum phenytoin level as the causative factor of movements.[7] In our report the patient had no history of using drugs, especially anticonvulsant drugs. In the case reported by Kurt et al, the patient was a 77-year-old woman who presented with generalized ballismus movements. She had diabetes, and an MRI showed no abnormal findings. She recovered by administration of insulin and a neuroleptic.[8] However, our patient had no history of medical problems, and her blood glucose was normal on presentation and admission.

The importance of our reported case is that the ballismus movements of the patient were a cardinal sign for her future stroke and a small lacuna was the causative factor for movements and an indicator for progression of the lesion. The ballismus movements, which occurred once several days earlier, can be considered part of her transient ischemic attack. According to the occurrence of movements once at home and three times in the emergency center on the admission day, her problem can be considered a recurrent transient ischemic attack or a stroke in evolution.

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