REVIEW ARTICLE

Osteoporosis and Orthodontics: A Review

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Abstract

Orthodontic tooth movement needs simultaneous bone formation and resorption. Patients with high risk of bone resorption (osteoporosis), due to systemic problems, may have a deleterious effect on tooth movement. With increased adults seeking orthodontic treatment, the number of such patients seeking orthodontic treatment have increased in last few decades. It so has become of prime importance for an orthodontist to diagnose such patients and take necessary actions. This article will deal with osteoporosis in general, its pathophysiology, the signs, and symptoms, its effect on orthodontic tooth movement, its treatment, and the necessary actions to be undertaken by the orthodontist.

Introduction

Orthodontic tooth movement needs simultaneous bone formation and resorption. Patients with high risk of bone resorption (osteoporosis), due to systemic problems, may have a deleterious effect on tooth movement. With increased adults seeking orthodontic treatment, the number of such patients seeking orthodontic treatment have increased in last few decades. It so has become of prime importance for an orthodontist to diagnose such patients and take necessary actions.

"Osteo" is a Latin word for bone. "Pores" means "full of pores or holes."[1] Thus, the term "Osteoporosis" literally means "condition of porous bone."[2]

In 1930, osteoporosis was simply defined as too little calcified bone.[3] More recently, it is defined as a condition of the generalized reduction in bone mass with no other abnormality.[4]

Nordin[3] proposed that the osteoporosis is present when the concentration of bone (mineral) lies more than two standard deviation below the mean of young adults of the same sex.

Mazess[5] suggested threshold value be based on the distribution of bone mass in fracture patients.

Clinically,[6,7] 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.

Ott *et al.*[8] reported that bone mass index correlated poorly with fracture index.

Reduced bone mass does not always leads to fracture.[9] Fracture risks may be the result of other age-related factors like neuromotor co-ordination, frequency and pattern of falling, arthritis, Parkinsons, etc.[10-13]

Osteoporosis before fracture is termed as "Silent disease."[14]

World Health Organization (WHO) in 1994 defined osteoporosis by a bone mineral density (BMD) measured as 2.5 standard deviations below average peak bone density achieved in young adults, matched by gender and race.[15]

Classification of Osteoporosis

Osteoporosis can be classified as:

- Localized osteoporosis[16]
- Generalized osteoporosis – This can further be classified as

- Primary osteoporosis
- Secondary osteoporosis

Primary osteoporosis[17] is the most common form and is diagnosed when other disorders known to cause osteoporosis are not present.

It can be classified according to age as:

1. Juvenile osteoporosis - It affects prepubescent boys and girls
2. Idiopathic osteoporosis - It affects young adults
3. Post-menopausal - It affects ladies 15-20 years after menopause
4. Senile - It affects elderly patients.

Secondary osteoporosis - It is diagnosed when the condition is related to another illness, nutritional complications or as a side effect of medications.[6]

Factors that contribute to secondary osteoporosis.[18]

Metabolic	Medication	Nutrition
Autoimmune Disease	Corticosteroids	Vitamin D/Calcium/Phosphours deficiency
Endocrine/Hormone Disorders	Vitamin D toxicity	Alcoholism
Digestive Disorders	Thyroid hormone	Malabsorption/Malnutrition
Blood Disorders	Heparin	Gastric surgery
Nervous System Disorders	Hormone agonists	Tobacco
Mental Illness	Anticonvulsants	Caffeine
Cancer	Chemotherapy drugs	Cola drinks

Risks Factors Associated with Osteoporosis

Factors that increase the chances of osteoporosis:[6]

1. History of fracture
2. Family history - small build
3. Race (Caucasians/Asian)
4. Gender - post-menopausal female
5. Physical inactivity
6. Poor calcium/vitamin D intake
7. Tobacco use
8. Excessive caffeine, alcohol consumption
9. Anorexia nervosa
10. Some medications and medical conditions
11. Amenorrhea.

Decreased risks:[6]

1. Estrogen replacement therapy
2. Large bone mass
3. Race - (African-American)
4. Gender - Male
5. Weight bearing exercise
6. Overall, adequate diet
7. Non-smokers.

Detection/diagnosis of osteoporosis:

- Osteoporosis is the process of quantitative loss of bone density per unit volume, with maintenance and reduction of the qualitative properties of mineralized bone.

The diagnosis of osteoporosis is done either by laboratory analyses or radiographic evaluation.

Laboratory analyses[19]

The instrumental diagnosis of osteoporosis requires radiological investigations and measurements of bone mineral content.

The laboratory analyses contemplate:

- Calcium and Phosphorus quantification in serum
- Electrophoresis of serum proteins
- Speed of erythro-sedimentation
- Alkaline serum phosphatase (increase in the presence of fractures)
- Dosing of the serum parathormone (PTH)
- Osteocalcine serum
- Calciuria
- Urinary excretion of peptides containing hydroxyproline and of the pyridine peptide.

Radiographic Evaluation

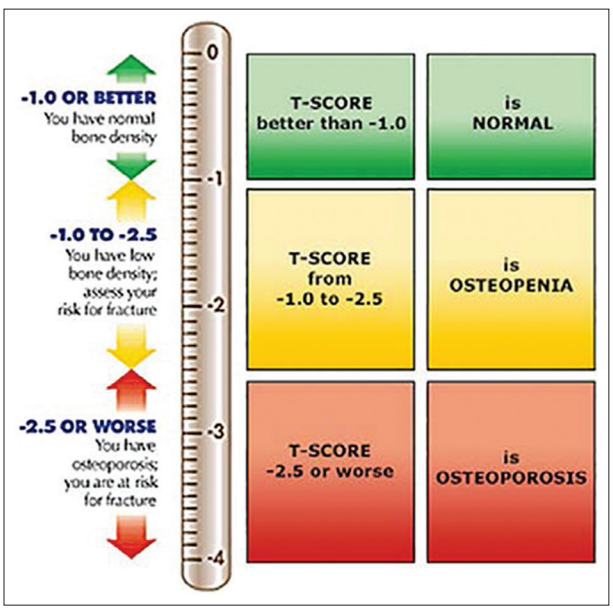
A bone density scan or quantitative ultrasound[20] is a way to measure bone density by using ultrasound waves. There are a number of advantages to this type of test:

1. Portability: Can reach a greater number of people in a public health setting.
2. Inexpensive: An important consideration for uninsured patients.
3. No radiation exposure: Better patient compliance.
4. A good predictor of risk fracture: Patient education.

FDA approved indications for BMD test is:

1. Estrogen deficient women
2. Vertebral abnormalities on X-ray
3. Gluco-corticoid treatment equivalent to or more than 7.5 mg prednisone per day or duration of therapy more than 3 months.
4. Primary hyperparathyroidism.

DEXA is short for dual X-ray absorptiometry. It is also known as bone densitometry or the BMD test/scan. It is really the "gold standard" of bone density measurement techniques. DEXA uses a very low radiation dose but is contraindicated during pregnancy.[20]



T-score classifications from the WHO.

Dental X-rays

1. Intraoral periapical X-rays
   a. Alveolar bone
2. Panoramic X-rays
   a. Panoramic mandibular index
   b. Morphology of mandibular inferior cortex
   c. Mandibular cortical width
   d. Panoramic analysis
   e. Antegonial index
   f. Residual ridge height[5]

Saliva as a biomarker

Bone turnover can be currently monitored by analyzing the biomarkers such as osteocalcin, pyridroline cross-links in serum and urine.[16] McGhee and Johnson (2004) determined salivary D-Pyr and osteocalcin as biomarkers of bone turnover.[21]

Osteoporosis and orthodontic tooth movement

Osteoporosis has been found to change the speed of tooth movement. Yamashiro *et al.*,[22] Tan *et al.*,[23] reported a correlation between increased tooth movement and an increased number of osteoclasts in ovariotomized rats. In an immuno-histochemical study, Yang *et al.*,[24] showed that expression of interleukin-1 beta, which is important for bone remodeling, was increased in osteoporotic rats when subjected to orthodontic forces. In ovariectomized rats, Arslan *et al.*,[25] showed that the osteoclast counts were higher, whereas the osteoblast and osteocyte counts were lower than in the control group. Orthodontic tooth movement may have a high tendency to relapse in patients with osteoporosis, regardless of whether it is of the postmenopausal or senile type.[26] Miyajima *et al.*,[27] classified osteoporosis on the basis of the results from the study by Orimo *et al.*,[28] into two types: (1) Postmenopausal osteoporosis with accelerated bone formation but with even more accelerated bone resorption; and (2) senile osteoporosis with depressed and imbalanced bone resorption and formation.

Corticosteroid-induced osteoporosis and orthodontic tooth movement

Corticosteroid-induced osteoporosis involves the uncoupling of the normal relationship between bone formation and resorption, resulting in a net decrease in bone formation. The osteoblastic and osteoclastic function is directly inhibited by corticosteroid.[29-31] Ashcraft *et al.*,[32] found that corticosteroid administered rabbits had elevated osteoclastic activity, increased alveolar bone resorption, and suppressed bone deposition, which accelerate tooth movement with subsequent greater relapse. Verna *et al.*,[33] found more root resorption in an acute corticosteroid treatment group when compared with the control and a chronic group and at the same time reduces bone turnover. Therefore, orthodontic treatment might best be postponed until a time when the patient is taken off medication.[34]

Osteoporosis and Orthodontic Considerations

- Assess periodontal condition
- Refer for periodontal treatment
- Enforce regular periodontal recalls during orthodontic treatment
- Discuss with patient's physician eventual temporary cessation of corticosteroid therapy
- Discuss with patient's physician eventual temporary cessation of estrogen supplement
- Adjust forces according to alveolar bone height
- Apply lighter and controlled forces
- Ensure less extensive treatment plan
- Ensure prolonged retention and effective retention regimen.

Drugs for the treatment of osteoporosis[19]

The principal pharmacological drugs for the therapy of osteoporosis are listed below (also in order by aggressiveness):

- Calcium (1500 mg/day)
- Vitamin D2 and vitamin D3 (altogether 400 international units/day)
- Sodium fluoride (50 mg/day)
- PTH (to daily dosing, low and intermittent)
- Salmon calcitonin (200 international units per day for inhalation; or 100 international units per day intravenous
- Strontium ranelate
- Bisphosphonates (with different types of dosage according to the molecule employed).

Bisphosphonates and osteoporosis

Bisphosphonates are synthetic analogs of inorganic pyrophosphates that have a high affinity for calcium. Bisphosphonates are drugs used to slow done or prevent bone damage. Bisphosphonates are often called "bone hardening" or "bone strengthening" drugs. There are several different types of bisphosphonates. The bisphosphonates are absorbed by the osteoclast cells, whereby increased activity of osteoclasts is reduced which leads to a reduction in breakdown of bone. They have been used in the management of systemic metabolic bone diseases including osteoporosis and osteopenia in postmenopausal women, corticosteroid-induced osteoporosis, multiple myeloma, bone metastasis of various cancers, hypercalcemia, and severe Paget's disease.

Recently, the literature shows a high rise of cases reported of osteonecrosis associated with the use of bisphosphonates.[35,36]

Bisphosphonates and orthodontic tooth movement

Animal studies have been undertaken to study the effect of bisphosphonate therapy on orthodontic tooth movement. Igarashi *et al.*,[37] found that in rats there was inhibition of tooth movement after topical administration of bisphosonates. Rinchuse *et al.*,[38] reported that in patients undergoing bisphosphonate administration, orthodontic tooth movement took longer, and bodily movements were limited.

Orthodontic considerations in patients treated with bisphosphonates

- Take good medical history
- Obtain consent after explaining risks and side effects of bisphosphonate therapy
- Assess risk level for orthodontic treatment and osteonecrosis
- Consider morning fasting serum test for C-terminal telopeptide test prior to invasive surgical procedures
- Take preventive measures against osteonecrosis
- Avoid invasive dental procedures in orthodontic treatment plan
- Consider alternative dental treatment plan
- Ensure retainers are passive with no pressure on the bone covering soft tissues
- Stay up-to-date with current research.

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