

OSTEOPOROSIS AND PERIODONTAL BONELOSS

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ABSTRACT

Periodontal diseases are characterized by alveolar bone loss resulting in mobility and tooth loss, several conditions like Periodontitis and other periodontal inflammatory conditions are implicated in alveolar bone loss leading to mobility and tooth loss, recently systemic condition like diabetes is also found to play an important role in causation of this kind of bone loss, in this perspective it has become necessary to evaluate whether diseases like osteoporosis which is effecting a very large percent of population world over is having any role in aggravating the periodontal bone loss in already affected patients, although some authors believe that osteoporosis acts as a cofactor in periodontal bone loss, to date there has been no clear cut evidence/study which directly correlates osteoporosis as an aggravating systemic condition in periodontal bone loss.

KEY WORDS: Osteoporosis, Periodontal disease, Bone loss.

INTRODUCTION

Osteoporosis is a disorder characterized by a generalized low bone mass. Osteoporosis is presently defined based on measurements of bone mineral density (BMD) taken at the femoral neck of young adults. The World Health Organization (WHO) ¹ has established four diagnostic levels for BMD as follows 1.Normal, 2.Osteopenia, 3.Osteoporosis and 4.Established Osteoporosis.

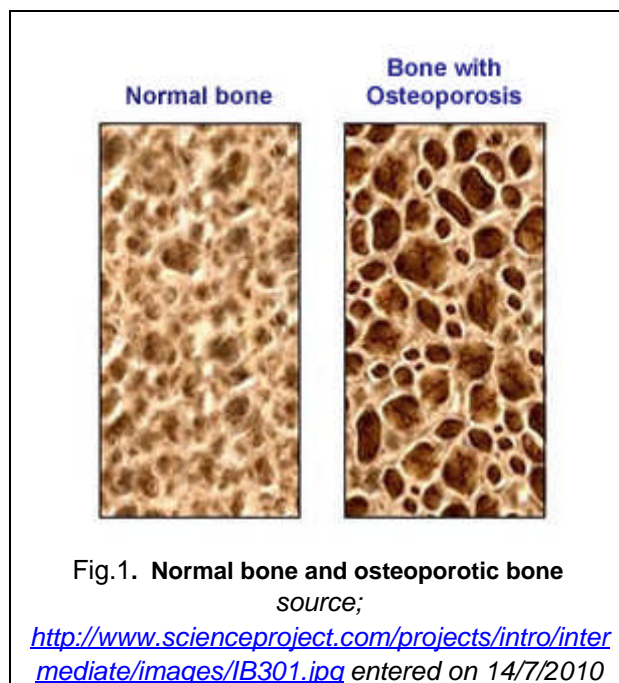
Although bone fracture was considered as essential criteria for diagnosis of osteoporosis, now a low BMD level is taken as a factor for diagnosis of osteoporosis. The diagnostic categories are based on BMD and the presence or absence of fractures.

The WHO committee has established a set of values and standard deviations (SD) for those values, based on young healthy individuals. The SD represents statistical variations 1. Osteopenia is a BMD between 1 SD and 2.5 SD below average for young adults. 2.osteoporosis is a BMD lower than 2.5 SD below average for young adults. 3. Established osteoporosis is a BMD lower than 2.5 SD in the presence of fractures.

Pathogenesis

The pathogenesis of osteoporosis is complex. The adequate health of bones is maintained by a precise balance between bone apposition by osteoblasts and bone resorption by osteoclasts. In childhood and adolescent period bone formation exceeds resorption, resulting in continued skeletal growth and denser, longer and heavier bones. This process slows down in adulthood, and peak bone

mass is attained at about 30 yr of age. After this, resorption begins to exceed formation. Normal bone loss averages 0.7 per cent per year. In females, it gets accelerated at the time of menopause to 2-5 per cent per year, which may continue for up to 10 years. Since cancellous bone is much more metabolically active than cortical bone, in periods of accelerated bone loss cancellous bone loss is 3-fold greater. Osteoporotic fractures, therefore,



commonly occur in vertebrae. Peak bone mass is primarily determined by genes but may be modified

to a considerable extent by certain factors like physical activity, calcium, vitamin D nutrition, smoking, alcohol, concurrent illnesses, and medications (glucocorticoids, antiepileptics).

The level of peak bone mass achieved at puberty is a major determinant of bone mass in later life and hence an important factor in the ultimate development of osteoporosis.

Aetiology and Risk factors (The cause(s) of osteoporosis)

Genetic factors: Studies have suggested that a major genetic component responsible for bone mass may be linked to polymorphism in the gene for vitamin D receptor (VDR)²

Advances made in the genetics of osteoporosis may have implications for racial differences in the clinical spectrum of disease. Since VDR gene may be a determinant of bone mass, differences in VDR gene polymorphism in different races could account for differences in bone mass. Polymorphism of the alleles of the vitamin D receptor gene may account for the major part of the heritable component of bone density in women, possibly mediated in part by impaired calcium absorption from the bowel but this association has not been found in group of men³. A recent study by Mitra *et al*⁴ also revealed that VDR gene polymorphisms were associated with BMD in postmenopausal Indian women and may influence determinants of bone metabolism. Another study done by Vupputuri *et al*⁵ reported that variation in BMD at spine and forearm was related to parathyroid hormone levels and VDR gene polymorphisms and at hip to vitamin D deficiency in vitamin D deficient/ insufficient urban Asian Indians. In addition, estrogen receptor α (ER α) gene polymorphisms may also be associated with BMD in Indian women and may influence some determinants of bone metabolism resulting in accelerated age related bone loss⁶.

Especially in women, estrogen deficiency is the principal pathogenic factor for osteoporosis. Several risk factors contribute to low bone mass. These include **non-modifiable** factors like **female sex, old age, small thinly built physique, Caucasian/ Asians and family history of fractures**. Ethnic differences in bone mineral density (BMD) are strongly influenced by body weight. Important **modifiable risk factors** include **calcium and vitamin D deficiency, sedentary life style, smoking, excessive alcohol and caffeine intake**. Studies have shown that postmenopausal women

having weight <60 kg, height <155 cm has significant risk of osteoporosis and regular consumption of milk, almonds, fruits as protective factors. Calcium intake, increased body mass index (BMI) and higher activity levels gives a significant protective effect on hip fracture in urban north Indian population^{5,6}.

Relationship between nutrition and osteoporosis

Calcium intake is of vital importance and it can be obtained by a proper diet. Dietary supplements of calcium are available in either chewable tablets or liquid form. Another dietary supplement is vitamin D (25 hydroxycholecalciferol) obtained as an oral tablet and as inject-able form. Intramuscular injection of vitamin D can vary from once a year to once a month. Proper serum levels of calcium and vitamin D are the two most important factors to achieve and maintain adequate bone density. It has been indicated that the risk of fractures, especially in the elderly, can be diminished with an adequate diet in calcium and vitamin D.

On the other hand, decreased agility increases the risk of hip fracture. Medical conditions like hypogonadism, thyrotoxicosis, Cushing syndrome, anorexia nervosa, malabsorption syndromes, chronic liver and renal disease, drugs like glucocorticoids and anticonvulsants, and chronic inflammatory conditions like rheumatoid arthritis may lead to secondary osteoporosis.

Is there any sex predilection? If so, why?

Forty percent of women above age 65 in Western societies present signs and symptoms of osteoporosis. Based on BMD measurements provided by a nationwide health survey it is known that in USA osteoporosis affects 6 million women and 2 million men; while osteopenia is reported as affecting 17 million women and 9 million men. The risk for an American man to suffer a bone fracture as a consequence to osteoporosis is higher than that of developing a prostatic carcinoma. It has been reported then 33% of osteoporotic men that fracture a hip die within a year of the fracture.

Diagnosis

Dual energy X-ray absorptiometry (DEXA) technology is the gold standard for diagnosing osteoporosis by measuring bone density.

Treatment

Pharmacologic agents can diminish the risk of fracture further than that obtained by dietary supplementation of calcium and vitamin D. Treatment with various medications is recommended for patients with osteoporosis in conjunction with intake of adequate level of calcium and vitamin D. Several therapeutic modalities are in use either individually or in combination such as, calcitonin, hormone replacement therapy (HRT), raloxifene, which is an estrogen receptor modulator (SERM) and several bisphosphonates.

Raloxifene has been proven to reduce the risk of vertebral fractures in around 30% of patients. Raloxifene does not prevent hip or other than vertebral fractures.

Bisphosphonates are synthetic analogs of pyrophosphate which bind to hydroxyapatite and they act as specific inhibitors of osteoclast-mediated bone resorption. The three best known to be the most efficient in the prevention of fractures in osteoporotic patients are alendronate, risedronate and zoledronic acid.

A recent publication reports that a single annual infusion of zoledronic acid (the most potent bisphosphonate) can achieve similar results as those obtained with other bisphosphonates administered daily for the treatment of postmenopausal osteoporosis.

Relationship between osteoporosis and periodontal disease

Systemic bone loss has been cited as a risk factor for periodontal disease⁷ but their association is still not well understood. A study⁸ have shown that after fifty years of age the porosity of the mandibular cortical bone increases markedly especially in the alveolar bone, at the same time there is a decrease in bone mass. These changes are greater in women than in men and this is reflected in the fact that women have a lower mandibular BMD than men. This sex difference in BMD value is also observed in other bones. It has been suggested that this increase in alveolar bone porosity in combination with local factors could be of etiological importance in the rate of periodontal alveolar bone loss which leads to periodontal disease⁸.

Some authors⁷ have experimentally concluded that in postmenopausal women BMD is related to interproximal alveolar bone loss. This conclusion points at postmenopausal osteopenia as

a possible risk factor for periodontal disease in postmenopausal women. Another study⁹ has shown that women with high calculus apposition and low BMD had greater clinical gingival attachment loss than women with normal BMD and similar calculus apposition. Still other authors¹⁰ have reported that serum estradiol supplementation, in early menopausal osteoporotic women, reduces gingival inflammation and attachment loss. A study¹¹ performed on digitized periapical radiographs of the maxilla and mandible obtained from osteoporotic patients and normal controls lends support to the hypothesis that osteoporotic patients present an altered trabecular pattern in the jaw bones when compared to normal controls.

Radiographic evaluation of alveolar bone loss was conducted in a 2-year longitudinal clinical study¹² on 21 women with normal BMD of the lumbar spine, and 17 women with osteoporosis or osteopenia of the lumbar spine at baseline. These 38 patients had a history of periodontitis and were non-smokers. The results of this study showed that osteoporotic/osteopenic women exhibited a higher frequency of alveolar bone height loss ($p < 0.05$) and crestal ($p < 0.025$) and subcrestal ($p < 0.03$) density loss relative to women with normal BMD. Additionally it was shown that estrogen deficiency in the osteoporotic/osteopenic women was associated with increased alveolar bone crestal density loss. This study¹² data suggests that estrogen deficiency and osteoporosis/osteopenia could be considered potential risk factors for alveolar bone loss in postmenopausal women with periodontitis. Pilgram et al¹³ have concluded that there is no definite association between clinical attachment level and BMD of the lumbar spine and the femur. They also conclude that there may be a weak association between BMD and longitudinal changes in attachment level.

CONCLUSION

The risk relationship between osteoporosis and periodontal status has been analyzed by many investigators for more than a decade and the various results postulated by those investigators are controversial. With scientific fairness in mind, presently, osteoporosis cannot be postulated as one of the main causative factors of periodontal disease, especially in menopausal females. As many researchers are indicating the proper knowledge of the level of participation of many systemic factors in the etiology of periodontal disease is still in infancy. Osteoporosis being one of those factors. Further

detailed and comprehensive research is still needed to definitely prove or disprove the participation of osteoporosis in the etiology of periodontal disease.

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