Etiologic Importance of the Intimal Flap of the External Carotid Artery in the Development of Postcarotid Endarterectomy Stroke

Wesley S. Moore, MD, Jeannette Y. Martello, MD, William J. Quiñones-Baldrich, MD, and Samuel S. Ahn, MD

A technically unsatisfactory end point (transition from the removed diseased plaque to normal distal intima) leading to an intimal flap of the external carotid artery has been identified as a source of perioperative stroke. The mechanism involves thrombus formation with retrograde propagation of the thrombus and subsequent embolization of the internal carotid artery. This report describes three cases illustrating this mechanism and methods of identification and correction. This mechanism of postoperative stroke adds further justification for the routine use of intraoperative surveillance studies to document the technical result of endarterectomy involving the internal and external carotid arteries. When an unsatisfactory end point is identified in the external carotid artery, it should be corrected with the same sense of concern as a similar finding in the internal carotid artery. (Stroke 1990;21:1497-1502)

The external carotid artery has been recognized as a significant source of collateral blood flow after occlusion of the internal carotid artery. However, when the internal carotid artery is patent, it generally has been assumed that the external carotid artery is of superfluous importance with respect to cerebral circulation. In this communication, we report our experience with three patients in whom an intimal flap of the external carotid artery, uncorrected at the time of carotid bifurcation endarterectomy, led to a postoperative neurological complication secondary to thromboembolic propagation into a patent internal carotid artery. The need to recognize the importance of this phenomenon as an etiologic mechanism in the development of perioperative stroke justifies the use of intraoperative imaging such as completion angiography.

Case Reports

This study is based on an analysis of three cases that presented to the Division of Vascular Surgery at the University of California at Los Angeles (UCLA) Center for Health Sciences between 1985 and 1988. These three cases document the etiologic importance of an intimal flap in the external carotid artery leading to retrograde propagation of thrombus with subsequent embolization via the internal carotid artery. The fourth case is described as an example of how an intimal flap of the external carotid artery was appropriately identified by completion angiography and corrected at the time of operation, resulting in the prevention of possible untoward sequelae.

Case 1

A 65-year-old right-handed black woman presented to the Vascular Surgery Clinic at UCLA November 25, 1985. She described a history of left hemisphere transient ischemic attacks manifested by episodes of expressive dysphasia and right-hand clumsiness. Previously, in April 1984, she suffered a left hemisphere stroke.

On physical examination, the patient had normal quality carotid pulses with a left carotid bruit. A neurological examination demonstrated decreased motor strength of the right hand and atrophy of the interosseous muscles. There was moderate spasticity of the right upper extremity with hyperreflexia. There was mild graphesthesia and some compromise in stereognosis and two-point discrimination of the right hand.

Noninvasive evaluation was performed with an ophthalmopneumoplethysmography (OPG) and a duplex scan. The OPG was markedly abnormal,
lateraling to the left side. Duplex scan demonstrated a 90% stenosis of the left carotid bulb with clear patency of the distal internal carotid artery. Carotid angiography was not performed because of a stated allergy to iodinated contrast material.

Based on the patient's history and noninvasive assessment, a left carotid bifurcation endarterectomy was performed on December 6, 1985. Intraoperative brain map monitoring was used, and no abnormality was noted. Because of the patient's sensitivity to iodinated contrast material, a completion angiogram was not performed. Perioperatively, she was maintained on dextran-40. Seven hours postoperatively, the patient complained of two episodes of amaurosis fugax of the left eye, and physical examination showed her to have an increase in her right hemiparesis. She was given an intravenous bolus of heparin and immediately was returned to the operating room. The artery was reexplored, and the patient was given an intravenous dose of hydrocortisone followed by an intraoperative angiogram. This demonstrated a normal left internal carotid artery. However, there was an acute near-total occlusion of the external carotid artery due to a presumed intimal flap. The arteriotomy was reopened, and a 1.5-cm-long thrombus, propagating back from the intimal flap toward the internal carotid artery, was removed. The intimal flap was identified and extracted. This left a clean external carotid artery.

The patient recovered and was discharged on her fourth postoperative day. She has remained asymptomatic on follow-up examination.

Case 2

This patient was a 68-year-old white man who suffered a right hemisphere stroke in February 1987. He was left with a residual mild paresis of the left upper and left lower extremities. On March 2, 1987, the patient underwent a right carotid endarterectomy at another hospital. This was uncomplicated, and the patient recovered uneventfully. On March 24, 1987, the patient underwent a left carotid endarterectomy for an asymptomatic, high-grade stenosis. An intraoperative completion angiogram was performed, which demonstrated a good technical result involving the carotid bifurcation and internal carotid artery. However, there was a significant filling defect, representing an intimal flap of the external carotid artery, 0.5 cm distal to its origin (Figure 1). The surgeon stated that the patient was experiencing some cardiac instability, and, for that reason, a decision was made not to correct the intimal flap. The patient recovered without event from that operation.

Three months later, while visiting relatives in Los Angeles, the patient was found by his wife to be slumped over in a chair and unresponsive. She described his speech as garbled and indecipherable, consistent with a marked expressive dysphasia referable to left hemisphere ischemia. There was also a right central facial weakness.

Approximately an hour after the event, the patient was brought to the UCLA emergency department. By that time, most of the findings had resolved. On physical examination, his blood pressure was 210/110. There was a harsh, high-pitched carotid bruit present over the left carotid bifurcation, and there was a soft bruit over the right carotid bifurcation. Both carotid pulses were full, and both temporal artery pulses were palpable. The patient was lethargic and disoriented with respect to name, place, and purpose. His memory appeared to be impaired. His speech was slow but otherwise normal. Physical examination revealed the residual from his right hemisphere stroke, but there were no other residual findings from the left hemisphere ischemic event.

An emergency duplex scan was performed. The internal carotid arteries were normal bilaterally. However, the left external carotid artery was interpreted as showing fresh thrombus. The patient was taken immediately to the operating room where an intraoperative angiogram demonstrated a 99% stenosis of the left external carotid artery at its origin. The carotid bifurcation was opened, and fresh thrombus, secondary to an intimal flap of the external carotid artery, was present. It had progressed, retrograde, adjacent to the orifice of the internal carotid artery. The intimal flap and thrombus were extracted with the use of an angioscope.

Unfortunately, when the patient awoke from anesthesia, he was found to have suffered an intraoperative stroke with a resulting right hemiparesis. The mechanism of this event is not certain but may represent further embolization of the thrombus that propagated from the external to internal carotid artery during mobilization of the artery.

Case 3

The patient was a 71-year-old white man with a history of peripheral vascular disease and hypertension. In September 1985, he presented to a hospital in Missouri with right hemispheric transient ischemic attacks. An angiogram demonstrated a 90% stenosis of the right internal carotid artery. The left internal carotid artery had a 50% stenosis. A right carotid endarterectomy was performed using a shunt. The arteriotomy was closed with a vein patch. Completion angiography was not performed. The patient was discharged, without event, on the second postoperative day.

The patient presented to UCLA Medical Center in March 1987 for follow-up. At that time, he was asymptomatic. A duplex scan demonstrated a 20% stenosis of the right carotid bulb and a 50% stenosis of the left carotid bulb. The patient was instructed to return in 6 months.

He returned in September 1987, having experienced a right hemisphere transient ischemic attack 1 month earlier. This was manifest by left upper extremity numbness and weakness associated with blurred vision and headache. On examination, the right superficial temporal artery pulse was absent. New bruits were...
detected over both the right and left carotid bifurcation. A computed tomogram (CT scan) was negative. A duplex scan demonstrated a totally occluded right external carotid artery with an image consistent with thrombus. No plaque was noted in the right internal carotid artery. The right carotid bulb showed a 20% narrowing. The left carotid bulb showed a stable 50% stenosis. An angiogram was performed and demonstrated a completely occluded right external carotid artery with a shallow, smoothly marginated stump. The patient was told to continue to take aspirin and maintain careful follow-up.

On January 25, 1988, the patient presented to a hospital in another state with a 3-day history of unsteady gait and uncoordination. Immediately before admission, he had experienced 10 minutes of altered consciousness as well as three episodes of left hand and face numbness and weakness. This was associated with slurred speech and occipital headache. A magnetic resonance imaging scan demonstrated a wedge-shaped right parieto-occipital infarction. The patient was transferred to the Vascular Surgery service at UCLA. On examination, a left carotid bruit was present, but the right carotid bruit was absent. He was noted to have lost peripheral vision. A repeat duplex scan was unchanged from the previous study. The patient was immediately taken to the operating room where fresh thrombus was identified at the orifice of the right external carotid artery, which was occluded by an intimal flap. The thrombus propagated into the internal carotid artery adjacent to the vein patch. The carotid bifurcation was resected and a 6-mm polytetrafluoroethylene graft was interposed between the common and internal...
FIGURE 2. Upper panel: Intraoperative completion angiogram of case 4, demonstrating defect created by an intimal flap. Lower panel: After the intimal flap is removed, the completion angiogram demonstrates a smooth technical result in both internal and external carotid arteries.
carotid arteries. The external carotid artery was excluded. The operation was performed over a Javid shunt, and the patient was monitored with brain mapping, which revealed no change during the operation. A completion angiogram demonstrated an excellent technical result. The patient recovered and was discharged asymptomatic.

Case 4

A 65-year-old white man presented to the UCLA Medical Center in October 1987 having suffered a left hemisphere stroke 4 years previously. At that time, he was found to have a 60% stenosis of the left carotid bulb and a 40% stenosis of the right carotid bulb.

On physical examination, his blood pressure was 110/60. It was noted that he had an obvious dense expressive aphasia. His carotid pulses were normal bilaterally. There was a harsh bruit over the right carotid bifurcation. The temporal pulse on the left side was diminished, and neurological examination revealed somewhat diminished strength of the left arm and leg. There was a parietic right lower extremity and mildly parietic right upper extremity. There was a sustained contracture of the fingers of the right hand, and hyperreflexia was present on the right side. Preoperative angiography demonstrated bilateral 95% stenoses of both internal carotid arteries with a marked bulb irregularity on the right side.

Because the most recent symptoms suggested activity of the right carotid bifurcation lesion, a right carotid endarterectomy was performed with intraoperative brain mapping. A completion angiogram demonstrated a technically good result. The patient was discharged on the third postoperative day.

One month later, the patient returned for surgery of the left carotid bifurcation. A preoperative duplex scan demonstrated a normal right carotid bifurcation and a stenosis greater than 90% of the left internal carotid artery. A left carotid bifurcation endarterectomy was performed with the use of an internal shunt. The carotid arteriotomy was closed with a patch. A completion angiogram demonstrated a technically excellent result involving the carotid bifurcation and internal carotid artery. However, there was an intimal flap present in the left external carotid artery (Figure 2, upper panel). Based on the angiogram, an arteriotomy was made on the external carotid artery. The intimal flap was identified and was associated with fresh thrombus. The intimal flap and thrombus were removed, and the external carotid artery was closed. A completion angiogram was repeated and demonstrated a clean carotid bifurcation endarterectomy involving both the internal and external carotid arteries (Figure 2, lower panel).

The patient tolerated the operation well and was discharged on the third postoperative day.

Discussion

The importance of documenting on the operating table the technical result of carotid endarterectomy has met with a lukewarm response by many surgeons.

In 1967, Blaisdell et al reported their experience with routine completion angiography after carotid endarterectomy. They noted that approximately 25% of the angiograms demonstrated a technical abnormality that might otherwise have escaped notice. Correction of these defects at the time of angiography resulted in a 100% late patency of the carotid arteries in their series. A similar experience was reported by Rosental et al, in which they documented unacceptable technical errors in 8% of their operations. Although the majority of the defects identified in both series consisted of intimal flaps of the external carotid artery, critics continued to argue that the frequency of abnormality found in the internal carotid artery would not justify the routine use of completion angiography.

Based on the three cases in this report, we wish to emphasize that an important mechanism for perioperative stroke includes thrombus formation associated with an intimal flap of the external carotid artery, resulting in retrograde propagation and subsequent antegrade embolization of the internal carotid artery (Figure 3). Reports have mentioned intimal flaps in the external carotid artery, but only one has cited its importance as a source of intracranial embolization as manifested by transient ischemic attacks postoperatively. This problem, which might be termed "the external carotid artery intimal flap syndrome," adds further merit to the importance of documenting the technical result of operation, not only in the internal carotid artery, but also the external carotid artery. This external carotid artery stump syndrome is the converse of the internal carotid artery stump syndrome described by Barnett et al. When an intimal flap is identified in the external carotid artery, it should be corrected. The technique we advocate is to clamp the origin of the external carotid artery, thus maintaining flow between the common and internal carotid artery. The distal vessel then can be controlled in a similar fashion. A transverse arteriotomy, positioned just
distal to the attachment of the intimal flap, will permit correction of the problem (Figure 4).

Opponents to the use of intraoperative completion angiograms argue that the addition of this study increases operative time. This is only true when completion angiography is done on an occasional basis. When done routinely, it adds only 10–15 minutes to the operative procedure. The cost for completion angiogram at our institution is currently $181, which includes technician time and interpretation. It has always seemed curious that completion angiograms are routinely recommended for peripheral reconstructions, when the worst that can occur with a technical error is a thrombosis and return to the operating room. When technical error occurs with carotid endarterectomy and leads to a thromboembolic complication, the resulting stroke often leaves a permanent deficit in spite of a timely correction. Routine completion angiography would appear to be a small price to pay for insurance against this complication.

References


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