Subarachnoid hemorrhage after carotid artery stenting

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We describe subarachnoid hemorrhage (SAH) in a 66-year-old man, who underwent technically successful carotid stenting for a string-stenosis of the right internal carotid artery (ICA) in a presence of contralateral ICA occlusion with recurrent right hemisphere transient ischemic attacks. At 2 hours, the patient developed headache and vomiting, but no focal neurological deficits. Performed transcranial color-coded Doppler (TCCD) showed over 2.8-fold increase of the peak systolic velocity in the right middle cerebral artery. The emergent CT of the brain showed SAH with the right hemisphere edema. Patient was treated with Nimodipine in continuous infusion, diuretics i.v. and additional hypotensive therapy depending on blood pressure values. Clopidogrel was stopped for 5 days. Over next 4 weeks, a gradual cerebral velocities decrease was observed on TCCD, which was related to clinical and CT resolution. (J Vasc Surg 2007;45: 1072-5.)

Reperfusion injury is a rare but life threatening complication following carotid endarterectomy (CEA) and carotid artery stenting (CAS).[1-3] It may manifest as hyperperfusion syndrome with brain edema, intracranial hemorrhage (ICH), or subarachnoid hemorrhage (SAH).[1-4] The latter one is described very rarely. Occurrence of ICH carries a risk of death estimated at 37% to 80%.[4]

CASE REPORT

We report the case of a 66-year-old man with left internal carotid artery (ICA) occlusion and right ICA string-stenosis on duplex ultrasound and multislice computed tomography angiography (Fig 1), referred to our department to determine further management. The right ICA stenosis was highly symptomatic with recurrent transient ischemic attacks (TIA), including amaurosis fugax and left arm weakness which occurred three times during the week prior to admission. CT scans showed no detectable brain lesions (Fig 2, *A2*).

The patient was a former smoker with drug-resistant hypertension, exertion angina, diabetes, and hyperlipidemia. Laboratory tests showed elevated serum creatinine level (1.54 mg/dl). Significant bilateral renal artery stenosis was detected by duplex ultrasound. The patient had been taking aspirin (75 mg o.d.) for 2 years and clopidogrel (75 mg o.d) for 5 days prior to admission.

Angiography confirmed 95% lumen reduction in the right ICA (string-stenosis) with plaque ulceration (Fig 3, *A*), significant cerebral flow impairment (Fig 3, *B*), and bilateral renal artery stenosis. In addition, the right coronary artery stenosis was detected. Further patient management was discussed with an endovascular surgeon and an independent neurologist. The decision of a two-stage endovascular treatment was made. The highly symptomatic ICA stenosis was to be treated first (CAS), with the coronary and renal angioplasty 2 to 3 weeks later.

Despite contralateral ICA occlusion, a proximal neuroprotection by flow reversal (Gore NPS, Flagstaff, Ariz) was chosen due to normal vertebral arteries and the functional collateral flow from the posterior cerebral arteries to the middle cerebral arteries (MCA) through the both posterior communicating arteries.[5] The procedure was uneventful and the angiographic result was optimal (Fig 3, *D*) with an excellent blood flow to the right middle and anterior cerebral arteries as well as to the left ones through the anterior communicating artery (Fig 3, *C*).

Arterial blood pressure prior to CAS was 150/90 to 170/100 mm Hg, but there were episodes of rapid elevations up to 200/110 mm Hg. During CAS, blood pressure measured with arterial catheter ranged 150/70 to 170/90 mm Hg. Just after CAS, blood pressure monitored and measured by an automatic cuff, ranged 130/70 to 140/80 mm Hg.

Two hours after CAS, the patient began to complain of headache, followed by nausea and vomiting with no focal neurological deficits. There was blood pressure elevation to 180/95 mm Hg. Transcranial color-coded Doppler (TCCD) demonstrated 2.8-fold increase in the peak systolic velocity (PSV) in MCA compared with the values prior to CAS (Fig 2, *A1, B1*). Emergent brain CT showed SAH with bilateral hemisphere edema which was significantly more prominent on the right (Fig 2, *B2*). Careful retrospective evaluation of cerebral angiography showed no vascular malformation or cerebral aneurysm.

The patient was administrated Nimodipine in continuous intravenous infusion (initial dose of 2 mg/hour), Mannitol 125 mg and Furosemide 10 to 20 mg every 8 hours (i.v.), and hypotensive drugs (beta-blockers, clonidine, spironolactone, ACE inhibitors) to keep blood pressure below 160/100 mm Hg. Clopidogrel, but not aspirin, was stopped for 5 days to possibly reduce the risk of recurrent cerebral bleeding. Blood pressure, ECG, and serum electrolytes concentration were controlled. The headache began to decrease 1 week after CAS and a full recovery occurred

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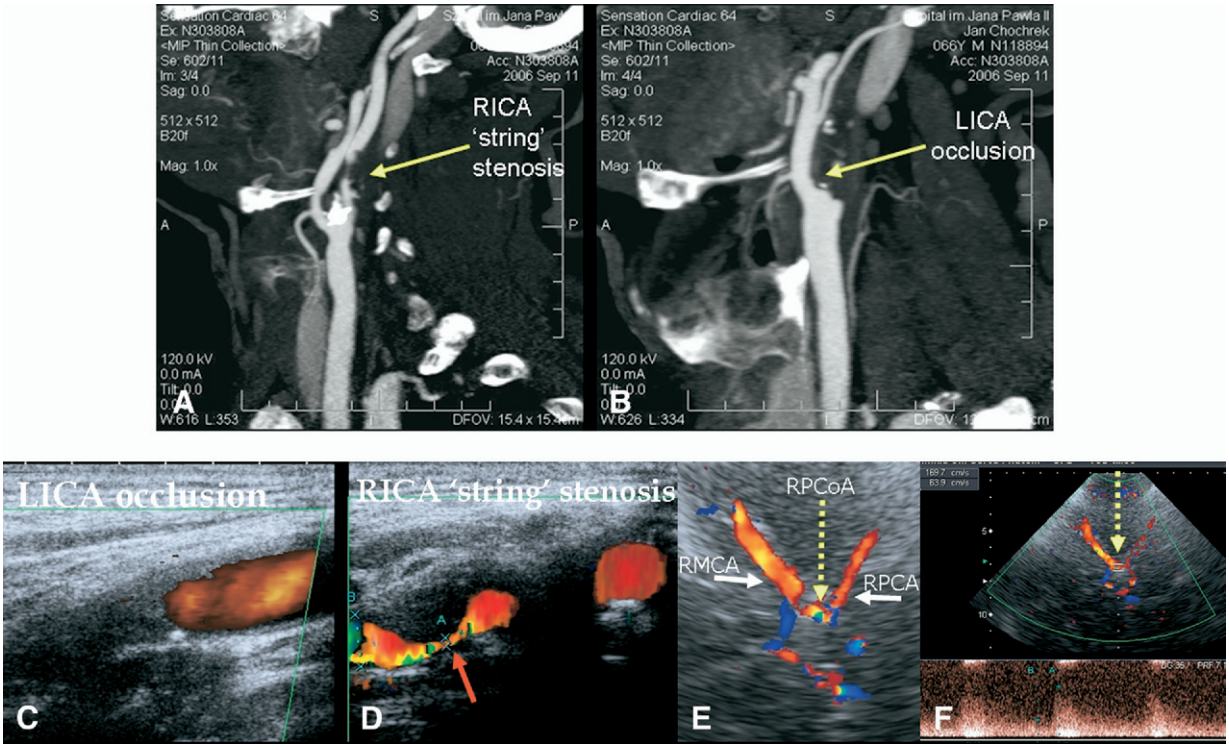

Fig 1. Multislice computed tomography angiography. **A**, the right ICA string-stenosis; **B**, occlusion of the left ICA. Doppler ultrasound. **C**, occlusion of the left ICA; **D**, the right ICA string-stenosis. Transcranial color-coded Doppler of the Willis circle. **E**, yellow arrow shows patent posterior communicating artery (PCoA) situated between the right middle cerebral artery (RMCA) and the right posterior cerebral artery (RPCA); **F**, spectrum registered in the PCoA indicates blood flow from the RPCA to the RMCA.

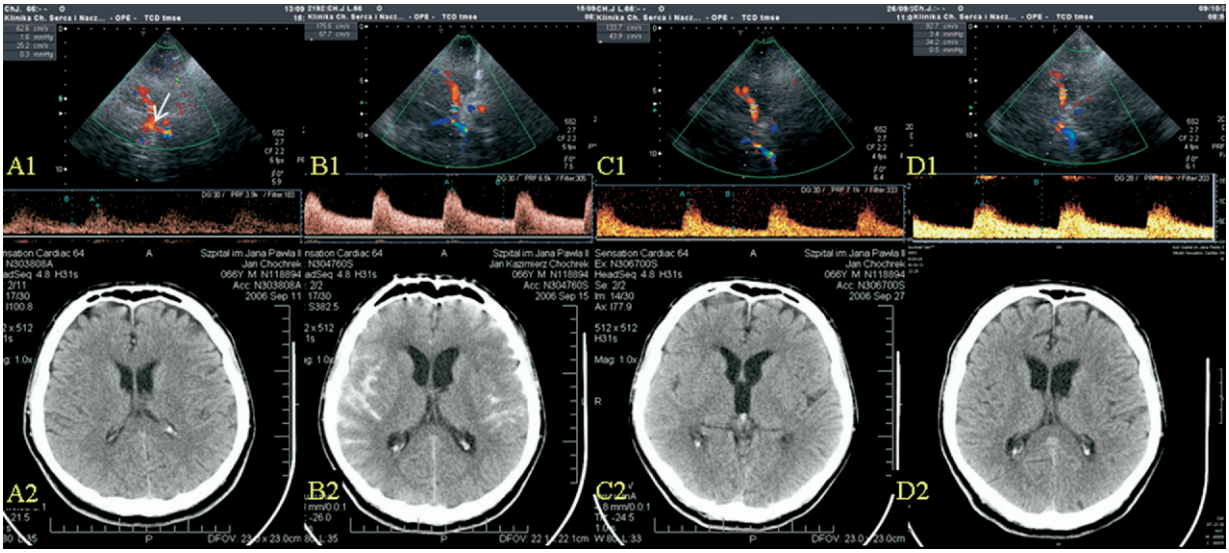

Fig 2. Images in the upper raw present TCCD findings: **A1**, prior to CAS, decreased PSV (62 cm/s) in the right MCA, posterior communicating artery (arrow); **B1**, after CAS, 2.8-fold PSV increase in the right MCA (PSV-175 cm/s); **C1**, 2 weeks after CAS, slow decrease of MCA PSV (133 cm/s); **D1**, 4 weeks after CAS, normalization of the right MCA PSV (92 cm/s) Images in the lower raw present CT findings: **A2**, prior to CAS, normal CT scan; **B2**, after CAS, subarachnoid hemorrhage with bilateral hemisphere edema; **C2**, 2 weeks after CAS, the right hemisphere edema, previously seen blood is absorbed; **D2**, 4 weeks after CAS, complete lesions' resolution.

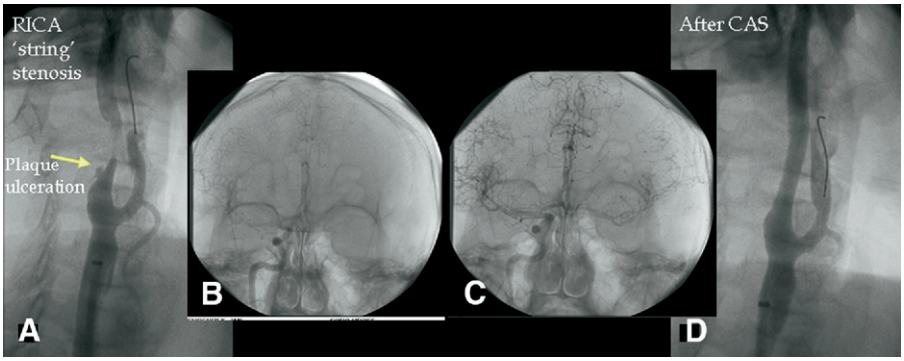

Fig 3. Angiography of the right ICA and cerebral arteries. **A, B,** prior to CAS, soft ulcerated plaque in right ICA causing string-stenosis; impaired cerebral flow. **C, D,** just after CAS, a good angiographic result after CAS and flow to the right and left middle and anterior cerebral arteries.

after 2 weeks. No neurological deficits occurred during the recovery.

Repeated duplex ultrasound showed patent stent without thrombus formation. Every day, follow-up TCCD showed a slow gradual PSV decrease in the right MCA from the day 8, with velocity normalization 4 weeks later (Fig 2, *C1, D1*). Control brain CT showed blood absorption on the day 13 and complete edema resolution on the day 29 (Fig 2, *C2, D2*).

The patient was discharged from the hospital after 5 weeks following CAS. One month later, he underwent successful stenting of coronary and both renal arteries. He remains asymtomatic and his blood pressure is below 140/80 mm Hg at the 4-month follow-up.

DISCUSSION

The presented patient required multisite interventions for multilevel symptomatic atherosclerosis. Recurrent cerebral TIAs prompted the decision to perform CAS first to prevent a disabling stroke. CEA was turned down due to the high risk of periprocedural complications related to symptomatic angina and contralateral ICA occlusion.[6,7] With the unfavorable right ICA plaque morphology, the significant risk of cerebral embolization during CAS was in part reduced by the choice of a proximal neuroprotection system that works already prior to lesion crossing.[8]

Despite the uneventful CAS procedure, the patient developed headache in the absence of any focal neurological deficit. The TCCD finding of the 2.8-fold PSV increase in the right MCA was highly suggestive of reperfusion injury and was followed by an immediate CT. The over twofold MCA PSV increase was described particularly often in ICH.[1,9,10] Surprisingly, CT scans showed SAH. The controversy remains about mechanism of SAH after CAS. This disorder occurs usually in the setting of coexisting malformation or cerebral aneurysm.[4] Those were not found on angio-CT and angiography in this case; however, the microscopic A-V malformations cannot be excluded.

Our TCCD findings of 2.8-fold MCA PSV increase may suggest similar mechanism of both ICH and SAH related to autoregulation loss. Prolonged ischemia due to bilateral ICA disease is responsible for maximal dilation of small arterioles to ensure adequate blood flow. However, when the normal perfusion pressure is restored with CEA or CAS, the arterioles are unable to reconstrict adequately in order to protect the brain tissue, leading to the reperfusion injury.[11] In those circumstances, TCCD can be very helpful in selection of patients at risk for reperfusion injury. We believe that in patients with marked postprocedural MCA PSV increase, a tight blood pressure control should be introduced. In our institution, TCCD examination is performed routinely prior to CAS. Postprocedural TCCD evaluation is performed in patients considered "high risk", and always in the setting of headache or neurological complications.

Reperfusion injury occurs in 0.4% to 4% of patients after CEA and in 0.67% to 5% of patients undergoing CAS,[1-4] and it occurs sooner after CAS (within hours or 1 to 2 days) than CEA (2 to 17 days). [2] In our database of 425 consecutive CAS procedures, there were four (0.9%) hemorrhages (three ICH, one SAH). Uncontrollable hypertension in the setting of bilateral renal artery stenosis in addition to contralateral ICA occlusion made the described patient very prone to reperfusion injury.[1-3,12] Also, routine dual antiplatelet therapy in patients undergoing CAS may contribute to its incidence.

SAH is the most rarely described type of reperfusion injury.[13,14] This devastating condition carries a 20% to 30% risk of rebleeding during first month, cerebral artery vasospasm (usually between 5 to 14 days), which may progress to cerebral infarction and neurological deficits.[15] Nimodipine is recommended to reduce poor outcome related to vasospasm.[15] Bed rest and antihypertensive therapy is frequently used to prevent rebleeding.[15] However, the issue of dual antiplatelet therapy remains unresolved. Two described cases of SAH following CAS were fatal.[13,14] Thus, we decided to stop clopidogrel (not aspirin) for 5 days.

In summary, TCTD can be useful in the diagnosis and monitoring of brain reperfusion injury after CAS.

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