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Review Article

Bone loss & Periodontitis- A Review

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ABSTRACT:
Periodontal diseases range from the relatively benign gingivitis to chronic and aggressive forms of the disease. Bone resorption is a basic physiologic process that is central to the understanding of many key pathologies, with its most common oral manifestation seen as the alveolar bone destruction in periodontitis. This review described the mechanisms of bone resorption as related to periodontal disease, at the molecular and cellular levels.
Key words: Bone loss, Periodontitis, Osteoclasts

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Introduction

Bone resorption is a basic physiologic process that is central to the understanding of many key pathologies, with its most common oral manifestation seen as the alveolar bone destruction in periodontitis. Periodontal diseases range from the relatively benign gingivitis to chronic and aggressive forms of the disease. There is inconsistent use of criteria to define the different forms of periodontitis in literature.[1] Throughout the 20th century, chronic periodontitis was considered as an inflammatory disease associated with local irritants and dental plaque on tooth surfaces. This concept prevails today. What is now known as "generalized aggressive periodontitis" was not clearly described until the latter part of 20th century. The objective of this review is to update the current understanding of the chronic and aggressive forms of periodontitis and their implications for the diagnosis and treatment.[2] This review aims to describe the prevailing understanding of mechanisms of bone resorption as related to periodontal disease, at the molecular and cellular levels.

Bone Homeostasis and Maintenance

Bone is a remarkably dynamic and active tissue, undergoing constant renewal in response to mechanical, nutritional, and hormonal influences. A balance between the coupled processes of bone resorption by osteoclasts and bone formation by osteoblasts is required in a healthy adult. Under physiologic conditions, these processes are very carefully regulated by systemic hormones and local factors and orchestrated by osteocytes and bone lining cells which fine-tune interstitial fluid and plasma calcium levels. Thus, bone resorption plays a major role in the homeostasis of skeletal and serum calcium levels, and the regulated coupling of resorption to new bone formation by osteoblasts is required for proper growth, remodelling, and skeletal maintenance. The overall quality and quantity of bone will be affected by any factors that influence either of these processes or perturb this balance.[3]

Bone Cells

Preosteoblasts, osteoblasts, osteocytes, and bone lining cells all arise from the osteogenic line of cells, which, in turn, arise from primitive mesenchymal cells in bone marrow stroma and from pericytes adjacent to connective tissue blood vessels. Their differentiation requires activation of the Osf2/Cbfa gene, which activates expression of osteocalcin, bone sialoprotein (BSP), osteopontin (OPN), and collagen synthesis, and is followed by stimulation from bone morphogenetic protein- (BMP-) 2 and transforming growth factor beta (TGF-β). Besides their primary

role in bone formation, osteoblasts express chemokines, prostaglandins, and growth factors (e.g., BMPs, TGF-β, colony-stimulating factor- (CSF-) 1, granulocyte colony-stimulating factor (GCSF), basic fibroblast growth factor (basic FGF), and insulin like growth factor (IGF)) with autocrine, self-regulatory, and/or paracrine activity that regulate osteogenic as well as osteoclastic cells. Osteoblastic cells have a major influence on the environmental responsiveness of osteoclasts through localisation, induction, stimulation, and inhibition of resorption.[4]

Degradation of the Mineral and Organic Matrix

Osteoclasts resorb bone in resorption lacunae by generating a pH gradient between the cell and bone surface, favouring the mineral-dissolving action of the osteoclast proteinases. Carbonic anhydrase (CA) II is the main cytoplasmic source of protons for the acidification of the lacuna. This hydrates carbon dioxide to carbonic acid, which ionizes into carbonate and hydrogen ions.[5] A vacuolar-type proton pump, V-ATPase, transports the protons generated by CAII into intracellular vesicles. These are then transported and fused to the RB membrane, releasing their proton content to the lacuna. Acidification is subsequently completed by passive potential driven chloride transport. The chloride channel of the ruffled border is identified as ClC-7, and it is transported along with the proton pump to the RB via endosomes.[6]

Local Mediators of Bone Resorption

Local formation of osteoclasts and their stimulation are required for alveolar bone loss. It has been shown that multiple mediators, such as IL-1, IL-6, IL-11, IL-17, TNF-α, TNF-beta, TGF-β, kinins, and thrombin, can stimulate bone resorption. Bone resorption is also directly regulated locally by ionized calcium generated as a result of osteoclastic resorption, and new evidence indicates that endothelial cells may also play a part via mediators including nitric oxide and endothelin.[7]

Immunopathogenesis of Periodontal Disease

In chronic periodontal disease, biologically active substances within bacterial plaque induce a local inflammatory response in the gingival soft tissues and periodontium. The resultant influx of inflammatory cells produces a host of cytokines, for example, PGE2, IL-1, and RANK-L, that promote resorption through osteoclasts, the primary bone resorbing cell. Thus, in pathologic inflammatory conditions, stimulatory inflammatory cell products initiate osteoclast activity and disturb the fine balance between protective and destructive processes.[8]

Role of Bacteria

Similar to other polymicrobial diseases, periodontitis is now characterized as a microbial-shift disease owing to a well characterized change in the microorganisms that are present (from mostly Gram-positive to mostly Gram-negative species) during the transition from periodontal health to periodontal disease. The pathogenic processes of periodontal diseases are primarily due to the host response, which propagates the destruction initiated by microbes. Harmful pathogenic products and enzymes such as hyaluronidases, collagenases, and proteases break down extracellular matrix components in order to produce nutrients for their growth.[9]

The Innate Immune Response, TLRs, and PAMPs

The host response against periodontopathic bacteria consists of innate and acquired immunity. The innate response meets the challenge of discriminating among large numbers of pathogens through recognition of conserved evolutionary molecular motifs called PAMPs (pathogen associated molecular patterns), which are expressed on pathogens but not by the host. The recently discovered Toll-like receptors (TLRs) are pattern-recognition receptors with key roles in detecting microbes and initiating inflammatory and host defense responses.[10]

Risk Factors of Periodontal Disease

Modifiable Risk Factors

Microorganisms and Periodontal Disease

The oral bacterial microbiome includes over 700 different phylotypes, with approximately 400 species found in subgingival plaque. The subgingival microflora in periodontitis can harbor hundreds of bacterial species but only a small number has been associated with the progression of disease and considered etiologically important. Subgingival plaque from deepened periodontal pockets is dominated by gram-negative anaerobic rods and spirochetes. Strong evidence has implicated Porphyromonas gingivalis and Aggregatibacter actinomycetemcomitans to the pathogenesis of adult periodontitis. In addition, Bacteroides forsythus, Prevotella intermedia, Peptostreptococcus micros, and Fusobacterium nucleatum have been strongly linked with the progression of adult periodontitis.[11]

Tobacco Smoking

There is accumulating evidence for a higher level of periodontal disease among smokers. Tobacco smoking exerts a substantial destructive effect on the periodontal tissues and increases the rate of periodontal disease progression. Risk factors including tobacco smoking modify the host response to the challenge of bacteria in dental plaque.

Diabetes Mellitus

One of the important oral signs of diabetes is gingivitis and periodontitis. Patients with undiagnosed or poorly controlled diabetes mellitus type 1 or type 2 are at higher risk for periodontal disease. There are many studies that demonstrate an association between

diabetes and an increased susceptibility to oral infections including periodontal disease.[12]

Obesity

Obesity has been reported to be an important risk factor for periodontal disease. Several explanations for the association between obesity and periodontal disease in younger adults have been provided. Younger people may have different dietary patterns than older study participants.

Nonmodifiable Risk Factors

Osteoporosis

Many of the studies conducted to date suggest there is a relationship between skeletal osteoporosis and bone loss to the extent that postmenopausal osteoporosis may result in dental osteopenia involving the jaws, and particularly the mandible. Osteoporosis was significantly associated with severe alveolar crestal bone loss and the prevalence of periodontitis cases in postmenopausal women.[13]

Hematological Disorders

Hemorrhagic gingival overgrowth with or without necrosis is a common early manifestation of acute leukemia. Patients with chronic leukemia may experience similar but less severe periodontal changes.

Chronic and aggressive periodontitis share many clinical features. They are both complex infections that occur in susceptible hosts and are caused by biofilms with indigenous oral microbiota on tooth surfaces. The host response to the biofilms are primarily responsible for the loss of periodontal attachment and alveolar bone supporting the teeth. The eventual outcome of these untreated diseases is tooth loss.[14]

In both generalized forms of chronic and aggressive periodontitis, the affected individuals have no known medical or general health conditions that might contribute to the development of their periodontitis. If the systemic disease profoundly impairs the ability of the host to cope with the bacterial challenge associated with periodontitis, the term "periodontitis as a manifestation of systemic disease" should only be used instead of and not "chronic or aggressive" according to the 1999 classification.

Although similar in many respects, chronic and aggressive forms of periodontitis have a number of significant clinical differences including: (i) age of onset (i.e., detection), (ii) rates of progression, (iii) patterns of destruction, (iv) clinical signs of inflammation, and (v) relative abundance of plaque and calculus. Indeed, combinations of these clinical differences are the primary basis for placing affected individuals into one of the three major categories of periodontitis (i.e., chronic periodontitis, localized aggressive periodontitis, and generalized aggressive periodontitis).[15]

Age of onset

The age of onset is an important feature that has traditionally been used to help place patients either in the aggressive or in the chronic periodontitis category. But there is considerable uncertainty about setting arbitrary upper age limits for certain forms of periodontitis. Given similar amounts of periodontal damage (i.e., probing depths, attachment loss, alveolar bone resorption), people with aggressive periodontitis are significantly younger than individuals with chronic periodontitis.[16]

It is pointless for clinicians to argue about what is the best cut-off age to distinguish between aggressive and chronic periodontitis as the treatment of a 30-year-old patient with severe periodontitis will probably be the same regardless of what the disease is called. However, for research purposes and depending on the research question, it may well be reasonable to include age limits in case definitions to reduce heterogeneity within study groups and to ensure that there is no overlap in disease categories.[17]

The rate at which loss of supporting periodontal tissues occurs has long been considered an important characteristic by which chronic and aggressive forms of periodontitis can be clinically distinguished. Chronic periodontitis has traditionally been viewed as a slowly progressing disease, whereas aggressive forms of periodontitis progress at a rapid rate. The most compelling argument indicating that aggressive periodontitis progresses at a rapid rate comes from case series and epidemiological reports showing extensive periodontal damage at some sites in adolescents and young adults. Rates of progression of various forms of periodontitis are difficult to study since there are many factors that influence how rapidly periodontal tissues are destroyed like the effectiveness of oral hygiene habits, access to dental care, genetic susceptibility to periodontal infections, systemic diseases (e.g., diabetes mellitus), and other powerful host-response modifiers (e.g., smoking).[18]

Patterns of destruction

In cases of chronic periodontitis, there is no consistent pattern to the number and types of teeth involved. The disease can be localized to a few teeth or can affect the entire dentition. In cases of generalized aggressive periodontitis, most permanent teeth are usually affected. There are no evidence-based criteria to determine when a localized periodontal infection becomes generalized.[19] The consensus at the 1999 Classification Workshop suggested the extent of the disease be considered localized if < 30% of the sites (or teeth) are affected and generalized if > 30% of the sites (or teeth) are involved to facilitate communication among colleagues as to the general location of the problem. The fallacy of rigidly using the 30% cut-off point between localized and generalized patterns of disease can be nicely demonstrated in a classic case of localized aggressive periodontitis in which 12 teeth are affected (i.e., all

incisors and first molars). If such a patient has only 28 teeth, then 12 / 28 or 42.9% teeth have the disease. Therefore, if the 30% figure is rigidly applied, some individuals with localized aggressive periodontitis paradoxically have generalized disease.[20]

Clinical signs of inflammation

One of the features of localized aggressive periodontitis is the relatively low level of gingival inflammation (e.g., redness, swelling) compared with other forms of periodontitis. In contrast, patients with generalized aggressive or chronic forms of periodontitis usually present with relatively intense gingival inflammation.[21]

Plaque and calculus formation

In many patients with localized aggressive periodontitis, there are only thin deposits of dental plaque (i.e., biofilm) with little or no calculus. However, sites affected by the disease are not biofilm-free. In contrast, teeth with chronic periodontitis usually have very complex and thick deposits of polymicrobial communities on affected root surfaces and this may hold true for generalized aggressive periodontitis cases as well.[22]

CONCLUSION

Author found that a wide range of host and microbial factors contribute to alveolar bone loss in periodontitis. Bone resorption via osteoclasts and bone formation via osteoblasts are coupled, and their dysregulation is associated with numerous diseases of the skeletal system.

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