Surgical treatment of symptomatic carotid stenosis and asymptomatic ipsilateral intracranial aneurysm

Case report

PORTNOY and Avellanosa recently reviewed the literature concerning the rare coexistence of carotid stenosis or occlusion with intracranial saccular aneurysm. They successfully operated on both the symptomatic carotid stenosis and the contralateral symptomatic intracranial aneurysm in one patient. Their approach to this case reflected Pool and Potts' advice: “the procedure of choice is first to perform an endarterectomy on the stenotic vessels and later to proceed with surgical treatment of the aneurysm on the opposite side.”

In contrast, Fields and Weibel reported two patients with symptomatic carotid stenoses and ipsilateral, incidentally-discovered, intracranial aneurysms. Because “many neurosurgeons have advocated common carotid ligation on such aneurysms,” the author expressed “concern . . . about the advisability of removing the cervical carotid lesion.” Nonetheless, both cases underwent endarterectomy without treatment of the intracranial aneurysms. Since arteriography 1 year later showed no growth of the untreated aneurysms, the authors concluded that “there is little risk in arterial reconstructive surgery in the presence of an unruptured asymptomatic aneurysm . . . on the distal portion of the same artery.”

We are presenting a case in which an asymptomatic middle cerebral artery aneurysm was incidentally discovered during angiographic study of a patient with symptomatic ipsilateral carotid bifurcation stenosis.

Case Report

This 58-year-old woman experienced two transient episodes of paresis of the left arm and left side of the face within the 24-hour period prior to admission. Both transient ischemic attacks (TIA) resolved sponta-
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Neurologically within 20 minutes. One TIA was associated with a poorly characterized blindness of several minutes’ duration. She had not experienced similar episodes previously. Past history revealed untreated labile hypertension.

Examination. General examination, including vital signs, was normal except for the findings of a harsh right carotid bruit and a slight drift of the left arm. Initial studies revealed normal complete blood count, urinanalysis, skull and chest films, and electroencephalogram. Prior to arteriography she experienced another TIA. Right retrograde-brachial and left carotid arteriography demonstrated a high-grade stenosis at the right carotid artery bifurcation (Fig. 1). The left-sided vessels were normal. An aneurysm 5 mm in the anteroposterior diameter was visualized at the right middle cerebral artery bifurcation.

Operations. With the patient’s fully informed consent, planned staged procedures were carried out. On June 16, 1972, a carotid endarterectomy was successfully accomplished. One week later angiography was repeated, showing satisfactory operative results and no growth of the aneurysm.

![Fig. 1. Arteriograms, anteroposterior (left) and lateral (right) views. The anteroposterior projection demonstrates severe stenosis of the right carotid bifurcation and an unsuspected aneurysm of the right middle cerebral artery.](image)
However, the emptying time of the contrast material from the aneurysmal sac was decreased by almost 2 seconds. On July 7, 1972, a plastic encasement of the entire broad-based aneurysm was accomplished using microsurgical technique. Postoperatively the patient has remained free of symptoms.

Discussion

We disagree with the conclusion of Fields and Weibel that a carotid stenosis may be corrected without imposing a potential risk to an ipsilateral aneurysm. Their assertion, based on two cases, not only appears theoretically unsound but also disregards available information about optimal management of incidentally-discovered intracranial aneurysms. We based our management both on theoretical considerations and on the clinical observations of others.

That a marked carotid stenosis should have a protective effect on a distal ipsilateral aneurysm appears theoretically unquestionable. The reduction of effective intracranial blood pressure constitutes the rationale behind carotid ligation as a definitive surgical treatment for intracranial aneurysms. In fact, Cronqvist and coauthors have demonstrated that even partial carotid occlusion, a situation not dissimilar to this case, may lead to a reduction of an aneurysm's size. By the same token, artificially lowering blood pressure reduces the risks of aneurysmal hemorrhage while awaiting definitive intracranial surgery. Ferguson’s recent demonstration that intracranial aneurysms are ordinarily subjected to the full force of systemic blood pressure experimentally supports the above clinical practices. Furthermore, Ferguson has shown that blood turbulence may exert a harmful fatigue stress on the aneurysmal wall. In our case, the decreased angiographic emptying time of the aneurysm after endarterectomy indicates increased velocity of blood flow, a factor which may increase turbulence and also reduce the likelihood of spontaneous thrombosis. If, as these thoughts suggest, severe carotid stenosis should protect an upstream aneurysm, then endarterectomy would theoretically increase the real risk of aneurysmal rupture.

Although recognized factors are felt to contribute to the growth and rupture of aneurysms, the growth rate itself remains a poorly defined process which may occur fairly rapidly or over the course of years. Therefore, Fields and Weibel's observation of no growth at 1 year does not strongly enforce their clinical assumptions that imply "little risk." Despite these vagaries of aneurysm growth rates, the known risks from rupture of the untreated incidental aneurysm may be greater than the hazards of elective surgery on the unruptured aneurysm. In this regard both Mount and Brisman and Moyes have presented convincing series of patients with asymptomatic aneurysms treated by elective surgery. These considerations we believe justified our staged surgical approach to this case, and have been strengthened by the result.

References

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