Smart Life Forum

Dr. Thomas Levy

DEATH BY CALCIUM, THE TOXICITY OF OSTEOPOROSIS THERAPY

Cubberly Community Center
4000 Middlefield Road, Room H1, Palo Alto, California

June 17, 2004 at 7:00 PM

Future Speakers:(on Third Thursdays)

- **July 15**, Dwight Jennings, DDS on Correcting Airway Interference Fields for Improved Oxidation and Health
- **August 19,** Garry Gordon, MD, on Nutritional Uses of RNA Therapies for Management of Many Diseases
- **September 16**, Frank Shallenberger, MD, on Mitochondria Energy Production and Measurement.
- **October 21**, Paris Kidd, PhD, on Ortho-molecular Synergy of Nutrients for Healthy Brain Aging.
- **November 11**, (tentative) Julian Whitaker, MD, on Orthomolecular Treatments for Chronic Diseases.

Mini-Presentation (2nd Monthly "Bio-Med 101")

This month's mini-presentation, starting at 7:00 pm will be by Steven Wm. Fowkes, our esteemed technical advisor and past president. Last meeting, several mechanisms of Alzheimer's disease were presented. John Furber discussed the metals-accumulation hypothesis of Alzheimer's, and the possible role of cloquinol in chelating iron from the brain. Dr. Wong mentioned several potential therapeutic benefits of enzymes to people with neurodegenerative conditions. Also, someone shared an experience with Steve Fowkes about cholinesterase inhibitors (medically prescribed drugs and over-the-counter herbal products) and Alzheimer's disease. Conspicuously missing was a discussion of the role of diminishing mitochondrial function and the decreased availability of ATP and NADH to the maintenance of brain neural function. Steve Fowkes will cover this in a 20 minute mini-presentation at the upcoming meeting. He will present evidence that lack of cellular energy production 1) is the first step in the Alzheimer's cascade and 2) can cause metal accumulation, loss of neurotransmitters, and oxidative stress.

Our Speaker

Meet Dr. Tom Levy (who changed the status of U.S. Vitamin C usage after his best selling book and presentation to us last year. Now he intends to change the status of Calcium usage worldwide, after this presentation and new book due out in December).Dr. Levy received his Bachelor of Arts degree in biology from the Johns Hopkins University in 1972. He later graduated from the Tulane University School of Medicine in 1976. Continuing his training at Tulane, he specialized first in internal medicine and then in cardiology, receiving board certification in both of these disciplines. After completing his postgraduate training, Dr. Levy served as an assistant professor of medicine at Tulane Medical School for another three years. After a private practice of adult cardiology, Dr. Levy started his research on the medical impact of dental toxicity with Dr. Hal Huggins in 1994. In 1998, he received his law degree from the University of Denver and was subsequently admitted to practice law in Colorado and the District of Columbia. Death By Calcium will be his fifth book, to be published by the end of the year. His previous books are Vitamin C Infectious Diseases, and Toxins, Uninformed Consent: The Hidden Dangers in Dental Care, co-authored with Dr. Huggins; The Roots of Disease: Connecting Dentistry and Medicine, co-authored with Robert Kulacz, D.D.S.; and Optimal Nutrition for Optimal Health. For further information on Dr. Levy, you can review his bio on the web at www.TomLevyMD.com

Overview

Many older adults today have much more calcium in their bodies than their osteoporotic bones might indicate. But, much of the calcium is abnormally deposited in tissues other than bone. The bones are thin, while the blood vessels and other tissues are literally lined and filled with rock-like depositions of calcium salts. Strongly consistent with this observation is that most deaths in patients with osteoporosis relate to the vascular system and not to the bones. Clearly, then, aggressive calcium supplementation would not appear to be the answer to restoring the integrity of osteoporotic bones since it further fuels the obstructive deposition of calcium salts in the blood vessels. A slightly decreased risk of an osteoporotic fracture is not worth a greatly increased risk of a fatal heart attack.

The following is an outline of Dr. Levy's talk:

I. Osteoporosis

- Overview
- Typical therapy
- Statistically associated morbidities and mortalities

II. Calcium

- Important nutrient and potent toxin
- Superhype: excess extremely common; deficiency rare; credit (blame) the milk industry
- The role of other toxins in the maintenance of a proper Ca/PO4 ratio and calcium metabolism
- Calcium homeostasis best regulated by non-calcium nutrients and metabolites

III. Vitamin D

- Emerging as an extremely critical vitamin/hormone/nutrient
- Too little as bad as too much
- NEVER to take with calcium; regulate with blood testing

IV. Physiological therapy

- Magnesium

- Vitamin K
- Vitamin C with a wide array of supporting antioxidant therapy
- Vitamin D
- Essential fatty acids
- Proper toxin removal

V. Monitoring the patient for optimizing therapy

- Hair analysis
- Routine blood chemistries
- Coronary calcium score
- Clinical correlations

Basic Vitamin D and Calcium Physiology

Vitamin D really functions as a hormone in the body even though it is usually classified as a fat-soluble vitamin. Generally, vitamins come from the diet and hormones are produced by the body. Vitamin D, however, can both come from the diet and be produced in the body. Dietary sources of vitamin D are relatively few unlike the many dietary sources of other vitamins, and skin exposure to the sun can supply all the vitamin D a given body needs. Data continues to accumulate indicating widespread hormonal functions for vitamin D. Consistent with the definition of a vitamin, however, is the fact that a deficiency of vitamin D will result in a deficiency disease known as rickets in children or osteomalacia in adults. Further complicating proper understanding of vitamin D is the double-edged nature of its effects. Even though too little vitamin D will cause rickets, it has also been demonstrated that feeding animals excessive amounts of vitamin D will cause rickets as well. Excess vitamin D actually moves calcium out of bones. It would appear that a little vitamin D is good, but more is not necessarily better, and a lot is definitely harmful. Although many new hormonal functions are being discovered for vitamin D, the role of vitamin D in maintaining normal levels of calcium and phosphorus in the blood is still regarded as its primary function. This function of vitamin D is primarily achieved by the ability of vitamin D to increase the absorption of both calcium and phosphorus from what is eaten, with the effects on calcium absorption being most pronounced. Vitamin D also works to increase calcium blood levels by enhancing the reabsorption of calcium back from the urine forming in the kidney tubules. When vitamin D is absent or severely deficient for a long enough period of time, the deficiency diseases mentioned above can appear. Bones can become thin and brittle, or even soft and pliable when these diseases become manifest.In addition to its production in the skin, vitamin D can also be obtained

from the diet. Just about the only foods that naturally contain a significant amount of vitamin D are fatty fish, like swordfish or mackerel, eggs, and chicken liver (Neer, 1975). However, the addition of vitamin D to foods and to animal feeds in variable amounts has resulted in an additional, although not easily quantifiable, source of vitamin D for many people. Finally, more vitamin D is ingested by many people through nutritional supplements, either separately as a direct vitamin D supplement or as a component of a multiple vitamin preparation. Not surprisingly, as Calikoglu and Davenport (2003) put it, the amount of vitamin D needed "to maintain adequate calcium metabolism and healthy bone development for all ages is very difficult to determine due to many confounding factors." With such a lack of precision in defining vitamin D intake, the continuing lack of awareness as to the toxicity inherent in excess vitamin D supplementation, and the persistent "fortification" of vitamin D in different foods, it should be easy to appreciate that while some people may not get enough vitamin D on a daily basis, many people get far too much. The impact of such a chronic over-dosage of vitamin D will be discussed later in detail.Hypercalcemia, or an elevated blood level of calcium, can result from excessive vitamin D hormonal effect and from excessive calcium supplementation. The two excesses together, as is very often seen today, will have an even more pronounced effect on keeping calcium blood levels high, with an increased propensity for abnormal deposition in tissues outside of the bone. Normally, of the approximately 1,000 to 2,000 mg of calcium found in the body, 98% is in the bones, 1% is in the teeth, and the remaining 1% is in the extra cellular fluids, the intracellular structures, and the cell membranes. In health, the small amount of calcium outside of the bones and teeth remains in solution, in contrast to the abnormal tissue depositions seen in many people today. While the most obvious effect of calcium is to help provide structural integrity and strength to the bones, calcium also plays very important roles in stimulating enzymatic reactions and cell membrane transport systems. These effects of calcium are involved in nerve excitability and conduction, cardiac and skeletal muscle function, cell membrane permeability, and cellular adhesiveness.

Metastatic Calcification

Metastatic calcification refers to the widespread deposition of calcium salts, usually amorphous calcium phosphate or crystalline hydroxyapatite, whenever the calcium-phosphate solubility product exceeds a defined level (Gupta and Hruska, 1990). It’s not so important to know this level or to remember the definition of a solubility product as it is important to realize that when enough excess calcium and/or phosphorus gets dissolved in the blood, it will start precipitating back out of the blood into various tissues in an inorganic, rock-like form. Allen and Shah (1992) pointed out that when this calcium

deposition occurred, it typically appeared in any of a number of the following locations:

- Brain basal ganglia
- Cornea and conjunctivae
- Fundus of the stomach
- Heart (endocardium and myocardium)
- Kidney
- Lung
- Arterial linings (media)
- Pancreas
- Soft tissue surrounding the joints
- Eardrum and the middle ear bones

Metastatic calcification occurs whenever calcium levels, vitamin D levels, or phosphate levels get high enough for a long enough period of time. Certainly, when more than one of these levels elevate, the tendency toward calcification is even more pronounced. Excessive intakes of vitamin D or one of its analogues in the treatment of various medical conditions have resulted in such metastatic calcification and other toxic effects. Excess vitamin D intake has been documented to result in cardiovascular, renal, and skeletal damage. Infants receiving excess vitamin D due to the routine fortification of foods have consistently had significant toxicity reported. Similarly, laboratory animals have also been demonstrated to suffer significant damage from excess vitamin D. A fatal widespread tissue calcification in two suckling puppies was felt to be secondary to high vitamin D levels in the diet of their lactating mother, an indication of the importance of avoiding excess maternal levels of vitamin D as well.Vitamin D and its metabolic derivatives are important factors in the promotion of metastatic calcification largely because of their effects on calcium and phosphorus absorption in the gut. In effect, giving vitamin D as a supplement is much the same as directly supplementing calcium and phosphorus, since the vitamin D results in a much greater absorption of these minerals from the diet. Since vitamin D increases both calcium and phosphorus levels in the blood, excess vitamin D intake will even more readily promote metastatic calcification than the excess supplementation of calcium alone. Furthermore, vitamin D supplementation in high enough doses will increase bone resorption, a process in which calcium is again removed from the bones, promoting the development of osteoporosis rather than inhibiting it. This process appears to routinely accompany the deposition of calcium salts in the non-bony tissues in the metastatic calcification process. In patients undergoing treatment for osteoporosis with vitamin D, Schwartzman and Frank (1987) concluded that the therapy was only pulling calcium out of the bone and causing a pathological degree of hypercalcemia associated

with kidney failure. They decided that "pharmacologic doses of vitamin D cannot be recommended for any form of osteoporosis." Another research team found that middle-aged males treated with doses of vitamin D ranging from 10,000 to 50,000 IU/day had marked increases in the calcium content of their urine, indicating a leaching of calcium out of their bones (Spencer et al., 1989).Excessive intake of vitamin D also tends to have long-lasting effects, as it can be stored in the fatty, liver, and muscle tissues for many months (Adams, 1989). These tissue stores result in the continued slow release of vitamin D for months after intake or supplementation has been discontinued.

Atherosclerosis, Osteoporosis, Vitamin D, and Magnesium

Moon et al. (1992) suggested that excessive vitamin D intake induces both atherosclerosis and osteoporosis in humans as well as laboratory animals. They noted that the incidence of atherosclerosis rose dramatically in women as they developed osteoporosis. They further noted that the use of vitamin D as a food supplement seemed to coincide with the onset of epidemic rises of both atherosclerosis and osteoporosis. A bit later, Demer (1995) pointed out that new technologies have revealed that most atherosclerotic lesions have mineral deposits in them and that 90% of patients with coronary artery disease have such lesions. This is especially significant since calcification in the coronary arteries had long been regarded as uncommon. Now it is becoming appreciated that much of the calcium lost in the evolution of osteoporosis is being re-deposited in arteries, resulting in significant atherosclerosis. The calcium depositions occurring outside of the bone may represent compensatory bone-forming mechanisms that inadvertently miss their bony osteoporotic targets.Atherosclerotic mineral deposits are predominantly calcium and phosphate, along with some magnesium, iron, and other trace mineral components. This is significant since it demonstrates that just getting calcium and/or phosphorus levels up in the blood and keeping them there will cause precipitates in the blood vessel walls that match the makeup of the typical atherosclerotic plaque. Since vitamin D excess will produce such mineral elevations, it is logical to conclude that an important part of the evolution of atherosclerosis proceeds when vitamin D excess persists.

Tissue Calcification Aggravated by Magnesium Deficiency

Researchers have also shown that the tissue calcification induced by vitamin D excess is further aggravated by a coexistent magnesium deficiency. This interrelationship between vitamin D and magnesium is significant, as it has been observed that one form of rickets (a primary vitamin D deficiency disorder) will not respond to vitamin D therapy until dietary sources of magnesium are increased. Increasing the dietary sources of magnesium has

also been shown to decrease the atherosclerosis in swine induced by excess vitamin D. It would seem that increased intracellular calcium levels is a precursor to and an accelerator of vascular calcification and atherosclerosis. The magnesium-vitamin D interrelationship suggests that supplementation with the right forms of magnesium should prove to be one of the important ways to treat metastatic calcification, a concept which is actually very well supported in the scientific literature.Vitamin D deficiency also appears to be related to the worsening of a possible risk factor for atherosclerosis and heart attack. Iron excess appears to accelerate the progression of atherosclerosis Vitamin D is known to increase iron absorption. Chicks fed a low calcium diet clearly had their iron absorption increased by vitamin D, with elevated levels being demonstrated in blood, liver, and bone. Children with lower plasma vitamin D levels had significantly lower levels of hemoglobin and serum iron. While this does not prove that chronic vitamin D excess will reliably produce chronic iron excess, it does represent an additional mechanism relating vitamin D intake to atherosclerosis aside from just promoting vessel wall calcification.

Tracking Calcium Toxicity

The ability to track the extent of the calcium deposits outside of your bones will give you a very important indication as to whether your health is headed in a positive or a negative direction. An objective decline in the calcium stores in your arteries and other non-bony tissues is a strong indicator that at least most of what you are doing is right. One should always stay open to new information that is well-grounded scientifically and that can promote good health. And then recheck their calcium status several months later to make sure the new intervention has not resulted in any new calcium accumulation.There are a number of different ways to track calcium status. Hair analysis, when properly understood and interpreted in a clinical context, can provide an inexpensive and fairly accurate reflection of calcium status. Echocardiography, or ultrasound of the heart, gives a good reflection of the calcium that has abnormally deposited in the proximal aorta and the valves of the heart. Finally, electron beam computerized tomography (EBCT), a test that gives an accurate coronary artery calcium "score," is now readily available.

Coronary Calcium Content

The calcium score is related to the total atherosclerotic plaque burden within the body. A very low or negative CS (0-10) generally excluded the presence of significant coronary artery narrowings. Tracking increases in this condition leads to coronary c alcification which also predisposes to calcification in other arteries and non-bony tissues.The literature on coronary calcium scoring shows that these levels are not only

associated with atherosclerosis, as noted above, they are also associated with myocardial infarction, or heart attack, as they progress to higher scores on serial measurements (Raggi et al., 2003). The more calcium found in the coronary arteries, the greater chance of death by any cause, including the more obvious cardiac causes. This finding alone strongly indicates that all chronic degenerative diseases eventually leading to death, including cancer, are intimately involved with abnormal calcium deposition throughout the body, as indirectly indicated by the levels of calcium in the coronary arteries. In general, the more calcium, the more disease, and the sooner death will come.While an association does not have to mean a cause-and-effect relationship, the accumulating data does also suggest that increased metastatic calcification, as reflected in higher coronary artery calcium levels, predisposes for any of a wide variety of chronic degenerative diseases. What came first, the calcium or the disease? It is likely that either one might come first, depending upon the patient. Excess calcium can cause disease, and disease can predispose to calcification.

Good-bye to Clara Felix

Recently we lost one of our treasured members. Clara Felix, who died from ovarian cancer at 81, shared her knowledge with us, so that we could understand and utilize it to improve our lives. The Felix Letter was a labor of love for Clara. Many of these newsletters appear on our website. Before her death, Clara and Sue Richfield were in discussions to compile her newsletters into a book. Clara's son is now interested in carrying on this project. If anyone would like to participate, please contact Sue@smart-life.net. Clara will be missed by all.