Cerebral hyperperfusion syndrome after carotid endarterectomy: case report

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Abstract

Prevention of disability from cerebrovascular accident through carotid endarterectomy, neurovascular stenting, and endovascular treatment of stroke are among the most promising treatments for neurovascular diseases. Cerebral hyperperfusion syndrome is a rare complication of these procedures, and this case report illustrates a patient with this condition after carotid endarterectomy. The mechanism of hyperperfusion is thought to be from chronic compensatory small vessel dilation to maintain cerebral perfusion in the context of vascular stenosis. Treatment is largely supportive with strict blood pressure control. As treatment options for neurovascular reperfusion become mainstay, emergency clinicians need to be appraised to post procedural complications such as cerebral hyperperfusion.

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Introduction

Prevention of disability from cerebrovascular accident through carotid endarterectomy, neurovascular stenting, and endovascular treatment of stroke are among the most promising treatments for neurovascular diseases. The rapid increase in these treatment modalities will undoubtedly be accompanied by postprocedural complications.[1,2] This case presents the uncommon complication of cerebral hyperperfusion syndrome after a carotid endarterectomy.

Case Report

The patient is a 70-year-old female with a past medical history of coronary artery disease and carotid stenosis presenting with left-sided weakness, left-sided facial droop, and right gaze deviation. She was 6-days days postoperative from a right carotid endarterectomy. She was last seen well when she went to sleep that evening.

Her family denied any trauma or concerns during the prior day. Her carotid endarterectomy was reportedly uncomplicated, with her only postoperative problems being elevated blood pressure, managed with oral medication dose adjustments, and feelings of right eye pressure.

Her initial vital signs showed a blood pressure of 137/86 mmHg, heart rate of 78 beats per minute, temperature of 36.1 degrees Celsius, respiratory rate of 17 breaths per minute, oxygen saturation of 100% on room air, and blood sugar of 118 mg/dL. Due to her initial physical exam showing left-sided weakness and left facial droop, the initial concern was an acute ischemic stroke, and computed tomography imaging was prioritized. Her blood sugar was 90 mg/dL. While in the computed tomography scanner, the patient had a generalized tonic-clonic seizure, which self-resolved within one minute.

The patient's computed tomography of the brain without contrast showed asymmetric, ill-defined hypodensities in the right frontal lobe periventricular white matter without intracranial hemorrhage (Figure 1). Computed tomography angiography did not show cerebrovascular stenosis or occlusion, and perfusion imaging showed symmetric flow and volume throughout the brain. These findings were discussed with the neurointerventional specialist and neurologist and determined to be secondary to cerebral hyperperfusion syndrome. The patient was admitted to the intensive care unit for strict systolic blood pressure control with a goal of less than 130 mmHg. This was achieved with intermittent intravenous

labetalol 10 to 20mg. Intravenous levetiracetam 1000mg followed by 500mg twice daily was given for seizure prophylaxis and the patient made gradual improvements in speech and strength. She was transferred to a rehabilitation facility and later she was discharged home with only minimal left leg weakness as her only neurologic deficit.

Discussion

Cerebral Hyperperfusion Syndrome (CHS) is a rare complication of Carotid Endarterectomy (CEA), Carotid Angioplasty with Stenting (CAS), and endovascular thrombectomy.[1,2] The proposed mechanism of hyperperfusion is related to the chronic compensatory small vessel dilation to maintain cerebral perfusion in the context of vascular stenosis. Autoregulation is initially impaired in these vessels when the stenosis is repaired. Endothelial dysfunction due to free radicals and the resulting hyperperfusion damages the capillary beds leading to cerebral edema and hemorrhage.[2-4] Presenting symptoms occur within two weeks of CEA including ipsilateral positional headaches, ipsilateral eye or facial pain, seizures, and intracerebral hemorrhage.[2]

Risk factors include revascularization of high-grade (greater than 80 percent) stenosis, recent cerebral infarction, contralateral carotid occlusion, and post-operative hypertension.[5] CHS after CEA occurs at rates of 1 to 3 percent. Associated intracerebral hemorrhages in this cohort have an incidence of 0.6 percent with mortality as high as 26 percent.[3,4,6]

CHS is primarily a clinical diagnosis. Identifying cerebral edema or intracranial hemorrhage associated with it is achieved with Computed Tomography (CT), Computed Tomography Angiography (CTA), or Magnetic Resonance Imaging (MRI) with T2 or FLAIR sequences. Imaging findings are ipsilateral to the side of the carotid artery procedure. Edema is classically within the white matter although the cortex may also be involved. These modalities can assist in ruling out alternative pathologies including vessel dissection or rupture. Diffusion weighted imaging on MRI distinguishes cerebral ischemia from edema. CT perfusion may see increased cerebral blood flow, increased cerebral blood volume, shortened mean transit time, and shortened time to peak. Flow alterations may also be identified on Near-Infrared Spectroscopy (NIRS) or transcranial doppler.[2,3,5,7-9] Evaluations with these modalities have shown cerebral artery flow velocity up to three times the contralateral hemisphere; however, the degree of increase does not correlate with disease severity. Even moderate hyperperfusion (20 to 44 percent increase) may still cause cerebral hyperperfusion syndrome.[6]

Prevention with strict post-operative hypertensive control is recommended with systolic blood pressure goals ≤140/90 mmHg or within 20% of baseline blood pressure in the context of pre-existing hypertension. Recommended medications include labetalol, clonidine, nitroglycerin, and, particularly, nicardipine, as it is rapidly titratable.[2,6]

Conclusions

Cerebral hyperperfusion syndrome is an uncommon complication of cervical or cerebrovascular reperfusion. As treatment options for neurovascular reperfusion continue to expand, vigilance of post procedural complications such as this will be critical to help prevent further disability.

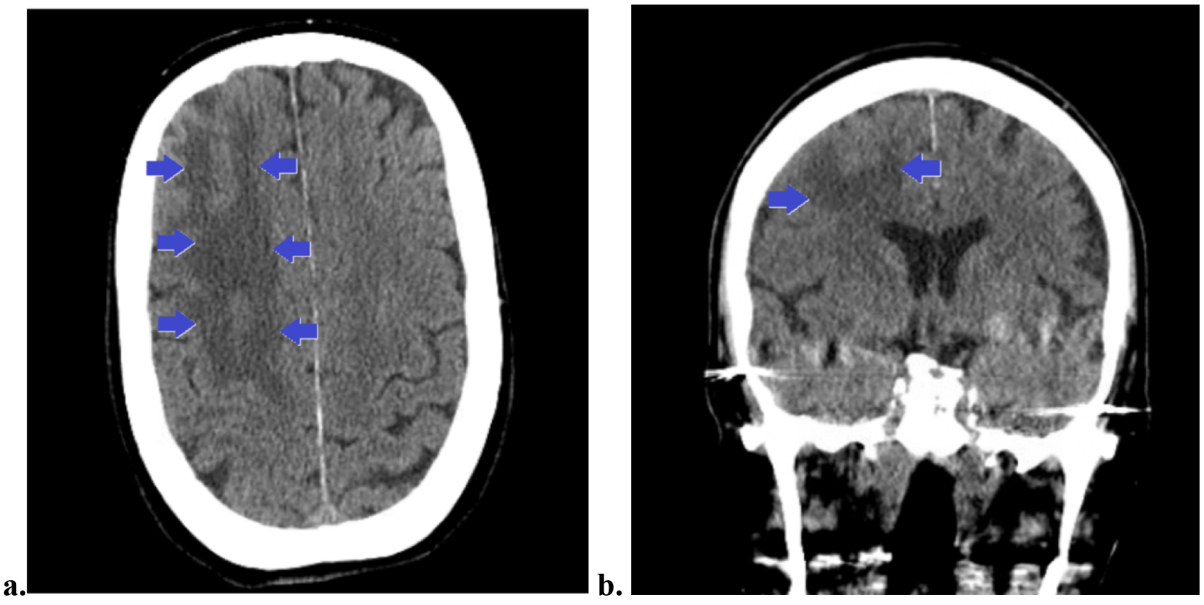

Figure 1. Axial view (**a**) and coronal view (**b**) of computed tomography of the brain showing right frontal hypodensities as indicated by the blue arrows.

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