Aphasia 1 Week After Carotid Endarterectomy
Hypoperfusion or Hyperperfusion?

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Case Description
An 83-year-old man with a history of arterial hypertension and dyslipidemia, presented to a neurologist with a tremor of the right hand. Magnetic resonance imaging of the brain was performed, which did not show major abnormalities (Figure A and C). A routine carotid ultrasound revealed an occluded right internal carotid artery (ICA) and high-grade stenosis (>75%) of the left ICA. An uncomplicated carotid endarterectomy (CEA) of the left ICA was performed, and the patient was discharged 2 days later.

Eight days post CEA, he was admitted at our hospital with sudden onset of aphasia and confusion. On neurological examination, severe aphasia and a mild right-sided hemiparesis were documented; in addition, his blood pressure was elevated (213/75 mmHg). Computed tomography (CT) of the brain was normal and a CT angiography showed a patent left ICA. He subsequently developed fever (38.6°C) for which a lumbar puncture was performed. Analysis of the cerebrospinal fluid was unremarkable. Shortly after admission, he experienced an epileptic seizure and treatment with levetiracetam was initiated. The diagnosis of a cerebral hypoperfusion syndrome (CHS) was considered for which the blood pressure was closely monitored. Magnetic resonance imaging of the brain (Figure B and D) revealed extensive white matter changes in the left hemisphere, with no signal permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the permeability of intracranial vessels. In addition, the
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blood pH can influence autoregulation. A second contributing mechanism is hypertension, possibly mediated by baroreceptor reflex failure after receptor denervation during CEA.6

Hyperperfusion and failure of autoregulation can lead to vasogenic brain edema, which is defined as extravasation and extracellular accumulation of fluid into the cerebral parenchyma (interstitium and astrocytes) as a result of disruption of the blood–brain barrier. This is in contrast to cytotoxic edema, which occurs in cerebral ischemia and is characterized by intracellular accumulation of fluid into brain cells and cell swelling.7 When intravascular pressure remains too high, microvessels can rupture resulting in cerebral hemorrhage.8

Unless there is severe edema or intracerebral hemorrhage, CT of the brain is usually normal, and magnetic resonance is required to diagnose CHS. Magnetic resonance can show several abnormalities, including vasogenic edema, focal infarction, and hemorrhage. Vasogenic edema is most pronounced in the posterior parieto-occipital lobes and is most easily seen on FLAIR as hyperintense lesions predominately involving the white matter. On the other hand, cytotoxic edema, also seen best on FLAIR as hyperintense lesions, involves both the white and the gray matter. DWI can help distinguish the 2 types of edema as vasogenic edema will be iso- or hyperintense on DWI and hyperintense on apparent diffusion coefficient mapping.

Cytotoxic edema will be hyperintense on DWI and hypointense on apparent diffusion coefficient.4 Imaging in the present case shows extensive FLAIR hyperintensity within the white matter of the left hemisphere without any change on DWI or apparent diffusion coefficient, consistent with vasogenic edema.

CHS is a neurological emergency that can be fatal because of severe cerebral edema or intracranial hemorrhage.9 Preoperative compromise of cerebrovascular reserve and postoperative hypertension are risk factors associated with CHS after carotid revascularization.10 Therefore, patients at risk should be closely monitored, and intensive postoperative blood pressure control is critical to prevent this syndrome. Rigorous blood pressure control is essential once symptoms of CHS have developed until cerebral autoregulation is restored, which can take several weeks.10 Anti-hypertensive drugs that can cause cerebral vasoconstriction are generally preferred, and direct vasodilators and calcium channel blockers are contraindicated.3,4

**TAKE-HOME POINTS**

- Cerebral hyperperfusion syndrome after carotid revascularization is a rare syndrome characterized by the following triad: cortical neurological symptoms, headache, and seizures, typically associated with hypertension.
- Magnetic resonance imaging of the brain in cerebral hyperperfusion syndrome typically reveals vasogenic edema characterized by white matter changes on FLAIR, which can be DWI–positive with increased signal on apparent diffusion coefficient.
- Early recognition and appropriate treatment of cerebral hyperperfusion syndrome are critical if serious complications of infarction and hemorrhage are to be avoided.

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**Disclosures**

None.

**References**


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