USANA Technical Bulletin

Osteoporosis

General Description

- Osteoporosis is the most common type of metabolic bone disease. In this disease, the rate of bone resorption accelerates, while the rate of bone formation decelerates, resulting in a decrease in bone mass.
- Bones affected by this disease lose calcium and phosphate, this causes the bones to become porous and brittle, and increases the risk of bone fractures.
- Osteoporosis may also be caused secondary to an underlying disease or disorder.[1]
- After peak bone density is reached, it remains stable for many years, then gradually declines as aging occurs, increasing the risk for osteoporosis.
- Evidence suggests that bone loss begins before menopause in women, and in men, bone loss may begin between ages 20-40.[2]

Types

- Type I (or postmenopausal) osteoporosis, usually affects women ages 51 to 75. It is related to the loss of estrogen's protective effect on bone. Vertebral and wrist fractures are common with type I osteoporosis.
- Type II (or senile) osteoporosis, most commonly occurs between ages 70-85. Fractures of the humerus, tibia, femoral neck, and pelvis are common with type II osteoporosis.[1]

Causes

- The exact cause of primary osteoporosis is unknown. However, researchers suspect that there are contributing factors involved in the development of this disease.
- Some of these contributing factors include calcium and mineral intake, sex, race, heredity, and lifestyle (e.g. nutrition and exercise), and the typical western diet (high in protein, salt and refined and processed foods).[3,4]
- Type I: the mechanism of estrogen deficiency leading to bone loss is not well established. Estrogen stimulates the production of growth factors that stimulate bone formation. Recent evidence suggests that an estrogen deficiency may increase the production of bone resorbing cytokines and tumor necrosis factor. This diminishes bone formation and increases skeletal sensitivity to the resorption effects of parathyroid hormone(the regulator of calcium and phosphorus metabolism). It is

hypothesized that this action may decrease renal 1,25-$(OH)_2$ vitamin D formation and limit calcium absorption in the intestines.

- Type II: the ability of the kidneys to make 1,25-$(OH)_2$ (vitamin D hormone) along with decreased intestinal sensitivity to 1,25-$(OH)_2$, causes diminished calcium absorption and a decrease in osteoblast (a bone forming cell) formation.[2]
- Other factors that increase the risk for developing osteoporosis include: pharmacological therapies, alcoholism, liver disease, malabsorption syndromes, scurvy, lactose intolerance, hyperthyroidism and other bone diseases.[1]

Prevention and Management

- Strategies aimed at preventing osteoporosis include optimizing peak bone mass, preventing bone loss at menopause and aging, thereby increasing bone quality. Other important factors include genetics, nutrition, and lifestyle.[5]
- Exercise can positively influence bone density, making the bones less susceptible to breaking. In the elderly, falls are often precipitated by fracturing of weakened bones. Exercise interventions in the elderly population have been reported to decrease fall frequency by 10%.[6]
- Estrogen replacement therapy is commonly prescribed for postmenopausal women.
- Calcium deficiency contributes to inadequate bone mineralization. Supplementing with calcium may slow bone loss in post menopausal women by 30 to 50%.[9]
- Manganese may play a role in bone development and remodeling[7]. There are reports that women with osteoporosis tend to have low levels of blood manganese.[8]
- Vitamin D plays a role in the regulation of calcium deposition (bone mineralization) and mobilization in bone tissue. Vitamin D levels are typically lower in the elderly population.[9]
- One animal study has shown that vitamin E offers protection against cellular lipid peroxidation in cartilage, thereby sustaining normal bone growth.[10]
- The vitamin K dependent proteins in bone appear to be involved in bone crystal formation and bone remodeling, this suggests a potential role of vitamin K in the treatment and prevention of osteoporosis.[11]
- Trace minerals may be important in maintaining bone quality through their role as metallo-enzymes in the synthesis of collagen and other proteins involved in bone formation.[12]

Abstracts

Fujita T. Vitamin D in the treatment of osteoporosis revisited. Proc Soc Exp Biol Med 1996 Jun;212(2):110-5. Interest in vitamin D treatment for osteoporosis has recently been revived because of the focus in various parts of the world on the elderly population, which is predominantly vitamin D deficient, in addition to postmenopausal osteoporosis due to estrogen withdrawal, which has been the central theme of osteoporosis research for many years. Combined use of other agents along with vitamin D has fortified the therapeutic armory against osteoporosis. The recent suggestion of a role of vitamin D receptor polymorphism in the development and progress of osteoporosis, possibly by interfering with its expected action, provoked intense discussions on the role of vitamin D in the pathogenesis and treatment of osteoporosis. Vitamin D receptor polymorphism may explain some of the racial differences in the incidence of osteoporosis and its complications. Responses to vitamin D treatment may also be predicted

by vitamin D receptor allelic analysis, though the currently proposed allelic patterns are yet far from being widely accepted. The outlook for vitamin D treatment for osteoporosis may require insight into vitamin D receptor, not only for vitamin D's given form, but also for a possible future form designed to intervene at the genomic level.

Kaufman JM. Role of calcium and vitamin D in the prevention and the treatment of postmenopausal osteoporosis: an overview. Clin Rheumatol 1995 Sep; 14 Suppl 3:9-13.
When discussing the use of calcium and vitamin D in the prevention and the treatment of osteoporosis one can make a distinction between the use as dietary supplementation to correct or prevent deficiencies, and the pharmacologic use of higher doses, whether or not in association with other drugs. However, in practical terms it is not always possible to clearly make this distinction. Available evidence suggests that increasing the calcium intake can favourably affect the build-up of bone mass in adolescence. In this population, the daily consumption of calcium in the diet should, optimally, be at least 1200 mg/day. In view of the lack of data pertaining to the effect on the final peak bone mass, there is at present time no basis for the systematic administration of calcium supplements to healthy children and adolescents. Calcium supplementation, aiming at a total calcium intake of at least 1500 mg/day, has a partial protective effect on postmenopausal bone loss, this effect being documented mainly in women more than 5 years after menopause. In the present state of our knowledge, there is no established role for vitamin D supplementation in the prevention of postmenopausal osteoporosis, except in elderly patients presenting with a higher risk for relative vitamin D deficiency and with low calcium intake. The results of a controlled trial suggest that in institutionalised elderly patients, systematic administration of calcium and vitamin D supplements can substantially reduce the risk of hip fracture. In the treatment of established postmenopausal osteoporosis, calcium supplementation has only a role as a general adjuvant therapeutic measure and as a specific complement to the treatment with other active compounds. There are indications that treatment alpha-calcidol or calcitriol has a positive effect on the evolution of bone mass, but awaiting further confirmation of a favourable effect on the incidence of osteoporotic fractures, treatment with these drugs remains experimental.

References:

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