HYPERPERFUSION SYNDROME COMPlicATING EXTRACRANIAL CAROTID ARTERY STENTING

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INTRODUCTION

Severe extracranial carotid artery disease is treated by arterial revascularization. Surgical endarterectomy or endovascular stenting can be complicated by hyperperfusion syndrome (HPS) in 0.3 to 5% of cases.¹² The complication occurs hours to days after a successful procedure despite immediate excellent angiographic results. The presentation is variable and includes atypical migrainous phenomena, confusion, focal deficit, seizure, intracerebral hemorrhage. Cerebral CT reveals focal edema or hemorrhage, which, when present, alters the prognosis. Mortality rates above 50% have been reported.¹

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CASEx REPORT

We report the case of a 56 years old male patient who was referred to our department with severe stenosis of the left carotid artery bifurcation. The patient was a treated hypertensive with statin-controlled hypercholesterolemia and had a history of multiple endovascular interventions. He was diagnosed with severe coronary artery disease and underwent a coronary endovascular procedure (stenting of the left anterior descending artery) in 2002.

Two years later the patient was asymptomatic however follow-up examination detected a right lateral cervical bruit. Carotid ultrasound evaluation was performed. Peak systolic velocities measured over both carotid arteries suggested severe 70-99% ostial stenosis of the right internal carotid and a 50-70% stenosis of the left internal carotid. The patient had no history of neurological symptoms or cerebral vascular acute event. Carotid angiography confirmed the presence and severity of bilateral obstructive extracranial carotid disease. Based on lesion severity the decision was made to treat only the right carotid artery.
After signing the informed consent the patient underwent endovascular angioplasty and stenting of the right common carotid bifurcation with a good procedural result and uneventful post-procedural evolution. Forty-eight hours after stenting the patient was doing well and was discharged. The daily drug regimen recommended at discharge included 75 mg of aspirin, 75 mg of clopidogrel, 8 mg of perindopril, 12.5 mg of hydrochlorothiazide, 10 mg of felodipin and 40 mg of simvastatin. Follow-up visits were scheduled every three months and there was very good compliance to antiplatelet, blood pressure lowering and lipid lowering therapy.

One year after right carotid stenting doppler examination diagnosed significant progression of the left carotid lesion. The patient underwent a new carotid angiography procedure that revealed very good patency of the right carotid stent, (Fig. 1) with no restenosis, but severe stenosis of the proximal segment of the left internal carotid artery. (Fig. 2)

The patient was taken to the Catheterization Lab. An 8F sheath was inserted in the right femoral artery and an intravenous heparin bolus of 5,000 units was administered. An 8F Judkins Right guiding catheter was positioned into the ostia of the right common carotid artery. The lesion was crossed with a 6 mm cerebral protection device (CPD) that was deployed in the right internal carotid artery distal to the lesion. To prevent bradycardia 1 mg of i.v. atropine was administered and the lesion was then predilated using a 3 mm diameter coronary balloon catheter. We stented the target arterial segment with a 8 mm diameter, 40 mm long self-expandable carotid stent and postdilated the stent with a 5.5 mm diameter balloon catheter. There was a 15% residual stenosis that was considered acceptable and the CPD was retrieved and inspected for debris. (Fig. 3) Procedural time (filter deployment to filter removal time) was 12 minutes. We performed the final angiogram that revealed patency of the extracranial carotid artery and of its intracerebral branches and no evidence of spasm, dissection or thrombus.

Severe hypotension occurred following stent postdilatation. Repeated boluses of ephedrine and intravenous dopamine were used to stabilize systolic...
blood pressure at 120-140mmHg. The patient was then transferred from the Cath Lab to the Coronary Care Unit.

![Figure 3. Final result of left CAS; there is a 15% residual stenosis with no angiographic evidence of dissection or thrombus.](image)

Postprocedural examination performed in the CCU was unremarkable. Femoral arterial introducer was retrieved and hemostasis was achieved with a compressive bandage. The ECG and blood pressure were monitored. Except for the procedural heparin dose no additional dose was administered.

Four hours later patient status was altered by severe headaches and vomiting. He rapidly developed confusion, right hemiparesis and motor aphasia. The ECG was normal, the blood pressure was 120/70mmHg. The symptoms were suggestive of a HPS complicating the revascularization procedure. The patient was immediately transferred to the Neurology Department of The County Hospital.

Initial neurological examination documented pure motor right hemiparesis of medium intensity, pyramidal signs, motor aphasia, and confusional status. CT imaging revealed a 1 cm diameter cortico-subcortical left frontal hematoma. The ECG was normal and the blood pressure was stable.

Intraparenchymal hemorrhage secondary to anticoagulant therapy was excluded. The blood pressure was normal. The onset signs suggested intracranial hypertension but this could not have been caused by the small hemorrhage detected on cerebral CT image. The location of the small hyperdense image was unusual for a primary intracerebral hemorrhage as was the rapid improvement that followed.

Clinical status showed significant improvement over the next hours with progressive remission of neurological dysfunction. Next morning there was almost complete recovery with only slight right hemiparesis persisting. The patient was transferred back to the Cardiology Department. Because of the patient’s favorable evolution and of the very high risk of stent thrombosis associated with antplatelet drugs discontinuation we decided to continue the combined aspirine-clopidogrel therapy. After another 48 hours there was complete resolution of symptoms and the patient was discharged.

One month later cerebral CT imaging showed complete resolution of the left frontal hematoma. Follow-up was performed every three month. At twelve month the patient was readmitted for a complete evaluation. He was asymptomatic, there was good control of his blood pressure and his plasma cholesterol and triglycerides were within therapeutic range. Echocardiography findings were unremarkable and the stress test showed no signs of myocardial ischemia. Neurological status was normal. Carotid angiography documented very good patency of both carotid stents with no signs of restenosis.

**DISCUSSION**

CAS is increasingly used for the treatment of carotid disease. It is less invasive than CEA, it offers excellent results in terms of acute and late patency of the treated artery and it was proved to be at least as safe and effective as surgery in stroke prevention. Like CEA, CAS is associated with rare but significant complications.

One potentially lethal complication is the HPS, first described by Sundt in 1981 after CEA and more recently observed during CAS. It occurs hours to days after the procedure. It was initially believed to be caused by the failure of normal cerebral autoregulation secondary to long-standing changes in perfusion pressure but now it seems more likely that microembolic showers are the real cause of a true HPS.

Risk factors for HPS include critical carotid artery stenosis, critical or occlusive contralateral carotid disease, decreased cerebral vascular reserve, periprocedural uncontrolled hypertension,
unprotected CAS, aggressive use of anticoagulants, use of IIb-IIIa inhibitors.\textsuperscript{6,7} Mortality rate is up to 80\% in cases associated with ICH.\textsuperscript{8}

In our case the HPS occurred in atypical conditions. No identifiable risk factor for this complication was present. There was adequate periprocedural control of blood pressure. The contralateral carotid lesion was treated one year prior to the actual procedure and the stent was patent providing very good blood flow to the brain. There was no aggressive use of antithrombotics. Seventy-five mg of aspirine and 75 mg of clopidogrel were administered daily. Procedural heparin was restricted to an i.v. bolus of 5,000 units and there was no additional dose of aspirin administered after the procedure. IIb-IIIa inhibitors were not an issue. There is no sound evidence of benefit with their use during carotid interventions and we do not use them.\textsuperscript{9}

We always perform carotid stenting using cerebral protection and this case was no exception. The short procedural time makes cerebral embolization of device related thrombus unlikely. Internal carotid artery showed no evidence of spasm at the place of CPD deployment and there was no local intraarterial administration of vasodilators. Moreover, the patient had a previous uncomplicated contralateral carotid revascularization procedure, suggesting good cerebral vascular reserve.

There was no accident or complication during angioplasty of the left carotid artery, the final angiographic result was very good and the patient was asymptomatic. Four hours later he developed severe deterioration and based on the neurological findings and cerebral CT images an HPS with associated ICH was diagnosed. Despite the use of a filter-type CPD it is possible that small embolic particles embolized through the 100μ pores of the filter membrane. Post CAS diffusion-weighted MR imaging demonstrated that CPD are not fully protective, embolization do occur and produce new small lesions.\textsuperscript{10} These microembolic events are asymptomatic in most of the cases. On rare occasions they can initiate a HPS, and it is likely that this happened in our patient.

Although ICH associated to HPS carries a high mortality risk, the patient had a fast and complete recovery over the next 72 hours. After discharge the evolution was very good. Within the next year there was no neurological event and both stented segments were patent.

**CONCLUSION**

Despite adequate case selection, optimal preprocedural patient preparation and good interventional technique CAS is not a risk-free intervention. HPS can complicate CAS. It is an unpredictable event and even good control of risk factors cannot always prevent it. Careful monitoring of the patient in the first 24 hour after the procedure is paramount for the rapid diagnosis of this condition. Although the mortality rates are high for patients with associated HPS and ICH, full recovery is possible and is associated with good long-term outcome.

**REFERENCES**