Intracranial Stents in SAH Cases

Intracranial stent placement is a sophisticated but challenging treatment option for subarachnoid hemorrhage as a result of aneurysm rupture.

BY DEMETRIUS LOPES, MD; ERWIN MANGUBAT, MD; KIFFON KEIGHER, APN; AND CHARLIE COGAN

Based on findings from the International Subarachnoid Aneurysm Trial (ISAT), coiling of ruptured cerebral aneurysms is associated with the lowest immediate morbidity and mortality rates compared to other treatment options.[1,2] Whenever anatomy permits, coiling is the preferred method for repair. Unfortunately, not all cerebral aneurysms are suitable for coiling, and the best treatment for aneurysms that cannot be coiled remains unclear. Adjunctive techniques such as surgical clipping, balloon remodeling,[3] use of two microcatheters,[4] and intracranial stents[5] can increase the likelihood of aneurysm thrombosis and parent vessel patency. The goal of this article is to describe our current practice using intracranial stents in appropriately selected patients with subarachnoid hemorrhage (SAH) as a result of aneurysm rupture.

POSSIBLE INDICATIONS FOR STENTING IN PATIENTS WITH SAH

At Rush University Medical Center (Chicago, IL), the use of intracranial stents and coils to treat patients with SAH from cerebral aneurysms has been extremely helpful in the following clinical situations:

- Fusiform or blister-like ruptured cerebral aneurysms (Figure 1);[6,7]
- Patients with SAH who require prolonged antiplatelet therapy, usually due to coronary disease;[8]
- Stent remodeling technique in wide-necked aneurysms (stent is retrieved after coiling; temporary stenting);[9]
- Bailout from coil stretching or coil migration;
- Staging ruptured aneurysm treatment by initially coiling the dome of the aneurysm in the acute phase, then before discharge placing a stent and further coiling as needed to completely occlude the aneurysm.

USEFUL ANTIPLATELET TIPS

When using intracranial stents in patients with recent SAH, antiplatelet therapy can be both friend and foe. Partial inhibition of the platelet population is required to maintain stent and parent-vessel patency, but the recent SAH can be aggravated by the dysfunctional platelets.

The most concerning complications associated with intracranial stenting involve ischemic and hemorrhagic strokes. The implantation of an intracranial stent without adequate antiplatelet inhibition will result in high rates of thromboembolic complications. Patients with SAH present a challenge because the usual dual-antiplatelet regimen recommended for unruptured aneurysms may be associated with an increase in hemorrhagic complications.[10,11] In our experience, platelet inhibition assessment, timing of platelet inhibition, intracranial computed tomography (CT) imaging, and careful postoperative management (team training and communication) are the most important aspects for safe use of antiplatelet therapy in cases of SAH.

Our center uses point of care assessment (VerifyNow Aspirin [ASA] Reaction and P2Y12 Assay [Accumetrics, San Diego, CA]) of platelet inhibition.[12] We have used ASA reaction unit (ARU) < 550 and P2Y12 reaction unit (PRU) > 250 or > 30% inhibition. This is very important in patients who are already taking antiplatelets. If the baseline

assessment shows platelet inhibition to ASA or clopidogrel, we have used intracranial stents without experiencing a significant increase in thromboembolic complications. If the patient has no significant platelet inhibition on baseline assessment and a stent was implanted, we prefer to use glycoprotein IIb/IIIa inhibitors. An intra-arterial loading dose of glycoprotein IIb/IIIa inhibitors is given after the aneurysm has been protected with a stent and coils.

Heparin administration during coiling is usually started after the first coil is in the aneurysm. Our goal is to achieve an activated clotting time of 250 seconds. Heparin administration is not reversed at the end of the case unless the head CT scan shows intracranial hemorrhage (ICH). We usually use heparin in the setting of SAH in prolonged and complex aneurysm repair cases. After the procedure, if the platelet inhibition was not therapeutic, we proceed with an oral loading dose of 325 mg ASA and 600 mg clopidogrel followed by daily 325 mg ASA and 75 mg clopidogrel for at least 3 months. Follow-up studies are performed in intervals to confirm that ASA and clopidogrel are working.

When the neck of the aneurysm is wide, coils can protrude through the neck into the parent vessel, causing occlusion. It is possible to place a temporary scaffold over the neck of the aneurysm, coil the aneurysm, and then remove the scaffold. In this way, antiplatelet therapy is not required in the perioperative period (Figure 2). In cases in which the temporary stenting technique (stent remodeling technique) is used, patients receive only heparin without additional antiplatelets.[9]

These recommendations for antiplatelet inhibition during SAH stenting have helped us to decrease our stroke complication rate. After reviewing all cases in which SAH is treated acutely (< 24 h) with stenting, we identified a few areas that need special attention. We will discuss the importance of focusing on the management of external ventricular drainage (EVD) (and any other open procedures), access site, and gastrointestinal hemorrhage (GIH).

EXTERNAL VENTRICULOSTOMY DRAINAGE

In some patients with SAH, blood causes obstruction of the normal cerebrospinal fluid pathways, at times resulting in hydrocephalus that requires EVD. Ventriculostomy requires a small burr hole be made in the calvaria (skull), followed by placement of a transcortical tube into the horn of the cerebral ventricle. This can be performed in the emergency department, intensive care unit, angiography suite, or operating room under sterile conditions.

We always consider EVD placement before starting the procedure for repair of a ruptured intracranial aneurysm. After ventriculostomy, a CT scan must be obtained before antiplatelet therapy administration. If an intracranial hemorrhage is seen on CT, no antiplatelet therapy is given.

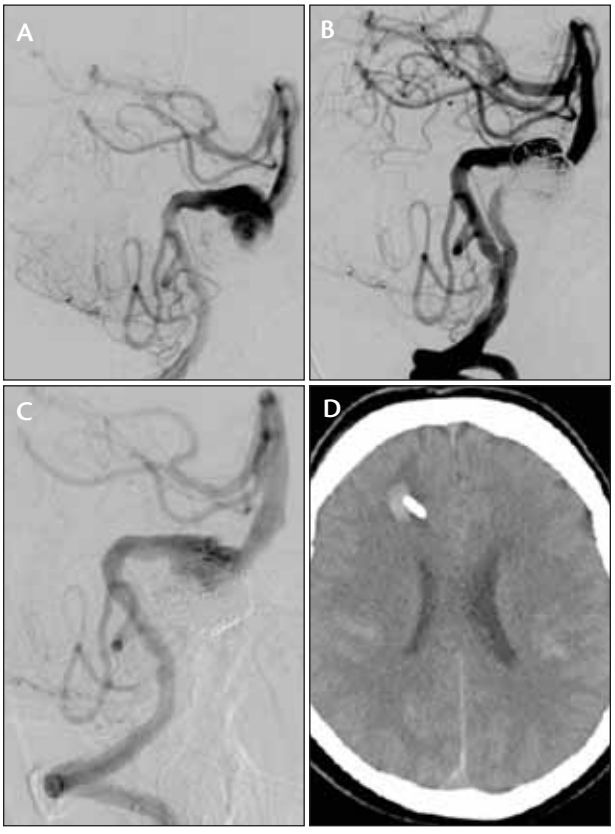

Figure 1. Before (A) and immediately after (B) stenting and coiling of ruptured aneurysm. Head CT showing EVD tract associated ICH (C). Six-month angiographic follow-up of stent and coil construct (D).

Intraoperative monitoring with electroencephalography, somatosensory, and motor evoked potentials are extremely useful. If hemorrhage is suspected during treatment, DynaCT (Siemens Healthcare, Malvern, PA) can be performed with the Siemens angiography unit without moving the patient from the angiography table.

Early diagnosis may help to avoid progression of ICH. One of the most important lessons for us has been to maintain communication with the critical care team on the daily management of the EVD. The EVD should not be manipulated or "flushed" in case of malfunction without discussing with the endovascular team. We have considered a platelet transfusion before EVD manipulation. Desmopressin can also be used to mitigate the effect of antiplatelet medications. This approach has been applied to other interventions such as placement of ventriculoperitoneal shunts, tracheostomy, and percutaneous endoscopic gastrostomy. If a head CT shows ICH associated with mass effect, we consider surgical decompression and single-antiplatelet coverage (minimal antiplatelet dose to achieve inhibition on VerifyNow ASA Reaction and P2Y12 Assay).

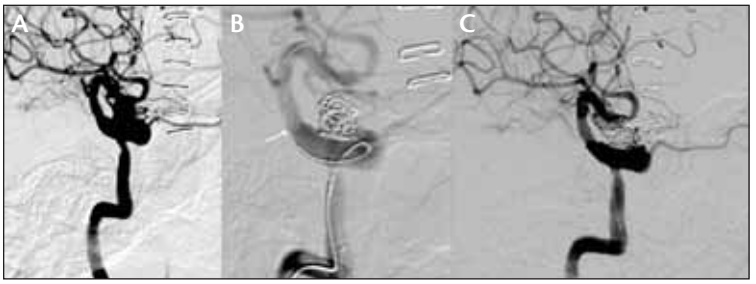

Figure 2. A case of a ruptured wide-necked aneurysm in which clipping was attempted but was unsuccessful. Preoperative angiogram (A). Stent partially deployed (arrow) after coiling (B). Stent retrieved with the coils occluding the aneurysm (C).

ACCESS SITE

We usually use a femoral 6-F arterial access. It is very important to obtain access in a single pass into a single wall of the femoral artery. After the intervention, we use a closure device for management of the access site. Every effort should be made to avoid a retroperitoneal hematoma because the patient will need to be kept on antiplatelet therapy.

GASTROINTESTINAL HEMORRHAGE

In our experience, GIH can be a significant complication in patients who undergo stent placement and coiling after SAH. The severity of the GIH may affect the ability to continue antiplatelet therapy during the hospital course. In theory, discontinuing antiplatelets before "healing of the stent" could result in thromboembolic complications. We use prophylactic H2 blockers to prevent GIH.

CONCLUSION

In our experience, implantation of an intracranial stent in the setting of SAH increases the complexity of patient management. As endovascular devices and techniques become more sophisticated, we have worked to increase our ability to offer these treatments to our patients when appropriate. The indications for the use of a stent and the management techniques described in this article have been helpful for us to be able to successfully treat challenging cases. The implantation of stents in ruptured aneurysms was necessary in only 4% of our SAH cases over the last 3 years. Our preference has always been to stage SAH cases that cannot safely be completely coil occluded and are not good open surgery candidates: first coiling to "protect" the dome of the ruptured aneurysm followed by oral loading of antiplatelets and stenting and coiling as needed before discharge.

Stenting for the treatment of unruptured wide-necked aneurysms has become integral to the practice of neurointervention. Intracranial stenting has allowed us to increase the number of cases that can be treated with minimally invasive techniques. The long-term results have been extremely positive with regard to concerns related to in-stent stenosis and complication rates. The knowledge and experience acquired with the use of intracranial stents for the treatment of unruptured aneurysms has been extremely helpful to the introduction and management of stents for the treatment of ruptured aneurysms. ■

Demetrius Lopes, MD, is an endovascular neurosurgeon with Rush University Medical Center in Chicago, Illinois. He has disclosed that he is a paid consultant to and receives grant/research funding from ev3 Inc. and Boston Scientific Corporation. Dr. Lopes may be reached at (312) 942-6644.

Erwin Mangubat, MD, is with Rush University Medical Center in Chicago, Illinois. He has disclosed that he holds no financial interest in any product or manufacturer mentioned herein.

Kiffon Keigher, APN, is with Rush University Medical Center in Chicago, Illinois. She has disclosed that she holds no financial interest in any product or manufacturer mentioned herein.

Charlie Cogan is with Rush University Medical Center in Chicago, Illinois. He has disclosed that he holds no financial interest in any product or manufacturer mentioned herein.

1. Molyneux A, Kerr R, Stratton I, et al. International Subarachnoid Aneurysm Trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: a randomised trial. Lancet. 2002;360:1267-1274.
2. Molyneux AJ, Kerr RS, Yu LM, et al., International subarachnoid aneurysm trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: a randomised comparison of effects on survival, dependency, seizures, rebleeding, subgroups, and aneurysm occlusion. Lancet. 2005;366: 809-817.
3. Moret J, Cognard C, Weill A, et al. [Reconstruction technique in the treatment of wide-neck intracranial aneurysms. Long-term angiographic and clinical results. Apropos of 56 cases]. J Neuroradiol. 1997;24:30-44.
4. Baxter BW, Rosso D, Lownie SP. Double microcatheter technique for detachable coil treatment of large, wide-necked intracranial aneurysms. AJNR Am J Neuroradiol. 1998;19: 1176-1178.
5. Wakhloo AK, Lanzino G, Lieber BB, et al. Stents for intracranial aneurysms: the beginning of a new endovascular era? Neurosurgery. 1998;43:377-379.
6. Fiorella D, Albuquerque FC, Deshmukh VR, et al. Endovascular reconstruction with the Neuroform stent as monotherapy for the treatment of uncoilable intradural pseudoaneurysms. Neurosurgery. 2006;59:291-300; discussion, 291-300.
7. Wakhloo AK, Mandell J, Gounis MJ, et al. Stent-assisted reconstructive endovascular repair of cranial fusiform atherosclerotic and dissecting aneurysms: long-term clinical and angiographic follow-up. Stroke. 2008;39: 3288-3296.
8. Almeda FQ, Lopes DK, Eybel CE. Management of a patient with ST segment elevation myocardial infarction immediately after successful coiling of a basilar tip aneurysm for a subarachnoid hemorrhage. J Invasive Cardiol. 2004;16:166-167; discussion 167-168.
9. Lopes DK, Wells K. Stent remodeling technique for coiling of ruptured wide-neck cerebral aneurysms: case report. Neurosurgery. 2009;65:E1007-E1008; discussion E1008.
10. Van den Bergh WM, Kerr RS, Algra A, et al. Effect of antiplatelet therapy for endovascular coiling in aneurysmal subarachnoid hemorrhage. Stroke. 2009;40:1969-1972.
11. Tahtinen OI, Vanninen RL, Manninen HI, et al. Wide-necked intracranial aneurysms: treatment with stent-assisted coil embolization during acute (<72 hours) subarachnoid hemorrhage—experience in 61 consecutive patients. Radiology. 2009;253:199-208.
12. Prabhakaran S, Wells KR, Lee VH, et al. Prevalence and risk factors for aspirin and clopidogrel resistance in cerebrovascular stenting. AJNR Am J Neuroradiol. 2008;29:281-285.