

# Post-stroke hemiballismus and Contralateral tremor: Report of two cases

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## Abstract

Involuntary abnormal movements may occur as part of the symptomatology of acute stroke or may be delayed or progressive. We report two cases of post-stroke hemiballismus and contralateral tremor. Both patients presented acute hemiballismus. In one of the patients, the tremor started with acute symptomatology and the other was delayed. We discuss the possible pathogenic mechanisms for their movement disorders. To our knowledge, this is the first report of two cases with the unusual presentation of post-stroke hemiballismus and contralateral tremor.

**Key words:** Hemiballismus, Contralateral tremor, Pathophysiology, Stroke.

## Resumen

Movimientos involuntarios anormales pueden presentarse como parte de la sintomatología de la enfermedad cerebrovascular aguda o pueden ser de inicio tardío o progresivo. Reportamos dos casos de hemibalismo y temblor contralateral después de enfermedad cerebrovascular aguda. Ambos pacientes presentaron hemibalismo agudo. En un paciente el temblor se inició en forma aguda y en otro fue tardía. Discutimos los posibles mecanismos patogénicos para sus desórdenes del movimiento. A nuestro conocimiento, éste es el primer reporte de dos casos con la presentación inusual de hemibalismo y temblor contralateral después de una enfermedad vascular cerebral.

**Palabras clave:** Hemibalismo, Temblor contralateral, Patofisiología, Enfermedad vascular cerebral.

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## Introducción

Chorea and ballismus often coexist and usually involve hemibody.<sup>1</sup> Chorea is reported as the most frequent post-stroke involuntary movement.<sup>2, 3</sup> It is often thought that hemiballismus is caused most commonly by a lesion in the contralateral subthalamic nucleus (STN),<sup>1</sup> but the localization is usually elsewhere.<sup>1-5</sup> The pathogenesis of hemiballismus has not as yet been sufficiently elucidated.<sup>1</sup> Lesions in the thalamus, basal ganglia, brainstem, cerebellum, white subcortical matter and cortex have been associated with post-stroke tremor.<sup>6-8</sup> Action tremor occurs with lesions in the cerebellum or in their outflow tracts.<sup>9</sup> Vascular Parkinsonian tremor may share a pathogenic basis identical to those associated with idiopathic Parkinsonism.<sup>10</sup>

## Case Reports

### Patient 1

In February 2003, while watching TV, an 80-year-old hypertensive, right-handed male developed sudden, mild left hemiparesis. In 1998, he had sudden ataxia of right limbs and gait, from which he recovered in three months. Since 1998, the patient had been taking antihypertensive drugs and aspirin on a regular basis. The patient and his wife indicated that, one month after the stroke of 1998, he presented with occasional mild right-hand tremor when he wrote.

At the Emergency Room, the patient's initial blood pressure was 200/110 mmHg. On neurological examination, the patient was alert and oriented and had mild left hemiparesis. In the hours following admission, the patient developed ballistic movements on the left side. He did not have any anosognosia or abnormal cortical sensations (two-point discrimination and stereognosis). Over the next weeks, his hemiballismus gradually worsened. He was treated with Haloperidol, up to 10 mg per day, with minimal improvement in abnormal movement.

Three weeks after admission, the patient was able to walk with difficulty and was then discharged. MRI scan was obtained four days after admission and showed in T2- Flair a right cerebellar hemisphere atrophy,

secondary to an old infarct (figure 1), and infarcts at the gray-white matter junction in the frontal and parietal regions (figure 2).



Figure 1: Axial T1W MR scan shows right cerebellar hemisphere atrophy secondary to an old infarct.

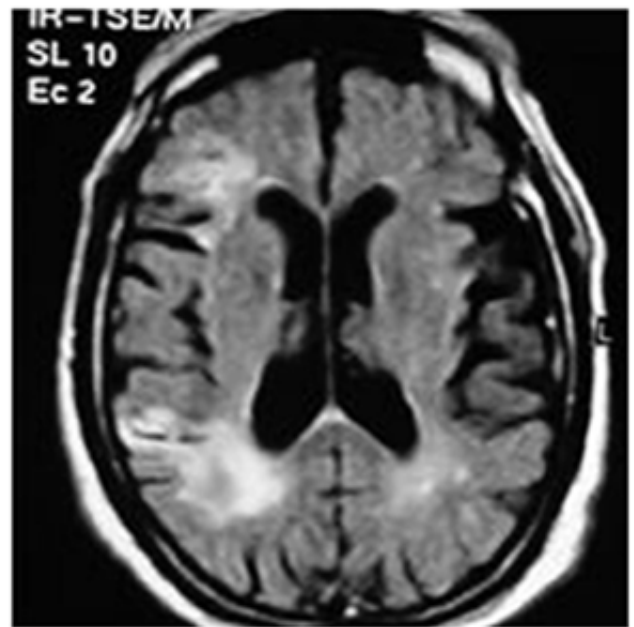


Figure 2: Axial FLAIR contiguous MR images show hyperintense ischemic lesions in the supratentorial white matter and right infarcts at the gray-white matter junction in the frontal and parietal regions.

Echocardiogram showed hypertrophy of the left ventricle and did not show evidence of thrombus formation. A carotid echo-doppler scan and complete blood test results were unremarkable. The patient had no history of treatment with neuroleptic drugs or exposure to toxins. There was no family history of movement disorders.

During the next months, the patient took Haloperidol, Olanzapine, Risperidone, Quetiapine, Clozapine, Mirtazapine and Clonazepam until he reached the maximum dose. In February 2004, the patient presented with a progressive form of postural and kinetic tremor in his upper right limb. Four weeks later, the tremor became disabling. There was no rest hand tremor or cogwheel rigidity in his right hand. The tremor was from 3 to 5 Hz and was shown as an irregular oscillation in an intended direction and was more prominent toward the end. A new MRI did not show new vascular lesions compared to that of February 2003. The patient had a partial improvement of the tremor and the hemiballismus, until the last follow-up in May 2005.

### **Patient 2**

A 78-year-old right-handed woman, who developed dysarthria and left hemiparesis-hemihypoesthesia while taking a nap. In the Emergency Room, the patient had a blood pressure of 190/105 mg Hg. Twelve hours later, she suddenly and progressively presented with ballistic movements in the left hemibody, with greater proximal severity in the upper limb. In the following three weeks, the hemiballismus disappeared spontaneously. On the third day after admission to the hospital, she progressively presented with intermittent rest and postural tremor of the right hand, which in less than 48 hours tended to become permanent. The rest tremor that appeared only during the day at a frequency of 4-7 Hz was characterized by flexion-extension movements of all the right fingers, mainly at the metacarpophalangeal joints.

Occasional mild adduction-abduction movements of the fingers were also noticed. The tremor had a tendency to be more prominent when the patient remained at rest with the arm and especially the hand on her bed or body. The postural tremor was intermittent, at a frequency of 4-6 Hz, and was more intense when the patient used the right hand and diminished when she moved her left arm, talked or focused attention on other activities. There was no cogwheel rigidity. At the last follow-up a year after the stroke, the patient was walking and her tremor did not show any significant changes.

Magnetic Resonance Imaging (MRI) was obtained three days after admission and showed in T2-Flair ischemic foci in the pontine tegmentum, and bilateral globus pallidus (figure 3). The angioresonance showed segmentary narrowing in the proximal segment of the left posterior cerebral artery, suggesting atheromatosis. The echocardiogram showed myocardiosclerosis. Complete blood test results were unremarkable. The patient did not have any history of movement disorders, nor had she been administered

any neuroleptic drugs or been exposed to toxins.

### Discussion

Lesions affecting the afferent or efferent pathways of the STN may result in hemiballismus.<sup>1-5</sup> In our patient 1, the infarcts in the frontal and parietal lobes may have induced changes in the firing patterns of the neural fibers being projected to the basal ganglia, reducing the excitatory inputs from the STN to the pars intern of the globus pallidus, which in turn disinhibits the thalamus and cortex resulting in hyperkinetic movement.<sup>1</sup> In patient 2, the bilateral lesion in the globus pallidus could explain the pathophysiology of hemiballismus.<sup>1-3</sup>

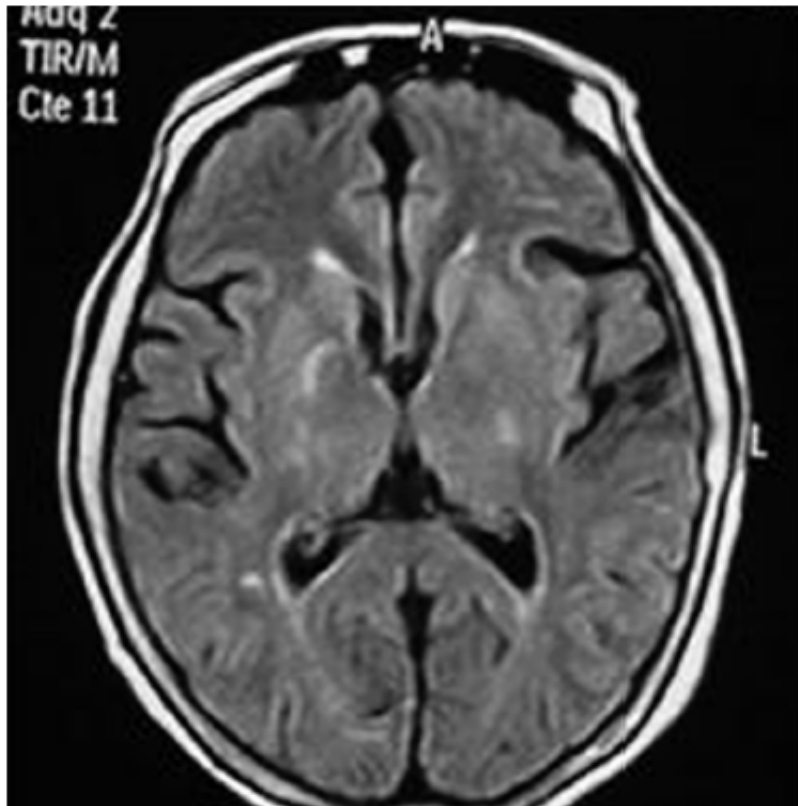


Figure 3: Axial FLAIR MR scan demonstrates small bilateral high signal intensities in the globus pallidus, in relation with ischemic foci.

Action tremor in the ipsilateral upper extremities occurs with lesions in the nucleus dentate and interpositus or brachium conjunctivum.<sup>9</sup> Patient 1 showed action tremor, which has characteristics similar to those of cerebellar tremor.<sup>9</sup> These tremors occur, as a result of the loss of cerebellar influence in motor control,<sup>9</sup> and appears to be a result of an excessive reliance on feedback control, which results in abnormal mechanical reflex oscillation.<sup>9</sup> The increase in severity of tremor until it is disabling, after the second stroke, may be related to neuroleptic drugs used to treat hemiballismus, to certain changes in brain plasticity,<sup>3</sup> or the development of post-synaptic denervation supersensitivity and release of the inferior olive.<sup>11</sup> Our patient 2 had a Parkinsonian rest tremor, characterized by a rhythmic “pill-rolling”- type finger tremor. Her tremor, however, was less monotonous than Parkinsonian tremor and occurred more intermittently.

Our patient did not have rigidity or bradykinesia and therefore could not be classified as having a vascular Parkinsonism.<sup>3</sup> In spite of this, her rest tremor may share a pathogenic basis similar to the tremor of idiopathic Parkinsonism. In this patient, the genesis of the rest tremor could be the result of a thalamic dysfunction,<sup>10, 11</sup> secondary to pallidal or midbrain damage.<sup>7</sup>

The coexistence of two movement disorders in the same patient suggests an imbalanced bilateral integration of the ganglia-sensorimotor cortex circuits.

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