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Arresting bone loss in a post-menopausal osteoporotic woman without the use of bisphosphonates: a case report

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ABSTRACT

There are many dietary recommendations to assist with the treatment of osteoporosis, and some appear to be helpful. However, as of this date no single dietary recommendation has emerged as a replacement for bisphosphonates. This study is a single case study that explored the effects of dietary treatment of a 71 year-old woman with advanced osteoporosis, a fifteen-plus year history of consistent and progressive bone mineral density (BMD) decline and loss of one inch in height. Over the fifteen-year course of illness the patient has consistently refused bisphosphonates. She came to the professional clinical practice of the researcher asking about possibilities for herbal or nutritional intervention and was accepted as a patient. This report describes the dietary intervention and the results. After fifteen months of herbal, mineral and dietary treatment the bone density scan test (DXA) showed zero bone loss.

Keywords: Osteoporosis, Bone Loss, Bisphosphonates, Vitamins, Isoflavones

1. Introduction

There are many dietary recommendations for bone health, and specifically for post-menopausal women dealing with bone mass density decline. Among the recommendations include vitamin D supplementation, calcium supplementation, and more recently vitamin K supplementation. Studies of supplementation with both vitamin D and calcium range fewer hip fractures to ineffective, and are therefore generally considered adjuncts to treatment [1]. It has also been recognized that sufficient protein is needed for osteoporosis prevention and treatment, although protein alone is also not a treatment for bone density loss [5].

Vitamin K and isoflavones are also considered as alternative therapies. Vitamin K plays a role in synthesis of bone proteins such as osteocalcin, which is involved in mineralization. Isoflavones have shown clearly to be useful in bone loss prevention; short-term RCTs indicate that there may be benefit with as little as 40-60mg isoflavones from soy or red clover [4]. However, even taken collectively, all of these substances have not been able to reduce the loss of bone density in Western patients.

2. The Case

A 69 year old woman presented in the professional practice office of the researcher in 2011 with long-term bone loss. Her bone density scan tests (DXA) records dated from 1999. They showed a steady loss of bone mass density in the spine – Table 1 - and femur, and a slower rate of decline in the hip. She reported no history of fracture. Further, the patient reported no use of hormones at any time in her life including no hormone replacement therapy.

Table 1: Bone mass density data for case study patient 1999-2013

Spine	1999	2001	2009	2011	2013
T score	-1	-1.7	-2.7	-2.9	-2.9

Having routinely declined Alendronic acid, the patient was advised by her physician to undertake a course of Zoledronic acid injection. She was unwilling. She sought the advice of the researcher about treatment for osteoporosis that did not include the use of bisphosphonates.

3. Discussion

Anticipated bone loss from the decline in hormone production in post-menopausal women

appears to be approximately +/- 5% as measured against FSH stage 4 [21]. The patient, who is the subject of this study, and many patients in the West, present upwards of this data as they age, and suffer relatively high rates of fracture [15]. Medicare's expenditure on fracture from falls exceeded $19 billion in 2000 [23].

Fracture rates can be traced and show equality across the US and much of Western Europe. However, fracture rates are significantly lower in China and India [15]. Given the distinctly different dietary cultures between the East and West, these patterns suggest that one of the root causes of high fracture rates in the West is diet.

The researcher designed a dietary protocol based on a multi-pronged approach towards calcium absorption and avoidance of bone resorption. Milk provides an excellent source of protein and calcium, as well as phosphatase. Components of milk are thought to favor the intestinal absorption of calcium by keeping it in a soluble form until it reaches the distal intestine, where it can be absorbed by unsaturable routes that are independent of vitamin D. The best known are lactose proteins and phosphopeptides [4].

Many *in vivo* and *in vitro* studies on proteins and phosphopeptides have demonstrated a positive effect of these molecules on calcium absorption [4]. Phosphopeptides–in particular those derived from the enzymatic hydrolysis of caseins - have been shown to sequester calcium [4]. Phosphopeptides therefore help to keep calcium in solution until it reaches the distal intestine, facilitating its absorption by passive diffusion.

It is now clear that lactose, like other slowly-absorbed sugars, must be at the site of absorption. In this case it prolongs the passive, vitamin D-independent absorption of calcium in the ileum. The effects may be spectacular, doubling absorption, if a high dose of lactose (15% to 30% of intake) is given [4].

Intestinal absorption does not necessarily reflect bio-availability because calcium must be retained and used in bone formation and mineralization. Phosphorus must also be present for the production of hydroxyapatite. The dissociation of calcium intake from that of phosphorus (if, for example, the calcium source is not ingested with the meal and/or this source contains no P) may restrict bone mineralization [4]. Unfortunately the high heat of pasteurization denatures both alkaline phosphatase and milk protein [3]. Lack of high-heat pasteurization should allow calcium to be more bio-available as both the phosphatase and the structure of the protein would be intact. The researcher's assumption is that along with continued vitamin D supplementation, calcium absorption would become more efficient with the consumption of unpasteurized milk.

While there are scare figures associated with unpasteurized milk, there are 515 times more illness from *L-mono* in delicatessen meats and 29 times more illness from *L-mono* in pasteurized milk. On a per-serving basis, delicatessen meats were ten times more likely than unpasteurized milk to cause illness [20]. The patient resides in a state where it is legal to buy unpasteurized milk from a farm that holds a Dairy Farm Certificate of Registration and meets bottling, testing and inspection requirements of the state's Department of Agricultural Resources.

Based on the above assumptions, the researcher formulated a breakfast that included the additional vitamins and minerals required for ideal bone health. The breakfast comprises unpasteurized milk plus six organic seeds, several types of nuts, two dried fruits, fresh fruit and unsulphured blackstrap molasses. In combination this provides high natural forms of calcium as well as magnesium and phosphorous and a small amount of phytoestrogen; also high levels vitamin E, balanced trace minerals and vitamin A. Additionally the researcher instructed the patient to reduce intake of grains which are high in phytic acid [7]. Phytic acid binds with calcium and other minerals rendering them unavailable. For the same reason, the researcher instructed the patient to eliminate soy products that are not fermented, and to add fermented soy to her diet. Unfermented soybeans have relatively high levels of phytic acid. The fermentation process reduces the phytic acid substantially and frees the isoflavones [6]. The researcher also asked the patient to use only sprouted nuts and seeds, as sprouting reduces the naturally-occurring phytic acid in both.

It is shown that isoflavones – specifically when daidzein and genistein are metabolized by intestinal bacteria into equol – inhibit bone resorption [6, 8]. Evidence from animal studies consistently shows that appropriate doses of isoflavones inhibit bone loss in osteoporotic animals without exhibiting adverse effects on reproductive organs or serum hormone levels [6, 8].

The researcher also instructed supplementation with vitamin K. Vitamin K is a necessary ingredient in bone building [12, 13]. In animals Vitamin K2 is the main storage form of Vitamin K and has several subtypes. Bacteria in the colon convert K1 into K2. Subtype Menaquinone-4 is synthesized by animal tissue and is found in meat, eggs and dairy products. Menaquinone-7 is synthesized by commensal gut bacteria or during fermentation. Osteoporosis [16, 17] and coronary heart disease [18] are strongly associated with lower levels of K2. Vitamin K2 (MK-7) deficiency is also related to severe aortic calcification and all-cause mortality [19].

The mineral-binding capacity of osteocalcin requires vitamin K-dependent gamma-carboxylation of three glutamic acid residues. Epidemiological studies have demonstrated a relationship between vitamin K and age-related bone loss (osteoporosis) [7, 13].

The Nurses' Health Study followed more than 72,000 women for ten years. In an analysis of this cohort, women whose vitamin K intakes were in the lowest quintile (1/5) had a 30% higher risk of hip fracture than women with vitamin K intakes in the highest four quintiles. A study in over 800 elderly men and women, followed in the Framingham Heart Study for seven years, found that men and women with dietary vitamin K intakes in the highest quartile (1/4) had a 65% lower risk of hip fracture than those with dietary vitamin K intakes in the lowest quartile (approximately 250 mcg/day vs. 50 mcg/day of vitamin K) [9, 15].

The patient was enthusiastic and reported full compliance. After fifteen months in total the DXA scan showed zero bone loss.

4. Bone-Building Plan

The next step is to increase bone mineral density (BMD). Toward that end the patient has begun a program that includes the following: breakfast as before, natural forms of vitamin K2 including both MK-4 and MK-7, bone broth, a bone-building vinegar containing Gu Sui bu, Xu duan, Bu gu zhi, Du zhong, Stinging nettle, Horsetail, Red clover and Pu gong ying. Documentation includes CTX, Bone-specific alkaline phosphatase, propeptide type 1 collagen (PICP), and Serum N-terminal osteocalcin. These tests will be repeated in six months. The DXA scan will be repeated in fifteen months.

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