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A review on decreased bone mineral density in chronic alcoholics.

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ABSTRACT

A significant percentage of population is at risk of alcohol's harmful effects on bone. The maintenance of healthy bone in human adults occurs through a process called" bone remodeling". This process can be interfered on long term consumption of alcohol, resulting in decreased bone density and increased risk of fracture. Heavy drinking is well known to be associated with osteoporosis and osteoporotic fractures in chronic alcoholic. Epidemiologic studies have shown that long-term heavy drinkers have multiple risk factors for bone loss, including low dietary calcium and other nutritional deficiencies, low body weight, smoking, and a high caffeine intake. Alcohol is directly toxic to bone and may disrupt bone metabolism. Those effects may be exerted directly or indirectly through the many cell types, hormones, and growth factors that regulate bone metabolism. Even more provocative, some recent studies find that moderate intake of alcohol may actually protect against the loss of bone mass that characterizes the disease osteoporosis. Despite the difficulty of establishing with certainty how and to what extent alcohol affects bone and the risk of fracture, this issue is an important one for public health. Modern-day scientific research on fracture prevalence in alcoholic subjects is based for the most part on small, inadequately controlled studies composed mostly of men. "Many people know about alcohol's effects on the liver and the damage it can cause to this organ after years of heavy drinking," said Terrence M. Donohue, Jr., VA Research Career Scientist at the Omaha VA Medical Center and professor of internal medicine at the University of Nebraska Medical Center. "Considerably fewer people know about alcohol-induced bone disease."

Key words: Alcohol, Bone mineral density, Alcoholics.

SKELETAL GROWTH

The process of skeletal growth and maturation involves three phases:

a) Growth and remodeling
b) Consolidation
c) Remodelling

Bone is a highly specialized supporting framework of the body, characterized by its rigidity, hardness, and power of regeneration and repair. It protects the vital organs, provides an environment for marrow (both blood forming and fat storage), acts as a mineral reservoir for calcium homeostasis and a reservoir of growth factors and cytokines, and also takes part in acid–base balance [3]. Bone remodeling is a dynamic, lifelong process

in which old bone is removed from the skeleton and new bone is added [8]. It consists of two distinct stages – resorption and formation – that involve the activity of special cells called osteoclasts and osteoblasts. Usually, the removal and formation of bone are in balance and maintain skeletal strength and integrity.

Between the age 20-40yrs bone density begin to decline and cumulative decline in the skeletal mass leads to fractures. Women experience accelerated decrease in the bone density following menopause.

Bone is composed of support cells, namely, osteoblasts and osteocytes; remodeling cells, namely, osteoclasts; and non-mineral matrix of collagen and non-collagenous proteins called osteoid, with inorganic mineral salts deposited within the matrix. During life, the bones undergo processes of longitudinal and radial growth, modeling (reshaping), and remodeling (Clarke 2008). Longitudinal growth occurs at the growth plates, where cartilage proliferates in the epiphyseal and metaphyseal areas of long bones, before subsequently undergoing mineralization to form primary new bone.

ALCOHOL AND BONE HEALTH

The holiday period is traditionally lubricated by alcohol and, as the holidays draw to an end and the empty bottles stack up in the recycling bin, thoughts turn to the toll that drinking has taken on the body. Although many people know about alcohol's effects on the liver, far fewer are aware of the adverse effects it has on bone. Alcohol decreases the estrogen levels and increases the two bone damaging hormones cortisol and parathyroid hormone. Alcohol kills the bone forming cells called the osteoblasts.

Achieving optimal peak bone mass during adolescence may reduce a person's risk of developing osteoporosis. Although peak bone mass appears to be largely under genetic control, it can be influenced by hormonal, environmental and lifestyle factors including tobacco and alcohol consumption.

The longitudinal growth rate and the rate of proliferation of cells in the growing region near the end of long bones stop during long term alcohol consumption. The decreased bone mass that occurs from early long term alcohol consumption could result in increased fractures and early onset of osteoporosis.

DISCUSSION

Schnitzler and Solomon 1984 stated that alcohol administration reduced bone formation and increased bone resorption. Human studies examining the relationship between alcohol abuse and markers of bone resorption have been somewhat contradictory. Several studies reported increased bone resorptive markers in both long-term alcoholic patients as well as during alcohol withdrawal (Laitinen et al., 1994; Nyquist et al., 1996; Pepersack et al., 1992). Dr.Vernejoul 1983, concluded that alcoholic osteoporosis is characterized by decxreased bone formation .Chappered et al 1991 conducted a microscopic analysis of bone tissue from men with osteoporosis and confirmed that alcohol leads to delayed amd impaired osteoblast activity associated with normal osteoclast function. Peng et al 1991 carried out an experiment in rats to measure a protein osteocalcin. It is a protein secreted by osteoblast during bone resoprtion. Peng reported decrease in osteocalcin level in response to alcohol administration suggesting that alcohol decreases osteoblastic activity. Sampson et al 1997 and 1998 demonstrated microscopic studies of bone tissues in rats which showed decreased trabecular bone volume, decreased number of osteoblast and decreased rate of bone formation and mineralization leading to inhibition of osteoblast proliferation which is indicative of osteoporosis. Klein et al 1997 conducted experiments in rats. The findings in rats agree with in vitro studies that demonstrate diminished osteoblast number and osteoblast function in humans. Conn 1985, Mathew 1992 and Sellee 1985 stated in their studies that alcohol abuse confer a high risk of skeletal facture. Saville 1965 was the first to identify the association between osteopenia and alcohol abuse

Apart from the harmful effects of alcohol, there are certain protective effects of social drinking. Several studies showed that chronic alcoholism leads to osteopenia and increased incidence of skeletal fractures. Studies carried out to understand the underlying mechanism suggested that alcohol may have a direct effect on bone cells and an indirect or modulatory effect through mineral regulatory hormones. Alcohol has been shown to

decrease the bone formation rate by decreasing the osteoblast number, osteiod formation, and osteoblast proliferation. However, the effect of alcohol consumption on bone resorption has not been established clearly. Some histomorphometric studies showed increased bone resorption in moderate and heavy drinkers, whereas others found no effect. In addition, calcitropic hormones, including serum parathyroid hormone (PTH) and vitamin D metabolites, were reported to be altered by alcohol consumption.

There are conflicting reports in the literature regarding the effect of moderate alcohol consumption on bone. Some researchers did not find a positive association between moderate alcohol intake and bone mineral density (BMD). However, recent studies, mostly in postmenopausal women, showed a positive correlation between BMD and moderate alcohol consumption. Holbrook and Barrett-Connor [26] reported that social drinking is associated with higher BMD in both men and women. Similar observations were made in a study by Felson et al, who found that elderly women with an alcohol intake of ≥210 mL (7 oz)/wk had higher BMD than did nondrinkers at most sites tested. Fescaniks et al [28] also showed a linear increase in spine density over increasing categories of alcohol intake in postmenopausal women.

CONCLUSION

Although available evidence suggests a favorable effect of alcohol consumption on bone density, a precise range of beneficial alcohol consumption cannot be determined. The degree to which alcohol contributes to osteopenia in the entire population is not yet known. Intriguing data at lower levels of consumption, however, suggest that more modest alcohol consumption is less likely to be associated with low bone density and may even be associated with higher bone density. Moderate alcohol intake may affect endogenous hormone levels, to indirectly augment skeletal mass. However, the evidence for a protective effect of moderate alcohol consumption is not entirely compelling and should be interpreted with caution. Experiments using well-defined osteoblastic model systems indicate that the observed reductions in bone formation result from a direct, antiproliferative effect of alcohol on the osteoblast itself.

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