

Reversible cerebral vasoconstriction syndrome is a rare cause of stroke after carotid endarterectomy

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Neurologic events after carotid endarterectomy (CEA) require prompt diagnosis and management to avoid potentially catastrophic sequelae. This report describes a 69-year-old gentleman who underwent a left CEA for a high-grade asymptomatic carotid stenosis with concomitant contralateral carotid occlusion. He had transient and crescendo neurologic events in the first 3 postoperative weeks that culminated in right hand weakness and paresthesia, despite dual antiplatelet therapy, maximal anticoagulation, and undergoing stenting of the endarterectomy site. Neurologic events recurred despite these measures and subsequent angiography showed reversible cerebral vasoconstriction syndrome that was successfully managed without further events. Reversible cerebral vasoconstriction syndrome is an unusual but important cause of neurologic events after CEA that requires aggressive and directed medical therapy. (*J Vasc Surg* 2016;■:1-4.)

Carotid endarterectomy (CEA) is often used for treatment of carotid stenosis to reduce future stroke risk.^{1,2} Postoperative increased cerebral perfusion can result in intrinsic arterial vasomotor dysregulation leading to two uncommon postoperative complications. The more common of these cerebrovascular dysregulation syndromes is cerebral edema or hemorrhage (reperfusion or hyperperfusion syndrome) and is thought to occur secondary to impaired intracranial vascular autoregulation due to prior chronic cerebral hypoperfusion.³ The other rare complication is intracranial cerebral vasoconstriction, a subtype of reversible cerebral vasoconstriction syndrome (RCVS), which is thought to also occur because of endothelial vascular dysregulation.⁴ This report describes RCVS after a routine CEA and we report on the diagnostic workup and treatment strategies, and discuss the possible pathophysiologic mechanisms. The patient consented to publication of this report.

CASE REPORT

A 69-year-old asymptomatic left-handed man was referred after his primary care physician noted a left carotid bruit. Carotid duplex ultrasound imaging demonstrated an occluded right internal carotid artery (ICA) with a high-grade stenosis of his proximal left ICA (velocity, 229/108; ICA/common carotid artery ratio, 4:1). His medical history was significant only for well controlled hypertension and remote atrial fibrillation in sinus rhythm with medical therapy. He had no history of migraine headaches,

antiphospholipid syndrome, or drug usage. Computed tomography angiography, obtained by the referring provider, showed an 80% to 90% stenosis of the proximal left ICA (*Fig 1, left*), occlusion of the right ICA, an intact circle of Willis, and no intracranial abnormalities. After careful counseling and in light of his good physiologic state, he underwent a left CEA. The endarterectomy, performed using a shunt and bovine pericardial patch, proceeded routinely including a normal completion Duplex ultrasound examination, well maintained perioperative and intraoperative hemodynamics, and he was discharged the following day. On postoperative day four, he experienced a very transient episode of right hand ataxia. Ten days later, he had another focal neurologic episode that manifested as transient right hand weakness and paresthesia, prompting admission. Computed tomography angiography and carotid duplex ultrasound imaging showed findings consistent with changes normal after CEA with no evidence of intracranial arterial pathology, subarachnoid hemorrhage, or vasospasm. After 2 days of hospitalized observation, neurology consultation, and without further symptoms he was discharged with dual antiplatelet therapy (aspirin and clopidogrel). Two days later, he experienced two similar episodes occurring 2 hours apart, manifested by the return of the same transient neurologic deficits in the right upper extremity. He again received systemic anticoagulation using intravenous heparin. Brain imaging revealed small punctate areas of infarction in the left caudate head on magnetic resonance imaging, and computed tomography angiography showed only postsurgical changes at the CEA site from the surgical clamps (*Fig 2, A*). Over the next several hours, the neurologic symptoms increased in frequency and severity (manifesting as arm weakness), despite therapeutic anticoagulation. Emergent cerebral angiography showed an area of focal irregularity at the proximal and distal extent of the CEA likely secondary to clamping (*Fig 2, B and C*). Within the intracranial circulation there was extensive left hemispheric vasculopathy noted, characterized by alternating regions of vessel narrowing and dilatation involving the middle cerebral and anterior cerebral artery branches as well as the very unusual involvement of the ophthalmic artery (unusual because it is rarely involved in cerebral vasculopathy because it is not in the subarachnoid space). Because of the repetitive clinical events despite maximal

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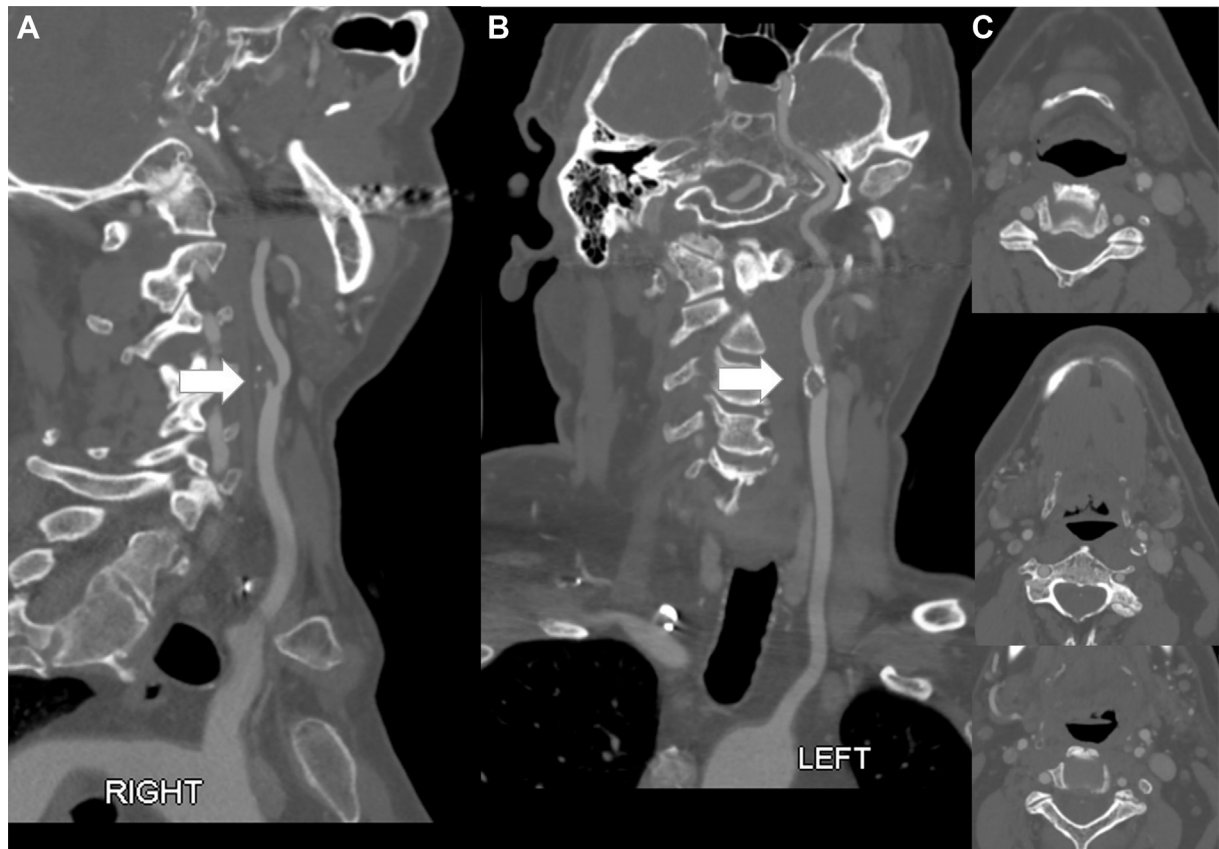


Fig 1. Preoperative computed tomography angiography showing **A**, an occlusion of the right internal carotid artery (ICA; *white arrow*), and **B**, 80% to 90% stenosis of the proximal left ICA (*white arrow*). **C**, The cross-sectional imaging at the level of the carotid bulb and proximal ICA.

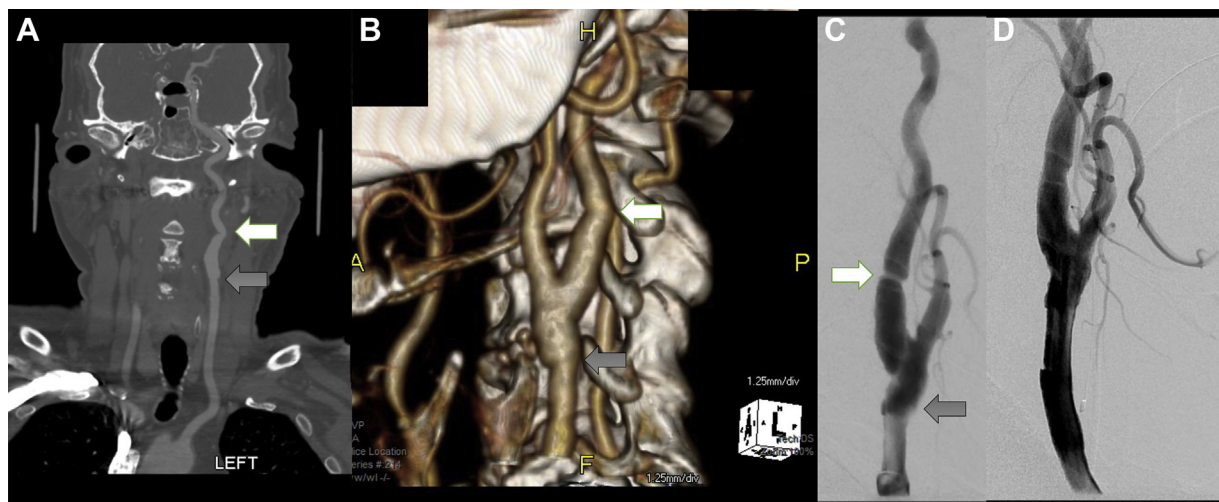


Fig 2. After the patient had repeated neurologic events, he underwent noninvasive imaging with ultrasound (not shown). **A** and **B**, Computed tomography angiography; and magnetic resonance angiography (not shown) (proximal endpoint depicted with the *gray arrow* and the distal endpoint noted with the *white arrows*). **C** and **D**, Subsequent cerebral angiography showed similar findings with a caliber change at the proximal endarterectomy site (*gray arrow*) and a possible kink/clamp site at the distal endpoint (*white arrow*). Because of the concern for these as possible embolic sources despite dual antiplatelet therapy, the carotid artery was stented using an uncovered 7- to 10-mm tapered stent.

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