Short Communication

External Carotid Artery Embolus from the Internal Carotid Artery “Stump” During Angiography

Case Report

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SUMMARY A case is reported in which during a single angiographic sequence, an embolus was dislodged from a thrombus in the internal carotid artery “stump” and progressed up the external carotid artery. This case supports the concept that emboli originating in the stump of an occluded internal carotid artery may progress through the external carotid system and via external carotid-internal carotid anastomoses, produce ischemic episodes in the distribution of the occluded internal carotid artery.

DISEASE OF THE PROXIMAL internal carotid artery as a cause of cerebral vascular disease, primarily through embolization, has been well accepted for almost thirty years. That disease of the proximal external carotid artery may contribute to cerebral vascular symptoms, especially amaurosis has been suspected for at least fifteen years. Barnett and associates have suggested a source of the symptoms due to external carotid artery disease may be emboli from thrombi in the residual origin of the occluded proximal internal carotid artery, a pouch they have labeled the “stump.” I was able to observe this phenomenon during angiography, lending credence to the theory of Barnett and his associates.

Case Report

This 62 year old male was admitted (in 1971) for the evaluation of cerebral vascular symptoms involving his left hemisphere. Approximately one year prior to admission, he noted the sudden onset of numbness and weakness in his right upper extremity and a slight clumsiness, if not weakness, of his right lower extremity. This was associated with, according to his wife, a confused, barely intelligible speech. The extremity symptoms cleared in approximately 24 hours. The speech difficulty improved to normal over three or four days. In the year prior to admission, he had three or four episodes of right upper extremity numbness, not associated with any other symptoms and lasting a few minutes to hours. The last such episode was one week prior to admission.

On admission he was noted to be alert and oriented. His blood pressure was 150/90 with a regular pulse of 80. He had no neck bruits and no abnormal neurological findings. An isotope brain scan was normal. Forty-eight hours after admission, under general endotracheal anesthesia, he underwent cerebral angiography consisting of right brachial artery and left percutaneous common carotid artery injections. During the left carotid injection he was noted to have an occluded left internal carotid artery with a residual stump. A thrombus was identified in the stump and a portion embolized, during the single angiographic series, into the external carotid artery (figs. 1–3). Following recovery from anesthesia he was noted to be somewhat disoriented as to place and time. He had no focal neurological deficits. Vision and fundoscopic examination were normal. On the fourth postangiographic day, the internal carotid artery stump was resected with removal of the residual thrombus that extended into the external carotid artery. His recovery was uncomplicated and when examined three months later, he reported no symptoms. Shortly thereafter he moved from the community and was lost to followup.

Discussion

Due to extensive anastomoses between the external carotid artery and the intracranial internal carotid artery system, it has been suggested that the external carotid artery, in the presence of internal carotid artery occlusion, may be the source of cerebral vascular ischemic syndromes. Hemodynamic studies show that, during internal carotid endarterectomy, restoration of flow in the external carotid artery with temporary internal carotid occlusion increased the internal carotid artery stump pressure by up to 21 mm of mercury. This suggests that external carotid artery stenosis may decrease blood flow through external carotid–internal carotid anastomoses and therefore, decrease blood flow in the distribution of the intracranial internal carotid artery.

Barnett and associates have suggested that thrombi...
present in the stump of an occluded cervical internal carotid artery may be the source of emboli into the external carotid artery which in turn by way of external carotid–internal carotid anastomoses, may be the source of cerebral vascular ischemia.\textsuperscript{4,5} They have reported a number of patients with hemispheric and retinal ischemic syndromes which developed in the presence of an occluded internal carotid artery with persistence of the stump. Angiographically and at surgery ulcerations and thrombi, which they felt could be the source of embolic disease, were identified within the stump. They noted the internal carotid stump might be the site of excessive turbulence based upon in vitro flow studies using glass tubing fashioned to resemble the carotid bifurcation. They thus concluded that platelet aggregation and thrombogenesis might occur due to pre-existing arteriosclerosis in the stump and that turbulent flow could cause emboli to disseminate.

In the presently reported case, turbulent flow in the stump may have been enhanced by the injection of the contrast material directly into the common carotid artery through the percutaneously placed needle. Figure 1 demonstrates an intraluminal filling defect in the distal common carotid artery and in the "stump" of the occluded internal carotid artery. Figures 2 and 3 reveal an apparent enlargement of the stump. This is due to greater filling of the stump as a result of dislocation of the thrombus, some of which may be seen at the origin of the external carotid artery. A well-defined intraluminal defect is seen within the external carotid artery just proximal to the angle of the mandible in figure 2. In figure 3, this defect has progressed distal to the angle of the mandible. The conclusion drawn from an examination of this angiographic sequence is that thrombus was present, partially filling the internal carotid artery stump. Increased turbulent flow created by the direct intra-arterial injection of the contrast material dislodged the thrombus, causing a fragment to migrate distally during the angiogram. This finding lends credence to the thesis of Barnett and his associates that this phenomenon may occur naturally.

Because the patient's subsequent neurological symptoms were not focal, it is difficult to state categorically that the embolus identified angiographically or other emboli which may not have been seen were
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Progression of external carotid artery embolus in later arterial phase.

responsible for his symptoms. The natural history of this condition is not known and, because of the increasing tendency for it to be managed surgically, may never be known. Nevertheless, the dramatic demonstration by this case that the theory of Barnett and his associates is reasonable, and the demonstration by them and others that obliteration of the stump along with external carotid endarterectomy relieves the symptoms of some patients with ischemic episodes in the distribution of the occluded internal carotid artery, make the recommendation of surgical management of these cases reasonable.

References

External carotid artery embolus from the internal carotid artery "stump" during angiography. Case report.

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