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 mm Hg). 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 leviracetam was initiated. The diagnosis of a cerebral hyperperfusion syndrome (CHS) was considered for which the blood pressure was closely monitored. Magnetic resonance imaging of the brain (Figure B and D) revealed extensive fluid-attenuated inversion recovery (FLAIR) hyperintense white matter changes in the left hemisphere, with no signal alteration on diffusion-weighted imaging (DWI), confirming the diagnosis of CHS. The white matter changes were new because magnetic resonance imaging of the brain a few months before admission had not revealed these changes (Figure A and C). The symptoms resolved completely during a period of several days.

Discussion
Ischemic stroke is a known complication after CEA,1 caused by carotid occlusion or embolization, and was therefore suspected in this patient with sudden onset of aphasia. Early ischemic changes were absent on CT of the brain, and CT angiography showed a patent left ICA.

The clinical syndrome of aphasia, an epileptic seizure and hypertension 8 days post CEA raised the suspicion of a CHS. CHS is a rare but serious complication after carotid revascularization and can develop after both CEA and carotid artery stenting with an incidence of 1% to 2%.2,3 CHS can occur up to several weeks after revascularization but is most often seen within the first day.3 CHS is clinically best defined as a triad of headache, neurological deficit, and seizures. Typically, the headache is ipsilateral to the revascularized vascular territory, can vary in intensity, but can also be absent. The neurological deficits involve changes in consciousness, confusion, and cortical deficits (hemiparesis, hemianopsia, and aphasia); elevated blood pressure is documented in most patients. Intracerebral hemorrhage is a rare complication of CHS and occurs in <1% of patients.4

Increased ipsilateral cerebral blood flow occurs in almost all patients immediately after CEA; in some patients, this increase results in hyperperfusion (defined as an increase of >2-fold over the baseline cerebral blood flow), which can lead to clinical symptoms in a minority of these patients. Increased cerebral blood flow reaches a maximum 3 to 4 days after revascularization, but can last for several weeks.5 Although many factors are thought to be important in the pathophysiology of CHS, 2 synergistic mechanisms are of particular interest. The first is impaired cerebral autoregulation after revascularization. In patients with extracranial carotid stenosis, maximal vasodilation of cerebral arterioles is required to maintain cerebral blood supply. After reestablishing cerebral blood flow by revascularization, autoregulatory mechanisms normally prevent hyperperfusion. Several mediators have been implicated in the failure of this autoregulation in CHS. The production of oxygen-derived free radicals produced during CEA can cause endothelium dysfunction. Another factor is nitric oxide that can impair autoregulation by increasing the permeability of intracranial vessels. In addition, the
blood pH can influence autoregulation. A second contributing mechanism is hypertension, possibly mediated by baroreceptor reflex failure after receptor denervation during CEA.

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. When intravascular pressure remains too high, microvessels can rupture resulting in cerebral hemorrhage.

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. 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. Preoperative compromise of cerebrovascular reserve and postoperative hypertension are risk factors associated with CHS after carotid revascularization. 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. Antihypertensive drugs that can cause cerebral vasoconstriction are generally preferred, and direct vasodilators and calcium channel blockers are contraindicated.

**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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None.

**References**


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