ELSEVIER

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

M.G. Delgado,[1] A. Gil,[2] V. Mateos,[1] S. Calleja,[1] P. Vega,[2] C.H. Lahoz,[1] and P. Michel,[3] Oviedo, Spain, and Lausanne, Switzerland

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

[1]*Neurology Service, Hospital Central de Asturias, Oviedo, Spain.*

[2]*Radiology Service, Hospital Central de Asturias, Oviedo, Spain.*

[3]*Neurology Service, Centre Hospitalier Universitaire Vaudois, Lausanne, Switzerland.*

Correspondence to: M. Gonzalez Delgado, MD, Neurology Service, Hospital Central de, Asturias, C/Julián Clavería, 3006, Oviedo, Spain, E-mail: mglezdelgado@yahoo.es

Ann Vasc Surg 2008; 22: 465-468
DOI: 10.1016/j.avsg.2007.12.010

Published online: May 8, 2008

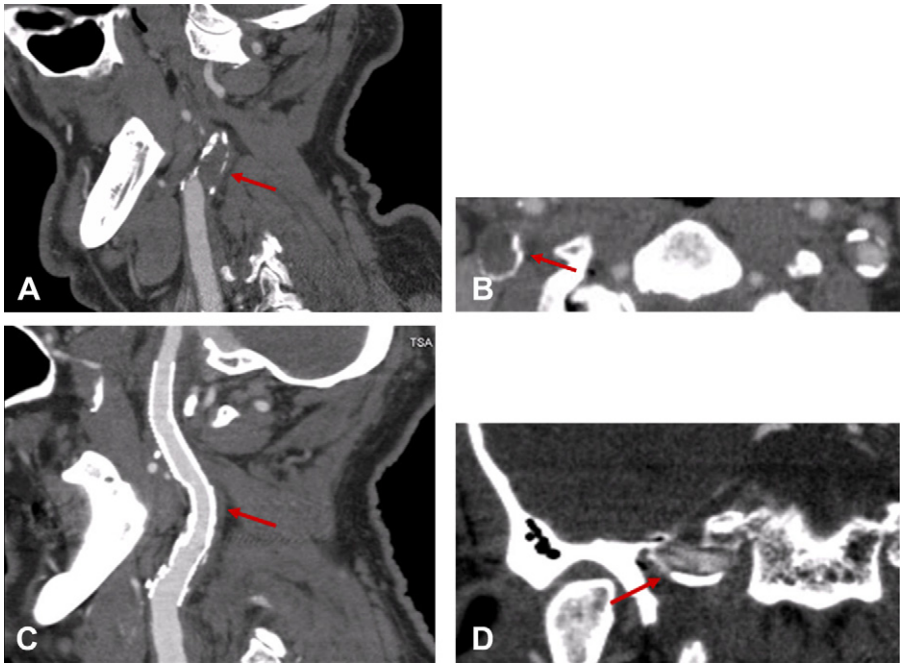

Fig. 1. CTA. **A** Lateral projections: right carotid artery occlusion (*arrow*) at the carotid bifurcation before the intervention. **B** Axial projection: ''ring sign'' (*arrow*) in right carotid artery. **C** Lateral projection: complete recanalization (*arrows*) of the right carotid artery after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

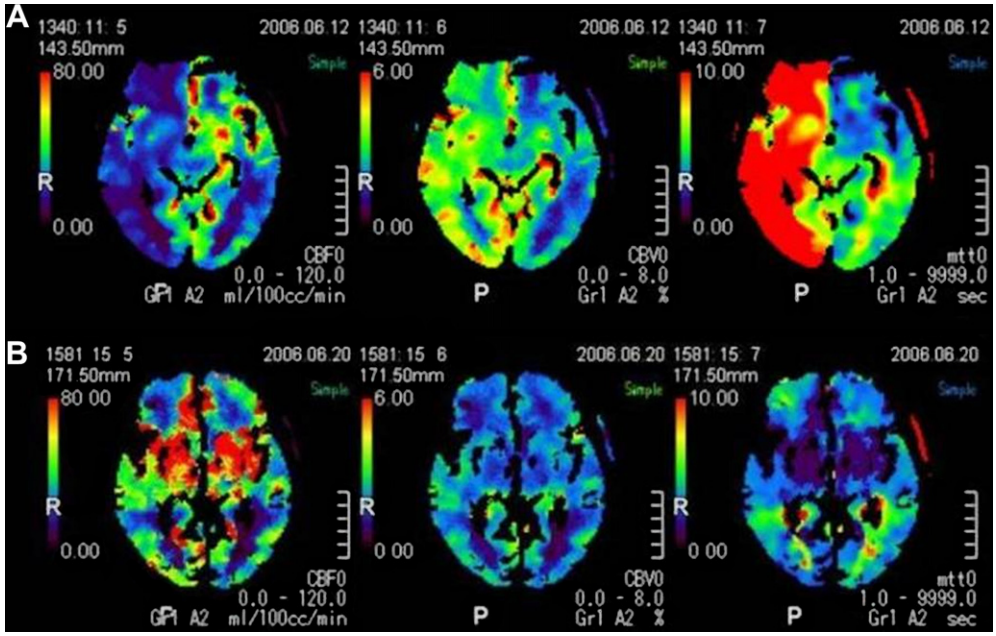

Fig. 2. A Perfusion CT showing increased mean transit time, increased cerebral blood volume, and decreased cerebral blood flow in the right hemisphere, indicating a large penumbral area. **B** Perfusion CT at 1 week after recanalization, showing normalization of perfusion except for a small right frontal region corresponding to a small hemorrhage on noncontrast CT.

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

1. Kasper GC, Wladis AR, Lohr JM, et al. Carotid thromboendarterectomy for recent total occlusion of the internal carotid artery. J Vasc Surg 2001;33:242-249.
2. Paty PSK, Adeniyi JA, Mehta M, et al. Surgical treatment of internal carotid artery occlusion. J Vasc Surg 2003;37:785-788.
3. Sugg RM, Malkoff MD, Noser EA, et al. Endovascular recanalisation of internal carotid artery occlusion in acute ischemic stroke. Am J Neuroradiol 2005;26:2591-2594.

4. Nedeltchev K, Brekenfeld C, Remonda L, et al. Internal carotid artery stent implantation in 25 patients with acute stroke: preliminary results. Radiology 2005;237:1029-1037.
5. Markus HS, Harrison MJG. Estimation of cerebrovascular reactivity using transcranial Doppler, including the use of breath-holding as the vasodilatory stimulus. Stroke 1992;23:668-673.
6. Michel P, Binaghi S, González-Delgado M, et al. The ''carotid ring sign'' on angio-CT may differentiate acute from chronic carotid occlusion. (Abstract). International Stroke Conference, New Orleans, LA. Stroke 2005;36:482.
7. Wintermark M, Reichhart M, Cuisenaire O, et al. Comparison of admission perfusion computed tomography and qualitative diffusion- and perfusion-weighted magnetic resonance imaging in acute stroke patients. Stroke 2002;33:2025-2031.
8. Wintermark M, Bogousslavsky J. Imaging of acute ischemic brain injury: the return of computed tomography. Curr Opin Neurol 2003;16:59-63.
9. Faught WE, van Bemmelen PS, Mattos MA, et al. Presentation and natural history of internal carotid artery occlusion. J Vasc Surg 1993;18:512-524.
10. Baquis GD, Pessin MS, Scott RM. Limb shaking—a carotid TIA. Stroke 1985;16:444-448.