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Chelation Therapy: for Atherosclerosis

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Abstract

Chelating agents are chemicals containing two or three ligands which are directly able to bind with metal ions to form stable, soluble complexes. In this way the physiological and toxic properties of the metal are neutralized. Being soluble, the complex is passed out in the urine. Typical uses of chelation include deferoximine for iron overload, EDTA for zinc toxicity, dimercaperol for arsenic poisoning and penicillamine for Wilson's disease (copper).

Non-conventional uses of EDTA chelation are a source of controversy. Initially proposed as an effective treatment for atherosclerosis, chelating physicians have promoted EDTA chelation as treatment for a wide variety of other conditions including degenerative brain disorders, arthritis, diabetes mellitus, for reduction of cancer mortality, nonspecific environmental toxin exposure, and in healthy individuals as a "preventative".

There does not appear to be any single coherent mechanism of action for the beneficial claims made of chelation therapy. An initial explanation was that EDTA was able to remove calcium from atherosclerotic plaques. This has subsequently been modified with speculation that EDTA has probably been the "prototype" calcium channel blocker; or that it is a stabilizer of cell membranes (possibly by preventing pathological cross linkages); lowers of blood viscosity; inhibits of platelet aggregation prostaglandin; and modifies synthesis. Finally by removing a wide variety of toxic metals, EDTA is said to prevent excessive free radical formation which is responsible for many pathologic processes, including peroxidation of LDL which may be important in atherogenesis.

No hard evidence is available to support the claims made for chelation therapy. The main body of evidence consists of anecdotal case reports; submissions on the breadth of their experience by chelation experts; testimonials by patients; and a large body of indirect scientific information. A single small pilot controlled study suggested significant benefit.

This has not been substantiated in two larger double-blind, controlled studies (below).

The placebo effect is dismissed outright by chelation physicians, and the beneficial effect of risk factor and lifestyle modification, essential components of the chelation protocol, have not been addressed. "Broad spectrum" vitamins are prescribed, including the antioxidants Vitamin A, b-carotene, Vitamin C and Vitamin E.

A controlled trial from Denmark demonstrated no benefit of chelation therapy in patients with intermittent claudication. Parameters measured included pain-free and maximal

walking distances and the ankle-brachial index. In a subgroup, pre and post arteriograms and cutaneous oxygen tensions were unaffected by chelation.

A more recent double-blind, randomized, controlled study from New Zealand concluded that chelation therapy has no significant benefit over placebo in patients with intermittent claudication. Furthermore, there was no significant difference in performance and perception of activities of daily living.

Based upon the available information no evidence has been presented to substantiate the belief that chelation with EDTA is of benefit in atherosclerosis and in other conditions treated non-conventionally with chelation therapy. Two double-blind, controlled studies conclude that chelation therapy is ineffective.

References

1. A Textbook on Chelation Therapy. New York: Human Sciences Press, Inc., 1989:416. (Cranton EM, ed.; vol 2). [Actually a textbook supplement to The Journal of Advancement in Medicine]
2. Blumer W, Cranton E. Ninety Percent Reduction in Cancer Mortality After Chelation Therapy With EDTA. Journal of Advancement in Medicine 1989;2(1/2):183-188. [Reduced cancer mortality, in individuals chelated for environmental toxin exposure]
3. Casdorff HR. EDTA Chelation Therapy, Efficacy in Arteriosclerotic Heart Disease. Journal of Holistic Medicine 1981;3:53-59.
4. Casdorff HR. EDTA Chelation Therapy. 11. Efficacy in Brain Disorders. Journal of Holistic Medicine 1981;3:101-117.
5. Clarke NE, Clarke CN, Mosher R. Treatment of Angina Pectoris With Disodium Ethylene Diamine Tetraacetic Acid. The American Journal of Medical Sciences 1956;232:654-666. [Chelation Physician's "classic"]
6. Cranton EM. Protocol of the American College of Advancement in Medicine for the Safe and Effective Administration of EDTA Chelation Therapy. Journal of Advancement in Medicine 1989;2(1/2):269-305. [Official EDTA chelation therapy protocol]
7. Lamar CP. Chelation Therapy for Occlusive Atherosclerosis. Journal of the American Geriatric Society 1966;14:272-294. [Another chelation physician's "classic"]
8. McGillem MJ, Mancini GB. Inefficacy of EDTA Chelation Therapy for Coronary Atherosclerosis. The New England Journal of Medicine 1988;318(24):1618-1619. [Case report on patient who underwent quantitative coronary angiography]

9. Sloth-Nielsen J, Guldager B, Mouritzen C, et al. Arteriographic Findings in EDTA Chelation Therapy on Peripheral Arteriosclerosis. *The American Journal of Surgery* 1991;162:122. [Subgroup who underwent arteriography in randomized controlled trial]
10. Guldager B, Jelnes R, Jorgensen SJ, et al. EDTA Treatment of Intermittent Claudication - a Double-blind, Placebo-controlled Study. *Journal of Internal Medicine* 1992;231:261-267. (Randomized control trial)