Hyperperfusion Syndrome Following Carotid Endarterectomy: Evaluation Using Diffusion-weighted Magnetic Resonance Imaging

—Case Report—

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Abstract

A 65-year-old female developed hyperperfusion syndrome following carotid endarterectomy for severe stenosis of the left internal carotid artery. Transcranial Doppler monitoring showed a sharp increase in flow velocity in the middle cerebral artery (MCA). Diffusion-weighted magnetic resonance (MR) imaging demonstrated diffuse hyperintensity in the region of the left MCA, which diminished markedly 9 days later. The abnormal hyperintensity decreased as the patient’s symptoms improved, suggesting that diffusion-weighted MR imaging can be used for evaluating the course of hyperperfusion syndrome.

Key words: hyperperfusion syndrome, carotid endarterectomy, magnetic resonance imaging, diffusion-weighted magnetic resonance imaging

Introduction

Hyperperfusion syndrome following carotid endarterectomy (CEA) is rare, but may be followed by intracerebral hemorrhage. In particular, patients with severe carotid artery stenotic lesions who show a marked postoperative increase in cerebral blood flow are at higher risk for developing hyperperfusion syndrome. Prediction of the syndrome is important, but evaluation of the events immediately after onset is important to ensure adequate treatment. Diffusion-weighted imaging can detect acute ischemic alterations, as well as discriminate between different types of white matter edema. However, little is known about the magnetic resonance (MR) imaging appearance following the onset of hyperperfusion syndrome. We describe a case of hyperperfusion syndrome following CEA in which several diffusion-weighted MR imaging was used to evaluate the events following the onset of the syndrome.

Case Report

A 65-year-old female with controlled hypertension and diabetes mellitus had suffered from a right hemisensory disturbance for 5 years. MR imaging showed a small infarct in the pons, and antiplatelet agents were administered. Two months prior to admission, she suffered transient loss of consciousness. T2-weighted MR imaging on admission showed multiple cerebral infarctions in the right frontal and occipital lobes, bilateral basal ganglia, thalamus, and pons (Fig. 1A, B). Intraarterial digital subtraction angiography demonstrated severe bilateral carotid artery stenosis (right 90%, left 99%) (Fig. 1C). MR angiography taken simultaneously revealed lower intensity of the left middle cerebral artery (MCA) compared to the right MCA (Fig. 1D). Vertebral angiography demonstrated the left MCA via the left posterior communicating artery, but right carotid angiography detected no collateral flow through the anterior communicating artery. Single photon emission computed tomography (SPECT) using technetium-99m-hexamethylpropyleneamine oxime (99mTc-HMPAO) showed a moderate decrease in perfusion...
in the left frontoparietal region.

Left CEA was performed without complications. Transcranial Doppler (TCD) ultrasonography was used for intraoperative monitoring of the left MCA mean flow velocity (Vmca). Decreased Vmca was evident after the left internal carotid artery was clamped and an internal shunt was inserted. Vmca became 130 cm/sec immediately after the shunt insertion. Following endarterectomy, Vmca increased from 43 cm/sec to 122 cm/sec.

Blood pressure was controlled at normotension using continuous intravenous injection of nitroglycerin. No complications were noted during the first 5 days postoperatively and the patient remained normotensive. On the 6th postoperative day, the patient showed disorientation, but without headache. Four hours later, she developed global aphasia. Her blood pressure was 162/90 mmHg and emergency computed tomography showed no hemorrhage. One hour after onset of aphasia, she developed generalized seizure and became hemiparetic on the right. Her blood pressure rose further to 204/107 mmHg and nicardipine treatment was initiated immediately. Mannitol was also intravenously injected continuously, but dexamethasone was not administered due to her diabetes mellitus. The seizure was controlled using intermittent injection of phenobarbital. No electrolyte disorders were detected throughout these treatments and blood glucose levels were maintained within the normal range. Diffusion-weighted MR imaging (spin-echo sequence, repetition time/echo time of 1500/100 msec, gradient b value of 220 sec/mm²) showed dramatic hyperintense lesions in the region of the left MCA 2 hours after onset (Fig. 2A–C), and T₂-weighted MR imaging showed moderate hyperintense changes predominantly in the gray matter of the left hemisphere (Fig. 2D–F). MR angiography taken simultaneously revealed a marked increase in the intensity of the left MCA compared to the right and to the preoperative observation (Fig. 2G). ⁹⁹ᵐTc-HMPAO SPECT showed a marked increase in perfusion of the left frontotemporal region 8 hours after onset. Follow-up TCD revealed Vmca of 129 cm/sec 2 days after onset and 95 cm/sec 1 week after onset. The diagnosis was hyperperfusion syndrome. Her hemiparesis, aphasia, and confusion continued, but improved gradually by the 10th postoperative day. Follow-up diffusion-weighted MR imaging showed a reduction in the hyperintense lesion in the left MCA region by the 15th day (Fig. 3A, B). MR angiography on the 15th postoperative day also demonstrated reduced intensity of the left MCA compared to the observation at onset (Fig. 3C). Diffusion-weighted MR imaging showed the lesion had disappeared by the 29th postoperative day (Fig. 4A, B). In addition, both T₁- and T₂-weighted MR imaging detected no deterioration in the left hemisphere compared to the preoperative images (Fig. 4C–F). However, MR angiography still revealed slightly increased intensity of the left MCA (Fig. 4G). The patient was discharged without neurological deficits on the 33rd postoperative day.

**Discussion**

Neurological complications associated with CEA
Fig. 2  A–C: Diffusion-weighted axial magnetic resonance (MR) images taken 2 hours after onset demonstrating diffuse hyperintensity in the left middle cerebral artery (MCA) region.  D–F: $T_2$-weighted MR images taken simultaneously showing moderate hyperintense changes predominantly in the gray matter of the left hemisphere.  G: MR angiogram taken simultaneously showing a marked increase in the intensity of the left MCA compared to the right and to the preoperative observation.

Fig. 3  A, B: Diffusion-weighted magnetic resonance (MR) images taken on the 15th postoperative day demonstrating slight hyperintensity in the left middle cerebral artery (MCA) region, which was greatly reduced compared to the images taken at onset.  C: MR angiogram taken on the 15th postoperative day demonstrating reduced intensity of the left MCA compared to the observation at onset.
have various causes. Most complications that occur hours or days after surgery are ischemic in nature, caused by either carotid artery occlusion or embolization. Hyperperfusion syndrome occurs in only approximately 1% of all CEA procedures, but postoperative neurological dysfunction in patients with severe stenotic carotid lesions could be related to this syndrome rather than to a lack of adequate cerebral blood flow. Our patient had severe bilateral carotid artery stenosis. Changes in the mean flow velocity of the MCA were shown by intraoperative TCD. A small delayed embolus may have caused the focal deficit and seizure, but the neurological deteriorations were more likely to be caused by the high flow because follow-up TCD revealed a marked increase of Vmca 1 week after onset. Any patient demonstrating a marked increase in cerebral blood flow following CEA should be recognized as potentially at risk for hyperperfusion syndrome.

Prediction of hyperperfusion syndrome is important, but the events that develop immediately after onset must be elucidated to ensure appropriate management. Intracerebral hemorrhage and/or cerebral edema are known to be preceded by hyperperfusion following reconstruction of high-grade carotid artery stenosis. In the present case, diffuse hyperintensity was demonstrated in the left MCA region compared to the chronic cerebral infarction in the right occipital lobe 2 hours after onset. Hyperintensity on diffusion-weighted MR images indicated slower water diffusion in the lesion, so acute cerebral infarction or cytotoxic edema probably occurred following CEA. Follow-up diffusion-weighted MR imaging revealed a marked decrease in hyperintensity 9 days later and showed total recovery by the 23rd day after onset. In addition, the clinical symptoms were ameliorated on the 4th day after onset. Therefore, diffuse cytotoxic edema appeared to have occurred in the left MCA...

Fig. 4 A, B: Diffusion-weighted magnetic resonance (MR) images taken on the 29th postoperative day showing complete recovery of signal changes in the left middle cerebral artery (MCA) region. C-F: T1-weighted (C, D) and T2-weighted (E, F) MR images taken on the 29th postoperative day showing no deterioration in the left hemisphere compared to the preoperative images. G: MR angiogram taken simultaneously showing slightly increased intensity of the left MCA compared to the right. The intensity of the left MCA has significantly improved following carotid endarterectomy.
region due to hyperperfusion following CEA, and resulted in global aphasia, right hemiparesis, and generalized seizure. The abnormal hyperintensity decreased as the patient's symptoms improved, suggesting that diffusion-weighted MR imaging can be used for evaluating neurological function.

Intracerebral hemorrhage is associated with a high mortality of approximately 50%. Therefore, diffusion-weighted MR imaging should be performed as soon as a high risk for hyperperfusion syndrome is predicted. Based on our experience, hyperperfusion probably results first in cerebral edema which can be reversed under strict blood pressure control. Although intracerebral hemorrhage occasionally occurs without any preceding symptoms such as ipsilateral headache, strict blood pressure maintenance at normotension or even slight hypotension during the early postoperative period following CEA is essential. TCD monitoring is useful for predicting the occurrence of hyperperfusion syndrome, as indicated by markedly increased flow velocities during reperfusion. Diffusion-weighted MR imaging will reveal slower water diffusion in the MCA region at onset of the syndrome, indicating the presence of cytotoxic edema.

References


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