

case report

Cerebral hyperperfusion syndrome after carotid angioplasty

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Background. Cerebral hyperperfusion syndrome after carotid endarterectomy is an uncommon but well-defined entity. There are only few reports of »hyperperfusion injury« following carotid angioplasty.

Case report. We report an unstable arterial hypertension and high-grade carotid stenosis in a 58-year-old, right-handed woman. After a stroke in the territory of middle cerebral artery carotid angioplasty was performed in the patient. Among risk factors, the long lasting arterial hypertension was the most pronounced. Immediately after the procedure, the patient was stable without any additional neurologic deficit. The second day, the patient had an epileptic seizure and CT revealed a small haemorrhage in the left frontal lobe.

Conclusions. The combination of a high-grade carotid stenosis and unstable arterial pressure is probably an important prognostic factor in the pathogenesis of hyperperfusion syndrome.

Key words: carotid artery diseases; angioplasty - adverse effects; reperfusion injury, hyperperfusion syndrome; hypertension

Introduction

Cerebral hyperperfusion after carotid endarterectomy is an uncommon, but well-defined entity.¹ Despite the increasing use of carotid anigoplasty, there are only few reports of »hyperperfusion injury« following carotid angioplasty in the literature.²⁻⁵ The syndrome occurs in a number of clinical settings and is characterised by the diagnostic triad of unilateral headache, seizures, and intracranial haemorrhage. In our case, the pa-

tient developed typical signs of hyperperfusion syndrome detected from typical computed tomography (CT) findings.

Case report

In September 2001, a 58-year-old, right-handed woman was referred to our Department after an ischemic stroke. In 1995, she had an anterior circulation cerebrovascular accident but she has made a good recovery. Her medical history included long-lasting arterial hypertension for more than 20 years and hyperlipoproteinemia. There was no history of cigarette smoking or excessive alcohol intake. In spite of regular ACE-inhibitor treatment, her blood pressure fluctuated. She had been treated with Aspirin 100 mg daily and statin 20 mg.

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When assessed in our hospital before carotid angioplasty she had a residual expressive dysphasia, as well as a mild weakness in the face and limb on the right. Her blood pressure was 180/110 mm Hg. Computed tomography scan (CT) of the brain revealed widened liquor spaces, pre-existed ischemic lesions up to 1 mm in size, located deep in the left cerebral hemisphere, in the left frontal lobe and subcortically in the left parietal lobe. There was no evidence of haemorrhage. All haematological and biochemical tests were normal with a normal platelet count and coagulation screen. Duplex ultrasonography made in 2001 revealed a 90-per-cent stenosis of the left internal carotid artery (ICA) produced by echolucent plaque, type I (according to the accepted international classification). The plaque was unstable and had an irregular surface (Figure 1).

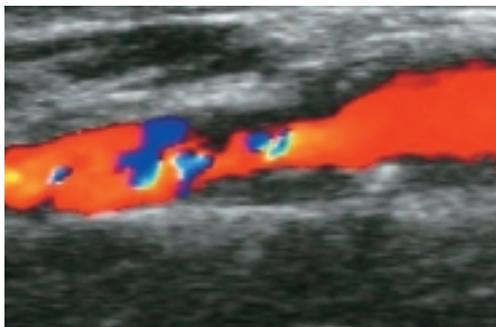


Figure 1. Colour Doppler ultrasound shows a 90-per-cent stenosis of the left internal carotid artery.

The patient underwent the left carotid angioplasty through the femoral approach under local anaesthesia. Intra-arterial digital subtraction angiography confirmed a 95-per-cent stenosis of the left ICA (Figure 2a). The patient was given 5000IU of heparin IV. The stenosis was crossed with a flexible coronary guidewire (V-18 Control Wire; Boston Scientific Corp). Glycopyrrolate and 0.5 mg atropine IV were administered during the procedure. The stenosis was predilated with a low-profile coronary balloon (4 x 20 mm

Bypass Speedy Monorail Catheter, Boston Scientific Corp) and stented with a 7 x 30 Carotid Wallstent Monorail (Boston Scientific Corp). The stent was dilated with a 5.5 x 20 mm Bypass Speedy Monorail Catheter (Boston Scientific Corp) that embedded it firmly into the vessel wall.

The blood pressure varied between 160/90 mm Hg and 175/105 mm Hg during the procedure, but there were no residual adverse neurological *sequelae*. Postprocedural angiogram showed no significant stenosis or dissection (Figure 2b). In the following 24 hours, the patient was treated with Aspirin



Figure 2a. Digital subtraction angiography. Lateral views of the left carotid artery bifurcation. High grade circumferential, atherosclerotic stenosis of the internal carotid artery origin before carotid stenting.

and clopidogrel, her blood pressure varied between 140 and 160/95 mm Hg and she was clinically stable. On the following day she was dismissed. She did not continue antihypertensive therapy when she was at home because she was convinced that she did not need this therapy after carotid stenting.

After 2 days she was urgently re-admitted because of a grand mal type epileptic seizure. After the seizure she had a transient left right-sided hemiplegia. Blood pressure at the time of admission was 180/100 mm Hg. An urgent brain CT revealed a small haemorrhage in the left frontal lobe (Figure 3). Colour Doppler ultrasound of the ICA revealed a visibly patent vessel (Figure 4). The peak systolic velocity rose to 2.3 m/s, with the end diastolic

velocity of 1.2 m/s. The patient was managed conservatively. Hypertension was easily controlled with 10 mg enalapril twice daily. The antiepileptic therapy was introduced. She recovered completely after two weeks.

Discussion

Cerebral hyperperfusion syndrome (CHS) may manifest as ipsilateral headaches, seizures, or intracerebral haemorrhages. Risk factors such as high-grade stenosis, contralateral carotid occlusion, poor collateral flow, chronic ipsilateral hypoperfusion, preoperative and postoperative hypertension, and perioperative use of anticoagulant or antiplatelet agents have been reported.¹ In our case, we do not have pathologic evidence to support hyperperfusion injury as a cause of the haemorrhage after CAS, but clinical feature and postprocedural systemic hypertension, together with the lobar appearance of the haemorrhage, indicate to the mechanism of hyperperfusion injury. Angioplasty performed in the patients with high degree carotid stenosis proved that the stenosis was associated with poststenotic drop in perfusion pressure. Therefore, it is likely that the patient suffered a chronic ischemia, which can cause a loss of autoregulation. This could be an important pathophysiologic mechanism of hyperperfusion injury.⁶

CHS has been defined as cerebral blood flow (CBF) in excess of that required for metabolic needs or a postoperative increase greater than 100% of the preoperative cerebral blood flow.⁷ Therefore, when cerebral autoregulation is impaired, the elevated blood pressure could increase CBF. In our case, the patient had unstable arterial pressure with tendency toward high pressure. During hospitalisation, the arterial pressure did not exceed 145/90 mm Hg and the patient did not use antihypertensive medication. According to our experiences, the heart rate

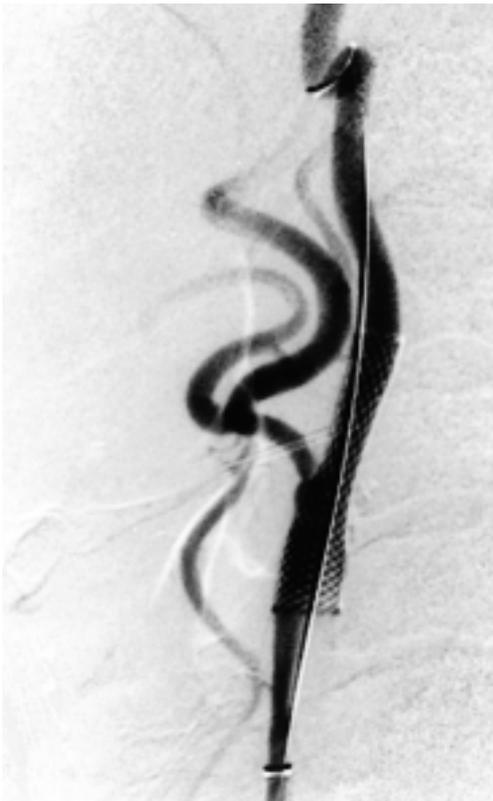


Figure 2b. Digital subtraction angiography. Lateral views of the left carotid artery bifurcation. No residual stenosis after carotid stenting.

and arterial pressure in most of the patients decrease after the carotid angioplasty. The reason could be iatrogenic stimulation of carotid baroreceptors, which seems to improve in the next few days. Thus, the arterial pressure could rise to hypertensive levels in the days after carotid stenting, thereby increasing regional CBF in the presence of impaired autoregulation and causing CHS.

CHS has also been widely reported in the surgical literature as an infrequent complication of carotid endarterectomy (CEA) with an incidence of approximately 0.6%.⁶ In our opinion and previous experiences, it may also occur after percutaneous transluminal carotid angioplasty and stenting with causal mechanism and clinical features similar to those of CEA.^{5,8} One cannot completely exclude the possibility of embolism and silent cerebral infarction with subsequent haemorrhagic transformation in response to hyperperfusion, but the CT scan appearances do not indicate to such a mechanism.

Conclusions

In conclusion, CHS may occur after carotid stenting. The combination of a high-degree carotid stenosis and unstable arterial pressure is probably an important cause in the pathogenesis of hyperperfusion syndrome. The arterial blood pressure monitoring seems to be important after carotid angioplasty of high-degree stenosis.

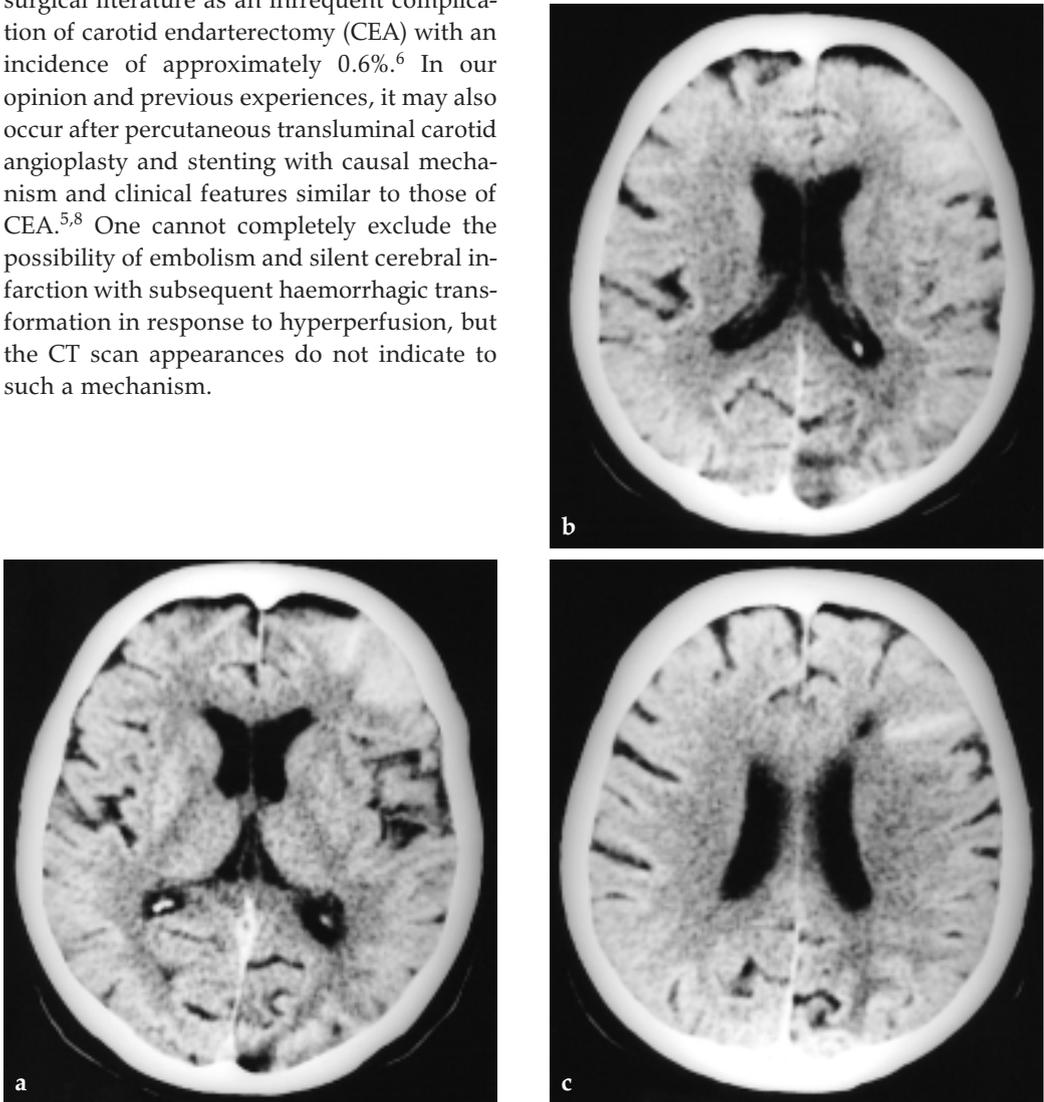


Figure 3a, 3b, 3c. CT of the brain demonstrates small haemorrhage in the left frontal region.

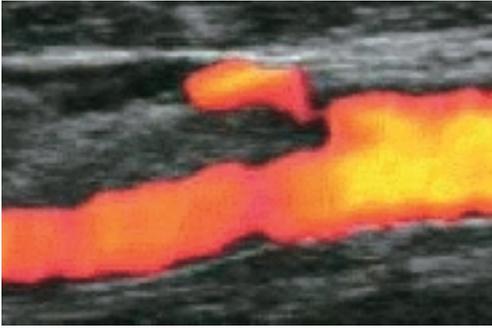


Figure 4. Colour Doppler ultrasound of the internal carotid artery shows a visibly patent vessel.

References

1. Keunen R, Nijmeijer HW, Tavy D, Stam K, Edelenbosch R, Muskens E, et al. An observational study of pre-operative transcranial Doppler examinations to predict cerebral hyperperfusion following carotid endarterectomies. *Neurol Res* 2001; **23**: 593-8.
2. Milosevic ZV, Zvan B, Zaletel M, Surlan M. Cerebral hyperperfusion syndrome after carotid stenting. [Online]. Mar 22, 2002. URL: <http://www.eurorad.org/case.cfm?UID=1563> Luxembourg, Euromedia.
3. Chuang YM, Wu HM. Early recognition of cerebral hyperperfusion syndrome after carotid stenting - a case report. *Kaohsiung J Med Sci* 2001; **17**: 489-94.
4. Morrish W, Grahovac S, Douen A, Cheung G, Hu W, Farb R, et al. Intracranial hemorrhage after stenting and angioplasty of extracranial carotid stenosis. *AJNR Am J Neuroradiol* 2000; **21**: 1911-6.
5. McCabe DJ, Brown MM, Clifton A. Fatal cerebral reperfusion hemorrhage after carotid stenting. *Stroke* 1999; **30**: 2483-6.
6. Hosoda K, Kawaguchi T, Shibata Y, Kamei M, Kidoguchi K, Koyama J, et al. Cerebral vasoreactivity and internal carotid artery flow help to identify patients at risk for hyperperfusion after carotid endarterectomy. *Stroke* 2001; **32**: 1567-73.
7. Nakase H, Sakaki T, Kempfski O. A scanning technique to measure regional cerebral blood flow and oxyhemoglobin level. *Neurosurgery* 2001; **48**: 1335-42.
8. Mansoor GA, White WB, Grunnet M, Ruby ST. Intracerebral hemorrhage after carotid endarterectomy associated with ipsilateral fibrinoid necrosis: a consequence of the hyperperfusion syndrome? *J Vasc Surg* 1996; **23**: 147-51.