Intimal Fibroplasia of the Internal Carotid Arteries

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Abstract: Intimal fibroplasia of the extracranial internal carotid arteries can cause symptoms of transient neurological deficit in young patients which are similar to the transient ischemic attacks of arteriosclerotic origin. The clinical, angiographical and histological features of such a case are presented and discussed.

ADDITIONAL KEY WORDS occlusive arterial lesions fibrous dysplasia arterial dysplasias transient neurological deficit

Introduction

Occlusive arterial lesions may result from intimal, medial or subadventitial fibroplasia as well as arteriosclerosis.1, 2 Fibromuscular hyperplasia, which involves the media, was first described in renal arteries as a cause of hypertension.1, 3-6 When it occurs in carotid or vertebral arteries, it may be an infrequent but recognized cause of transient neurological deficit.7-10 Intimal fibroplasia, a lesion of the intima and elastica interna, which has also been described in renal arteries,10 has heretofore not been noted in the carotid arteries. The following is the first reported case of bilateral intimal fibroplasia of the internal carotid vessels.

Case Report

A 29-year-old Caucasian female had a two-week history of transient weakness, paresthesias of the right arm and leg, and lightheadedness. On the day prior to admission, the patient had a sudden episode of left upper extremity weakness, slurred speech, and facial paresthesias that resolved within 20 minutes. The history was not remarkable and the patient denied use of contraceptive medications. The general physical examination was within normal limits and the neurological examination revealed only a slight proprioceptive deficit involving the left upper extremity. The CBC, urinalysis, sedimentation rate, L.E. prep, cholesterol, BUN, and blood sugar were normal. The brain scan and spinal fluid were unremarkable. The electroencephalogram revealed a grade 2 generalized dysrhythmia. An aortic arch study with selective carotid catheterization revealed 90% bilateral stenotic lesions of the internal carotid arteries with marked poststenotic dilatation. The intracranial, visceral and renal vessels were found to be unremarkable.

The patient underwent exploration of the right carotid artery under general anesthesia and hypercarbic technique. A 1-cm segment of internal carotid artery was excised and an interposition saphenous vein graft was placed between the common carotid and internal carotid arteries. The postoperative period was entirely uneventful. The patient was readmitted one month later for a vascular reconstruction of the opposite side. A retrograde brachial arteriogram revealed a patent reconstruction on the right side. The patient then underwent excision of the lesion of the left internal carotid artery with an end-to-end anastomosis. During the immediate postoperative period she had a brief series of grand mal seizures. They were readily controlled with diphenylhydantoin and phenobarbital. The slight proprioceptive deficit present preoperatively improved during the period of convalescence.

Pathology

GROSS The segment of right carotid artery measured 1.5 cm in length and up to 1.2 cm in greatest

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FIGURE 1

This photomicrograph reveals the classical changes seen in intimal fibroplasia. The lumen is markedly narrowed and a subintimal collagenous zone is seen which appears to be superimposed on the internal elastic membrane. Elastic tissue stains demonstrate a marked thickening of the intimal portion by connective tissue. The medium also reveals a prominent elastic tissue component. The externa is not remarkable.

diameter. The specimen had been opened in a longitudinal direction partially distorting the most narrowed aspect. No yellow discoloration of the intima was encountered and there was no evidence of calcification.

The specimen from the left carotid artery was received a little over a month after the above specimen and it consisted of a portion of vessel measuring 0.3 cm in length and up to 0.7 cm in diameter. There was a pinpoint lumen somewhat eccentric to the central axis of the vessel segment. The intimal aspect had a glistening tan appearance without evidence of subintimal calcification or plaque formation.

MICROSCOPIC

Histologically, a prominent increase in subintimal collagenous tissue was evident between the endothelium and the internal elastic membrane. This was most conspicuous utilizing Verhoeff's stain for elastic tissue. There was an absence of lipid or lipid-containing histiocytes utilizing the Sudan IV stain. Microscopic calcification was not encountered. The toluidine blue and colloidal iron stains
Selective carotid arteriograms revealed marked focal bilateral stenoses at the bifurcations of the carotid arteries with poststenotic dilatation. The lesions appeared identical bilaterally.

Discussion
Arterial dysplasias have been increasingly recognized as a cause of occlusive vascular disorders, especially in young patients. McCormick et al. have correlated the arteriographical and pathological findings of vessels involved with fibrous dysplasia. Although their study was concerned with renal arteries in particular, it would appear that their findings are applicable to arteries in general, including the carotid arteries. Lesions producing arterial stenosis have been divided into those affecting the intima (arteriosclerosis and intimal fibroplasia) and those affecting the media (medial fibroplasia with aneurysm, fibromuscular hyperplasia, and subadventitial fibroplasia). Intimal fibroplasia is a lesion of the intima and elastica interna with a segmental circumferential accumulation of cellular collagen (fig. 1). This differs from a circumferential type of atherosclerotic process which may show not only collagen between the endothelium and internal elastic membrane but also accumulations of lipid and often areas of calcification.

The angiographical picture of intimal fibroplasia (a symmetrical narrowing of the artery with poststenotic dilatation [fig. 2]) may be difficult to distinguish from the stenosis produced by arteriosclerosis. It may be differentiated from the “string of beads” arteriogram usually considered to be characteristic of fibromuscular hyperplasia. Although arteriosclerosis and various forms of medial fibroplasia are known to occur in the carotid artery, intimal fibroplasia as a cause of vascular occlusive disease has not received recognition and documentation.

The differential diagnosis of the case herein reported included demyelinating disease, brain tumor, or vascular insufficiency syndrome. The finding of intimal fibroplasia of the extracranial carotid artery, a surgically remedial lesion, adds to the types of occlusive lesions known to produce cerebral ischemia. The necessity of thorough investigation of these patients even in the early decades of life is emphasized.

References
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