Review Article

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RELATIONSHIP BETWEEN OSTEOPOROSIS AND PERIODONTAL DISEASE: A REVIEW

Mohammed Shereef[1],Susanth Sachithananda Baliga[2],Andres F. Munoz[3]

1.Periodontist (Reader, Amrita School of Dentistry, Kochi), 2. General Dentist, Dr. Shereef's Cosmetic Dental Speciality Clinic,St. George Shopping Complex,Pump Junction,Railway Station Road, Aluva, India

3. General Dentist, Pontificia Javeriana University, Bogota DC, Colombia

ABSTRACT:

It is a disease characterized by low bone mass and deterioration of bone structure that causes bone fragility and increases the risk of fracture. Osteoporosis results from an imbalance between the rate of bone formation and resorption that leads to loss of bone mineral mass. Loss of the mineral component of the bone, leads to a greater tendency of the bone to be broken· Osteoporosis and osteopenia are characterized by reductions in bone mass and may lead to skeletal fragility and fracture.

Periodontitis is an inflammatory disease characterized by loss of connective tissue and alveolar bone. Like osteoporosis, it is a silent disease, not causing symptoms until late in the disease process when mobile teeth, abscesses and tooth loss may occur. While the causative agent in periodontitis is a pathogenic bacterial plaque in a susceptible patient, periodontitis and osteoporosis have several risk factors in common. Familiarity with the risk factors could help identify these individuals and aid in earlier diagnosis.

Keywords: Bone mineral mass, osteoporosis, osteopenia, risk factors, skeletal fragility.

INTRODUCTION:

Osteoporosis is a skeletal disease characterized by reduction in bone mass and micro architectural changes in the bone, which leads to an increased bone fragility and an increased risk of fracture. Osteoporosis results from an imbalance between the rate of bone formation and resorption that leads to loss of bone mineral mass. Loss of the mineral component of the bone, leads to a greater tendency of the bone to be broken[1]. Osteoporosis and osteopenia are characterized by reductions in bone mass and may lead to skeletal fragility and fracture. In fact, the exact definition of osteoporosis differs around the world. In much of Europe, osteoporosis implies a reduced bone mass that results in a predisposition to fracture. In 1994, the World Health Organization defined osteoporosis as a bone mineral density level more than 2.5 standard deviations below the mean of young, normal women[2].

*Corresponding Author Address: Dr Susanth Sachithananda Baliga Email: susanthbaliga90@gmail.com

Risk factors for osteoporosis:

Non-modifiable risk factors

- Age
- Race
- Sex
- Family history of osteoporosis/fracture
- Early menopause

Modifiable risk factors

- Sex hormone
- Insufficient Calcium intake
- Insufficient Vitamin D intake
- Weight
- Physical activity
- Chronic glucocorticoid use.
- Antiepileptic agents
- Cigarette smoking
- Gonadotropin-releasing hormone agonists
- Excessive thyroxine doses, lithium, and anticoagulants.

Women are at a greater risk for osteoporosis after menopause. Estrogen levels present prior to menopause are protective against loss of bone mineral, as in hormone replacement therapy after menopause. Early menopause, either naturally occurring or drug or surgically induced, without hormone replacement therapy predisposes to osteoporosis. The decision of whether or not to use hormone replacement therapy is dependent on the risk-benefit ratio for the individual woman.

Age is major non-modifiable risk factor for osteoporosis [5]. In most women, bone mass reaches its peak in the third decade of life (twenties or thirties) and declines thereafter. This decline in bone mass is accelerated with the onset of menopause.

Other non-modifiable factors include a thin-framed body, and the fact that Caucasian and Asian women are at higher risk than African –American women [5], as women with a history of osteoporosis in the family. Modifiable contributors to low bone mass include lack of sufficient calcium intake [6], lack of exercise, smoking and alcohol. Certain medications, such as steroids, will alter the balance between bone formation and resorption resulting in a net loss of bone mass.

Periodontitis is an inflammatory disease characterized by loss of connective tissue and alveolar bone[7]. Like osteoporosis, it is a silent disease, not causing symptoms until late in the disease process when mobile teeth, abscesses and tooth loss may occur. While the causative agent in periodontitis is pathogenic bacterial plaque in a susceptible patient, periodontitis and osteoporosis have several risk factors in common.

U.S. Survey of oral health in adults has shown an increased prevalence of periodontitis with increasing age. In addition to the increased prevalence with increasing age, risk factors in common for both diseases include smoking, and influence of disease or medications that may interfere with healing[8]. Whether the rate of progression of periodontitis in women sharply increases immediately after menopause is presently unknown.

Osteoporosis is categorized into primary or secondary. Primary osteoporosis is associated with increased age and/or decreased sex hormones. Secondary osteoporosis implies an underlying cause such as usage of glucocorticoids, systemic diseases affecting bone turnover, or low calcium intake [9]. Periodontal disease is a chronic destructive disease that may occur in adults, young people and children. Periodontal pathogens which are found in the dental biofilm result in inflammation of the gingiva which is called gingivitis. When periodontal tissue destruction and alveolar bone loss happen, it is called periodontitis [10]. Periodontal disease and periodontal pathogen have been linked to several systemic diseases [11].Since both osteoporosis and periodontal diseases are bone destructive diseases, it has been hypothesized that osteoporosis could be a risk factor for the progression of periodontal disease. But some of the literature concluded that osteoporosis in human organs has no effect on the maxilla and mandible bone density [12].

Criteria for defining osteoporosis and osteopenia[13]:

World health organization criteria for defining osteoporosis and osteopenia	
Condition	Description
Normal	BMD ≤1 SD below the mean for a young, healthy adult (T ≥ -1.0)
Osteopenia	BMD >1 SD, but < 2.5 SD below the mean for a young, healthy adult (-1.0 >T > -2.5)
Osteoporosis	BMD ≥ 2.5 SD below the mean for a young, healthy adult (T ≤ -2.5)
Established osteoporosis	BMD ≥ 2.5 SD below the men for a young, healthy adult (T ≤ -2.5), with 1 or more fragility fractures
T score = 1 SD difference from the BMD in a young, healthy adult of the same gender. BMD, bone mineral density; SD, standard deviation; WHO, World health organization. Modified from report of a WHO study group.	

Assessment of osteoporosis[14, 15, 16]:

A number of methods are currently used to assess bone density, including single-photon absorptiometry, dual-photon absorptiometry (DPA), dual-energy x-ray absorptiometry (DXA), quantitative computed tomography, and radiographic absorptiometry (RA). Of these, DXA is considered the preferred technique for measurement of BMD[14]. DXA measures bone density as "area density" in units of grams per square centimeter. It is the most precise and the diagnostic measure of choice as the quantitative computed tomography through being more sensitive, causes greater radiation exposure. The sites most often used for DXA measurement of BMD include central sites such as the spine or hip or peripheral sites such as the radius.

Literature review regarding periodontal disease and osteoporosis:

In a classic series of studies, **Kribbs et al[17] (1983)**, addressed this relationship in both normal and osteoporotic women. In their study in 1983, total body calcium as assessed by neutron activation analysis was found to be associated with mandibular bone density as measured by quantitative analysis of intraoral radiographs.

In a study by **Daniell AW[18] (1983)**, 208 women aged 60 to 69 were assessed for smoking habits, periodontal disease, and current osteoporosis severity based upon the percentage of cortical area at the metacarpal mid shaft. In this study, 52% of the smokers, 26% of the nonsmokers, and only 8% of non-osteoporotic non-smokers required dentures since reaching the age of 50. This series of case reports suggests that osteoporosis and smoking are important factors promoting tooth loss.

In a later study by **Kribbs et al[19] (1990)**, in normal, non-osteoporotoic women revealed that bone mass was not affected by age but was significantly associated with skeletal bone mass at the spine and wrist.

A comparison of 85 osteoporotic women with 27 normal women showed less mandibular bone mass and density and a thinner cortex at the gonion in osteoporotic compared with non osteoporotic women.

In another study, **Kribbs[20] (1990)** compared osteoporotic patients with controls and found that the osteoporotic group had a greater percentage of subjects who were edentulous or had greater tooth loss. However, the reason for tooth loss is difficult to determine, and it is not clear whether tooth loss was weakly or significantly associated with more severe periodontitis.

Similarly, **Von Wowern et al[21] (1994)**, studied the relationship in both normal and osteoporotic women and reported that 12 osteoporotic subjects with a history of fractures had less mandibular bone mineral content as measured by dual photon absorptiometry than 14 normal women.

In a cohort study from the leisure world Cohort, **Paganini-Hill[22] (1995)**, noted in 3,921 postmenopausal women that tooth loss was reduced by 36% in estrogen users as compared to non-users.

Grodstein et al[23] (1996) data from the Nurses' Health study Cohort observed a 24% reduction in the risk of loss of more teeth in current users of postmenopausal estrogen replacement as compared to never-users.

Wactawski-Wende and coworkers[24] (1996) reported a direct association between skeletal and mandibular osteopenia and destructive periodontal disease as measured by loss of interproximal alveolar bone in post-menopausal women.

Implants and osteoporosis: Osteoporosis results in decreased bone quality and therefore may affect the outcome of dental implant therapy. In an animal study evaluating the effect of glucocorticoid-induced osteoporosis on implant osseointegration, animals received intramuscular injections of glucocorticoids (7.5 mg/kg) for 8 weeks before, simultaneous with, or after implant placement, with a fourth group serving as the control. Although there was no difference in interfacial strength between the test and control groups, bone-to-implant contact (BIC) was significantly lower in the osteoporosis groups (range, 24%±16% to 42%±16%) compared with the control group (49%±10%). Similarly, another study utilizing an oophorectomized rat model found that an osteoporotic state resulted in decreased BIC compared with controls. Furthermore, the greatest decrease in BIC was noted when an osteoporotic state was induced after osseointegration had occurred (BIC = 50% compared with 79% in the control group). The results of these studies imply that although osseointegration of implants in osteoporotic bone is possible, the long-term stability of the implants may be compromised by the disease.

OSTEOPOROSIS-PREVENTION STRATEGIES[16] (formulated by the National Osteoporosis Foundation):

•All women should be counselled on the risk factors.

•An evaluation of bone mineral density should be performed on all post - menopausal women who present with fractures to determine the diagnosis and disease severity.

•Bone mineral density testing is recommended for all post- menopausal women younger than 65 years, who have one or more risk factors for osteoporosis in addition to menopause.

•Bone mineral density testing is recommended for all women above 65 years and older regardless of additional risk factors.

•All diagnosed patients are counselled to obtain an adequate dietary intake of calcium.

•Regular weight bearing and muscle strengthening exercises to reduce the risk of falls and fractures are recommended.

•Alcohol intake should be at a moderate level (about one drink per day for women and two drinks per day for men.

• Patients should be advised against smoking, and smoking cessation should be implemented.

•All post-menopausal women who present with hip fractures or vertebral fractures should be considered candidates for osteoporosis treatment.

Manangement: Several pharmacological agents are available to increase bone mineral density and therefore treat or prevent osteoporosis. They include hormone replacement therapy, bisphosphonates, calcitonin, selective estrogen receptor modulators, parathyroid hormone or combination of these medications [16].

Bisphosphonates: They are analogues of pyrophosphonate and bind selectively to bone mineral. During bone resorption they are taken up by the osteoclast, resulting in osteoclast de-activation and apoptosis'. Bisphosphonates are often considered the first-line therapy for the treatment of post-menopausal osteoporosis. They are the most widely prescribed anti-resorptive agents. Bisphosphonates binds avidly to apatite crystals, mainly on remodeling surfaces and inhibits their growth, aggregation and dissolution. The more potent nitrogen containing member of this drugs class includes alendronate, risedronate, ibandronate and zoledronic acid. Alendronate has been shown to reduce active bone resorption significantly without interfering with bone mineralization and quality. Clinical trials have shown that bisphosphonates also decrease bone turnover and increased bone mass and bone strength[25, 26].

Hormone replacement therapy: Rapid loss of bone density is observed because of estrogen deficiency in the early post-menopausal years. The rationale for HRT is to delay this bone loss. Estrogen therapy can inhibit osteoclast formation and function and can also extend the lifespan of osteoblasts and osteocytes. HRT should not be recommended for prevention of osteoporosis in post-menopausal women, unless the woman are at a significant risk of osteoporosis, and other osteoporosis medications are unable to be considered. It is therefore important that women discontinuing HRT receive appropriate screening for their risk for complications of osteoporosis and should be counselled regarding alternative forms of therapy to prevent fracture.

Selective estrogen receptor modulators: They were developed to provide the benefits of estrogen therapy without its unwanted side effects. Their mechanism of action such as that of raloxifene is similar to that of the estrogens[16].

Calcitonin: Calcitonin is an inhibitor of osteoclast activity. Both nasal and subcutaneous calcitonin are available for treatment of post-menopausal osteoporosis. Treatment of women with osteoporosis with nasal calcitonin has been shown to reduce the incidence of vertebral fractures in a single randomized study, by 33% when compared to placebo [28].

CONCLUSION:

Osteoporosis is a systemic skeletal disease manifested by reduced bone strength, decreased bone mineral density (BMD), and altered macrogeometry and microscopic architecture, and resultant increased risk of fractures. Both osteoporosis and periodontitis are common bone-resorptive, host-dependent, multifactorial diseases that generally affect older patients. Both diseases are stimulated by bone-resorptive proinflammmatory cytokines such as IL-1 and TNF-α, but the end result of this stimulation differs in the 2 diseases. Osteoporosis results in bone loss that is generalized throughout the skeleton, whereas periodontitis results on bone loss that is localized to the alveolus. The effects of osteoporosis on both systemic health and oral health need to be well understood. Understanding the association between these common diseases and the mechanisms underlying those associations will aid health professionals to provide improved means to prevent diagnose and treat these very common diseases.

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