STENT-MEDIATED WEDGING OF A CALCIFIC EMBOLUS TO RECANALIZE AN OCCLUDED MIDDLE CEREBRAL ARTERY: TECHNICAL CASE REPORT

Clemens M. Schirmer, M.D.

Department of Neurosurgery, Cerebrovascular and Endovascular Division, Tufts Medical Center, and Tufts University School of Medicine, Boston, Massachusetts

David E. Thaler, M.D., Ph.D.

Department of Neurology, Tufts Medical Center, and Tufts University School of Medicine, Boston, Massachusetts

Adel M. Malek, M.D., Ph.D.

Department of Neurosurgery, Cerebrovascular and Endovascular Division, Tufts Medical Center, and Tufts University School of Medicine, Boston, Massachusetts

Reprint requests:
Adel M. Malek, M.D., Ph.D., Tufts Medical Center, 750 Washington Street, Proger 7, Boston, MA 02111.
Email: amalek@tuftsmedicalcenter.org

Received, March 14, 2007.
Accepted, April 17, 2008.

OBJECTIVE: Calcific cerebral emboli are rare yet pose a difficult therapeutic challenge because of their lack of response to thrombolytic therapy and their propensity for fragmentation.

CLINICAL PRESENTATION: A 75-year-old woman developed aphasia and hemiparesis after cardiac catheterization from a near-occlusive calcific embolus to the left middle cerebral artery origin. The calcific embolus was shown to be hemodynamically limiting by computed tomographic perfusion and diffusion-weighted magnetic resonance imaging.

INTERVENTION: The patient was treated medically for 6 days with anticoagulation, antiplatelet, and hypertensive therapies but continued to deteriorate. A decision was made to attempt retrieval of the calcific embolus. Conventional intracranial mechanical clot and coil retrieval systems were used initially but were unsuccessful and caused migration of the calcific embolus, leading to complete occlusion of the left middle cerebral artery origin. At this point, the decision was made to wedge the calcific embolus against the vessel wall. A balloon-mounted coronary stent was used to achieve this successfully, resulting in recanalization of the middle cerebral artery trunk and flow restoration.

CONCLUSION: Symptomatic calcific emboli, which are poorly responsive to pharmacological therapy and difficult to recover using conventional retrieval systems, may be treated by immobilization against the vessel wall using a balloon-mounted stent.

KEY WORDS: Acute stroke, Calcific aorta, Cardiac catheterization, Complication, Embolus

Neurosurgery 63:E180–E181, 2008 DOI: 10.1227/01.NEU.0000319637.09212.0A www.neurosurgery-online.com

Cerebral infarction caused by calcific embolus (CE) is a rare but increasingly recognized cause of acute stroke (8, 15, 23). Spontaneous calcific emboli are associated with calcified cardiac valves (16) and aortic arch atherosclerosis (3, 7) and have been reported as a complication after cardiac catheterization (23). The recognition of CE does little to help in treatment because it is not likely to respond to conventional thrombolytic therapy (4), and other therapeutic options have not been established. We present a case of symptomatic CE to the left middle cerebral artery (MCA) origin that occurred during cardiac catheterization and resulted in near-total occlusion. The patient was initially managed medically, but a more aggressive endovascular approach was undertaken after clinical worsening.

CASE REPORT

A 75-year-old woman with hypertension, hyperlipidemia, and coronary and peripheral vascular disease developed aphasia, right hemiplegia, and neglect after cardiac catheterization. She was anticoagulated with heparin and administered oral aspirin (325 mg) after a head computed tomographic (CT) scan and CT angiogram showed a calcium signal at the left MCA origin (*Fig. 1*, *A* and *B*). CT scanning confirmed a calcified aortic arch (*Fig. 1C*) as a possible source of the embolus. Despite medical therapy with aspirin and heparin, her symptoms worsened over the next 24 hours, coincident with either decreases in blood pressure or increases in head elevation. A CT perfusion scan confirmed increased transit times (*Fig. 1E*); serial magnetic resonance imaging scans demonstrated an increasing number of areas of restricted diffusion within the hypoperfused zone between Days 2

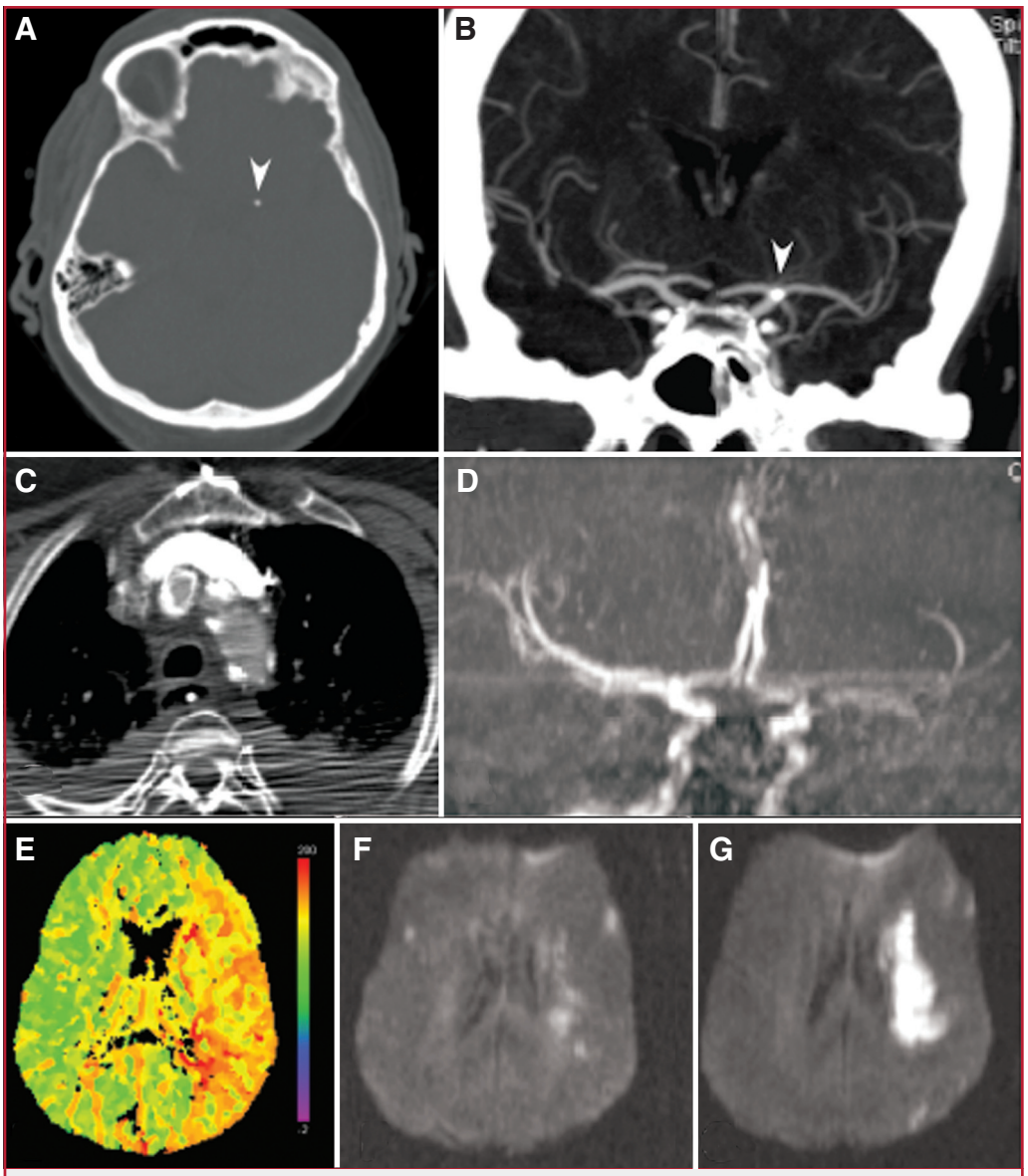


FIGURE 1. *Axial computed tomographic (CT) scan (**A**) and coronal maximum intensity projection CT angiogram (**B**) revealing the calcific embolus at the left internal carotid artery bifurcation measuring 2.5 to 3 mm* (arrowhead)*. Axial thoracic CT scan revealing heavy calcifications in the aortic arch. **C**, coronal magnetic resonance (MR) angiogram revealing decreased flow in the left middle cerebral artery (MCA) and its branches. **D**, CT perfusion confirmed delayed transit time to the left hemisphere. **E**, Diffusion-weighted MR imaging (**F**) showed ischemia in the deep white matter and repeat imaging 3 days later confirmed progression (**G**).*

(*Fig. 1F*) and 5 (*Fig. 1G*). After discussion with members of the family, who were resistant to endovascular therapy at first, the patient was taken to the endovascular suite on Day 6 according to the family's wishes, where digital subtraction angiography identified the occlusive CE (*Fig. 2A*) at the left MCA M1 segment origin. After intravenous anticoagulation with heparin to an activated clotting time of 250 seconds, an Alligator retrieval device (Chestnut Medical, Menlo Park, CA) (5) was used unsuccessfully in an attempt to grab or manipulate the embolus. Next, the Merci Retrieval System (Concentric Medical, Mountain View, CA) (21) was advanced in the M1 branch in an unsuccessful attempt to dislodge the embolus. At this point, flow in the left M1 was completely lost (*Fig. 2B*) as a result of distal wedging of the embolus. An exchange microwire was navigated past the CE into a distal superior M2 segment MCA branch and used to enable sequential angioplasty of the M1 origin with a 1.5 × 9-mm followed by a 2.0 × 9-mm Gateway intracranial angioplasty balloon (Boston Scientific, Natwick, MA) (*Fig. 2C*); angioplasty restored flow only transiently with reocclusion (*Fig. 2D*). A decision was made to use a coronary balloon-mounted stent to wedge the CE against the wall of the vessel and restore MCA flow. Clopidogrel (300 mg; Bristol Myers Squibb, New York, NY) was administered by nasogastric tube, and a 2.5 × 8-mm Taxus stent (Boston Scientific) was deployed successfully with M1 recanalization (*Fig. 2, E* and *F*); despite progressive overinflation to 2.75 mm, significant recoil near the left M1 origin across from the CE resulted in residual stenosis. Surveillance digital subtraction angiography performed after the balloon was withdrawn showed progressive thrombus formation on the stent with flow reduction. The glycoprotein IIb/IIIa inhibitor eptifibatide (Integrilin; Millennium Pharmaceuticals, Cambridge, MA) was administered intravenously as a 120-μg/kg bolus followed by 2 μg/kg/min with subsequent flow restoration (*Fig. 2G*). The patient remained stable afterward, with CT imaging showing the relationship of the deployed stent and the CE wedged between the proximal stent struts and the vessel wall (*Fig. 3A*). Magnetic resonance angiography revealed persistent flow in the left MCA branches (*Fig. 3B*) and stable MCA territory infarcts (*Fig. 3C*). The patient was discharged to rehabilitation 10 days later. At 3 months, the patient was ambulating with a walker and maintaining full oral nutrition, and had significant improvement in her aphasia and right hemiparesis. Follow-up CT angiographic images after 8 months and magnetic resonance imaging and magnetic resonance angiographic imaging after 1 year showed unchanged flow

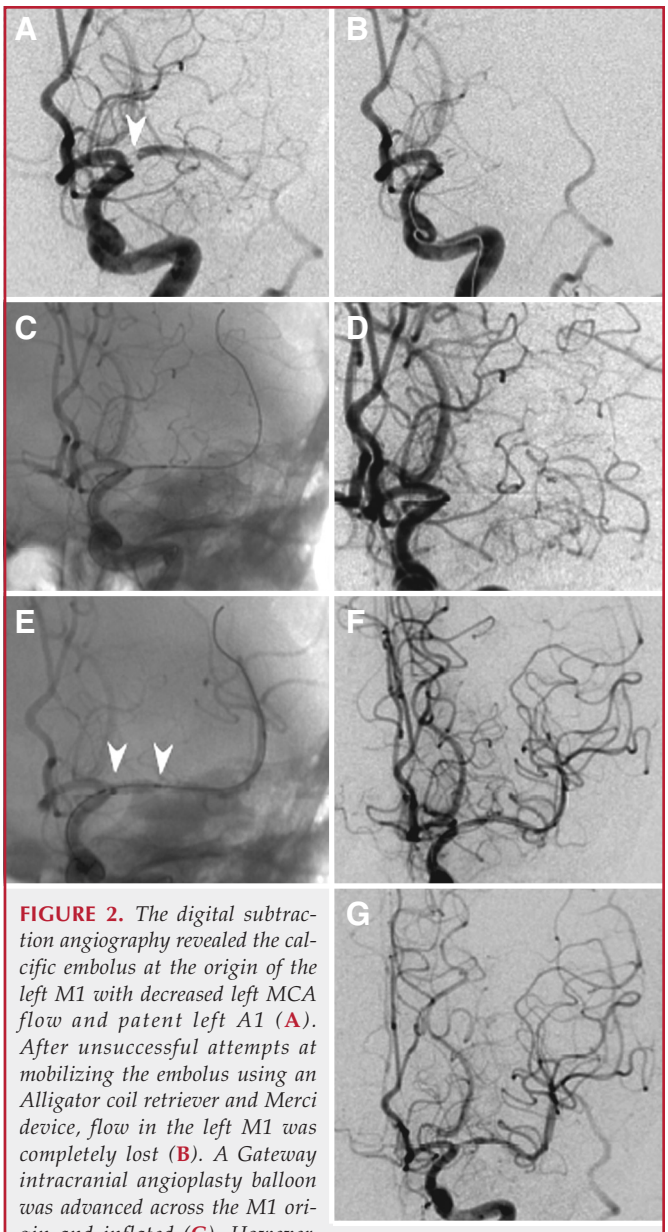


FIGURE 2. *The digital subtraction angiography revealed the calcific embolus at the origin of the left M1 with decreased left MCA flow and patent left A1 (**A**). After unsuccessful attempts at mobilizing the embolus using an Alligator coil retriever and Merci device, flow in the left M1 was completely lost (**B**). A Gateway intracranial angioplasty balloon was advanced across the M1 origin and inflated (**C**). However, flow could not be restored in a sustained fashion because of reocclusion. A Taxus stent was navigated across the stenotic origin (delimited by* arrowheads *(**D**). **E**) with resumption of left MCA flow (**F**). Final result after intravenous glycoprotein IIb/IIIa inhibitor for progressive occlusive stent thrombosis shows residual stenosis at the proximal end of the stent but unimpeded flow (**G**).*

in the left MCA with a patent stent and mild to moderate stenosis immediately distal to the stent, with unchanged appearance of the chronic infarcts in the left watershed area between the left anterior, middle, and posterior cerebral arterial territories (*Fig. 3, D* and *E*). The patient's hemiparesis and aphasia improved further, but she refused catheter-based angiographic follow-up.

DISCUSSION

The rate of stroke occurrence after cardiac catheterization is roughly 1 per 1000 procedures, and is slightly higher if a coronary intervention is performed (19, 24). Systemic (20) and intra-arterial (1) thrombolysis have been advocated as treatment options, although no therapy has been proven to be safe and effective. Thrombolysis would likely not have been effective in this case.

This report illustrates the absence of a device specifically suited for safe retrieval of a calcific embolus. The Merci Retriever relies on engaging thrombus, although it is conceivable that a calcified embolus could be engaged by the Merci device, the safe retrieval of the embolus will not always be possible. Snares and clasps such as those found in the Alligator devices are better suited for engaging slender, man-made devices such as coils and stents than a hard, round, friable CE. Although the use of a distal protection-type filter device was considered, none of the currently available devices are able to reach beyond the petrous segment of the internal carotid artery without risking iatrogenic injury.

Acute occlusions of the MCA were managed surgically before the advent for current endovascular techniques (13); surgery remains a viable alternative if thrombolysis and endovascular techniques are not successful (6). Thrombus originating in the heart has been found to be more amenable to embolectomy when compared with atherosclerotic plaque or debris such as that encountered in the case described here (12). Stenting has been described previously as a salvage method in the treatment of ischemic stroke after acute intracranial vessel occlusion (9, 17) and after unsuccessful recanalization with the Merci device (18); the rate of angiographic recanalization is high, between 79 and 90%, but long-term outcome is currently unclear.

This case also illustrates the pitfalls and dilemmas of aggressive endovascular therapy for a stable, partially occlusive CE. Unlike thrombus, which can be macerated into smaller fragments that are more effectively treated chemically using superselective arterial infusion, dislodging a stable CE can potentially result in a worsened angiographic outcome. This occurred in our case, when flow to the M1 trunk was completely lost after an attempt to retrieve the embolus and before angioplasty. Another requirement for management of cases such as the current one is a stiff stent with relatively small cell size and sufficient radial force to maintain patency and contain the embolus against the vessel wall. The recently approved self-expanding Wingspan intracranial stent (Boston Scientific) (2) offers increased navigability and flexibility over a coronary stent system; however, the same features that would be advantageous in most lesions might have proven disadvantageous in this case: the open-cell design would not necessarily ensure a smooth radial force application for such a focal and calcified lesion, and although the Wingspan stent offers significant radial force, it is by design not as rigid as a coronary stent.

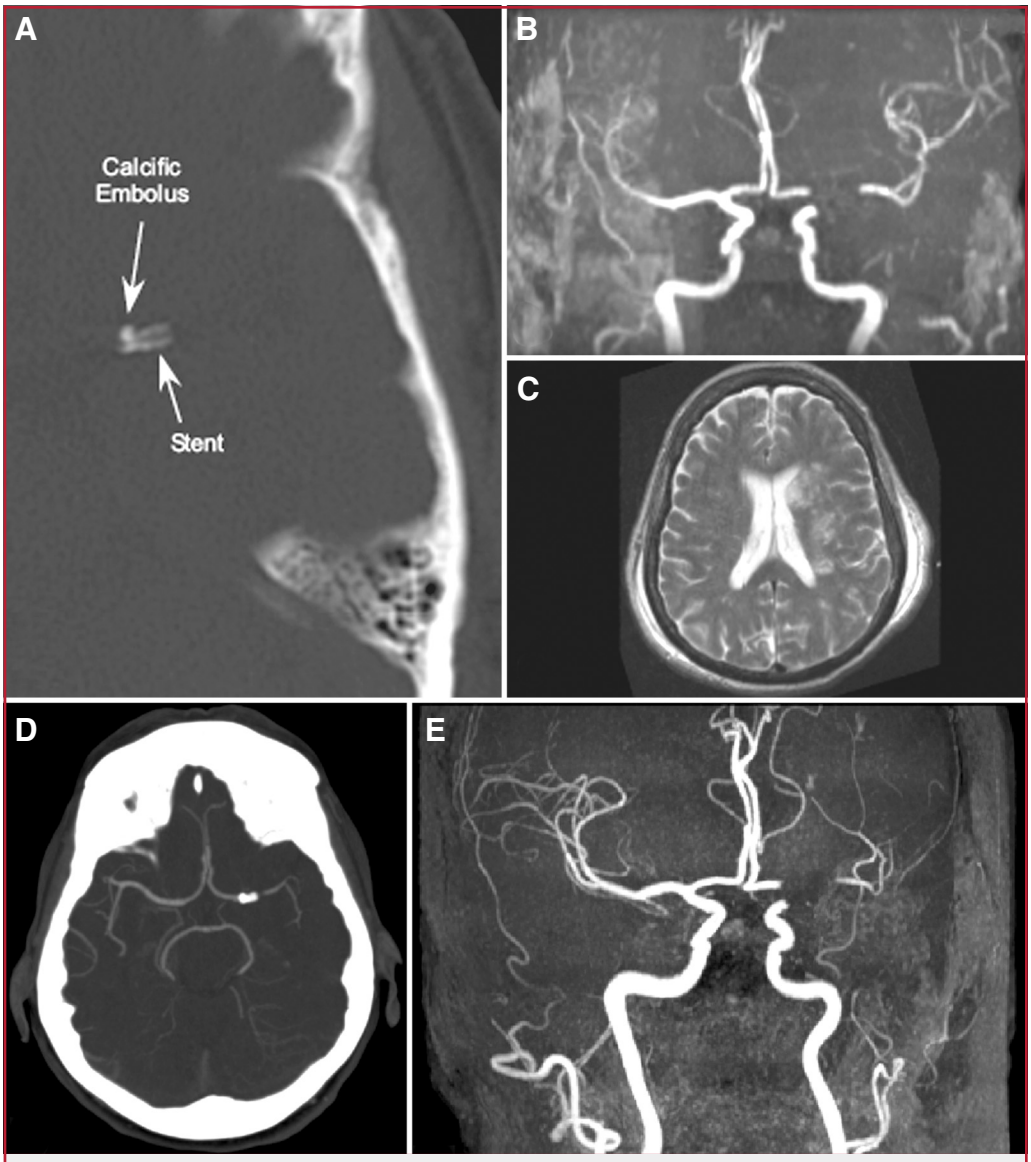


FIGURE 3. *Postprocedural CT* **A**, *revealing the position of the calcific embolus (CE) with respect to the deployed stent and demonstrating the CE impinging on the proximal stent. Magnetic resonance angiography at postprocedure Day 2 revealing persistent restoration of left MCA flow, with metal void artifact around the stent.* **B**, *T2-weighted axial MR imaging scan demonstrating persistent subcortical infarcts.* **C**, *follow-up CT angiogram after 8 months (***D***) and follow-up MR angiogram (***E***) after 1 year showing durable blood flow with patent stent and mild to moderate stenosis immediately distal to the stent.*

Angioplasty in the setting of a calcific lesion is a high-risk procedure. It is important to proceed with slow inflation of the angioplasty balloon and to select a relatively undersized stent to minimize the risk of vessel rupture, given the size of the CE. The danger of possible vessel dissection and rupture has to be weighed against the risk of the cerebrovascular territory at risk. Other risks include the disintegration of the CE into multiple smaller fragments that may then embolize distally or a hemorrhage after reperfusion of the infarcted area of the brain. Fortunately, these problems did not occur here; however, this eventuality must be discussed with the patient's health care proxy when considering intervention. In the current case, the importance of the threatened territory and the complete occlusion of the left M1 origin after migration of the CE warranted the aggressive attempt at recanalization.

Reperfusion hemorrhage has been described in the setting of carotid angioplasty and stenting, commonly associated with hyperperfusion syndrome (11, 14, 22). Hyperperfusion has also been described after MCA angioplasty (10). The role of anticoagulation and antiplatelet therapy as a risk factor for hemorrhage in the setting of hyperperfusion seems intuitive, but no definitive correlation has been demonstrated (11).

In our case, clopidogrel was administered before deployment of the stent, but to minimize the risk of reperfusion hemorrhage, a 300-mg dose was administered rather than the frequently used 600-mg loading dose. Optimal titration and timing of antiplatelet therapy is a known problem, and the timing of clopidogrel administration may have contributed to the acute formation of thrombus in the stent.

CONCLUSION

In summary, stent angioplasty to wedge and immobilize a CE against the vessel wall is a useful technique that can be used when flow restoration is paramount and other methods of treatment have been exhausted.

REFERENCES

1. Chan AW, Henderson MA: Immediate catheter-directed reperfusion for acute stroke occurring during diagnostic cardiac catheterization. **Catheter Cardiovasc Interv** 67:314–318, 2006.

2. Fiorella D, Levy EI, Turk AS, Albuquerque FC, Niemann DB, Aagaard-Kienitz B, Hanel RA, Woo H, Rasmussen PA, Hopkins LN, Masaryk TJ, McDougall CG: US Multicenter experience with the wingspan stent system for the treatment of intracranial atheromatous disease: Periprocedural results. **Stroke** 38:881–887, 2007.
3. Gearry RB, Sharr JP, Avery SF: Spontaneous calcific cerebral embolus. **Australas Radiol** 49:154–156, 2005.
4. Halloran JI, Bekavac I: Unsuccessful tissue plasminogen activator treatment of acute stroke caused by a calcific embolus. **J Neuroimaging** 14:385–387, 2004.
5. Henkes H, Lowens S, Preiss H, Reinartz J, Miloslavski E, Kühne D: A new device for endovascular coil retrieval from intracranial vessels: Alligator retrieval device. **AJNR Am J Neuroradiol** 27:327–329, 2006.
6. Horiuchi T, Nitta J, Sakai K, Tanaka Y, Hongo K: Emergency embolectomy for treatment of acute middle cerebral artery occlusion. **J Neurosurg** 106:257–262, 2007.
7. Kapila A, Hart R: Calcific cerebral emboli and aortic stenosis: Detection of computed tomography. **Stroke** 17:619–621, 1986.
8. Kavanagh EC, Fenton DM, Heran MK, Lapointe JS, Nugent RA, Graeb DA: Calcified cerebral emboli. **AJNR Am J Neuroradiol** 27:1996–1999, 2006.
9. Levy EI, Mehta R, Gupta R, Hanel RA, Chamczuk AJ, Fiorella D, Woo HH, Albuquerque FC, Jovin TG, Horowitz MB, Hopkins LN: Self-expanding stents for recanalization of acute cerebrovascular occlusions. **AJNR Am J Neuroradiol** 28:816–822, 2007.
10. Liu AY, Do HM, Albers GW, Lopez JR, Steinberg GK, Marks MP: Hyperperfusion syndrome with hemorrhage after angioplasty for middle cerebral artery stenosis. **AJNR Am J Neuroradiol** 22:1597–1601, 2001.
11. McCabe DJ, Brown MM, Clifton A: Fatal cerebral reperfusion hemorrhage after carotid stenting. **Stroke** 30:2483–2486, 1999.
12. Meyer FB: Emergency embolectomy for treatment of acute middle cerebral artery occlusion. **J Neurosurg** 106:255–256, 2007.
13. Meyer FB, Piepgras DG, Sundt TM Jr, Yanagihara T: Emergency embolectomy for acute occlusion of the middle cerebral artery. **J Neurosurg** 62:639–647, 1985.
14. Meyers PM, Higashida RT, Phatouros CC, Malek AM, Lempert TE, Dowd CF, Halbach VV: Cerebral hyperperfusion syndrome after percutaneous transluminal stenting of the craniocervical arteries. **Neurosurgery** 47:335–345, 2000.
15. Moustafa RR, Antoun NM, Coulden RA, Warburton EA, Baron JC: Stroke attributable to a calcific embolus from the brachiocephalic trunk. **Stroke** 37:e6–e8, 2006.
16. O'Donoghue ME, Dangond F, Burger AJ, Suojanen JN, Zarich S, Tarsy D: Spontaneous calcific embolization to the supraclinoid internal carotid artery from a regurgitant bicuspid aortic valve. **Neurology** 43:2715–2717, 1993.
17. Phatouros CC, Higashida RT, Malek AM, Smith WS, Mully TW, DeArmond SJ, Dowd CF, Halbach VV: Endovascular stenting of an acutely thrombosed basilar artery: Technical case report and review of the literature. **Neurosurgery** 44:667–673, 1999.
18. Sauvageau E, Samuelson RM, Levy EI, Jeziorski AM, Mehta RA, Hopkins LN: Middle cerebral artery stenting for acute ischemic stroke after unsuccessful Merci retrieval. **Neurosurgery** 60:701–706, 2007.
19. Segal AZ, Abernethy WB, Palacios IF, BeLue R, Rordorf G: Stroke as a complication of cardiac catheterization: Risk factors and clinical features. **Neurology** 56:975–977, 2001.
20. Serry R, Tsimikas S, Imbesi SG, Mahmud E: Treatment of ischemic stroke complicating cardiac catheterization with systemic thrombolytic therapy. **Catheter Cardiovasc Interv** 66:364–368, 2005.
21. Smith WS, Sung G, Starkman S, Saver JL, Kidwell CS, Gobin YP, Lutsep HL, Nesbit GM, Grobelny T, Rymer MM, Silverman IE, Higashida RT, Budzik RF, Marks MP; MERCI Trial Investigators: Safety and efficacy of mechanical embolectomy in acute ischemic stroke: Results of the MERCI trial. **Stroke** 36:1432–1438, 2005.
22. Sundt TM Jr, Sharbrough FW, Piepgras DG, Kearns TP, Messick JM Jr, O'Fallon WM: Correlation of cerebral blood flow and electroencephalographic changes during carotid endarterectomy: With results of surgery and hemodynamics of cerebral ischemia. **Mayo Clin Proc** 56:533–543, 1981.
23. Sylaja PN, Hill MD: Stroke due to calcific embolus following coronary angiography. **Neurology** 67:E16, 2006.
24. West R, Ellis G, Brooks N; Joint Audit Committee of the British Cardiac Society and Royal College of Physicians of London: Complications of diagnostic cardiac catheterisation: Results from a confidential inquiry into cardiac catheter complications. **Heart** 92:810–814, 2006.

COMMENTS

Schirmer et al. present a well-illustrated case of a calcific embolus with near internal carotid artery-T occlusion with a rationally described, stepwise escalation in therapy, leading to eventual use of a short balloon-mounted coronary stent within the M1 segment. As noted by the authors, stenting for embolic stroke has been described previously. However, the application of this technique to a calcific embolus resistant to mechanical embolectomy serves to highlight some of the limitations of existing methods for intra-arterial stroke salvage.

It should be kept in mind that balloon-mounted coronary stents are less flexible (and therefore less navigable) than self-expanding intracranial stents, such as the Wingspan (Boston Scientific, Natick, MA) or Enterprise (Cordis, Miami Lakes, FL) stents, and can be associated with high complication rates, at least in the posterior circulation (1). In the situation encountered by Schirmer et al., one worries about vessel perforation as balloon inflation pushes a calcific embolus against the vessel wall. The authors discuss undersizing the stent to minimize this risk, although balloon overinflation was used in their patient. In addition, although the authors chose a coronary stent because of its high outward radial force, its superiority in recanalizing a calcified embolus remains unknown relative to existing intracranial stents with lower radial force. Nevertheless, Schirmer et al. performed a difficult intervention with good results.

Babak S. Jahromi
L. Nelson Hopkins
Buffalo, New York

---

1. Fiorella D, Chow MM, Anderson M, Woo H, Rasmussen PA, Masaryk TJ: A 7-year experience with balloon-mounted coronary stents for the treatment of symptomatic vertebrobasilar intracranial atheromatous disease. **Neurosurgery** 61:236-243, 2007.

This is an interesting article presenting the case of a 75-year-old patient with a left middle cerebral artery calcific embolus and near complete occlusion of the M1 segment. After failure of medical therapy, Schirmer et al. proceeded to use a mechanical retrieval approach unsuccessfully, which resulted in complete occlusion of the M1 segment. They then decided to deploy a balloon-mounted coronary stent to tack the calcific embolus against the arterial wall. This was done successfully, and middle cerebral artery flow was restored. This case report confirms the fact that one cannot rely on only one approach when treating acute stroke interventionally. The pros and cons of all the potential tools have to be weighed carefully, and the specific features of the embolus in this case made the particular stent choice rational. Timely revascularization is emerging as one of the most important factors in improving outcome in strokes resulting from large vessel occlusion. The story of how we will evolve the approach to achieving this revascularization will be filled with exciting innovations and opportunities for research. One of my concerns with the use of stenting in acute stroke is the need for aggressive antiplatelet medications, which may increase hemorrhage rates. Drug-coated, retrievable, or perhaps biodissolvable stents may address this issue.

Ziad A. Hage
Bernard R. Bendok
Chicago, Illinois

Schirmer et al. describe the wedging of a calcified embolus against the wall of a middle cerebral artery by use of a balloon-mounted

stent. Novel stent technology appears to be evolving into an effective means of addressing acute intracranial thromboembolic events. This report adds to the growing body of literature that supports the use of this aggressive revascularization technique. Given the calcified nature of the embolus, this particular case was challenging, and the technical achievement of Schirmer et al. was excellent. Deployment of the stent effectively lodged the embolus against the wall of the middle cerebral artery. However, given the small nature of this vessel and the deployed stent, only long-term radiographic follow-up will determine the clinical efficacy of this technique. Nonetheless, both self-expanding and balloon-mounted stents remain important weapons within the endovascular armamentarium and for the management of both acute strokes and chronic intracranial atherosclerotic disease.

Felipe C. Albuquerque
Phoenix, Arizona