Case reports

Carotid artery stenting in the presence of contralateral carotid occlusion: mind the hyperperfusion syndrome!

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Stroke is the third cause of death in western countries and its complications lead to significant socio-economic problems related to the prolonged hospitalization and rehabilitation of patients with neurological lesions. Severe atherosclerotic lesions of the carotid artery are the main cause of stroke and transient ischemic attacks. Their incidence may reach 5-7% per year in patients with carotid artery stenosis > 70% with or without symptoms.

Time-honored carotid endarterectomy is still regarded as the gold standard therapy for primary and secondary prevention of stroke. However, surgery is not free of complications and the rate of perioperative stroke ranges from 5.1 to 14.3%.

A group of patients at a particularly high risk of stroke during surgical endarterectomy is represented by patients with significant carotid stenosis in the presence of an occluded contralateral artery. Indeed, carotid cross-clamping during operative surgery in the absence of an adequate collateral flow may result in a critical flow reduction during the operation and therefore increases the risk of periprocedural strokes. In the North American Symptomatic Carotid Endarterectomy (NASCET) trial, the overall risk of stroke was 5.1%, whereas it increased up to 14.3% in patients with an occluded contralateral carotid artery.

Recently, carotid stenting has been increasingly used as an endovascular technique for carotid revascularization, especially after the introduction of neuroprotection devices which improved the safety of the procedure. Therefore, it may be an attractive alternative to carotid endarterectomy, especially when the surgical risk is too high.

We describe the immediate and late outcomes of 3 patients treated with carotid artery stenting in the presence of contralateral carotid occlusion.

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Introduction

Stroke is the third cause of death in western countries and its complications lead to significant socio-economic problems related to the prolonged hospitalization and rehabilitation of patients with neurological lesions. Severe atherosclerotic lesions of the carotid artery are the main cause of stroke and transient ischemic attacks. Their incidence may reach 5-7% per year in patients with carotid artery stenosis > 70% with or without symptoms[1].

Time-honored carotid endarterectomy (CEA) is still regarded as the gold standard therapy for primary and secondary prevention of stroke[2]. However, surgery is not free of complications and the rate of perioperative stroke ranges from 5.1 to 14.3%[1,3].

A group of patients at a particularly high risk of stroke during surgical endarterectomy is represented by patients with significant carotid stenosis in the presence of an occluded contralateral artery. Indeed, carotid cross-clamping during surgery in the absence of an adequate collateral flow may result in a critical flow reduction during the operation and therefore increases the risk of periprocedural strokes[4-6]. In the North American Symptomatic Carotid Endarterectomy (NASCET) trial[2,3], the overall risk of stroke was 5.1%, whereas it increased up to 14.3% in patients with an occluded contralateral carotid artery.

Recently, carotid artery stenting (CAS) has been increasingly used as an endovascular technique for carotid revascularize-

tion, especially after the introduction of neuroprotection devices which improved the safety of the procedure[7-12]. Therefore, it may be an attractive alternative to CEA, especially when the surgical risk is too high[3,13,14].

We describe the immediate and late outcomes of 3 patients treated with CAS in the presence of contralateral carotid occlusion.

Description of cases

Case 1. The first patient was a 73-year-old male, hypertensive, current smoker and with a family history of cerebrovascular disease. His cardiac history included an anterior myocardial infarction (MI) in 1986, a second anteroseptal MI in 1998, and subsequent angioplasty to the right coronary artery in 1999. At the time of presentation at our center he was only symptomatic for dyspnea on exertion (NYHA functional class II-III). The echocardiogram showed an ejection fraction of 25%. He was admitted to our clinical center for the recent onset of dizziness. He did not have history of previous transient ischemic attack or stroke. A carotid artery ultrasound revealed occlusion of the right carotid bulb and the presence of a fibrocalcific plaque causing a 70% stenosis at the origin of the left internal carotid artery.

Therefore, he underwent: 1) a coronary angiogram (which showed occlusion of the left anterior descending coronary artery and a 70% lesion of the right coronary artery, distal to the previously implanted stent); 2) a carotid angiogram which confirmed the presence of a 90% lesion at the origin of the left internal carotid artery and the occlusion of the contralateral artery. The right subclavian artery was very tortuous and a severe lesion was found at the origin of the right vertebral artery, whereas the left subclavian and vertebral arteries were free of obstructive disease.

Our strategy was to perform a carotid angioplasty to the left internal carotid artery and, possibly, a coronary angioplasty to the right coronary artery. We decided not to treat the right vertebral artery percutaneously due to the excessive tortuosity of the anonymous truncus.

After placement of an 8F sheath, an 8F Hochey Stick guiding catheter (Boston Scientific Medi-Tech, Boston, MA, USA) was placed into the left common carotid artery and mounted on a rigid 0.035" Amplatz guidewire (Boston Scientific Medi-Tech) placed previously in the left external carotid artery to maintain support. Heparin was administered intravenously as a 5000 U bolus after sheath insertion; additional heparin was given as needed to maintain an activated clotting time of 200 s. The patient's neurologic status was continuously monitored both during and after CAS by simple contralateral hand-gripping maneuvers. An angiogram of the lesion was taken to obtain the best road map for the procedure. The lesion was crossed with an extra-support floppy guidewire. A protection-filter Angioguard 6.0 (Cordis Corporation, Miami, FL, USA) was placed to avoid distal embolism. The lesion was predilated with a 2.0/20 mm balloon (Hayate Terumo, Tokyo, Japan) at 6 atm. A 8/40 mm self-expandable stent (Smart, Cordis, Johnson&Johnson, Miami, FL, USA) was deployed and post-dilated with a 5.5/20 mm balloon (Viatrac 14 Plus, Guidant, Temecula, CA, USA) at 14 atm. The filter was removed using a dedicated retrieval system. A final angiogram taken in two orthogonal views showed no residual stenosis and a good distal flow. There were no neurological complications during the procedure.

Following carotid angioplasty, a direct stenting of the lesion of the right coronary artery was performed, using an 8/3.0 mm stent (Smart, Cordis, Johnson&Johnson), expanded to 14 atm. There was no residual stenosis and a good distal flow was obtained.

The introducer sheaths were removed after the procedure when the activated clotting time was < 180 s and no further anticoagulation was administered.

The patient made an uneventful recovery and was discharged 2 days after the procedure on aspirin (325 mg/day) and ticlopidine (250 mg twice daily).

At the 6-month clinical follow-up, the patient reported no new neurological events or episodes of dizziness. Carotid artery ultrasound showed a well-expanded stent, with no signs of in-stent restenosis.

Case 2. The second patient was an 81-year-old hypertensive male, with a previous history of coronary artery bypass grafting in 1998. Following the finding of a murmur at the left side of the neck, he underwent an echo-color Doppler evaluation of the epiaortic vessels. A subocclusive stenosis of the left internal carotid artery with an occlusion of the contralateral carotid artery was found. Therefore, he was admitted to our hospital to undergo carotid angiography in view of a possible angioplasty.

Carotid angiography confirmed the echo findings: total occlusion of the right internal carotid artery and subocclusive stenosis of the origin of the left internal carotid artery.

Our strategy was to perform a carotid angioplasty on the left internal carotid artery. The lesion was crossed with a floppy guidewire. A cerebral protection device Neuroshield 6.0 (Mednova, Horsham, West Sussex, UK) was placed proximally and distally to the origin of the carotid canal. The lesion was predilated with a 20/2.0 mm balloon (Maverick Boston Scientific Scimed, Boston, MA, USA). A self-expanding 9.0/40 mm stent (Smart, Cordis, Johnson&Johnson) was deployed and post-dilated using a 7.0/30 balloon (Viatrac 14 Plus, Guidant). The patient's neurological status was continuously monitored both during and after coronary artery stenting by simple contralateral hand-gripping maneuvers. A post-procedure angiography in two projections showed no residual stenosis and a good distal flow.

The patient made an uneventful recovery and was discharged 2 days after the procedure on aspirin (325 mg/day) and ticlopidine (250 mg twice daily).

At the 6-month clinical follow-up, the patient reported no new neurological events or other symptoms. Carotid artery ultrasound showed a well-expanded stent, with no signs of in-stent restenosis.

Case 3. The third patient was a 63-year-old woman. She had many risk factors for vascular diseases: diabetes, hypertension, hypercholesterolemia, obesity, and a family history of cerebrovascular disease.

She was symptomatic for angina and effort dyspnea at low workloads and complained of recent onset of dizziness and headache. A carotid artery ultrasound performed at the time of admission revealed severe atherosclerotic disease of both the internal carotid arteries, with a 90% stenosis of the right internal carotid artery and occlusion of the contralateral artery.

The coronary angiogram revealed a two-vessel disease of the left anterior descending coronary artery and of the left circumflex artery, and the carotid angiogram confirmed the echo findings.

Our strategy was to initially treat the right carotid artery percutaneously, in view of a coronary artery bypass operation to be performed 1 month later.

The lesion of the right internal carotid artery was crossed with an extra-support floppy guidewire. A protection-filter Angioguard (Cordis Corporation) was placed to avoid distal embolism. The lesion was predilated with a 20/2.5 mm balloon (Hayate Terumo) at 10 atm. An 8.0/40 mm self-expanding stent (Precise stent, Cordis, Johnson&Johnson) was deployed and post-dilated with a 6.0/30 mm balloon (Viatrac 14 Plus, Guidant) at 7 atm. The filter was removed using a dedicated retrieval system. The final angiogram showed no residual stenosis and a good distal flow.

There were no neurological events during the procedure and neurological examination repeated at 24 hours did not show any deficit. Antiplatelet medications included clopidogrel (75 mg daily) and aspirin (325 mg daily).

Two days after the procedure the patient developed headache associated with systolic hypertension. Therefore, she was placed on oral nimodipine, intravenous nitrates and beta-blockers to better control the blood pressure. A computed tomography (CT) scan performed immediately did not show any signs of possible intracranial bleeding. Adequate control of her blood pressure was achieved by means of oral beta-blockers, calcium antagonists and ACE-inhibitors and a new CT scan performed 1 week later did not show any significant difference. Unfortunately, the night before discharge she complained of severe migraine and in a few minutes became unconscious. A CT scan revealed cerebral hemorrhage and the patient died 3 days later.

Discussion

With medical therapy alone, patients with bilateral carotid artery disease in which one artery is totally occluded have a poor prognosis[4,15]: asymptomatic patients, when followed for 6 years on medical therapy, have been reported to have a 38% incidence of stroke[16]; symptomatic patients have a 69% risk of ipsilateral stroke at 2 years[3].

Surgical revascularization of the carotid arteries is an accepted modality of treatment of atherosclerotic obstructive disease[2,17,18]. Although endarterectomy in patients with contralateral occlusion reduces the long-term risk of ipsilateral stroke, the procedure in this group of patients may imply a higher incidence of perioperative neurologic complications compared with those without contralateral occlusion[3,6]. Even though many authors[4,19-21] suggest that in their experience CEA in patients with contralateral occlusion is not associated with an increased perioperative morbidity/mortality rate, the reduced collateral circulation during carotid clamping, cerebral hemorrhage secondary to the hyperperfusion syndrome, and the overall advanced status of the vascular disease[22], always constitute a threat during the surgical approach.

CAS is being increasingly used for the treatment of carotid artery stenosis[7-12]. An attractive application of CAS may be the treatment of patients at a potential surgical risk for neurological complications, such as patients with contralateral occlusion[1,4,15].

We report 3 cases of CAS in the presence of contralateral carotid occlusion. In none of the patients periprocedural neurological complications were observed.

Our results are in line with those of many studies which showed that carotid angioplasty with stent placement may be performed with an excellent technical success and an acceptable complication rate, even in high-risk patients[23,24].

Mathur et al.[15], in their series of 26 patients treated with CAS in the presence of contralateral carotid occlusion, had a 96% procedural success rate of carotid stenting with only 1 (3.8%) minor stroke in a patient who had air embolism during baseline angiography. No neurologic events were observed within a mean follow-up of 16 ± 9.5 months after the procedure.

All our patients had concomitant coronary artery disease, which was stable at the time of admission. One of them also had a ischemic dilated cardiomyopathy with a very low ejection fraction. Shawl et al.[24] had a series of very high-risk patients that included patients with unstable angina, previous ipsilateral CEA, contralateral carotid artery occlusion and other severe comorbid illnesses. Their procedural success rate was 99%, including 73 patients who had a coronary intervention. The total 30-day stroke rate was 2.9% for treated patients and only one major stroke had occurred. There were no myocardial infarctions or deaths during

or within 30 days of CAS. At a mean follow-up of 19 ± 11 months no other major strokes or neurologic deaths occurred and 3 patients (2%) had asymptomatic restenosis.

Finally, Dangas et al.[23] performed CAS in 39 carotid arteries of 37 consecutive patients who met the criteria for high-risk surgical anatomy: previous ipsilateral CEA (20/39, 51.3%), a common carotid bifurcation above the mandibular angle (5/39, 12.8%), contralateral carotid artery occlusion (15/39, 38.5%), or previous radiation therapy to the neck (1/39, 2.6%). The procedural success rate was 100%, with no major in-hospital complications. Neurological events were rare. Only 1 (2.6%) transient ischemic attack occurred prior to discharge; at 30 days, 1 (2.6%) additional minor stroke had been observed, reaching a 2.6% cumulative 30-day "death plus any stroke" rate. Over an 11 ± 6 month follow-up, 2 (5.4%) patients died of non-neurological causes, but there were no strokes.

In our experience of carotid artery angioplasty performed in 3 patients with an occluded contralateral carotid artery, we report one death at 15 days after the procedure and due to cerebral hemorrhage.

Intracerebral hemorrhage is an uncommon complication of CEA[25], and carries a high rate of mortality and morbidity. Traditionally, attention has been focused on the cerebral hyperperfusion syndrome as the leading cause of intracerebral hemorrhage after CEA. Other mechanisms, such as a perioperative cerebral ischemic event, cerebral infarction, and the use of postoperative anticoagulation therapy, may also be relevant. Hyperperfusion is defined as hypertension with symptoms of either severe headache, seizures, or confusion, or a doubling of the intraoperative cerebral blood flow values[25]. According to some authors[26], the cerebral hyperperfusion syndrome occurs in 0.5 to 1% of patients undergoing CEA. Other authors[27] report an incidence of 0.3 to 1.2% after CEA. The incidence of cerebral hyperperfusion after endovascular revascularization procedures of the craniocervical arteries remains unknown. Meyers et al.[27], in their series of 140 patients treated with percutaneous transluminal angioplasty/stenting between 1996 and 2000, report an incidence of 5% of patients who developed clinical or radiological manifestations of cerebral hyperperfusion. They also report that 2 patients with cerebral hemorrhage recovered during a protracted period after the procedure (range 3 weeks to 6 months).

Patients undergoing endovascular stenting procedures should be closely monitored for evidence of hyperperfusion, with careful monitoring of the blood pressure, heart rate, and anticoagulation. Further research is needed to confirm that cerebral hyperperfusion is the pathogenesis of this condition. Moreover, sentinel headache could solely indicate the early sign of hyperperfusion injury after carotid stenting, especially in the presence of arterial hypertension[28]. Patients with sentinel headache after angioplasty should be identified early and deserve intensive study for other features of cerebral hyperperfusion injury and prompt early management.

In conclusion, our cases show that CAS may be an attractive alternative to CEA, especially in patients at high surgical risk, such as patients with an occluded contralateral carotid artery. However, we are aware of the small number of patients enrolled and treated, and many doubts remain about the real short- and long-term outcomes.

Moreover, patients undergoing endovascular stenting procedures should be closely monitored for evidence of the hyperperfusion syndrome, whose prevalence may be higher in the high-risk cohort commonly referred for endovascular treatment[27].

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