OSTEOPOROSIS AND PERIODONTITIS: A BIDIRECTIONAL RELATIONSHIP.

Paramjit Kaur Khinda, Jyotika Pasricha*, Amarjit Singh Gill, Deepkanwar Kaur

Department of Periodontology and Oral Implantology, Genesis Institute of Dental Sciences and Research, Ferozepur, India.

Email: resmile.zenflox@gmail.com

Abstract

Osteoporosis is a condition of compromised bone strength that predisposes an individual to an increased risk of fracture and is a major cause of morbidity in older susceptible individuals. Osteoporosis is related to various endocrinial abnormalities, metabolic and nutritional factors, postmenopausal hormonal changes and consumption of certain drugs such as cortisone. Emerging clinical and molecular evidence suggests that inflammation also exerts significant influence on osteoporotic bone changes. Numerous pro-inflammatory cytokines have been shown to be associated with regulation of osteoblast and osteoclast differentiation, resulting in a shift towards activated immune profile; hypothesized as an important risk factor for the condition. Chronic inflammatory conditions and the immune system remodeling characteristic of aging may be determinant pathological risk factors for osteoporosis. The present article reviews the current perspective on the interaction between bone morphology and immune system in the inflammatory condition (periodontitis), unleashing the link between two chronic conditions.

Keywords: Osteoporosis; periodontitis; pro-inflammatory cytokines; inflammation; bone remodeling; dual-energy X-ray absorptiometry.

INTRODUCTION

Periodontitis, an inflammatory disease characterized by resorption of alveolar bone as well as loss of soft tissue attachment of the tooth, is a major cause of tooth loss in adult population. Advances in science and technology over the last century have greatly expanded our knowledge on the pathogenesis of periodontal disease. Though periodontal disease is a infectious disease, but environmental, physical, social and various host stresses may affect and modify disease expression. There is an essential bacterial etiology, and there are specific pathogenic bacteria (periodontal pathogens) associated with destructive periodontal disease. However, these pathogens do not invariably cause disease by their presence alone.

Current concept states that host response varies among different individuals and that an insufficient host immune response or an exaggerated immune response to bacterial pathogens may lead to more severe forms of the disease. Furthermore, certain systemic disorders and conditions alter host tissue physiology, which may impair host barrier integrity and immune response to bacterial pathogens, resulting in more destructive periodontal disease. Many systemic diseases and disorders have been implicated as risk indicator and/or risk factor in periodontal disease. Growing understanding of physiological bone remodelling suggests that factors involved in inflammation are linked with those critical for the aetopathogenesis of certain chronic bone disorders like osteoporosis. Recent studies quoted also suggest an association between systemic conditions like osteoporosis and periodontal infection.

Osteoporosis

Osteoporosis is a systemic skeletal disorder; characterized by low bone mass density and micro-architectural deterioration of bone tissue without any change in its chemical composition and subsequent increase in bone fragility and susceptibility to fracture. In this perspective osteoporosis may reflect a state of disequilibrium between structural demand for calcium and phosphate and their biologic demand during metabolically active states such as inflammation. According to National Osteoporosis Foundation, osteoporosis is a major public threat for an estimated 44 million of the US population (55% of people > 50 years of age) and almost twice that number have low bone mass and osteopenia (a condition of compromised bone strength that predisposes an individual to an increased risk of fracture and usually asymptomatic, becoming symptomatic when functional demand exceeds the structural viability of the skeleton). Osteoporosis and osteopenia can be differentiated on the basis of bone mineral density assessed using dual energy X-ray absorptiometry. Bone mineral density quantified using dual-energy X-ray absorptiometry scan can be defined in terms of T score. The T score compares the bone mineral density with the peak bone mineral density for an individual of the same age and gender and is reported as the number of standard deviations below that average. According to World Health Organization (WHO) osteoporosis is defined as bone mineral density (BMD) 2.5 standard deviations below normal. Bone mineral density scores 1.0 to 2.5 standard deviations below normal are diagnostic for osteopenia and scores of 0 to 1.0 are considered normal. The incidence of osteoporosis is much higher in women (80%) than in men (20%). Approximately one in two women over the age of 50 is susceptible to bone fracture because of osteoporosis. This may be attributed to the fact that women at peak mass attainment achieve less bone mineral content than males. In women, 98% of the skeletal mass built occurs by age of 20 years much earlier than in men; responsible for increased risk of osteoporosis. Whereas low levels of estrogen mainly causes accelerated bone loss perimenopausally.

Osteoporosis can be categorized into primary and secondary. Primary osteoporosis is associated with increased age and/or decreased sex hormones. A multitude of systemic diseases are also associated with an increased risk of osteoporosis, including hypogonadal states, endocrine disorders (Cushing’s syndrome, thyrotoxicosis), rheumatologic disorders (rheumatoid arthritis) and certain inherited diseases (osteogenesis imperfecta), hypophosphatasia, multiple myeloma, leukaemia and lymphoma. Secondary osteoporosis implies an underlying systemic cause which may include usage of certain medications, systemic factors affecting bone turnover, and low calcium intake.

Osteoporosis and Periodontal disease

Osteoporosis, characterized by imbalance in normal bone turnover physiology with net loss of bone and bone mineral density do affect oral bone turnover. Now, question arises to what extent is oral bone affected by altered bone physiology, and does it contribute to oral bone loss? In addition to the academic relevance of this...
question, it is fundamental to proper clinical management of dentate and edentulous patients suffering from both osteopenia and osteoporosis. A rational dental treatment plan in such patients is not complete if proper management of systemic bone loss is not included.

Co-Risk factors for osteoporosis and periodontal disease:
Osteoporosis and periodontal disease are chronic multifactorial diseases. Thus, it is not surprising that both diseases share common risk factors. These risk factors can be classified as nonmodifiable or modifiable.[table 1]

Table(1) Risk Factors Common To Periodontal Disease And Osteoporosis.

<table>
<thead>
<tr>
<th>RISK FACTOR</th>
<th>MODIFIABLE</th>
<th>PREVENTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>Early menopause (Estrogen deficiency)</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>Nutritional factors (Lack of Calcium, several vitamins)</td>
<td>Yes</td>
<td>Diet high in calcium and vitamins</td>
</tr>
<tr>
<td>Smoking</td>
<td>Yes</td>
<td>Smoking cessation</td>
</tr>
<tr>
<td>Alcohol</td>
<td>Yes</td>
<td>Decreased alcohol consumption</td>
</tr>
<tr>
<td>Heredity</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>Diseases (e.g. Hyperparathyroidism,)</td>
<td>To some extent</td>
<td>Treatment</td>
</tr>
</tbody>
</table>

Estrogen deficiency is dominant pathogenic factor for osteoporosis in postmenopausal women. Estrogen either directly or indirectly, modulates the production of cytokines and growth factors which in turn act as local regulators of the remodelling process. Cytokines under estrogen control with direct effects on bone cells include OPG, RANKL/RANK, IL-1 alpha, IL-1 beta, TNF-alpha, and granulocyte-macrophage colony-stimulating factor (M-CSF) secreted by monocytes and IL-6 secreted by osteoblasts. IL-1 induces the synthesis of IL-6, which increases bone resorption through osteoclast recruitment. Colony-stimulating factor plays a role in the maturation of osteoclasts. IL-1 and TNF-alpha stimulate mature osteoclasts, modulate bone cell proliferation, and induce bone resorption in vivo. In addition, IL-1, TNF-alpha, and GM-CSF contribute to bone resorption by promoting osteoclast recruitment and differentiation from bone marrow precursors. Thus, estrogen deficiency causes an increase in the number of osteoclasts, driven by the higher levels of same cytokines that down-regulate osteoblast generation in normal physiological conditions. This creates imbalance in normal physiological metabolism, favouring bone resorption.

In addition, estrogen may affect bone turnover indirectly by acting as an antagonist to PTH. But, the bone sparing effect of estrogen may be explained by its fundamental ability to interact with bone cells and modulate the cytokine circuitry.[15]

Norderyd and colleagues[16] reported lower, although not statistically significant, levels of clinical attachment loss and gingival bleeding in postmenopausal women receiving estrogen supplementation compared with estrogen-deficient postmenopausal women. A 5-year longitudinal study of 69 women (with menopause receiving hormone replacement therapy) compared lumbar spine BMD, with mandibular bone mass assessed by quantitative measures of standardized intraoral radiographs. A statistically significant but moderate correlation was observed between mandibular and lumbar spine bone mass and that estrogen replacement therapy after menopause had a positive effect on bone mass not only of the lumbar spine but the mandible as well.[17] Payne and colleagues showed,[18] in a 1-year longitudinal study of 24 postmenopausal women, that estrogen-deficient women displayed a mean net loss in alveolar bone density compared with estrogen sufficient women, who displayed a mean net gain in alveolar bone density.[table 2]

Table(2) Relationship between estrogen status and periodontal disease

Smoking
Smoking interferes with efficient calcium absorption resulting in accelerated bone loss. A meta-analysis of 29 studies including 2,156 smokers and 9,750 nonsmokers examined the effect of cigarette smoking on skeletal bone mineral density. While bone density in premenopausal women was comparable in smokers and nonsmokers, in postmenopausal women bone loss was greater in current smokers compared with non-smokers,[19] suggesting that the effect of smoking on skeletal BMD is modulated by estrogen.

A meta-analysis of available literature indicates that smokers have 2.5 times the risk for severe periodontal disease compared with nonsmokers, independent of the effects of age, socioeconomic factors, diabetes mellitus, or dental plaque. Furthermore, the risk is cumulative and dose dependent in that the severity of periodontal disease is related to the duration and amount of smoking. The greater severity of periodontal disease in males in part is explained by smoking as a risk factor.

Thus, smoking is considered the single most important modifiable risk factor for periodontal disease and osteoporosis.

Dietary Factors:

Adequate dietary calcium is essential for the growth and development of a normal skeleton. Insufficient calcium intake during childhood and adolescence can reduce peak bone mass attainment and enhance postmenopausal and age-related osteoporosis.[20] Individuals with diets deficient in calcium had statistically higher levels of periodontal disease compared with those with calcium...
sufficient diets. This association was especially strong in younger and premenopausal women. Individuals with diets deficient in vitamins C, A, alpha-caroteneoids, selenium, and lutein also showed increased risk for periodontal disease, independently of the confounding effects of age, dental plaque, and smoking. Therefore, a diet complete in vitamins and minerals plays an important role, not only in ensuring achievement of peak bone mass and protection from age-related bone loss, but also protects against the destruction of connective tissue and alveolar bone resulting from periodontal infection.

**Genetics Factors:**

Osteoporosis is a multifactorial, polygenic condition involving multiple genes regulating the attainment of peak bone mass and possibly the control of bone turnover.

The vitamin D receptor (VDR) is required for normal calcium absorption from the gut. Common allelic variants in the gene encoding the vitamin D receptor has direct effect on bone density. Other polymorphisms imparting susceptibility to osteoporosis include the binding site in collagen type I alpha 1 (COLIA1) gene, transforming growth factor-beta (TGF-β) gene, the estrogen receptor as well as genes regulating cytokines involved in bone turnover. Susceptibility to periodontal infection appear to be under genetic regulation as well influenced by several genes. Candidate genes for susceptibility to periodontal disease include genes defining the FcyRII receptor, genes regulating immunoglobulin synthesis, especially IgG2, and genes regulating cytokine synthesis determining bone turnover.

**Is Osteoporosis an inflammatory condition?**

Clinical observations reveal coincidence of systemic osteoporosis with period of systemic inflammation as well as co-localization of regional osteoporosis with areas of regional inflammation. Different epidemiological studies report an increase in the risk of developing osteoporosis in various inflammatory conditions. Immunological dysfunction, autoimmune and various inflammatory conditions, hyper IgE syndrome, rheumatoid arthritis, haematological disorders, particularly myeloma and inflammatory bowel diseases are associated with osteoporosis.

C-reactive protein (CRP), a pentameric protein found in blood plasma is elevated during conditions of active inflammation. C-reactive protein production in liver is upregulated by various pro-inflammatory cytokines like IL-1, IL-6 and TNF-alpha and is regarded as sensitive marker of systemic inflammation. An association between circulating levels of high sensitive (hs) CRP and bone mineral density has been observed in several immune and inflammatory conditions, suggesting an association between subclinical systemic inflammation and osteoporosis.

On the other hand, an intriguing aspect of immunosenescence is the increased production of pro-inflammatory cytokines with aging (inflamm-aging). As age advances, continuous exposure to chronic antigenic load and oxidative stress may impair the normal physiological counter-regulatory mechanism; which inhibits bone resorption following T-cell activation. This would contribute, together with low grade systemic inflammation, to increasing incidence of osteoporosis during senescence.

Excessive osteoclastic resorption is a common feature of chronic inflammatory processes such as periodontal disease. The underlying mechanism of increased bone resorption may be directed by increased systemic/local osteoclastic activity, or by elevated local cellular or cytokine profile. In physiological bone remodelling, the cell-to-cell contact between receptor activator of nuclear factor-κB ligand (RANKL)-expressing osteoclasts and RANK-expressing monocyte/osteoclast precursor cells is crucial. In inflammatory processes activated T lymphocytes express higher levels of RANKL, increasing the possibility of osteoclast differentiation and synthesis. RANKL is inhibited by osteoprotegerin (OPG) released by stromal cells and osteoblasts. B lymphocytes may also participate in osteoclast formation, either by expressing RANKL or by serving as osteoclast progenitor cells themselves. Interestingly, studies have shown that RANKL mRNA is upregulated in the gingiva of patients with advanced periodontitis. On the other hand, osteoprotegerin (OPG) mRNA is downregulated. The hypothesis linking osteoprotegerin and periodontal disease is strengthened by studies involving many gram-negative bacteria. Bacterial infection by these pathogens may trigger RANKL activation and subsequent osteoclast proliferation and activation, inducing osteoporotic bone changes in patients with periodontal infection.

Since the expression of RANKL/RANK may be controlled by sex hormones, it is possible to speculate that this system may control gender specific differences in immunity and could be involved in higher incidence of autoimmune diseases and osteoporosis in women. RANKL, RANK and OPG are considered as interesting molecular links between bone remodelling, immunity and inflammation.

Another factor, nitrous oxide (NO) has both anabolic and catabolic effects on bone metabolism. The role of NO is controversial, in that low levels of NO maintain homeostasis, whereas high levels of NO induce bone resorption as seen in many inflammatory conditions. Also, NO is an important element of the host defense mechanism against P. gingivalis, a primary periodontal pathogen. Thus activation of the inducible NO synthesis pathway by cytokines, such as IL-1 and TNF-α, inhibits osteoblast function in vitro and stimulates osteoblast apoptosis shifting bone physiology towards resorption.

Recently, some studies have reported an association between osteoporosis and oral bone loss in periodontal disease, first attempt for which was made as early as 1960. Most of the research carried out on mandibular bone revealed a relationship between systemic and oral bone loss evaluated by means of radiography, histology (microradiography), single-photon absorptiometry (SPA), dual-photon absorptiometry (DPA), quantitative CT (QCT) and more recently, dual-energy X-ray absorptiometry (DXA).

**Relationship of Skeletal bone mass to Mandibular Bone Density:**

It has long been postulated that mandibular bone density may be indicative of systemic bone mineral density. In a classic series of studies, Kribbs and colleagues addressed this relationship in both normal and osteoporotic women. In an early study total body calcium as assessed by neutron activation analysis, was found to be associated with mandibular density as measured by quantitative analysis of intraoral radiographs. Later study in normal, non-osteoporotic women, revealed that mandibular bone mass was not affected by age but was significantly associated with skeletal bone mass at the spine and wrist. [TABLE 3] A study conducted by Melescanu-lmre et al 2009 revealed no relationship between alveolar bone mass and skeletal BMD but mandibular cortical thickness was influenced by estrogen levels.

**Alveolar Crestal Height and Osteoporosis:**

Several studies were conducted to determine the relationship between crestal bone level and skeletal BMD. Elders et al and Kalmetti et al failed to determine any positive co-relation between...
alveolar bone and skeletal bone mass. Wactawski-Wende et al, in a study of 70 postmenopausal women, found a significant relationship between alveolar crestal bone height as a measure of periodontitis and skeletal osteopenia (femur and lumbar spine) measured by DXA. This relationship was seen after controlling for possible confounders such as dental plaque, years of menopause, and smoking. In addition, there was a relationship between osteopenia at the hip and probing attachment loss in this same group. Payne et al and Tezal et al were able to establish positive co-relation between alveolar bone height and BMD spine and hip. [TABLE 4]

**Tooth loss and Osteoporosis:**

Several studies have demonstrated a relationship between tooth loss and systemic osteoporosis in both dentate and edentulous individuals. Daniell and colleagues suggested that systemic bone loss was a risk factor for edentulism. Women with severe osteoporosis, defined as extreme thinning of the metacarpal cortical area, were three times more likely (44% versus 15%) to have no teeth compared with healthy, age-matched controls. In a study of 329 healthy postmenopausal women, for each additional tooth present, spinal BMD increased 0.003 g per cm². Taguchi and colleagues showed that a decrease in mandibular bone density, estimated as mandibular cortical width, correlated with tooth loss for women in their sixties. Collectively, this evidence indicates that osteoporotic women have lost significantly more teeth, and more are edentulous compared with non-osteoporotic women. [TABLE 5]

**Periodontal Disease and Osteopenia/Osteoporosis:**

Payne and his colleagues demonstrated in a prospective study, positive co-relation between periodontal destruction and BMD at spine and hip. Yoshihara and colleagues also demonstrated similar results in another prospective study. von Wowern and colleagues in a case-control study comparing 12 female patients with osteoporotic fractures and 14 normal women, reported significantly greater periodontal attachment loss in the osteoporotic women compared with the normal women. They found that the osteoporotic women had less mandibular bone mineral content, as measured by dual photon absorptiometry, than the 14 normal women. This association was increased even further in postmenopausal females. [TABLE 6]

Hence, though limited, the evidence from various studies suggest an association between osteopenia, osteoporosis, and periodontal disease.

Evidence suggests that hormone replacement therapy improves bone density in postmenopausal women. In a 3-year randomized trial in postmenopausal women with moderate to advanced periodontal destruction, estrogen therapy significantly improved alveolar bone density compared with placebo (p<0.04), along with increase in bone mineral density of the femur but not in the lumbar spine. Furthermore, women receiving hormonal therapy had significantly less gingival inflammation, lower plaque scores, and lesser loss of attachment.

Using osteoporosis treatment as a basis, many studies have tried to use similar approaches in treating periodontal disease, deepening the relationship between the two diseases. Parathyroid hormone (PTH) produces several distinct effects on the entire bone remodelling process, because it influences both bone formation and bone resorption. Recent studies have indicated that intermittent doses of PTH have an efficient systemic anabolic effect, reducing bone resorption especially in estrogen deficient osteoporotic cases. A study conducted by Marques MR concluded that systemic administration of PTH reduces alveolar bone loss in ovariectomized rats, despite the presence of periodontal disease inducer and estrogen deficiency. DISCUSSION:

Although number of studies have found that the density of the alveolar bone in the mandible correlated with the density of the bone in the rest of the skeleton and that generalized bone loss may render the jaw susceptible to accelerated alveolar bone resorption, these findings are not universal. Many other cross-sectional studies and longitudinal studies fail to establish association between systemic bone loss, periodontal disease, and edentulism. Thus, further studies should be attempted to clarify this correlation; as majority of studies determining the relationship between periodontitis and osteoporosis have been hindered by small sample size, limited control of other confounding factors, varying definitions of both periodontal disease and osteoporosis and few perspective studies where association between periodontal disease and osteoporosis had been established. Other types of study design including long term follow up, intervention before menopause and investigation of oral conditions during the menopausal phase need to be practiced.

Characterization of the functions of RANKL-OPG axis has significantly contributed to the emergence of osteoimmunity, helping us in examination of the interplay between active immunity and maintenance of bone homeostasis. In the relationship between periodontal disease and osteoporosis, detailed knowledge of the molecular mechanisms involved in RANKL-RANK activation and downstream signalling could generate new pharmacological principles for the inhibition of excessive bone resorption in various periodontal pathological conditions. Though modulating the immune system is a delicate work, targeting the alterations of the axis may form the basis for rational drug therapy in treating periodontal infection effectively.

**CONCLUSION**

As stated earlier periodontal disease is multifactorial disease and the main factor responsible for periodontal disease is microbial plaque, osteoporosis therefore, cannot be the aetiological agent causing the onset of periodontal disease, but after the outbreak of the disease, it may be a predisposing factor in the exacerbation, or persistence of the disease. Periodontal disease itself an inflammatory condition, could alter the course of several chronic conditions like osteoporosis due to generation of activated immune profile. Hormonal Replacement Therapies found beneficial in preventing the progression of periodontal disease in postmenopausal women indicate that osteoporosis is definitely a risk factor for periodontal destruction within the female population.

An important implication as a healthcare provider would be to serve as a pre-screener of patients with the potential risk for osteopenia and osteoporosis as familiarity with the risk factors could help identify these individuals and aid in early diagnosis.

**References**

27) Yun AJ, Lee PY. Maladaptation of the link between inflammation and bone turnover may be a key determinant of osteoporosis. Med Hypotheses 2004;63:532-537.
The relationship between general osteoporosis of the organism and periodontal diseases. Medicina (Kaunas) 2006;42:613-618.


Table(2) Relationship between estrogen status and periodontal disease

<table>
<thead>
<tr>
<th>Author</th>
<th>Periodontal parameter</th>
<th>Osteoporosis assessment</th>
<th>Study design</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Norderyd et al, 1993</td>
<td>CAL, gingival bleeding and levels of plaque</td>
<td>234 postmenopausal women (57 ERT; 177 non-ERT)</td>
<td>Cross-sectional</td>
<td>ERT associated with less gingivitis in postmenopausal women</td>
</tr>
<tr>
<td>Payne et al, 1997</td>
<td>Alveolar bone density</td>
<td>24 postmenopausal women (10 estrogen sufficient; 14 estrogen deficient)</td>
<td>Longitudinal</td>
<td>Estrogen status may influence alveolar bone density status</td>
</tr>
<tr>
<td>Jacobs et al, 1996</td>
<td>BMC; mandible</td>
<td>BMC; lumbar spine</td>
<td>Longitudinal</td>
<td>Estrogen status directly related to mandibular bone mass</td>
</tr>
</tbody>
</table>

Table(3) Relationship between Skeletal and Mandibular bone density

<table>
<thead>
<tr>
<th>Author</th>
<th>Oral measure</th>
<th>Osteoporosis assessment</th>
<th>Study Design</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kribbs et al, 1983</td>
<td>Mandibular bone density</td>
<td>Total body calcium</td>
<td>Cross-sectional</td>
<td>Positive</td>
</tr>
<tr>
<td>Kribbs et al, 1989</td>
<td>Mandibular bone mass</td>
<td>Total body calcium, bone mass at radius and bone density at spine</td>
<td>Cross-sectional</td>
<td>Positive</td>
</tr>
<tr>
<td>Kribbs et al, 1990</td>
<td>Mandibular bone density</td>
<td>Bone mass at wrist and spine (normal women) Osteoporotic group had less mandibular bone mass and density</td>
<td>Cross-sectional</td>
<td>Positive</td>
</tr>
<tr>
<td>Homer et al, 1996</td>
<td>BMD mandibular body, ramus, symphysis</td>
<td>BMD lumbar spine, femoral neck, forearm</td>
<td></td>
<td>Positive</td>
</tr>
<tr>
<td>Melescanu-Imre et al, 2009</td>
<td>Alveolar bone mass</td>
<td>Skeletal BMD</td>
<td></td>
<td>Negative</td>
</tr>
<tr>
<td>Melescanu-Imre et al, 2009</td>
<td>Mandibular angular cortex density</td>
<td>Estrogen use</td>
<td></td>
<td>Positive</td>
</tr>
<tr>
<td>B Cakur et al, 2009</td>
<td>Mandibular cortical index</td>
<td>Skeletal BMD</td>
<td></td>
<td>Negative</td>
</tr>
<tr>
<td>Ducnea et al, 2013</td>
<td>Panoromic mandibular index</td>
<td>BMD hip, femoral neck</td>
<td></td>
<td>Positive</td>
</tr>
</tbody>
</table>

Studies performed to determine relationship between Mandibular Bone Density and Osteoporosis.
Table 4: Studies Performed To Determine The Relationship Of Osteoporosis And Alveolar Crestal Height.

<table>
<thead>
<tr>
<th>Author</th>
<th>Oral Measure</th>
<th>Osteoporosis Assessment</th>
<th>Epidemiological Design</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Humphries et al 1989 52</td>
<td>Residual ridge resorption</td>
<td>Gender, age</td>
<td>Cross sectional</td>
<td>Positive</td>
</tr>
<tr>
<td>Elders et al 199253</td>
<td>Alveolar bone height</td>
<td>BMD spine, Metacarpal cortical thickness</td>
<td>Cross sectional</td>
<td>Negative</td>
</tr>
<tr>
<td>Klemetti et al 1993 54</td>
<td>Crestal alveolar bone loss</td>
<td>Cortical or trabecular density</td>
<td></td>
<td>Negative</td>
</tr>
<tr>
<td>Hirai et al 1993 55</td>
<td>Residual ridge resorption</td>
<td>Osteoporosis</td>
<td>Cross sectional</td>
<td>Positive</td>
</tr>
<tr>
<td>Wactawski-Wende et al 1996 56</td>
<td>Alveolar crestal height</td>
<td>BMD spine, hip</td>
<td>Cross sectional</td>
<td>Positive</td>
</tr>
<tr>
<td>Payne et al 2000 57</td>
<td>Alveolar crestal height</td>
<td>BMD spine</td>
<td>Prospective</td>
<td>Positive</td>
</tr>
<tr>
<td>Tezal et al 2000 58</td>
<td>Alveolar crestal height</td>
<td>BMD spine, hip</td>
<td>Cross sectional</td>
<td>Positive</td>
</tr>
</tbody>
</table>

Studies determining the relationship of Osteoporosis and Alveolar Crestal Height.

Table 5: Studies Linking Osteoporosis and Number of Teeth Present.

<table>
<thead>
<tr>
<th>Author</th>
<th>Oral Measure</th>
<th>Osteoporosis Assessment</th>
<th>Epidemiological Design</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daniell et al 1983 59</td>
<td>Edentulism</td>
<td>Metacarpal index</td>
<td>Cross sectional</td>
<td>Positive</td>
</tr>
<tr>
<td>Kribbs et al 1990 60</td>
<td>Edentulism</td>
<td>Osteoporosis</td>
<td>Cross sectional</td>
<td>Positive</td>
</tr>
<tr>
<td>Astrom et al 1990 61</td>
<td>Tooth loss</td>
<td>Hip fracture</td>
<td>Prospective</td>
<td>Positive</td>
</tr>
<tr>
<td>Krall et al 1994 62</td>
<td>Number of Teeth</td>
<td>BMD spine/ forearm</td>
<td>Cross sectional</td>
<td>Positive</td>
</tr>
<tr>
<td>Taguchi et al 1995 63</td>
<td>Number of Teeth</td>
<td>Mandibular cortical width</td>
<td>Cross sectional</td>
<td>Positive</td>
</tr>
<tr>
<td>Taguchi et al 1995 64</td>
<td>Number of Teeth</td>
<td>Fracture spine</td>
<td>Cross sectional</td>
<td>Positive</td>
</tr>
<tr>
<td>Krall et al 1997 65</td>
<td>Number of Teeth</td>
<td>Estrogen use</td>
<td>Cross sectional</td>
<td>Positive</td>
</tr>
<tr>
<td>Mohammad et al 1997 66</td>
<td>Tooth loss</td>
<td>BMD spine</td>
<td>Cross sectional</td>
<td>Negative</td>
</tr>
<tr>
<td>Hildebolt et al 1997 67</td>
<td>Number of Teeth</td>
<td>BMD spine, hip</td>
<td>Cross sectional</td>
<td>Negative</td>
</tr>
<tr>
<td>Earnshaw et al 1998 68</td>
<td>Number of Teeth</td>
<td>BMD</td>
<td>Cross sectional</td>
<td>Negative</td>
</tr>
</tbody>
</table>

Studies on relationship between Osteoporosis and number of teeth present.

Table 6: Studies Determining Relationship Between Osteoporosis and Clinical Attachment Level.

<table>
<thead>
<tr>
<th>Author</th>
<th>Periodontal Parameters evaluated</th>
<th>Osteoporosis Assessment</th>
<th>Epidemiological Design</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yoshihara et al 2004 69</td>
<td>CAL</td>
<td>BMD</td>
<td>Prospective</td>
<td>Positive</td>
</tr>
<tr>
<td>Payne et al 2000 57</td>
<td>Bleeding on probing, plaque</td>
<td>BMD spine, hip, wrist</td>
<td>Prospective</td>
<td>Positive</td>
</tr>
<tr>
<td>Tezal et al 2000 58</td>
<td>CAL</td>
<td>Osteoporosis</td>
<td>Cross sectional</td>
<td>Positive</td>
</tr>
<tr>
<td>Richardt et al 1999 70</td>
<td>CAL</td>
<td>Serum Estradiol levels</td>
<td>Prospective</td>
<td>Negative</td>
</tr>
<tr>
<td>Mohammad et al 1997 66</td>
<td>CAL, Gingival recession</td>
<td>BMD spine</td>
<td>Cross sectional</td>
<td>Positive</td>
</tr>
<tr>
<td>Hildebolt et al 1997 67</td>
<td>CAL</td>
<td>BMD spine, hip</td>
<td>Cross sectional</td>
<td>Negative</td>
</tr>
<tr>
<td>Mohammad et al 1996 71</td>
<td>CAL</td>
<td>BMD spine</td>
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