Editorial

Hemorrhagic complications associated with stent-assisted coil embolization

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There is little doubt that the development and implementation of intracranial stents such as the Neuroform (Boston Scientific) and more recently the Enterprise stent (Cordis Neurovascular) in the US has enabled the successful endovascular treatment of intracranial aneurysms previously not amenable to endovascular procedures. Stent-assisted coil embolization is an important technique in the armamentarium of most neurointerventionalists, although the proportion of cases in which it is used varies considerably among practitioners. Like any other technique, stent-assisted coil embolization is associated with risks and limitations, some of which have been incompletely defined. In this report, the highly experienced group from Emory University raises concerns about hemorrhagic complications associated with stent-assisted coil embolization and dual antiplatelet therapy in patients who also require periprocedural cerebrospinal fluid (CSF) diversion procedures.

Tumialán and colleagues report a series of 37 patients who underwent stent-assisted coil embolization of either ruptured or unruptured aneurysms over a 2-year period. All patients were given a loading dose of 325 mg aspirin and 375 mg clopidogrel prior to sheath placement and diagnostic angiography; they received heparin for anticoagulation during the procedure and were maintained on 325 mg aspirin and 75 mg clopidogrel daily. The authors emphasized a subset of 7 patients who required periprocedural external ventricular drainage or permanent CSF diversion for the management of hydrocephalus. Six of these 7 patients underwent treatment of acutely ruptured aneurysms. Five patients underwent placement of an external ventricular drain (EVD) prior to the procedure, whereas in 2 an EVD was placed following the procedure. Six of these 7 patients experienced hemorrhagic complications, including 3 with intraventricular hemorrhage at the time of initial EVD placement, 1 with rehemorrhage from the aneurysm during the endovascular procedure, 1 with rehemorrhage from an incompletely coil-occluded aneurysm 2 weeks after the procedure, and 1 with a subdural hematoma (SDH) after ventriculoperitoneal (VP) shunt placement. Three of these complications were fatal. Only 1 of the 7 patients in this group did not experience a hemorrhagic complication. Although the authors indicate that they will report their overall Neuroform stent placement experience in a later submission, they state that they had an overall 16.2% hemorrhagic complication rate but that there was an 85% hemorrhagic complication rate in patients who required both stent-assisted coil embolization and placement of an EVD.

The authors have provided a timely contribution that serves to highlight an increasingly important management dilemma encountered by those caring for patients with acutely ruptured, wide-necked intracranial aneurysms. There are multiple treatment strategies that may be used under these circumstances. One strategy, as the authors describe, includes a staged endovascular treatment paradigm in which the aneurysm dome is secured using incomplete coil occlusion, followed by delayed definitive stent-assisted coil embolization after dealing with the issues of CSF diversion. This strategy is not always feasible (for instance, some aneurysms will not accept a single coil in a stable configuration without significant coil herniation), and the degree of protection afforded against rerupture from an intentionally incompletely coil-occluded aneurysm is not clear. We have generally favored a single-stage treatment strategy (clip ligation or coil embolization).

Another strategy involves placement of an EVD prior to the procedure and initiation of dual antiplatelet therapy. This may decrease the immediate risk of insertion-related hemorrhage; however, under normal circumstances, EVD manipulation and/or permanent shunt placement is necessary over the next 7–14 days. The liberal application of this strategy may also expose patients to the risks associated with a procedure that they may not have ultimately needed. For example, radiographic evidence of hydrocephalus is quite common in the early stage of subarachnoid hemorrhage (SAH). However, many patients will compensate and remain asymptomatic or minimally symptomatic and do not require urgent placement of an EVD. The potential risk of delayed EVD insertion after a patient receives dual antiplatelet therapy must now be weighed against the risks of immediate (and perhaps somewhat prophylactic) insertion prior to initiation of antiplatelet therapy. As we are all aware, the risks associated with EVD insertion are not zero, and hemorrhagic complications can occur in patients who have not received anticoagulants or antiplatelet agents.
An alternative strategy for a highly select group of patients may involve placement of a lumbar drain for temporary CSF diversion or a lumbarperitoneal (LP) shunt for permanent CSF diversion. Many patients are not eligible for lumbar drain insertion; these include high-grade patients with raised intracranial pressure and those with obstructive hydrocephalus. With lumbar drainage or LP shunt placement there is still a risk of SDH formation, and LP shunts are notorious for frequent malfunctions. Although the risk of intracranial hemorrhage may be lessened, spinal hematomas may also be problematic.

The authors have thoughtfully highlighted the periprocedural considerations applicable to stent-assisted coil embolization for acutely ruptured wide-necked aneurysms. In our practice, we have generally delayed initiation of dual antiplatelet therapy until after the diagnostic angiogram is performed and a definitive decision has been made to proceed with stent-assisted coil embolization, unless it is absolutely clear based on noninvasive imaging that stent-assisted coil embolization will be the safest and most effective treatment. Platelet transfusions can be given to patients who have received antiplatelet therapy in preparation for stent-assisted coil insertion but who ultimately undergo surgical clip ligation; however, our strategy facilitates an expeditious transition from the endovascular suite to the operating room and potentially avoids unnecessary exposure to platelet inhibition in a patient with an unsecured, ruptured aneurysm. Short-term use of heparinization during and after securing of the aneurysm endovascularly, followed by administration of antiplatelet agents, is another strategy that we have used with success.

Another option for wide-necked aneurysms is balloon remodeling, which obviates the need for prolonged dual antiplatelet therapy. Nevertheless, balloon remodeling is not possible for all wide-necked aneurysms, and it carries its own set of limitations and risks. The Emory group has a vast experience with this technique, and we presume that the aneurysms treated with stent-assisted coil embolization were not appropriate candidates for balloon remodeling.

Stent-assisted coil embolization has clearly added another important option for the treatment of acutely ruptured wide-necked intracranial aneurysms. Nevertheless, its long-term efficacy and consequences have not been defined. In this honest appraisal of their own results, the authors have highlighted the potential increased risk of hemorrhagic complications in patients undergoing stent-assisted coil embolization who also require CSF diversion procedures, and they emphasize the importance of considering this risk in formulating a treatment plan. We have also noted hemorrhagic complications in such patients and have always maintained a highly selective approach to stent-assisted coil embolization for acutely ruptured, wide-necked aneurysms.

A very important message contained in this report is that treatment of intracranial aneurysms requires a multidisciplinary approach individualized to a given patient. The treatment plan formulated for each patient should be, in the estimation of the treating team, the plan associated with the highest degree of efficacy and safety for that particular patient after taking all data into consideration. We look forward to receiving further information from the Emory group and from others that will help to define the best way to use stent-assisted coil embolization as a treatment option for acutely ruptured wide-necked aneurysms.

RESPONSE: We are grateful for the thoughtful appraisal of our institution’s experience with stent-assisted coil embolization provided by Dumont and colleagues. As mentioned in their editorial, the introduction of the Neuroform stent has vastly increased the neurointerventionalist’s capacity to perform successful treatment of wide-necked aneurysms, which were previously outside the realm of endovascular therapy. This technology is of unquestionable benefit in the treatment of a patient with an unruptured aneurysm. In the context of SAH, however, stent-assisted coil embolization has the capacity to become the proverbial double-edged sword. The liability edge of this sword is the inherent thrombogenic nature of the nitinol alloy stent. In a patient with an unruptured aneurysm, the requirement to neutralize platelet function is of little consequence. Conversely, in the context of SAH, this requirement may be catastrophic. Whereas an acutely ruptured wide-necked aneurysm may be successfully embolized with this technology, the requirement for prolonged dual antiplatelet therapy creates a treacherous postprocedural course for the neurosurgeon to navigate. In particular, the need for EVDs and VP shunts under these conditions has been of great concern at our institution. Over the past several years, we have observed hemorrhagic complications in patients with SAH who underwent stent-assisted coil embolization and required either temporary or permanent CSF diversion. The absence of reports of these complications in the literature prompted a focused retrospective review of this particular subset of patients and ultimately yielded the case series presented here.

As Dumont and colleagues astutely point out, several management strategies for using stent-assisted coil embolization in patients with ruptured aneurysms still require further study. Other investigators have acknowledged the theoretical risk of hemorrhage in these patients and have recommended placement of the EVD prior to the institution of antiplatelet therapy. Although this may address the immediate concern of hemorrhage from the insertion of the EVD, the requirement for prolonged antiplatelet therapy leaves the patient at risk for hemorrhagic complications should he or she require subsequent manipulation, replacement, or permanent CSF diversion. First, we agree with Dumont and colleagues that the implementation of this strategy may not be indicated in all cases and that it exposes the patient to the risk of bleeding and infection. Second, the putative safeguard of placing a ventricular catheter prior to initiation of antiplatelet therapy was unfortunately not realized in our experience. Five of the 7 patients in our series had preprocedural EVD placement, but 4 of these still suffered hemorrhagic events from subsequent replacement of EVDs or VP shunt placement. Furthermore, although the reversal of antiplatelet therapy may minimize the risk of hemorrhagic complications for a procedure, such a reversal may in theory lead to thromboembolic complications. The risk of such a strategy has yet to be appropriately stratified. Finally, the role of lumbar drains in lower-grade patients as discussed by Dumont and colleagues needs to be further explored.