Intracranial Embolization Via External Carotid Artery: Report of a Case with Angiographic Documentation

ROGER W. COUNTEE, M.D., AND THURAIRASAHI VIJAYANATHAN, M.D.

SUMMARY This report describes our experiences with a patient who developed delayed recurrent retinal and hemispheric ischemia distal to an old internal carotid artery occlusion in the neck. Fundoscopy and sequential cerebral arteriography documented that recurrent ischemic symptoms in this individual were the result of embolic fragments arising from the "stump" of the occluded internal carotid artery and from a diseased external carotid artery. These emboli traversed the external carotid artery and its orbital and intracranial anastomotic connections to reach the symptomatic eye and hemisphere. Ischemic symptoms in this patient were effectively terminated with anticoagulant therapy.

We believe that this patient graphically documents that post-occlusion microembolism via the external carotid artery does indeed occur, and probably accounts for post-occlusion recurrent ischemic attacks more frequently than is currently appreciated. Recognition of this phenomenon is of importance because of its significant therapeutic implications. In these situations treatment modalities which terminate embolic phenomena would appear to have a more rational basis than do surgical procedures designed primarily to augment collateral blood flow to the symptomatic organ(s).

IT IS RECOGNIZED that a significant number of patients will initially sustain an internal carotid artery occlusion with minimal or no neurological deficit but, some time later, develop symptoms of ipsilateral recurrent ischemia. The pathogenesis of recurrent transient ischemia and/or delayed infarctions in such situations, however, remains unclear. Several authors have suggested that microembolism via the external carotid artery homolateral to the internal carotid artery in the neck with a residual "stump" of its origin (See fig. 1). There was excellent retrograde perfusion of the right middle cerebral artery via an anastomotic connection to reach the symptomatic hemisphere. Ischemic symptoms in this patient were effectively terminated with anticoagulant therapy.

We believe that this patient graphically documents that post-occlusion microembolism via the external carotid artery does indeed occur, and probably accounts for post-occlusion recurrent ischemic attacks more frequently than is currently appreciated. Recognition of this phenomenon is of importance because of its significant therapeutic implications. In these situations treatment modalities which terminate embolic phenomena would appear to have a more rational basis than do surgical procedures designed primarily to augment collateral blood flow to the symptomatic organ(s).

Patient Report

A 54-year-old, right-handed, normotensive, non-diabetic, generally healthy, white male policeman was admitted to the hospital in May, 1979, after the sudden onset of a dense left hemiparesis. Within 8 hours of his admission, the left-sided weakness had dramatically improved. It later completely resolved in the subsequent 2 weeks following the ictus. Complete cerebral arteriography performed 2 weeks after admission demonstrated total occlusion of the right internal carotid artery in the neck with a residual "stump" of its origin (See fig. 1). There was excellent perfusion of the right middle cerebral artery via an enlarged right ophthalmic artery which was filled in retrograde fashion from the ipsilateral external carotid artery. The right anterior cerebral artery was filled by the left carotid system which was normal save for a modest deformity at the common carotid bifurcation. There was additional collateral flow to the right anterior circulation from the normal vertebro-basilar arterial system.

Within days following arteriography the patient began having recurrent episodes of monocular blindness in the right eye. Although these episodes were fleeting, they became progressively more frequent and, on one occasion, platelet fragments were observed in several temporal branches of the right central retinal artery. It was believed that the "stump" of the occluded right internal carotid artery was the source of these embolic fragments. Consequently, a right external carotid endarterectomy with resection of the "stump" was performed. The "stump" was found to be filled with atheromatous debris and thrombus.

The patient's initially uneventful course after operation was punctuated on the 4th postoperative day by an episode of transient clumsiness of his left hand. He later reported fleeting episodes of numbness in the left side of his face and his left hand, as well as several attacks of amaurosis fugax in the right eye. On the 7th postoperative day a selective right common carotid arteriogram was performed (fig. 2). Slight irregularities in the lumen of the distal common carotid and proximal external carotid arteries were noted. In addition, a filling defect in the external carotid artery was seen at the upper limits of the endarterectomy segment. This filling defect was believed to represent a small thrombus around a raised intimal flap. The distal cavernous and supra-clinoid segments of the right internal carotid artery were again filled by retrograde flow through the right ophthalmic artery. However, at the distal end of the supraclinoid carotid artery near the origin of the middle cerebral artery an intraluminal embolus was now visualized.

It was recognized that technical complications of the operative procedure had replaced the "stump" of the occluded internal carotid with another source of arterial-to-arterial emboli within the external carotid artery itself. Anticoagulant therapy was, therefore, initiated and the patient's symptoms promptly ceased.

From the Sections of Neurological Surgery (Dr. Countee) and Neuroradiology (Dr. Vijayanathan), College of Medicine and Dentistry of New Jersey, New Jersey Medical School, Newark, NJ, and Veterans Administration Medical Center, East Orange, NJ. Reprints: Dr. Countee, Section of Neurological Surgery, New Jersey Medical School, 100 Bergen St., H592, Newark, NJ 07107.
He has remained symptom free and neurologically intact on Coumadin therapy throughout the subsequent 12 months of follow up.

Discussion

Recurrent ischemic symptoms distal to internal carotid occlusions in the neck are frequently presumed to result from intermittent hemodynamic insufficiency in the intracranial circulation. It is theorized that regions of neural tissue precariously sustained by marginal arterial perfusion manifest transient neuronal dysfunction during subtle and episodic decrements in regional cerebral blood flow. As a logical extension to this assumption, it is believed that in such patients augmentation of collateral blood flow to these symptomatic areas should be the major goal of operative therapy. Several authors, however, have suggested that microembolism beyond internal carotid occlusions may frequently be the mechanism responsible for recurrent transient ischemic symptoms. This factor may well explain continued recurrent ischemia in some patients in spite of a successful extracranial-intracranial microvascular bypass procedure and/or angiographic evidence of apparently ample collateral reserves. Widespread acceptance of an embolic phenomenon occurring after an internal carotid occlusion has been slow in coming. The evidence in support of this thesis, however, continues to grow.

Convincing arguments for post-occlusion microembolism have been based on certain important observations made in some patients with angiographically documented "old" internal carotid occlusions. First, embolic fragments in the ipsilateral retina have been visualized in some of these patients after arteriographic procedures, after common carotid compression, after surgery on the homolateral external carotid artery, as well as after their spontaneous occurrence. Third, recurrent transient ischemia has often been promptly terminated after operative procedures have removed sources of arterial-to-arterial emboli through the ipsilateral external carotid artery, as well as after the spontaneous occlusion of a diseased common carotid artery which formerly supplied the offending external carotid artery. Previously reported arteriographic observations have given inferential support for a post-occlusion embolization.
**Figure 2.** Postoperative right AP (AP) and lateral (LAT) common carotid angiogram. Slight irregularity in the endarterectomized segment of the distal common carotid and the external carotid origin (A) are seen. A raised intimal flap with intraluminal thrombus (white arrows) is seen in the external carotid at the upper level of the endarterectomy. A distal embolus (small black arrows) is also seen in the supraclinoid segment of the internal carotid artery (C) near the origin of the middle cerebral artery. Internal maxillary (M), middle meningeal (MM), and superficial temporal (ST) branches are seen to fill the ophthalmic artery (B) in retrograde fashion.

Embolization phenomenon as well. Einsiedel-Lechtape has demonstrated angiographically that “secondary occlusions” of intracranial vessels distal to extracranial internal carotid occlusions may occur, and probably more frequently than is generally recognized. Arteriographic visualization of these distal emboli is often difficult, however, both because of their tendency to dissolve rapidly as well as because of the technical difficulties often encountered in optimally opacifying the intracranial circulation formerly irrigated by an occluded internal carotid artery. Although Einsiedel-Lechtape concluded that these emboli arise from fragments of stagnation thrombus in the distal portions of proximally occluded internal carotid arteries, Barnett et al. recognized that more likely sources of this embolic debris could be frequently identified angiographically in the ipsilateral extracranial collateral circulation. In earlier communication we have reported experiences similar to those reported by Barnett et al. Moreover, we have pointed out that enhanced arterial flow via the ipsilateral external carotid artery to the symptomatic eye and/or hemisphere in these patients can invariably be demonstrated angiographically when appropriate catheter techniques are employed. The arteriograms of the patient described in this report typify the characteristic features of these altered arterial flow patterns which we have previously described (fig. 1).

The possibility that a primary thrombosis was the cause for the supraclinoid carotid artery filling defect seen on postoperative angiograms in our patient was also considered. However, because of the excellent antegrade flow through this vessel visualized on preoperative arteriograms, as well as the likelihood of such an event occurring spontaneously in an uninstrumented vessel with no evidence of intrinsic disease, we believe that the probability of thrombosis in situ is quite remote. Furthermore, the possibility that an embolus reached the right supraclinoid carotid from the left carotid system also seemed improbable in view of the fact that the right internal carotid artery could not be opacified angiographically by selective left carotid injections either pre- or postoperatively. Moreover, there was no identifiable source of embolic fragments visualized in the left carotid or the vertebrobasilar systems. Although there was some
deformity in the angulation of the origins of the internal and external carotid arteries at the left common carotid bifurcation, close inspection of the bifurcations in 3 different views failed to disclose any suggestion of significant intraluminal disease such as intimal erosions or ulcerations. Sequential arteriography clearly demonstrated that the dominant, if not the exclusive, arterial flow to the right supraclinoid carotid artery was from the ipsilateral external carotid artery via reversed ophthalmic artery flow. In addition, reversed ophthalmic artery flow also opacified the cavernous segment of the right carotid and no propagation of thrombus from the proximal internal carotid beyond this segment could be demonstrated. Most importantly, an obvious source of emboli was clearly demonstrated within the lumen of the dominant arterial conduit to the symptomatic right retina and intracranial carotid artery.

In the patient described in this report we have documented with fundoscopy and arteriography that microembolism to the retinal and intracranial circulations distal to an "old" internal carotid occlusion in the neck can occur. These emboli traverse the ipsilateral external carotid artery and its orbital and intracranial anastomotic connections when this alternative pathway has been recruited to compensate for a unilateral internal carotid occlusion. These emboli arise from intraluminal lesions at or near the origin of the functionally important external carotid artery and their source, as well as the route by which they reach the symptomatic organ(s), can be invariably identified angiographically with proper arteriographic techniques. We believe that embolic phenomena probably account for post-occlusion recurrent ischemia far more frequently than heretofore appreciated. Recognition of this pathogenic mechanism is most important in view of its highly significant therapeutic implications. When this phenomenon is identified for its actual or potential role in causing recurrent ischemia in patients with internal carotid occlusions, treatment directed at terminating microembolism would seem to be the most rational therapeutic priority. The efficacy of such therapies in this select group of patients has been well documented. On the other hand, surgical procedures designed primarily to augment collateral cerebral blood flow would seem to have dubious benefit in these particular situations, especially when those procedures utilize branches of a diseased external carotid artery as a conduit. As this patient also clearly demonstrates, surgery on the external carotid artery in these situations demands a cautious and meticulous operative technique which minimizes the risk of intraoperative as well as postoperative embolization.

References

Intracranial embolization via external carotid artery: report of a case with angiographic documentation.
R W Countee and T Vijayanathan

Stroke. 1980;11:465-468
doi: 10.1161/01.STR.11.5.465

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1980 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/11/5/465

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/