Rescue Stenting in Endovascular Treatment of Acutely Ruptured Cerebral Aneurysms

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Summary

Thromboembolic events and major artery occlusion following cerebral aneurysm coiling may lead to serious complications and even death if not treated. The use of an intracranial stent in the setting of subarachnoid hemorrhage (SAH) is risky due to the need for antiplatelet therapy. However in some conditions it could be an effective solution for this major problem. This study describes a revascularization technique using a Solitaire stent for treatment of anterior cerebral artery (ACA) occlusion following coiling of anterior communicating artery (Acom) aneurysms. Three cases of ruptured Acom aneurysms treated during the course of SAH underwent unplanned deployment of an intracranial stent. Complete occlusion of the ACA at the origin of the A2 segment developed during or shortly after coiling. Emergent CT brain scan was done in two cases to exclude rebleeding. Follow-up CT or MRI scans were performed 24 hours after stenting. Technical success was achieved in all cases. Complete revascularization of the Acom was achieved post stent deployment (TIMI grade 3). Time from onset of symptoms to full revascularization in the three cases was 35 minutes, one hour 50 minutes and two hours 40 minutes respectively. No intracranial bleeding occurred in any case following the procedure. No neurological changes occurred in case 1; mild neurological and radiological changes occurred in cases 2 and 3. Deployment of an intracranial stent achieved complete revascularization of the occluded Acom. Its use in a context of SAH is relatively risky but the technique resulted in a significant improvement of symptoms following flow restoration and

probably helped prevent symptoms worsening, major disability or even death. A study on a larger patient sample with long-term follow-up will be of value.

Introduction

Thromboembolic complications following endovascular treatment of cerebral aneurysms are the most common complications occurring during or shortly after aneurysm embolization using coils^{1,2}. The thrombus may develop at the coil surface, from the coil mass exerting a stenotic effect on the arterial lumen, or from the catheter and microwire despite adequate heparinization and achieving target ACT levels.

This study reports on three cases where a Solitaire stent (ev3 Neurovascular, Irvine, CA, USA) was used to treat anterior cerebral artery (ACA) occlusion following coiling of an anterior communicating (Acom) aneurysm. Stent deployment allowed complete revascularization of the occluded artery. The objective of this study is to describe the early impact of stent deployment following ACA occlusion by coil loops. The clinical presentations and endovascular technique are described.

Case 1

A 42-year-old teacher was referred to our institution from a rural area in Egypt 15 days after subarachnoid hemorrhage (SAH). Upon admission she was slightly confused with behavioral changes. She complained of bilateral

Figure 1 Cerebral angiography shows a wide-neck Acom aneurysm.

Figure 2 Post coiling cerebral angiography shows no parent artery compromise.

paraparesis (grade 2 motor power in both lower limbs). CT scan revealed bifrontal infarction adjacent to the interhemispheric fissure.

The endovascular procedure was done under general anesthesia with systemic heparinization keeping ACT between 250-300 s. The angiogram revealed an Acom aneurysm 5 mm in diameter $\times$ 4 mm in length with a neck size of 4 mm (Figure 1). The procedure was done under protection of a remodeling balloon but a coil loop protruded into the left A2 segment leading to thrombus propagation and occlusion of the left A2 segment. Intra-arterial injection of 200 μ g of Tirofiban, a glycoprotein (GP) IIb/IIIa receptor blocker, through the microcatheter over five minutes did not succeed in parent artery reopening. The patient was loaded with 300 mg clopidogrel and 500 mg ASA. Finally a Solitaire stent (ev3 Neurovascular, Irvine, CA, USA) was successfully deployed allowing TIMI grade 3 revascularization (Figure 2).

The patient recovered with no changes in her neurological state. CT scan done 24 hours later revealed no newly developed lesions. Two, six and 18 month control angiography showed significant radiological improvement with no filling of the sac or the neck (Raymond 1 scale)¹. The patient regained full motor power in both lower limbs over three months and returned to work.

Case 2

A 54-year-old man presented with SAH grade I Hunt and Hess scoring. Upon admission he was fully conscious, oriented complain-

ing of mild to moderate headache. His blood pressure was 180/100 mm HG he was kept on labetalol IV to keep his systolic blood pressure below 160 mm HG. CT revealed Fischer II SAH. CTA showed a bilobed Acom aneurysm.

Under general anesthesia, catheterization of both ICA using an Envoy 6F guiding catheter (Cordis, Miami, FL, USA) revealed an agenetic RT A1 segment with a trilobed Acom aneurysm. The aneurysm was fully packed (Raymond 1 scale). Mild stenosis exerted from the coil mass was noted at the left A2-A1 junction. The ACT was kept between 250-280 s throughout the procedure. As prophylaxis against further parent artery compromise, 500 mg IV ASA was given then systemic heparinization at 1000IU/h was maintained for 20 hours targeting a PTT three times the baseline value.

The patient recovered with no change in his neurological state. Twenty-four hours later and four hours following heparin discontinuation he became confused, aphasic with dense RT hemiplegia. Emergent CT scan was done revealing no rebleeding. He was loaded immediately with 300 mg of clopidogrel bisulfate and 500 mg of ASA IV. The patient was urgently transferred to the angiosuite. Under general anesthesia the angiogram revealed complete occlusion of the left ACA (Figure 3). Treatment consisted of 0.25 mg/Kg Abciximab (ReoPro; Centocor B.V./Eli Lilly, Indianapolis, IN, USA) injected over five minutes through a Rebar 24 microcatheter (ev3 Neurovascular, Irvine, CA, US) at the left A1 segment. This did not allow reopening of the occluded artery. A Solitaire 4 $\times$ 20 mm stent was deployed between A1 and A2 segment allowing

Figure 3 Cerebral angiography shows the occluded left ACA.

Figure 4 Post-stenting angiography shows the revascularized ACA with in-stent thrombus.

TIMI grade 3 revascularization (Figure 2). One hour 52 minutes from onset of symptoms was needed to regain full revascularization of the ACA. The patient recovered with Grade 3 motor power in the upper limb and grade 2 motor power in the lower limb. Following anesthesia recovery he regained full consciousness and recovered from the aphasia. Twenty-four hours later his clinical condition improved with full recovery of motor power at the upper limb and motor power improvement to grade 4 at the lower limb. CT scan done 24 hours post stenting revealed an infarcted area in the left frontal lobe (Figure 2). Before discharge he was able to walk without support. He is scheduled for a control angiogram at six months.

Case 3

A 57-year-old man presented with SAH grade II Hunt and Hess scale. He was referred to our institute at day six after SAH. Upon admission he was conscious, oriented with no motor deficit. CT brain scan revealed Fischer II SAH. Under general anesthesia, bilateral ICA angiography revealed an Acom aneurysm measuring 6mm in diameter $\times$ 5.5 mm in length $\times$ 4.8 mm neck with an agenetic RT A1 segment. Multiple coils for a total length of 28 cm allowed exclusion of the sac with partial filling of the neck Raymond 2 scale¹. The patient recovered with no neurological changes. Four hours after heparin discontinuation, he became confused, aphasic with RT side dense plegia at the lower limb and monoparesis at the upper

limb (grade 2 motor power). Emergent CT brain revealed no rebleeding, he was loaded with 300 mg of Clopidogrel, 500 mg IV ASA and then transferred to the angiosuite. Under general anesthesia Left ICA angiography showed complete occlusion of the left A2 segment origin. The ACT was kept at 250 s throughout the procedure; 200 μ g of Tirofiban was injected locally over five minutes. This did not allow full reopening of the occluded artery. A Solitaire 4 $\times$ 20 mm stent was deployed allowing full revascularization of the whole left ACA (TIMI) grade 3 (Figure 3) (Figure 4). The time from onset of symptoms to full revascularization of the ACA was two hours 40 minutes. The patient recovered from the aphasia four hours post stent deployment. Six hours later the motor power improved to grade 4 in the upper limb and grade 2 in the lower limb. MRI diffusion sequence done 24 hours from the onset of symptoms showed areas of diffusion restriction at the cingulum cerebri and centrum semiovale (Figure 5). Five days later and before discharge, the neurological examination revealed more improvement in the lower limb, with a motor power of 3. He was discharged pursuing physiotherapy. All patients were kept on dual antiplatelet 75 mg clopidogrel for six months and 150 mg aspirin for life.

Discussion

Thromboembolic events are the most common complications during or shortly after aneurysm coiling, varying in incidence between

Figure 5 Diffusion MRI image shows spots of diffusion restriction at the cingulum cerebri and centrum semiovale.

2.5 and 28%²⁻⁴. The Solitaire stent, a closed cell design, has a radial force of 0.0106 N/mm that allowed ACA reopening in the cases of this study.

Major vessel occlusion represents a true clinical hazard which may lead to serious morbidity or even death. This complication can be managed pharmacologically or mechanically. In the three cases presented in this study, pharmacological measures to reopen the occluded vessel failed. The pharmacological measures included IV heparin reaching an ACT between 250 and 300 seconds, IV acetylsalicylic acid, clopidogrel, abciximab or tirofiban. Tirofiban and abciximab are chimeric monoclonal antibody fragments against the platelet glycoprotein IIb/IIIa receptor complex inhibiting the final common pathway of platelet aggregation. Bruening et al. used IV tirofiban to treat 16 patients with ruptured cerebral aneurysms who developed thrombus formation during aneurysm coiling. Eight out of ten patients who had total or partial occlusion of the parent vessel showed complete recanalization after IV tirofiban⁵. Mounayer et al. used intra-arterial abciximab to treat 13 cases that developed intraprocedure thrombus formation during aneurysm

coiling of whom four presented with SAH. They achieved complete recanalization in 12 patients (92%)⁶. Song et al. used intra-arterial abciximab during aneurysm coiling in seven cases that developed intraprocedure thrombus formation. Four cases were ruptured; they achieved complete or almost complete recanalization in six patients⁷. Cronqvist et al. treated 19 patients with intra-arterial urokinase, achieving complete recanalization in ten patients and partial recanalization in nine. However three out of six ruptured aneurysms developed devastating intracerebral hemorrhage⁸. Aggour et al. found 10% thromboembolic events related to aneurysm coiling. They used combined IA and IV abciximab to treat the developing thrombi in 39 patients of whom 11 had ruptured aneurysms. They observed 56.5% complete recanalization, amelioration in the TIMI score in 73.9% yet no change was observed in 26.1%⁹.

Qureshi et al. used local RTPA in seven patients during endovascular treatment of cerebral aneurysms. Five out of seven presented with SAH. They achieved TIMI 2 or 3 in four patients but four patients had an MRS score of 3 or more at three months¹⁰.

Two risk factors in our cases probably predisposed to thrombus formation or propagation: the wide aneurysm neck and the coil loops encroachment on the origin of the A2 segment. The dome-to-neck ratio in the three cases was less than 1.5. According to Workman et al. thrombus propagation in wide-neck aneurysms treated by coils may occur by promoting a larger coil surface area on which to form or greater access of the thrombus formed inside the aneurysm sac to the parent artery¹¹.

The use of intracranial stents in treating wide-necked aneurysms necessitates pre and post procedure dual antiplatelet therapy consisting of clopidogrel and acetylsalicylic acid. This antiplatelet therapy is risky in acute SAH or non-secured aneurysms¹². However in some situations with difficult wide-neck aneurysms intracranial stents may sometimes be the only solution. Intracranial stents have been used to treat acutely ruptured aneurysms. Tähtinen et al. treated 61 patients with ruptured aneurysms using intracranial stents reporting 21% of technique-related complications. Two patients in their series developed thromboembolic complications requiring insertion of Neuroform stents (Boston scientific corporation, Natick, MA, USA) as a bail-out procedure. They had throm-

boembolic complications post stent deployment or in-stent thrombosis in seven out of 61 patients (11%)¹². Yoo et al. used a bail-out stent during intracranial aneurysm embolization in 16 patients after deployment of one or more coils. Three patients in their study presented with SAH¹³. Lodi et al. used intracranial stents in treating wide-neck ruptured aneurysms in 22 patients. They had no intraprocedure hemorrhagic events but two in-stent thromboses developed, one was symptomatic requiring intra-arterial injection of glycoprotein IIb IIIa receptor antagonist¹⁴.

Thrombus development during coiling of ruptured aneurysms is problematic because in such a situation we will have three objectives: first to secure the aneurysm which is the initial goal; second to achieve lysis of the thrombus and reopening of the occluded artery; third to avoid perforation of the aneurysm after giving antiplatelet and intra-arterial thrombolytic drugs if additional coils are required to achieve satisfactory aneurysm occlusion post stent deployment.

Angiographic follow-up was only performed for case 1; the other two cases are scheduled for regular post coiling six month angiography. No

in-stent stenosis was noted up to the 18 months angiographic follow-up in case 1. When stents are used as an adjunctive tool for wide-neck aneurysm treatment, the in-stent stenosis rate is lower than when used to treat atherosclerotic stenotic lesions. Fiorella et al. reported a 5.8% in-stent moderate or severe delayed stenosis for stent-remodeled coil embolization¹⁵. We are aware of the small sample included in this study, but deployment of intracranial stents during endovascular treatment of ruptured aneurysms is a rescue measure in an exceptional situation. This study also lacks a long-term follow-up.

Conclusion

Deployment of an intracranial Solitaire stent achieved complete revascularization of the occluded ACA. Its use in the setting of SAH is relatively risky but it succeeded in significantly improving symptoms following flow restoration and probably helped to prevent symptoms worsening, major disability or even death. A study on a larger patient sample with long-term follow-up will be of value.

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