

# Spontaneous Early Recanalization of an Acute Symptomatic Critical Stenosis of the Extracranial Internal Carotid Artery: A Case Report

Fu-Chi Yang,<sup>1</sup> Jiann-Chyun Lin,<sup>1</sup> Cheng-Yu Chen,<sup>2</sup> and Giia-Sheun Peng,<sup>1</sup> Taipei, Taiwan

We present a 63-year-old female with critical stenosis of the extracranial portion of the internal carotid artery (ICA), diagnosed using color-coded Duplex ultrasonography (CCDU) and magnetic resonance imaging. Nine days later, the patient showed profound clinical improvement, at which time spontaneous recanalization of the previously highly stenosed ICA was seen in follow-up CCDU and magnetic resonance angiography (MRA) and confirmed by three-dimensional computed tomography angiography (CTA). The detection of recanalization is important in predicting the patient's prognosis and deciding a suboptimal secondary prophylaxis strategy of medical or surgical treatment. Therefore, we suggest performing imaging studies immediately in patients, especially potential surgical or stenting candidates showing great changes in neurological function, through examination with CCDU and confirmation with MRA or CTA, all safe and minimally invasive methods, to see whether recanalization has occurred.

Carotid arterial stenosis is a major risk factor for ischemic stroke. Its incidence has been increasing as lifestyles have become Westernized.<sup>1</sup> For patients with symptomatic strokes and high-grade carotid stenosis, several large randomized trials have provided a strong evidence base for advising and selecting patients to undergo carotid surgery rather than medical treatment.<sup>2,3</sup> Spontaneous recanalization of the internal carotid artery (ICA) has previously been reported, but most such reports are anecdotal and/or describe recanalization of the intracranial portion of this artery.<sup>4,5</sup> Recanalization of the extracranial ICA is thought to be rare, especially at early time periods and when stenosis is caused by a thromboembolus.<sup>6,7</sup>

We report a case in which spontaneous recanalization of a critically stenosed ICA was observed on follow-up imaging with color-coded Duplex ultrasonography (CCDU), magnetic resonance angiography (MRA), and three-dimensional computed tomography angiography (3D-CTA) 9 days after admission. This finding influenced the subsequent clinical management of the patient.

## CASE REPORT

A 63-year-old female with a history of hypertension, type 2 diabetes, and hypercholesterolemia arrived at our emergency department in the morning after suffering sudden weakness in her right-sided limbs. On admission, her blood pressure was 156/77 mm Hg and her heart rate 65 beats per minute, with no arrhythmia. The patient was alert and presented with right central facial palsy and right-sided hemiparesis, but these symptoms were not accompanied by sensory disturbance, motor aphasia, or cerebellar ataxia. She had Medical Research Council grade 3/5 muscle power in the right upper extremity, grade 4/5 in the right lower extremity, and grade 5/5 in the left extremities. Deep tendon reflexes were brisk in the right extremities. The plantar response was extensor on the right. Blood biochemistry tests revealed no abnormalities, except hyperglycemia (blood sugar 124 mg/dL).

<sup>1</sup>Department of Neurology, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan, ROC.

<sup>2</sup>Department of Radiology, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan, ROC.

Correspondence to: Giia-Sheun Peng, MD, PhD, Department of Neurology, Tri-Service General Hospital, National Defense Medical Center, No. 325, Section 2, Cheng-Kung Road, Neihu 114, Taipei, Taiwan, ROC. E-mail addresses: fuji-yang@yahoo.com.tw; penggs@ndmctsh.edu.tw

*Ann Vasc Surg* 2008; 22: 469-472

DOI: 10.1016/j.avsg.2007.12.009

© *Annals of Vascular Surgery Inc.*

Published online: April 4, 2008

On hospitalization day 1, hydration and an antiplatelet agent were prescribed, but a profound downhill course in the muscle power of the right-sided limbs (grade 0/5 in the right upper extremity, grade 1/5 in the right lower extremity) and severe dysarthria developed. The CCDU showed high-grade stenosis of the left ICA with obvious thrombus formation, a conspicuous decrease in peak velocity (25 cm/sec), and a dampened diastolic trace (Fig. 1A) but normal forward flow in the ophthalmic artery. Magnetic resonance scans of the brain disclosed lacunar infarctions involving the left genu of the internal capsule and the anterior corona radiata (Fig. 2A, B). Time-of-flight MRA showed occlusion of the extracranial left ICA (Fig. 1B) and, on the identical side in the middle cerebral artery (MCA), a gradual tapering and significant decrease in flow, as well as a number of distal branches. Prominent left posterior cerebral artery (PCA) branches serving as collaterals to the MCA were seen on the brain MRA (Fig. 1C).

Additional treatment with subcutaneous low-molecular weight heparin, fraxiparine (GlaxoSmithKline, Evreux, France) 3,800 IU twice a day, was started and continued for 3 days. Thrombolytic therapy was given up because the worst symptoms had occurred more than 6 hr after the onset of stroke. Two-dimensional and color-coded Duplex transthoracic echocardiography did not disclose thrombus formation, valvular disease, or intracardiac shunt pathways. Later transesophageal echocardiography (TEE) was also negative for patent foramen ovale (PFO) and aortic atheroma. Routine blood parameters and values for prothrombin and partial thromboplastin times; albumin, fibrinogen, factor VIII, and antithrombin III concentrations; protein C; protein S; platelet count; platelet aggregation; and bleeding time were normal.

In the following days, the patient showed dramatic clinical improvement in her previous hemiplegia and dysarthria, and her National Institutes of Health Stroke Survey scale decreased from 18 to 3. Nine days after admission, the follow-up CCDU showed that the previous thrombus was now absent and that the blood flow velocity and waveform of the left extracranial ICA had returned to normal (Fig. 1D). The spontaneous recanalization was confirmed by MRA (Fig. 1E) and 3D-CTA (Fig. 1G), both of which showed that the left extracranial ICA was patent and did not have the typical appearance of dissection or the existence of atherosclerotic plaque. The intracranial vessels, including the bilateral ICAs, as well as the anterior, middle, and posterior cerebral arteries, were well visualized, without stenosis or cut-off (Fig. 1F, H).

## DISCUSSION

Early spontaneous recanalization of the extracranial portion of the ICA has rarely been reported. Most studies are of the intracranial ICA, and the follow-up imaging to detect recanalization was performed only weeks to months after the original occlusive event.<sup>4,5,8-13</sup> One recent case history reported

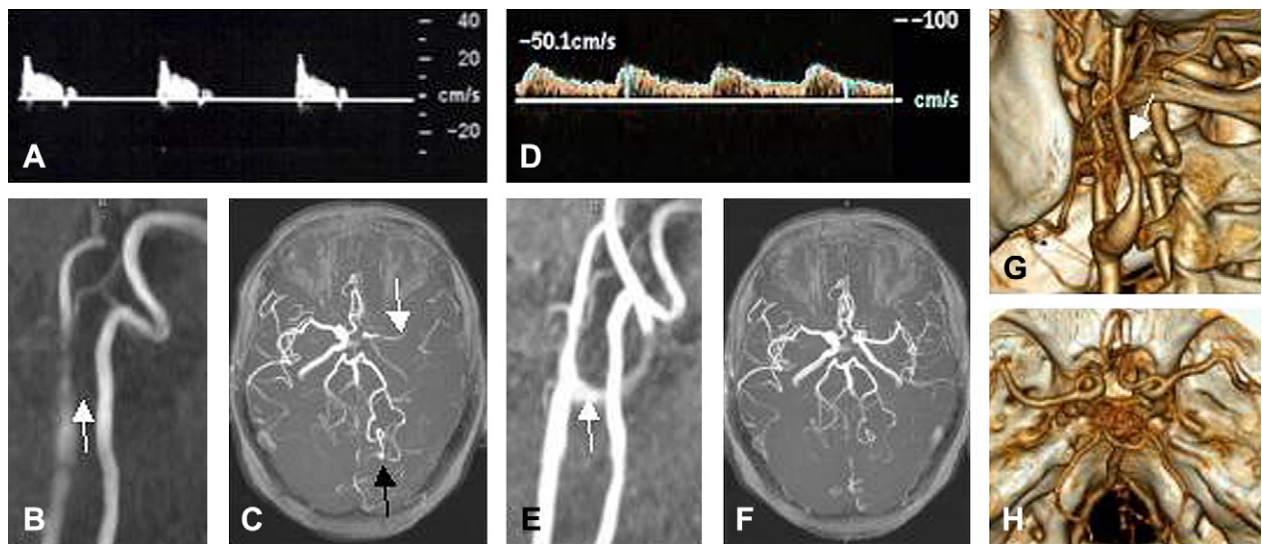
spontaneous recanalization that permitted “stenotic flow” 8 days after occlusion and residual plaque and clot in this section of the artery when it was later excised.<sup>12</sup> Our results showed normal flow in the artery on day 9 and no evidence of atherosclerosis or clot.

We currently use a widely accepted noninvasive protocol—CCDU combined with MRA—as a screening test for stroke patients to determine the severity of carotid stenosis.<sup>14-17</sup> Our patient was diagnosed as having a high-grade stenosis of the left ICA, based on CCDU findings, and total occlusion, based on MRA findings. This discrepancy is not unexpected, for MRA overestimates stenosis in tightly stenosed arteries and cannot reliably distinguish tight stenosis from occlusion.<sup>16,18</sup> In addition, the observation at the time of the duplex study of diffuse heterogeneous low echogenic thrombus as well as a peak systolic velocity of 25 cm/sec and a dampened diastolic flow in the cervical ICA, along with anterograde flow in the ipsilateral ophthalmic artery, indicated that there might exist a high-grade stenosis at the distal ICA with retrograde thrombus formation.

Our investigations to discover the reason for the high-grade stenosis were all negative. The patient had not suffered recent neck trauma and had no characteristic features of carotid dissection, such as tapering of the ICA lumen distal to the bulb, mural hematoma, irregular membrane crossing the vessel lumen, or demonstration of a true lumen with flow and a false one without flow, in the CCDU and MRA studies.<sup>19</sup> Blood biochemistry and clotting studies showed no abnormality in hemostasis. Cardiovascular screening tests showed no abnormalities, such as atrial fibrillation, myocardial infarction, valvular lesions, cardiomyopathy, PFO, or aortic atheroma, likely to cause cardioembolism.

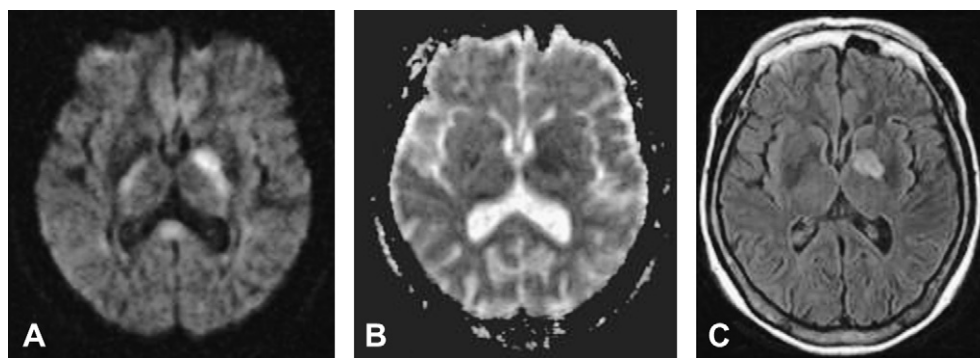
The patient had three risk factors—hypertension, hyperlipidemia, and type 2 diabetes—that might account for the formation of a vascular atherothromboembolus. Therefore, we initially thought that the ischemic event might be caused by the release of a fragment from an atherosclerotic plaque. However, no typical atherosclerotic changes were detected in the affected lumen, and both intracranial and extracranial ICAs were well delineated, without stenosis or cut-off in the rechecked CCDU, MRA, and 3D-CTA (Fig. 1D-H). We were therefore left with the speculation that the high-grade stenosis might have been caused by a cryptogenic thromboembolus.

Because the patient's condition initially worsened, we considered cerebral contrast angiography, to determine more precisely the severity of the critical stenosis, and follow-up, if necessary, with surgery such as stent implantation or endarterectomy.



**Fig. 1.** Day 1: **A** CCDU of the left ICA shows the conspicuously decreased peak blood flow velocity (25cm/sec) and dampened diastolic tracing typical of high-grade stenosis at distal ICA. **B** MRA of the neck shows absence of flow in the left ICA (arrow). **C** Brain MRA shows gradual tapering of the left MCA, a significant decrease in flow, and a number of distal branches (white arrow) compared to the right MCA. Prominent left PCA branches (black

arrow) are also seen. Day 9: **D** CCDU now shows a normal waveform in the left extracranial ICA. **E** MRA of the neck shows a return of blood flow (arrow). **F** Brain MRA shows a completely patent left ICA. The previously prominent left PCA branches have returned to normal. **G** Cervical (arrow) and **H** cerebral CTA show spontaneous recanalization of the left ICA.



**Fig. 2.** Day 1: **A** Axial diffusion-weighted image shows a hyperintense lesion in the left genu of the internal capsule and anterior corona radiata. **B** Apparent diffusion coefficient map shows a hypointense region in a site

corresponding to the site of the lesion in **A**. Day 9: **C** Axial fluid attenuation inversion recovery (FLAIR) image clearly demonstrates an increased signal intensity only in the same area as that seen in **A**.

Fortunately, the patient soon underwent spontaneous and complete recanalization of the ICA and did not need further surgical treatment.

The reasons that spontaneous recanalization of a nearly occluded vessel sometimes occurs are still unknown. Previous studies, using evidence from angiography<sup>20,21</sup> and TEE,<sup>22</sup> have reported that anticoagulation treatment alone could facilitate the lysis of intraluminal thrombi in ischemic stroke patients with high-grade ICA stenosis. This evidence indicates that prompt institution of anticoagulation

therapy may be of benefit to ischemic stroke patients under specific circumstances. Heparin exerts its anticoagulant activity through inhibition of the formation of thrombin, thus preventing new thrombus formation and extension of existing thrombi in vivo. Although endogenous fibrinolysis probably plays a role in clot resolution, whether heparin exerts any direct influence on clot thrombolysis remains unknown.

Several mechanisms to explain spontaneous relief of stenosis have been proposed, including relief



of vasospasm, distal embolization of an occlusive clot, endothelial spontaneous thrombolysis, and vasa vasorum formation.<sup>23</sup> The administration of low-molecular weight heparin and antiplatelet agents, such as were given to our patient, may play a role also. Although the incidence, mechanism, and timing of spontaneous recanalization of an extracranial ICA are not clear at present, its detection is important in order to determine whether surgery or stenting should be performed or is unnecessary.

## CONCLUSION

This case illustrates an infrequent phenomenon, early spontaneous recanalization of a highly stenosed extracranial ICA, and suggests the advisability of checking up immediately in patients who experience a large improvement in neurological function, especially those who are potential surgical or stenting candidates, and examining them to see if recanalization has occurred, using CCDU and confirming the results with MRA or CTA, all safe and minimally invasive methods. CCDU is a better follow-up tool to use than MRA or CTA before considering invasive treatment in ischemic stroke patients with high-grade carotid stenosis because it is feasible to use this imaging technique in all stroke patients and it carries little risk of overestimating carotid stenosis and no risk for allergy to contrast medium. Failure to recognize recanalization could result in inappropriate surgical decisions and suboptimal secondary prophylaxis strategies and affect the patient's clinical outcome.

## REFERENCES

1. Primary prevention of ischemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council: cosponsored by the Atherosclerotic Peripheral Vascular Disease Interdisciplinary Working Group; Cardiovascular Nursing Council; Clinical Cardiology Council; Nutrition, Physical Activity, and Metabolism Council; and the Quality of Care and Outcomes Research Interdisciplinary Working Group: the American Academy of Neurology affirms the value of this guideline. *Stroke* 2006;37:1583-1633.
2. North American Symptomatic Carotid Endarterectomy Trial Collaborators. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. *N Engl J Med* 1991;325:445-453.
3. MRC European Carotid Surgery Trial. Interim results for symptomatic patients with severe (70-99%) or with mild (0-29%) carotid stenosis: European Carotid Surgery Trialists' Collaborative Group. *Lancet* 1991;337:1235-1243.
4. Sindermann F, Brugel R, Giedke H. Spontaneous recanalization of internal carotid artery occlusions. *Neuroradiology* 1974;7:53-56.
5. Hoshino H, Takagi M, Takeuchi I, et al. Recanalization of intracranial carotid occlusion detected by duplex carotid sonography. *Stroke* 1989;20:680-686.
6. Keller HM, Schubiger O, Turina M, et al. Disappearing obstructions in extracranial cerebral arteries. *Acta Neurol Belg* 1980;80:19-29.
7. Schelp R, Bickel A, Kolominsky P, et al. Spontaneous recanalization of the totally occluded internal carotid artery under heparinisation—a neglected phenomenon? Two cases reports and review of the literature. *Fortschr Neurol Psychiatr* 1995;63:277-282.
8. Manganaro A, Ruggeri M, Ando G, et al. Endothelial functions in pathophysiology of thrombosis and fibrinolysis: late recanalization of an occluded internal carotid artery. A case report. *Angiology* 2002;53:99-103.
9. Kassem-Moussa H, Graffagnino C. Non-occlusion and spontaneous recanalization rates in acute ischemic stroke. *Arch Neurol* 2002;59:1870-1873.
10. Calleja S, De la Vega V, Llaneza JM, et al. Spontaneous recanalization of acute internal carotid artery occlusion. *Ann Vasc Surg* 2004;27:490-493.
11. Camporese G, Verlato F, Salmistraro G, et al. Spontaneous recanalization of internal artery occlusion evaluated with color flow imaging and contrast arteriography. *Int Angiol* 2003;22:64-71.
12. Nguyen-Huynh MN, Lev MH, Rordorf G. Spontaneous recanalization of internal carotid artery occlusion. *Stroke* 2003;34:1032-1034.
13. Meves SH, Muhs A, Federlein J, et al. Recanalization of acute symptomatic occlusions of the internal carotid artery. *J Neurol* 2002;249:188-192.
14. Bridgers SL. Clinical correlates of Doppler/ultrasound errors in the detection on internal carotid artery occlusion. *Stroke* 1989;20:612-615.
15. Polak JF, Bajakian RL, O'Leary DH, et al. Detection of internal carotid artery stenosis: comparison of MR angiography, color Doppler sonography, and arteriography. *Radiology* 1992;182:35-40.
16. Edelman RR, Heinrich PM, Wallner B, et al. Extracranial carotid arteries; evaluation with "black blood" MR angiography. *Radiology* 1990;177:45-50.
17. Heiserman JE, Drayer BP, Fram EX, et al. Carotid artery stenosis: clinical efficacy of two-dimensional time-of flight MR angiography. *Radiology* 1992;182:761-768.
18. Sameshima T, Futami S, Morita Y, et al. Clinical usefulness of and problems with three-dimensional CT angiography for the evaluation of arteriosclerotic stenosis of the carotid artery: comparison with conventional angiography, MRA, and ultrasound sonography. *Surg Neurol* 1999;51:301-308.
19. Sturzenegger M, Mattle HP, Rivoir A, et al. Ultrasound findings in carotid artery dissection: analysis of 43 patients. *Neurology* 1995;45:691-698.
20. Pelz DM, Buchan A, Fox AJ, et al. Intraluminal thrombus of the internal carotid arteries: angiographic demonstration of resolution with anticoagulant therapy alone. *Radiology* 1986;160:369-373.
21. Gonzalez A, Mayol A, Gil-Peralta A, et al. Angioplasty of symptomatic high-grade internal carotid artery stenosis with intraluminal thrombus: therapeutic approach. *Neuroradiology* 2004;46:313-317.
22. Corrado G, Tadeo G, Beretta S, et al. Atrial thrombi resolution after prolonged anticoagulation in patients with atrial fibrillation. *Chest* 1999;115:140-143.
23. Colon PG, Deveikis JP, Dickinson LD. Revascularization of occluded internal carotid arteries by hypertrophied vasa vasorum: report of 4 cases. *Neurosurgery* 1999;45:634-637.