Case Reports

Endovascular Recanalization of Basilar Artery Occlusion 80 Days After Symptom Onset

Wengui Yu, MD, PhD; Varoujan Kostanian, MD, PhD; Mark Fisher, MD

Background and Purpose—Acute basilar artery occlusion portends high risk of stroke and death. Thrombolysis or endovascular therapy has been limited to patients who present within hours of symptom onset. Without recanalization, acute basilar artery occlusion almost always results in death or severe disability.

Summary of Case—We report a case of basilar artery occlusion and successful endovascular recanalization 80 days after symptom onset.

Conclusions—Endovascular therapy can be feasible and safe for symptomatic basilar artery occlusion at chronic stage. (Stroke. 2007;38:1387-1389.)

Key Words: basilar artery occlusion — endovascular treatment — stroke — therapy — vertebrobasilar disease

Asilar artery (BA) supplies brain stem, cerebellum, thalamus, occipital lobes, and medial temporal lobes. Acute occlusion of this artery produces high risk of stroke and death.1–3 Intravenous or intra-arterial thrombolysis result in 40% to 65% recanalization, with 22% to 24% good outcome.3–7 Without recanalization, the likelihood of good outcome is ≈2%.7 We describe a case of BA occlusion and its successful treatment with endovascular stenting 80 days after symptom onset.

Case Reports

The patient was a 55-year-old right-handed man who initially presented to a local hospital with sudden onset of headache, vomiting, slurred speech, right-sided weakness, and marked alteration of vision seeing only “snow”. He had no history of hypertension, diabetes or coronary artery disease. However, he smoked 1 to 2 packs per day for 40 years and had chiropractic neck manipulations. His laboratory tests and cardiac work-up were unremarkable. A brain MRI 2 days later showed multiple infarcts in the posterior circulation (Figure 1A through 1C). There was a high intensity signal (thrombus) within the distal BA on fluid-attenuated inversion recovery (Figure 1A through 1C) and T1 image (Figure 1D). The BA occlusion was shown extending from mid-segment to the apex on the sagittal T1 MRI (arrows in Figure 1E) and MR angiography study (Figure 1F).8 Over the following weeks, the patient showed gradual improvement in speech and right-sided weakness. However, he continued to have episodic headaches and blurred vision. The headache had been diffuse, nonthrombing, and lasting up to days at a time. He was given topiramate and nortriptyline for headache and discharged to a rehabilitation center.

In the following months, he had chronic headaches, blurred vision, and 4 to 5 episodes of transient loss of peripheral vision. The symptoms were exacerbated by activities such as reading or watching TV. His wife also noted substantial personality changes, confusion and memory loss. When referred to us ≈80 days after symptom onset, his neurological examination was significant for disorientation to time, anterograde amnesia, and mild right-sided hyperreflexia. His immediate recall was intact, but he could not retain information for a few minutes and was unable to tell the history of his present illness. He was given aspirin for stroke prevention. A repeat brain MRI showed old infarcts without new lesion (not shown). The repeat MR angiography (Figure 2A) and a CT angiography showed persistent BA occlusion. There was no significant stenosis or calcification in other cerebral vasculature. Laboratory studies showed total cholesterol 114, triglycerides 86, LDL cholesterol 86, and HDL cholesterol 19. His symptoms were debilitating, and the patient and his wife gave informed consent for endovascular therapy. He was loaded with clopidogrel (300 mg). Selective microcatheter angiograms via right vertebral artery injection confirmed mid-BA occlusion (Figure 2B). Right internal carotid artery angiogram revealed a small posterior communication artery supplying collateral blood flow to both posterior cerebral arteries with delayed fillings. There was no retrograde flow in the distal BA and superior cerebellar arteries. The left internal carotid artery angiogram showed no significant cross-fillings of the posterior circulation. After a complete diagnostic evaluation, the BA occlusion site was passed by the use of a microcatheter over a guide wire. Repeat biplane angiography showed a focal, occlusive BA dissection with a true lumen.

Received August 8, 2006; final revision received November 6, 2006; accepted November 15, 2006.
From the Departments of Neurology (W.Y., M.F.), Radiology (V.K.), Anatomy & Neurobiology (M.F.), University of California, Irvine.
Current affiliation for W.Y.: Department of Neurosurgery, UT Southwestern Medical Center, Dallas, Tex.
Correspondence to Wengui Yu, MD, PhD; Department of Neurosurgery, UT Southwestern Medical Center, 5323 Harry Hines Blvd, Dallas, TX 75390-8855. E-mail wyu@yahoo.com
© 2007 American Heart Association, Inc.

Stroke is available at http://www.strokeaha.org DOI: 10.1161/01.STR.0000260186.93667.a2

1387
canulated with the microcatheter. Because of filling defect and meniscus signs at the dissection site, suggesting superimposed intraluminal thrombus, a 4×12-mm balloon-expandable stent was placed and deployed across the lesion (Figure 2C). After stenting, a distal BA thrombus was visualized and lysed with intra-arterial tissue plasminogen activator (10 mg) and abciximab (20 μg). Despite combined thrombolysis, the thrombus was only partially lysed and a
second, smaller stent (4×8 mm) was placed to reopen the distal BA (Figure 2D). Intravenous heparin was administered during the procedure to maintain an activated clotting time between 2 and 2.5 times normal. The systolic blood pressure was kept at 120 to 140 mm Hg to prevent hyperperfusion syndrome. After the procedure, the patient was placed on IV abciximab infusion (0.125 μg/kg per minute) for 12 hours. Follow-up MRI showed only small asymptomatic new infarcts in the right cerebellum (Figure 2E and 2F). He was discharged home with aspirin and clopidogrel 3 days after the procedure. His headache resolved completely and his memory also gradually improved after the treatment. CT angiography 6 months later showed patent BA with excellent distal flow.

Discussion

Improved technology and expertise have enabled recanalization of totally occluded coronary, subclavian or internal carotid artery in the chronic stage. Successful percutaneous intervention for chronic total occlusion of coronary artery is associated with improved clinical outcome and lower cumulative rates of cardiac death and infarction.

To the best of our knowledge, there has been no report of endovascular therapy of a totally occluded BA during the chronic stage. Thrombolysis or endovascular therapy have been limited to acute BA occlusion with the longest reported time-delay of 79 hours after symptom onset. Extracranial-intracranial arterial bypass may be feasible. However, superior temporal artery-superior cerebellar artery or superior temporal artery-posterior cerebral arteries bypass was not only technically challenging but also associated with high risks of serious complications and mortality.

The patient described in this report had debilitating symptoms of posterior circulation insufficiency from BA occlusion. The symptoms were exacerbated by activities such as watching TV or reading. Although the limited collaterals appeared to maintain baseline perfusion, they often failed to provide sufficient blood flow during periods of increased oxygen demand, resulting in lifestyle-limiting symptoms. His symptoms resolved after successful endovascular stenting. It should be emphasized that endovascular recanalization of chronic BA occlusion is extremely risky, and a prospective randomized trial may be warranted to evaluate the safety and efficacy of such therapy.

Disclosures

None.

References

Endovascular Recanalization of Basilar Artery Occlusion 80 Days After Symptom Onset
Wengui Yu, Varoujan Kostanian and Mark Fisher

*Stroke*. 2007;38:1387-1389; originally published online February 22, 2007;
doi: 10.1161/01.STR.0000260186.93667.a2

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2007 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://stroke.ahajournals.org/content/38/4/1387

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Stroke* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to *Stroke* is online at:
http://stroke.ahajournals.org//subscriptions/