

The Consequence of Unmanaged Hypotension After TCAR

BY ANGELA A. KOKKOSIS, MD, FACS, RPVI

A 71-year-old man presented to our institution for evaluation of left carotid stenosis noted on duplex imaging to be 70% to 99% and on CTA to be about 90% (Figure 1).

He had a complex medical history of hypertension, hyperlipidemia, antiphospholipid syndrome on lifelong warfarin (INR maintained > 3), right lower extremity deep venous thrombosis in 2003 with subsequent pulmonary embolism and resultant pulmonary hypertension, inferior vena cava filter placement and subsequent filter occlusion, and bone cancer (he underwent localized radiation and chemotherapy, with good prognosis).

In 2010, he suffered an ischemic stroke to his posterior intracranial circulation with visual changes and headaches. He reported residual partial peripheral vision loss to his left eye.

The patient also reported frequent vasovagal episodes, which was never fully elucidated. He was maintained

on 81 mg of aspirin and a statin in addition to his anticoagulation. He had never smoked cigarettes in his life.

TREATMENT OPTIONS

The patient was deemed high risk for left carotid endarterectomy based on his poor pulmonary status secondary to the significant pulmonary hypertension. There was concern for alterations in right ventricular preload or afterload induced by fluid shifts, medications, or changes in the autonomic nervous system, as can occur in open surgery under general anesthesia. Although carotid endarterectomy is not solely performed under general anesthesia and can be accomplished with a regional nerve block, other concerns, such as the long surgical procedure time, were taken into consideration. Ultimately, transcatheter carotid artery revascularization (TCAR) with local anesthesia and minimal sedation was offered to the patient.

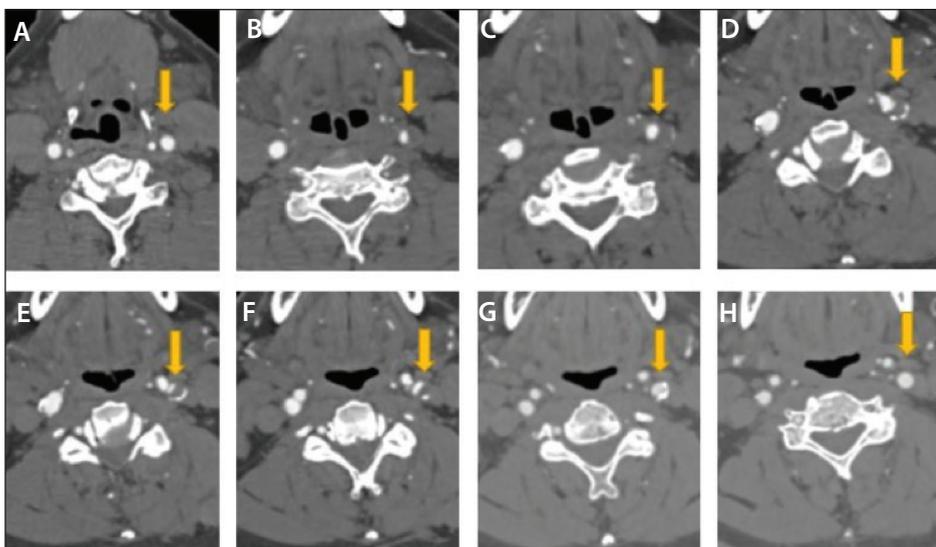


Figure 1. CTA depicting the left carotid stenosis (yellow arrows) from proximal (A) to distal (H). Of note, the right carotid and vertebral arteries were widely patent, and the left vertebral artery was hypoplastic. Not shown here is a hypoplastic posterior circulation secondary to the patient's previous cerebrovascular event.

MEDICAL MANAGEMENT

Aside from the pulmonary hypertension, the most treacherous component of this patient's perioperative management was the anticoagulation and antiplatelet therapy. Given his significant history for arterial and venous thrombotic events, there was concern for stent thrombosis. After discussion with the hematology and cardiology services, it was decided to maintain the patient on 81 mg of aspirin indefinitely. Standard of care for carotid stent placement includes dual antiplatelet therapy (DAPT), so he was

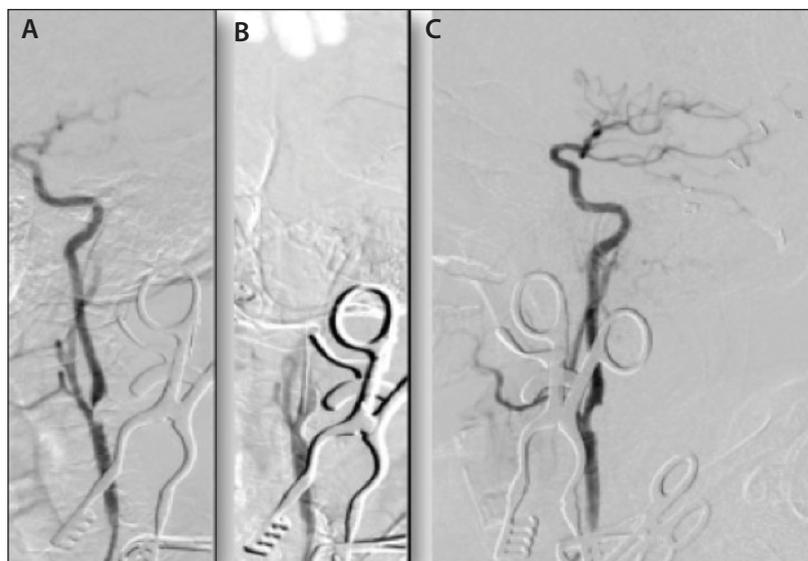


Figure 2. Intraoperative angiograms of the left carotid artery: pretreatment (A), after stenting (B), and after stenting with the intracranial circulation (C).

started on clopidogrel 1 week before surgery, with the intent to discontinue it 1 week after surgery to address the bleeding concern from DAPT and anticoagulation. The warfarin was bridged with enoxaparin 1 week prior to surgery, and the morning dose on the day of surgery was held. Thereafter, enoxaparin was resumed the evening after surgery, with the intent to restart warfarin within a couple days.

PROCEDURE

The patient received local anesthesia (1% lidocaine/0.25% bupivacaine) for the access site, and he was under mild conscious sedation for the carotid exposure before being wakened for the stenting.

The ENROUTE® Transcarotid Neuroprotection System (Silk Road Medical) was connected and placed on the high setting, as is the standard practice. The patient was

asked to squeeze a toy in his hand and answer questions, which he was able to do, confirming a maintained baseline neurologic status.

Several other intraoperative conditions were also confirmed prior to proceeding with stent placement: activated clotting time > 250 seconds after intraoperative administration of heparin, a systolic blood pressure (SBP) of 140 to 160 mm Hg, and adequate flow reversal once the common carotid artery was clamped to occlude the proximal inflow.

The patient was again asked to squeeze the toy and answer questions; however, he now could not perform these actions, demonstrating intolerance to the flow reversal. The carotid artery was unclamped, and after a few seconds, he regained full neurologic function. Based on the principle of “preconditioning,” the carotid was again clamped to test

the patient's tolerance. This time, he was able to squeeze the toy and answer questions. We then proceeded with the case.

The lesion was crossed with a 0.014-inch wire and predilated with a 5- X 20-mm balloon. In anticipation of hypotension with the balloon, atropine was preemptively administered. However, despite the atropine administration, the patient became bradycardic (heart rate dropped to the 40s) and hypotensive (60/40 mm Hg) for about 2 seconds. Another dose of atropine was administered, along with initiation of a phenylephrine drip. The patient's heart rate was normalized to the 70s, and his blood pressure returned to an SBP of 150 to 160 mm Hg.

In assessing the patient's neurologic status, he was unable to follow commands. His blood pressure increased to SBP > 160 mm Hg, and the 9- X 30-mm ENROUTE® Transcarotid Stent was quickly deployed.

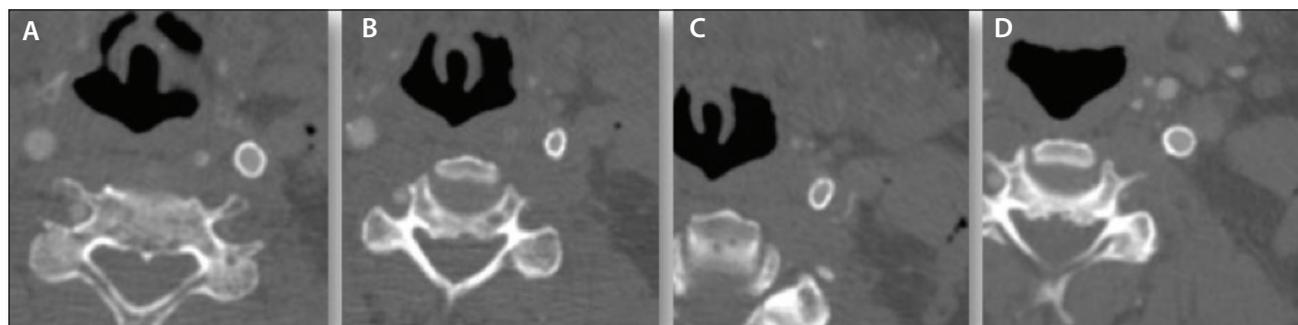


Figure 3. Postoperative CTA demonstrating stent patency from proximal (A) to distal (D).

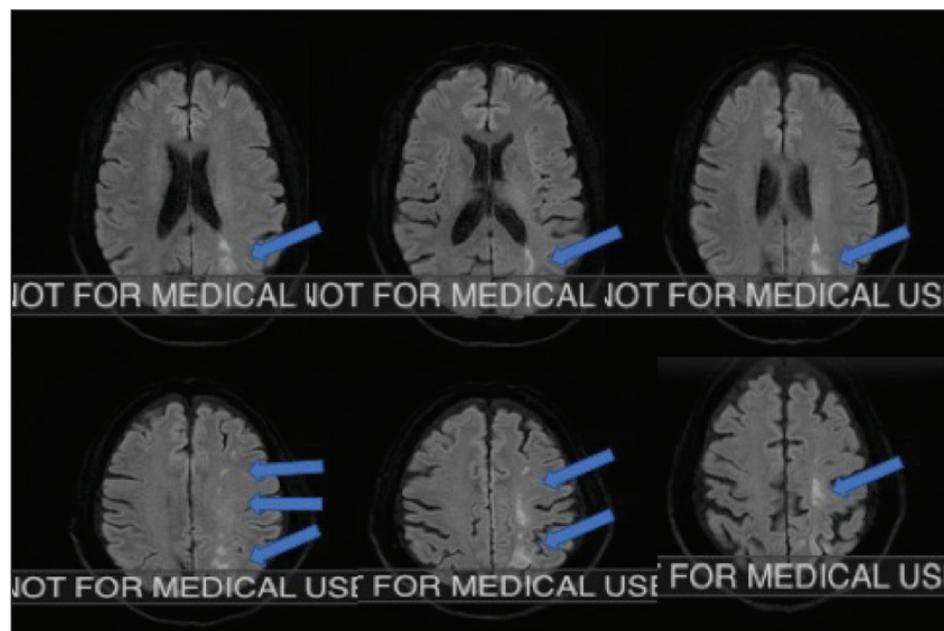


Figure 4. Diffusion-weighted MRI demonstrating the watershed infarct (blue arrows) in the left hemisphere.



Figure 5. Duplex ultrasound 1 year after TCAR demonstrating a patent left carotid stent on B-mode (A), color flow (B), and Doppler (C).

After stent deployment, a completion angiogram of the carotid stent in two views demonstrated brisk filling without any narrowing of the stent and no significant residual stenosis (Figure 2). An intracranial angiogram was also performed, demonstrating brisk filling of the left side of the brain with maintenance of the intracranial circulation and no evidence of distal embolization (Figure 2C).

The flow reversal system was then disconnected. The common carotid artery was unclamped. The sheath was removed from the common carotid artery, and the preplaced purse-string stitch was cinched to achieve hemostasis.

At this point, the patient's neurologic status returned completely to baseline (alert and oriented, full motor sensory function of upper and lower extremities, no dysarthria, and

no facial droop). The heparin was reversed with 30 units of protamine. He was then taken to the recovery room in stable condition.

RESULTS

Upon arrival to the recovery room, however, the patient demonstrated right hemiparesis and hemineglect while he was being connected to the monitors. His blood pressure dipped to an SBP of 60 mm Hg, and his phenylephrine dose was increased. Both neurology and neurosurgery services were consulted.

A quick bedside duplex ultrasound confirmed that the carotid stent was patent, so he was taken for a CTA (Figure 3) and CT perfusion scan. The CTA demonstrated patent intracranial vasculature and carotid stent. The results of the perfusion scan were normal as well.

With these negative studies, we proceeded to obtain a diffusion-weighted MRI (Figure 4), which demonstrated a left-sided watershed infarct. This finding was likely due to the

few seconds of hypotension with the carotid ballooning, the second hypotensive event in the recovery room, and the patient's baseline dependence of intracranial blood flow on the left carotid artery (with his chronically occluded left vertebral and hypoplastic posterior circulation on preoperative CTA).

The patient regained some neurologic function over the ensuing 2 to 3 hours, was maintained with permissive hypertension (SBP, 160–180 mm Hg), and as previously planned, enoxaparin was resumed that evening.

On follow-up, he regained motor sensory function of his right arm and leg, but sustained permanent loss of his peripheral vision bilaterally. At 1 year, his carotid stent was still widely patent (Figure 5).

DISCUSSION

As a result of this early experience, my practice has changed in the perioperative management of patients undergoing TCAR. It became exceedingly clear that hemodynamic management is vital to the treatment of these patients, and that this point needs to be understood by all parties involved in the patient's care (anesthesia, surgery, trainees, and nursing staff). Every second truly matters when cerebral function is at stake. Recently published data from the Vascular Quality Initiative (VQI) confirms that postoperative hypotension is significantly more common in stented patients as compared to carotid endarterectomy patients in whom hypertension is more common.¹

Instead of atropine administration, which becomes more of a reactive response rather than a protective treatment for hemodynamic changes, I now administer glycopyrrolate in every case once the carotid artery is surgically exposed. The duration of action is longer, and there are less hemodynamic swings. Typically, intracranial views are not recommended during the TCAR procedure due to the potential risk of showering emboli from a freshly angioplastied and stented lesion because the protective flow reversal is temporarily

stopped for the angiogram. However, in this case due to the intraoperative concern of a stroke event, intracranial views were performed.

Education to the whole perioperative team has been clarified to emphasize the high likelihood of hypotension after stenting (secondary to the radial force of the stent on the carotid baroreceptors), and the patient leaves the operating room with the phenylephrine drip and a portable monitor. ■

1. Schermerhorn ML, et al. In-hospital outcomes of transcatheter carotid artery revascularization and carotid endarterectomy in the Society for Vascular Surgery Vascular Quality Initiative. Published online June 18, 2019. *J Vasc Surg.* 2019.



Angela A. Kokkosis, MD, FACS, RPVI

Associate Professor of Surgery
Director of Carotid Interventions
Division of Vascular and Endovascular
Surgery

Stony Brook Medicine
Stony Brook, New York

*Disclosures: Consultant and faculty proctor
for Silk Road Medical.*