Intraoperative Decision-Making for TCAR Complications

BY EDGAR GUZMAN, MD, FACS, AND SUMAIRA MACDONALD, MD, PHD, MBCHB, MRCP, FRCP, FRCR, EBIR

Edgar Guzman, MD, FACS
Vascular Surgeon
Orlando Regional Medical Center
Orlando, Florida
edgar_guzman@sbcglobal.net
Disclosures: Consultant to Silk Road Medical.

Sumaira Macdonald, MD, PhD, MBChB, MRCP, FRCP, FRCR, EBIR
Vascular Interventional Radiologist
Executive Medical Director
Silk Road Medical
Sunnyvale, California
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The technical strengths of transcarotid artery revascularization (TCAR) are leading to ever broader application in complex anatomy, often in the face of substantial physiologic challenges. Although careful preoperative planning and openness to consider all treatment modalities can reduce intraprocedural complications, these are not fully avoidable. As with any complication, the single most important step in salvage is recognition of the problem, which may be difficult in the context of angiography under flow reversal and rendered more so given that these events are so infrequently encountered and may not be immediately identified.

Given the low overall incidence of intraprocedural complications and youth of TCAR as a surgical technique, there is no high-level evidence to guide management of these events. However, much has been learned in the field, as well as from case reviews. We offer experience-based recommendations to manage three scenarios that may be encountered.

CAROTID DISSECTION

In our experience, the etiology of carotid dissection (Figure 1) was mostly traceable to difficulties with needle or microsheath dislodgement, often coupled with unfavorable conditions at the intended access zone. Preoperative CTA assessment from the aortic arch to the circle of Willis allows identification of significant disease at the "clamp and stick" zones of the common carotid artery (CCA), as well as other anatomic features that may lead to complication. Highlighting the importance of access acquisition during TCAR, the use of the neuroprotection system (NPS) through a poor-quality CCA is considered off-label.

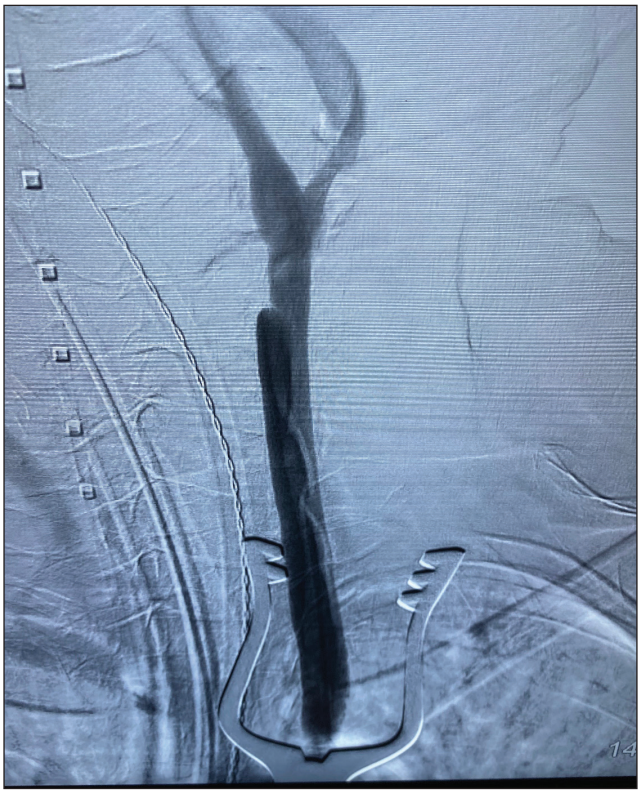

Figure 1. Carotid dissection propagating from the microsheath access site in the common carotid artery.

In the event of loss of access, the limited CCA exposure

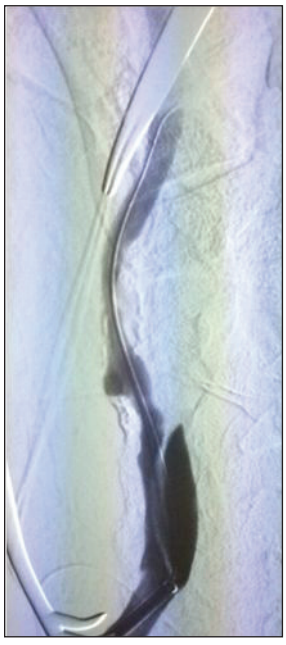

Figure 2. Carotid dissection imaged under flow reversal. Note the tip of the sheath abutting the vessel wall and presence of an intimal flap.

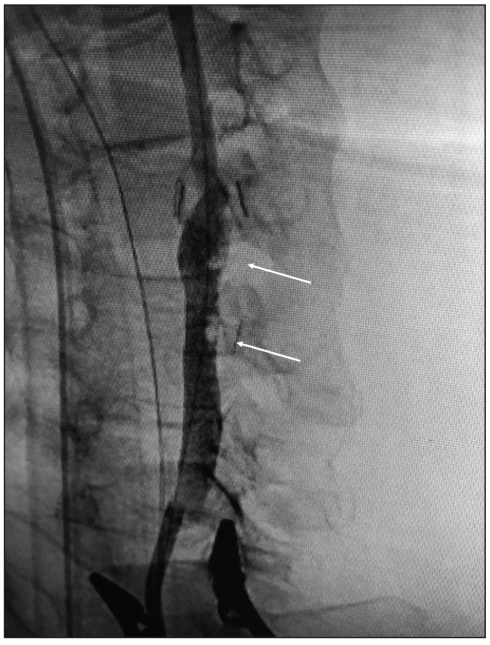

Figure 3. Plaque prolapse through stent struts.

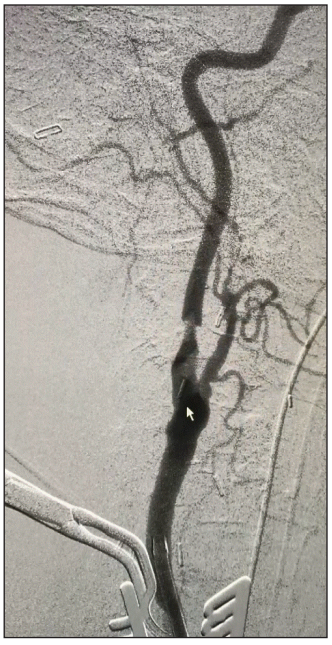

Figure 4. Carotid flame sign.

in TCAR and the presence of a preclosure suture make it tempting to reuse the arteriotomy site, but this can lead to entry into a false plane. It is best to close the original arteriotomy and obtain a new access in a more proximal location.

Identification of a dissection from a microsheath is challenging, even with antegrade flow, and may be extremely difficult under flow reversal (Figure 2). The diagnosis of this complication is insinuated by subtle changes in wire behavior, which are much less pronounced than what is often experienced in the arteries of the lower extremities. The inability to complete what should be straightforward access into the external carotid artery (ECA) through the micropuncture sheath or internal carotid artery (ICA) (after the arterial sheath of the NPS is placed) may be the only clue. If one finds it necessary to try multiple wires and catheters to traverse the area of stenosis, there is a high possibility that a dissection has occurred.

Even if distal true lumen access is established and a stent is successfully deployed, there can be a gap of untreated dissection between the caudal aspect of the stent and the access site. Angiographic diagnosis of this anatomy is difficult, leading to identification during postoperative surveillance.

The mechanisms of stroke from a carotid dissection include thrombosis, embolization, and static occlusion. The dynamics of flow reversal address all of these concerns. Flow reversal should be maintained until definitive repair is achieved. Given the inability to stent down to the access site, open repair is our preferred approach. Early recognition should avoid wire dissection distal to what is surgically accessible.

Although transfemoral CCA stenting is possible, it does require giving up flow reversal and potentially deploying an embolic protection device across a newly placed carotid stent, neither of which are desirable. Although we have performed deferred transfemoral CCA stenting to manage this scenario, we think it best to address the issue at the time of the original procedure. Endarterectomy and CCA to ICA bypass are both viable options, the suitability of which is best judged in the context of the status of the vessel.

PLAQUE PROLAPSE

Plaque prolapse consists of extrusion of thrombotic or atherosclerotic debris across the interstices of the stent (Figure 3). It is highly correlated with plaque morphology and, as such, potentially avoidable with careful patient selection. Intraluminal filling defects, described as a "flame sign" (Figure 4) and "life saver sign" (Figure 5) on preoperative CTA are both easily recognizable and indicative of a lesion with high potential for extrusion. Movement of the filling defect during intraprocedural

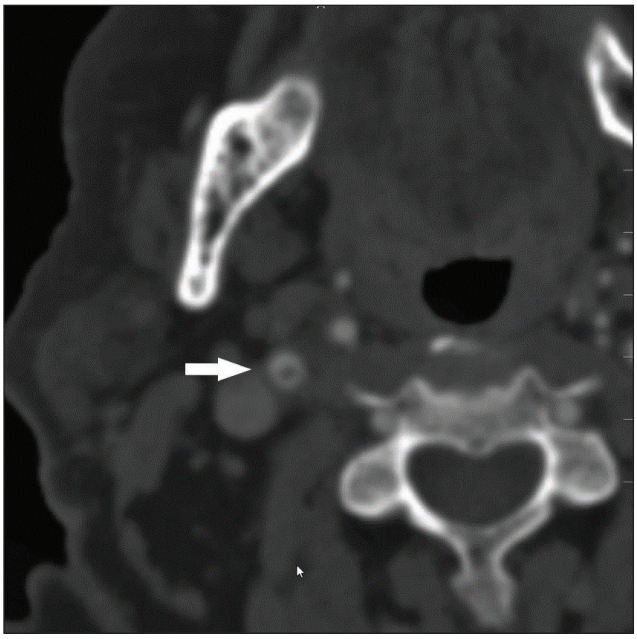

Figure 5. Life-Saver sign.

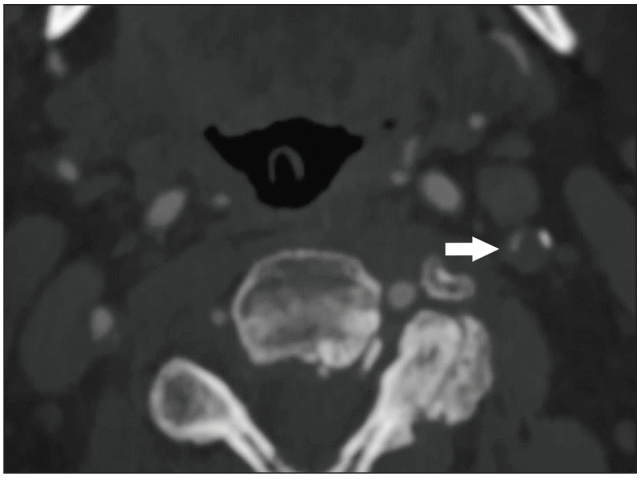

Figure 6. Swollen carotid sign.

angiography (mobile thrombus) is an ominous finding. A more subtle lesion morphology with a high risk of extrusion is the "swollen carotid sign" (Figure 6). This consists of the presence of soft, low-density plaque, often devoid of calcification, in a vessel that is noticeably larger than the contralateral counterpart (eg, 9 mm in diameter at the level of maximal disease compared with 4 mm contralaterally), without necessarily reaching aneurysmal dimensions.

If any of these described lesions are identified intraoperatively, or if plaque prolapse is noticed after stent deployment, preservation of flow reversal is the first step in management as these lesions are highly thrombogenic and carry elevated embolic potential.

Open repair, either via endarterectomy or bypass is recommended. Endovascular repair with layered bare-metal stents has been performed with good outcomes; however, there is the potential for extrusion of smaller emboli. The deployment of a self-expanding stent graft, such as the Viabahn (Gore & Associates) or Covera (BD Interventional) through the flow reversal system is an off-label application of these devices. This bailout maneuver has been successful, however, there is no evidence base to support it in this clinical scenario. There are also size constraints in terms of NPS sheath compatibility, as well as the potential for wire length issues as these are all over-the wire systems. Furthermore, these stent grafts lack the tapering capacity of bare-metal stents, leading to pleat formation. The failure to develop a neointima within these stent grafts generates concerns for ongoing platelet aggregation and thrombosis. In a manner analogous to what is seen in peripheral vessels, a covered stent graft may fail suddenly and catastrophically.

It should be stressed that the flow reversal system is not an embolectomy system. Therefore, attempts at balloon disruption or even pull-back embolectomy are to be discouraged. The debris may occlude the flow reversal sheath or become trapped between the sheath and the CCA, waiting to embolize once antegrade flow is restored.

CAROTID RUPTURE

The vessels most susceptible to carotic rupture are those that present heavy calcification, be it in a circumferential fashion (napkin ring sign, Figure 7) or with exophytic intraluminal projection (coral reef sign, Figure 8). These lesions often require high-pressure predilation and are associated with incomplete stent expansion leading to further high-pressure postdilatation. In the awake patient, acute pain during angioplasty should be considered a warning of possible rupture.

Once rupture has occurred (Figure 9), hemorrhage control is facilitated by the flow reversal system because it impedes antegrade flow into the area of injury and reduces the pressure within the carotid. Low-pressure inflation of a balloon sized to the intact ICA eliminates the main source of retrograde flow and pressure, leaving the ECA as the only bleeding branch. After the hemorrhage has been temporized, assessment and protection of the airway are critical steps for the awake patient. Furthermore, establishment of general anesthesia leads to a more expedient surgical exposure.

Definitive repair usually takes the form of CCA to ICA bypass. Given the circumstances, use of a prosthetic graft is usually most practical. Covered stenting of the lesion leads to the limitations of self-expanding stent grafts described previously. Furthermore, depending on plaque morphology, persistent failure of stent expansion may be

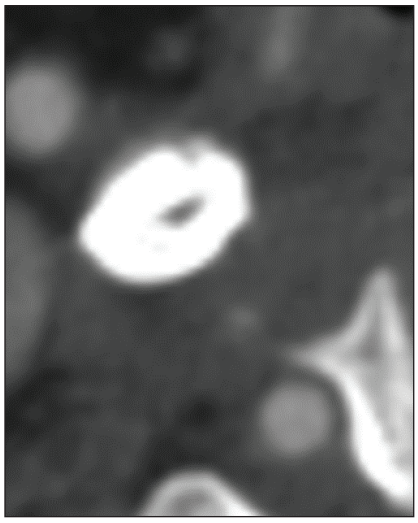

Figure 7. Napkin ring sign.

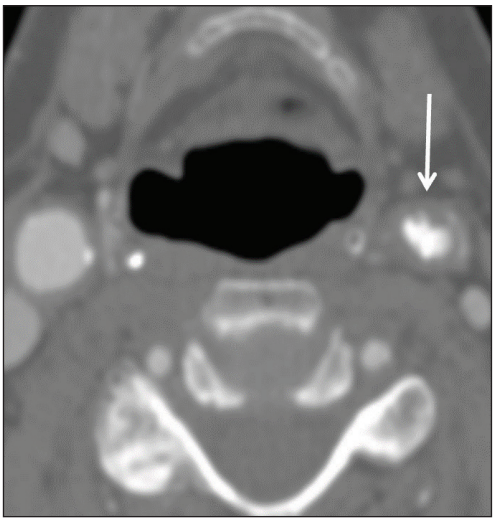

Figure 8. Coral reef sign.

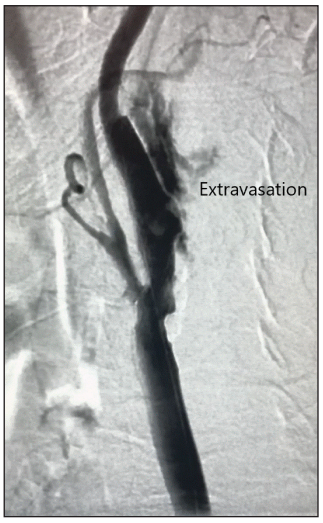

Figure 9. Carotid rupture with extravasation.

an issue. Even with successful bridging from intact CCA to ICA, covered stenting does not address bleeding from the ECA. The use of balloon-expandable stents or stent grafts is ill advised in the carotid arteries given the risk for extrinsic compression on neck movements.

Open repair has the added benefit of allowing hematoma evacuation. Ongoing airway compression is a concern after endovascular repair.

CONCLUSION

Although growing familiarity with TCAR may allow treatment of cases of higher complexity, familiarity alone does not overcome the anatomic limits of the technique; especially given that these scenarios occur with low frequency. Fortunately, in the event of complication, flow reversal provides a valuable bridge to definitive care. ■