Coincidental Multiple Asymptomatic Intracranial Aneurysms and Symptomatic Carotid Stenosis

ROBERT D. SHOUMAKER, M.D.,* WILBUR S. AVANT, M.D.,† AND MAJOR GILBERT H. COHEN, M.C.‡

SUMMARY A patient experiencing transient cerebral ischemic attacks (TIAs) was studied angiographically and found to have a symptomatic stenosis of the appropriate internal carotid artery (ICA) and three asymptomatic intracranial aneurysms. A therapeutic approach to this type of problem is discussed in this article.

IN PATIENTS undergoing cerebral angiographical examination for evaluation of occlusive cerebrovascular disease, about 5% will show an unexpected aneurysm. The number with multiple aneurysms is difficult to ascertain from the literature, but must be considerably less. The following case illustrates the rare association of three asymptomatic intracranial aneurysms and a symptomatic carotid stenosis demonstrated on angiographical evaluation.

Case Report

A 67-year-old white woman gave a history of a five-minute episode of left arm numbness occurring three months prior to admission. On the day of admission, April 3, 1974, she noted the onset of complete numbness and slight weakness of her left arm and leg lasting about 45 minutes. By the time she was hospitalized, the symptoms had nearly resolved.

She had a history of hypertension for at least 20 years, which had been under fair control with multiple medications. She also had chronic bronchitis, headaches, and occasional depression and was being treated for hypothyroidism. There was no history of diabetes mellitus, angina or prior neurological deficits. Family history was negative for any neurological disorders.

Examination on admission revealed an alert apprehensive woman with a blood pressure of 160/92 mm Hg, regular pulse of 96 per minute. Funduscopic examination showed mild arteriolar narrowing. No hemorrhages, exudates or bright plaques were seen. Ophthalmodynamometry showed a 25% difference in systolic pressures, with the left greater than the right. Her neck was supple and bruits were heard over both carotids, most prominent at the midcervical region, right greater than the left. Cardiac examination suggested left ventricular enlargement but no murmurs or gallops were heard. The rest of the general examination was normal. Neurological examination was normal except for some extinction of the left arm with two-point simultaneous stimulation testing and slight downward drift of her left arm when extended. No pathological reflexes were elicited.

Complete blood count, urinalysis, blood chemistries, partial thromboplastin time, and prothrombin time were all normal. Cholesterol was 200 mg/dl, and triglyceride was 235 mg/dl. Arterial gases showed a pH of 7.44, P0₂ of 81 mm Hg, and Pco₂ of 32 mm Hg. Chest and skull x-rays were normal. EKG was suggestive of an old anteroseptal myocardial infarct. A radioisotope brain scan and flow study with technetium was normal. During the 12 hours after admission, the patient had two more episodes of left arm and leg numbness with minimal weakness lasting 20 minutes. A lumbar puncture showed an opening pressure of 175 mm H₂O. Clear fluid with two mononuclear cells and no RBCs; glucose and protein were normal. She was treated with intravenous heparin, but had two more episodes of similar duration during the next 18 hours. Subsequently, she underwent cerebral angiography via transfemoral artery catheterization. Radiography revealed a 90% stenosis of the right internal carotid artery. In addition, aneurysms of the right middle cerebral artery, right anterior cerebral artery and left posterior cerebral artery (fig. 1) and left posterior cerebral artery (fig. 2) were noted. There was also a 30% stenosis of the left internal carotid artery at the bifurcation.

The patient then underwent a right carotid endarterectomy which revealed an estimated 95% stenosis of the internal carotid artery. Neurosurgical consultants felt that because of the patient’s multiple medical problems she was not a good candidate for intracranial surgery at that time. Her postoperative course was uneventful and ophthalmodynamometry revealed equal pressures. She was discharged on acetylsalicylic acid and dipyriramole (Persantin), in addition to her antihypertensive and thyroid medications. Her course remained stable and she was readmitted seven months after her surgery for re-evaluation. Cerebral angiography was performed via transfemoral catheterization and there was no measurable change in the size of the aneurysms. The right internal carotid artery was patent; however, the stenosis in the left internal carotid artery had progressed. A left carotid endarterectomy was successfully performed. Because no increase in the size of the aneurysms was noted, intracranial surgery was not recommended.

Discussion

In a review of the literature by Portnoy and Avellanosa on the association of extracranial carotid stenosis or occlusion and intracranial aneurysm, no cases showing the association with multiple aneurysms were identified. They reported one case with a right internal carotid aneurysm and left internal carotid artery stenosis. Their surgical approach was identical to that advocated by Pool and Potts, i.e., endarterectomy of the stenotic vessel followed by surgical treatment of the aneurysm. Fields and Weibel briefly reported two similar cases of ipsilateral symptomatic carotid stenosis with incidental single intracranial aneurysm, in which they...
MULTIPLE ASYMPTOMATIC INTRACRANIAL ANEURYSMS/Shoumaker et al. 505

FIGURE 1. Anteroposterior view of right common carotid injection showing 90% stenosis (circumferential measurement in two planes) of the internal carotid artery (small open arrow). Also seen is a 9 × 6 mm aneurysm of right anterior cerebral artery (large black arrow) with a 7 × 6 mm aneurysm of the right middle cerebral artery (small black arrow).

FIGURE 2. Anteroposterior view of left vertebral injection showing an 8 × 5 mm aneurysm of the left posterior cerebral artery (large open arrow) and residual of contrast media in right anterior cerebral artery aneurysm from previous injection (large black arrow).

only performed a carotid endarterectomy. Because follow-up angiography after one year showed no increase in the size of the aneurysm, they felt “there was little risk in arterial reconstructive surgery of the internal carotid artery in the presence of an unruptured aneurysm, even though the aneurysm may be on the same artery.” Denton and Gutmann reported a case with a symptomatic stenosis of the right internal carotid artery and an incidental aneurysm of the right middle cerebral artery. They performed a carotid endarterectomy and after one week (repeat angiogram showed no growth of the aneurysm) surgically treated the aneurysm via plastic encasement. It was their own opinion that “endarterectomy would theoretically increase the real risk of aneurysmal rupture.”

The natural history of an aneurysm is not uniform for it may: (1) remain the same size, (2) increase in size, (3) rupture, or (4) rarely disappear. The increase in size may be gradual or abrupt.10

Those factors felt to be significant in determining what changes, if any, the aneurysm will undergo include the degree of developmental defect, hypertension, turbulence within the aneurysms and degree of atherosclerosis.11-14

Previous studies have shown that the pressure to which an aneurysm is subjected is equal to the mean systemic pressure and that the turbulence within the aneurysm increases as the pressure increases.15-17 A stenotic lesion causing a greater than 80% decrease in cross-sectional area, as was the case in our patient, will cause a pressure gradient that increases markedly with any further stenosis.15-17 Thus, theoretically, correction of a significant internal carotid stenosis ipsilateral to an intracranial aneurysm should increase the pressure and turbulence to which the aneurysm is subjected and result in an increase in its size and probability of rupture. This was not noted in our patient, albeit follow-up was brief. It was our opinion that the risk of a cerebral infarct in this patient outweighed the possibility of an intracranial hemorrhage from rupture of one of the aneurysms after the endarterectomy, thus dictating our initial surgery.

The risk of a subarachnoid hemorrhage, in a patient with multiple intracranial aneurysms, from a previously asymptomatic aneurysm appears to be about 10% to 20% (four to five years’ average follow-up).18, 19 Since the mortality from a subarachnoid hemorrhage in patients with multiple intracranial aneurysms is about 60% in the first six months,20, 21
then the probability of a fatal hemorrhage in the patient with asymptomatic aneurysms should be around 6% to 12%. Thus, any proposed elective surgical procedure for these patients should have a mortality risk less than 6% to 12%. Whereas the surgical approach to multiple asymptomatic aneurysms has tended to be relatively conservative, others have taken a more aggressive approach. If the patient is properly selected (accessibility and size of the aneurysms; patient’s age and neurological, mental and physical condition; patient’s informed consent), and if the experience and skill of the neurosurgeon are taken into consideration, the mortality rate from elective surgery can be less than that of the nonsurgically treated patient.

A patient with symptomatic internal carotid artery stenosis and asymptomatic single or multiple aneurysms represents a therapeutic dilemma. Based on a review of the literature, this group of patients should first undergo a carotid endarterectomy if clinically and angiographically indicated. Subsequently, the course is not as certain, but if all of the very selective criteria are met, the use of one or more elective intracranial surgical procedure(s) on the aneurysm(s) should be considered. If the patient does not meet all the criteria but is still a surgical candidate, he should be followed with angiograms every 6 to 12 months. If an increase in the size of the aneurysm(s) is documented, elective intracranial surgery should be reconsidered.

References

Coincidental multiple asymptomatic intracranial aneurysms and symptomatic carotid stenosis.
R D Shoumaker, W S Avant and G H Cohen

Stroke. 1976;7:504-506
doi: 10.1161/01.STR.7.5.504
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1976 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/7/5/504

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/