Osteoporosis, Fracture Risk, and Prosthodontic Implications

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Osteoporosis, Skeletal Fracture Risk Assessment, and Medications

According to the National Osteoporosis Foundation (U.S.), osteoporosis, or porous bone, is a disease characterized by low bone mass and structural deterioration of bone tissue leading to bone fragility and an increased susceptibility to fractures, especially of the hip, spine, and wrist, although any bone can be affected.[1] In simpler terms, osteoporosis is a condition in which the bones become weak and can break from a minor fall or, in serious cases, from a simple action such as a sneeze.

The use of bone mineral density as a surrogate for bone strength and resistance to fracture has become the standard of care to predict fracture risk for the last 3 decades. However, the importance of other risk factors such as weight, age, gender, past fracture history, maternal fracture history, smoking history, glucocorticoid history, secondary osteoporosis, and alcohol use are well recognized.[2] Recently presented, the World Health Organization Fracture Risk Assessment Tool (FRAX calculator) combines the parameters of these different risk factors to provide a 10-year probability of major osteoporotic and hip fracture.[3] The anticipated benefit of this new tool is to provide a more sensitive and specific prediction of fracture in a way that supersedes what measures of bone mineral density alone can provide. Guided by a threshold of 10-year fracture risk, which supports institution of pharmacotherapy, it is believed that the Risk Assessment Tool will facilitate shared decision-making and ultimately adherence to pharmacotherapy to reduce the risk of osteoporotic fracture.[4]

Medications

There is no cure for osteoporosis. Pharmacologic therapy has therefore focused on slowing net bone loss by using antiresorptive medications such as sex hormones or bisphosphonates. Hormone therapy (HT) has diminished and quite markedly so, since the publication of results from the Women's Health Initiative indicate that HT along with smoking predisposes patients to an increased risk of thromboembolism, stroke, and breast cancer.[5]

The drop in HT use has led to increased use of bisphosphonates such as alendronate, risedronate, ibandronate, and zoledronic acid. The former two are available as oral tablets, whereas zoledronic acid is available in the United States as a once-yearly intravenous (IV) infusion. Ibandronate can be administered either in oral form or by IV infusion once every 3 months. It is likely that the IV infusions will become more common as poor patient compliance with oral forms is rendered moot.[6] Both HT and bisphosphonates act to retard bone resorption. Also available to inhibit bone resorption are a family of medications known as SERMs, or selective estrogen receptor modulators. Calcitonin may be used to diminish the rate of bone resorption in women who are more than 5 years postmenopause. In addition, and distinctly different to the aforementioned medications, is teriparatide. A form of parathyroid hormone given intermittently as a daily subcutaneous injection, it has anabolic effects on bone, ie, enhances bone formation. Teriparatide is used most often when a patient has very low bone mineral density (BMD), is of a young age, and/or has a high risk of fracture.

Prosthodontic Implications

There is conflicting evidence supporting an association between an osteoporotic and osteopenic condition, BMD with periodontitis,[7–9] and tooth loss.[10] Intriguingly, the use of bisphosphonates may protect women with low bone mineral density from alveolar bone loss.[11] Although intuition suggests that the osteoporotic individual would suffer from accelerated bone or tooth loss, it is unclear whether the jaw bones are responsive to menopause in a manner similar to other susceptible bones.

The relationship between osteoporosis and dental implant survival is poorly understood because there are few studies that directly link bone mineral density, the historically accepted method of diagnosing osteoporosis, with implant survival. Most literature has utilized surrogates of osteoporotic condition, eg, effect of age[12] or hormone replacement therapy,[13–14] to indicate the limited association between the osteoporotic condition and implant survival or has offered only exploratory findings.[15] Recently, Holahan et al[16] demonstrated that neither bone mineral density nor osteoporotic condition was associated with dental implant survival, regardless of whether the implants presented

a machined titanium surface or an anodized "rough" surface. Collectively, these data support the notion that dental implant survival in postmenopausal women is not influenced by systemic bone mineral density or a diagnosis of osteoporosis.

Management of the patient undergoing bisphosphonate therapy who needs a dental extraction or desires implant placement has been complicated by case reports of bisphosphonate-associated osteonecrosis of the jaw (ONJ).[17] Current evidence indicates that the risk of ONJ in patients receiving IV bisphosphonate therapy to prevent skeletal events secondary to cancer or Paget disease is reasonably high (from 5% to 16%).[18–19] However, prophylactic antibiotic use prior to and immediately postextraction may prevent ONJ in some cancer patients and is indicated for these patients.[20] In patients receiving bishphosphonate therapy for osteoporosis or osteopenia, the risk of developing ONJ subsequent to a dental extraction is relatively low. The currently reported incidence of ONJ in this population ranges from 1:300 of those needing a dental extraction[21] to 1:250,000 in the general population of osteoporotic/osteopenic individuals.[22] The wide range is indicative of the considerable paucity of data upon which to base clinical recommendations.[23] For patients wishing to receive dental implant therapy, our experience at Mayo Clinic has been favorable and pursuant to proper disclosure regarding the unknown degree risk of ONJ, and implants are not being withheld from patients who have a past or current history of bisphosphonate use.

Marx et al[24] have recently suggested the use of morning fasting levels of a serum marker of systemic bone turnover (C-terminal telopeptide of collagen or CTX) to indicate the risk of developing ONJ and to guide treatment decisions. At this time, it is the opinion of the authors that the lack of validated objective measures of healing presented in this paper along with concern regarding both the lack of sensitivity and specificity of serum CTX levels and the inter-assay and intra-assay variability of the CTX assay utilized by Marx et al indicate that CTX testing as a method to assess either risk of developing ONJ or guide treatment decisions is unjustifiable and, hence, may amount to unnecessary testing.

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