

Episode #42 – Osteoporosis and Oral Health August 13, 2021

Osteoporosis[1]

Osteoporosis is a progressive metabolic bone disease characterized by low bone density, deterioration of bone tissue, and an increase risk of fracture. [1] [2] Skeletal weakness leads to fragility fractures, especially in the thoracic and lumbar spine,[2] wrist, hip, and shoulder. [3] [4] Fragility fractures are bone fractures that occur from minor stresses (e.g., falling out of bed, falling from standing height, lifting, bending, sneezing) or inapparent trauma. Osteoporosis can occur at any age and affects both women and men. However, it is more common among women and older individuals. [2]

Osteoporosis is diagnosed by dual-energy x-ray absorptiometry (DXA). Prevention and treatment include risk factor modification; calcium and vitamin D supplementation;[3] physical activity to maximize bone and muscle strength, improve balance, and minimize the risk of falls; and pharmacologic therapy to preserve bone mass or stimulate new bone formation. [3]

Osteoporosis and oral health

Osteoporosis can impact the bone of the jaws, including the alveolar bone. Several studies have found a link between alveolar bone loss and an increase in tooth mobility and tooth loss. For example, women with osteoporosis are three times more likely to experience tooth loss than those without the disease. [5]

Low jaw bone density can also cause other oral health problems. For example, older women with osteoporosis may be more likely to have difficulty with loose or ill-fitting dentures and may have reduced optimal outcomes after oral surgery procedures. [5]

Osteoporosis and periodontal disease

Both osteoporosis and periodontitis are diseases characterized by bone resorption. Osteoporosis involves systemic bone loss that leads to decreased bone density and strength and possible bone fracture, whereas periodontitis involves local infection with a host inflammatory response within the periodontium that results in alveolar bone loss and possible tooth loss. Both conditions share multiple risk factors, including age,

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[1] Refer to Episode #41 for additional information on osteoporosis.
[2] The thoracic spine is comprised of twelve vertebral bodies (T1-T12) that make up the mid-region of the spine. The lumbar spine is the lower back that begins below the last thoracic vertebra (T12) and ends at the top of the sacral spine, or sacrum (S1). It consists of five lumbar levels (L1-L5), although it is not unusual to have 6 (L6).
[3] Refer to Episodes #22 and #24 for additional information on vitamin D.

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genetics, smoking, hormonal change, and low calcium and vitamin D levels. Several studies have shown a link between osteoporosis and periodontal disease. [6]

The 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions published several review papers and consensus reports that considered osteoporosis.[4] For example, Albander et al. (2018) reviewed systemic disorders and conditions that affect the periodontal attachment apparatus. Osteoporosis was included in the review of metabolic and endocrine disorders associated with the loss of periodontal tissues. Their review found significant strength of association between osteoporosis and loss of periodontal tissues. [7]

Further, Jepsen et al. (2018) formulated a consensus report from the World Workshop on the periodontal manifestations of systemic diseases and developmental and acquired conditions that affect the periodontal supporting tissues. The authors noted there was conflicting evidence regarding the association between osteoporosis and periodontitis. [8] However, they highlighted the findings by Penoni et al. (2017) that postmenopausal women with osteoporosis or osteopenia[5] exhibited a modest but statistically significant greater loss of periodontal attachment compared with women with normal bone mineral density. [9]

A meta-analysis by Xu et al. (2020) demonstrated osteoporosis was an independent risk factor for periodontitis. Although the study considered possible mechanisms for this link, further research is needed to explain the specific association between periodontitis and osteoporosis. [10]

Gil-Montoya et al. (2021) conducted a prospective study of generally healthy perimenopausal women and found low bone mineral density was significantly linked with periodontal disease in women over 58 years old, independent of tobacco consumption or oral hygiene. [11]

Chou et al. (2021) conducted a population-based cross-sectional study with 7,298 participants aged 35-44 years to investigate the association between osteoporosis and periodontal disease in young adults. A total of 31% had periodontal disease. The prevalence of osteopenia and osteoporosis was 36.2% and 2%, respectively. The data revealed the prevalence of periodontal disease was high in adults with osteoporosis and osteopenia. For example, the prevalence was 40% in adults with osteoporosis and 33% in those with osteopenia. The authors concluded low bone mass increased the risk of periodontal disease in young adults.

However, the authors noted several limitations of the study, such as probing depth as a dependent variable to represent periodontal disease severity. Probing depth readings may not provide an appropriate measurement of periodontal history. Furthermore,

[4] Proceedings of the World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions https://aap.onlinelibrary.wiley.com/toc/19433670/2018/89/S1
[5] Osteopenia – reduced bone mass of lesser severity than osteoporosis.

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because it remains unclear whether osteoporosis causes periodontal disease or vice versa, more studies are needed. [12]

Hong et al. (2021) conducted a cross-sectional study of 9,969 participants with periodontitis and 125,324 control individuals without periodontitis to identify the association between periodontitis and osteoporosis and fractures. The study demonstrated osteoporosis and fractures were associated with periodontitis. The authors suggested medical and oral health clinicians should consider possible interactions between osteoporosis and periodontitis and assess accordingly. [13]

Osteoporosis and dental radiographs

Research by Faber et al. (2004), supported by the National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS), suggested dental radiographs may be beneficial as a screening tool for osteoporosis. The researchers analyzed maxillary and mandibular periapical radiographs of 26 women with osteoporosis and 23 control individuals using three types of computer image analyses: Fourier, strut, and wavelet. The researchers found dental radiographs were very effective in distinguishing individuals with osteoporosis from those with normal bone density. For example, x-rays of individuals with osteoporosis revealed an altered trabecular pattern compared to the control x-rays.

The researchers noted it may be advisable to integrate other factors into this kind of screening, such as age, sex, medication use, familial history of osteoporosis (i.e., risk factors for osteoporosis), and other x-ray measurements. [14]

Verheij et al. (2009) used age[6] and trabecular pattern on dental radiographs of 671 women between 45-71 years to predict the presence of osteoporosis. The authors found that the trabecular pattern predicted the presence of osteoporosis just as well as age did. Combining the trabecular pattern with age increased the sensitivity and specificity. However, only the specificity increase was statistically significant. [15]

Calciolari et al. (2015) conducted a systematic review and meta-analysis to determine if panoramic radiographs could be used to detect early signs of osteopenia/osteoporosis. The authors found mandibular cortical width,[7] panoramic mandibular index,[8] and Klemetti index[9] were useful tools that potentially could be used to screen for low bone

[6] The risk of osteoporosis increases with age in both women and men.
[7] Mandibular cortical width may also be referred to as mental index or mandibular cortical thickness.
[8] Panoramic mandibular index is the ratio between the mandibular cortical width at the mental foramen region and the distance from the lower border to the inferior edge of the mental foramen. [16]
[9] Klemetti index (mandibular cortical index or cortical erosion) describes porosity of the inferior border of the mandible and is related to mandibular bone mineral density. It involves bilateral measurements at the inferior mandibular cortex distal to the mental foramen, based on the following scale:
C1: normal cortex (endosteal margin of the cortical jaw is smooth and continuous on both sides).
C2: slightly or moderately eroded cortex (endosteal margin appears to have resorption cavities and cortical residues consisting of 1 to 3 thin layers).
C3: cortex is severely eroded (cortical bone is clearly porous and there is a significant amount of residue). [35]

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mass density. Limitations of the indices were mainly related to the experience and agreement between different clinicians and the different image quality and magnification of the panoramic radiographs.

Also, the authors noted that based on current evidence, it is not recommended to prescribe panoramic radiographs to detect clients with osteopenia/osteoporosis. Further studies are needed to determine the accuracy of panoramic indices in detecting reduced skeletal bone density. [16]

Tounta (2017) proposed oral health professionals could screen for osteoporosis risk through radiographic indices, trabecular bone pattern changes, and tooth loss from periodontitis.

For example, osteoporosis risk could be screened mainly through dental radiographs, especially panoramic x-rays, utilizing several radiographic indices, such as mandibular cortical width, Klemetti index, gonial index,[10] antigonial index,[11] panoramic mandibular index, and alveolar crest resorption degree.[12] Furthermore, periapical and panoramic x-rays could be assessed for changes in jaw trabecular bone pattern (e.g., fewer number and thinner trabecular plates). However, these indices only suggest low skeletal bone mineral density. As a result, medical history and osteoporosis clinical risk factors should be taken into account before referring for an osteoporosis evaluation.

Also, osteoporosis may accelerate bone loss in periodontitis leading to tooth loss and research has shown there is a positive correlation between periodontitis and skeletal bone mineral density. However, tooth loss is a multifactorial issue and reduced bone mineral density is only a small part of the cause. Consequently, osteoporosis alone cannot be determined as the main reason for tooth loss. Nonetheless, when clinical history and radiographic findings signal low bone mineral density, severe tooth loss is an additional positive marker for referral for osteoporosis assessment. [17]

Other dental indications of low bone density may include mobile teeth, gingival recession, increased pocket depths, and ill-fitting or loose dentures, if other causes are not found. [5]

Implants and osteoporosis

Holanhan et al. (2008) conducted a retrospective chart review of 192 women who were 50 years or older at the time of implant placement to determine if osteoporosis affected the survival rate of osseointegrated dental implants. Out of the 192 women, 49% (94) were not diagnosed with osteopenia or osteoporosis, 29% (57) had osteopenia, 21% (41) had osteoporosis. A total of 37 implant failures were noted out of 646 implants. Those with osteoporosis or osteopenia were not significantly more likely to develop implant failure. However, those who smoked during the time of implant placement were

[10] Gonial Index – thickness of the mandible at the gonial region (angle of the mandible). [17]
[11] Antigonial index – thickness of the mandible at the antigonial region (anterior to angle of the mandible. [17]
[12] Alveolar crest resorption degree is calculated by dividing the total mandibular height by the height from the center of the mental foramen to the inferior mandibular border. [17]

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2.6 times more likely to have implant failure than nonsmokers. The authors concluded osteoporosis or osteopenia was not a contraindication to dental implant therapy. [18]

Tsolaki et al. (2009) completed a literature review in 2007. The authors concluded there were no data to contraindicate dental implants therapy in individuals with osteoporosis. However, surgical technique may need to be adjusted, along with a longer healing period to achieve osseointegration. Further, there was limited data on bisphosphonates and implants so a conclusion could not be drawn. [19]

Ouanounou, Hassanpour, and Glogauer (2016) conducted a narrative review to analyze the effects of systemic medications, including bisphosphonates, on osseointegration. The researchers found insufficient data to suggest implant therapy should be avoided in individuals receiving bisphosphonates. Nevertheless, dental professionals who place implants must to be cognizant of the risk of treating patients who are under oral or intravenous bisphosphonate therapy. [20]

Venkatakrishnan et al. (2017) conducted a literature review on dental implants and osteoporosis. The authors found the success rate of dental implants in individuals with osteoporosis was comparable to individuals without osteoporosis. Further, they found implants can be placed with predictable results in individuals with osteoporosis receiving oral bisphosphonates. Discontinuation of oral bisphosphonates is not mandatory during implant placement.

Considerations for implant therapy for individuals with osteoporosis include appropriate oral self care, good oral health prior to placement, as well as proper treatment planning, assessment of the bone quality (e.g., via CBCT scan), and preparation of the implant site. The authors concluded dental implants were not contraindicated in individuals with osteoporosis. [21]

A review paper from the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions by Schwarz et al. (2018) found no association between osteoporosis/osteopenia and peri-implantitis. [22]

A review from the World Workshop by Hämmerle and Tarnow (2018) highlighted the prolonged medication of high doses of bisphosphonates increases the risk of bone necrosis of the jaws in conjunction with implant therapy. The authors noted there was evidence demonstrating implants in individuals with certain systemic diseases may experience increased rates of hard tissue deficiencies after implant placement. Therefore, it is important to understand the etiology of hard tissue deficiencies and develop strategies to correct these deficiencies to improve clinical outcomes of implant therapy. [23]

Temmerman et al. (2018) conducted a multicenter controlled, clinical trial to verify the long-term outcome of implants in individuals with osteoporosis. After implant placement, oral examination and implant follow-up were performed at 6 months after loading and then yearly up to 5 years. Demographic, clinical, and radiographic data were collected

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and all participants underwent dual x-ray absorptiometry (DXA) to determine bone mineral density.

A total of 148 implants were placed in 48 female participants >60 years of age. Sixty-three implants were placed in 20 participants with osteoporosis and 85 in participants without osteoporosis (control group). At the 5-year follow-up, 117 implants (38 in those with osteoporosis and 79 in the control group) in 37 patients were assessed.

The authors concluded implant therapy was a reliable treatment option for individuals with osteoporosis, with survival rates and marginal bone level alteration values comparable to the control group. [24]

As per the American Dental Association (2019), antiresorptive therapy does not appear to be a contraindication for dental implant placement. However, larger and longer-term studies are needed to determine if implants placed in individuals exposed to antiresorptive agents perform as well as those placed in individuals who have not been exposed to these agents. [25]

Osteonecrosis of the jaw

Osteonecrosis of the jaw (ONJ) is a rare condition that typically presents as an area of exposed bone in the maxilla and/or mandible that does not heal after a period of more than 8 weeks. It usually manifests with pain and purulent discharge, although it may be asymptomatic. Most cases have occurred in individuals with previous or ongoing treatment with intravenous bisphosphonates, usually in high doses for treatment of cancer. In addition to bisphosphonate therapy, most had received dental treatment, such as tooth extraction, dental implants, or treatment for an oral infection, or had local trauma to the oral cavity prior to the development of ONJ. In a few individuals, there was no history of dental treatment or injury prior to the development of ONJ. [26]

ONJ has been observed in individuals receiving high doses of intravenous zoledronic acid or denosumab[13] to reduce the complications associated with cancer that has metastasized to the bones. The risk of ONJ increases with higher doses and with longer treatment duration. ONJ has been estimated to occur in 1% to 15% of individuals with cancer receiving high dose bisphosphonate or denosumab therapy. ONJ associated with use of antiresorptive drugs, such as bisphosphonates or denosumab, is referred to as medication-related osteonecrosis of the jaw (MRONJ). [27]

MRONJ is the preferred term by the American Association of Oral and Maxillofacial Surgeons to describe osteonecrosis of the jaw associated with bisphosphonate, other antiresorptive (denosumab) medications, and antiangiogenic therapies. Other terms previously used to describe medication-related ONJ include: antiresorptive agent-induced osteonecrosis of the jaw (ARONJ) and bisphosphonate-related osteonecrosis of the jaw (BRONJ). [28]

[13] Zoledronic acid is a bisphosphonate; denosumab is a RANK ligand inhibitor (brand name: Prolia). Refer to Episode #41 for additional information.

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To treat osteoporosis and osteopenia, much lower doses of denosumab or bisphosphonates are used. The risk of MRONJ with low dose bisphosphonate or denosumab osteoporosis treatment is approximately between 1 in 10,000 and 1 in 100,000 per year of therapy and the risk appears to be only minimally increased, if at all, compared with ONJ risk in the general population without history of osteoporosis therapy.

Also, ONJ can occur in the general population without a history of bisphosphonate, denosumab, or other drugs, and usually heals within 12 weeks. ONJ can also develop in people who have risk factors for ONJ, such as poor blood supply to the bone cells in the jaw. It can also develop after major dental surgery involving bone cutting; chemotherapy; steroid therapy (e.g., prednisone); and from poor oral health and diabetes.

Note: ONJ does not affect the temporomandibular joint (TMJ), and TMJ pain or discomfort is not related to ONJ or to bisphosphonate or denosumab therapy. [27] [29]

Medications related to MRONJ

Antiresorptive medications

Intravenous bisphosphonates are used to manage cancer-related conditions, such as:

- Hypercalcemia of malignancy, a common condition typically found in individuals with advanced stage cancers. Hypercalcemia is an increase in the serum calcium level above the upper limit of normal).
- Skeletal-related events associated with bone metastases from solid tumours such as breast, prostate, and lung cancers.
- Management of lytic lesions from multiple myeloma. Lytic lesions are areas of bone damage, which appear as holes on an x-ray.

Oral bisphosphonates are used to treat osteoporosis and osteopenia. They are also used to treat less common conditions, such as Paget's disease of bone and osteogenesis imperfecta.

The receptor activator of nuclear factor κ B ligand (RANKL) inhibitor (i.e., denosumab) inhibits osteoclast development and activation. It is administered subcutaneously every 6 months to:

- Decrease in the risk of vertebral, nonvertebral, and hip fractures in individuals with osteoporosis.
- Increase bone mass in men with nonmetastatic prostate cancer on androgen deprivation therapy with high fracture risk.
- Increase bone mass in women with nonmetastatic breast cancer on adjuvant aromatase inhibitor therapy with low bone mass and high fracture risk.
- Increase bone mass for the treatment and prevention of glucocorticoid-induced osteoporosis in women and men with high fracture risk.

In contrast to bisphosphonates, RANKL inhibitors do not bind to bone and their effects on bone remodelling are mostly diminished within 6 months of treatment cessation. [28] [30]

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Antiangiogenic medications

Antiangiogenic medications block angiogenesis (growth of new blood vessels) and are used to treat some types of cancers, such as gastrointestinal tumours, renal cell carcinomas, and neuroendocrine tumours. [28]

Pathophysiology of MRONJ

The pathophysiology of MRONJ is not fully understood even though cases of MRONJ were first described in 2003. [28] [31] Proposed hypotheses to explain the localization of MRONJ solely to the jaws include altered bone remodelling or over-suppression of bone resorption, angiogenesis inhibition, continual microtrauma, innate or acquired immune suppression, vitamin D deficiency, soft tissue bisphosphonate toxicity, and inflammation or infection. [28]

Altered bone remodelling

An increased remodelling rate in the jaws may explain the predisposition of osteonecrosis to occur in the jaws compared with other bones of the skeleton. [28]

Angiogenesis inhibition

Osteonecrosis or avascular necrosis is the death of bone tissue due to a lack or interruption of blood supply. In vitro experiments and studies in patients with cancer have shown a decrease in angiogenesis with zoledronic acid. Therefore, angiogenesis inhibition has been proposed in ONJ pathophysiology. However, inhibition of angiogenesis has not been reported with denosumab. [28]

Immune dysfunction

The immune system is closely associated with bone loss and bone regeneration. Studies have shown mucosal ulceration, delayed healing, exposed bone, and histologic necrosis and inflammation when bisphosphonates and chemotherapy were administered in rodents undergoing extractions.

Gamma delta T lymphocytes (innate immune cells) are important in bone regeneration. These T cells are greatly reduced in individuals with osteoporosis being treated with bisphosphonates, denoting a connection between gamma delta T cell deficiency and MRONJ.

Neutrophils help to promote wound healing. Nitrogen-containing bisphosphonates (e.g., alendronate, risedronate, zoledronic acid, pamidronate disodium[14]) alter neutrophil defence capabilities and impair normal wound healing, which may possibly play a role in the pathogenesis of MRONJ. [28] [32]

[14] Pamidronate disodium is administered intravenously to treat conditions such as lytic bone metastases, multiple myeloma, and symptomatic Paget's disease of the bone. [34]

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Soft tissue bisphosphonate toxicity

Although bisphosphonates primarily target osteoclasts and bone tissue, soft tissue toxicity has been reported. Oral epithelial cells have exhibited increased apoptosis[15] and decreased proliferation after exposure to bisphosphonates in vitro. Mucosal ulcerations may be the initial pathologic event that occurs in MRONJ. There may also be reduced oral mucosal soft tissue repair and wound healing in relation to bisphosphonate exposure. In contrast to bisphosphonates, soft tissue toxicity has not been reported with denosumab. [28] [32]

Inflammation and Infection

Oral disease or bacterial infection have been implicated in ONJ pathogenesis. Tooth extraction has been reported in cases of ONJ; correspondingly, these extracted teeth commonly had existing periodontal or periapical disease. Also, the presence of bacteria, especially *Actinomyces* species, have been noted in biopsied specimens of necrotic bone removed from patients with ONJ. [28] [32]

Many hypotheses exist to explain MRONJ; however, animal and human studies demonstrate the disease is probably multifactorial. More research is needed to uncover the exact mechanisms of MRONJ. [28]

MRONJ case definition

MRONJ may be considered if an individual has all three of the following criteria:

- Current or previous treatment with antiresorptive or antiangiogenic agents;
- Exposed bone or bone that can be probed through an intraoral or extraoral fistula in the maxillofacial region that has persisted for longer than 8 weeks; and
- No history of radiation therapy to the jaws or metastatic disease to the jaws. [28] [33]

Signs and symptoms of ONJ

Signs and symptoms may include:

- Sudden presence of intraoral pain and presence of roughness (i.e., exposed sharp necrotic bone) that irritates adjacent soft tissues, with the exposed bone being asymptomatic. Initially, individuals may be asymptomatic or have nonspecific jaw pain (i.e., no tooth-related causes can be identified).
- Loose teeth not associated with periodontal disease.
- Periapical or periodontal fistula not associated with pulpal necrosis due to caries.
- Pain, swelling, difficulty chewing, bad taste, and fetid odour from secondary infection.
- Paresthesia, altered neurosensory functions, and extraoral fistulas are late signs indicating disease progression, as with osteomyelitis.
- Exposed, nonhealing bone after dental surgery (e.g., extraction, periodontal, or endodontic surgery) or trauma.

[15] Apoptosis, also called programmed cell death, is one method the body uses to get rid of unneeded or abnormal cells.

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- Purulent discharge without bone exposure after tooth extraction or other oral surgery. [26]

ONJ diagnosis
Diagnosis of ONJ is based on a detailed medical and dental history and a clinical examination (extra- and intraoral). Medical history should include questions on drug history (e.g., drug names, route of administration, duration, reason for treatment), history of radiation therapy, concurrent illnesses and conditions, smoking, etc.

Extraoral examination would include palpation of head and neck lymph nodes and examining for extraoral draining sinuses. Intraoral examination would include examining all oral mucosal surfaces and exposed bone and assessing for purulent drainage (e.g., from small pinpoint areas) and signs of inflammation.

Radiographic examination may also be necessary to detect extent of bone destruction and location of sequestra (pieces of dead bone tissue). [25] [26] [27]

Differential diagnosis of MRONJ
The differential diagnosis of MRONJ includes conditions such as alveolar osteitis, sinusitis, gingivitis, periodontitis, osteomyelitis, caries, periapical pathology, odontalgia, atypical neuralgias, fibro-osseous lesions, sarcoma, chronic sclerosing osteomyelitis, and temporomandibular joint disorders. Note: exposed bone or sequestra can occur in individuals not exposed to antiresorptive or antiangiogenic agents. [25] [26] [28]

Risk factors for MRONJ
The following are risk factors for MRONJ:
- >65 years
- Pre-existing inflammatory dental disease, such as periodontal disease or periapical pathology
- Females have a higher prevalence likely due to underlying disease (i.e., osteoporosis, breast cancer) that require prescribed medications
- Poor oral hygiene
- Dentoalveolar surgery (e.g., tooth extraction, implant placement, endodontic or periodontal procedures that require exposure and manipulation of the bone)
- High dose and/or prolonged use of antiresorptive agents (>2 years)
- Tobacco use
- Cancer (e.g., lung, breast, and prostate cancer; multiple myeloma)
- Chemotherapy, corticosteroid, or antiangiogenic therapy
- Diabetes
- Wearing dentures (possibly due to mucosal trauma by ill-fitting dentures) [25] [28]

Treatment of ONJ
Treatment of ONJ usually includes client reassurance and education (e.g., maintaining good oral hygiene, staying hydrated, good nutrition low in sugar), pain control, treatment of secondary infection with antibiotics and antimicrobial mouth rinses; and prevention of

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lesion extension or new areas of necrosis. In certain circumstances, surgical debridement of the dead bone is required. [28] [27] [29]

For individuals who are diagnosed with MRONJ while being treated with bone-modifying agents (e.g., high dose intravenous bisphosphonate or denosumab therapy in individuals with cancer), there is insufficient evidence to support or refute the discontinuation of these medications. Bone-modifying agents may be deferred at the discretion of the treating physician, in conjunction with discussion with the patient and the oral health provider. [33]

Prevention of MRONJ

The prevention on MRONJ requires a multidisciplinary approach to address modifiable risk factors for MRONJ as early as possible, which may include:

- Consultation with an oral health professional when it is determined an individual needs antiresorptive or antiangiogenic therapy in order to initiate early oral health screening and treatment to decrease incidence of ONJ and to attain optimum oral health.
- Education on importance of maintaining good oral hygiene and regular oral health care, with specific instructions to report any pain, swelling, or exposed bone.
- Initiation of smoking cessation.
- Correction of ill-fitting dentures.
- Modification of lifestyle and/or medication to address uncontrolled diabetes mellitus.
- Coordination of oral health care with attending oncologist prior to initiation of bone-modifying agents. This would include a detailed dental examination with x-rays, as indicated; development of a treatment plan; and initiation of oral health treatment prior to bone-modifying treatment (e.g., extraction of non-restorable teeth and teeth with poor prognosis, other necessary elective dentoalveolar surgery). Dental prophylaxis, caries control, and conservative restorative dentistry are critical to maintaining functionally sound teeth and should be continued indefinitely.

The recommendations for individuals with osteoporosis receiving low dose bisphosphonate or denosumab therapy are similar to those which apply to all individuals. They include maintenance of good oral hygiene and visiting an oral health professional on a regular basis. If oral surgery is needed, it is ideal to have the surgery completed before starting low dose oral or yearly intravenous bisphosphonate therapy or denosumab therapy if possible. [27] [28] [33]

Take home messages

Overall, oral health professionals have the opportunity to refer their clients to appropriate medical providers when low bone density is identified on dental x-rays taken in the course of dental examination or treatment. Considering many people see their oral health provider more regularly than they see their medical provider, oral health professionals can facilitate early identification of osteoporosis for prompt interventions to help avoid the complications associated with osteoporosis. [5]

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Osteoporosis is generally not a contraindication for implant therapy. However, the type of osteoporosis medication and the timing of its administration has to be taken into consideration for implant placement. A referral to a periodontist or an oral surgeon should be considered.

Osteoporosis medication is prescribed to help prevent a fracture. Individuals are much more at risk of suffering a fracture than developing MRONJ. The benefits of osteoporosis medication outweigh the possible risks from these medications. Individuals should always discuss with their physician prior to stopping or changing their osteoporosis medication. [29]

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