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Cerebral hyperperfusion syndrome following carotid endarterectomy

Thomas Saliba[1*], Olivier Cappeliez[2], Henri Maisonnier[2], Sanjiva Pather[2], Ilias Bennouna[2]

[1]CHUV Service de Radiodiagnostic et Radiologie Interventionnelle, Rue du Bugnon 46, CH-1011 Lausanne, Vaud, Suisse

[2]Radiology department, Hôpital de Braine L'Alleud, Braine L'Alleud, Belgium

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Abstract

Introduction: Cerebral hyperperfusion syndrome (CHS) is a rare but potentially fatal complication following carotid revascularization procedures, particularly carotid endarterectomy (CEA), which is commonly used to treat extracranial carotid stenosis, which is a cause of approximately 15% of all strokes. CHS is characterized by non-ischemic headaches, seizures, and neurological deficits.

Case Report: A 73-year-old woman presented with convulsions three days after undergoing a left CEA for 97% stenosis. Initial symptoms included blurred vision, vertigo, and facial paralysis. Imaging revealed left cerebral hemisphere hypervascularization on CT and vasogenic oedema on MRI, consistent with CHS. Despite clinical deterioration, the patient fully recovered following corticosteroid treatment and was discharged without sequelae.

Discussion: CHS symptoms range from mild headaches to seizures and coma. Although uncommon, the condition carries a high risk of mortality. Pathophysiology involves impaired cerebral autoregulation due to chronic ischemia. Diagnosis relies on imaging, though no standardized criteria exist.

Conclusion: Given its delayed onset and potentially devastating consequences, CHS must be considered in post-CEA or stenting patients. Early recognition and management are essential to prevent adverse outcomes.

Keywords: *cerebral hyperperfusion, carotid endarterectomy, complication*

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Introduction

Extracranial carotid stenosis causes approximately 15% of all strokes, with carotid endarterectomy (CEA) being shown to be an effective treatment in patients under 75 years old. (1)

Cerebral hyperperfusion syndrome (CHS) is a clinical syndrome following revascularisation procedures. (2) It is a rare but potentially life-threatening complication that was first described in patients having undergone CEA. (2) The syndrome comprises headaches, neurological deficits and seizures that are non-ischemic in origin. (2)

As the rate of procedures to treat carotid occlusions increases, the syndrome is becoming better known, with several studies being conducted to establish the prevalence of the condition. (2)

Some studies show that CHS is more likely to occur post CEA in patients who have intracranial stenosis >90%, severe contralateral intracranial atherosclerosis and chronic hypertension, with one study suggesting that the risk of CHS was 16% in patients with all three criteria. (2) Other suggested risk factors are female sex, chronic renal failure, left sided carotid atherosclerosis, previous haemorrhage, microvascular disease, previous cerebral injury and progressive neurological deficits. (2,3) However, some studies put state that only 0-3% of patients who underwent CEA will develop CHS, with around half of the patients going on to have strokes. (1,3) When CHS does occur, the

results can be devastating, with a mortality rate of up to 50% (3).

Case Report

A 73-year-old woman presented to the emergency department for convulsions. The patient had undergone a left CEA three days prior for stenosis of 97% in the left common carotid artery (Fig. 1). Upon admission, the patient related that she had suffered blurred vision followed by vertigo and left facial paralysis. This prompted an immediate contrast-enhanced cerebral CT-scan which revealed left-sided hypervascularisation when compared to the contralateral side (Fig. 2). The patient's condition rapidly degraded in the 30 minutes following the CT scan. The patient entered a coma with clonic forehead movements, trismus, laboured breathing and hypersalivation.

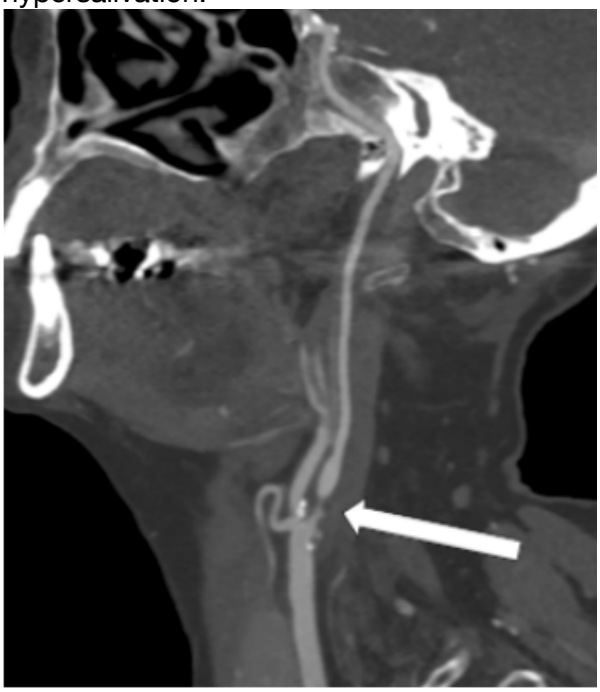


Figure 1
Contrast-enhanced angio-CT of the arteries of the neck showing a nearly complete stenosis of the left common carotid artery (arrow).

The patient was given an emergency brain MRI, revealing sub-cortical vasogenic edema in the left fronto-parietal and thalamic regions, which were interpreted as signs of hyperperfusion after CEA endarterectomy (Fig. 3 and 4). The patient was transferred to the intensive care unit, where she received an electroencephalogram that was shown to have no abnormalities. The patient received corticosteroids to ease the inflammation and was discharged home two days later, having fully recovered. The patient attended a neurology consultation one month later, confirming that she had fully recovered from the incident and had no sequelae.

Discussion

The type of symptoms varies greatly, from mild to potentially life-threatening. (2) The most common presentation is a severe ipsilateral, though sometimes diffuse, headache, with eye and facial pain. (2) More severe symptoms can be seizures, loss of consciousness, nausea, vomiting, hypertension or neurological deficits. (2) Some reports indicate that CHS occurs significantly sooner in patients having undergone carotid stenting, peaking at 12 hours after the procedure, compared to 6 days in patients having undergone CEA, though a large meta-analysis found most cases also occurred around 12 hours post-procedure with no cases found after 6 days. (1,2) Subsequently, intracranial haemorrhages also occur sooner in stenting patients, peaking at around 2 days post-procedure, compared to 11 days for endarterectomy patients, though some cases can present up to one month later, possibly leading to an underestimation of the associated risks. (1,2) Patients should therefore be made aware of the risk of developing CHS after discharge (1).

The exact pathophysiological mechanism is unclear, though the most widely accepted suggestion is the impairment of cerebral autoregulation, which would normally constrict the cerebral blood vessels in response to a sudden increase in blood flow, due to chronic ischemia. (2,3) This reaction includes both a neurogenic and myogenic component, with the myogenic being the to react. (2) If this path is deficient, the sympathetic system should take over but if this also fails CHS can develop (2).

The most common radiological modality used for detecting CHS is transcranial doppler, used to measure the velocity of the blood flow in the middle cerebral artery, which correlates with cerebral blood flow, though this technique is not always reliable or practically feasible. (2,4) Cerebral perfusion imaging, either SPECT-CT, CT perfusion or perfusion weighted imaging can be used to estimate cerebral blood flow. (2) CT perfusion is the preferred method due to availability, short acquisition time and technical feasibility in most patients, alongside the ability to compare the blood flow in each cerebral hemisphere. (2) New MRI techni-

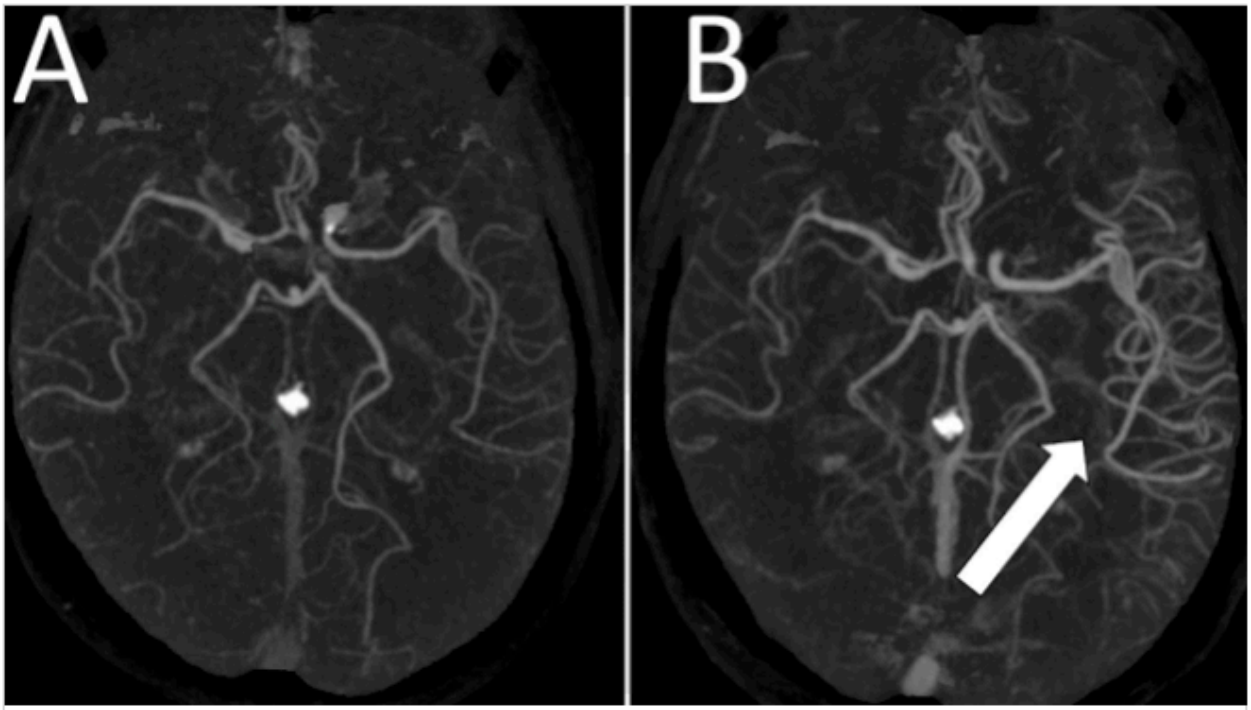


Fig. 2 Contrast-enhanced CT-angiography of the head showing imagery acquired before the patient underwent a carotid endarterectomy (part A) and during the patient's admission to the emergency department (part B), where there is increased vascularisation on the left side hemisphere (arrow).

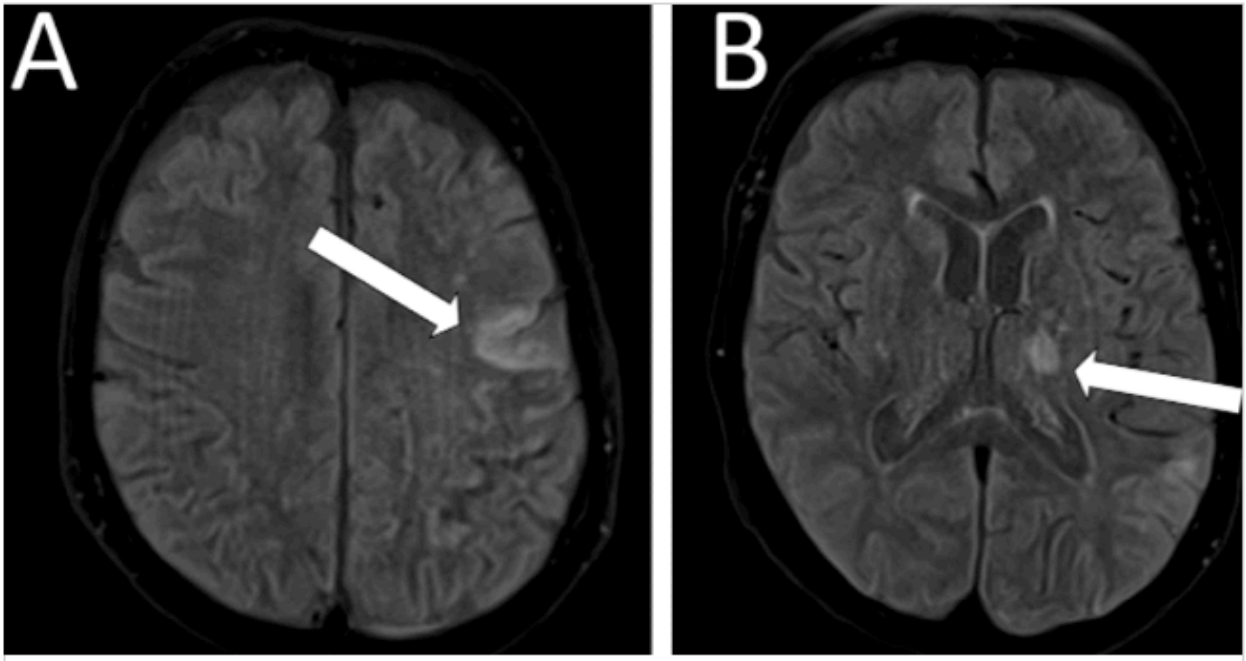


Fig. 3 1.5T brain MRI with FLAIR sequences showing a hyperintense signal in the left frontal cortex (part A, arrow) and the thalamic region (part B, arrow).

ques such as arterial spin labelling can be used to estimate cerebral blood flow without the use of contrast agents and predict the risk of patients developing CHS in a pre-intervention exam (2,4).

When using quantitative methods like SPECT -CT, cerebral hyperperfusion is defined as cerebral blood flow that exceeds a baseline by 100% or more compared to what was established before the vascular procedure, though in practice few patients have pre-procedure exams for comparison (2,4). Therefore, a comparison with the contralateral side is most commonly done, though this is unreliable in patients with contralateral stenosis. (2) For transcranial doppler, increased velocity by 1.5 – 2 times the baseline may be predictive of CHS. (2) The proposed

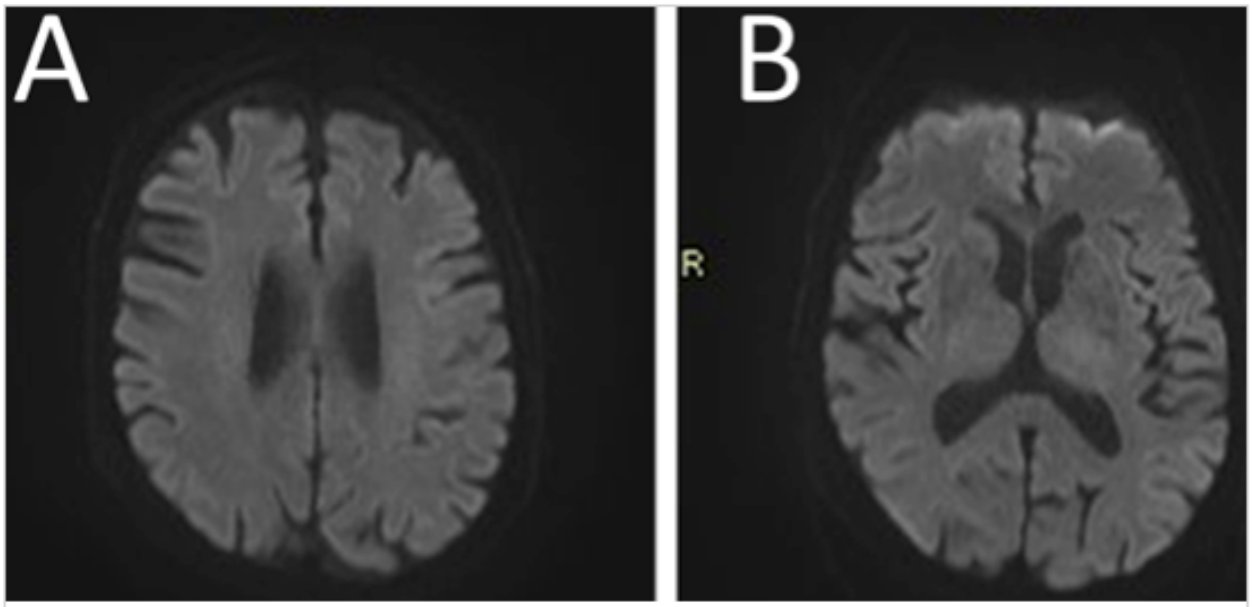


Fig. 4 1.5T brain MRI with diffusion weighted sequences showing the lack of diffusion restriction in the left frontal cortex (part A) and the thalamic region (part B), proving that the edema is of vasogenic nature.

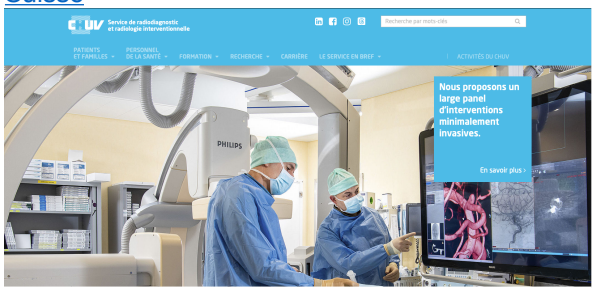
criteria for CT perfusion are elevated post-operative cerebral blood volume, mean transit times thresholds of <2s or >4s, regionally augmented cerebral blood flow and blood flow, decreased mean transit time and decreased time to peak in the clinically linked artery. (2) Overall, however there is no consensus on the diagnostic criteria. Furthermore, one must note that a negative radiological exam cannot exclude CHS.

Other conditions that can cause CHS are in post-operative intracranial atherosclerosis and acute ischemic stroke patients and after recanalization of the vertebral, subclavian or external carotid arteries (2).

Conclusion

CHS is a rare but potentially life-threatening condition that occurs after vascular surgery of the arteries supplying the brain. As it can occur with a varying amount of delay after the procedure, often after patient discharge, clinicians and radiologists must be vigilant when examining patients with a recent history of CEA and carotid stenting.

Correspondence to
Dr. Thomas Saliba
https://orcid.org/0000-0001-6989-9577
CHUV
Service de Radiodiagnostic et Radiologie Interventionnelle
Rue du Bugnon 46
CH-1011 Lausanne, Vaud
Suisse



Conflict of interest:

The authors declare that there were no conflicts of interest within the meaning of the recommendations of the International Committee of Medical Journal Editors when the article was written.

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