FATAL SUBARACHNOID HEMORRHAGE AFTER CAROTID STENTING

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Background and Purpose: Cerebral hyperperfusion syndrome with intracerebral hemorrhage (ICH) following carotid angioplasty and stent placement (CAS) of the internal carotid artery (ICA) is well known. We report the occurrence of fatal subarachnoid hemorrhage in a patient undergoing CAS.

Case report: A 77-year-old woman experiencing a left-hemispheric transient ischemic attack underwent CAS for a 95% stenosis of the left ICA. CAS was performed without acute complications. At 5 hours the patient suddenly deteriorated. Her level of consciousness changed and she developed neck stiffness. CT of the brain revealed diffuse SAH with acute hydrocephalus.

Conclusions: Like ICH, SAH may develop as a severe complication after CAS. There are no reliable clinical symptoms preceding this fatal complication. However, several factors such as long-standing severe carotid stenosis with contralateral occlusion and increasing blood pressure after CAS accompanied by the extensive use of antithrombotic agents may predispose to this fatal complication.

Key words: carotid angioplasty, subarachnoid hemorrhage, reperfusion injury, antithrombotic therapy.

INTRODUCTION

Carotid angioplasty and stent placement (CAS) has been introduced as an alternative to carotid endarterectomy (CEA) for the treatment of carotid artery stenosis. Hemodynamic instability consisting of hypertension, hypotension, or bradycardia and cerebral hyperperfusion syndrome with intracerebral hemorrhage (ICH) after CEA or CAS are well-known complications, with an incidence between 0.3% and 2.7% [2, 6, 8, 9, 15]. We describe a patient who suffered fatal subarachnoid hemorrhage after CAS.

CASE REPORT

A 77-year-old woman was referred for internal carotid stenting after experiencing a left-hemispheric transient ischemic attack. She did not have a history of hypertension or other vascular risk factors. Extracranial Doppler revealed an occlusion of the right internal carotid artery (ICA) and severe stenosis at the origin of the left ICA. Transcranial Doppler ultrasound showed a slightly diminished blood flow velocity in the left cerebral arteries under very poor conditions for transcranial insonation. Diagnostic angiography confirmed the occlusion of the right ICA and showed a short, concentric, 95% stenosis (NASCET) of the left ICA (figure 1a). There were no other stenoses in this vascular territory. Furthermore, the left ICA showed irregular fusiform enlargement in the cavernous segment, probably due to atherosclerotic vascular disease (ASVD). There were no other vascular malformations such as AVM, AVF, or aneurysm in the anterior or posterior circulation. A brain computed tomography (CT) scan before CAS was normal, without evidence of acute or previous infarction.
Written informed consent for CAS and treatment during follow-up was obtained. The patient received continuous intravenous infusion of heparin (300 units/kg/24h) and was started on aspirin 300mg/d and clopidogrel 75mg/d 72 hours before the procedure. The procedure was performed under local anesthesia via a femoral arterial route. Once the diagnostic study was completed and the stenotic internal carotid artery was identified, the 5-F catheter was withdrawn and 7-F 90-cm guiding sheath (Shuttle; Cook, Inc.) was selectively placed into the left common carotid artery via a femoral arterial route. The patient was given 2000 IU of heparin IV to achieve a periprocedural activated clotting time (ACT) ≈ 250 seconds. The stenosis was crossed with a 0.014-inch guidewire (Trooper; Boston Scientific/Scimed Maple Grove, MN). Atropine (0.5 mg) was injected as a single bolus intravenously, and the stenosis was predilatated with a 2.5 × 20-mm balloon catheter (Hayate, Terumo, Leuven, Belgium), which was inflated at 6 atm. The balloon catheter was replaced by a 7 × 40 mm self-expanding stent (Carotid Wallstent; Boston Scientific). The stent was postdilated with a 4 × 20 mm balloon (Bypass Speedy; Boston Scientific) at 7 atm. The control angiogram of the left ICA showed a residual stenosis of less than 15% (figure 1b) and good intracranial perfusion without any evidence of complications (figure 3). The neurological examination performed immediately after the procedure was normal. The patient was transferred back to the stroke unit and a heparin infusion (25,000 U/24 h) maintained a PTT of less than 70 seconds. For the following 5 hours the patient did not suffer headache or demonstrate any seizure activity. Within the first three periprocedural hours her blood pressure increased, but never exceeded 180 mmHg (systolic). After 5 hours the patient’s condition suddenly deteriorated, her level of consciousness changed, and neck stiffness developed. A brain CT was performed immediately and revealed diffuse subarachnoid hemorrhage (SAH) with acute hydrocephalus (figure 2). She subsequently died 17 hours later.

At autopsy, the brain showed massive, diffuse SAH, particularly above the left cerebral hemisphere and in the basal cisterns. There was marked evidence of increased intracranial pressure, including flattened gyri and closed sulci. The circle of Willis was normal except for the ICA. The bilateral carotid siphons showed severe atherosclerotic lesions. However, an aneurysm, AVM, AVF, or any other cause for the SAH could not be demonstrated. On coronal sections fresh secondary hemorrhages were detected within the white matter of the left cerebral hemisphere beside the striatum and in the brainstem. The SAH had extended through the basal cisterns into the third and the left lateral ventricle. These autopsy findings were consistent with a hyperperfusion phenomenon after carotid stenting.

DISCUSSION

CAS is an alternative technique for treating extracranial carotid stenotic disease, so far on an experimental basis. Beside the three major complications embolism, hemodynamic instability and early stent thrombosis, there is also a risk of hyperperfusion syndrome which can also be complicated by an ICH, known as reperfusion hemorrhage [3, 6, 7, 13]. Although the use of CAS is increasing tremendously, to the best of our knowledge, there are only two reports of SAH after CAS in the literature [2, 13].

The cause of SAH is a ruptured aneurysm in 85% of cases, non-aneurysmal perimesencephalic hemorrhage in 10%, and a variety of rare conditions in 5%
In our patient neither diagnostic angiography nor autopsy revealed intradural aneurysm, vascular malformation, or intracranial dissection. It is unlikely that the fusiform aneurysm of the left ICA in the cavernous segment would cause a SAH because the location is extradural and, if it had ruptured, it would have produced a spontaneous carotid-cavernous fistula (CCF), probably accompanied by cranial nerve palsy. Furthermore, the pattern of distribution of the subarachnoid blood in our patient on the postictal CT resembles that of nonaneurysmal perimesencephalic SAH [12, 17]. The blood was predominantly located in the posterior fossa with downward extension anterior to the brainstem as far as the medulla. In addition, blood had leaked into the Sylvian and anterior interhemispheric fissures but no rupture into the ventricular system had developed. The cause of nonaneurysmal perimesencephalic SAH is unknown. Diagnosis can only be made on the basis of negative findings at digital subtraction angiography (DSA) or CT angiography [18]. On the other hand autopsy revealed a predominant distribution of the subarachnoid blood in the left-hemispheric basal cisterns in our patient. Perhaps impaired autoregulation as described for the hyperperfusion syndrome with ICH after CEA or CAS [3, 6, 7, 11, 15] is also responsible for SAH after CAS. Long-lasting hypoperfusion distal to a high-grade stenosis with maximal vasodilatation and impaired autoregulation might result in high-pressure autoregulatory failure. Thus, very high perfusion pressure after CEA or CAS overwhelms the vasoconstriction ability of the arterioles, disrupting the tight junctions of the capillary endothelial cells and causing ICH and also likely SAH. The assumption of high-pressure autoregulatory failure is supported by two further case reports with SAH after CAS [2, 13]. Schoser et al. [13] reported a patient with SAH after simultaneous angioplasty of multiple extracranial artery stenoses including both vertebral arteries, the right subclavian artery and a high grade stenosis of the left ICA. Al-Mubarak et al. [2] reported one case with SAH after CAS of 90% ICA stenosis with contralateral ICA occlusion. The intraparenchymal petechial hemorrhages found at necropsy in our patient are a further argument for a diagnosis of reperfusion hemorrhage. SAH can be caused by rupture of a berry aneurysm, dissecting aneurysm to the perforating vessels, or rupture of the vessel wall alone. However, necropsy cannot definitely exclude a berry aneurysm. Some non aneurysmal SAH are thought to result from microaneurysms obliterated by the hemorrhage or undergoing spontaneous thrombosis after rupture [1, 4, 5]. Furthermore we can postulate that the combination of high-grade stenosis and occlusion of the contralateral ICA, as in our patient, may weaken the vessel walls and enhance the possibility of
an aneurysm to form due to the augmented hemodynamic stress associated with increased flow in the collateral vessels [14]. Therefore, increased flow and perhaps elevated blood pressure after CAS may cause vessel or aneurysm rupture. In contrast to hyperperfusion syndrome after CEA, where symptoms usually develop within a few days, symptoms following CAS occur within a few hours. The extensive periprocedural use of antiplatelet agents and heparin may additionally enhance the risk of reperfusion hemorrhage with SAH. Therefore we now discontinue i.v. heparin immediately after successful CAS.

CONCLUSION

ICH after CAS has been increasingly reported, but there are only two reports of SAH after CAS in the literature. The frequency of the condition has not yet been determined and there is no single answer as to the cause of this potentially fatal complication. As in our patient, no reliable clinical symptoms precede this fatal complication; however, several factors may predispose to this fatal course. These are long-standing severe carotid stenosis with contralateral occlusion and increasing blood pressure after CEA accompanied by extensive heparinization and the use of antiplatelet agents. Therefore, meticulous periprocedural monitoring of clinical symptoms, aggressive procedural and periprocedural blood pressure monitoring, prompt treatment of hypertension, and close control of the periprocedural coagulation status are mandatory. Complications of carotid stenting will never be entirely avoidable, but with these precautions we will be able to reduce fatal complications.

RÉFÉRENCES


