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Review Article

Periodontal medicine: a comprehensive review

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ABSTRACT:
Periodontal disease is primarily associated with bacterial infection such as dental plaque. Dental plaque, an oral biofilm harboring a complex microbial community, can cause various inflammatory reactions in periodontal tissue. In many cases, the local bacterial invasion and host-mediated immune responses lead to severe alveolar bone destruction. To date, plaque control, non-surgical, and surgical interventions have been the conventional periodontal treatment modalities. Although adjuvant therapies including antibiotics or supplements have accompanied these procedures, their usage has been limited by antibiotic resistance, as well as their partial effectiveness. Therefore, new strategies are needed to control local inflammation in the periodontium and host immune responses. In recent years, target molecules that modulate microbial signaling mechanisms, host inflammatory substances, and bone immune responses have received considerable attention by researchers.
Key words: Periodontal medicine, Inflammation

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INTRODUCTION

Periodontitis is inflammation and infection of the ligament and alveolar bone supporting the teeth that can have significant effects on general health and vice versa, i.e., a number of systemic diseases and conditions can be potential risk factors for periodontitis as well. There is an increasing interest over recent years in the relationship between periodontal and systemic health that has labeled periodontal–systemic interlink as a two-way road.[1, 2]
Periodontitis was once, generally believed to be an inevitable consequence of aging. However, with an increasing body of epidemiological and experimental work, specific risk-factors and risk-indicators for periodontitis such as tobacco smoking, demographic factors, socio-economic status, several general diseases, and conditions and psychological stress have been identified and acknowledged, permitting a better understanding of what makes an individual more susceptible to periodontal diseases. This knowledge has given an increasing emphasis to the important role that systemic factors, diseases, and conditions ranging from the hormonal changes during puberty and pregnancy to disease entities involving immune dysfunction, connective tissue disease and malignancy, play in the causation and progression of periodontal disease. Dentistry has also become more cognizant of the extent to which behavioral factors play a role as a risk-factor for periodontal diseases. The dynamics of the periodontium are a product of its circulation, hormonal changes and immune response mechanisms. Changes in systemic health that affect any of these factors can be reflected as changes in periodontal health. This side of the link has long been established indubitably. In fact the influence of diabetes on periodontal health was found to be so compelling that Loe in 1993 regarded periodontitis as the 6th complication of diabetes.[1- 5]

PERIODONTAL MEDICINE

The endodontic community has remained steadfast in its rejection of the infected root canal as a cause of distant, non-infectious disease. The position of the American Association of Endodontists (AAE) has been that (i) bacteraemia occurs as part of normal

daily activity such as chewing and tooth brushing; (ii) there is no evidence on the inoculum size needed to generate a metastatic disease – the only consistency between the turn of the century animal studies was that the inoculum sizes were unrealistically large; and (iii) dental extractions produce a larger circulatory bacterial load than endodontic therapy. The AAE does, however, recognize that untreated peri-apical infections may cause distant disease by releasing bacteria and bacterial products into the circulation.[6]

On the other hand, untreated periodontal disease has continued to be examined as a source of circulatory bacteria. This became especially important when the American Heart Association released a position paper on the role of oral streptococci in bacterial endocoarditis. The last decades of the 20th century saw the emergence of new techniques for bacterial identification and classification, especially oral microorganisms. Non-targeted molecular assays such as 16S sequencing revealed the presence of novel and hitherto unsuspected organisms in the oral cavity, while innovations in culturing and microscopical approaches allowed the identification of uncommon phenotypes in known species. Several systemic pathogens, ranging from respiratory pathobionts such as Hemophilus influenzae, Pseudomonas aeruginosa and Acinetobacter baumanii to gut pathogens such as Trophyrema whipplei, have been identified in significant numbers in the periodontal pocket. Explorations of atheromatous plaques, knee implants, placenta, amniotic sac, the tracheobronchial tree, joint cavities and the pancreas have revealed the presence of periodontal pathogens, for example Porphyromonas gingivalis, Treponema denticola, Fusobacterium nucleatum and Campylobacter rectus, in these areas, especially in regions that were previously considered sterile. These advances in microbiological methodologies and clinical techniques produced data that suggested that the oral cavity could indeed act as a reservoir of bacteria that might metastasize to distant sites in the body and cause disease in susceptible individuals. The World Workshop in Periodontics introduced the term ‘periodontal medicine’ in 1996 to describe the role played by periodontitis in exacerbating or initiating systemic diseases. Thus, the last two decades have seen what may be considered a resurrection of the focal infection theory; however, investigators are using an abundance of caution in advocating therapy based on these links.[7- 9]

While several lines of evidence are emerging to suggest that periodontitis may be linked to osteoporosis, diabetes, atherosclerotic circulatory disease, rheumatoid arthritis, pregnancy-related complications, pulmonary disorders, pancreatic cancer, chronic renal disease, obesity and Alzheimer's disease, there is little evidence at this point in time that oral bacteria or bacterially driven pathways play a role in all of these linkages.[10]

MORE RECENT ASSOCIATIONS BETWEEN PERIODONTITIS AND OTHER DISEASES

Despite the tremendous progress made by periodontal medicine since the early 1990s, new associations and, more importantly, the elucidation of the biological mechanisms for such associations continue to emerge. A systematic review conducted in 2016 revealed that periodontitis has been linked to 57 other systemic diseases. Periodontal medicine has been shown to be quite dynamic and in constant evolution, however, a future challenge will be to demonstrate convincingly that the prevention and treatment of periodontitis will bring significant benefits to other diseases. Among the associations that have gained more attention recently, we can highlight inflammatory bowel disease, several types of cancer, and Alzheimer’s disease. In this sense, the interrelation between the oral and intestinal ecosystems appears as a new frontier for periodontal medicine. Changes in the oral microbiota concerning oral and systemic diseases have been the focus of constant investigation. Several reports have shown that microorganisms from the oral cavity can overcome physical and chemical barriers and colonize other sites, potentially contributing to the emergence and/or aggravation of other diseases. Studies have shown the growth of oral bacteria in the intestinal microbiota of patients with several diseases. Experimental studies have shown that oral administration of P. gingivalis causes changes in the intestinal microbiota and intestinal epithelial barrier, together with changes in the liver and adipose tissue. In line with these findings, individuals with periodontitis have shown changes in the intestinal microbiome, such as less diversity and an increase in the Firmicutes / Bacteroidetes ratio.[11]

Epidemiological studies have shown that individuals with inflammatory bowel disease were more likely to have periodontitis. On the other hand, the presence of periodontitis increased the risk of developing ulcerative colitis, one of the types of inflammatory bowel disease, over a 13-year follow-up. Despite limited epidemiological evidence pointing to periodontitis as a possible risk factor for inflammatory bowel disease, substantial studies in animal models are beginning to point out the possible biological mechanisms by which periodontal disease and oral pathogens contribute to inflammatory bowel disease. Oral bacteria have been shown to colonize and persist in the intestine, activating the immune system and causing chronic inflammation in a susceptible host. Klebsiella strains isolated from saliva can induce a strong T helper 1 cell response in the intestine in the context of intestinal dysbiosis and induce colitis in genetically susceptible hosts. More recently, it has been shown that oral pathobionts, including Klebsiella species, increase their number in periodontitis and can trigger an inflammatory response in the intestine through two mechanisms. First, when colonizing the intestine, oral pathobionts activate the local immune system, triggering the production of IL-1β and colitis.

Second, T helper 17 cells that appear during periodontitis can migrate to the intestine and be activated by oral pathobionts that colonized the intestine and lead to the development of colitis. These discoveries reveal new mechanisms of how periodontitis can influence other pathogenic processes in sites far from the oral cavity and open new paths for investigating the clinical and therapeutic impact of periodontitis.[12- 15]

BONE REGENERATION: ANABOLIC AGENTS

Teriparatide (FORTEO®): Teriparatide (FORTEO®) which con-sists of the bioactive portion of parathyroid hormone (PTH), was the first anabolic drug approved by the FDA to treat osteoporosis. Because PTH plays a role in the WNT/β-catenin signaling that downregulates sclerostin, which is an inhibitor of WNT-LRP5/6, and stimulates bone formation, Teriparatide is a research target among periodontists as a potential means of achieving alveolar bone regeneration. Teriparatide activates a signaling cascade that includes protein kinase-1, cyclic adenosine monophosphate, and protein kinase. A number of preclinical studies have shown that teriparatide has potential as a therapeutic target for alveolar bone defects. Moreover, clinical studies involving the systematic administration of teriparatide for periodontal tissue regeneration resulted in successful outcomes of significant bone formation. In a clinical study, a daily subcutaneous injection of teriparatide (20 μg) over the course of 6 weeks resulted in significant bone formation in the osseous defect, accompanied by a 33% reduction of periodontal probing depth, and a 22% gain in clinical attachment level. In another study, short-term (28 days) daily systemic administration of teriparatide positively affected the osseointegration of a titanium implant. While the clinical effects of teriparatide have been observed, the inconvenience of daily injections and the risk of osteosarcoma has prompted a search for new candidate drugs that would offer similar benefits without these drawbacks, including sclerostin antibodies. Alternatively, teriparatide may be incorporated into current treatment regimens for periodontal disease and dental implants if new methods of oral or local drug delivery are developed.[16]

Sclerostin antibody: Second only to bone morphogenetic proteins, the Wnt/β-catenin signaling pathway has received much attention due to its importance in osteoblast differentiation and bone formation. Similar to other signaling mechanisms, Wnt signaling is also regulated by an agonist and antagonist. Sclerostin, encoded by the SOST gene, is primarily expressed in the osteocyte and achieves an anti-anabolic effector by inhibiting Wnt signaling. Historically, it was believed that sclerostin was a BMP antagonist, however, it was subsequently determined that sclerostin bonded to the WNT co-receptor (low density lipoprotein receptor–related protein 5/6 (LRP5/6)) and thus inhibited the Wnt signaling pathway. Previous authors identified that mutations in LRP5 decreased sclerostin binding, and that the resulting lack of sclerostin induced high bone mass (a condition referred to as sclerosteosis). These findings generated significant interest in both the academic and private sectors, where researchers have spent the last decade exploring the role of WNT signaling in bones through a wealth of genetic studies of mice and humans. All of these studies have confirmed the importance of this pathway in bone biology and disease. Building on these studies, sclerostin antibody (Scl-Ab) has been widely studied as a bone anabolic agent for possible treatment of osteoporosis, and numerous preclinical and clinical studies have proved its effects on bone formation. As alveolar bone and skeletal bone rely on a similar mechanism during bone remodeling, including the balance of osteoblast and osteoclast, drugs that target skeletal bone may be suitable candidates for treatment of periodontal disease. Previous authors reported that sclerostin and the sclerostin:RANKL ratio were higher in the gingival crevicular fluid of periodontitis patients, and some pre-clinical studies have shown that sclerostin inhibition decreased bone resorption and promoted bone regeneration in animal periodontitis models. For the same reasons that sclerostin inhibition using Scl-Ab has proven helpful to patients with osteoporosis, sclerostin modulation may prove to be an effective pharmaceutical target for the treatment of periodontal disease.[17- 19]

CONCLUSION

Irrespective of the significant or non-significant correlation and varying strength of periodontal-systemic interlink that a number of studies have depicted, these exhaustive researches by providing scientific evidence of the possible interactive mechanism between oral and systemic health, have contributed extensively in expanding one's horizon, from viewing periodontal disease as a “localized entity” to “one effecting the whole body.” This is potentially of great public health significance, as periodontal disease is largely preventable and in many instances readily treatable.

REFERENCES

1. Genco RJ. Current view of risk factors for periodontal diseases. J Periodontol. 1996;67:1041–9.
2. Garcia RI, Henshaw MM, Krall EA. Relationship between periodontal disease and systemic health. Periodontol 2000. 2001;25:21–36.
3. Klokkevold PR, Mealey BL. Influence of systemic conditions on the periodontium. In: Newman MG, Takei HH, Klokkevold PR, Carranza FA, editors. Carranza's Clinical Periodontology. 10th ed. St. Louis: Saunders Elsevier; 2006. pp. 284–311.
4. Taylor BA. Periodontal diseases and systemic health: Associations, directions, implications. NSW Public Health Bull. 1998;10:14–6.
5. Löe H. Periodontal disease. The sixth complication of diabetes mellitus. Diabetes Care. 1993;16:329–34.

6. Erdemir EO, Nalcaci R, Caglayan O. Evaluation of systemic markers related to anemia of chronic disease in the peripheral blood of smokers and non-smokers with chronic periodontitis. Eur J Dent. 2008;2:102–9.
7. Hunter W (1900). Oral sepsis as a cause of disease. Br Med J 2, 215–216.
8. Ingle JI, Bakland LK & Baumgartner JC (2008). Ingle's Endodontics 6 BC Decker, Hamilton, Ontario.
9. Jamieson GA, Maitland NJ, Wilcock GK, Yates CM & Itzhaki RF (1992). Herpes simplex virus type 1 DNA is present in specific regions of brain from aged people with and without senile dementia of the Alzheimer type. J Pathol 167, 365–368
10. Kamphuis S, Kuis W, de Jager W, Teklenburg G, Massa M, Gordon G, Boerhof M, Rijkers GT, Uiterwaal CS, Otten HG, Sette A, Albani S & Prakken BJ (2005). Tolerogenic immune responses to novel T-cell epitopes from heat-shock protein 60 in juvenile idiopathic arthritis. Lancet 366, 50–56.
11. Kell D, Potgieter M & Pretorius E (2015). Individuality, phenotypic differentiation, dormancy and 'persistence' in culturable bacterial systems: commonalities shared by environmental, laboratory, and clinical microbiology. F1000Res 4, 179.
12. Kinloch AJ, Alzabin S, Brintnell W, Wilson E, Barra L, Wegner N, Bell DA, Cairns E & Venables PJ (2011). Immunization with Porphyromonas gingivalis enolase induces autoimmunity to mammalian α-enolase and arthritis in DR4-IE-transgenic mice. Arthritis Rheum 63, 3818–3823.
13. Puig Silla M, Montiel Company JM, Almerich Silla JM. Use of chlorhexidine varnishes in preventing and treating periodontal disease. A review of the literature. Med Oral Patol Oral Cir Bucal. 2008;13:E257–60.
14. Gunsolley JC. Clinical efficacy of antimicrobial mouthrinses. J Dent. 2010;38(Suppl 1):S6–10.
15. Sharma N, Charles CH, Lynch MC, Qaqish J, McGuire JA, Galustians JG, et al. Adjunctive benefit of an essential oil-containing mouthrinse in reducing plaque and gingivitis in patients who brush and floss regularly: A six-month study. J Am Dent Assoc. 2004;135:496–504.
16. Mythri H, Ananda SR, Prashant GM, Subba Reddy VV, Chandu GN. The efficacy of antiseptic mouth rinses in comparison with dental floss in controlling interproximal gingivitis. J Int Soc Prev Community Dent. 2011;1:31–5.
17. Bauroth K, Charles CH, Mankodi SM, Simmons K, Zhao Q, Kumar LD. The efficacy of an essential oil antiseptic mouthrinse vs. Dental floss in controlling interproximal gingivitis: A comparative study. J Am Dent Assoc. 2003;134:359–65.
18. Deep P. Screening for common oral diseases. J Can Dent Assoc. 2000;66:298–9.
19. Chen ZY, Chiang CH, Huang CC, Chung CM, Chan WL, Huang PH, et al. The association of tooth scaling and decreased cardiovascular disease: A nationwide population-based study. Am J Med. 2012;125:568–75.