Is Osteoporosis a Risk Factor for Osseointegration of Dental Implants?

T.T.T. Dao, DMD, MSc/J.D. Anderson, BSc, DDS, MScD/George A. Zarb, BChD, DDS, MSc, MS, FRCD(C), Odont Dr(HC)

The success of osseointegration depends in part on the state of the host bed. Concerns have therefore been raised about osteoporosis, a condition believed to be associated with a decrease in bone quality and quantity. However, the orthopedic literature indicates that osteoporotic fractures heal readily and that the level of bone mass and estimates of the parameters associated with bone remodeling present considerable overlap between patients with osteoporosis and control subjects. It also appears that osteoporosis, as diagnosed at one particular site of the skeleton, is not necessarily seen at another distant site. Although the prevalence of osteoporosis increases among the elderly and after menopause, the results of this study indicate that implant failure rate is not correlated with age and sex. A review of the literature and of results of a series of patients treated does not provide a compelling theoretical or practical basis to expect osteoporosis to be a risk factor for osseointegrated dental implants. (INT J ORAL MAXIFOLLAC IMPLANTS 1993;8:137-144.)

Key words: dental implant, osseointegration, osteoporosis

The use of osseointegrated implants in the treatment of edentulous patients has become an accepted alternative to conventional prosthetic dentistry. For the maladaptive edentulous patient or the patient with a severely resorbed ridge, an implant-supported dental prosthesis may be the treatment of choice. However because the success of osseointegration depends in part on the state of the host bone bed and its healing capacity, concerns have been raised about conditions affecting its quality and quantity. Osteoporosis, which is a disorder characterized by a generalized diminution in bone mass, may therefore represent "didactic contraindications" or a risk factor for osseointegration.

Epidemiologic studies indicate that bone loss occurs after the fourth or fifth decade in both sexes and after menopause in all populations studied. As bone loss increases with age and becomes a universal phenomenon among the elderly, the prevalence of osteoporosis also rises with age. Based on bone density measurements of the middle phalange of the fifth finger, Mangaroo et al reported that within a sample from the general population of the United States, about 25% of white women in the age range of 45 to 54 years and about 45% of white women in the age range of 65 to 74 years had evidence of osteoporosis. Based on radiographic
evidence of moderate or severe osteoporosis in the dorsolumbar spine of a group of unselected Michigan women, it was reported that in the age ranges of 45 to 49 years and 50 to 54 years, about 17.9% and 39.2% of the women, respectively, had osteoporosis; by age 75, almost all women did. On the other hand, it has been reported that at all ages after 50 years (in the United States), the incidence of hip fracture rises dramatically and is about twice as high for white women as white men. Thus, the literature clearly indicates that osteoporosis increases with age and is more prevalent in women than in men. Because age and gender are reported to be important risk factors for osteoporosis, the rate of implant loss caused by failure of osseointegration may also be expected to increase correspondingly, and a large proportion of the target population for dental implants may have a higher risk of implant failure. If this is the case, the choice of prosthodontic treatment (ie, conventional versus implant-supported prosthesis) in elderly patients would need to be reassessed.

The concern that osteoporosis is a contraindication for dental implants presumes that: (1) osteoporosis affects the mandible or the maxilla in the same manner as the other parts of the skeleton that serve as diagnostic markers of the disease and (2) the impaired bone metabolism in osteoporotic bone may reduce the healing capacity of bone around implants. The objective of this article is to discuss whether osteoporosis should be considered a risk factor for dental implants based on a review of the literature pertaining to osteoporosis and a survey of the patients in the Toronto implant study.

Definition of Osteoporosis
In the 1930s, osteoporosis was defined as "too little calcified bone." More recently, osteoporosis has been used to refer anatomically to a condition of generalized reduction in bone mass with no other abnormality. However, there seems to be no general agreement regarding the interpretation of the level of bone mass loss as being indicative of osteoporosis. Nordin proposed that "osteoporosis is present when the concentration of bone (mineral) lies more than two standard deviations below the mean of young adults of same sex." Thus, the threshold value is set to the mean of young normal adults. Mazess suggested that the threshold value be based on the distribution of bone mass in fracture patients. Meunier (as cited in Kanis et al) considers that patients with iliac crest bone volume of 11% or less of the sectional area have osteoporosis in the sense that their risk of fracture is extremely high. Hence, definitions of osteoporosis made solely on the basis of bone mass remain empirical and arbitrary.

Clinically, the definition of osteoporosis may be based on the presence of nonviolent fracture or on a fracture threshold, such as a reduction of bone mass that increases susceptibility to fracture. The disease is then studied in its manifestations or clinical signs, rather than with a simple measure of bone quantity. However, it is well known that clinicoradiologic definitions and anatomic definitions...
often lead to conflicting results. Ott et al\textsuperscript{12} reported that bone mass index correlated poorly with fracture index. Reduced bone mass does not always lead to fracture,\textsuperscript{13} because fracture risks may be the result of other age-related factors,\textsuperscript{4,14-16} (eg, frequency and pattern of fallings, neuromotor coordination, specific diseases such as Parkinson's, arthritis). Furthermore, an anatomic diagnosis of osteoporosis can be made in the absence of clinical and radiologic signs.\textsuperscript{13,17} Many subjects without fracture may have lost a significant amount of bone mass,\textsuperscript{11} and many patients with fracture may have a level of bone mass that is indistinguishable from that of the control subjects matched for age and sex.\textsuperscript{13,18,19} In fact, the overlap between the two groups in terms of bone mass is considerable \textsuperscript{8,10,18-21}

In reference to the orofacial complex, Kribbs\textsuperscript{22} reported significant differences "between normal and osteoporotic populations in mandibular bone mass and density, in cortical thickness at the gonion" and concluded that "these differences suggest that osteoporosis does have an effect on mandibular bone." However, the "differences" reported are overwhelmed by the considerable overlap shown in the data, and the statistical test performed to confirm the results was not reported. Rather, the data seem to indicate that these two populations are indistinguishable from one another in terms of mandibular bone mass measurements.

Definitions of osteoporosis based on reduced bone mass or nonviolent fracture seem not to be synonymous. Therefore, a patient diagnosed as osteoporotic does not necessarily have an "abnormal" amount or quantity of bone either in the jaws or in the other parts of the skeleton. Because osteoporosis is difficult to define and measurements of bone mass are difficult to interpret, the disease is mostly diagnosed in the presence of its clinical manifestations. Thus, the definition of osteoporosis in the literature to be reviewed herein refers mostly to the presence of fractures unless otherwise specified.

**Do Changes at one Skeletal Site Reflect Changes Elsewhere?**

On the assumption that osteoporosis impairs the success of dental implants, the question may arise whether information derived from one part of the body affected by the disease (eg, spine for crush fracture, wrist for Colles' fracture) can consistently reflect changes elsewhere in the skeleton (eg, the jaws). It has been reported that (1) the rate of bone loss may differ for the axial and for the appendicular skeleton\textsuperscript{23}; (2) osteoporotic bone loss affects preferentially the more metabolically active trabecular bone; and (3) fractures occur at sites predominantly composed of trabecular bone, such as the spine.\textsuperscript{8} Krolner and Nielsen\textsuperscript{24} reported that although the correlation between the measures of bone mineral content of the lumbar spine and the forearm was significant for normal individuals, it is not significant in patients with osteoporosis. The lack of correlation of bone mass between trabecular bone and cortical bone in such patients was consistent with results of several other studies.\textsuperscript{18,20,25} This was confirmed by histomorphometric data showing that bone modeling is focal, varying between skeletal sites at any given time and varying from...
time to time within one site.\textsuperscript{26,27}

The relationship between mandibular and skeletal bone mass has also been studied. The mandible consists primarily of cortical bone, even at the broadest part of the mandibular body.\textsuperscript{28} In the normal population, mandibular bone mass does not appear to be related to bone mass of other parts of the skeleton that consist mainly of trabecular bone (e.g., the iliac crest).\textsuperscript{29,30} Von Wovern\textsuperscript{30-32} reported that mandibular bone mass was related to bone mass in other skeletal cortical bone, although this relationship is rather weak.

In osteoporotic populations, Kribbs et al\textsuperscript{33,34} reported a statistically significant correlation in bone density between mandibular residual ridge, alveolar bone density, distal radius measurements, and total skeletal bone mass. However, there was a substantial spread around the regression line of data correlating mandibular to skeletal bone,\textsuperscript{34} so that the results obtained by one procedure at a given site cannot be used to reliably predict those acquired with the other technique or from another site. Moreover, different techniques were used for different sites, although it is reported that each technique of measuring bone mass may produce a different assessment of bone mass as well as measure somewhat different characteristics of bone.\textsuperscript{3,35-37}

It appears that changes in bone mass in one site do not always reflect changes at another site. It would therefore be inappropriate to conclude that maxillary and/or mandibular bones are osteoporotic in patients with spinal or Colles' fracture, unless the diagnosis is based on the measurement made at the latter sites.

**Bone Metabolism in Osteoporosis**

It is generally believed that bone loss may be the result of an abnormal accentuation of the disequilibrium between bone resorption and formation, resulting from a decrease in bone formation, an increased bone resorption, or a combination of the two.\textsuperscript{38,39} Although this may be true for some patients with osteoporosis, the disease is also characterized by anatomic and histopathogenetic heterogeneity, with different disturbances in bone remodeling at the organ tissue and cell levels, leading to the same syndrome.\textsuperscript{40} Remodeling occurs by the continuous removal and replacement of bone tissue, which help to maintain the biomechanical competence of the skeleton, that is, its capacity to withstand load without accumulating fatigue damage. The speed and extent of the replacement regulate the rates of bone loss or gain at specific locations and times.\textsuperscript{41} Although bone remodeling may be hyperkinetic and lead to accelerated bone loss in some osteoporotic patients, the process is low or normal in most patients. This has been confirmed by bone histomorphometry, which is accepted as the only method that provides a direct precise analysis of the static and dynamic cellular and tissue abnormalities.\textsuperscript{40} Iliac crest biopsy specimens have shown bone turnover to be high in about 25\%, normal in about 45\%, and low in about 30\% of patients.\textsuperscript{42,45} From an analysis of 154 iliac bone samples, Meunier et al\textsuperscript{46} reported that "there are 48\% of the patients without detectable abnormality of remodelling
surfaces and appositional rate at the time of the biopsies"; only 33% of the patients had trabecular osteoclastic resorption surfaces higher than two standard deviations above the normal.

These results appear to be consistent with the observation that the biochemical tests of bone metabolism are generally normal. Estimates of parameters associated with bone remodeling, such as serum alkaline phosphatase, urinary hydroxyproline, fasting urine calcium/creatinine ratio, radio-calcium kinetics, or the bone gamma carboxyglutamic acid containing protein (osteocalcin), may be mildly elevated but are more frequently normal.

Thus, as with bone mass measurements, a substantial overlap is present when one compares the rate of bone remodeling between osteoporotic patients and normal populations.

**Bone Healing in Osteoporosis**

The presumption that osteoporosis represents a risk factor for osseointegration may partly be derived from the belief that the disease is associated with a deficiency in bone formation, thus compromising the healing capacity and the apposition of bone at the bone-implant interface. Although data comparing the healing rate of bone in control and osteoporotic populations are not available, as previously noted, histomorphometric studies have shown that bone remodeling was normal in a large proportion of patients diagnosed as being osteoporotic. The clinical heterogeneity observed with bone remodeling in osteoporotic patients may reflect the phasic fluctuation of the disease, but it is also possible that bone metabolism has already returned to its normal state by the time the condition is diagnosed. Nonetheless, the observation that osteoporotic fractures usually heal readily suggests that the repair process in osteoporotic patients remains satisfactory. This may also be related to the fact that woven bone formation, which plays an important role in postfracture bone union, is less susceptible to endocrine and other regulation than is lamellar bone. As commented by Parfitt, "The controlled production of woven bone in specific locations may turn out to be an important strategy for the restoration of normal bone structure in severe osteoporosis."

Another hypothesis is that changes in the quality of bone (bone architecture, sizes of mineral crystal) may reduce the strength of bone containing an adequate amount of mineral. This would predispose bone to fracture or compromise the state of the host bed receiving the dental implants. However, those changes are at present either clinically undetectable or only accurately assessed by invasive technique such as bone biopsy. Thus, whether osteoporosis affects bone quality, bone quantity, or both remains a matter of controversy, with no other clear trends or distinguishing factors evident.

**Survey of Patients in the Toronto Implant Study**

An informal review of the Toronto implant study patient series was done to
determine whether age and sex, which have been recognized as important risk factors for osteoporosis, are also risk factors for patients with dental implants.

The sample population included 93 women and 36 men, aged 20 to 76 years at the time of implant placement, having Brånemark implants in function for 2 to 11 years. Sixty-six patients were completely edentulous and 63 were partially edentulous. Inclusion and exclusion criteria related primarily to the dental needs of the patients and therefore were not related to osteoporosis. No patients were excluded because of preoperative radiographs indicating poor bone quality; therefore, no osteoporotic patients would have been excluded on this basis. However, because this is a retrospective study, the prevalence of osteoporosis in the population could not be established.

Implant failure was defined using the criteria of Smith and Zarb\(^5\)\(^4\) and was expressed in terms of the number of subjects having at least one implant lost in each age range. This method respects the independence required between the variables. Comparison was made between (1) women and men over age 50 years and (2) women above and below age 50, using the chi-square test (and Fisher's exact test where the sample size was too small). The chi-square test was also used to assess the relationship of age at the time of implant placement with prevalence of implant failure. A logistic regression model using six age groups and gender to predict the probability of implant failure was completed.

The distribution of subjects according to gender and age is shown in Table 1. There were 45 women and 18 men over age 50, and 48 women and 18 men under age 50. The rates of failure in each group were 22.2% versus 22.2%, and 18.8% versus 11.1%, respectively (Table 2). The difference between men and women above age 50 was found to be not significant according to the Fisher's exact test (\(P = 1\), Table 2). Likewise, no statistical difference in implant failure rates between women over and under age 50 could be shown (\(P = .68\), \(\chi^2\); Table 2). The association between age at the time of implant placement and the prevalence of failure was not significant for women (\(P = .67\)) or for the whole study sample (\(P = .19\)). In both cases the highest failure rates appeared in the younger age groups (Figs 1 and 2), a trend opposite to what may be expected. Similarly, logistic regression revealed no association between gender or age and prevalence of implant failure (\(P = .22\) for gender; \(P = .93\) for age). Our data suggest that the subjects at risk for osteoporosis were not at risk for implant failure. Also, the rate of implant failure was not correlated with age and sex. This observation is consistent with the data reported by Köndell et al.\(^5\)\(^5\). It must be emphasized that the strength of this study is limited by its retrospective nature, its small and possibly nonrepresentative sample size, and the variable length of time elapsed since implant placement. Also, the prevalence of osteoporosis in this sample size was not established. With these difficulties clearly understood, great caution must therefore be exercised in arguing that osteoporosis outside the jaws is not a risk factor for the placement of dental implants. This
argument is based on the lack of correspondence between age and sex as risk factors for implant failure and age and sex as established risk factors for osteoporosis. To that extent, these findings are not in conflict with the literature.

As already noted, the prevalence of osteoporosis in asymptomatic women has been reported to be as high as 25% for the age range of 45 to 54 years\(^5\) and 39.2% for the age range of 50 to 54.\(^6\) If osteoporosis is indeed a risk factor for dental implants, one might expect, with the previous caveats in mind, that the number of subjects with implant loss would approximate the prevalence of undiagnosed instances of osteoporosis reported in individuals of same sex and age range. The lack of association of implant failure with gender, as well as the opposite age trends observed in the implant failure rate (a decrease with age) and the prevalence of osteoporosis (an increase with age), suggest that it is unlikely that osteoporosis has had any influence on implant failure.

The present review also indicates that osteoporosis at one particular site of the skeleton is not necessarily seen at another distant site. The correlations reported, even if positively and statistically significant in a population, are sufficiently poor\(^32\) to be of no predictive value on an individual basis. For instance, a diagnosis of osteoporosis established on a history of hip fracture or Colles' fracture does not necessarily imply that the bone quality or quantity of the mandible (or maxilla) is reduced. This is not surprising because bones in different locations are subjected to different biomechanical stress,\(^41\) which in turn is known to influence the remodeling of the bony tissue.\(^56,57\) As suggested by von Wovern,\(^32\) "The best method for the evaluation of bone mass changes in the mandible is to analyze the mandible itself." Moreover, while the correlation of bone mass levels between various skeletal sites can be used to estimate the extent to which the bone mass at one site is reflected by that of another site, it cannot be used to quantitate the strength of a relationship with risk.\(^58\) For instance, low bone mass in the radius may lead to an increased risk of fracture, but low bone mass in the mandible is not necessarily an indication of increased risk of implant loss, even though the correlation between the bone mass of the two sites is positively correlated.

Because the state of the host bed for dental implants could not be extrapolated from that of a distant bone the prediction of implant failure may be better determined by a local radiographic or clinical assessment of the surgical site. For instance, the rate of implant failure has been reported to be higher in the maxilla than in the mandible,\(^59-61\) although, discounting the learning curve, no difference in failure rate was found in the present population. The failure rate is also greater in the posterior mandible when compared with the region anterior to mental foramina.\(^62\) The situation in which the host bed for an implant appears to be of bone quality type IV, as classified by Lekholm and Zarb,\(^63\) may be analogous to the circumstance where osteoporosis is diagnosed primarily in the maxilla or the mandible. Bone quality type IV is believed to be unfavorable for osseointegration possibly because of the marked
porosity of bone, because this may offer little mechanical anchorage to the implant to ensure its stability. The implant failure rate in such sites has been reported to be greater than that in the bone qualities type I to III.\textsuperscript{62,64} However, these results need to be confirmed by further clinical trials because the method of classification of bone type was merely based on subjective evaluation of the surgeon\textsuperscript{62} or failed to be calibrated between operators in radiographic interpretation or assessment of bone quality.\textsuperscript{64}

The long-term failure rate of orthopedic implants used in the treatment of osteoporotic fractures is high and increases dramatically with time.\textsuperscript{65} However, those results cannot be automatically extrapolated to the field of osseointegration because orthopedic implants are usually cemented or screwed in place, and their attachment mechanism to the bone or their mode of failure is completely different from that of an osseointegrated implant.\textsuperscript{65,66} Moreover, in contrast to conventional orthopedic implants, it has been well established that the overall success rate of osseointegrated fixtures remains stable over time.\textsuperscript{65}

**Conclusion**

A review of the literature and a separate descriptive analysis of our patient treatment series do not provide a compelling theoretical or practical basis to confirm osteoporosis as a risk factor for osseointegration of dental implants. Therefore, denying implant treatment to a patient whose diagnosis of osteoporosis is based on a decrease in bone mass or on the presence of atraumatic fracture in a site other than the jaw itself cannot be supported at this time. It is important that treatment planning for dental implant therapy be based on a local assessment of the potential surgical site.

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Table 1 Distribution of Subjects According to Gender and Age at the Time of Implant Placement

<table>
<thead>
<tr>
<th>Age (y)</th>
<th>20-29</th>
<th>30-39</th>
<th>40-49</th>
<th>50-59</th>
<th>60-69</th>
<th>70-79</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of women</td>
<td>6</td>
<td>17</td>
<td>25</td>
<td>30</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>No. of men</td>
<td>1</td>
<td>4</td>
<td>13</td>
<td>12</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>7</td>
<td>21</td>
<td>38</td>
<td>42</td>
<td>14</td>
<td>7</td>
</tr>
</tbody>
</table>

Table 2 Number of Subjects (and Implant Failure Rate*) Among Men and Women Above and Below Age 50 Years

<table>
<thead>
<tr>
<th>Age</th>
<th>Women</th>
<th>Men</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;50 y</td>
<td>48 (18.8%)</td>
<td>18 (11.1%)</td>
</tr>
<tr>
<td>≥50 y</td>
<td>45 (22.2%)</td>
<td>18 (22.2%)</td>
</tr>
</tbody>
</table>

*Failure rate was expressed in terms of percentage of subjects in each age group having implant failure. The failure rates were not significant between men and women above age 50. Likewise, no difference could be shown between women over and under age 50.
The rate of failure was expressed in terms of percentage of subjects in each age group having implant failure(s). The association between age at the time of implant placement and the rate of implant failure was not significant. The group sizes vary between the age groups as shown in Table 1.

The rate of failure was expressed in terms of percentage of subjects in each age group having implant failure(s). The association between gender or age at the time of implant placement and the rate of failure was not significant. The group sizes vary between the age groups as
shown in Table 1.