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Hormonal effect on the Periodontium: A Brief Review

Abstract

Periodontitis is a chronic bacterial infection of the supporting structures of the teeth. The host response to infection is an important factor in determining the extent and severity of periodontal disease. Periodontitis is now seen as resulting from a complex interplay of bacterial infection and host response, often modified by behavioral factors. Systemic factors modify periodontitis principally through their effects on the normal immune and inflammatory mechanisms. These systemic disorders have been documented as capable of affecting the periodontium and/or treatment of periodontal disease. In order to rationally treat and prevent periodontal disease, we need to know the etiologic agents for specific patients, and the mechanism of bacterial pathogenesis in periodontitis. In systemic diseases in which the periodontal tissues are affected as well, early detection and carefully managed therapeutics with the physician and periodontist working together may prove beneficial to the patient's general health and quality of life. In this article, we attempt to outline the effect of systemic hormones and body changes on the periodontium.

Key Words

Hormone; disease; periodontium

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INTRODUCTION

Periodontal diseases include a group of inflammatory diseases characterized by progressive destruction of the periodontium. Dental plaque is the initiator of periodontal disease, but disease severity and response to treatment are determined predominantly by host-based risk factors. When dealing with periodontal problems, particularly in children and adolescents, it is advisable to establish a differential diagnosis of periodontal disease due to systemic conditions.[1] Although specific microorganisms have been identified as putative periodontal pathogens, it has become apparent that pathogens are necessary, but not sufficient, for tissue breakdown to occur.[2] Their presence, in fact, is a crucial factor in the onset of periodontitis, but disease susceptibility, progression, and severity as well as response to treatment is determined predominantly by host-based risk factors.[3] These factors operate by affecting primary host defense mechanisms at the microbial-host interface (humoral immune response, polymorphonuclear leukocyte phagocytosis and killing) and the production of pro-inflammatory cytokines, prostaglandins, and matrix metalloproteinases in periodontal connective tissue. Therefore, systemic diseases and genetic disorders that affect immune function, inflammatory response, and tissue organization are considered major determinants of susceptibility and severity of periodontitis.[4]

Periodontal diseases and cardiovascular diseases

Cardiovascular diseases (CVD) are a group of diseases that include congestive heart failure, cardiac arrhythmias, coronary artery disease (including atherosclerosis and myocardial infarction), valvular heart disease and stroke. CVD and periodontitis are both chronic and multifactorial diseases, and share some of their risk factors: age, male gender, lower socioeconomic status, smoking and psychosocial factors such as stress.[5] Recently, periodontal disease (PD) has been investigated as a potential factor contributing to the onset and development of CVD.[6] Several mechanisms that could explain this association have been investigated. The host response to the presence of periodontal pathogens may trigger the production of

inflammatory mediators such as C-reactive protein, TNF-α, PGE2, IL-1β and IL-6, which can accelerate the progression of pre-existing atherosclerotic plaques[7] and are related to an increased number of adverse cardiovascular events.[8] Also, several studies demonstrated the ability of periodontal pathogens to induce platelet aggregation and the formation of atheromas.[9,10]

Pregnancy and Periodontal Diseases

The first study to report the influence of poor oral health on the birth of low weight and preterm infants was performed by Offenbacher and colleagues.[11] The etiology of preterm birth is multifactorial, but inflammation is the common pathway that leads to uterine contractions and cervical changes with or without premature rupture of membranes. Biological plausibility of the link between both conditions, periodontal disease and preterm birth, does exist and can be summarized in three potential pathways.[12,13] One of them refers to the hematogenous dissemination of inflammatory products from a periodontal infection, while the second potential pathway involves the fetomaternal immune response to oral pathogens. The third pathway proposed to explain the theoretical causal relationship between periodontal disease and preterm birth involves bacteremia from an oral infection. There appears to be an association between both conditions, but whether periodontitis is a confounding factor, a marker or one of the causes of preterm birth remains unclear.[14]

Diabetes and Periodontium

Diabetes is a group of metabolic diseases characterized by hyperglycemia and results from either a deficiency in the secretion of insulin and/or reduced insulin action.[15] Chronic periodontal disease and diabetes mellitus are common chronic conditions in adults throughout the world.[16] Severe periodontal disease often coexists with diabetes and is considered the sixth most common complication of the disease.[17] A number of studies have demonstrated that poor blood sugar control may contribute to poor periodontal health[18-24] and that such individuals have a 2.8-fold greater chance of developing destructive periodontal disease[19] as well as a 4.2-fold greater chance of having progressive alveolar bone loss.[25] The increased risk of developing periodontal disease cannot be explained by age, gender or hygiene.[26] The interrelationship between periodontal disease and diabetes provides an example of a systemic disease predisposing individuals to oral infection and, once the infection is installed, it exacerbates the systemic disease.[16] The interrelationship between diabetes and periodontal disease is established through a number of pathways[27] and is bidirectional.[28] Diabetes is a risk factor for gingivitis and periodontitis.[29,30] Blood sugar control is an important variable in the relationship between diabetes and periodontal disease. Individuals who have poor control over glycemia have a greater prevalence and severity of gingival and periodontal inflammation.[31-33] It has been suggested that hyperglycemia promotes periodontitis and its progression.[28,34-39]

Periodontal Diseases and Respiratory Diseases

Respiratory diseases is the term for diseases of the respiratory system, including lung, pleural cavity, bronchial tubes, trachea, and upper respiratory tract. There is increasing evidence that a poor oral health can predispose to respiratory diseases, especially in high-risk patients (nursing home residents, older subjects, intensive care unit patients and hospitalized individuals requiring mechanical ventilation). The oral cavity is contiguous with the trachea and may be a portal for respiratory pathogen colonization. Dental plaque can be colonized by respiratory pathogens,[40] which may be aspirated from the oropharynx into the upper airway and then reach the lower airway and adhere to bronchial or alveolar epithelium.[41] There is fair evidence of an association of pneumonia with oral health, but there is poor evidence of a weak association between COPD and oral health. Improved oral hygiene and professional oral health care reduces the progression or occurrence of respiratory diseases among high-risk elderly adults.

Oral contraceptives and Periodontium

Hormones are specific regulatory molecules that have potent effects on the major determinants of the development and the integrity of the skeleton and oral cavity including periodontal tissues. It is clear that periodontal manifestations occur when an imbalance of these steroid hormones take place. Women using hormonal contraceptives can be considered to be a 'risk group' for periodontal disease, due to prolonged, sustained serum levels of oestrogens and progesterone.[42] It is clear that endogenous sex steroid hormones play significant roles in modulating the periodontal tissue responses. A better understanding of the periodontal changes to varying hormonal levels throughout life can help the dental practitioner in diagnosis and treatment. The women under contraceptive seem to set up a group

at risk for developing a periodontal disease, it is thus necessary to systematise periodontal appraisal before and during contraceptive use period.[43]

CONCLUSION

The connection between systemic diseases and chronic destructive periodontitis (CDP) has received increasing attention in recent years. A major unanswered question is how disease in one part of the body (e.g., the joints and skeletal tissues) can transmit signals to the periodontium. Current treatment approaches available to the periodontist and dentist include: (i) antimicrobial therapy, including mechanical debridement and surgical reduction of probing depth to reduce the bacterial "load" in the periodontal pocket, combined (as needed) with topical and systemically administered antimicrobials; and (ii) host-modulation therapy (using FDA-approved, MMP-inhibitor sub-antimicrobial-dose doxycycline by itself or, after confirmation by additional research, using other pharmaceuticals, such as non-steroidal anti-inflammatory drugs [NSAIDs] and bisphosphonates, or combinations of these). There appears to be more than a casual relationship between serum lipid levels and systemic health (particularly cardiovascular disease, diabetes, tissue repair capacity, and immune cell function), susceptibility to periodontitis, and serum levels of pro-inflammatory cytokines. In terms of the potential relationship between periodontitis and systemic disease, further research is warranted for the better treatment of the patients.

REFERENCES

1. Pizzo G, Lo Re D, Piscopo MR, Pizzo I, Giuliana G. Genetic disorders and periodontal health: a literature review. Med Sci Monit 2009;15(8):167-78.
2. Socransky SS, Haffajee AD: The bacterial etiology of destructive periodontal disease: current concepts. J Periodontol 1992;63:322-31.
3. Kinane DF, Peterson M, Stathopoulou PG: Environmental and other modifying factors of the periodontal diseases. Periodontol 2000 2006;40:107-19.
4. Hart TC, Atkinson JC. Mendelian forms of periodontitis. Periodontology 2000 2007;45:95-112.
5. Paquette DW, Brodala N, Nichols TC. Cardiovascular disease, inflammation, and periodontal infection. Periodontol 2000 2007;44:113-26.
6. Weidlich P, Cimões R, Pannuti CM, Oppermann RV. Association between periodontal diseases and systemic diseases. Braz Oral Res 2008;22(1):12-14.
7. Lalla E, Lamster IB, Hofmann MA, Bucciarelli L, Jerud AP, Tucker S, *et al*. Oral infection with a periodontal pathogen accelerates early atherosclerosis in apolipoprotein E-null mice. Arterioscler Thromb Vasc Biol 2003;23(8):1405-11.
8. Scannapieco FA, Bush RB, Paju S. Associations between periodontal disease and risk for atherosclerosis, cardiovascular disease, and stroke. A systematic review. Ann Periodontol 2003;8(1):38-53.
9. Khader YS, Albashaireh ZS, Alomari MA. Periodontal diseases and the risk of coronary heart and cerebrovascular diseases: a meta-analysis. J Periodontol 2004;75(8):1046-53.
10. Meurman JH, Sanz M, Janket SJ. Oral health, atherosclerosis, and cardiovascular disease. Crit Rev Oral Biol Med 2004;15(6):403-13.
11. Offenbacher S, Katz V, Fertik G, Collins J, Boyd D, Maynor G, *et al*. Periodontal infection as a possible risk factor for preterm low birth weight. J Periodontol 1996;67(10):1103-13.
12. Pretorius C, Jagatt A, Lamont RF. The relationship between periodontal disease, bacterial vaginosis, and preterm birth. J Perinat Med 2007;35(2):93-9.
13. Goldenberg RL, Hauth JC, Andrews WW. Intrauterine infection and preterm delivery. N Engl J Med 2000;342(20):1500-7.
14. Klebanoff M, Searle K. The role of inflammation in preterm birth/ - focus on periodontitis. BJOG 2006;113(3):43-5.
15. Almas K, Al-Lazzam S, Al-Quadairi A. The effect of oral hygiene instructions on diabetic type 2 male patients with periodontal diseases. J Contemp Dent Pract. 2003;4(3):24-35.
16. Kiran M, Arpak N, Unsal E, Erdogan MF. The effect of improved periodontal health on metabolic control in type 2 diabetes mellitus. J Clin Periodontol. 2005;32(3):266-72.
17. Loe H. Periodontal disease. The sixth complication of diabetes mellitus. Diabetes Care 1993;16(1):329-34.
18. Cianciola LJ, Park BH, Bruck E, Mosovich L, Genco RJ. Prevalence of periodontal disease in insulin-dependent diabetes mellitus (juvenile

diabetes). J Am Dent Assoc 1982;104(5):653-60.
19. Emrich LJ, Shlossman M, Genco RJ. Periodontal disease in non-insulin-dependent diabetes mellitus. J Periodontol 1991;62(2):123-31.
20. Jones JA, Miller DR, Wehler CJ, Rich SE, Krall-Kaye EA, McCoy LC, *et al*. Does periodontal care improve glycemic control? The Department of Veterans Affairs Dental Diabetes Study. J Clin Periodontol 2007;34(1):46-52.
21. Novaes Jr AB, Gutierrez FG, Novaes AB. Periodontal disease progression in type II non-insulin-dependent diabetes mellitus patients (NIDDM). Part I - Probing pocket depth and clinical attachment. Braz Dent J 1996;7(2):65-73.
22. Safkan-Seppala B, Ainamo J. Periodontal conditions in insulin-dependent diabetes mellitus. J Clin Periodontol 1992;19(1):24-9.
23. Taylor GW, Burt BA, Becker MP, Genco RJ, Shlossman M, Knowler WC, *et al*. Non-insulin dependent diabetes mellitus and alveolar bone loss progression over 2 years. J Periodontol 1998;69(1):76-83.
24. Unal T, Firatli E, Sivas A, Meric H, Oz H. Fructosamine as a possible monitoring parameter in non-insulin dependent diabetes mellitus patients with periodontal disease. J Periodontol 1993;64(3):191-4.
25. Taylor GW, Burt BA, Becker MP, Genco RJ, Shlossman M. Glycemic control and alveolar bone loss progression in type 2 diabetes. Ann Periodontol 1998;3(1):30-9.
26. Rodrigues DC, Taba MJ, Novaes AB, Souza SL, Grisi MF. Effect of non-surgical periodontal therapy on glycemic control in patients with type 2 diabetes mellitus. J Periodontol 2003;74(9):1361-7.
27. Tan WC, Tay FB, Lim LP. Diabetes as a risk factor for periodontal disease: current status and future considerations. Ann Acad Med Singapore 2006;35(8):571-81.
28. Grossi SG, Genco RJ. Periodontal disease and diabetes mellitus: a two-way relationship. Ann Periodontol 1998;3(1):51-61.
29. Papapanou PN. Periodontal diseases: epidemiology. Ann Periodontol 1996;1(1):1-36.
30. Mealey BL. Periodontal implications: medically compromised patients. Ann Periodontol 1996;1(1):256-321.
31. Tervonen T, Oliver RC. Long-term control of diabetes mellitus and periodontitis. J Clin Periodontol 1993;20(6):431-5.
32. Ervasti T, Knuuttila M, Pohjamo L, Haukipuro K. Relation between control of diabetes and gingival bleeding. J Periodontol 1985;56(3):154-7.
33. Campus G, Salem A, Uzzau S, Baldoni E, Tonolo G. Diabetes and periodontal disease: a case-control study. J Periodontol 2005;76(3):418-25.
34. Taylor GW. The effects of periodontal treatment on diabetes. J Am Dent Assoc 2003;134:41-8.
35. Taylor GW. Bidirectional interrelationships between diabetes and periodontal diseases: an epidemiologic perspective. Ann Periodontol 2001;6(1):99-112.
36. Taylor GW. Periodontal treatment and its effects on glycemic control: a review of the evidence. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;87(3):311-6.
37. Iacopino AM. Periodontitis and diabetes interrelationships: role of inflammation. Ann Periodontol 2001;6(1):125-37.
38. Heitz-Mayfield LJ. Disease progression: identification of high-risk groups and individuals for periodontitis. J Clin Periodontol 2005;32(6):196-209.
39. Almas K, Al-Qahtani M, Al-Yami M, Khan N. The relationship between periodontal disease and blood glucose level among type II diabetic patients. J Contemp Dent Pract 2001;15;2(4):18-25.
40. Didilescu AC, Skaug N, Marica C, Didilescu C. Respiratory pathogens in dental plaque of hospitalized patients with chronic lung diseases. Clin Oral Investig 2005;9(3):141-7.
41. Scannapieco FA. Role of oral bacteria in respiratory infection. J Periodontol 1999;70(7):793-802.
42. Tilakaratne A, Soory M, Ranasinghe AW, Corea SM, Ekanayake SL, de Silva M. Effects of hormonal contraceptives on the periodontium, in a population of rural Sri-Lankan women. J Clin Periodontol 2000;27(10):753-7.
43. Seck-Diallo A, Cissé ML, Benoist HM, Diouf A, Ahnoux-Kouadio A, Diallo T, *et al*.

Periodontal status in a sample of Senegalese women using hormonal contraception. Odontostomatol Trop. 2008;31(121):36-42.