Carotid endarterectomy performed after progressive carotid stenosis following angioplasty and stent placement

Case report

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Carotid endarterectomy (CEA) is the treatment of choice for asymptomatic and symptomatic disease causing greater than 60% internal carotid artery (ICA) stenosis. Recently, percutaneous transluminal angioplasty (PTA) with stent placement has been investigated as a therapeutic option for the treatment of ICA stenosis. In this report the authors document CEA performed after PTA with stent placement and describe the pathological findings.

A standard CEA was performed. The surgical intervention was more difficult secondary to the following variables: the length of the exposure necessary to dissect out the metallic stent, the difficulty with opening and cutting the artery, and the care required to remove the stent to avoid vessel wall perforation. Pathological examination of the specimen demonstrated classic atherosclerotic changes revealing persistence of native disease. The metallic stent was embedded within the plaque. Many questions remain unanswered regarding the physiological and biological changes that occur in the carotid vessel wall after PTA with stent placement. It is concluded that CEA of a stent-containing carotid artery is feasible and should be considered as an alternative when recurrent stenosis occurs after PTA.

Key Words • angioplasty • carotid artery • carotid endarterectomy • stent • pathology

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Case Report

History. This 55-year-old left-handed woman with insulin-dependent diabetes mellitus developed symptoms of a left hemispheric transient ischemic attack in April 1995. Evaluation at another institution revealed a high-grade stenosis in the left ICA and probable right ICA occlusion. The patient underwent a left CEA with no complications. She was later referred to UAB for further evaluation and treatment of the presumed right ICA occlusion. It was determined by means of high-resolution angiography that the right ICA was not completely occluded, but severe stenosis (almost 99%) was present and allowed only trickle flow distally (Fig. 1 left). The right anterior cerebral circulation was supplied primarily via the vertebrobasilar system through a patent right posterior communicating artery. There was an associated 50 to 60% stenosis of the right external carotid artery (ECA). Injection of contrast material into the left carotid artery demonstrated satisfactory caliber after endarterectomy with 60% short segment stenosis proximal to the surgical site (left common carotid artery).

Stent Placement. The patient was enrolled in the UAB carotid angioplasty trial after PTA of the right ICA in May 1995. To prevent arterial collapse and/or subintimal flaps, stainless steel stents were placed within the right ICA and ECA. Two 20 × 4-mm cardiostent stents (Cook Inc., Bloomington, IN) were used in the right ICA. To prevent forced occlusion of the ECA caused by compression from the dilation of the ICA, a third 20 × 4-mm stent was placed in the right ECA. This type of stent needs to be
dilated, and this was achieved by means of a 20 × 4-mm high-pressure balloon. The final angiographic results demonstrated less than 15% residual stenosis (Fig. 1 center). The procedure was complicated by transient hypotension and bradycardia that required a temporary procedural pacemaker.

Postoperative Course. The patient was discharged home in stable condition after 2 days. A follow-up Doppler ultrasound study of the carotid artery obtained 6 months postsurgery demonstrated a 60 to 79% restenosis of the right ICA. A carotid angiogram revealed a 50% restenosis of the right ICA within the area of stent placement (Fig. 1 right). The right ECA demonstrated severe proximal stenosis (>90%). The left ICA exhibited satisfactory vessel diameter in the postendarterectomy segment. Proximal focal narrowing of the left ICA (approximately 70%) was again demonstrated.

Operation. Six months after the original PTA, a CEA was performed via a standard approach. Gross inspection of the ICA revealed a nonpulsatile artery along the span of the stent, and the length of the stent was easily recognized by visualization and palpation. The arteriotomy was difficult because of the enclosed stent, but could be performed with standard surgical instruments. The stent and the artery were cut simultaneously. The arterial opening was made unusually long and involved the ECA to assure total stent removal. The plaque and stent were removed en bloc. The artery was closed in a routine fashion and the patient was discharged home 3 days later in good health. Doppler studies performed 6 weeks postoperatively revealed no recurrent stenosis or other abnormality.

Pathological Examination. Gross inspection of the surgical specimen showed a cylindrical segment of yellow plaquielike material, with the metallic stent embedded within the plaque. On sectioning, it appeared that the plaque material was growing in and around the metallic stent. The lumen of the specimen was very small throughout. The deeper layers of the plaque contained some reorganized thrombi. A larger percentage of the plaque was composed of atheromatous debris than fibrous proliferation, and diffuse dystrophic calcifications were present. The atheroma contained numerous cholesterol clefts (Fig. 3) and occasional foreign body giant cells reacting to the cholesterol. Numerous hemosiderin-filled and foamy macrophages were present and neovascularized endothelium-lined capillaries were frequently seen. A fragmented internal elastic lamina was identified in a portion of the specimen. The luminal side of the plaque displayed a smooth endothelial surface. Marked intimal hyperplasia was present throughout the specimen, but was more pronounced near the central portion of the debrided material.

Discussion

Restenosis in the coronary and peripheral circulation has been reported as a common problem after angioplasty and stent placement. Recurrence rates for stenosis in
groups of patients with longer than 12 months of follow-up review range between 25% and 50%.\textsuperscript{4,6,13,15} Recurrence in the carotid artery territory could also represent a potential problem requiring long-term follow-up. Alternatives such as repeated angioplasty and/or surgical intervention are available to deal with this potential problem. Surgical experience with removal of peripheral circulation stents has been documented previously.\textsuperscript{7} To our knowledge, this represents the first report of removal of a carotid stent during CEA. The fact that the vessel could be opened and the stent removed in its entirety is an important surgical observation. The normal cleavage plane between the vessel wall and the atheroma was present in spite of the stent (the pathology specimen corroborated that the plane of cleavage was preserved). There were, however, several areas where the stent had caused visible indentations and vessel wall thinning. In these areas the coils had lain against the vascular layers, causing what appeared to be pressure changes, but no vessel penetration was identified. The surgical procedure was clearly more challenging because of: 1) the length of the exposure necessary to dissect out the entire stent; 2) the difficulty involved in opening the operative field and cutting the artery caused by the metallic stent; 3) the care that was required during removal of the stent to avoid vessel wall penetration because of the vessel wall thinning from the stent coils; and 4) the need to perform a CEA of the external carotid artery with stent removal.

The in vitro observation of the stent and the pathological findings are also unique. At the time of surgery, the stent could be easily visualized through the vessel wall. The nonpulsatile nature of the vessel wall may in some way play a very important role in the escalated progression of the residual atheroma. The pathological examination of this specimen revealed a combination of atheromatous debris and intimal hyperplasia in the luminal area. Although the percentage of intimal hyperplasia in the specimen was less than that of the atheromatous debris, it represents more than is generally seen in plaque material. This is likely to be the cause of the rapid reaccumulation of plaque over a 6-month period and is thought to be the usual cause of restenosis after CEA.\textsuperscript{2} Intimal hyperplasia is a nonspecific response to injury regardless of the device used and accounts for approximately 80% of cases of restenosis in the coronary circulation.\textsuperscript{15} Angiographic studies in this case showed that intimal hyperplasia appeared to occur with a more rapid onset when compared with disease in the contralateral carotid artery that had undergone endarterectomy. Examination of the pathological specimen corroborated that the plaque was primarily confined to the intima as previously reported.\textsuperscript{10} The fact that the stent was located in the midsection of the plaque indicates that the stent did little to alter the atheroma and its formation. Although this hypothesis is unproven, plaque persistence may signify the potential for future embolic symptoms or act as a stimulus for intimal hyperplasia formation.

It remains unclear why the plaque recurred within a 6-month period. Of the various risk factors for atherosclerosis, four are considered of prime importance: hyperlipidemia; hypertension; cigarette smoking; and diabetes. In this patient, only diabetes was present as a risk factor for recurrent carotid stenosis, but recent evidence suggests that restenosis may not be related to this disease.\textsuperscript{3} Many questions arise as to the physiological and biological changes that occur in the vessel wall after angioplasty and stent placement.\textsuperscript{16} Is pulsatile flow important to prevent further plaque formation? Is the presence of plaque a mediator of a more advanced atheromatous change? Does luminal dimension (and therefore improved flow) caused by PTA prevent restenosis? Recent experimental studies in the femoral and iliac arteries have demonstrated that intravascular stents in normal arteries are associated with a certain degree of thrombogenicity and formation of neointimal hyperplasia.\textsuperscript{10,12} The deposition of platelets and neutrophils at the site of the stent was significantly increased when compared with the site where angioplasty alone was performed.\textsuperscript{16} We emphasize the need for: 1) an understanding of the causes of carotid
plaque and the effect of stents on it; 2) shorter and removable stents (so that if and when surgical intervention takes place, unusual exposures or techniques are not required); and 3) skilled placement of these stents (within the reach of surgical approaches) to avoid future complications. Only with further investigation and study can these problems be addressed.

In our case, angioplasty with stent placement did not seem to result in vessel wall indentations or thinning sufficient to disallow safe resection of the plaque if recurrent stenosis develops. We conclude that CEA is feasible in this setting and should not involve special pre- and postoperative care. Further testing with newer self-expanding stents may be required to confirm their resectability, because they may be more difficult to remove without vessel wall disruption.

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References

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