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Cerebral Hyperperfusion Syndrome: A Case Report and Review of Literature

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Abstract

Large clinical trials have established the superiority of carotid endarterectomy for stroke prevention in patients with symptomatic critical carotid artery stenosis. Although postoperative complications are commonly ischemic, cerebral hyperperfusion syndrome represents a rare but potentially treatable diagnosis. Outcomes are contingent on early identification, prevention and management of precipitating factors. Herein, we report the case of a patient who presented with altered mentation, hemiparesis, aphasia and seizures, nine days after left carotid endarterectomy. The underlying pathophysiological mechanism, associated risk factors, screening, prevention and management of cerebral hyperperfusion syndrome are discussed.

Keywords: Stenosis; Syndrome; Hyperperfusion injury

Introduction

Carotid Endarterectomy (CEA) largely remains the preferred modality of treatment for symptomatic severe (>70% diameter) carotid artery stenosis [1], with a possible benefit in patients with asymptomatic carotid stenosis [2]. Post-operative neurological complications are usually ischemic in nature; patients rarely develop reperfusion or hyperperfusion injury. Although the terms are often used interchangeably, reperfusion suggests normalization of cerebral blood flow as opposed to hyperperfusion which implies excessive blood flow (classically >100% increase) when compared to a pre-or intra-operative baseline [3,4]. Cerebral hyperperfusion syndrome (CHS) is a clinical diagnosis characterized by a triad of ipsilateral headache, seizure, and focal neurological deficits [5] which commonly presents during the first month post-operatively [4,5]. Outcomes are contingent on timely recognition and prevention of precipitating factors. The underlying pathophysiology is multi-factorial with complex interplay between cerebral dysautoregulation, hypertension, oxidant production and microvascular hyperpermeability secondary to ischemic-reperfusion injury. We present a patient who developed CHS nine days after carotid endarterectomy.

Case Presentation

An 83-year-old female presented with a grand mal seizure, emesis, inability to speak, and leaning towards her right. Nine days prior to presentation, she had undergone left carotid endarterectomy for an asymptomatic stenosis, which rapidly progressed to 80%. Post-operatively, she was discharged to an assisted living facility. Her attendant at the assisted living reported that her blood pressure had been "very high" since her recent surgery despite adherence to her medication regimen. On examination, she was awake, non-verbal and not following commands, with right hemiparesis and a blood pressure of 231/94 mmHg. Her past medical history was significant for chronic hypertension, dyslipidemia, and severe right internal carotid stenosis for which she underwent endarterectomy thirteen years earlier. She had no known drug allergies, and had been taking aspirin, lisinopril, amlodipine, nebivolol and atorvastatin.

Computerized Tomography (CT) of the head showed diffuse left hemispheric subcortical hypodensities, and CT angiography revealed no abnormal vasculature. A rapid-sequence magnetic resonance imaging (MRI) was performed, which revealed diffuse subcortical hyperintense signals on FLAIR. Diffusion-weighted imaging showed minimal increase in signal. Based on her recent surgery, hypertension and imaging findings, CHS was suspected, and intravenous nicardipine was initiated to achieve blood pressure <140/90 mmHg. She also received mannitol and levetiracetam. Aspirin and nebivolol were discontinued. Her mental status gradually improved over the next several days with no focal neurologic deficits, and she was discharged to a rehabilitation facility after adequate titration of her antihypertensive medications.

Discussion

Sundt first defined CHS after a successful CEA in 1981 as the combination of increased arterial blood pressure and the clinical trial of ipsilateral headache, seizure and focal neurologic deficits in the absence of cerebral ischemia [5]. Estimated to occur in only 0.7-3.0% of patients [4,6], CHS is an important complication that can lead to significant morbidity and mortality if not recognized and adequately treated [4]. Impaired cerebral autoregulation in patients with severe internal carotid artery stenosis may be the initial step in the cascade leading to CHS [3]. The duration and severity of hypoperfusion determines the degree of microvascular dysautoregulation and production of vasodilatory substances leading to structurally defective and weaker capillaries. Additional microvasculature damage secondary to chronic hypertension and amyloid deposition, and baroreceptor denervation, which is common after successive bilateral carotid endarterectomies, may compound the risk for hyperperfusion post-operatively [7,8].

Immediate reperfusion after CEA in previously hypoperfused tissue results in increased intracapillary pressure in already maximally dilated vessels. This ischemic-reperfusion injury, characterized by oxidant production and complement activation, prompts disruption of capillary endothelial cells, breakdown of the blood brain barrier, and edema in the cerebral white matter [9].

There are no data from randomized clinical trials for optimal peri-operative management of patients with CHS. Therefore, prevention is critical and is based on early recognition of risk factors for hyper-

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perfusion after carotid revascularization. Adequate control of postoperative hypertension is believed to be the single most crucial preventative strategy [4,6]. In the background of defective cerebral auto-regulation, increased systemic blood pressure places an inordinate burden on these dilated vessels, which then leads to edema and hemorrhage [3]. This loss of reflex microvascular contractility can be identified pre-operatively by assessing cerebrovascular reactivity to acetazolamide using single-photon emission CT (SPECT) [4], or to carbon dioxide using transcranial Doppler ultrasonography (TCD) and more recently, Blood Oxygen Level-Dependent (BOLD) functional Magnetic Resonance imaging (fMRI) [7].

Conclusion

These patients who at risk for hyperperfusion are likely to benefit from aggressive post-operative blood pressure monitoring and control, and education regarding the importance of reporting symptoms such as major headache, confusion, or seizures that occur within the first month after CEA. Post-operatively, hypertension should ideally be controlled using beta-blockers, rather than angiotensin converting-enzyme inhibitors that may further increase cerebral blood flow or nitrates with their vasodilatory effects. CHS is a rare but serious complication of carotid revascularization, including carotid endarterectomy and carotid stent placement. Patients should be educated about the importance of early reporting of symptoms such as major headache, confusion, or seizures occurring within the first month after CEA.

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