

A case of Hemichorea-Hemiballism Induced by Acute Infarction of Bilateral Corona Radiata and Cortex

Mengwei Hao · Xiaoling Qin · Hui Gao

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Abstract Movement disorders are a recognized complication of stroke. Here we present a case of hemichorea-hemiballism (HCHB) after stroke. Basal ganglia and thalamus are typically recognized as sites responsible for HCHB. The MRI scan showed acute infarction which was unexpectedly present in both sides of corona radiata and cortex, but not in basal ganglia. This cortical HCHB could have evolved due to hypoperfusion of basal ganglia undetectable at the MRI scan or due to interruption of excitatory connections from the cerebral cortex to basal ganglia.

Keywords Hemichorea-hemiballism · Stroke · MRI · Corona radiata · Cortex

Introduction

Movement disorders are a recognized complication of stroke. Many different types of hyperkinetic and hypokinetic movement disorders, including hemichorea, hemiballism, and hemichorea-hemiballism, were reported after ischemic and hemorrhagic stroke [1]. Hemichorea is defined as unilateral, rapid involuntary motions of flexion and extension, rotation or crossing, which predominantly affects distal parts and may also involve other body parts [2].

Hemiballismus (hemiballism) is a severe, violent, arrhythmic, and large amplitude excursion of a limb from a proximal joint with an element of rotation [2]. Simultaneous hemichorea-hemiballism (HCHB) can occur because hemichorea and hemiballism may be driven by the same mechanism [2]. Cortical HCHB includes cases with lesions only in the frontal and parietal cortices, with normal findings in the basal ganglia and thalamus in both MRI and SPECT images [3]. Usually, basal ganglia and thalamus are the sites responsible for hemichorea [4], [5], although other regions were also reported in some cases.

The symptoms mentioned above are involuntary hyperkinetic disorders and are uncommon after stroke. Since most publications report isolated cases or relatively small series of cases compiled retrospectively from stroke registries, morbidity is still unclear. One case series study reported that the occurrence of movement disorder after stroke reached 3.7 %, and that hemichorea was the most common disorder [6]. Another publication reported the incidence of post-stroke hemichorea being as low as 0.54 % [7].

Here we present a case of a 62-year-old man who was hospitalized because of repeated involuntary movements of the left side limbs.

Case Report

In this patient, involuntary movements of the left arm and leg developed suddenly and without any obvious provoking factor on October 22, 2013. The movements comprised severe, violent, arrhythmic, and large amplitude excursions of the left arm and rapid oscillations of the left leg. These were accompanied by facial involuntary movements. The symptoms lasted dozens of minutes and recurred several

M. Hao · X. Qin (✉)
Department of Neurology, Xuzhou Central Hospital, Jiefang Street 199, Xuzhou, Jiangsu 221000, People's Republic of China
e-mail: doctorqx0@126.com

H. Gao
Department of Nutrition and Food Hygiene, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, People's Republic of China

times in the subsequent two days. The patient had a history of hypothyroidism and received relevant medications. However, specifics of the treatment and the outcome were unknown. There were no history of hypertension or diabetes mellitus, and no family history of these diseases.

Neurological examination showed a superimposed dystonic posturing on the left side when the symptoms attacked, but all changes returned to normal condition after the symptoms subsided. The muscle tone and strength of the upper and lower limbs were normal on both sides. There was no evidence of sensory impairment, and cranial nerves were normal.

Laboratory examination confirmed hypothyroidism: free T3 2.82 pmol/L (normal range: 3.5–6.5 pmol/L), free T4 5.26 pmol/L (normal range: 11.5–22.7 pmol/L), and thyroid-stimulating hormone 50.136 μ U/L (normal range: 0.35–5.5 μ U/L). Other biochemical markers were normal: free blood glucose 4.79 mmol/L (normal range: 3.8–6.2 mmol/L), triglycerides 1.02 mmol/L (normal range: 0.45–1.75 mmol/L), and LDL cholesterol 2.7 mmol/L (normal range: 2.2–3.6 mmol/L). The brain MRI scan showed an irregular area of high signal intensity on both sides of the corona radiate and parietal cortex. The apparent diffusion co-efficient (ADC) imaging excluded

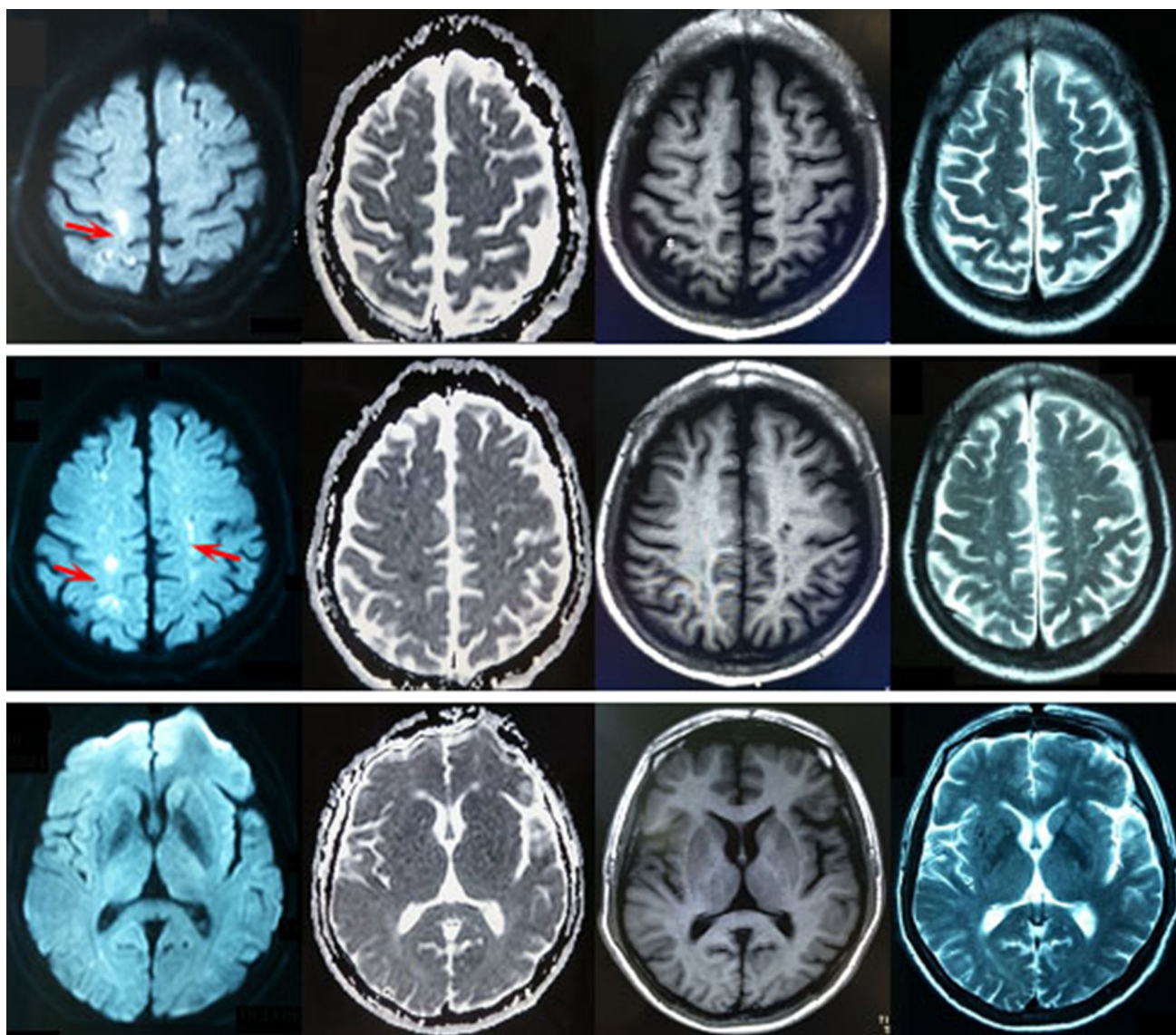


Fig. 1 Magnetic resonance imaging. The diffusion-weighted imaging (DWI) shows acute infarction of bilateral corona radiate and the cortex. The apparent diffusion co-efficient (ADC) imaging excludes inflammation or intoxication. The T1-weighted axial and T2-weighted

axial imagings show infarction of bilateral corona radiate and the cortex. In addition, DWI, ADC, T1- and T2-weighted axial imaging show no focus in area of basal ganglia

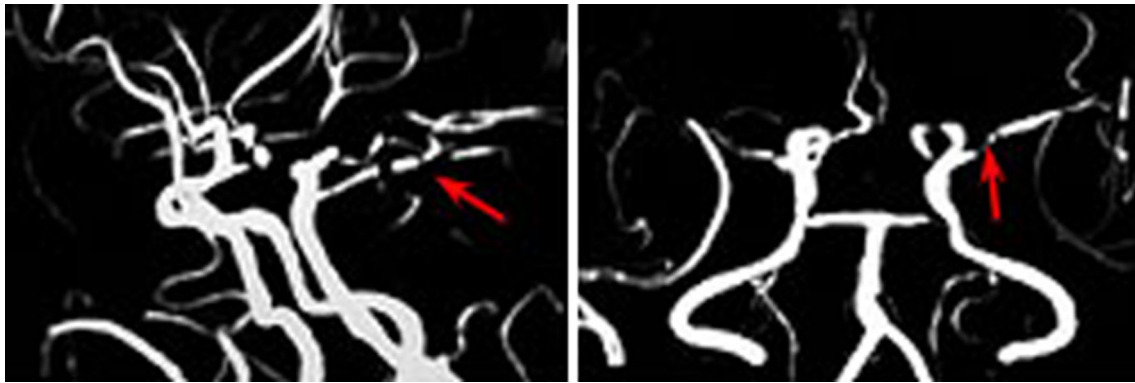


Fig. 2 Magnetic resonance angiography. Magnetic resonance angiography shows intracranial atherosclerosis. The left anterior cerebral artery is not seen, while the middle and posterior cerebral arteries show extensive stenosis

inflammation and intoxication. Furthermore, DWI, ADC, T1-weighted axial imaging, and T2-weighted axial imaging showed no lesions in basal ganglia (Fig. 1). The brain magnetic resonance angiography (MRA) scan showed intracranial atherosclerosis. The left anterior cerebral artery was not seen, while the middle and posterior cerebral arteries showed extensive stenosis (Fig. 2).

The treatment of this patient included aspirin, ozagrel, and Chinese traditional medicine promoting blood circulation to remove blood stasis. Interestingly, the symptom did not attack any longer during the time of hospitalization.

Discussion

Clinical manifestations in this patient comprised repeated involuntary movements of left limbs, which can be diagnosed as HCHB. The MRI scan showed fresh infarction on both sides of corona radiata and cortex, but not in basal ganglia. In this patient, we excluded other causes of HCHB by collecting prior medical history and by laboratory testing. Therefore, this case can be defined as cortical HCHB.

A number of brain regions have been identified as areas causing these movement disorders after stroke. Thus, a study on 27 patients demonstrated that the lesions can be located in the caudate and putamen, cortex, thalamus and subthalamic area, subthalamus, and the globus pallidus [7]. Basal ganglia are the areas most commonly implicated in the post-stroke movement disorders. In a classical model of movement disorders, either structural damage or functional alteration is thought to be the central problem. Pathologic signals from the diseased basal ganglia go to the affected body parts [3]. HCHB may result from hypoperfusion of basal ganglia, mild enough not to be detected by MRI. This speculation is supported by MRA scan of this patient (Fig. 2), which showed intracranial atherosclerosis.

Intracranial atherosclerosis leads to ischemia of basal ganglia. Alternatively, interruption of excitatory connections from the cerebral cortex to the basal ganglia may result in functional derangement of the striatum. There may be an independent contribution of cortical areas to the pathogenesis of cortical HCHB even without significant involvement of basal ganglia and thalamus [3]. Derangement of the wiring between the frontal cortices and parietal cortex may be another possible mechanism of cortical HCHB [3]. This would require PET or functional MRI to uncover.

Conclusion

In this case report, we describe a rare patient who developed repeated involuntary movements of left limbs after stroke. Acute infarction was located in bilateral corona radiata and cortex, but not in basal ganglia or thalamus. We diagnosed this case as cortical post-stroke HCHB. As potential mechanisms, hyperperfusion of basal ganglia, contribution of cortical areas without significant involvement of basal ganglia and thalamus, and derangement of the wiring between the frontal cortices and parietal cortex were discussed.

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