CASE REPORT

Salvage of distal non-target coil embolization with stent placement and intravenous eptifibatide in a ruptured, unsecured, atypical aneurysm

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SUMMARY

Introduction Small aneurysms may be challenging to embolize. In cases of subarachnoid hemorrhage (SAH) where treatment is delayed, physicians may have to balance the risks of certain required therapies (antiplatelet agents) with the risk of rupture. We describe a case of a technically challenging anterior cerebral artery aneurysm requiring eptifibatide infusion prior to definitive aneurysm treatment.

Case report A 57-year-old woman with SAH, underwent coil embolization of a small fenestrated A1–A2 junction aneurysm. The procedure was complicated by downstream coil migration which was then treated with Enterprise stent placement in the pericallosal artery. This required subsequent infusion of a glycoprotein IIb/IIIa inhibitor until the aneurysm could be repaired surgically.

Conclusions Revascularization with a stent in a distal cerebral vessel may salvage inadvertent coil migration. Although it is undesirable to administer antiplatelet agents to patients with SAH, in these circumstances short acting agents may be used.

BACKGROUND

Aneurysm repair in subarachnoid hemorrhage (SAH) may be delayed secondary to patient instability or lesion complexity. In such instances, extenuating circumstances requiring antiplatelet agents present a difficult dilemma. We describe a case of SAH due to a technically challenging aneurysm. Initial attempted treatment complication required stent placement and the need for antiplatelet agents, before definitive microvascular surgery could be performed. The interventional techniques used to salvage the dislodged coil and pharmacological management provide an example to others in similar circumstances.

CASE PRESENTATION

A 57-year-old women presented to another hospital 1 day previous to presentation at our hospital with confusion, headache, right hemiparesis (Hunt and Hess grade 3), and cranial CT notable for SAH and hydrocephalus (figure 1). She underwent ventriculostomy and angiography on SAH day 1.

TREATMENT

Left internal carotid artery angiography revealed an anterior cerebral artery (ACA) complex, with an accessory fenestrated ACA filling across the anterior communicating (ACOM) artery from the left side. A 1.6–1.8 mm aneurysm was noted (figure 2) from this ACOM–A2 junction. Because of the patient’s poor clinical condition with a ruptured aneurysm, endovascular embolization was preferred over open surgical intervention. Although the morphology of the aneurysm was less favorable for unassisted coiling, we were reluctant a priori to place a stent in the setting of acute SAH. Balloon supported coiling was considered but access to the left ACA proved difficult due to the sharp curvature of the vessel origin. Ultimately, this was achieved using an Excelsior SL-10 microcatheter and Synchro 0.010 inch microwire (Target Therapeutics, Fremont, California, USA). Given the difficulty of navigating the treatment catheter, we did not attempt placement of the balloon catheter or a stent delivery catheter. Of note, microcatheter placement within the aneurysm led to flow arrest here, so subsequent coil embolization was guided by prior guide catheter angiography. A 1.5 mm×1 cm Cerecyte coil (Codman Neurovascular, Raynham, Massachusetts, USA) was advanced through the microcatheter and detached in a standard fashion.

On microcatheter removal, the detached coil migrated distally through the ACA system, lodging...
in the callosomarginal artery. Attempts to retrieve the coil using a 2 mm Alligator Retrieval Device (Chestnut Medical, Menlo Park, California, USA) resulted in its migration to the pericallosal artery (figure 3A). Thrombosis from the pericallosal occurred down through the proximal A2. It was felt that further attempts at retrieval might result in loss of the coil more proximally in the anterior circulation. Thus we adopted a strategy to revascularize the artery using an Enterprise Vascular Reconstruction Device (Codman Neurovascular). The vessel was selected past the coil with the SL 10 catheter, which was then exchanged for a Prowler Plus microcatheter (Codman Neurovascular). Next, a 4.5×14 mm Enterprise stent was advanced through the microcatheter and unsheathed (figure 3B). Eptifibatide, 135 μg/kg bolus followed by 0.5 μg/kg/min infusion intravenously, and heparin 1000 U intravenously, were administered, which recanalized this segment (figure 3C). Intra-arterial abciximab, 5 mg/5 ml volume, was administered in aliquots for a total of 10 mg into the proximal A2, which further improved flow. She was maintained on eptifibatide until surgical intervention could be performed 2 days later.

OUTCOME AND FOLLOW-UP
Postoperative angiography (SAH day 5) showed no residual aneurysm, and an unchanged patent appearance of the ACA. During her postoperative course, as it was anticipated that she would require ventriculoperitoneal shunt placement, no antiplatelet agents were begun. A ventriculoperitoneal shunt was placed on SAH day 20. The patient was subsequently transferred to a rehabilitation facility where antiplatelet agents were restarted. After 4 months of follow-up she was in subacute rehabilitation, ambulating independently, with normal language, and 4/5 strength in the upper and lower extremities (modified Rankin Scale score 2). The patient declined follow-up angiography when seen at follow-up at this time but brain MR angiography suggested patency of the stented vessel.

DISCUSSION
This case highlights the difficulty of treating very small ruptured aneurysms as well as the clinical scenario of managing SAH patients with antiplatelet agents. Key points of the interventional aspect include attempts at coil embolization in cases of aneurysm flow arrest and management of distal coil dislodge-ment. We chose to undersize the coil to avoid intraprocedural rupture. However, a larger coil (1.5 mm×2 cm) may have had greater packing density within the aneurysm and not have migrated out of the sac. In addition, with flow arrest, a potential useful strategy prior to coil detachment might be to retract the microcatheter along the pusher wire to permit control angiography. Distal intracranial stent placement, which we used as a salvage technique, has been described in cases of distal posterior cerebral artery aneurysms, with a Neuroform stent (Boston/Stryker, Kalamazoo, Michigan, USA).1 Although the Enterprise stent is indicated for vessels of a minimum diameter of 2 mm, its wire based, rather than catheter based, delivery system makes this more navigable in the distal intracranial vasculature.

From a clinical standpoint, the major dilemma was use of antiplatelet agents despite having an unsecured ruptured aneurysm. We felt that in the setting of already having suffered SAH causing focal deficit, a further ACA stroke would be poorly tolerated. Rebleeding occurs in 6–8% of patients with SAH, and is a major cause of poor outcome. Most events occur within the first 24 h.2–4 The risk remains high for the first 2 weeks, then declines daily after the initial 24 h,5 peaking again at about 2–4 weeks after the event.6 7 Therefore, the time period during which she was exposed to eptifibatide was a relatively lower risk period. Rebleeding risk also increases with aneurysm size.8 Accordingly, this strategy may have been deleterious in a patient with a larger aneurysm. Additionally, we chose this very short acting9 glycoprotein IIb/IIIa inhibitor which could be immediately discontinued if necessary. This is commonly utilized to bridge patients with myocardial infarction to coronary bypass
surgery. We also elected to use a dose studied in the setting of acute ischemic stroke, much lower than that typically used for myocardial infarction patients.9 It is also possible that inherent biological mechanisms in some patients may minimize platelet aggregation around and within foreign bodies. This is unpredictable in the clinical setting, so caution in such cases, prior to treatment, should be exercised.

Another justifiable strategy may have been to perform stent assisted coiling in the ACOM segment after the initial attempt, which would have lent more ease to administration of intravenous eptifibatide once the aneurysm was secure. However, although we were successful in placing a stent in the pericallosal artery which is more distal and smaller in caliber than the aneurysm parent vessel, the angulation of the latter was less favorable to navigation of stiff catheters, and we felt we could not successfully catheterize the parent vessel with a stent delivery system. We also wished to minimize further potential for treatment related injury at this juncture.

Our reasons for not choosing surgery as the initial mode of repair relate to data favoring endovascular therapy as a less treatment related injury at this juncture.


REFERENCES

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