

Acute Thrombosed Carotid Stent Recanalization

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A 70-year-old man presented with new-onset right handed weakness, and MRI confirmed that he had an acute left temporoparietal infarct. He has a history of untreated bilateral frontal stroke with expressive aphasia and flat affect. Carotid ultrasound showed bilateral 80% to 99% carotid stenosis, and he underwent a left carotid endarterectomy without complication. The patient complained of dysphagia after the carotid endarterectomy, and workup revealed a squamous cell carcinoma of the right supraglottis requiring chemotherapy and radiation. Repeat duplex 6 months after the previous study showed increased right internal carotid artery (ICA) stenosis (peak systolic velocity, 451 cm/sec; end diastolic velocity, 124 cm/sec). Given the high-grade stenosis and recent cervical radiation, transcarotid artery revascularization (TCAR) was recommended.

PROCEDURE

Under general anesthesia, a right supraclavicular transverse incision was made, and the common carotid artery (CCA) was exposed; 100 units/kg intravenous heparin was administered for an activated clotting time > 250 seconds. Left common femoral vein access was achieved, and an 8-F venous sheath was inserted. The CCA was accessed with a micropuncture system, and an angiogram confirmed a high-grade stenosis. An 8-F arterial sheath was inserted over a J-wire. Flow reversal was established with the ENROUTE® Transcarotid Neuroprotection System (Silk Road Medical), and the CCA was occluded with a vessel tie. The lesion was crossed with a 0.014-inch wire without significant difficulty. A 3- X 20-mm Sterling angioplasty balloon (Boston Scientific Corporation) was used to predilate the lesion. A 9- X 40-mm ENROUTE® Transcarotid Stent (Silk Road Medical) was placed across the lesion. The stent was postdilated with a 5- X 40-mm Sterling angioplasty balloon. Angiography showed a patent stent with a patent CCA, external carotid, and distal ICA into the cerebral circulation (Figure 1). The total flow reversal time was 10 minutes. Protamine was administered for full heparin reversal.

The patient was slow to arouse, unable to follow major commands, and not moving the left upper or left lower extremity. Immediate CTA showed acute occlusion of the ICA stent without intracranial bleed. On axial imaging, the stent was noted to be compressed into an ovoid shape (Figure 2). The patient emergently returned to the operating room and the previous incision was reentered. Digital subtraction angiography was suspicious for a stent fracture (Figure 3). A Bentson wire was passed into the external carotid artery and a 6-F sheath was placed in the CCA. Arteriography demonstrated thrombus burden within the stent but cross-filling through the external carotid artery into the distal ICA. Next, antegrade flow was occluded with vessel tie of the CCA. Abciximab (4 mg), tissue plasminogen activator (4 mg),

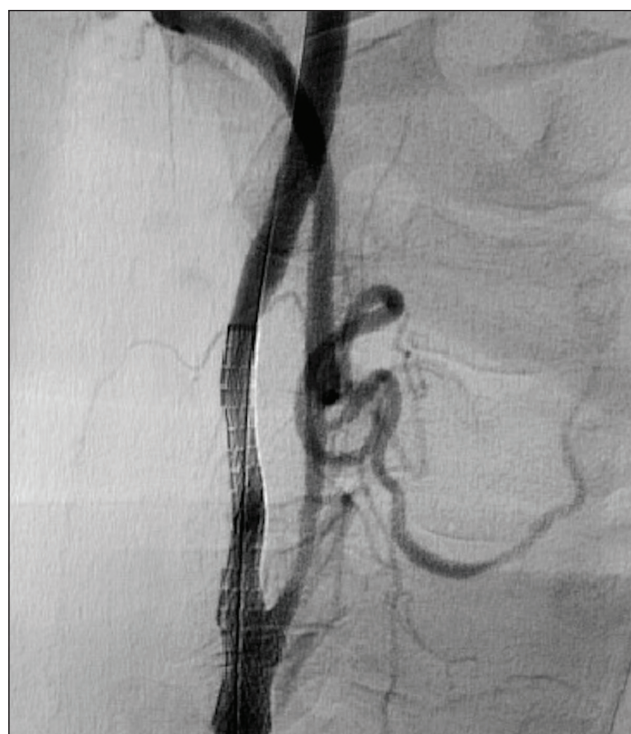


Figure 1. Poststenting angiogram showed a patent stent, CCA, external carotid, and distal ICA into the cerebral circulation.

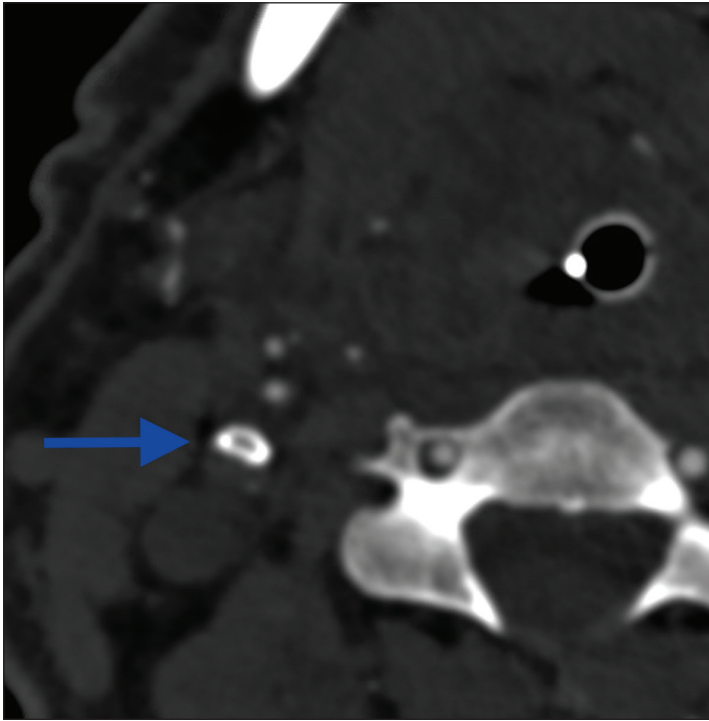


Figure 2. Axial imaging showed the stent compressed into an ovoid shape.

and verapamil (10 mg) were administered slowly through the sheath and allowed to dwell for 5 minutes. Angiography initially demonstrated large air bubbles and a stagnant column of blood in the proximal ICA. This was immediately evacuated via a 20-mL syringe applied to the sheath. Repeat angiography showed a now-patent stent with flow into the distal ICA. Due to the potential stent fracture, the previous stent was relined with a 6- X 40-mm Zilver PTX drug-eluting stent (Cook Medical) and postdilated with a 5- X 40-mm angioplasty balloon. Completion angiogram showed a patent stent and distal ICA without notable kink or defect (Figure 4).

RESULTS

The patient awoke from general anesthesia and could move all extremities and follow commands. Repeat CT scan showed no new infarcts. The patient was back to his baseline by the morning of postoperative day 1. At 6-month follow-up, repeat CTA showed a widely patent stent without kinks or deficits (Figure 5).

DISCUSSION

The etiology of acute stent thrombosis in this case is unknown. There is radiographic evidence as shown for possible stent fracture (Figure 3), incomplete stent apposition (Figure 2), and possible failure to exclude the entire lesion (Figure 1). With thrombolysis and vasodilation, mechanical aspiration, and relining the lesion with an additional stent to extend proximally as well as further aggressive post dilation yielded successful radiographic and clinical outcomes.

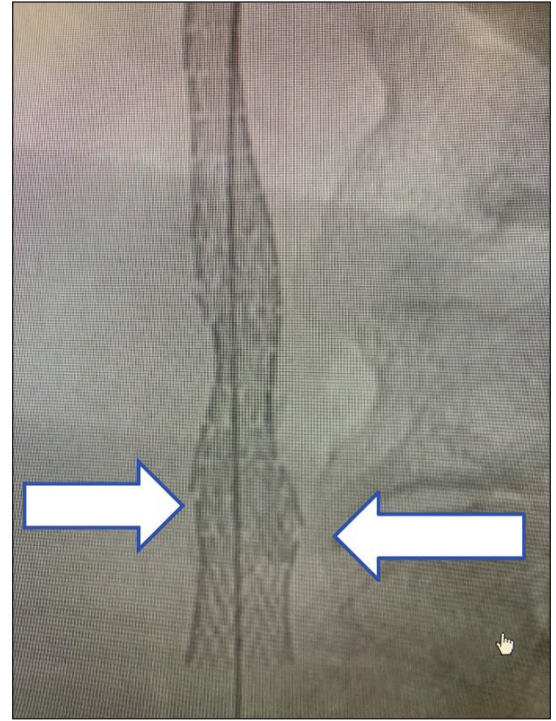


Figure 3. Digital subtraction angiography showed a potential stent fracture.



Figure 4. Completion angiogram showed a patent stent and distal ICA without notable kink or defect.



Figure 5. CTA at 6-month follow-up showed a widely patent stent without kinks or deficits.

There have been numerous other TCAR procedures performed since without any complications. This case represents one of the earlier experiences. A major change in protocol after this experience has been aggressive predilation of the lesion prior to stent placement. This step allows for improved stent deployment, apposition, and prevention of any kink or stent fracture. ■



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Disclosures: None.



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Disclosures: None.