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Hyperkinetic Movement Disorder Secondary to Punctate Hemorrhage in Lateral Ventricle Lining

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Key Words
Chorea · Hemiballismus · Hemorrhage · Periventricular lining

Abstract
We present the case of an elderly male with hyperkinetic movements of the right arm and leg due to a small hemorrhage in the lateral aspect of the left lateral ventricle atrium. As per our database search, this is a unique presentation of a stroke in this particular location.

Introduction

Hemiballismus is defined as the unilateral involuntary flinging or flailing movement of an extremity. Chorea is the randomly appearing sequence of ≥1 discrete involuntary movements of a limb, while athetosis is defined as slow, continuous, involuntary writhing movements. Hemiballismus involves the proximal limbs, while athetosis involves the distal extremities [1]. These hyperkinetic movements are commonly associated with contralateral lesions of the basal ganglia [especially the subthalamic nucleus (STN)]. Less than 1% of patients with stroke (ischemic or hemorrhagic) present with hemiballistic or choreoathetoid movements [2, 3]. Earlier, it was believed that ballistic and choreoathetoid movements were solely subthalamic in origin. However, various case series demonstrate that they may in fact manifest from various pathologies originating in the caudate, putamen, striatus, and globus pallidus [3]. A few studies with MRI showed that cortical strokes may also cause hemiballism [4]. This is the first ever reported case of an acute presentation of a hyperkinetic movement disorder as a result of periventricular stroke.

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Case Report

An 82-year-old male with a past medical history of prostate cancer status following radiation and brachytherapy presented with an acute onset of right facial droop and right hand jerky movements. According to the patient, he initially experienced involuntary jerky movements involving the right leg, which partially resolved soon afterwards. This was followed by flailing of the right arm that the patient had no control over. During the event, the patient was conversant and completely aware of what was going on. In addition, the patient's family reported a mild right-sided facial droop. He did not have any focal weakness other than right hand distal muscle weakness.

In the emergency department, he was worked up for stroke/intracranial hemorrhage. A CT and MRI revealed a very small hemorrhage in the intraparenchymal area near the left ventricle. MRI/MRA of the brain showed a punctate hemorrhage in the lateral aspect of the left lateral ventricle atrium, which was thought to be a subacute punctate hemorrhage of uncertain etiology. Figures 1 and 2 demonstrate the exact location of the punctate hemorrhage from the selected MRI sequences. M1 and P2 vessels showed mild to moderate stenosis. No pathology could be found in the basal ganglia, thalamus, or subthalamic region. His electroencephalogram was normal, with no ictal or interictal spikes or discharge.

Leviteracetam was initially started in the emergency room, but showed no improvement in his symptoms. The patient was then given haloperidol 2 mg, which was able to control the hyperkinetic movements. We reviewed the MRI with a radiologist in order to determine the presence of ischemic changes in the region of interest (STN and basal ganglia). However, there was no identifiable pathology. The patient's response to haloperidol was reassuring, and the patient was subsequently discharged. His symptoms had significantly improved 3 weeks after discharge, to an extent that the patient no longer required haloperidol.

Conclusion

In this patient, 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 interna of the globus pallidus. This, in turn, disinhibited the thalamus and cortex and therefore resulting in the hyperkinetic movements experienced by the patient.

There is extensive data to support STN lesions as a cause of hemiballismus and choreoathetosis [2, 5]. Animal models reinforce the concept that lesions in the STN produce contralateral hemiballism. However, in multiple clinical case series, non-STN lesions have also been shown to cause hyperkinetic movements, especially in stroke patients [5]. Pathology of the incoming and outgoing fibers to and from the STN may manifest as hemiballismus, which may explain the few cases of frontal and parietal strokes (MCA distribution) that presented with hyperkinetic movements in rare case reports [1, 6]. However, the exact cortical circuit projecting into the basal ganglia is still not completely known. Our hypothesis is that the punctate hemorrhage in the left lateral ventricle lining in our patient may have affected the fibers in the basal ganglia leading to hemiballistic and choreoathetoid movements of the right side of the body.

Stroke-induced hyperkinetic disorders are symptomatically managed with neuroleptics and atypical antipsychotics [1, 3, 7]. Correspondingly, our patient showed a marked improvement in his symptoms after being administered haloperidol.

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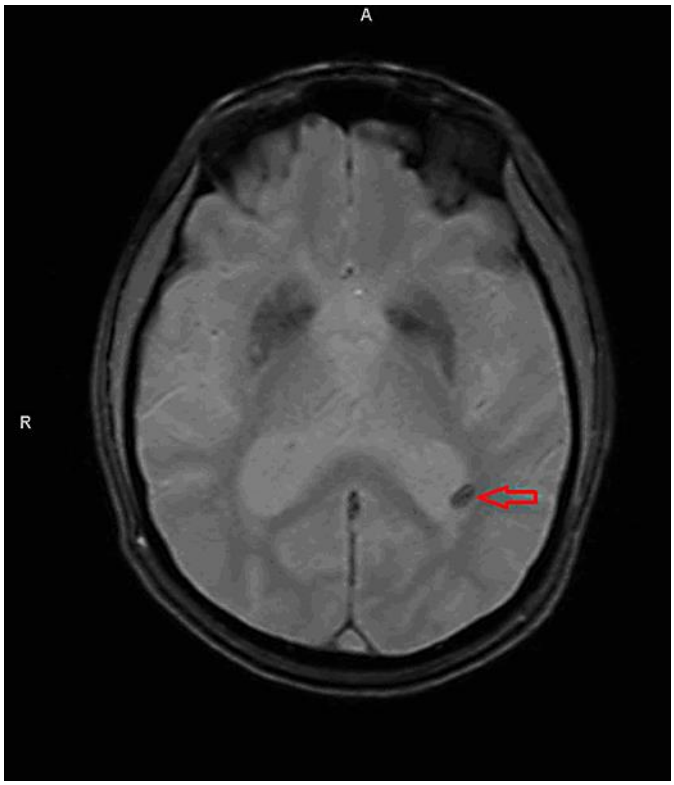

Fig. 1. Gradient echo sequence demonstrating the small punctate hemorrhage as an oval-shaped hyperintense lesion (red arrow) located adjacent to the left lateral ventricle atrium.

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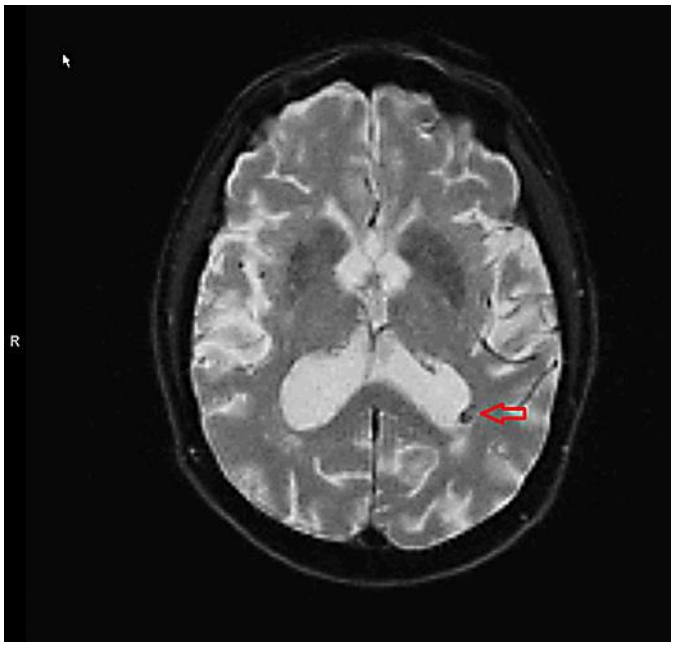

Fig. 2. Brain MRI (T2 sequence) demonstrating the small hemorrhagic lesion (arrow) lateral to the ventricle atrium.