Case Reports

Carotid Stent Thrombosis
Report of 2 Fatal Cases

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Background—Carotid stenting is receiving increased attention as an alternative to carotid endarterectomy. Carotid stent thrombosis is a rare complication of this procedure.

Case Descriptions—We present 2 case reports from a university medical center. Two patients had angiographically confirmed carotid stent (CS) thrombosis with subsequent fatal strokes. The first was a 63-year-old woman who developed stent thrombosis after the procedure was performed for a postendarterectomy intimal flap. The second patient was a 57-year-old man who underwent CS placement for transient ischemic attacks and developed internal carotid artery occlusion at the level of the stent 3 days later. Neither patient was treated with combination antiplatelet therapy before or after CS placement. Both patients died.

Conclusions—CS thrombosis can be a cause of fatal iatrogenic stroke. Combination antiplatelet therapy should be administered to decrease the incidence of this complication. (Stroke. 2001;32:2700-2702.)

Key Words: carotid artery ■ stents ■ stroke, ischemic ■ thrombosis

Carotid stenting (CS) has become an alternative to carotid endarterectomy (CE) for select patients in the last decade.1 One of the early concerns with angioplasty of the proximal internal carotid artery (ICA) was the possible production of distal emboli and subsequent ischemic stroke. There are several possible mechanisms for stroke development in the periprocedural period. These include distal embolization at the time of the procedure, delayed embolization due to dissection, and hyperperfusion syndrome.2–4

We describe 2 patients with fatal stroke due to a novel mechanism, carotid stent thrombosis. Both of the cases were documented angiographically.

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Case 1
The patient was a 63-year-old woman who underwent left CE due to severe, symptomatic stenosis. She received aspirin preoperatively as part of the Aspirin and Carotid Endarterectomy (ACE) trial (dose range 81 mg to 1300 mg). She had experienced a left posterior cerebral artery stroke 8 years previously.

Following surgery, the patient developed a right hemiparesis. An angiogram performed 11 days postoperatively revealed an intimal flap at the operative site. A stent was placed at the site of the intimal flap, but thrombus was noted during the procedure (Figure 1). The thrombus was resistant to treatment with 750 000 U urokinase. Aspirin was discontinued. Approximately 12 hours later, the patient became poorly responsive, and right hemiplegia and global aphasia were noted. CT of the head revealed a large, evolving left middle cerebral artery (MCA) infarct. She died shortly thereafter from massive hemorrhagic conversion.

Case 2
The patient was a 57-year-old man with a history of hypertension, diabetes mellitus, coronary artery disease, congestive heart failure (CHF), and morbid obesity. He presented with episodes of transient visual loss in the left eye. He was placed on intravenous heparin initially. A cerebral angiogram revealed severe stenosis at the origin of the left ICA (Figure 2). Heparin was resumed after the angiogram. Initial consideration was given to possible CE, but because of his obesity and CHF, CS was performed instead. The stent was placed uneventfully, with excellent reconstruction of the proximal ICA postprocedurally (Figure 3). No antiplatelet agents were administered before the procedure.

Heparin was resumed after the procedure, although it was subtherapeutic at several time points. Two days after the procedure, the partial thromboplastin time was 32.7 seconds. The patient developed right-sided weakness 3 days after the procedure. He was started on aspirin after a stat angiogram showed carotid occlusion due to stent thrombosis (Figure 4). Treatment with thrombolysis was not felt to be indicated, and consideration was not given to the use of a IIb/IIIa inhibitor. There was no documented cardiac arrhythmia. Six days after carotid stent placement, the patient had a flaccid right hemiplegia and was minimally responsive. CT revealed a large left MCA territory infarct with developing mass effect (Figure 5). He died 11 days after stent placement.
Discussion

To the best of our knowledge, these patients are the first reported cases of angiographically documented carotid stent thrombosis with subsequent fatal stroke. Both patients had large hemispheric strokes and died shortly thereafter.

Because these patients had poor outcomes, it is important to review aspects of their treatment that may have been associated with the stent thrombosis. One possible factor that may be linked with the occurrence of stent thrombosis is the periprocedural antithrombotic medication. In the coronary circulation, initially aspirin and heparin followed by oral warfarin were commonly used after stent placement. However, acute and subacute coronary stent thrombosis still occurred in approximately 3.5% of patients. Subsequent clinical trials showed that combination antiplatelet therapy with aspirin and ticlopidine was superior to treatment with heparin followed by warfarin for prevention of coronary stent thrombosis. In a study of 1965 patients who received coronary stents and then were randomly assigned to treatment with aspirin alone, aspirin and warfarin, or aspirin and ticlopidine, a statistically significant benefit of aspirin and ticlopidine was shown, with the rate of angiographically evident thrombosis being 0.5%. Animal studies have also shown that heparin is relatively ineffective at preventing coronary stent thrombosis. With the currently used approach of 2 antiplatelet agents, acute and subacute stent thrombosis is relatively uncommon in the coronary vessels. In a retrospective study of 1620 patients who had undergone coronary...
stenting, early reintervention was necessary in 26 patients (1.6%), with half of these cases due to stent thrombosis.7

Recent data has also found that aspirin and clopidogrel can be used for prevention of coronary stent thrombosis and that there may be fewer adverse events than with the combination of aspirin and ticlopidine.8 In a series of 283 patients with coronary stents who were pretreated with aspirin and clopidogrel, the 1-month incidence of stent thrombosis was 1.4%. Estimates regarding the incidence of carotid stent thrombosis are imprecise, but it appears to be a relatively rare phenomenon. The Lenox Hill carotid stenting team reported that 18 of 368 patients (4.9%) had early stent thrombosis.9 Similarly, a recent review of 5210 CS procedures from 36 international centers did not mention stent thrombosis as a cause of procedure-related death.10

With regard to antithrombotic therapy, our first patient did receive aspirin alone in the week before stent insertion, although the dose was not known because of the double-blind nature of the ACE trial. The second patient was treated with intravenous heparin only, and it was not consistently in the therapeutic range. Tong et al11 recently described a patient with acute carotid occlusion due to stent thrombosis, in whom the vessel was recanalized after administration of the glycoprotein IIb/IIIa inhibitor abciximab. Their patient, like our 2 cases, was not pretreated with combination antplatelet therapy, suggesting the importance of combined antplatelet treatment. Neither of our 2 patients was treated with abciximab or another glycoprotein IIb/IIIa inhibitor.

Combination antplatelet therapy is being increasingly used in CS protocols. In the upcoming Carotid Revascularization Endarterectomy versus Stenting Trial (CREST), for example, the protocol mandates pretreatment with aspirin (325 mg/d) and clopidogrel (75 mg/d) in patients randomized to the stenting arm (R. Hobson, MD, unpublished data, 2001). The clopidogrel is to be started 2 to 4 days before the stent insertion (or a 450-mg loading dose can be used) and continued for 30 days after the procedure.

In summary, we describe 2 patients with large, fatal MCA strokes following carotid stent deployment. Neither patient was treated with combination antplatelet therapy before the procedure. Meticulous attention to the antplatelet regimen is important, and intravenous heparin alone appears to be insufficient to prevent stent thrombosis.

References

Figure 5. Large left MCA infarct.