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 successfully 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. 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. On the
same day, perfusion CT showed an increased mean transit
time, increased cerebral blood volume, and decreased ce-
rebral 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 vertebral-
silar system were normal. However, the anterior commu-
nicating artery was not present and the P1 segment of the
right posterior cerebral artery was hypoplastic. After ob-
taining informed consent from the patient, the right inter-
cal carotid was reopened by traversing the occlusion with
a guidewire and introducing three stents into the occlu-
sion. A distal protection device was used, and no distal
thrombus was seen after contrast passed through the site
of occlusion, although an intrapetrous, probably iatroge-
nous, carotid dissection was observed. A bolus of intrave-
nous 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) af-
after the procedure. One hour after the procedure, transcra-
nial 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 pos-
terior communicating artery. The ophthalmic artery was
not detected. Blood pressure was kept below 160/90.

Clinically, the patient became asymptomatic in all po-
sitions, 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 cere-
brovascular hemodynamic reserve were normal. At 2
weeks, the patient remained asymptomatic and brain
CT showed hematoma reabsorption. 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 hemo-
dynamic symptoms may occur and prevent mobili-
ization.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 cir-
culation. 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
the emergency setting because of its availability, being well tolerated and not time-consuming. Besides, conventional CT and CTA can be realized as part of the study.\(^8\) Although perfusion CT has not yet been studied systematically to identify ischemic tissue at risk in the subacute or chronic state of carotid occlusions, the perfusion CT results 13 days after the presumed carotid artery occlusion showed a persistent area of critical but reversible hypoperfusion in our symptomatic patient, as usually seen in the acute state of ischemia.\(^7\) This hypoperfused area also included the right posterior cerebral artery territory, suggesting that deviation of its blood into the MCA territory contributed to the ischemia. Similar information about the state of perfusion might be obtained with perfusion-diffusion magnetic resonance imaging, even though this method has not been validated in more chronic hypoperfusion either.

The importance of showing viability of threatened tissue before internal carotid artery recanalization has been stressed recently, and the feasibility and safety of endovascular recanalization have been demonstrated in several series.\(^3,4\) In order to mobilize the thrombus during the intervention, the demonstration that the carotid occlusion is recent is also useful. CTA may be able to do so and was used in our patient before attempting to recanalize. Based on the good correlation between symptoms, noninvasive arterial imaging, and perfusion imaging, late recanalization treatment was considered in this patient.

Intra-arterial recanalization in the acute or subacute stage may lead to intracerebral hemorrhage, dissection, and hyperperfusion.\(^3,4\) Our patient experienced both an asymptomatic hemorrhage and an asymptomatic, nonstenosing dissection. The optimal antithrombotic strategy after such intervention remains open.

**CONCLUSIONS**

Internal carotid artery occlusions may remain symptomatic after the occlusion. Using noninvasive imaging by Doppler/duplex, CTA, and perfusion CT may allow us, in unstable patients like ours, to plan and perform late endovascular recanalization, with complete resolution of symptoms and no significant complications.

**REFERENCES**


