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Hemichorea secondary to cerebral border zone infarction with middle cerebral artery stenosis: A case report

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Abstract

Hemichorea is a rare presentation of stroke. Post-stroke hemichorea is often caused by a lesion affecting the basal ganglia. Herein, we present an unusual case of hemichorea in a patient with border zone infarction and middle cerebral artery stenosis. A 64-year old man visited the emergency department due to acute onset left-sided hemichorea. Brain MRI showed several dot-like multifocal high signal intensities in the right border zone area between the right anterior cerebral artery and the middle cerebral artery. Transfemoral cerebral angiography revealed severe focal stenosis in the right M1 segment. Mechanical thrombectomy was performed and the stenosis partially improved. Computed tomography perfusion showed a delayed mean transit time in the subcortical area around the stroke lesions and no abnormal perfusion defect in the deep structures. Six days after symptom onset, clonazepam was administered, and the chorea ceased. This case highlights that hemichorea can occur in patients with dot-like, scattered stroke lesions in the border zone.

Keywords: Stroke; chorea; cerebral infarction; perfusion imaging

INTRODUCTION

Hemichorea is a rare symptom of acute stroke.[1,2] One study reported that 0.54% of 5,009 patients with acute stroke presented with hemichorea.[2] Post-stroke hemichorea is most often caused by a lesion affecting the basal ganglia.[1-3] However, hemichorea secondary to cortical or subcortical stroke, sparing the deep structures has been reported.[1-5] In particular, post-stroke hemichorea associated with dot-like scattered lesions of the border zone is very rare.[6] Herein, we present an unusual case of hemichorea in a patient with border zone infarction and middle cerebral artery (MCA) stenosis.

CASE REPORT

A 64-year-old man presented to another emergency department with an abrupt involuntary left-side movement that occurred an hour before presentation. He was a smoker and took medications for hypertension and heart disease. The patient had no family history of cerebrovascular disease or movement disorders. Diffusion-weighted magnetic resonance imaging (DWI) of the brain showed several dot-like multifocal high signal intensities in the right border zone area between the right anterior cerebral artery (ACA) and the MCA (Figure 1).

Magnetic resonance angiography of the brain revealed stenosis of the right MCA. The patient was treated with intravenous thrombolysis and transferred to our hospital for further management. On presentation, the patient had involuntary, flinging, and continuous random muscle contractions in his left arm and legs. Muscle strength, sensory functions, and deep tendon reflexes were symmetrical. Laboratory examinations, including serum electrolytes, glucose, liver enzymes, creatinine, and thyroid function tests, were normal. Transfemoral cerebral angiography revealed severe focal stenosis in the right M1 segment (Figure 2A). Mechanical thrombectomy was performed and the stenosis partially improved (Figure 2B).

He was prescribed aspirin, statin, and risperidone (1 mg), and his symptoms improved slightly. No additional lesions were observed

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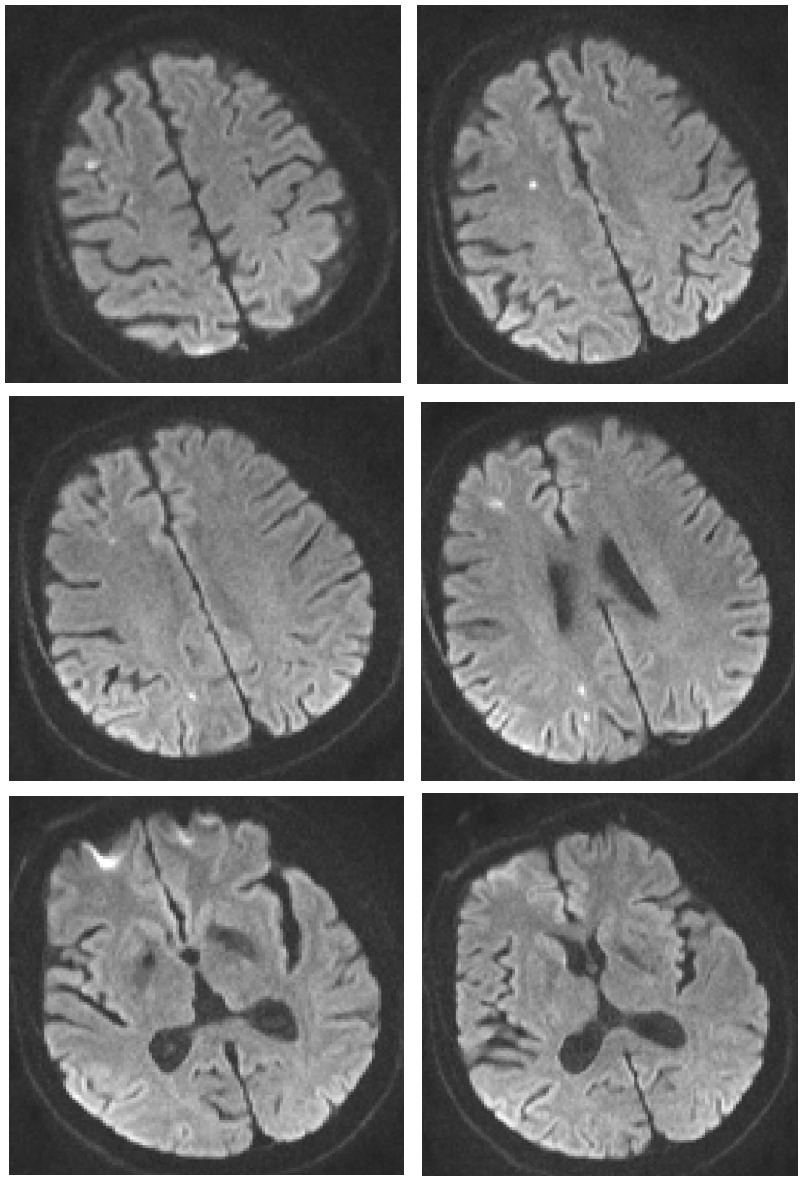

Figure 1. Diffusion weighted imaging shows several dot-like multifocal high signal intensities in the right border zone area between the right anterior cerebral artery and the middle cerebral artery.

on DWI the next day. Computed tomography perfusion (CTP) during the symptomatic phase showed a delayed mean transit time in the subcortical area around the stroke lesions (Figure 3A) and no abnormal perfusion defect in the deep structures, including the basal ganglia (Figure 3B).

Six days after symptom onset, clonazepam 0.5 mg was administered, and the chorea disappeared. At the 2-month follow-up, the patient remained symptom-free.

DISCUSSION

Abnormal involuntary movements associated with stroke, including hemichorea, are often associated with lesions in the basal ganglia.[3] The basal ganglia play an important role in motor control and form circuits with the cerebral cortex and thalamus.[3] However, the mechanism of post-stroke hemichorea remains unclear, because hemichorea does not always appear when stroke occurs in the basal ganglia and related structures, and post-stroke hemichorea is relatively rare.[1,2]

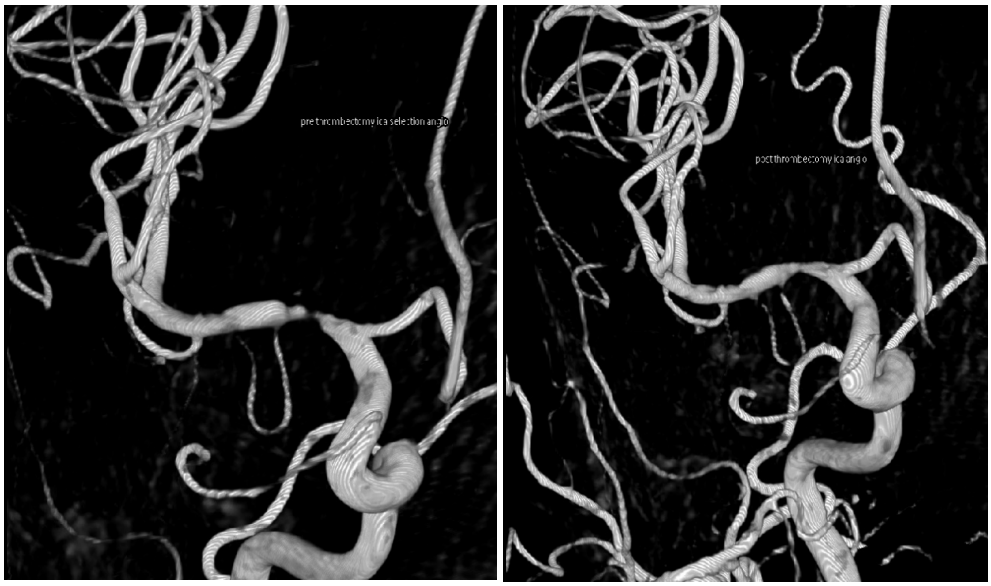

Figure 2. Transfemoral cerebral angiography shows focal severe stenosis in the right M1 segment (A). The stenosis was partially improved after mechanical thrombectomy (B).

It has been documented that lesions other than those affecting the basal ganglia can cause post-stroke hemichorea. Strauss *et al*. reported a case of hemichorea after stroke of the pure parieto-occipital cortex without involvement of subcortical structures and suggested interruption of the intercortical and/or cortical-ganglionic connection as a pathophysiologic mechanism.[5] Dong *et al*. reported that hemichorea occurred after an isolated temporal stroke.[7] In this case, CTP showed delayed perfusion in the parietal cortex and corona radiata due to severe MCA stenosis. The authors suggested that hypoperfusion could disturb the metabolism of the basal ganglia. Wang *et al*. reported two cases of acute hemichorea secondary to acute ischemic stroke in the temporal cortex.[8] In the first case, severe stenosis of the MCA was observed on the same side of the ischemic lesion, and CTP revealed marked hypoperfusion in the MCA territory. Following angioplasty, perfusion

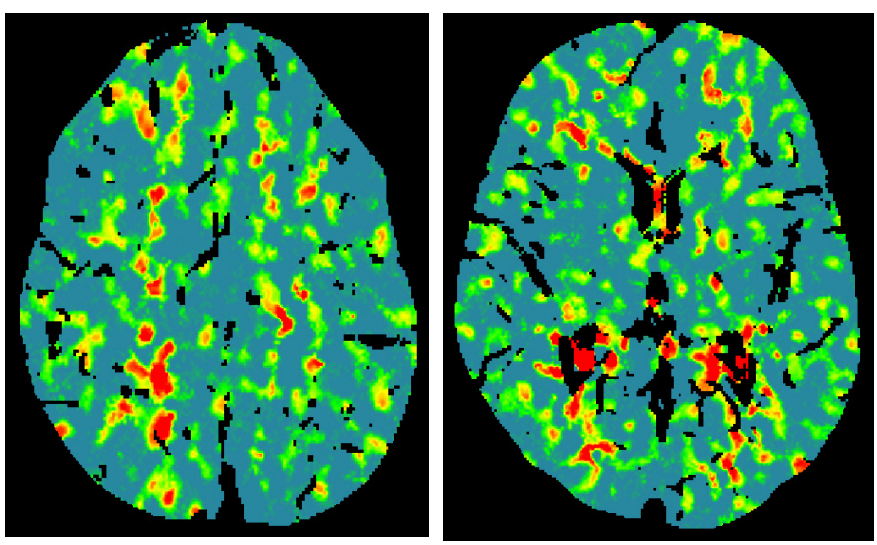

Figure 3. Computed tomography perfusion during the symptomatic phase shows a delayed mean transit time in the subcortical area around the stroke lesions and no abnormal perfusion defect in the deep structures including the basal ganglia (A, B).

in the affected area improved, and hemichorea was significantly relieved. These findings suggest that hypoperfusion may be associated with hemichorea and that surgical revascularization could be an effective treatment option. In the second case, CTP revealed mild cortical hypoperfusion, with preservation of the basal ganglia on the same side of the ischemic lesion. This finding may provide evidence for the cortical contribution to the pathophysiology of hemichorea.

To the best of our knowledge, only two cases of hemichorea with small dispersed lesions that did not involve deep structures have been reported in the MCA-ACA border zone.[6,9] In the first case, the border zone ischemic lesions were dot-like, and the MCA stenosis was located on the same side of the ischemic lesion as in our case; however, perfusion imaging was not performed.[6] Another report involved a patient with acute hemichorea associated with border zone infarction and ipsilateral severe carotid artery stenosis.[9] The ischemic lesion was larger than that in our patient, and perfusion studies showed impaired cerebral blood flow in the subcortical region, sparing deep structures similar to that observed in our case. Hemichorea persisted despite haloperidol and clonazepam administration and disappeared after few weeks with normalization of perfusion after carotid artery angioplasty and stent placement. Hemodynamic insufficiency may be a contributing factor to hemichorea, as hemichorea improves after carotid artery stenting.

Suri *et al*. reviewed 284 cases of post-stroke movement disorders and reported that, among patients with chorea for whom outcome data were available, 15 experienced improvement or resolution of symptoms, while 13 had persistent symptoms.[10] Given that post-stroke hemichorea can lead to injury and increased morbidity, appropriate treatment is needed. Pharmacologic treatments include neuroleptics and benzodiazepines, as used in our patient, as well as other agents such as sodium valproate and topiramate.[3] In refractory cases, surgical interventions, including deep brain stimulation and pallidotomy, have been reported to result in favorable outcomes.[11,12] Additionally, in cases where significant stenosis is present in an artery associated with the ischemic lesion, revascularization procedures may offer prognostic benefits in the management of hemichorea. However, although border zone infarction is usually caused by hemodynamic insufficiency, the fact that it rarely causes hemichorea suggests that further research is warranted.

The mechanism of hemichorea in our patient was unclear; however, our case highlights that hemichorea can occur in patients with dot-like, scattered stroke lesions in the border zone.

A limitation of our case is that CTP was not performed before thrombolysis or thrombectomy. Further studies using advanced techniques are required to clarify the mechanisms underlying post-stroke hemichorea.

DISCLOSURE

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