Mandibular Panoramic Indexes Predictors of Skeletal Osteoporosis for Implant Therapy
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ABSTRACT The aim of this study is to evaluate the relationship between osteoporosis and mandibular panoramic indexes in order to assess the possibility of using these parameters as an indicator of osteoporosis. Bone quality relates to bone turnover rate, architecture, damage accumulation, and degree of matrix mineralization. Risk factors for osteoporosis can be categorized as non-modifiable (sex, age, early menopause, body frame, race, heredity) or modifiable (lack of calcium, exercise, smoking, alcohol, systemic diseases etc). Various studies have demonstrated that individuals with osteoporosis have altered morphology of the mandible and various investigators reported different aspects: mandibular bone mineral density and cortical index (MCI), mandibular alveolar bone mass, inferior Cortex of the Mandible, alveolar bone resorption, the number of teeth present and special considerations for implant therapy in osteoporotic patients. In conclusion, this paper is a review of the literature on the possible association between osteoporosis and oral bone loss, with an emphasis on radiological studies and dentists are a potentially valuable resource for initial patient screening for signs of osteoporosis, as individuals with osteoporosis have altered architecture of the inferior border of the mandible as seen on panoramic radiographs.

KEY WORDS osteoporosis, mandibular bone, panoramic index, implant therapy

Introduction
Given an apparent association between skeletal bone loss and tooth loss, it has been suggested that dental x-rays may in fact serve as good screening tools for osteoporosis. In a study published in 1996, researchers at the University of Washington School of Dentistry found dental x-rays to be a highly effective method for distinguishing patients with osteoporosis from patients with normal bone density. A British study by Horner and others concluded that mandibular bone density measurements significantly correlated with bone density measurements at other skeletal sites.

As osteoporosis is a systemic skeletal disease, it also affects bone density and structure of the jaws. The relationship between oral signs and osteoporosis was investigated to assess the possibility of using this as an indicator of osteoporosis. The results of dental panoramic radiographs signs are questioning in osteoporosis risk prediction.

Osteoporosis
Osteoporosis is a complex disease and not all of its causes are known. The bones become fragile and more likely to break due to a loss of bone mineral density (bone mass) which makes bone more porous and subsequently much weaker.

However, a large body of evidence now indicates that skeletal fragility is not explained only by diminished density alone. The strength of bone depends not only on its mass but also on the mechanical properties of materials composing it and on its 3-dimensional structure.

Bone quality relates to bone turnover rate, architecture, damage accumulation, and degree of matrix mineralization. It is continuously remodeled. Remodeling consists of the resorption of packets of old bone by osteoclasts followed by replacement with new bone by osteoblasts. In the younger individual, lower rates of bone remodeling result in increased repair of microdamage. In contrast, a higher rate of remodeling or bone turnover, as seen in postmenopausal females, is the primary source of structural fragility. After reaching peak bone mass, bone continues to undergo bone remodeling but the mass remains relatively constant.

At some time during early middle life, the tight coupling of bone resorption and bone formation is disrupted. There is a gradual age-related reduction in the amount of bone formed in resorption cavities and bone mass begins a very gradual decline. Accelerated bone loss occurring after declines in estrogen production accentuates the uncoupling of bone formation and resorption and increase bone turnover rates. Vital to this intercellular signaling system are the bone morphogenic proteins, proteins that regulate osteoblast function and development osteocalcin (which has a pro-osteogenic effect on osteoblasts), and receptor activator of nuclear factor leading to bone resorption. Any disruption therefore in the normal formation or function of either cell type, or
imbalance in secreted signaling proteins, may have profound effects for the maintenance of bone density and thus result in osteopenia or osteoporosis.

There seems to be a disconnect between true osteoporosis and the condition known as osteopenia. Osteoporosis is the clinical manifestation of a substantial decrease in bone density/mass to the point that there is a high probability of failure of osseointegration. (post-menopausal women). Virtually all of us become osteopenic with age, manifested by a decrease in bone mass/density and changes in bone metabolism. The point at which osteopenia becomes osteoporosis is relative. The clinical manifestation of the systemic disease of osteoporosis is fracture.[1]

Risk factors for osteoporosis can be categorized as no modifiable (sex, age, early menopause, body frame, race, heredity) or modifiable (lack of calcium, exercise, smoking, alcohol, systemic diseases etc). [2]

**Clinical Diagnosis of Osteoporosis**

The clinical diagnosis of osteoporosis is made in 2 ways:
- occurrence of an osteoporotic fracture and
- the World Health Organization's (WHO) bone density criteria.

The World Health Organization has established diagnostic criteria for osteoporosis based on bone density measurements determined by dual-energy x-ray absorptiometry (DEXA)/3/. According to these criteria, a patient is classified as having low bone mass, e.g., osteopenia, if the bone mineral density measures between 1 and 2.5 standard deviations below the mean of a young population. Osteoporosis, however, has been defined as a bone mineral density level of 2.5 standard deviations or below from the mean of a young population [3].

However, DEXA is not universally available, is not portable, and is an imperfect predictor of future fractures. Therefore, it is important to evaluate non-DEXA osteoporosis tests that are sensitive, inexpensive, and easily implemented.

**Osteoporosis and mandibular morphology.**

The earliest suggestion of an association between osteoporosis and oral bone loss was made in 1960. The dentist is often the most regularly visited doctor in the elderly population, and dental radiographs are the most frequently used imaging modalities for these patients. Various studies have demonstrated that individuals with osteoporosis have altered morphology of the mandible.[4]

The concern that the impaired bone metabolism affects the mandible or maxilla in a manner similar to its effects on other bones, since a potential relationship between osteoporosis and decreased oral bone mass or density, is controversial and it is actually not easy to assess whether bone quantity and quality in the mandible and maxilla parallel those in the rest of the skeleton. This problem is due to the fact:

- that reported studies have been preliminary, most of them having involved small numbers of subjects, biased sample selection, and differing definitions and measurements of bone loss (i.e., interchangeable use of the terms "bone mass" and "bone density") etc.;
- the peak BMD and rate of change of BMD vary at different skeletal sites depending on the ratio of trabecular to cortical bone and on the average use at the site;
- numerous factors can affect BMD and the development of osteoporosis, including extrinsic factors such as nutrition, medication use, amount of physical activity, lifestyle factors (e.g., smoking and alcohol abuse), and intrinsic factors (e.g., adrenal and gonadal hormone levels);
- the process of bone remodeling is a non-uniform process, it differs from one bone to another, between cortical and trabecular bone, and from one trabecular bone site to another. In contrast to cortical bone, trabecular bone is much more affected by metabolic changes of the skeleton. The decrease in trabecular bone density after menopause exceeds that of cortical bone. For this reason, bone like the maxilla, which consists largely of trabecular bone, is more susceptible to rapid and severe atrophy under conditions of disease and/or metabolic demand for calcium than the mandible, which consists primarily of cortical bone.[5]

A number of mandibular indices based on panoramic radiographs, and image processing and analysing techniques have been developed to allow quantification of mandibular bone mass and trabecular architecture in order to discriminate individuals with osteoporosis from those without osteoporosis. Cortical width (CW), panoramic mandibular index (PMI), alveolar crest resorption degree (M/M) ratio, cortical index (CI) and fractal dimension (FD) are among them.

In various studies, it has been shown that the decreased bone mineral density (BMD) affects the morphometric, densitometric and architectural properties of mandibular bone in the osteoporotic patients on radiographs.[6]

A large number of quantitative and qualitative measurements of mandibular bone from
radiographs have been devised for this purpose, including densitometry and morphometry. The most common radiographic method for application in general practice is panoramic radiograph, which is often used for the diagnosis and treatment planning.

The earliest bone loss in osteoporosis patients occurs in areas of trabecular bone. The metabolic turnover of trabecular bone is approximately eight times greater than that of cortical bone. This is the reason why areas of predominantly trabecular bone such as the vertebral body have been preferred sites for measuring bone mineral density.[7]

The earliest bone loss in osteoporosis patients occurs in areas of trabecular bone. The metabolic turnover of trabecular bone is approximately eight times greater than that of cortical bone. This is the reason why areas of predominantly trabecular bone such as the vertebral body have been preferred sites for measuring bone mineral density.[7]

The mandible is a site consisting predominantly of trabecular bone. Trabecular bone is clearly visible on dental radiographs thus lending itself to quantitative analysis of bone mineral density. Recent studies have demonstrated a significant correlation between bone mineral density in the mandible or maxilla and that in the axial skeleton such as the spine and hip.

Mandibular anatomic indicators on panoramic radiography can be useful in the evaluation of bone resorption in different age groups of women to determine the existence of osteoporosis.[8]

Various investigators reported different levels of repeatability for CW, PMI, M/M ratio and CI. Generally authors used correlation coefficients in describing agreement between two quantitative variables but high correlation does not imply good agreement.[9,10]

Mandibular bone mineral density

The mineral content of cortical bone in the mandible is likely to be related to the mineral content of the skeleton. As a matter of fact, the mandible seems to be the bone within the human skeleton that is most exposed to severe decrease in its mineral content as it is one of the primary source of the available calcium in the body.[6,10,11]

The mineral content of the mandible is low in patients with osteoporotic fractures so that BMD (bone mineral density) of the mandibular bone is related with osteoporosis that is a low skeletal BMD, even if the conformity at other different levels of the skeleton is reduced. Using densitometry, the major part of the researchers, but not all of them, found that the optic radiological density of the mandible is grown in patients with osteoporosis and is related with low vertebral BMD.[3,12,13]

Mandibular cortical index (MCI)

Mandibular cortical index (MCI), the cortical thickness of the lower border of the mandible, could be easily observed on each panoramic radiograph and therefore it is a useful parameter for the assessment of the status of the mandibular bone.[12,14]

Cortical index has a relatively easier application, is a useful oral sign in screening the patients for osteoporosis and it might be used as a subsidiary diagnostic tool in referring the patients to bone densitometry clinics. In order to evaluate the CI of the mandible, the morphology of mandibular inferior cortex was visually examined distally from the mental foramen bilaterally using Klemetti’s classification. [5]

Cl 1: The endosteal margin of the inferior cortex is smooth on both ends.

Cl 2: The endosteal margin shows semilunar defects or appears to form endosteal cortical residues.

Cl 3: The cortex is obviously porous with dense endosteal residues.

Measurement of cortical thickness and subjective assessment of cortical porosity on panoramic radiographs are methods previously reported for diagnosing osteoporosis. The thickness of the mandibular cortical bone is decreased in osteoporotic patients and inferior border of the mandible is more porous than controls.[14-17]

The evaluating panoramic radiographs, showed that only those patients with the thinnest mandibular cortices (i.e., <3 mm) should be referred for further osteoporosis investigation. Mandibular cortical index is found to be useful in evaluating the patients for the risk of osteoporosis in various studies being significantly related to bone mineral density of the body. Because mandibular cortical bone is evaluated visually there is a limitation in its repeatability, especially between different observers, is reported to be a serious problem for the method to be used clinically, especially in inter-observer.[10,18-20]

Mandibular alveolar bone mass

The changes in the mandibular alveolar bone do reflect changes in the skeletal BMD, and these may be estimated on periapical radiographs by changes in their grey-level value and their texture.[21,22]. The association between skeletal bone mineral density (BMD) and mandibular alveolar bone mass has been reported to be rather weak, probably due to local functional factors.[23-24]
Inferior Cortex of the Mandible

The inferior bony cortex of the mandible is dense, wide, and appears as a very radiopaque strip of bone along the inferior border of the mandible. One of the most useful bony landmarks to use as an indicator for the analysis of bone metabolism is the mandibular angular cortex. The cortex layer of the angle of the mandible is not visible before the age of 15. After age 15, the thickness of the cortex is almost fixed except in women. In women it will become thinner over time. Cortical changes of the mandibular angular area were observed in women who were more than 20 years old. [25,26,27]

The diagnostic performance of mandibular inferior cortical shape detected on dental panoramic radiographs is useful for identifying postmenopausal and in postmenopausal women with histories of hysterectomy, oophorectomy, or estrogen use.[28]

Alveolar bone resorption

Alveolar bone loss is atrophy of the maxillary and mandibular bones that underlie and support the teeth, with reduction in bone height and volume. The primary cause is periodontitis, although tooth loss and osteoporosis may also contribute. The alveolar bone (the bone that used to surround the teeth) simply melts away (resorbes) after the teeth are extracted. The association between skeletal bone mineral density (BMD) and mandibular alveolar bone mass has been reported to be rather weak, probably due to local functional factors. The local factors played an important role in the posterior mandibular segment and their effect might partly explain the low correlation between MABM and skeletal BMD.[29,30]

The radiographic alveolar bone structure may be estimated on periapical radiographs by changes in their grey-level value and their texture.[31] Measurements of the mandibular alveolar process can be used as one of several parameters to predict skeletal bone density.[32,33] Changes in alveolar bone vary considerably across individuals and depend directly on local factors.

Many studies have investigated associations between alveolar bone status and bone mass at other skeletal sites.[34] It was demonstrated that, in healthy women, density of maxillary alveolar process bone is significantly related to the density of the mandibular alveolar process, lumbar spine, hip, and radius and that maxillary alveolar process bone density declines with age.[35]

Osteoporosis and periodontitis are two independent diseases. It is thought that these diseases are related as both damage bone tissue, share common risk factors, are most common in middle-aged and elderly women, and are very prevalent.[36,37]

The number of teeth present

The analysis of the relationship between the results of densitometric examinations and the number of teeth present showed strongly negative correlation with skeletal osteoporosis. There is any statistically significant interdependence between the number of teeth in the mandible and the mineral density of the examined bones (in normal and post-menopausal women).

There was not any influence observed of the decreased mineral status of the organism on the number of own teeth and the degree of periodontal disease advancement.[38,39]

Special considerations for implant therapy in osteoporotic patients

Treatment of partial and total edentulism with dental implants has evolved into a predictable procedure for the majority of patients and is expected to play a significant role in oral rehabilitation in the future. The long-term outcome studies which are now available for many of the implant techniques used indicate that successful integration and restoration of implants are now the expected therapeutic outcome. Today, in the general population, long-term success rates of over 90% to 95% are considered to be realistic treatment outcomes. However, there are few guidelines on dental implant therapy in this patient category, so that numerous issues regarding pre- and post-operative management remain unclear to the dental clinician.[40]

Before any form of endosseous implant therapy is considered in any patient, the medical history must be thoroughly reviewed and, if appropriate, a physical examination performed. The concern that dental implants are at an increased risk for failure in osteoporotic patients is based on the assumption that the impaired bone metabolism affects the mandible or maxilla in a manner similar to its effects on other bones. However, since a potential relationship between osteoporosis and decreased oral bone mass or density is controversial it is actually not easy to assess whether bone quantity and quality in the mandible and maxilla parallel those in the rest of the skeleton. This problem is due to the fact that reported studies have been preliminary, most of them having involved small numbers of subjects, biased sample selection, and
differing definitions and measurements of bone loss (i.e., interchangeable use of the terms "bone mass" and "bone density") and osteoporosis, as well as to the cross-sectional nature of the study design.

Another matter of concern is the assumption that impaired bone metabolism as it occurs in osteoporosis may affect osseointegration of implants. A more detailed look at the process of bone remodeling reveals that it is a non-uniform process. This process of bone remodeling differs from one bone to another, between cortical and trabecular bone, and from one trabecular bone site to another. In contrast to cortical bone, trabecular bone is much more affected by metabolic changes of the skeleton and is lost at an annual rate of 0.7% and 1.2% in males and pre-menopausal females, respectively. After menopause, the decrease in cortical and trabecular bone density accelerates to 1% and 6%, respectively, i.e., the decrease in trabecular bone density after menopause exceeds that of cortical bone. For this reason, bone like the maxilla, which consists largely of trabecular bone, is more susceptible to rapid and severe atrophy under conditions of disuse and/or metabolic demand for calcium than the mandible, which consists primarily of cortical bone. However, the observation that osteoporotic fractures usually heal readily suggests that the repair process in osteoporotic patients remains satisfactory and less susceptible to endocrine regulation, thus indicating that bone remodeling processes after implant placement in osteoporotic patients may also not differ fundamentally from those seen in healthy patients.[41]

It appears prudent for clinicians to adhere to the following guidelines when oral implants are to be placed in osteoporotic patients. Prior to implant surgery, a careful assessment of nutrition and systemic health in patients at risk for metabolic bone disease is recommended. In cases of insufficient bone volume, the implant sites should be augmented before or during implant surgery. Various bone augmentation methods are available. In addition, the occlusal load should be properly distributed throughout the dentition to avoid overloading the implant, which may contribute to implant loss.[42]

Preference should be given to implant designs that will have close bone-implant contact on insertion to ensure primary stabilization in less dense osteoporotic bone. In contrast to the concept of early mobilization in orthopedic treatment of fractures in osteoporotic patients, aimed at improving bone formation by mechanical stimulation, there is actually no precedent for advocating immediate or "provisional" loading of dental implants to enhance osteogenesis in osteoporotic bone.[43,44]

Implant therapy in systemic osteoporosis appears in certain textbooks of implantology as an absolute contraindication, which is relative in others’ opinion, or is not even mentioned, although it is a disease characterized by the loss of bone mass and bone density in all bones, including the jaws. In osteoporosis the metabolism is impaired, so theoretically implants osseointegration is hard to be achieved. In spite of this, systemic osteoporosis doesn’t mean that jaws can’t integrate endosseus implants, so it shouldn’t be an absolute contraindication of implant therapy. Although it was established a correlation between systemic bone loss, bone mass and bone density loss in jaws, no connection between systemic osteoporosis and implant’s failure was established.

Thus, even if osteoporosis appears more often in postmenopausal women, in studying the association between dual x-ray energy absoriometry and postmenopausal women and implant failure, did not find a higher failure rate for of implants placed in women older than 50 years as compared with women younger than 50 or between women and men older than 50. Neither of these studies have differentiated between maxillary and mandibular implants, but there are studies that show a higher failure rate of maxillary implants in postmenopausal women comparatively to premenopausal women. They reasoned that because osteoporosis affects trabecular bone more than cancellous bone and the maxilla has more trabecular bone content than the mandible, the maxilla is more susceptible to the effects of systemic osteoporosis.

Osteoporosis should not be an absolute contraindication for endosseus implants therapy, but cautiousness is needed when deciding maxillary for implant placement in postmenopausal women.

No surgical procedure, including the placement of implants, is without risk. The risks associated with implant placement include postoperative bleeding, numbness if the mandibular nerve is disturbed, infection and lack of osseointegration.

Implant loss has been extensively studied in relationship to type of prosthesis and arch, timing of the loss, effect of implant length, effect of bone quality, and relationship to systemic conditions. However, negative effects have not been reported in conjunction with osteoporosis. It appears prudent for clinicians to adhere to the following guidelines when oral implants are to be placed in osteoporotic patients:
1-Prior to implant surgery, a careful assessment of nutrition and systemic health in patients at risk for metabolic bone disease is recommended;

2-In cases of insufficient bone volume, the implant sites should be augmented before or during implant surgery. Various bone augmentation methods being now available;

3- the occlusal load should be properly distributed throughout the dentition to avoid overloading the implant, which may contribute to implant loss.

4-choosing implant designs that will have close bone-implant contact on insertion to ensure primary stabilization in less dense osteoporotic bone. In contrast to the concept of early mobilization in orthopedic treatment of fractures in osteoporotic patients, aimed at improving bone formation by mechanical stimulation, there is actually no precedent for advocating immediate or "provisional" loading of dental implants to enhance osteogenesis in osteoporotic bone.

Conclusions

This paper is a review of the literature on the possible association between osteoporosis and oral bone loss, with an emphasis on radiological studies. Such an association was first suggested in 1960.

Dentists are a potentially valuable resource for initial patient screening for signs of osteoporosis, as individuals with osteoporosis have altered architecture of the inferior border of the mandible as seen on panoramic radiographs.

It has noted that abnormalities in the width and morphologic structure of the mandibular inferior cortex, as imaged on a conventional panoramic radiograph, may be indicative of systemic osteoporosis (especially in postmenopausal).

Because the diagnostic accuracy of cortical width measurements is less than perfect, the dental panoramic radiograph would not be taken for osteoporosis screening per se, but could be evaluated for osteoporosis if the radiographs were exposed for dental purposes. The researchers also caution that radiographic measurements cannot be used as the sole basis for referral, and that the patient's medical history must be evaluated before undertaking further referral or investigation.[41]

As technology develops, dentists may become ideally situated to observe and monitor bone loss in their patients (regardless of age or gender), which may be a sign of bone loss in other parts of the body. Dentists are encouraged to refer patients suspected of being at risk for osteoporosis—based on medical history, including risk factors, and results of clinical and X-ray examination—to their primary-care physician for a complete health assessment.

References


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