

Possible relationship between Osteoporosis and Periodontal Disease

Ivan M*

Department of Dentistry, Medical faculty, University of Nis, Serbia

***Corresponding author:** Minic Ivan, Department of Dentistry, Medical faculty, University of Nis, Nikole Tesle 63/8, 18000 Nis, Serbia. Tel: +381643004883; E-mail: ivanminic32@gmail.com

Received: May 18, 2020

Published: June 19, 2020

Abstract

Osteoporosis is a systemic disorder characterized by decreased bone mass and micro architectural deterioration of bone tissue, leading to bone fragility and increased susceptibility to fractures of the hip, spine, and wrist. Periodontal diseases are disease processes involving the periodontium, a term used to describe the supportive apparatus surrounding a tooth, which includes the gums (gingiva), alveolar bone, cementum and periodontal ligament. Gingivitis is the mildest form of periodontal disease and can be found in up to 90% of the population. A possible pathway in which systemic bone loss may lead to more severe periodontal destruction is that the reduced bone mineral density (BMD), caused by osteoporosis in the alveolar bone, may facilitate local bone resorption caused by the periodontal disease. Another possibility is that systemic factors of bone remodeling could modify local tissue response to periodontal infection. Accordingly, individuals with systemic bone loss who have periodontitis may react differently to the increased production of cytokines and inflammatory mediators, therefore presenting more severe periodontal disease.

Key words: Osteoporosis; Periodontal disease; Mineral bone density

Introduction

Osteoporosis is a systemic disorder characterized by decreased bone mass and micro architectural deterioration of bone tissue, leading to bone fragility and increased susceptibility to fractures of the hip, spine, and wrist. Due to its important prevalence worldwide, osteoporosis is considered a serious public health concern associated with disability and pain [1].

The pathophysiology of osteoporosis is an imbalance between bone resorption and bone formation. In osteoporosis, bone resorption takes place to a greater extent than bone formation, so a negative balance occurs with a net loss of bone and an accompanying increasing risk of fractures, resulting in deformity and chronic pain. Nociceptive pain is considered to be chronic when it has been present for at least 3 months. The imbalance between bone formation and bone resorption might occur as a result of one or a combination of the following factors:

- Increased bone resorption within a remodeling unit.
- Decreased bone formation within a remodeling unit (incomplete coupling) [2]

Many of the risk factors for osteoporosis are environmental and therefore, are preventable. Established risk factors include older age; female gender; postmenopause; Caucasian or Asian race; a low body mass index; cigarette use; alcoholism; inadequate calcium and vitamin D intakes; physical inactivity; taking medications such as glucocorticoids and anticonvulsants; and anorexia nervosa [3,4]. Although osteoporosis and osteopenia can affect people of all ages, they occur most often in middle-aged and elderly people [5].

Osteoporosis is categorized into primary or secondary. Primary

osteoporosis is associated with increased age and/or decreased sex hormones. Secondary osteoporosis implies an underlying cause such as usage of glucocorticoids, systemic diseases affecting bone turnover, or low calcium intake [6, 7]. The gold standard for the diagnosis of osteoporosis is the measurement of bone mineral density (BMD) by dual energy x-ray absorptiometry (DXA).

Periodontal diseases are disease processes involving the periodontium, a term used to describe the supportive apparatus surrounding a tooth, which includes the gums (gingiva), alveolar bone, cementum and periodontal ligament. Gingivitis is the mildest form of periodontal disease and can be found in up to 90% of the population [8].

When periodontal disease affects the bone and supporting tissue, it is termed periodontitis and is characterized by the formation of pockets or spaces between the tooth and gums. This may progress and cause chronic periodontal destruction leading to loosening or loss of teeth. The dynamics of the disease are such that the individual can experience episodes of rapid periodontal disease activity in a relatively short period of time, followed by periods of remission.

Though the majority of adults are affected by gingivitis, gingivitis fortunately does not always develop into periodontal disease. Progression of gum disease is influenced by a number of factors which include oral hygiene and genetic predisposition. One of the challenges for early detection of periodontal disease is its "silent" nature – the disease does not cause pain and can progress unnoticed.

In its early stages, bleeding gums during tooth brushing may be

the only sign; as the disease advances and the gums deteriorate, the bleeding may stop and there may be no further obvious sign until the teeth start to feel loose. In most cases, periodontal disease responds to treatment and although the destruction is largely irreversible its progression can be halted [9, 10].

Since both osteoporosis and periodontal diseases are bone destructive diseases, it has been hypothesized that osteoporosis could be a risk factor for the progression of periodontal disease. A possible pathway in which systemic bone loss may lead to more severe periodontal destruction is that the reduced bone mineral density (BMD), caused by osteoporosis in the alveolar bone, may facilitate local bone resorption caused by the periodontal disease. Another possibility is that systemic factors of bone remodeling could modify local tissue response to periodontal infection. Accordingly, individuals with systemic bone loss who have periodontitis may react differently to the increased production of cytokines and inflammatory mediators, therefore presenting more severe periodontal disease [11, 12].

Osteoporosis is a systemic bone resorption disease affecting mostly precancels bone whereas periodontal disease involves local infection of the periodontium that first attacks the cortical bone and results in dimensional changes of the alveolar ridge. There is a possibility that the osteoporotic changes of the alveolar bone directly contribute to premature loss of teeth through noninfectious mechanism.

Menopausal effect of women, such as estrogen level reduction that will effect calcium absorption and result in deficiencies are major risk factor for osteoporosis. Reduce level of estrogen also induce osteocyte apoptosis which disrupt the homeostasis of bone. Hormonal changes also effect systemic bone homeostasis and inflammatory responses [13].

Studies assessing the association between osteoporosis and periodontal disease differ widely in their methodology, techniques for periodontal examination and bone mineral density (BMD) assess. Despite such heterogeneity in study designs, most of them showed an association of low systemic BMD with periodontal disease and tooth loss.

Periodontal disease is more common in women with osteoporosis and vitamin D deficiency. Supplementation of calcium and sufficient levels of vitamin D may lead to an increase in mandibular bone mass for postmenopausal women [14].

Antiresorptive therapy for low bone mass is a protective bone therapy. Bisphosphonates (BP) and denosumab are commonly prescribed drugs for managing osteoporosis. Bisphosphonates are highly concentrated in the jaws and have shown to act as blockers of tooth-supporting alveolar bone destruction. Postmenopausal women with low BMD who received BP therapy showed better periodontal status, alveolar bone and decreased tooth mobility, than those who did not have treatment [15, 16]. There is a slight risk of developing osteonecrosis of the jaw (ONJ) in individuals who use antiresorptive drugs for osteoporosis treatment. Despite of the low incidence of ONJ, it is important for patients with osteoporosis to be aware of this condition, and the preventive measures to avoid this complication. Dental care prior to starting antiresorptive therapy is recommended.

In routine dental practice clinicians come across many patients who are receiving bisphosphonates as part of their therapy. Most commonly postmenopausal female patient who are receiving bisphosphonates as a treatment for osteoporosis which is very common for their age group, are encountered. These pa-

tients are at increased risk of developing osteonecrosis in jaw when any dental treatment is done or patient is suffering from dental disease. So it becomes important to identify such patients and follow a suggested protocol to avoid complications.

Conclusion

Health care professionals and patients should be aware of the influence of systemic bone condition on the periodontal status. Managing osteoporosis, maintaining good oral hygiene, and complying with dental visits for biofilm control are especially important measures for avoiding periodontitis in susceptible individuals.

References

1. Lane NE. Epidemiology, etiology, and diagnosis of osteoporosis. *Am J Obstet Gynecol.* 2006;194(2 Suppl);S3-11.
2. Grinspoon S, Thomas E, Pitts S, Gross E, Mickley D, Miller K, et al. Prevalence and predictive factors for regional osteopenia in women with anorexia nervosa. *J Ann Intern Med.* 2000;133(10);790-4.
3. Takedachi M, Murakami S. Present status of periodontal regeneration - FGF-2 and Teriparatide. *Clin Calcium.* 2012;22(1);99-104.
4. Wright N, Looker A, Saag K, et al. The recent prevalence of osteoporosis and low bone mass in the United States based on bone mineral density at the femoral neck or lumbar spine. *J Bone Miner Res.* 2014;29(11);2520-2526.
5. Cosman F, de Beur S, LeBoff M, et al. Clinician's guide to prevention and treatment of osteoporosis. *Osteoporos Int.* 2014;25(10);2359-2381. Erratum in: *Osteoporos Int.* 2015;26(7);2045-2047.
6. Erdoğan O, Shafer DM, Taxel P, Freilich MA. A review of the association between osteoporosis and alveolar ridge augmentation. *Oral Surg Oral Med Oral Pathol Oral Radiol.* 2007;104(6);738-44.
7. Dawson-Hughes B, Heaney RP, Holick MF, Lips P, Meunier PJ, Vieth R. Estimates of optimal vitamin D status. *Osteoporos Int.* 2005;16(7);713-6.
8. Irfan UM, Dawson DV, Bissada NF. Epidemiology of periodontal disease: a review and clinical perspectives. *J Int Acad Periodontol.* 2001; 3(1);14-21.
9. Nakashima K, Roehric N, Cimasoni G. Osteocalcin, prostaglandin E-2 and alkaline phosphatase in gingival cervical fluid: their relations to periodontal status. *J Clin Periodontol.* 1994;21(5);327-333.
10. Gapski R, Barr JL, Sarment DP, et al. Effect of systemic matrix metalloproteinase inhibition on periodontal wound repair: a proof of concept trial. *J Periodontol.* 2004;75(3);441-452.
11. Vitte C, Fleisch H, Guenther HL. Bisphosphonates induce osteoblasts to secrete an inhibitor of osteoclast-mediated resorption. *Endocrinology.* 1996;137;2324-2333.
12. Zamboni G, Colucci S, Cantatore F, Grano M. Response of Human Osteoblasts to Polymethylmetacrylate In Vitro. *Calcif Tissue Int.* 1998;62;362-364.
13. Jeffcoat M. The association between osteoporosis and oral bone loss. *J Periodontol.* 2005;76; 2125-2132.
14. Sandeep Kalra, Veena Jain. Dental complications and management of patients on bisphosphonate therapy: A review article. *J Oral Biol Craniofac Res.* 2013;3;25-30.
15. Fugazzotto P, Lightfoot WS, Jaffin R, Kumar A. Implant placement with or without simultaneous tooth extraction in patients taking oral bisphosphonates: Postoperative healing, early follow-up, and the incidence of complications in two private practices. *J Periodontol.* 2008;78;1664-1669.
16. Bell BM, Bell RE. Oral bisphosphonates and dental implants: a retrospective study. *J Oral Maxillofac Surg.* 2008; 66;1022-1024.