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Osteoporosis, periodontal disease and aging

Abstract

Periodontitis and osteoporosis are multifactorial bone disorders frequently considered separately but they could have a reciprocal influence and require a holistic treatment. Tooth loss as a consequence of a gradual alveolar bone resorption and higher fracture risk increase with aging. This oral and skeletal decline contribute to systemic deterioration and frailty. Currently, novel approaches are needed to complement and improve conventional periodontal and osteoporosis therapies. The aim of this review is to highlight both the potential interaction between periodontal disease and osteoporosis and the emerging approaches to treat these bone disorders.

Keywords: osteoporosis, periodontal disease, aging, bone loss

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Introduction

Periodontal disease and osteoporosis are chronic inflammatory disorders resulting in a gradual alveolar bone loss and higher risk of fracture, respectively. A difference between both diseases is that periodontitis is initiated by a localized bacterial infection. Osteoporosis, in contrast, could be attributed to a "sterile inflammation" (indicating an absence of detectable pathogens) as a consequence of cellular senescence.[1] However, in both conditions cells of the immune system, such as T cells, B cells, macrophages or dendritic cells become activated and produce inflammatory cytokines.[2] These secreted pro-inflammatory signals, such as IL-1 and TNF-α not only promote osteoclast differentiation, but also oppose BMP-2 induced osteoprogenitor cells differentiation into osteoblasts.[3] This immune-inflammatory response favors bone loss by both an aberrant increased bone resorption and a diminished osteoblast bone formation.

Periodontitis and osteoporosis are also influenced by age-related endocrine disorders that directly or indirectly affect bone homeostasis. Osteoporosis mostly affects people over 50 years of age and one of the major contributing factors is the drop in estrogen during menopause in women.[4] This drop in estrogen has not been found to have a major direct effect on osteoclast activity but its withdrawal enhances the production of pro-inflammatory cytokines and this promotes osteoclast resorption indirectly.[4] In addition, it is also known that the levels of melatonin decrease with aging. This age-related decrease in melatonin levels coincides with menopause and is also linked to osteoporosis.[5] Interestingly, patients with chronic and aggressive periodontitis have lower levels of melatonin both in gingival crevicular fluid and saliva than healthy subjects.[6] Moreover, melatonin could protect periodontal tissues against inflammatory injury by its antioxidant, anti-inflammatory and immune-modulatory properties.[7] This hormonal withdrawal suggests a decline of an endocrine regulatory system controlling the immuno-inflammatory response in bone and an increased resorption with aging.

Mechanical removal of calcified plaque affecting periodontal tissues is required to regenerate such tissues. Furthermore, because gingival and mucosal tissue thinning often occurs, the use of an extrasoft toothbrush, mouth rinses with a low alcohol concentration and dentifrices with minimally abrasive particles should be considered.[8] However, the standard treatment may not be sufficient or definitive therapy that results in clinical improvement, requiring a more sophisticated biological approach.[9] Currently, novel therapeutic strategies to regenerate periodontal tissues have been used. These include the use of growth factors[10] microRNAs[11] active ingredients derived from natural products[12] and tissue engineered constructs.[13]

People with osteoporosis and low bone mineral density also present an important disruption in the clinical periodontal attachment level and an excessive gingival recession. This relationship suggests a higher predisposition for periodontal disease.[14,15] It has been recognized that osteoporosis also influences the treatment and recurrence of periodontitis.[16] Estrogen treatment has been commonly used to prevent bone loss in osteoporotic women and this has been associated with a decrease in the risk of tooth loss.[17] On the other hand, periodontitis has been associated with and recognized as a potential risk factor for rheumatoid arthritis, diabetes, cardiovascular disease, Alzheimer's disease and certain eye diseases.[18,19] The dissemination of pathogens and toxins from periodontal lesions is increasingly recognized as a link between periodontal disease and these systemic diseases.[20] Conversely, diverse results have been published considering this relationship between osteoporosis and periodontitis. Some of these have reported that non significant differences exist when healthy and osteoporotic women are compared.[21,22] Lack of standarization in the definition and measuring of osteoporosis and periodontitis could account for these different results.[21–23] Therefore, osteoporosis and periodontitis could be risk factors for each other and have a mutual impact that requires concomitant management.[24]

Conventional pharmacological osteoporosis therapies exert either an antiresorptive or an anabolic effect independently although they could be combined. Currently, new approaches are emerging promoting integrated therapies rather than those considering two separated elements of the same pathological condition. Already in the early 2000s experimental studies demonstrated that melatonin at pharmacological doses increases bone mass by suppressing resorption[25] and promote osteoblast differentiation and bone formation.[26] More recently, a causal role for senescent cells in bone loss with aging has been reported.[27] Targeting and eliminating these senescent cells has both anti-resorptive and anabolic effects on bone. Concurrently, this approach could prevent multiple aging comorbidities by using senolytic drugs.[27]

Conclusion

Abnormal immuno-inflammatory responses promote bone loss with aging. It has been recognized that osteoporosis could affect the periodontal condition. Similarly, increasing evidence suggests that chronic bacterial infection from periodontitis (and its products released into the blood stream) also could be associated with several systemic diseases. Periodontitis and osteoporosis frequently are considered separately but they could have a reciprocal influence and require a holistic treatment. More sophisticated approaches are emerging to treat bone loss and could circumvent the current limitations and drawbacks of conventional treatments. A better understanding of the potential relationship between these bone related disorders could contribute to the improvement of such novel strategies.

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Conflict of interest

The authors have no conflict of interest.

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