

Endovascular embolectomy of acute basilar artery occlusion

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Abstract—Acute basilar artery occlusion has a mortality rate approaching 90%. The authors describe a case of acute basilar artery occlusion managed successfully with endovascular embolectomy. A 31-year-old man sought treatment for confusion, dysarthria, and right-sided weakness. He soon became unresponsive and was found to have a vertebral artery dissection and an associated basilar artery embolism. The dissection was managed with endovascular stenting, and the basilar artery embolus was removed with a clot retriever at 7 hours. The patient recovered without neurologic deficit.

NEUROLOGY 2003;61:1421–1423

Acute basilar artery occlusion is a life-threatening event with mortality rates of 75 to 91%.^{1,2} Patients who survive rarely regain functional independence and are at risk for recurrent strokes.^{1,3} In a case report, some patients were noted to have a benign outcome if they had short segment basilar artery occlusion of atherothrombotic origin and adequate collateral supply.⁴ Although anticoagulation has been used for decades as the therapy of the choice, the mortality rate of acute basilar artery occlusion was 88% with heparin therapy.³ Recently, a number of thrombolytic agents have been investigated for the management of acute basilar artery occlusion. Despite good recanalization rate with aggressive intra-arterial (IA) thrombolysis, the mortality rate in most case series has remained 65 to 75%.^{2,5,6} Therefore, it is imperative to develop more effective therapy for this devastating condition. In this report, we describe a case of vertebral artery dissection and associated acute basilar artery occlusion and the successful endovascular therapy of both lesions.

Case report. The patient was a healthy 31-year-old Asian man without previous illness. He was struck on his face with head twisting sideways during a football game. He was dazed and had brief episodes of confusion. His neurologic examination and CT of the head at a local hospital were reported as normal. A few hours later, he developed “seizure-like activity” and was sent to a trauma center for further evaluation. On arrival, he was noted to have dysarthria and right-sided weakness. A repeat CT showed a hyperdense basilar artery on two consecutive 5-mm cut images, indicative of acute basilar artery embolism. He soon became incoherent and unresponsive with marked vertical nystagmus. Neither systemic anticoagulation nor IV thrombolytic therapy was initiated because of a preliminary report of possible subarachnoid hemorrhage. After intubation for airway protection, he was emergently transferred to the University of California, San Francisco 4 hours after symptom onset. Family consent was obtained for endovascular therapy after full discussion of the benefit and risks of

such therapy under an approved Food and Drug Administration (FDA) protocol for use of an experimental clot-retrieval device for acute cerebrovascular emboli. After an IV bolus of 2,000 U heparin, diagnostic angiography of the right vertebral artery demonstrated complete occlusion with an abrupt cutoff at the midcervical portion of the vertebral artery at C₄-C₅ (figure 1, A and B). The obstruction was gently traversed with a UCSF-2 catheter over a Bentson guidewire (Cook Incorporated, Bloomington, IN), and an injection of contrast medium confirmed the patency of the distal right vertebral artery above the dissection site. IV abciximab was initiated with a bolus injection of 0.25 mg/kg followed by an infusion of 0.125 μg/kg/min for 12 hours. A 6- × 40-mm Magic Wall Stent (Boston Scientific, Natick, MA) was then placed across the area of the dissection site with excellent restoration of blood flow (figure 1, C and D) at approximately 6 hours after symptom onset. Angiography of the now patent right vertebral artery demonstrated a large embolus in the distal basilar artery occluding both superior cerebellar arteries (SCAs) and the right posterior cerebral artery (PCA) (figure 2A). An initial attempt at embolectomy was made with the Neuronet (Guidant Corporation, Santa Clara, CA) to capture the embolus in the distal basilar artery but was unsuccessful. The Concentric Retriever (Concentric Medical, Mountain View, CA) was then used for clot removal. The device was advanced through a 7-French guide catheter in the right vertebral artery, and the tip was placed in the right PCA across the embolus over an Agility 0.014-inch microguidewire (Cordis Neurovascular, Miami, Florida) (figure 2B). The clot was captured with the helical coils of the device (figure 2C) and pulled back into the 7-French guide catheter. It was then removed in toto from the guide catheter through the femoral artery sheath at 7 hours after stroke onset. The embolus recovered from embolectomy measured 5 × 25 mm (figure 2D). Follow-up angiography demonstrated complete recanalization of the basilar artery with full restoration of blood flow to both SCAs and the right PCA (figure 2E). The patient regained complete consciousness after the stent and embolectomy and was extubated with only mild residual nystagmus and left-sided dysmetria for a few hours. A follow-up brain MRI showed only a small infarction in the left cerebellum and no infarct in the brainstem (figure 3). The patient was discharged home with aspirin and clopidogrel 2 days after the procedure. Two months later, he complained of episodes of lightheadedness and was found to have a high-grade stenosis at the proximal segment within the stent in the right vertebral artery. The stenosis was managed with balloon angioplasty without

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Supported as part of the MERCI Clinical Investigation by Concentric Medical, Mountain View, CA. W.S.S. had stock ownership of the company. None of the other authors received financial support or compensation from the corporate sponsor.

Received September 3, 2003. Accepted in final form September 22, 2003.

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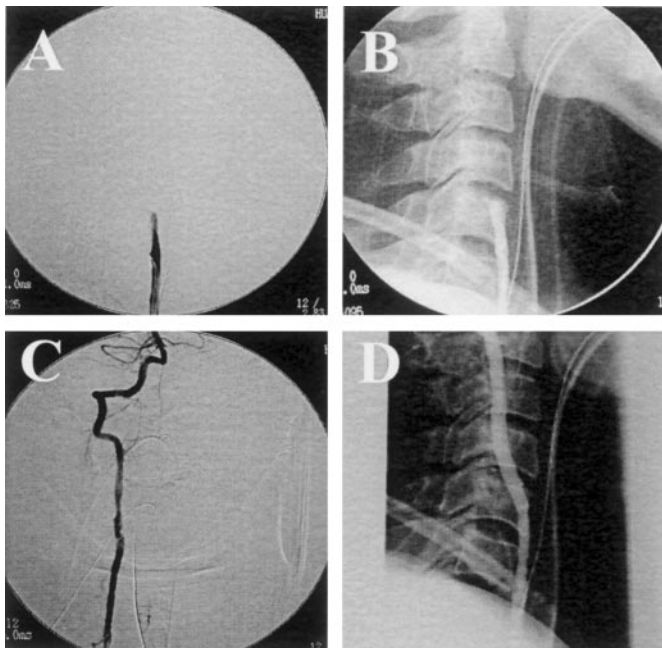


Figure 1. Diagnostic angiography of right vertebral artery showed complete occlusion with an abrupt cutoff at the midcervical portion of the vertebral artery at C₄-C₅ level (A, B). After mechanical recanalization and abciximab infusion, the right vertebral dissection was visualized (C) and stented (D).

complication. He has returned to work and remained neurologically intact 9 months after embolectomy.

Discussion. Until recently, anticoagulation with IV heparin was the therapy of choice for acute basilar artery occlusion based on anecdotal reports. However, in a retrospective study of 25 patients who had acute basilar artery occlusion requiring mechanical ventilation, the mortality rate was 88% despite anticoagulant therapy.³ Three patients survived with a locked-in syndrome without further neurologic improvement. Therefore, the therapy is of little benefit. Recently, thrombolytic agents, including urokinase, streptokinase, and tissue plasminogen activator (tPA), have been administered either IV or IA for acute basilar artery occlusion. Although recanalization rates have approached 44 to 83%, the mortality rate has remained 65 to 75% because of rethrombosis, hemorrhagic complications, and irreversibility of the stroke itself.^{2,5-7} Of note, patients with distal basilar artery occlusion and adequate collateral circulation tended to have a lower mortality rate of approximately 50% with IA thrombolysis.⁶ However, the overall good outcome with no or mild deficit was only 20 to 25%.^{6,7}

Stenting of dissected extracranial arteries has been reported. However, mechanical removal of emboli from intracranial arteries has only been described in three case reports. Microsnares have been used to retrieve foreign bodies, such as detachable coils and catheter fragments, in the circulation. Such a device was first used successfully to retrieve an embolus from the mid-

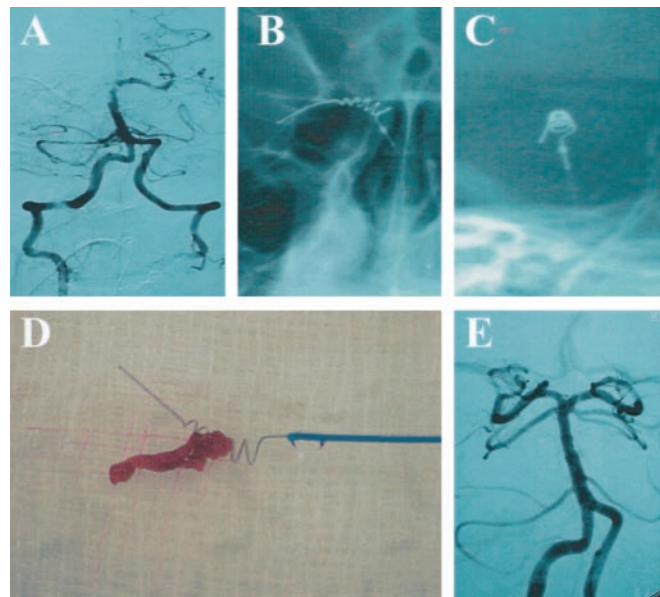


Figure 2. Angiography of the stented right vertebral artery demonstrated a large embolus occluding both superior cerebellar arteries (SCAs) and right posterior cerebral artery (PCA) (A). The clot was captured by the soft helical coils of the Concentric Retriever and removed in toto from the guide catheter through the femoral artery sheath (B, C). The embolus recovered measured 5 × 25 mm (D). Repeat angiography demonstrated complete recanalization of the distal basilar artery with full restoration of blood flow to both SCAs and the right PCA (E).

dle cerebral artery in one patient with an acute stroke.⁸ Subsequently, there are two reports of successful retrieval of clots from the basilar artery using the snare and Neuronet.^{9,10} In one of these reports, five patients with basilar artery occlusion underwent mechanical removal of an embolus using the Neuronet.⁹ The device failed to retrieve clots in the initial two patients but succeeded in the subsequent three patients with flow reversal by temporarily occluding the vertebral artery. In this report, we initially attempted embolectomy with the Neuronet but were unable to capture the embolus with this device. However, the embolus was suc-

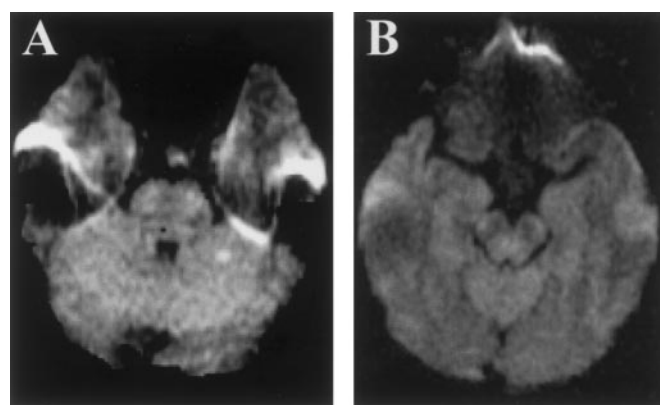


Figure 3. Diffusion-weighted MRI at day 2 after embolectomy showed a small cerebellar infarction (A) and no infarct in the brainstem (B).

cessfully captured with the Concentric Retriever and pulled back into the guide catheter without using flow reversal as described.⁹ The patient recovered quickly without neurologic deficit and remains healthy 9 months after the procedure.

Compared with thrombolytic therapy, endovascular embolectomy theoretically has a lower risk of hemorrhagic complications and can be used in patients with contraindications for thrombolytic therapy. Per FDA-approved protocol, patients evaluated within 8 hours of symptom onset are eligible for the clinic trial of embolectomy. It must be noted that experienced neurointerventional specialists may currently perform this procedure at 25 investigational sites in the United States. Although large randomized controlled studies are required to evaluate the safety and effectiveness of various clot-retrieval devices, our report corroborates previous reports^{9,10} and indicates that endovascular embolectomy may be a promising therapy for life-threatening basilar artery occlusion and other types of strokes.

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Compound heterozygous *PANK2* mutations confirm HARP and Hallervorden-Spatz syndromes are allelic

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Abstract—The authors describe a patient with hypoprebetalipoproteinemia, acanthocytosis, retinitis pigmentosa, and pallidal degeneration (HARP) who has two compound heterozygote mutations of the *PANK2* gene. IVS4–1 G>T segregates with the lipid and erythrocyte changes in the mother and sister. No other family members have the lipid, erythrocyte, or clinical abnormalities. The father and two brothers are heterozygous for Met327Thr. One other mutation has been found in this *PANK2* region associated with the HARP phenotype, suggesting a local genotype effect.

NEUROLOGY 2003;61:1423–1426

Hypoprebetalipoproteinemia, acanthocytosis, retinitis pigmentosa, and pallidal degeneration (HARP; OMIM 200150) syndrome^{1,2} is a rare disorder that shares many common clinical features with Hallervorden-Spatz syndrome (HSS), also termed neurodegeneration with brain iron accumulation 1 (NBIA1). HSS is characterized by early onset dystonia, spasticity, and dementia. Rare clinical features include pigmentary retinopathy, choreoathetosis, ri-

gidity, seizures, and optic atrophy.^{3,4} Neuropathologically, HSS is characterized by bilateral degeneration of the globus pallidus and substantia nigra pars reticulata with deposition of iron in the affected regions and more widely distributed neuronal axonal spheroids.^{5,6} Lewy bodies and alpha-synuclein immunoreactivity have also been identified in HSS, suggesting a link with other neurodegenerative disorders such as Parkinson disease and multiple

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Supported by the Wellcome Trust. H.H. was recipient of a Bogue Research fellowship.

Received March 27, 2003. Accepted in final form June 13, 2003.

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