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Percutaneous Direct-Puncture Acrylic Embolization of a Pseudoaneurysm after Failed Carotid Stenting for the Treatment of Acute Carotid Blowout

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Summary: We report a difficult case of recurrent carotid blowout syndrome in a patient who had a rupture of the common carotid artery with formation of a large pseudoaneurysm. Conventional management of this emergency, consisting of composite occlusion of the internal and common carotid arteries, was not possible owing to prior clinical failure of balloon test occlusion. This led to an initial attempt to cover the site of rupture with overlapping self-expanding stents, which was only temporarily successful in controlling the hemorrhage. When a subsequent episode of carotid rupture with life-threatening hemorrhage occurred, percutaneous direct-puncture acrylic embolization with temporary flow arrest was used to successfully obliterate the pseudoaneurysm.

An increasingly recognized delayed complication of aggressive primary and salvage radical neck surgery for squamous cell carcinoma of the head and neck is spontaneous rupture of the carotid artery, or so called "carotid blowout" (1, 2). In our experience and that of others (1–7), a variety of clinical presentations of actual and potential carotid rupture may occur, justifying the concept of carotid blowout syndrome (ie, a spectrum of clinical manifestations and possible pathoetiologies of carotid rupture) (2). Consequently, our center has instituted a clinical protocol for multidisciplinary management of carotid blowout syndrome in which, after initial stabilization measures have been executed, meticulous diagnostic angiography is performed to identify the known heterogeneous group of angiographic pathoetiologies of hemorrhage that may occur in this patient population (1, 2). These findings enable the physician to tailor the best endovascular embolotherapeutic approach to the specific lesion responsible for the hemorrhage (2). Such an approach has resulted in substantially improved outcomes (89% survival with 0% major and 15% minor neurologic morbidity) as compared with most prior surgical series (2). However, we have encountered some limitations with this protocol in the subpopulation of patients with carotid blowout syndrome who require therapeutic occlusion of the internal or common carotid artery owing to either threatened or actual rupture of the vessel but who do not tolerate balloon test occlusion (2).

We recently encountered such a patient, who presented on four separate occasions with carotid blowout syndrome, in whom a spectrum of pathoetiologic substrates were addressed initially by conventional endovascular and surgical approaches. Because the affected carotid system was shown to be intolerant to balloon test occlusion, treatment strategies for subsequent episodes of threatened and acute carotid blowout were limited to less conventional parent artery preservation techniques.

Case Report

Our patient is a 66-year-old woman with a history of T2N0M0 stage II squamous cell carcinoma of the right tonsillar fossa, diagnosed in June 1995. She was treated with external-beam radiotherapy, but had a local recurrence (T2N2bM0) in February 1996. Further treatment included a composite resection and modified radical neck dissection with intraoperative brachytherapy and a left radial forearm free flap to the right side of the neck. In December 1996, the patient was found to have palpable disease in the left side of the neck. Treatment consisted of a right-sided composite resection, bilateral supraomohyoid neck dissections, and a right fibular free flap to the right side of the neck. Within a week, the free flap became ischemic, requiring a left fibular free flap with an end-to-end anastomosis of the left peroneal artery to the left facial artery to revise coverage and vascularization of the right-sided neck wound.

On postoperative day 20, the patient returned with brisk bleeding from the left side of the neck, which was only temporarily controlled with a pressure dressing (ie, acute carotid blowout [2]), and she was immediately taken to the operating room for exploration. No source for the bleeding could be identified; however, a Jackson-Pratt drain was placed and emergency angiography was performed.

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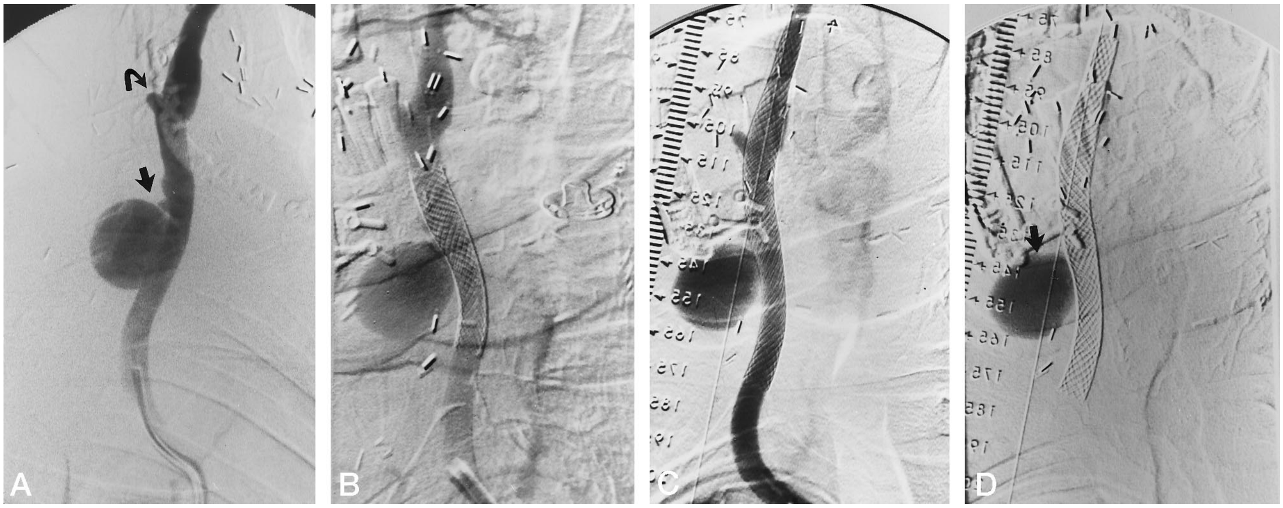

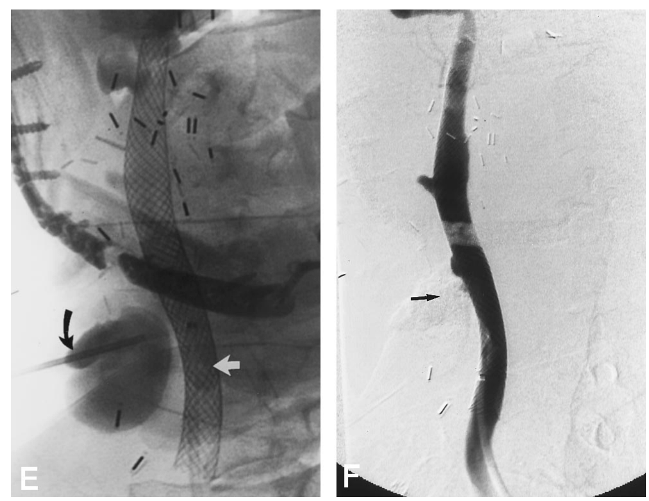

FIG 1. 66-year-old woman with recurrent carotid blowout syndrome in whom a large pseudoaneurysm developed.

A, Oblique view of a right common carotid artery injection shows large pseudoaneurysm (*straight arrow*). Note evidence of prior surgery, including ligation of the external carotid artery (*curved arrow*).

B, Angiogram after deployment of an 8 × 20-mm Wallstent across the rent in the carotid artery shows good distal runoff but persistent filling of the pseudoaneurysm.

C, Repeat angiogram after deployment of a second, overlapping (8 × 60-mm) Wallstent shows slower filling of the pseudoaneurysm.

D, Delayed image shows stagnation of contrast material within the pseudoaneurysm (*arrow*).

E, Fluoroscopic spot film shows a balloon occlusion catheter within the stents, with the balloon partially inflated (*straight arrow*). This allowed some retrograde flow of opacified blood to fill the pseudoaneurysm, which was percutaneously punctured with a 200-gauge needle (*curved arrow*).

F, Control angiogram after two direct-puncture acrylic embolizations shows complete obliteration of the pseudoaneurysm with patency of the carotid artery. Subtraction artifact is seen where NBCA was injected at the site of rupture (*arrow*).

Superselective angiography showed active extravasation of contrast material from a branch of the left facial artery into the recently placed surgical drain. This was promptly treated by transarterial embolization with several fibered platinum microcoils, resulting in complete cessation of bleeding.

One month later (January 1997), the patient was readmitted with a right-sided neck wound dehiscence and an exposed carotid artery (ie, threatened carotid blowout [2]). Repeat angiography showed no pseudoaneurysm, vascular narrowing, or irregularity. The external carotid artery had been surgically ligated at its origin. In preparation for further surgical resection and reconstruction, a carotid balloon test occlusion was performed (2). However, the patient became confused and hemiparetic within 15 minutes of occlusion. Neurologic signs returned to baseline immediately after the balloon was deflated.

A heavily radiated local field and extensive prior reconstructive surgery deterred surgical replacement of the artery at risk. Subsequently, a right musculocutaneous pectoralis flap was mobilized to cover the exposed carotid artery. The mobilized flap healed well and the patient was discharged to home 1 week later. Unfortunately, she was home for only 1 week when bleeding developed from the right side of the neck that was not completely controlled with a pressure dressing (ie, a second episode of acute carotid blowout). She was admitted emergently for supportive therapy, and repeat angiography showed a large (3.0-cm) pseudoaneurysm of the distal right common carotid artery, with a relatively wide neck arising from the posterolateral wall (Fig 1A). With no surgical bypass options and prior failed balloon test occlusion, a decision was made to treat the pseudoaneurysm primarily with intravascular stents.

A 9F multipurpose guiding catheter (Cordis, Europa, NY) was initially advanced to the proximal right common carotid artery, and a 0.035-inch exchange length hydrophilic guidewire (Terumo, Tokyo, Japan) was placed in the distal cervical internal carotid artery. Under fluoroscopic guidance, an 8 × 20-mm Wallstent (Schneider, Minneapolis, MN) was deployed across the neck of the pseudoaneurysm without difficulty. Repeat angiography showed good distal runoff but persistent filling of the pseudoaneurysm (Fig 1B).

At this time, the patient was hemodynamically stable with minimal bleeding. We waited 15 minutes to allow for potential progressive thrombosis of the pseudoaneurysm subsequent to the change in flow dynamics created by the stent. Control angiography showed only minimally slower filling and delayed emptying of the pseudoaneurysm, therefore prompting us to position and deploy a second overlapping Wallstent (8 × 60 mm) to decrease the porosity of the endoluminal prosthesis and to increase the resistance across the rent. Unfortunately, the pseudoaneurysm continued to fill.

Again, time was allowed to elapse, and a 5.4F double-lumen temporary balloon occlusion catheter (Zeppelin, 8 mm; Medtronics-MIS, Sunnyvale, Calif) was inflated for approximately 15 minutes across the rent in a further attempt to promote pseudoaneurysmal thrombosis. Subsequent angiography showed slow filling of the pseudoaneurysm with stagnation of contrast material (Fig 1C and D). At this time, the bleeding appeared to be well controlled. Consequently, we decided to observe the patient closely in the hope that complete progressive thrombosis of the pseudoaneurysm would occur.

Although she did well overnight, approximately 18 hours after stent implantation she had a repeat episode of acute carotid blowout from rupture of the pseudoaneurysm. Emergent recatheterization was performed in which initially a 6.4F double-lumen balloon occlusion catheter (Zeppelin, 10 mm) was positioned and inflated across the carotid rent to control active bleeding.

The balloon was inflated without immediate change in the patient's neurologic signs. However, flow arrest could only be maintained for short intervals owing to intolerance of prolonged balloon test occlusion. Treatment options were limited. No covered stent was readily available. Because of the potential technical difficulties and lack of a true endothelial wall or connective tissue support surrounding the pseudoaneurysm, we did not think that transarterial or direct-puncture endosaccular coil embolization would be beneficial. For all these reasons, plus our access to and familiarity with *n*-butyl cyanoacrylate (NBCA) (Histoacryl, Braun, Germany), we decided to treat the pseudoaneurysm by percutaneous direct-puncture acrylic embolization.

Percutaneous Direct-Puncture Embolization

The balloon occlusion catheter was initially fully deflated and repositioned under fluoroscopic guidance and road-mapping to lie across the rent within the carotid artery. The balloon was then partially inflated to arrest anterograde flow while leaving some retrograde flow of opacified blood to fill the pseudoaneurysm (Fig 1E). This then permitted visualization of the rupture site for localization and accurate direct-puncture embolization while minimizing the risks of uncontrolled hemorrhage. A 20-gauge blunt-tipped needle was inserted percutaneously into the pseudoaneurysm (Fig 1E) and approximately 150 mL of blood was aspirated. Using orthogonal fluoroscopic guidance, we injected contrast material through the puncture needle to confirm placement at the rent in the carotid artery, in apposition to the posterolateral aspect of the stent. The balloon catheter was then fully inflated across the rent to provide complete flow arrest and to prevent potential distal embolization of the acrylic material. Two slow, controlled injections of approximately 0.5 mL of a 1:1 mixture of NBCA and ethiodized oil (with tungsten powder) were administered at the site of rupture.

Control angiography performed after the second direct-puncture embolization showed complete obliteration of the pseudoaneurysm with preserved patency and normal runoff of the carotid artery (Fig 1F). At this time, the patient was mildly hemiparetic on her left side, presumably because of the prolonged period of balloon occlusion needed to permit setting up this therapeutic intervention. Fortunately, her neurologic signs rapidly returned to baseline over the next several minutes. Angiographic studies of the anterior circulation were unchanged and without evidence of thromboembolism. The patient remained neurologically and hemodynamically stable.

Color duplex Doppler sonography performed 1 day and 2 weeks after treatment confirmed complete obliteration of the pseudoaneurysm and normal flow within the carotid artery. The patient continued to do well and was eventually discharged from the hospital.

Discussion

The vascular injuries that occur during aggressive treatment of head and neck cancer, which manifest as the carotid blowout syndrome, appear well suited to endovascular therapeutic intervention. The specific role of endovascular therapy in terms of indications and appropriate selection of techniques and technology continues to evolve (2). By using pathoanatomic data obtained from angiography combined with provocative testing, the interventional neuroradiologist can match the best endovascular approach(es) to the lesion responsible for a given hemorrhage. In our experience, the majority of such cases can be effectively managed by therapeutic embolization, typically consisting of either large parent artery occlusion (eg, common, external, and internal carotid arteries) or small-vessel occlusion (eg, external carotid branches, tumor hemorrhage) (1, 2).

This particular case illustrates some of the most important therapeutic challenges with which neurointerventionalists may be confronted when treating patients with carotid blowout syndrome. This patient had repeated episodes of carotid blowout that initially could be managed by conventional endovascular and surgical approaches. However, when balloon test occlusion failed in the face of impending carotid blowout, therapeutic options were limited to those that would preserve the parent artery. Initially, pectoralis flap mobilization was used in an attempt to cover the exposed carotid artery for prevention of subsequent rupture. When this failed, few additional conventional options remained. Common-internal or external-internal carotid arterial bypass with subsequent parent artery occlusion of the diseased carotid system was precluded by prior radiation and radical and reconstructive surgeries in the neck. Since both the patient and her family wished to aggressively pursue treatment for her carotid rupture, intravascular stenting was initially performed in an attempt to obliterate the large carotid pseudoaneurysm.

The use of intravascular stents for the treatment of various diseases of the extracranial carotid artery is being increasingly reported. Preliminary studies in two relatively large series have shown both the technical feasibility and relative safety of combined percutaneous transluminal angioplasty with stenting in selected patients with carotid occlusive disease (8, 9). Recently, stents have been used successfully to treat arterial dissection–related pseudoaneurysms of the cervical internal carotid artery in a small number of patients (10, 11) (Lee LI, Ferguson RD, Holbrook JT, et al, "Stent Therapy of Cervical Internal Carotid Artery Pseudoaneurysms," presented at the annual meeting of the American Society of Neuroradiology, Seattle, June 1996). The rationale for this application of endovascular techniques comes predominantly from animal studies of experimentally constructed side-wall aneurysms and arteriovenous fistulas (12–19). Ongoing advances in device technology have led to further investigation with regard to the best materials for potential use in a number of specific clinical circumstances.

Graves et al (12) have shown that it is possible to induce thrombosis by altering flow dynamics within aneurysms. Several authors have since shown that it is feasible and efficacious to implant intravascular stents across the neck of an experimental aneurysm to promote thrombosis and organized fibrosis within the aneurysmal sac, with preservation of the parent artery (13, 14). The time required to obliterate an aneurysm can be highly variable. This prompted some investigators to introduce electrical detachable coils into the aneurysms through the stent mesh, successfully effecting aneurysmal occlusion while maintaining patency of the parent vessel (15, 16).

Clinically, stents have proved useful in some cases of angioplasty-induced dissection of the coronary and peripheral vasculature by effectively obliterating the false lumen and theoretically strengthening the parent vessel. There have been anecdotal reports of endovascular stenting for internal carotid artery dissection with eradication of the associated pseudoaneurysms (10, 11) (Lee LI, et al. ASNR meeting 1996). Recently, Parodi et al (20) successfully treated a large traumatic pseudoaneurysm of the common carotid artery with placement of an endoluminal stent wrapped with a basilic vein graft. Their patient was asymptomatic and treated on the basis of the high potential for complications.

We chose to use the Wallstent in our patient because of its flexibility, atraumatic self-expanding deployment mechanism, and relatively decreased porosity. A covered stent would have been ideal in this situation, but was not readily available. A second, overlapping stent was placed to further decrease the relative porosity of the endoluminal prostheses in an attempt to increase resistance across the rent into the pseudoaneurysm. Theoretically, this may enhance spontaneous thrombosis of lateral wall aneurysms (13, 18). A secondary goal of this approach was to provide additional structural support to a long segment of the diseased carotid system in an attempt to prevent repeated rupture at other sites. This latter hypothesis is based on our knowledge of the pathologic substrate of carotid blowout syndrome in which there is typically extensive and multifocal radiation fibrosis, medial necrosis, inflammatory infiltration, loss of adventitia, secondary atherosclerosis, and lack of external supportive tissues (21, 22).

Unfortunately, this technique was only temporarily successful. When the carotid stents failed to control recurrent bleeding, we were forced to consider alternative treatment options directed at obliteration of the pseudoaneurysm. Time remained a factor with regard to ongoing hemorrhage and the patient's intolerance of prolonged carotid balloon occlusion. Because of the potential technical difficulties and the lack of a true endothelial wall or supporting tissues surrounding the pseudoaneurysm, we did not think that transarterial or direct-puncture endosaccular coil embolization would be effective. The accessibility of NBCA and our relative expertise with its use prompted us to consider direct-puncture acrylic embolization.

Although initially described by Casasco et al (23) for preoperative or palliative embolization of hypervascular tumors, we have recently applied direct-puncture acrylic embolization to the treatment of extracranial high-flow vascular malformations and hypervascular tumors (24, 25). This technique has the advantage of allowing direct access to anatomic regions that may be difficult or dangerous to reach by conventional transarterial routes. In the present case, the rapid direct placement of a needle into the aneurysmal neck permitted the simultaneous use of a double-lumen temporary balloon occlusion catheter within the stented artery to control anterograde flow. This combination allowed a precise and controlled injection of NBCA to seal the carotid rent. This approach, although unconventional, was successful in treating a life-threatening carotid blowout.

In conceiving and implementing our therapeutic approach, we had to weigh the risks of attempting this unproven procedure against the potential benefits. Possible risks include laceration of the carotid artery or rupture of the pseudoaneurysm, resulting in uncontrollable hemorrhage; permanent occlusion of the carotid artery by gluing the balloon occlusion catheter or by inadvertently filling the lumen of the carotid with adhesive; and thromboembolic events from either the catheter or the NBCA. Considering that without treatment the patient would have certainly died from exsanguination or would have suffered a major stroke if permanent balloon occlusion of the carotid artery had been performed, our decision appears justified.

Conclusion

As advances in endovascular technology continue to be made, covered stents may provide the optimal treatment for patients with ruptured carotid pseudoaneurysms. In the event of encountering a patient with life-threatening carotid blowout syndrome, we have described a reasonable alternative technique that proved safe and successful in arresting acute hemorrhage.

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