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Carotid Restenosis: A Case for EDTA Chelation

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ABSTRACT: Carotid restenosis has been found in up to 25% of patients after carotid endarterectomy. The most common cause of restenosis is continuation of the atherosclerotic process. Surgery can be beneficial in stroke prevention and should be considered in those patients at high risk for stroke. However, surgery does not arrest the disease of atherosclerosis. This report demonstrates a 10% reduction in the degree of stenosis in a patient treated with EDTA chelation for restenosis of a carotid artery after endarterectomy. EDTA chelation does arrest and reverse atherosclerosis and should be used in conjunction with surgery or as primary treatment for carotid restenosis as well as for vascular occlusive disease in any artery whether initial or recurrent.

Introduction

An exciting alternative for the treatment of vascular disease is now a reality. For over forty years many have implicated EDTA as a beneficial treatment for arterial occlusive disease. The chemistry and pharmacology of this synthetic amino acid have been more completely defined. The concept of free radical tissue damage has provided a better explanation of the pathophysiology of most if not all maladies. (1) These two areas of understanding can now be combined to explain abnormalities leading to disease and to design a treatment plan that actually attacks the disease process. This case is presented to illustrate the potential benefit of nonsurgical care of carotid restenosis using EDTA technology which offers a unique approach to reversing

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the effects of free radical pathology as it relates to atherosclerotic vascular disease.

Materials & Methods

This seventy-four year old gentleman presented in March, 1993 with symptoms of cerebrovascular insufficiency. Arteriography confirmed a total occlusion of the left internal carotid artery and 80-85% stenosis of the right internal carotid artery. A right carotid endarterectomy was performed without complication. A post-operative ultrasound was performed according to protocol and was normal until October, 1994, when a 65-70% restenosis was demonstrated. The degree of stenosis progressed to 70-75% by March, 1995. After considering the options of surgery or EDTA chlelation, he chose the nonsurgical approach. The treatments were performed on a weekly basis according to ACAM protocol (2). The Cockcroft-Gault equation was used for dose adjustment. He received a total of twenty treatments and was then placed on a monthly maintenance program. To the carrier solution of 500 cc D_6W was added 1000 units of heparin, 200 mg of magnesium sulfate, 10 cc of 1% Xylocaine, and the EDTA solution. The treatments were given over a three-hour period of time. He also received natural vitamin and mineral supplementation including the antioxidants Vitamin C 1 gram, Vitamin E 800 units, Beta Carotene 20,000 units, Folic acid 800 mcg, Thiamin 13.5 mg, Riboflavin 15.3 mg, Niacin 180 Mg., Vitamin B_6 18 mg, Vitamin B_{12} 54 mcg, Biotin 600 mcg, Pantothenic acid 90 mg, selenium 20 mcg, and zinc 30 mg. These were taken by mouth daily and with each treatment. The only laboratory abnormalities noted were an abnormally low serum selenium and an abnormally high twenty-four hour urinary zinc. His daily selenium intake was increased to 40 mcg and a similar dose given with each infusion. Zinc intake was increased to 60 mg with each treatment in addition to 45 mg daily. The serum zinc remained within normal range. The selenium was successfully restored to normal level. He tolerated the chelation treatments without incident or complication.

Results

The carotid arteries were evaluated before and after twenty EDTA treatments using Siemens Quantum 2000 Triplex Angiodynography. Before chelation, color flow and spectral analysis revealed changes consistent with a 70-75% restenosis of the right internal carotid artery. The systolic velocity was 246 cm/sec with a diastolic velocity of 58 cm/sec. The systolic velocity ratio was 2.6 with a diastolic velocity ratio of 4.1. Spectral broadening was determined to be 30 cm/sec.

After twenty treatments, color flow and spectral analysis demonstrated changes consistent with a 60-65% stenosis in the right inter-

nal carotid artery. The systolic velocity was 220 cm/sec with a diastolic velocity of 58 cm/sec. The systolic velocity ratio was 2.2 with a diastolic velocity ratio of 4.8. The spectral broadening was recorded at 25 cm/sec. Color flow analysis revealed a 10% larger flow channel after EDTA chelation.

These data demonstrate a 10% reduction in the degree of stenosis of the right internal carotid artery with a decrease in both the systolic and diastolic velocities. The decrease in the systolic velocity ratio also demonstrates evidence of improvement regarding this patient's recurrent atherosclerotic carotid disease.

Discussion

Recurrent carotid stenosis following carotid endarterectomy has been reported as low as 3.8% in one series (3). Other authors have reported post-operative recurrent stenosis as high as 25% (4). My experience, after performing two hundred endarterectomies, includes nine restenoses for a recurrence rate of 4.5%. One patient required a second operation which was accomplished without complications. In the immediate post-operative period, carotid restenosis is most commonly caused by surgical technical error. Medial fibrodysplasia is associated with restenosis at one to two years after surgery. Restenosis after three to five years is most likely the result of progression of the atherosclerotic disease process. Conventional medical practice standards favor surgical treatment over medical management for significant carotid artery atherosclerotic disease (5). Surgical endarterectomy can be very effective in preventing stroke when performed in the right setting by a surgeon who can perform the surgery with a low morbidity and mortality rate. Carotid endarterectomy, however, does not treat the disease process as can be determined in part by incidence of recurrent stenosis. Conventional medical management offers nothing to reverse atherosclerosis. Disodium EDTA chelation represents a medical treatment modality that both arrests and reverses the atherosclerotic process. McDonagh, Rudolph and Cheraskin reported fifty-seven patients with cerebrovascular disease who were treated with EDTA chelation. They showed an 18% reduction in the degree of arterial stenosis and 88% of the patients showed objective improvement in cerebrovascular flow (6). Casdorph demonstrated significant improvement in cerebral blood flow studies after twenty intravenous infusions of EDTA (7). Rudolph and McDonagh reported

a single patient with severe carotid occlusive disease, noting a reduction from 98% stenosis to 33% stenosis after thirty intravenous infusions of EDTA (8).

EDTA is known to reduce the number of cross-linkages in connective tissue, improving elasticity sufficiently to allow an artery to dilate. This benefit is in addition to the effect on existing plaque (9). In a vessel with a turbulent flow, such as a diseased artery, a 10% increase in diameter can allow the blood flow to double (10). EDTA chelation removes heavy metals and abnormally situated iron and copper, thus arresting the progress of free radical damage. Other benefits occur from uncoupling of disulfide and metallic cross-linkages. Some of these benefits are: 1) normalization of calcium metabolism, 2) reactivation of enzymes poisoned by lead and other toxic metals, 3) restoration of normal prostaglandin production along blood vessel walls. The restoration of normal prostaglandin production counteracts the spasm and platelet adhesion caused by the unopposed effect of thromboxane (1).

Surgery will most likely continue to serve a significant role in the treatment of carotid atherosclerotic disease in those patients with high grade stenosis and an impending threat of stroke. EDTA chelation provides an exciting approach providing nonsurgical treatment that reduces the degree of blockage. If endarterectomy is necessary, maintenance chelation can be used post-operatively to prevent recurrence. Surgery may be avoided when patients with a strong family history for vascular disease or those who develop atherosclerosis are treated primarily with EDTA chelation. Whether used as primary treatment or in conjunction with surgery, EDTA chelation should be included in the treatment plan because, unlike surgery, it actually arrests and reverses the disease process.

Conclusion

Disodium EDTA chelation for treatment of atherosclerotic arterial disease is an excellent option and in many cases can be considered the treatment of choice. This mode of therapy actually treats the disease process rather than bypassing or dilating the area of stenosis which do not treat the atherosclerotic abnormality. Surgery for recurrent carotid stenosis is not without difficulty and potential complications. Complication from EDTA therapy is negligible if certain guidelines are followed and if there is adherence to the American College

for Advancement in Medicine protocol (2). This report is only a single case study, but does demonstrate significant improvement in both hemodynamics of flow and reduction in degree of stenosis of the right internal carotid artery with EDTA chelation therapy.

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