Reversible Vasospasm in Migrainous Infarction
A Transcranial Doppler Follow-up Study

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Migraine is a common disorder in young women, but it is rarely associated with ischemic stroke. According to previous stroke registries, migrainous infarcts account for 0.5% to 1.5% of all ischemic strokes and about 10% to 14% of young ischemic strokes. The pathophysiologic mechanism of migrainous infarction is still unclear. Here we report a patient with a migraine history in whom acute cerebral infarction developed during a migraine attack due to an arterial vasospasm, which was illustrated by serial transcranial Doppler sonography (TCD).

Case Report

A 42-year-old woman had a history of migraine since she was a teenager. Her migraines were characterized by episodic unilateral throbbing headache with nausea, vomiting, and limitation of daily activity. No visual aura had been noted. Her headache frequency had increased in the past year. She had no history of smoking, substance abuse, diabetes mellitus, hypertension, or other medical diseases. She did not take any oral contraceptives or ergot alkaloids. Her mother also had a migraine history for years.

One day before admission, the patient had recurrent severe right throbbing headache with dizziness and vomiting, similar to her previous migraines. She had taken nonsteroidal anti-inflammatory drugs, but it was in vain. Meanwhile, acute onset of left limb weakness and slurred speech was noted. Her consciousness was still preserved without vertigo, diplopia, dysphagia, or heart discomfort.

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There was no recent fever or trauma. The patient was sent to the emergency department on the next day. On arrival, her neurologic examination revealed clear consciousness, left facial palsy, dysarthria, and left hemiparesis. Her muscle power was 4/5 (Medical Research Council grading) in the left limbs and full on the right side. Brain computed tomography showed faint hypodensity in the right subcortical area.

After admission, the patient underwent brain magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA). Magnetic resonance imaging showed acute cerebral infarction at the right basal ganglion and corona radiata with mild focal brain swelling (Figure 1A). Contrast-enhanced MRA revealed no obvious atherosclerosis of neck and head arteries or evidence of dissection (Figure 1B). Laboratory and young stroke workup results, including white blood cell count, homocysteine, protein C, protein S, antithrombin, and anticardiolipin antibody, were all normal except for an elevated total cholesterol level (287 mg/dL). The low-density cholesterol was 161 mg/dL, and the high-density cholesterol was 45 mg/dL. Electrocardiography showed a normal sinus rhythm. We also arranged advanced echocardiography, and there was no visible thrombus or valvular or congenital abnormalities. The patient then received further carotid duplex and TCD examinations. Her blood pressure was 126/82 mm Hg during the sonographic examinations. The carotid duplex scan disclosed only mild atherosclerosis at the right carotid bulb, but TCD illustrated a very high flow velocity at the right middle cerebral artery (MCA) M1 segment with a turbulent flow pattern (237/99 cm/s; Figure 2A). The flow velocities of the other intracranial arteries, including the left MCA and bilateral anterior and posterior arteries, were normal (Table 1).

In addition to aspirin, flunarizine was prescribed for a suspicious vasospasm and frequent migraine attacks. Follow-up TCD revealed stepwise improvement of the right MCA M1 flow velocity 1 week (176/80 cm/s) and 1 month (156/69 cm/s) later, without a notable change in the flow velocities of the other vessels. Right MCA infarction resulting from a vasospasm was diagnosed on the basis of the clinical manifestations and serial TCD findings. Clinically, the left limb weakness and slurred speech improved greatly after medical treatment.

Her headache subsided after naproxen therapy. The migraine frequency decreased considerably from several times per week to once per season. Six months after the stroke, TCD showed a normal flow velocity at the same segment of the right MCA (123/53 cm/s; Figure 2B).

Discussion

Although stroke and migraine are two different diseases, recent reports have documented a substantial association between them. Migraine is relatively frequent in young female patients with ischemic stroke. According to many observational studies, the risk of stroke is increased not only in people who have migraine with auras but also in those without auras. It is particularly higher among users of oral contraceptives and smokers. As for migrainous infarction, it is a direct consequence of severe hypoperfusion during a migraine attack; the precise cause in unclear. Of the variety of putative mechanisms, cortical spreading depression, intracranial large vessel spasms, and hypercoagulation are the most recognized causes.

For young patients with stroke, a series of investigations should be conducted to find the underlying etiology. The main differential diagnoses includes cardiac emboli, arterial dissection, coagulation, and autoimmune and metabolic abnormalities. For our 42-year-old patient, no remarkable abnormality was found in the young stroke workup. Her MRA study did not show obvious atherosclerosis or evidence of dissection. Instead, TCD during the acute stroke showed a very high velocity at the right MCA with turbulent flow, which reduced rapidly on follow-up sonography after medical treatment. In patients with migraine, a vasospasm putatively may result from the ictal release of potent vasoconstrictive substances such as endothelin and serotonin or from the use of vasoconstrictive drugs, including ergotamines and triptans. This patient had never taken vasoconstrictive drugs before or during her attacks. Thus, our serial TCD examinations clearly illustrated that migraine with a right MCA vasospasm was the most likely cause leading to the patient’s infarction. This finding provides useful information about the mechanism of stroke.
Transcranial Doppler sonography has been successfully applied to detection of vasospasms after subarachnoid hemorrhage. There is an inverse relationship between the degree of arterial narrowing and the velocity, and the correlation is great between the mean velocity of the MCA by TCD and the diameter of the vessel lumen on angiography. A mean flow velocity of 120 cm/s is classified as a moderate spasm, and 200 cm/s is classified as a severe spasm. In comparison with angiography, TCD has good sensitivity and specificity for the diagnosis of MCA vasospasms in patients with subarachnoid hemorrhage. As for headache, vasospasms had been recorded occasionally by TCD in a few patients with migraine, but there were no serial TCD follow-up examinations of migrainous infarction. Our case had a clear depiction of a vasospasm, and it suggests that TCD is a valuable method for detecting vasospasms in migrainous infarction.

Figure 1. Brain MRI and MRA of the patient showed acute cerebral infarction with mild focal brain swelling in the right MCA territory. A, Diffusion-weighted imaging showed high signal intensity with mild focal brain swelling, consistent with acute cerebral infarction. B, Brain and neck MRA showed neither atherosclerosis nor evidence of dissection such as an intima flap or a false lumen.

Figure 2. Serial TCD studies of the right MCA M1 segment. A, A very high-velocity and turbulent flow pattern (237/99 cm/s; mean flow velocity, 145 cm/s) was noted on the first study of the right MCA M1 segment during the acute stroke. B, Final TCD follow-up showed a completely normal flow velocity at the same segment of the right MCA (123/53 cm/s; mean flow velocity, 77 cm/s) 6 months after the stroke.
Given these circumstances, a vasospasm is an important factor to be considered in migrainous infarction. Transcranial Doppler sonography is noninvasive and safe compared with the traditional angiography and may be a good diagnostic tool to reveal this hemodynamic change. It deserves to be reported because serial TCD examinations can not only show vasospasms clearly and but also provide therapeutic implications in these patients. They should avoid the use of vasoconstrictive drugs, including triptans and ergotamines, during acute attacks. With regard to prophylaxis, propranolol may be prescribed with caution because of the limited compensatory vasodilator capacitance. Instead, calcium channel blockers and angiotensin receptor blockers may be the alternative choices for migraine prophylaxis.

References


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Table 1. Mean Blood Flow Velocities and Insonation Depths in all Vessels of the Circle of Willis of Both Sides

<table>
<thead>
<tr>
<th>Vessel</th>
<th>Right Depth, cm</th>
<th>MBFV, cm/s</th>
<th>PI</th>
<th>Left Depth, cm</th>
<th>MBFV, cm/s</th>
<th>PI</th>
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</thead>
<tbody>
<tr>
<td>MCA (proximal)</td>
<td>4.5</td>
<td>145</td>
<td>0.95</td>
<td>5.0</td>
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<td>MCA (distal)</td>
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<td>4.0</td>
<td>45</td>
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<td>61</td>
<td>1.09</td>
<td>6.6</td>
<td>79</td>
<td>0.94</td>
</tr>
<tr>
<td>PCA</td>
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<td>54</td>
<td>0.88</td>
<td>7.0</td>
<td>33</td>
<td>0.99</td>
</tr>
</tbody>
</table>

ACA indicates anterior cerebral artery; MBFV, mean blood flow velocity; PCA, posterior cerebral artery; and PI, pulsatility index.