Recurrent Atherosclerotic Obstruction of the Internal Carotid Artery Five Years After Endarterectomy

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Abstract:
A patient is described in whom an obstructive atherosclerotic plaque recurred in the cervical portion of the internal carotid artery five years after endarterectomy. The lesion appeared to be a new formation of atheromatous material contiguous to the site of the previous arteriotomy and resulted in neurological symptoms almost identical to those experienced by the patient five years before.

This case suggests that the treatment of cerebral ischemia by surgical removal of stenotic lesions in the extracranial cerebral arteries may produce only temporary relief of symptoms and emphasizes the need, following endarterectomy, for medical therapy of the underlying atherosclerotic disease.

ADDITIONAL KEY WORDS surgery transient ischemic attacks angiography ophthalmodynamometry arterial stenosis cerebral infarction

During the past decade, endarterectomy has become an established form of treatment for cerebral ischemia caused by stenotic atherosclerotic lesions in the cervical portions of the carotid artery. Since most of the patients with this disorder also have atherosclerotic lesions in the other extracranial or intracranial cerebral arteries, it would be expected that cerebral ischemia will recur due to progression of the disease in these vessels. Indeed, several cases have recently been reported in which stenosis from newly formed atherosclerotic plaques developed several years after contralateral carotid endarterectomy. In three other patients, restenosis was observed at the site of the carotid endarterectomy two to seven years after the initial operation. In each case, the obstruction was caused by tough fibrous thickening of the arterial wall suggesting chronic deposition of fibrin in the endarterectomized area. There was no evidence of recurrent atherosclerosis.

The following case is the first reported instance in which an obstructive atheromatous lesion developed contiguous to the site of the carotid arteriotomy five years after successful endarterectomy. This case emphasizes the need for long-term follow-up observation of patients undergoing surgical treatment of cerebral arterial occlusive disease.

Case Report
J. B., a white male bookkeeper, born in 1907, was admitted to Duke University Hospital for the first time in July, 1962. One month before admission, transient numbness of the right foot and lower leg developed, followed two weeks later by weakness and clumsiness of the right arm and hand. Several days before admission he had noted intermittent difficulty in speaking. There were no other neurological symptoms. He had mild hypertension and an episode of chest pain in 1953 which was diagnosed as a myocardial infarction. Since that time, he had noted claudication of the calf muscles after walking a few hundred yards.

The patient's blood pressure on admission to the hospital was 135/70 mm Hg. A long, loud bruit was heard over the bifurcation of the left
carotid artery. No bruits were heard over the right carotid. Arterial pulses could not be palpated in either foot. There were moderate expressive dysphasia and slight impairment of fine movements of the right arm and hand. The right biceps tendon reflex was increased, but no other neurological abnormalities were noted. The ophthalmic artery pressures in the left eye were lower than in the right, being 75/50 and 135/70 respectively. The electrocardiogram showed evidence of an old myocardial infarction. Bilateral retrograde carotid arteriograms revealed a markedly stenotic lesion in the left internal carotid artery just above its origin (fig. 1). No other significant lesions were noted in the other extracranial or intracranial vessels.

Several days later a carotid endarterectomy was carried out and a large atherosclerotic plaque was removed from this vessel. The lesion at operation extended from the carotid bifurcation proximally about 1 cm into the common carotid artery, and distally about 1.5 cm into the internal carotid vessel. The atheromatous plaque was removed in its entirety. No patch graft was applied and the vessel was closed by direct suture. Before closure of the cervical incision, the blood pressures were measured in the distal portion of the internal carotid artery and in the external carotid artery and were found to be equal: 120/80 mm Hg. The patient had no surgical or neurological complications following the operation. The ophthalmic artery pressures measured...
postoperatively were equal: i.e., 125/50 in the left eye and 120/45 in the right. Examination of the specimen removed at operation revealed a firm, calcified, thick-walled plaque with a small lumen approximately 3 mm in diameter. Several areas of the wall were found microscopically to be covered with an organizing mural thrombus.

At follow-up examination eight months later, in March, 1963, the patient stated that he had had no recurrence of his cerebral ischemic attacks. He had not regained full dexterity of the right arm, however, and continued to have difficulty using a typewriter. His mild speech difficulty remained and he had some hesitancy in choosing words, particularly when excited. A repeat arteriogram of the left carotid artery at this time revealed good filling of the common, internal and external carotid vessels (fig. 2). No intimal lesions or evidence of stenosis were evident. The ophthalmic artery pressures remained essentially equal: 120 to 125/65 in the left eye and 130/65 in the right. The serum cholesterol level was 242 mg %.

Three years later, in April, 1966, the patient returned complaining of low back pain. He was found to have a large, diffuse aneurysm of the abdominal aorta which was successfully resected and replaced with a prosthetic graft. No bruits were heard over the carotid arteries at that time.

In January, 1967, some four and one-half
years after carotid endarterectomy, the patient again noted transient difficulties in speech. He was treated by his family physician with anticoagulant agents. In November, 1967, recurrent transient clumsiness and weakness of the right hand lasting 15 to 20 minutes developed and he was readmitted to the hospital. On examination, he was found to have hesitating speech without frank dysphasia. There was also a right superior quadrant homonymous field defect. A loud bruit was heard over the bifurcation of the left carotid artery. The arterial blood pressure was 200/105 mm Hg. The ophthalmic artery pressures were again unequal, the left being definitely lower than the right (90/40 versus 135/70 respectively). A retrograde femoral arteriogram of the aortic arch and cervical vessels showed a well-localized, severely stenotic lesion in the left internal carotid artery. This lesion was approximately 1 or 2 cm distal to the carotid bifurcation (fig. 3). The left carotid siphon showed minor plaque formation with no evidence of occlusive lesions in the intracranial branches of the carotid artery. The right vertebral artery was completely occluded at its origin and focal irregularities were noted in the bifurcation of the right common carotid artery. No crossover of the intracranial blood supply could be demonstrated from the right to left cerebral hemisphere. A roentgenogram of the chest showed a thoracic aneurysm. The electroencephalogram revealed a mild asymmetry with slightly slower rhythms over the left temporal area. The electrocardiogram showed left ventricular ischemia.

Endarterectomy was again carried out in the left carotid artery. At operation, an obstructing lesion was found in the internal carotid vessel 1½ to 2 cm above the carotid bifurcation just distal to the sutures outlining the upper end of the incision made at the previous arteriotomy. A large obstructing plaque containing shaggy atheromatous material was removed from this area. The previous arteriotomy site was carefully inspected and there appeared to be new endothelium lining the vessel with slight atheromatous changes. There was some roughening of the intimal surface which was dotted with fine yellow, glistening plaques. The new obstructive lesion appeared to be an entirely new formation of atherosclerotic material dating from the previous operation. The patient had mild dysphasia in the postoperative period, but this quickly cleared.

The patient has continued under observation. In June, 1969, he reported by correspondence that he was working full time and that he has had no new neurological symptoms. However, a malignant growth in his throat had developed which required high-voltage radiation therapy.

Discussion
The patient described in this report had a recurrent atherosclerotic obstructive lesion in the internal carotid artery several years after successful surgical removal of the initial stenotic plaque. This case is unique in that the atheromatous lesion recurred contiguous to the site of the previous carotid endarterectomy and reproduced neurological symptoms almost
RECURENT ATHEROSCLEROTIC OBSTRUCTION

identical to those experienced five years before. The plaque removed at the second operation appeared to be a new formation rather than progression of a residual lesion since the carotid arteriogram carried out eight months after the initial endarterectomy showed no evidence of intimal involvement. The ophthalmic artery pressures had become equal and a vascular bruit could no longer be heard over the area. Moreover, the intimal surface of the carotid artery at the site of the original arteriotomy was found at the second operation to be relatively clear of atheromatous material.

Assuming that the recurrent atheromatous plaque had arisen de novo, the question arises whether it may have been due in part to an alteration in local hemodynamics produced by the initial carotid arteriotomy. There is experimental evidence to indicate that segmental turbulence in blood flow may be a significant factor in the formation of atherosclerotic plaques. It has been shown, for example, that surgical alterations in the configuration of blood vessels in experimental animals will augment the development of segmental areas of atherosclerosis. Model hydraulic systems using flexible or rigid tubes to simulate human vessels suggest that the area of predilection for atherosclerotic lesions corresponds to the areas of reduced lateral wall pressure. The localization of atherosclerotic material to the bifurcation or branchings of large intracranial or extracranial cerebral arteries is probably due to such hemodynamic mechanisms. Local alterations in hydraulic forces may also account for the fact that in many patients, including the case described previously, the bifurcation of the carotid artery may be severely infiltrated with atheromatous material, whereas the intima immediately adjacent is spared and appears remarkably healthy.

Despite these considerations, it is unlikely that the initial surgical procedure in our patient resulted in sufficient disturbance in local blood flow to produce formation of a new atherosclerotic lesion. Care had been taken at endarterectomy to avoid the usual causes of turbulent flow. A patch graft was not employed in the surgical repair of the arteriotomy and all loose ends of intima had been carefully trimmed away. The arteriographic appearance of the vessel after the surgical procedure showed only slight dilatation of the vessel and no roughening of the intimal surface. In all probability, the development of the new carotid lesion was due to progression of the atherosclerotic process in a patient with extensive vascular disease involving the abdominal and thoracic aorta as well as the peripheral and coronary arteries. This explanation would seem most likely despite the unusual location of the atheromatous plaque, which was confined to a short segment of carotid artery several centimeters above, but not including, the carotid bifurcation.

It seems evident that patients undergoing surgical treatment for occlusive cerebrovascular disease in the extracranial vessels should also receive medical therapy directed at the underlying atherosclerotic process. Although the efficacy of most forms of medical treatment for this condition has not been fully established, the use of low-fat or low-cholesterol diets or medications such as clofibrate (Atromid) or cholestyramine (Cuemid, Questran) should be considered.

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References

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