CASE REPORT

Braz J Cardiovasc Surg 2007; 22(1): 116-118

Cerebral hyperperfusion syndrome occurring three weeks after carotid endarterectomy

Síndrome de hiperperfusão (pós-operatória) após três semanas da endarterectomia de carótida

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RBCCV 44205-877

Abstract

Cerebral hyperperfusion syndrome is a recognized complication of carotid endarterectomy. Various studies have documented an incidence of 0.3% to 1.2%. It occurs in the setting of sudden reperfusion of a chronically hypoperfused hemisphere. We present here a case of a 48-year-old lady who developed cerebral hyperperfusion syndrome three weeks after undergoing a carotid endarterectomy for high-grade carotid artery stenosis.

Descriptors: **Carotid endarterectomy, complications. Cerebrovascular accident. Cerebrovascular disorders, etiology.**

Resumo

A síndrome de hiperperfusão pós-operatória (SH) é uma complicação conhecida após a endarterectomia de carótida. Vários estudos têm demonstrado uma incidência de 0,3% a 1,2%. Isso ocorre em situações de reperfusão súbita de um hemisfério com hipoperfusão crônica. Apresentamos, neste trabalho, o caso de uma paciente de 48 anos que desenvolveu SH três meses após ser submetida a uma endarterectomia de carótida por estenose grave da artéria carótida.

Descritores: **Endarterectomia das carótidas, complicações. Acidente cerebrovascular. Transtornos cerebrovasculares, etiologia.**

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Article received in November 19th, 2006
Article accepted in January 18th, 2007

INTRODUCTION

Embolization is one of the leading causes of perioperative stroke following carotid endarterectomy. Cerebral hyperperfusion may cause complications in the immediate postoperative period too, presenting at times with a devastating intracranial hemorrhage. Though cerebral hyperperfusion syndrome (CHS) commonly presents early after carotid endarterectomy, it may at times present later or may be missed in the immediate aftermath of surgery due to its subtleness with devastating consequences later on. We present a patient who developed the full hyperperfusion syndrome three weeks after surgery and discuss her presentation and management.

CASE REPORT

A 48-year-old lady presented to our emergency room with headache, slurring of speech and right arm and leg weakness. Blood pressure at the time of presentation was recorded as 235/115 mm Hg. Her history was significant for high-grade carotid artery stenosis.

She had complete occlusion of the internal carotid artery on the right and an 88% stenosis of the left internal carotid artery. She had undergone a left carotid endarterectomy in another hospital three weeks prior to her current hospitalization with no complications reported during the surgery or at the start of the immediate post operative period apart from the development of left sided headaches. She had no other risk factors for cerebral atherosclerosis apart from triglyceridemia (serum triglycerides 378 mg/dL, Normal range 150-200mg/dL) and a strong family history of carotid artery disease; both her parents had history of carotid artery stenosis.

A neurological examination revealed a conscious young woman who had expressive and a component of receptive aphasia. She had dense right-sided hemiplegia (power 1/5 right arm and leg Medical Research Council grade). She had loss of fine touch, two-point discrimination and graphesthesia on the right. Right plantar was equivocal and left was down-going. Computed tomography of the head showed left parieto-occipital hypodensity with vasogenic edema predominantly in the surrounding white matter (Fig. 1 and 2).

There was an intracranial hemorrhage. Computed angiography showed a widely patent left internal carotid artery and complete occlusion of the right internal carotid artery at the level of the carotid siphon. Her blood pressure was difficult to control requiring intravenous boluses of Labetalol. She was transferred to the medical ICU for better control of her blood pressure.

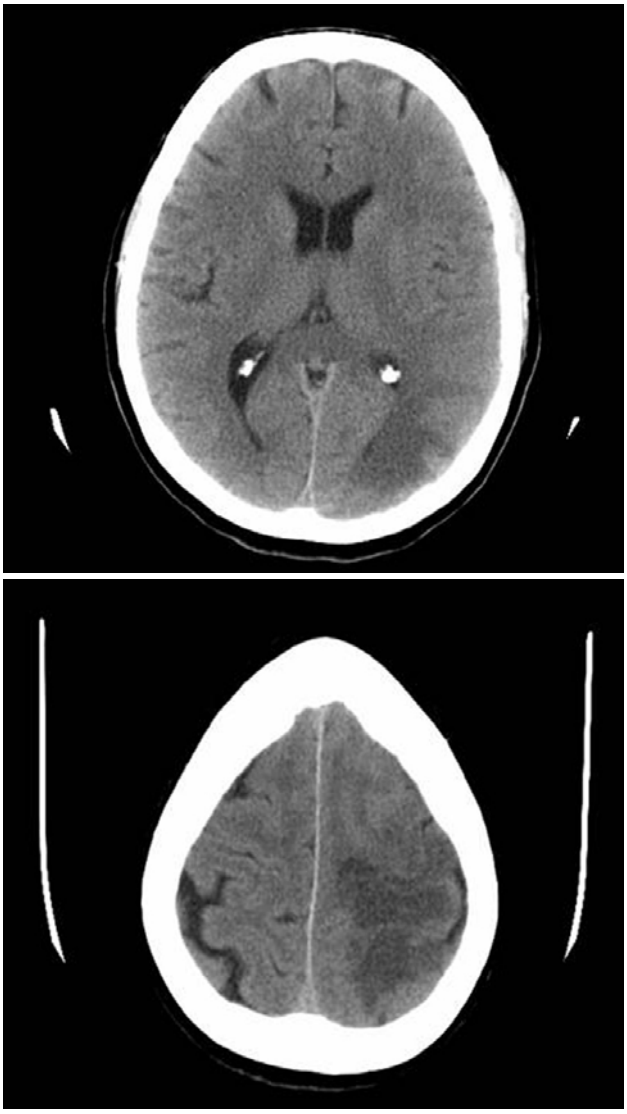

Fig. 1 and 2 - CT scans showing left parieto-occipital hypodensity with surrounding white matter edema suggestive of hyperperfusion injury

DISCUSSION

Cerebral hyperperfusion syndrome (CHS) is generally considered to occur as a result of impaired autoregulation of cerebral blood flow in a chronically hypoperfused hemisphere. CHS after carotid endarterectomy is characterized by ipsilateral headache, hypertension, seizures

and focal neurological deficits [1,2]. The understanding is that hypoperfusion induced by severe carotid artery stenosis results in compensatory dilation of cerebral blood vessels distal to the stenosis as part of the normal autoregulatory response to maintain adequate cerebral blood flow [3]. The syndrome usually occurs in the immediate post-operative period and may present with fatal cerebral hyperperfusion hemorrhage [4].

It has been reported after both carotid endarterectomy and carotid stenting [5]. Delayed cerebral hyperperfusion syndrome has been described before though it is rare and is thought to occur due to prolonged impairment of cerebrovascular autoregulation. Our patient developed the full cerebral hyperperfusion syndrome three weeks after her carotid endarterectomy; it is possible however that the syndrome started at the time she started complaining of ipsilateral headaches, in the immediate post-operative period following her carotid endarterectomy.

Her blood pressure was hard to control requiring transfer to the medical ICU. We attempted a neuroprotective strategy using magnesium and attempted to lower the intracranial edema with the use of albumin [6].

In spite of our best and aggressive efforts, she was left with a dense right sided hemiplegia and receptive as well as expressive aphasia. When last seen at follow up, two months after her first presentation to our hospital, she is able to ambulate from bed to bathroom with the aid of a walker. Expressive aphasia still persists, though at times she is able to formulate a 5 to 6 word sentence with effortful speech. Knowledge of CHS among physicians is limited and if the syndrome is not detected and aggressively treated it can lead to fatal cerebral edema. Further studies are warranted to predict accurately which patients are at high risk of developing this potentially fatal complication of carotid surgery [4].

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