

Nutritional Factors are not Sufficient to Check Osteoporosis in Women after Menopause- A Justification

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Abstract

Osteoporosis in women after menopause is a common disease. It is extremely painful disease which shows frequent fracture of bones especially in vertebrae, especially in very elderly women usually over 62 to 65 years of age. Many orthopedics advise them to consume more calcium rich foods, to ameliorate the painful suffering. Therapeutically, these patients are given chewable calcium tablets or syrups or intramuscular calcium injections also containing Vitamin D3. In either case, it is of very little help. In fact those patients, prone to renal calculi, will have to suffer an additional pain. This article attempts to justify why nutritional factors alone is not sufficient to help these women.

Keywords: Osteoporosis; Menopause; Calcium; VitD3; Osteogenesis

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Introduction

It has been observed in many literature surveys, in the field of Nutrition that women who are suffering from osteoporosis after menopause, are given high oral dose of calcium which is also followed by parenteral administration of calcium. However, in spite of such calcium administration mostly by the oral route (nutritional mode), these women show very insignificant recovery from osteoporosis. This article is aimed at an attempt to summarize, as to what might have gone wrong in such a calcium therapy.

Here are excellent reviews that explain how such a suffering can be minimized for these elderly women by having diet rich in calcium (Swaminathan, 1999). It has been observed in-vitro studies that both osteocytes and osteoblasts are responsible for osteogenesis of mesenchymal stem cells found in bone (Birmingham., *et al.* 2012). Remembering the basic biochemistry involved in calcium absorption and desorption during osteogenesis of these stem cells of the bone, it is observed that Vitamin D3 (cholecalciferol) plays the most important role. It is usually found in the fatty deposition of the liver. It is actually not a vitamin it is a steroid with one ring open and hence it is called as secosteroid. It is found in liver oils of certain fishes like Cod or Shark. Basically it serves as an antioxidant preventing certain cancers like colorectal cancers in mice (Wactawski-Wende., *et al.* 2006). This vitamin is found in only a few food items and mostly made by UV light from sunlight using the cholesterol in the skin.

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Role of Vitamin D3

Therefore, as such this compound (Vitamin D3) does not affect absorption and desorption of calcium by the bones. It must undergo certain metabolism like formation of calcifediol (25 hydroxycholecalciferol) by the endoplasmic reticulum of hepatocytes. This is then passed onto the kidney where it further hydroxylated to form $1\alpha, 25$ dihydroxycholecalciferol (Calcitriol) (Norman, 2008). This is the active form of vitamin D3, which is responsible for absorption and desorption of calcium in the bones and this is now a hormone. Deficiency of this can cause hyperparathyroidism, hyperphosphatism and Fanconi syndrome (Hamilton, 2015). Excessive intake of the inactive Vitamin D3, can result in nausea, vomiting and formation of renal calculi of kidney in certain patients. This will leave the readers, asking why not administer calcitriol directly in the patients? That is not possible due to certain important pharmacokinetic and pharmacodynamic reasons.

Conclusions

First of all it must be remembered that nutrition or diet plays the least significant role in prevention of osteoporosis. Calcitriol formation is triggered by parathyroid hormone when it senses a low level of Ca^{2+} in the blood. A low level of Ca^{2+} indicates hypoglycemia (a condition where blood glucose level falls too low and the patient can become unconscious if not attended). Calcitriol formed in the kidney will send back the Ca^{2+} from the kidney either in the blood or in the bones. In case of menopausal women, where there is lack of the hormone like estrogen, there will be more osteoclastogenesis resulting in inhibition of bone formation as there will be increased osteoclastic resorption (instead of apoptosis of osteoclasts), which at times also shows symptoms of osteoporosis (Horowitz, 1993). Therefore, in addition to nutritional factors, possibility of other therapeutic measures like hormone replacement therapy should also be considered.

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