Periodontitis, Diabetes and Smoking

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Periodontal diseases have, for a half of a century, been known to be initiated by the accumulation of bacterial plaque (R). It has however been increasingly apparent in recent years that both intrinsic and extrinsic risk factors influence the progression of periodontitis. Of these the most important are Diabetes and Smoking. Since both of these factors are common in the UAE this is of particular importance to UAE dentists. It is the purpose of this paper to review the current associations between these risk factors and periodontitis, and to discuss the likely bi-directional relationship between management of periodontitis and diabetes control.

Periodontitis and Diabetes
Diabetes mellitus occurs when the level of sugar (glucose) in the blood becomes higher than normal. There are two main types of diabetes. In type 1 diabetes the body stops making insulin and the blood glucose level becomes very high (R). Type 2 diabetes, also called adult-onset diabetes and noninsulin-dependent diabetes, is a chronic condition caused by high levels of glucose (sugar) in the blood. Although some people can overcome the symptoms by losing weight and following a healthy diet and exercise plan, most people with type 2 diabetes will have it for life (R).

Both diabetes and periodontitis are chronic diseases. Diabetes has many adverse effects on the periodontium, and conversely periodontitis may have deleterious effects further aggravating the condition of diabetes. The potential common pathophysiologic pathways include those associated with inflammation, altered host responses, altered tissue homeostasis and insulin resistance. A recent study also showed that scaling, polishing and root planing of the periodontal tissue can lower blood sugar levels potentially offering a practical means of reducing the numbers who develop full type 2 diabetes (R).

Diabetes has many adverse effects on the periodontium, including decreased collagen turnover, impaired neutrophil function, and increased periodontal destruction. Neutrophil chemotaxis and phagocytic activities are compromised in diabetic patients, which can lead to reduced bacterial killing and enhanced periodontal destruction. Inflammation is exaggerated in the presence of diabetes, insulin resistance, and hyperglycemia (R).

Neelima et al (R) examined 1500 diabetic patients; the prevalence of periodontal disease in these patients was 86.8%, significantly higher than the general population. By the early 1990s periodontitis was referred to as the “sixth complication of diabetes”, and in 2005 the ADA formally acknowledged that periodontal disease is more often found in diabetics (R). Epstein (R) demonstrated that essentially all the aspects of bone growth and mineralization are diminished in the absence of insulin i.e. hyperglycemia. Vascular changes also increase with increase in blood glucose levels (Oliiver and Tervonen (R).

Periodontitis and Smoking
Second to bacterial plaque, smoking is the strongest of the modifiable risk factors for periodontal disease. Smokers harbor a higher prevalence of potential periodontal pathogens, and smoking impairs various aspects of immune responses, including neutrophil function, antibody production, fibroblast activities, vascular factors and inflammatory mediator production. In smokers inflammation in response to plaque accumulation is reduced compared with nonsmokers and smokers have a decreased expression of gingival inflammation and bleeding on probing in the presence of plaque accumulation when compared with smokers (R).

Smoking has been associated with a twoto eight-fold increased risk for periodontal attachment and/or bone loss, depending on the definition of disease severity and smoking dose. One of the largest epidemiological studies reporting an association between smoking and periodontitis included 12,329 U.S. adults 20 years and older. In this study, current smokers were four times as likely to have periodontitis compared to nonsmokers after adjusting for age, race ethnicity, income, and educational level. Heavy smokers (≥ 51 cigarettes per day) using a stricter definition of periodontitis (mean whole mouth attachment loss of ≥4 mm), the adjusted odds ratio was increased to 25.84 among smokers aged 50 years or more (R). Longitudinal studies have demonstrated that young individuals smoking more than 15 cigarettes per day showed the highest risk for tooth loss (R).

Smokers have a greater extent of colonization by periodontal pathogens than nonsmokers or former smokers particularly at shallow sites (pocket depth ≥ 4 mm) with no differences between smokers, former smokers, and nonsmokers in pockets ≥ 4 mm. This colonization, along with low gingival blood flow may lead to an increased prevalence of periodontal breakdown.

Smoking exerts a major effect on the protective elements of the immune response, resulting in an increase in the extent and severity of periodontal destruction. Neutrophils obtained from peripheral blood or saliva of smokers, have been shown to demonstrate functional alterations in chemotaxis, phagocytosis, and the oxidative burst. Smoking has been shown to impair the chemotaxis and phagocytosis of neutrophils obtained from the oral cavity, and in vitro studies of the effects of tobacco products on neutrophils have shown detrimental effects on cell movement and the oxidative burst (R). Smoking aids in the development of atherosclerosis, which is one of the most important risk factors for periodontal disease. Smokers have a decreased expression of genes, and smoking impairs various aspects of immune responses, including neutrophil function, antibody production, fibroblast activities, vascular factors and inflammatory mediator production.

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Fig. 1. A significant inflammatory response with bone loss in a poorly controlled diabetic.

Fig. 2. Advanced periodontal and imminent tooth loss in a heavy smoker.

The relationships between Periodontitis, Diabetes and Smoking are major areas of research interest at Dubai School of Dental Medicine.

Editorial note: (R) References are available from the authors.

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