THERAPEUTIC BASILAR ARTERY OCCLUSION FOR MANAGEMENT OF MEDICALLY REFRACTORY BASILAR ARTERY STENOSIS: CASE REPORT

Michael Horowitz, M.D.

Departments of Neurosurgery and Radiology, University of Pittsburgh Medical Center and Presbyterian University Hospital, Pittsburgh, Pennsylvania

Tudor G. Jovin, M.D.

Department of Neurology, VA Pittsburgh Medical Center, University of Pittsburgh Medical Center, and Presbyterian University Hospital, Pittsburgh, Pennsylvania

James M. Gebel, M.D.

Department of Neurology, University of Pittsburgh Medical Center and Presbyterian University Hospital, Pittsburgh, Pennsylvania

Reprint requests:
Michael Horowitz, M.D., Department of Neurosurgery, University of Pittsburgh Medical Center and Presbyterian University Hospital, Suite B400, 200 Lothrop Street, Pittsburgh, PA 15213.
Email: horowitzmb@upmc.edu

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OBJECTIVE AND IMPORTANCE: To describe a novel therapeutic approach (endovascular basilar artery occlusion) to a notoriously difficult-to-manage clinical condition (actively symptomatic high-grade basilar artery stenosis) on the basis of assessment of the patient-specific mechanism of disease.

CLINICAL PRESENTATION: An 81-year-old woman presented with recurrent episodes of brainstem ischemia refractory to aggressive medical therapy. Cerebral angiography revealed a high-grade proximal basilar artery stenosis. On the basis of clinical presentation and angiographic findings, the pathogenesis of this complex of symptoms was thought to be embolic rather than hemodynamic.

INTERVENTION: Endovascular coil occlusion of the basilar artery was used, with excellent outcome (cessation of ischemic symptoms and independent level of functioning at 1 yr).

CONCLUSION: Successful endovascular management of intracranial occlusive disease requires understanding of the mechanism responsible for the patient's symptoms.

KEY WORDS: Basilar artery stenosis, Medically refractory, Therapeutic occlusion

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Basilar artery (BA) stenosis can present in a variety of ways, ranging from transient ischemic events secondary to brainstem, cerebellar, or occipital emboli to disabling stroke or death. Like other large-vessel cerebral occlusive syndromes, ischemia in this setting is believed to be caused by distal embolization, hemodynamic insufficiency, or a synergistic combination of the two (3). Treatment has traditionally included medical therapy aimed at control of atherosclerotic risk factors combined with antiplatelet agents and/or anticoagulants. More recently, endovascular therapy using coronary angioplasty balloons and stents has broadened our ability to treat this often severely debilitating disease. Several case series in the literature attest to the feasibility and relative safety of this procedure, but no randomized prospective trial has yet been undertaken to formally evaluate the efficacy of this procedure compared with medical therapy alone. Moreover, the optimal endovascular treatment modality for this condition is not known. Options include pharmacological thrombolysis with or without angioplasty/stenting, angioplasty alone, and angioplasty followed immediately or as a staged procedure by stenting. With the availability of newer devices and medications, consideration of the disease mechanism underlying the patient's complaints allows the treating physician to choose the appropriate treatment modality. This report presents a novel approach to the management of medically refractory BA stenosis in a patient whose stroke mechanism was thought to be embolic rather than hemodynamic. Consequently, endovascular BA occlusion, a procedure that is technically less challenging and therefore less prone to complications, was chosen over flow-restoring therapy with BA angioplasty/stenting.

CASE REPORT

An 81-year-old right-handed, independently living woman presented with 3-minute-long episodes of dizziness associated with right hemiparesis and dysarthria. Her stroke risk factors were hypertension, hyperlipidemia, and diabetes mellitus. A brain

magnetic resonance imaging (MRI) scan/magnetic resonance angiogram revealed a critical proximal BA stenosis and a recent right superior cerebellar artery distribution infarct (*Fig. 1*). Intravenously administered heparin was initiated, and the patient initially became free of symptoms. She was offered participation in the Warfarin-Aspirin Symptomatic Intracranial Disease trial but declined to participate. While on intravenous heparin, transitioning to oral anticoagulation, she developed new symptoms (alteration in level of consciousness, left hemiparesis), prompting addition of aspirin to her anticoagulation regimen. She continued, however, to have recurrent posterior circulation ischemic events, all transient and all different from one another with respect to clinical manifestation. These spells occurred on a daily basis for 1 week despite progressive addition of clopidogrel, simvastatin, and fludrocortisone to her medication regimen and blood pressure augmentation therapy with intravenous fluids. A repeat brain MRI scan revealed several new small infarcts (*Fig. 2*).

The patient underwent cerebral arteriography, which demonstrated a 99% stenosis of the proximal BA measuring 6.5 mm in length (*Fig. 3*). Because of persistent symptoms despite maximal medical therapy, consideration was given to various endovascular options, including angioplasty and angioplasty/stenting of the BA. A left posterior communicating artery was present, with opacification of the basilar system seen on left common carotid arteriography (*Fig. 4*). Because the patient had excellent collateral opacification of the posterior circulation yet continued to have transient ischemic events, we thought that the cause of her symptoms was emboli forming at the stenosis. Rather than attempt a primary angioplasty or angioplasty/stenting, we opted to simply eliminate the embolic source by completely occluding the BA at the stenosis. This was achieved by use of a microcatheter and several detachable platinum Guglielmi coils (Boston Scientific/Target, Fremont, CA) (*Fig. 5*). The procedure was complicated by a brief episode of alteration in level of consciousness without focal neurological symptoms. This was followed by complete resolution of her spells. She was maintained on warfarin, and at 1 year, she remained asymptomatic and continued to live independently at home.

DISCUSSION

The natural history of BA stenosis has been described by the Warfarin-Aspirin Symptomatic Intracranial Disease trial investigators, who followed up 28 patients with 50 to 90% stenosis, all of whom had had previous transient ischemic attacks or strokes in the basilar territory (12). The investigators reported a 10.7% stroke rate in the territory of the stenotic vessel (10.7 per 100 patient-yr) despite medical therapy (12). The most common transient ischemic attack symptoms include diplopia, dizziness with or without vertigo, lower-extremity weakness, and/or weakness alternating between different limbs in different attacks. BA thrombosis carries an 80 to 100% mortality without therapy, although patients with distal BA occlusion may have a higher survival rate than those with proximal or midbasilar occlusion (4, 12). In patients who survive, BA stenosis with subsequent embolization or thrombosis and occlusion can result in BA distribution strokes. These may present with extremity paralysis; bulbar or pseudobulbar palsy; abnormalities of eye movements, including nystagmus, ocular skewing, and ptosis; and coma or reduction in level of consciousness.

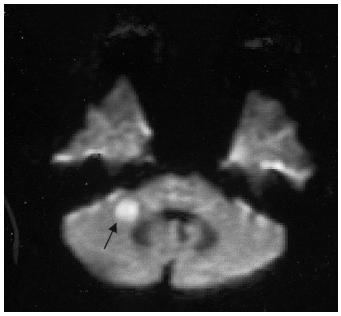

FIGURE 1. *Diffusion axial MRI scan showing right middle cerebellar peduncle ischemic change.*

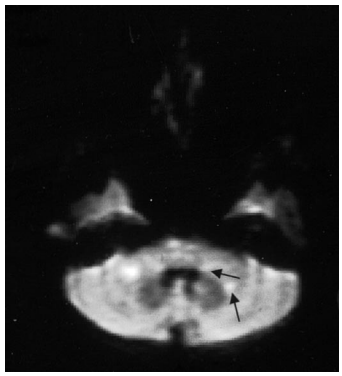

FIGURE 2. *Diffusion axial MRI scan showing new ischemic changes* (arrows).

Considerable effort has been exerted during the past few years by a number of investigators, including ourselves, to manage medically refractory symptomatic BA stenosis by use of percutaneous transluminal angioplasty (PTA) with or without arterial stenting. In 1992, Ahuja et al. (1) reported the use of PTA for symptomatic BA stenosis. Although the vessel failed to remain patent at 6-month follow-up, this report marked the first step in endovascular management of this potentially debilitating disease. In 1996, Nakatsuka et al. (11) reported two cases of BA angioplasty for stenosis. In 1999, Jimenez et al. (8) published one case of successful BA angioplasty and reviewed 36 published cases relating to BA stenosis and PTA. The concept of using stenting along with PTA stems from the cardiac intervention experience. Abrupt vessel closure after coronary PTA occurs in 5 to 28% of cases (2). In 1997, Higashida et al. (6) placed a Palmaz-Schatz PS 1540 articulated stent (Johnson & Johnson Interventional Systems, Warren, NJ) into the BA to help treat a fusiform basilar aneurysm. In 1999, Horowitz et al. (7) described three cases of BA PTA and stenting for basilar stenosis. No immediate procedure-related complications were reported, although one patient did develop an in-stent aneurysm, seen on follow-up angiography. In 1999, Gomez et al. (5) reported 12 BA stenosis cases managed with stents, with no procedure-related complications. Levy et al. (9) reported an additional 11 cases in 2001. Four procedure-related deaths, one case of in-stent hyperplasia, and one in-stent aneurysm point to the dangers related to this procedure and the need for improved technology to reduce procedure-related complications. Mori et al. (10) reviewed the risk of stroke or ipsilateral bypass surgery in patients undergoing coronary PTA for intracranial disease and found it to be related to the morphological characteristics of the atheroscle-

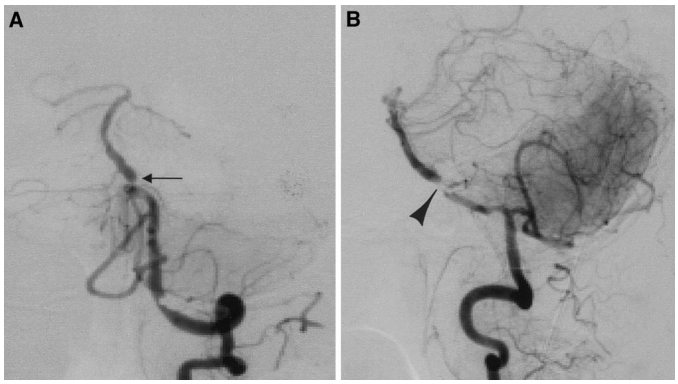

FIGURE 3. A, *anteroposterior left vertebral artery angiogram showing BA stenosis (*arrow*).* B, *lateral left vertebral artery angiogram showing BA stenosis (*arrowhead*).*

rotic plaque: Mori Type A lesions were associated with a relatively low risk (8%), whereas Mori Type B and especially Mori Type C lesions were associated with high risk (26 and 87%, respectively).

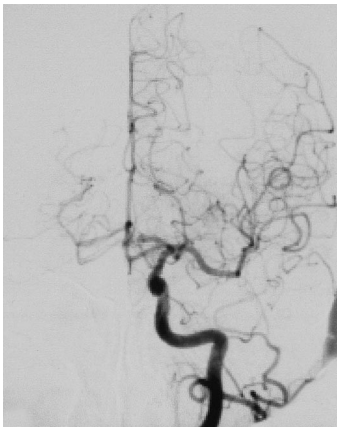

FIGURE 4. *Anteroposterior left common carotid artery arteriogram before therapeutic BA occlusion showing left posterior cerebral artery opacification via left posterior communicating artery.*

Despite the urge to provide complete restoration of flow in a stenotic vessel, it is important for the interventionalist to first determine the pathogenesis of a patient's symptoms, understand all sources of collateral circulation, and finally choose the safest means of eliminating the anatomic defect. In the case presented here, the pathogenesis of the patient's transient ischemic events despite maximal medical therapy was not hemodynamic insufficiency but rather repeated embolic events secondary to an unstable atherosclerotic lesion. Perforator disease was also considered as a possibility but was thought to be less likely, given that some of the infarcts occurred in the right anteroinferior cerebellar artery and superior cerebellar artery territories whose origins are distal to the atherosclerotic plaque, supporting the embolic nature of the patient's symptoms. Ideally, the absence of hemodynamic impairment below and above the stenosis should have been confirmed by perfusion studies (xenon computed tomography, computed tomographic perfusion, magnetic resonance perfusion, single-photon emission computed tomography) that were available for clinical decision-making at our institution. However, none of these imaging modalities have been shown to be capable of reliably assessing perfusion in the brainstem, which is the area affected by ischemia in this patient. We therefore chose to base our assessment of the hemodynamic situation on clinical grounds (nonstereotyped spells that had no relationship with change in blood pressure or body position) and angiographic data (good collateral flow above and below the stenosis). Balloon test occlusion of the BA was considered but was not performed, for the following reasons: 1) at 99% stenosis, the vessel is practically occluded; 2) occlusion at or distal to the stenosis would have required passage of a microguidewire and balloon through the stenotic area, potentially increasing the risk of embolic events; and 3) occlusion below the stenosis could have involved proximal BA perforators, potentially giving misleading information with regard to the patient's symptoms.

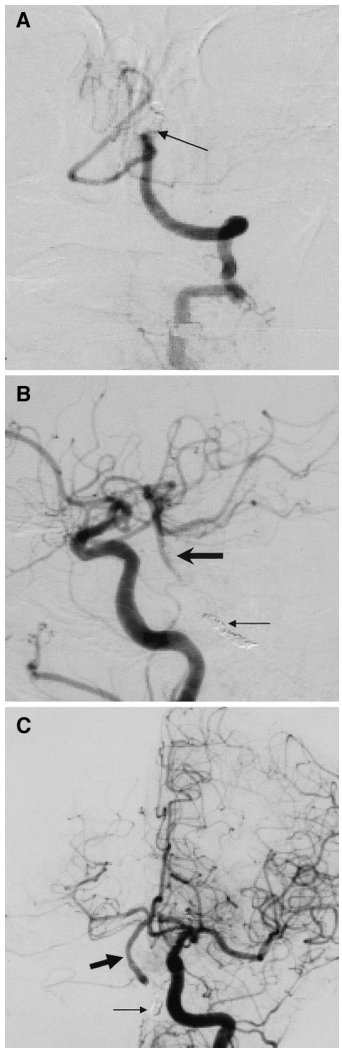

FIGURE 5. A, *left vertebral artery arteriogram after BA sacrifice.* Arrow *points to platinum coil mass.* B, *lateral left common carotid artery arteriogram showing retrograde filling of BA via the left posterior communicating artery (*large arrow*) after coil sacrifice of the proximal BA at the site of the previous symptomatic stenosis (*small arrow*).* C, *anteroposterior left common carotid artery arteriogram showing retrograde filling of BA via the left posterior communicating artery (*large arrow*) after coil sacrifice of the proximal BA at the site of the previous symptomatic stenosis (*small arrow*).*

The risks of angioplasty and stenting to alleviate the stenosis in this Mori Type B lesion included vessel rupture and death, acute vessel occlusion, and delayed instent occlusion or restenosis and were considered higher than the risks of occluding an already nearly occluded vessel, given the low clinical suspicion of a flow-limiting lesion in view of the patient's excellent collateral flow. Furthermore, the risks were reduced further by performing the procedure in a controlled setting with anticoagulation in place and pharmaceutical blood pressure control available. By eliminating the source of the patient's emboli, we thought we could successfully treat her symptoms without exposing her to the additional risks of a more invasive and manipulative procedure.

CONCLUSION

This case report underscores the crucial importance of understanding the pathogenesis of a patient's symptoms as a prerequisite for successful management of intracranial vascular occlusive disease. In selected cases of failed maximal medical therapy with no hemodynamic component to the patient's symptomatology, endovascular occlusion of the BA may be considered a potential therapeutic option if the morphological aspect of the lesion is considered unfavorable for flow-restoring therapy. Only by determining whether or not symptoms are secondary to hemodynamic insufficiency or embolic events can one successfully select appropriate therapy. Once the pathogenesis is surmised, a careful evaluation of collateral circulation is necessary so that a determination can be made concerning the importance of maintaining normal vascular patterns. With the above data in hand, the physician can weigh the risks and benefits of various therapies and choose the treatment that it is thought will provide a long-term solution with minimal risk for immediate and delayed complications.

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COMMENTS

The authors present a very interesting case of a patient with posterior circulation transient ischemic attacks that are referable to a severe basilar artery (BA) stenosis. On the basis of the available information, the case was diagnosed as being related to emboli rather than hemodynamic insufficiency. It is also well known that with carotid artery stenosis, most of the neurological problems that result from carotid artery disease relate to embolic phenomena and not hemodynamic deficiency. Therefore, proximal occlusion is an effective treatment when revascularization procedures are more dangerous than a deconstructive procedure. Given the situation in this case report, the authors made the correct decisions, and the therapy was successful.

At our institution, the complications from BA angioplasty and stenting have been very significant. This procedure should be reserved for patients who have no other therapeutic options. Proximal occlusion is a good alternative when conditions are favorable.

Robert A. Solomon
New York, New York

The authors describe the unconventional but successful management of an octogenarian presenting with medically refractory posterior circulation ischemia. After a thoughtful analysis that led them to conclude that the patient's symptoms were most likely embolic, they proceeded with occlusion of the highly stenotic proximal BA under full anticoagulation. The patient tolerated the procedure well, after an apparent transient disturbance of consciousness, and made an excellent recovery without further ischemic manifestations during a year of follow-up.

There can certainly be no argument with the successful outcome. The authors have taken a thoughtful approach to a difficult case. Often, we are reminded that we tend to treat these patients on the basis of the "accepted paradigm of the day," but these problems remain very risky, and no treatment to date has been shown to be reliably safe or effective in every patient. Hence, a thoughtful and individualized approach, as the authors used, remains paramount.

Still, there should be clear words of caution, because the successful management of one such case involves an element of clinical good fortune and does not ensure reliable safety and effectiveness in all similar cases. In this patient, the likelihood of an embolic mechanism was quite high, given the patchy multifocal ischemic lesions on magnetic resonance imaging and the variable symptomatology associated with transient ischemic attacks. However, such a distinction is not always this clear or reliable. And often, thromboembolism accompanies situations of significant hemodynamic vulnerability. Also, the presence of a patent posterior communicating artery and filling of the rostral BA on carotid injection do not guarantee tolerance to BA occlusion. A diagnostic balloon test occlusion with hypotensive challenge under full anticoagulation may enhance the predictability of safe tolerance to BA occlusion. It is not clear why the authors

did not use such a diagnostic test in this patient before deploying the coils for BA occlusion.

Also, even in patients who tolerate test occlusion with hypotensive challenge, some risk of delayed collateral failure remains, despite full anticoagulation. Elderly patients with advanced vascular risk factors may be most vulnerable to such complications. Although this fortunately did not happen in this case, the risk of delayed ischemia with deliberate parent vessel occlusion, especially in patients with ischemic occlusive cerebrovascular occlusion, remains unknown. Although attempted angioplasty and stenting would not have been without risk, it could be argued that the patient would have tolerated a stent if she in fact tolerated the deliberate therapeutic coiling. Hence, a trial of balloon test occlusion and an attempt at angioplasty and stenting may introduce added layers of potential safety in such clinical scenarios. Still, this patient clearly did well, and the authors provided wise and thoughtful management, daring to use an unconventional weapon to secure easier victory against a formidable clinical challenge.

Issam A. Awad
Evanston, Illinois

Horowitz et al. report a novel endovascular approach to the management of a patient with medically refractory BA stenosis. This 81-year-old woman presented with recurrent episodes of brainstem ischemia despite aggressive anticoagulation, and her angiographic evaluation demonstrated a 99% proximal BA stenosis with good collateral flow through the left posterior communicating artery. The authors concluded, on the basis of these angiographic findings, that the symptoms were the result of emboli arising from the stenosis as opposed to being hemodynamic in origin. They elected to treat the embolic source by completely occluding the BA at the stenosis. The patient experienced only a brief change in her level of consciousness without any other focal neurological symptoms and has had no further episodes for 1 year.

The concept of arterial sacrifice to treat difficult vascular lesions in the brain is by no means new, but this procedure has been used primarily for giant aneurysms or high-flow fistulae. Extracranial-to-intracranial bypass procedures, once commonly performed for intracranial stenosis, have fallen out of vogue, which has left anticoagulation as the mainstay of intracranial stenosis management. However, recent advances in endovascular devices have created the possibility of alternative treatment strategies. Microwires, catheters, and balloons can now be passed safely into the distal intracranial circulation to treat atherosclerotic lesions. Unfortunately, not all lesions respond equally well to endovascular therapy. Mori et al. (3) have demonstrated that the degree, length, and eccentricity of a stenotic lesion were directly correlated with the rate of both procedural complications and restenosis. The lesion presented in the present report is a Type B stenosis, and although the risk of treatment approximates 26%, endovascular revascularization is far from prohibited.

As a general practice, neurosurgeons strive to protect neural structures whenever possible. We use cranial base approaches and use stereotactic biopsy to minimize brain retraction and injury at surgery. The same should also be true with regard to endovascular surgery. Every effort should be made to preserve the patency of a vessel supplying the brain. True, the collateral circulation afforded by the circle of Willis makes arterial sacrifice of a carotid or vertebral artery possible, but the BA, because of its unpaired status and plethora of brainstem perforators, poses a problem when sacrifice is considered. In a study of 407 patients with posterior circulation infarctions, Glass et al. (1) reported that occlusion of the BA was responsible for the greatest risk of mortality and major disability in this population. The patient described in the present study was safely treated with elective occlusion of the BA at the stenosis; however, we think that this patient could have undergone endoluminal revascularization effectively with staged angioplasty and stenting. Whether this approach would have entailed more immediate risk than that associated with basilar occlusion is not known, but we think that the long-term benefit of revascularization exceeds that associated with occlusion of the artery and life-long anticoagulation with warfarin. With the staged approach, the lesion is opened initially with a gentle angioplasty, and then a stent and further angioplasty are performed 4 to 6 weeks later. This interval allows for endothelialization, which has a protective effect on the stent placement and secondary angioplasty (2). The authors have shown astute clinical judgment in recognizing the source of ischemia as embolic in nature and achieved a satisfactory result, but the reader is urged to understand that arterial sacrifice as a treatment for intracranial stenosis should not be considered a first-line endovascular option.

Jay U. Howington
L. Nelson Hopkins
Buffalo, New York

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