Acute Embolic Event as a Result of CAS

With the increasing popularity of catheter-based therapy for carotid artery stenosis it is important to recognize and treat neurologic complications when they occur.

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A 64-year-old, right-handed man with underlying ischemic cardiomyopathy (LVEF, ~25%), and hypertensive nephropathy (baseline Cr, ~1.7) was referred to our institution after he had undergone evaluation of his recurrent right hemispheric TIA; he was found to have severe bilateral carotid stenosis as determined by carotid ultrasound. The patient underwent concurrent cardiac catheterization and carotid angiography. The cardiac catheterization showed severe and diffuse coronary artery disease that would be best managed by medications. Carotid angiography showed severe bilateral internal carotid stenosis exceeding 95% on both sides. It was believed that the patient's underlying heart disease made him a poor surgical candidate; therefore, carotid angioplasty and stenting (CAS) was offered and accepted by the patient. The patient underwent CAS on the right side without postoperative complications. He was discharged the next day with his cardiac medications, as well as a regimen of aspirin (81 mg/d) and clopidogrel (75 mg/d). The patient returned 1 month later to undergo elective angioplasty and stenting of the left internal carotid artery.

The patient was taken to the cardiac cath lab, and the left carotid artery was engaged with an 8-F, JR 4 (Cordis Corporation, a Johnson & Johnson company, Miami, FL) without difficulty from the right groin (Figure 1). The 7-mm AngioGuard (Cordis Corporation) was placed distally from the lesion after 3,000 units of heparin were administered. The AngioGuard device was deployed, and the lesion was predilated with a 5-mm X 20-mm

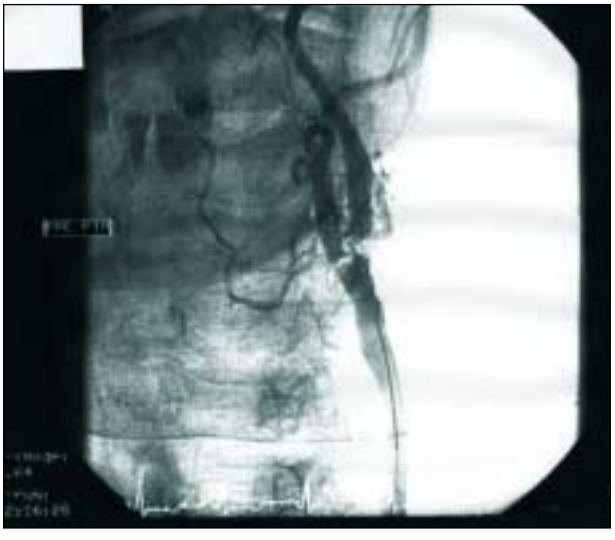

Figure 1. This pre-PTA picture shows severe complex plaque involving the distal left common carotid artery extending into both the internal and external carotid origins. Stenosis exceeds 95% in each vessel. The lesion appears typically atherosclerotic.

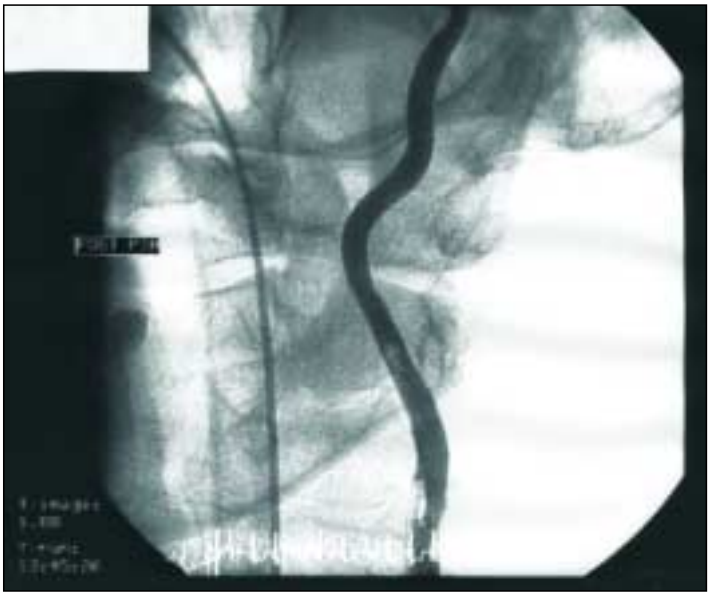

Figure 2. After PTA, the internal carotid is open, but there has been a plaque shift resulting in an occlusion of the external carotid artery. There is also an intraluminal filling defect not seen previously, 3 cm beyond the internal carotid origin.

balloon for 8 seconds. The patient had a brief episode of bradycardia during the balloon inflation when his heart rate was 40 beats per minute. This rapidly resolved with balloon deflation and the administration of 0.4 mg of atropine. The 10-mm X 20-mm, self-expanding stent was deployed without an episode of bradycardia. The patient was alert, awake, and able to use his right arm and hand; however, soon after the stent was deployed, he became aphasic with a hypertensive response (BP 226/104), tachycardia (HR 106). He experienced left gaze deviation and immediate right hemiplegia. Injection of the left carotid artery showed sluggish flow, and neither the internal nor the external carotid artery filled. The patient required immediate intubation for airway protection because of rapidly progressive obtundation.

The patient was intubated for airway protection and was loaded with 25 g of albumin and 1,000 mL of 0.9% normal saline after the central line was placed through the right femoral vein. At that time, it was believed that the stagnation could be due to either air emboli or thrombosis. t-PA was not administered because of the possibility that the defect was an air embolus. The previously used 5-mm balloon was reinserted and inflated in the distal common carotid, the proximal internal carotid, and just beyond to evaluate whether any residual stenosis was responsible for the flow defect. No defects were noted; therefore, sodium nitroprusside 0.2 mg was administered directly into the carotid artery. Significant improvement in flow and outflow in both the internal and external carotids was immediately present. Intracranial flow was also brisk.

Final angiography demonstrated multiple filling defects in the cervical portion of the left internal carotid and in the bifurcation of the left middle cerebral artery (Figure 2). Eptifibatide was then given per the standard dose. The patient's neurologic deficit was rapidly resolved to normal after flow was established. He was then transferred to the coronary care unit and was extubated an hour later without any complications. Warfarin therapy was initiated the same night and continued with fractionated heparin overlap (Lovenox, Aventis Pharmaceuticals Inc., Bridgewater, NJ) until the INR was in a therapeutic range at 2.5. The following day, the patient had TIA symptoms, and a noncontrast CT was ordered. The study was normal. He was sent home on the fifth hospital day on warfarin, aspirin, and clopidogrel. He will be followed by his primary care physician with a target INR of 2.5.

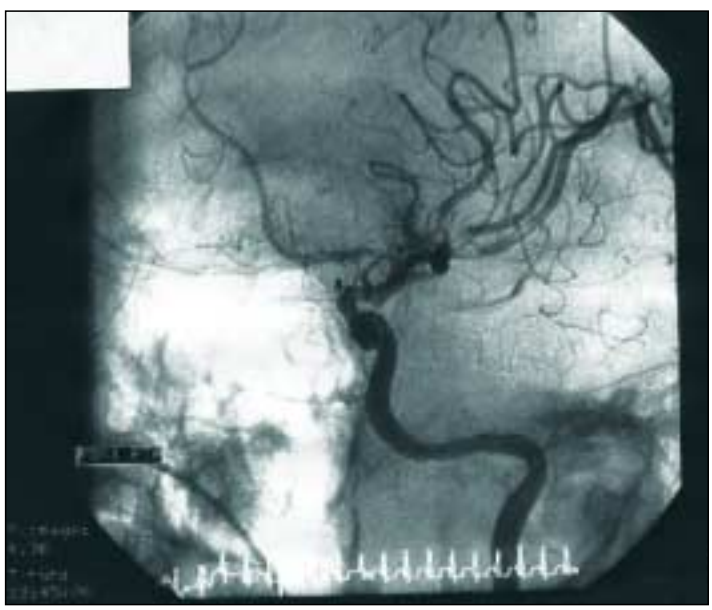

Figure 3. This post-PTA lateral intracranial view shows multiple filling defects involving the angular artery, the posterior temporal, and the posterior parietal arteries. These findings are consistent with multiple emboli.

DISCUSSION

The case presents some interesting issues that are worthy of discussion. First, the initial angiogram provides a clue that the original lesion contains thrombus. Whether or not this was consistent with a plaque hemorrhage will not be known; however, the clinical presentation was not one that suggested this. We know of at least two other cases that were suspicious for plaque hemorrhage, and both were treated with warfarin for 1 month prior to the carotid intervention. Each case responded quite well to catheter-based therapy and was subsequently treated with clopidogrel in the long term.

The initial lesion in this case was certainly complex, and had there been any doubt initially that clot was present, we would have not recommended the procedure without first treating conservatively for 1 month using warfarin at full dose anticoagulation to permit fibrinolysis from the patient's own intrinsic fibrinolytic pathway.

The next issue is the decision to give sodium nitroprusside. This potent arterial vasodilator is frequently in the armamentarium of cardiac pharmacology, but not frequently given as an acute intra-arterial bolus injection. Given the extremely low resistance of the internal carotid artery, it seems that the drug in this case was capable of profound intracranial vasodilation without any significant effect on the patient's systemic blood pressure. The initial thought in this case had been to give nitroglycerin, but because of the patient's low cardiac output (baseline ejection fraction, <30%) and chronic low blood pressure (90/65) we were very reluctant to use it. The effect of the nitroprusside was to permit nearly complete washout of the obstructing emboli as evidenced by this patient's near complete instantaneous recovery.

The most valuable point in this case is one of a multidisciplinary approach to treating carotid stenosis with catheter-based therapy. In this case, an interventional neurointensivist collaborated with an interventional cardiologist to complete a very successful outcome. ■

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