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Summary: We report the case of a supraclinoid carotid rupture during the delivery of a balloon-expandable stent in a 59-year-old patient with incidental paraclinoid berry aneurysms for whom stent-assisted coiling was planned. The deployment of the stent resulted in immediate rupture of the artery. We describe the emergent management of this complication with prolonged balloon inflation to occlude the site of rupture, a treatment that led to the discharge of the patient 2 weeks later without any sequelae.

The balloon-assisted, so-called remodeling, technique emerged several years ago as a supplemental tool in the treatment of wide-necked aneurysms or of aneurysms with an unfavorable dome-to-neck ratio (1). Four years ago, the introduction of three-dimensional coils and neck-bridge devices augmented the spectrum of intracranial aneurysms suitable for coiling (2, 3). More recently, the stent-assisted coiling technique—which consists of the prior placement of a stent into the parent vessel across the aneurysm neck, followed by the coil deposition into the sac through the stent mesh—has rapidly gained acceptance within the interventional community (4-6). The latter technique is still limited by the relatively poor ability of the various stent devices to navigate adequately along the intracranial arterial tree, specifically in the anterior circulation. We report a case of stent-assisted coiling that was complicated by rupture of the parent vessel during stent delivery and the emergent treatment that followed and led to a favorable clinical outcome.

Case Report

A 59-year-old woman was referred to us for the endovascular treatment of two ipsilateral incidental paraclinoid aneurysms. Cerebral angiography revealed that the aneurysms existed in a series in the C2 segment of the right internal carotid artery (ICA). The three-dimensional reformatted images of the right carotid angiogram obtained from rotational angiography revealed the right carotid siphon to be dysplastic (Fig 1).

The treatment strategy consisted of the coiling of the two aneurysms. To do so, it was decided first to deploy a stent in the C2 segment of the ICA to cover both aneurysm necks, allowing further deposition of the coils into the aneurysms. The endo-

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vascular procedure was carried out under general anesthesia and full anticoagulation (5000 IU of heparin, given as an intravenous bolus injection, followed by continuous infusion of 2500–3000 IU/h). The goal of anticoagulation therapy was to keep the activated clotting time at two to three times above the normal value during catheterization, stent deployment, and coil deposition. In addition, 250 mg of aspirin was given intravenously as a single dose at the beginning of the procedure.

To begin, a four-vessel cerebral angiogram was used to assess the functionality of the circle of Willis. The cross-flow from the left ICA to the right hemisphere was judged sufficient to maintain right hemispheric perfusion in case of right ICA flow impairment. We chose first to navigate a self-expandable (4 mm in diameter, 15-mm-long) stent (Neuroform; Boston Scientific, Natick, MA) through a 6F guiding catheter (Envoy; Cordis, Miami Lakes, FL) over a 0.014-inch guidewire (Choice; Boston Scientific/Scimed, Maple Grove, MN). After several unsuccessful attempts to deliver the stent, we exchanged the Choice guidewire for a stiffer one (Luge; Boston Scientific/Scimed). Unfortunately, even with the use of the Luge guidewire, it was not possible to deliver the stent into the targeted vessel segment.

At this point, we decided to exchange the self-expandable Neuroform stent for a balloon-expandable Cerebrence stent (Medtronic, Minneapolis, MN). This stent was 4 mm in diameter with a length of 12 mm when deployed at its nominal pressure of 6 atm. This over-the-wire stent was then advanced over the 0.014-inch Luge guidewire, and its distal tip was secured in the M3 portion of the right middle cerebral artery. Once the Cerebrence stent was positioned in the C2 portion of the ICA across the aneurysm necks (Fig 2), we gently and gradually inflated the balloon to 5 atm to expand the stent diameter to 3.8 mm as indicated by the manufacturer. The balloon was then deflated and left across the deployed stent. The control angiogram immediately demonstrated a massive contrast material extravasation distal to the distal end of the stent, attesting to the carotid rupture into the subarachnoid space and cavernous sinus; there was no more distal contrast material filling of the arterial tree via right ICA injection (Fig 3). Heparin was reversed with intravenous protamine sulfate.

We carefully reinflated the balloon to 2 atm to close the right ICA. The endovascular procedure was discontinued while we performed CT. CT revealed a subarachnoid hemorrhage and slight ventricular enlargement. Twenty minutes after having reinflated the balloon at low pressure to stop the hemorrhage, the balloon was deflated, but an immediate right ICA angiogram showed the recurrence of the massive contrast medium extravasation. The decision to deflate the balloon was made to assess whether this temporary balloon occlusion at the site of rupture would have been sufficient enough to obtain local hemostasis, but the severity of the vessel tear precluded this attempt. The recurrence of the subarachnoid hemorrhage assured us that the severity of the vessel tear should prompt emergent and definitive occlusion of the right ICA.

The balloon was then reinflated for hemostatic purposes. At this point, we decided to perform an occlusion of the ICA proximal to the site of rupture with coils while keeping the balloon inflated to prevent back bleeding. Another option would have been to consider proximal ICA coiling as well as coiling of the most distal right ICA portion through the ante-

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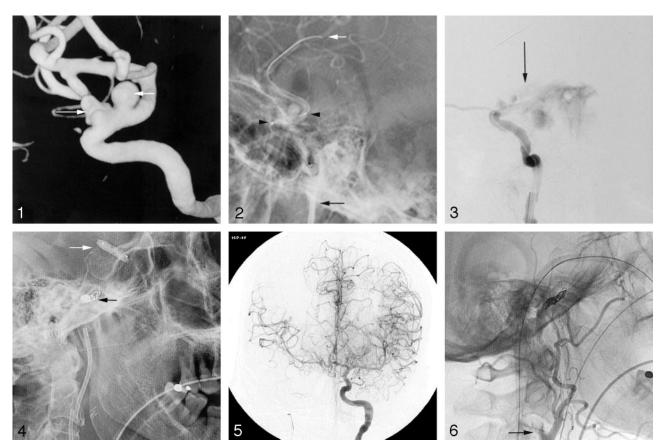


Fig 1. Tridimensional representation of the right ICA, showing the dysplastic carotid siphon harboring two saccular aneurysms (arrows).

- Fig 2. Right ICA unsubtracted angiogram (working projection) showing the positioning of the stent (*arrowheads*) before inflation of the delivery balloon. The distal tip of the Luge guidewire (*white arrow*) is secured into the M3 segment of the right middle cerebral artery. The 6F Envoy guiding catheter (*black arrow*) has been navigated up to the skull base.
- Fig. 3. Right ICA subtracted control angiogram (working projection) immediately after the stent delivery, showing the massive contrast medium extravasation in keeping with the ruptured carotid.
- Fig. 4. The stent delivery balloon (white arrow) has been reinflated for hemostatic purpose into the stent. A second 6F guiding catheter has been navigated into the right ICA to allow the occlusion with coils (black arrow) of the petrous portion of the right ICA.
- Fig. 5. Left ICA angiogram during the occlusion of the right ICA, showing good cross-flow through the anterior communicating artery.
- Fig 6. Right common carotid unsubtracted angiogram (lateral projection) after the removal of the balloon, showing the persisting occlusion of the right ICA (arrow).

rior communicating artery or through the right posterior communicating artery. These two options appeared unrealistic, because it was not possible to determine the exact length of intact right supraclinoid ICA between the tear and the right ICA termination.

With the patient still on the angiographic table, to control the raising intracranial pressure and to place an external drainage site, a right frontal burr hole was made to puncture the right lateral ventricle. Once the external drainage was set, the petrous portion of the right ICA was occluded with coils (TruFill; Cordis) via a second 6F Envoy guiding catheter navigated into the right ICA via a left femoral approach (Fig 4). After the procedure, the patient was left intubated and sedated. Anticipating that the right ICA occlusion with coils would not be sufficient to protect the patient from further hemorrhage in this acute setting, the right femoral sheath was left in place with the 6F guiding catheter into the right cervical ICA as well as the balloon catheter connected to the inflation device (Indeflator; Boston Scientific). The left ICA control angiogram showed cross-flow from left to right via the anterior communicating artery with good filling of the right hemisphere (Fig 5). After the first procedure, monitoring of the patient was achieved in the intensive care unit. Seven days later, a follow-up angiogram showed the persistence of the ICA occlusion with both the coils and the balloon left in place.

At this time, because we thought that after 7 days of carotid occlusion both at the site of rupture and more proximally with coils, the ICA would have clotted sufficiently to allow the balloon to be retrieved without excessive risk, we decided to deflate the balloon. Doing so sooner would have probably been more hazardous because of the possibility of insufficient clotting of the carotid, but waiting longer could have made retrieval of the balloon more difficult as the clot became more organized. Thus, the balloon was completely deflated by using a 50-mL syringe to create a vacuum, with the plunger kept retracted by use of a clamp. Both the guiding catheter and the balloon were then retrieved en bloc from the supraclinoid ICA across the coil mesh down to the femoral sheath. A control angiogram showed the persistence of the right ICA occlusion after balloon was removed (Fig 6). The next day, sedation was discontinued. The external ventricle drainage was removed 10 days after the procedure, and the patient was discharged home 14 days after the intervention without any neurologic deficit.

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Discussion

Endovascular ICA occlusion is a widely accepted and effective method in the treatment of large and giant carotid aneurysms with anticipated surgical or endovascular difficulty; however, despite the use of various testing techniques to determine whether a patient could tolerate permanent carotid occlusion, 5–22% of patients undergoing balloon occlusion of the carotid artery develop ischemic complications (7–9), making the preservation of the ICA patency a more desirable goal when achievable. Several reports have similarly documented contralateral aneurysm formation or growth after permanent ICA occlusion (7, 10). The selective endovascular treatment of wide-necked intracranial aneurysms remains challenging and leads to less favorable treatment results and long-term angiographic outcome than for small aneurysms. The stent-assisted coil embolization technique is still limited to some difficult cases because of the unavailability of stent devices that adequately negotiate tortuous intracranial vessels.

On the other hand, many studies have already reported the use of intracranial stents for the reconstruction of arteries affected with aneurysms, stenosis, and dissections, allowing vessel patency and a scaffold for endothelial growth. In particular, these devices are instrumental to preventing coil protrusion during the treatment of wide-necked, dissecting, and fusiform aneurysms. They also play a major role in angioplasty of atherosclerotic stenoses to prevent plaque disruption and intimal flap, thus reducing the risk of abrupt vessel closure and distal embolization and preventing early rethrombosis after successful thrombolysis and may be used to trap accidentally protruding coils during aneurysm treatment (11).

The inherent potential complications of stent-assisted aneurysm coiling can be divided into mechanical, ischemic, and hemorrhagic events. Mechanical complications encompass the unintentional detachment from the balloon before reaching the lesion, the inability to reach the targeted lesion specifically when the aneurysm is located along the course of the intracranial ICA, the backward stent dislodgment when removing the delivery balloon, and the inability to traverse the stent mesh with the microcatheter for secondary coil deposition (12). Although ischemic complications appear less likely than during intracranial angioplasty, they may be due to abrupt or delayed stent closure in keeping with inadequate anticoagulation protocol or intimal damage leading to platelet aggregation and subsequent thrombus formation.

Hemorrhagic complications of stent-assisted aneurysm treatment are secondary to dissection or perforation of distal cerebral arteries where the guidewire tip has been secured. This later issue is of paramount importance because the use of more rigid guidewires is necessary for stent tracking along tortuous arterial trees such as the intracranial ICA, which in many instances require a more distal placement of the guidewire tip. One arterial rupture during stent-assisted coil embolization has been recently reported by Lylyk et al (12). This dramatic complication occurred in one of 62 pa-

tients treated with the combination of stent and coils. The ICA rupture occurred during the stent placement at the neck of a giant carotid-cavernous aneurysm and resulted in the patient's death. In our case, the ICA rupture took place distal to the aneurysm neck, at the junction of the C1 and C2 ICA segments. One can argue that the relative rigidity of the stent did not conform to the slight curve separating the C1 and C2 carotid portions, making the use of a self-expandable stent more judicious as a first attempt. The self-expandable stents are supposed to have a better conformation to the vessel and should be more versatile to adapt to vessel tortuosities such as those of the intracranial ICA (13). For these reasons, our first choice was the self-expandable Neuroform stent, which certainly would have been less traumatic for the carotid siphon and would have conformed more amenably, but our efforts to deploy it were in vain.

In this case, the good clinical outcome of our patient was due to the anticipated excellent collateral flow through the anterior communicating artery and also to the emergent external shunt surgery of the ventricles. The closure of the petrosal ICA with coils secured the distal occlusion of the supraclinoid ICA and allowed a definitive vessel occlusion once the balloon occlusion was discontinued 7 days later.

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