ELSEVIER

Perfusion Computed Tomography–Guided Subacute Endovascular Reperfusion in a Patient with Carotid Occlusion

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Most patients with symptomatic internal carotid artery occlusion have a single minor or major hemispheric stroke. A minority of patients have ipsilateral retinal ischemia, recurrent strokes, or transient ischemic attacks. Whereas spontaneous carotid recanalization is rare, acute surgical recanalization has been attempted, with mixed results. Recently, acute endovascular recanalization has been performed and described as feasible and relatively safe. We describe a patient with symptom recurrence related to hemodynamic factors after occlusion of the carotid artery who was succesfully treated 14 days after symptom onset.

Most patients with symptomatic internal carotid artery occlusion have a single minor or major hemispheric stroke. A minority of patients have ipsilateral retinal ischemia, recurrent strokes, or transient ischemic attacks (TIAs). Whereas spontaneous carotid recanalization is rare, acute surgical recanalization has been attempted, with mixed results.[1,2] Recently, acute endovascular recanalization has been performed and described as feasible and relatively safe.[3,4]

We describe a patient with symptom recurrence related to hemodynamic factors after occlusion of the carotid artery who was succesfully treated 14 days after symptom onset.

CASE REPORT

A 60-year-old male hypertensive smoker was admitted because of an acute left faciobrachiocrural hemisyndrome and hemianopia (National Institutes of Health Stroke Scale [NIHSS] = 14) upon awakening from general anesthesia after a hip replacement. Rapid improvement was observed (NIHSS = 2), and he became asymptomatic 36 hr after symptom onset. Low-weight molecular heparin was initiated (nadroparin calcium, 0.6 cc subcutaneous daily). The patient reported a similar hemisyndrome 1 month earlier, lasting about 4 hr. Noncontrast brain computed tomography (CT) at admission showed a small right subcortical hypodensity. Transcranial Doppler showed inversion of the flow in the right ophthalmic artery. Flow in the right middle (R-MCA) and anterior cerebral arteries (R-ACA) was decreased by more than 30% compared to the left side (R-MCA 50 to 35 cm/sec, R-ACA 25 to 15 cm/sec, L-MCA 100 to 45 cm/sec, L-ACA 75 to 25 cm/sec). The right posterior cerebral artery was accelerated, and the right posterior communicating artery was detected. Right cerebrovascular hemodynamic reserve capacity was abolished in response to the apnea test.[5] Carotid duplex showed right internal carotid occlusion. Antiplatelet treatment with aspirin (325 mg daily) was initiated.

The patient remained asymptomatic while lying down but started to have blurred vision in the right eye each time he sat up, lasting a few minutes. The patient also reported recurrent left leg and arm jerking, independent of body position and of visual symptoms. His blood pressure was well controlled (over 140-170 systolic/70-90 diastolic) without antihypertensive treatment. Full-dose anticoagulation with low-molecular weight heparin (nadroparin calcium, 0.6 cc subcutaneous/12 hr) was given for 1 week, but symptoms persisted. CT Angiography (CTA) done on day 13 showed internal carotid occlusion, with a ''ring sign'' with peripheral hyperdensity of the carotid wall and hypodense central thrombus (Fig. 1A, B), suggesting a subacute occlusion.[6] On the

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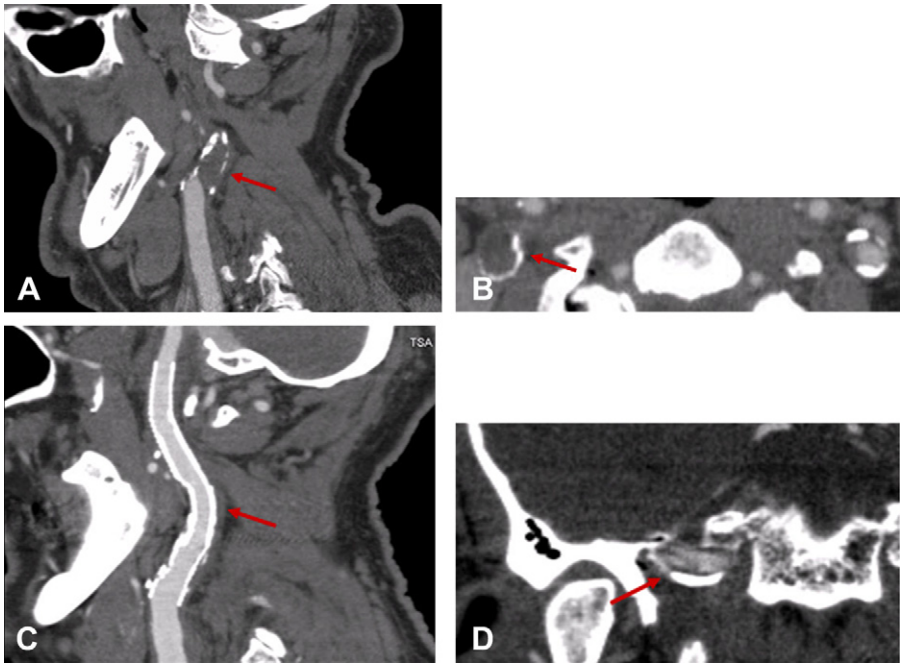

Fig. 1. CTA. **A** Lateral projections: right carotid artery occlusion (*arrow*) at the carotid bifurcation before the intervention. **B** Axial projection: ''ring sign'' (*arrow*) in right carotid artery. **C** Lateral projection: complete recanalization (*arrows*) of the right carotid artery after the intervention with stenting. **D** Coronal projections: intrapetrosal right carotid artery dissection (*arrow*).

same day, perfusion CT showed an increased mean transit time, increased cerebral blood volume, and decreased cerebral blood flow in the right hemisphere, consistent with an extensive penumbral area (Fig. 2A).[7,8] Conventional angiography was done to confirm carotid occlusion. It showed a 30% left internal carotid artery stenosis, whereas both external carotid arteries and the vertebrobasilar system were normal. However, the anterior communicating artery was not present and the P1 segment of the right posterior cerebral artery was hypoplastic. After obtaining informed consent from the patient, the right internal carotid was reopened by traversing the occlusion with a guidewire and introducing three stents into the occlusion. A distal protection device was used, and no distal thrombus was seen after contrast passed through the site of occlusion, although an intrapetrous, probably iatrogenous, carotid dissection was observed. A bolus of intravenous heparin (100 UI/kg) was administered during the procedure, and the patient was maintained on a regimen of aspirin (325 mg daily) and clopidogrel (75 mg daily) after the procedure. One hour after the procedure, transcranial Doppler showed an increased velocity in the R-MCA and R-ACA (R-MCA 150 from 70 cm/sec, R-ACA 110 from 40 cm/sec), with normal flow in the right posterior cerebral artery and disappearance of flow in the right posterior communicating artery. The ophthalmic artery was not detected. Blood pressure was kept below 160/90.

Clinically, the patient became asymptomatic in all positions, and control brain CT 1 week after the procedure showed a small right frontal hemorrhage. Perfusion CT was normal, except for a small perfusion deficit around the hemorrhage (Fig. 2B). Transcranial Doppler and cerebrovascular hemodynamic reserve were normal. At 2 weeks, the patient remained asymptomatic and brain CT showed hematoma reabsortion. CTA demonstrated a patent carotid artery with a persistent nonstenotic intrapetrous carotid dissection (Fig. 1C, D).

DISCUSSION

Once an atheromatous carotid artery has become occluded, hemispheric TIAs or strokes, retinal ischemia, or no symptoms occur. Recurrent hemodynamic symptoms may occur and prevent mobilization.[9] Typical manifestations of hemodynamic TIAs in the carotid territory are amaurosis fugax and limb shaking when standing up.[10]

Noninvasive imaging by transcranial Doppler allows accurate assessment of flow and collateral circulation. The observed inversion of the flow in the ophthalmic artery and significant flow from the right posterior cerebral artery through the posterior communicating artery suggest a collateral supply to the right MCA, still insufficient in our patient.

Perfusion imaging techniques may allow the identification of ischemic tissue at risk. Among them, perfusion CT can be performed quickly in