## PERIODONTAL DISEASE IN DIABETIC PATIENTS

### Michael A. Leman

Dental Study Program, Faculty of Medicine, University of Sam Ratulangi Manado Email: micpatlem@yahoo.com

**Abstrak:** Diabetes melitus banyak ditemukan di negara berkembang, dengan konsekuensi cukup seringnya pasien diabetes yang berkunjung ke tempat praktek dokter gigi. Diabetes melitus merupakan penyakit yang kompleks dengan berbagai tingkat komplikasi sistemik dan pada rongga mulut. Jaringan periodonsium juga merupakan sasaran kerusakan akibat penyakit diabetes melitus. Beberapa tahun terakhir ini, hubungan antara periodontitis dan diabetes melitus telah dikemukakan. Dengan demikian penanganan gigi dan mulut yang tepat pada pasien dengan riwayat diabetes mellitus perlu ditingkatkan.

Kata kunci: diabetes melitus, penyakit periodontal, penanganan gigi dan mulut

**Abstract:** Diabetes mellitus is pandemic in both developed and developing countries. As a consequence, diabetic patients are commonly encountered in the dental office. Diabetes mellitus is a complex disease with varying degrees of systemic and oral complications. The periodontium is also a target for diabetic damage. In recent years, a link between periodontitis and diabetes mellitus has been postulated; therefore, a proper dental management in diabetic patients has to be improved.

Keywords: Diabetes mellitus, periodontal disease, dental management

Diabetes mellitus represents a spectrum of metabolic disorders and has emerged as a major health issue worldwide. 1 Changes in human behavior and lifestyles over the last century have resulted in a dramatic increase in the incidence of diabetes in the world.<sup>2</sup> Diabetes mellitus is a metabolic disorder characterized by hyperglycemia with metabolic disturbances of carbohydrates, fats and proteins resulting from defects in insulin secretion, insulin action, or both.<sup>1,3</sup> Periodontal disease is a chronic inflammatory disease of the oral tissues that result in the loss of attachment, bone destruction, and eventually the loss of teeth which are caused by gram-negative bacteria. 1,4 Persons with diabetes mellitus are at a greater risk of developing periodontal disease.<sup>4</sup> Both diabetes mellitus and periodontitis are chronic diseases affecting large numbers of worldwide populations.<sup>2</sup>

There are two major types of diabetes,

type 1 and type 2. Type 1 diabetes mellitus, formerly insulin-dependent diabetes mellitus, is caused by a cell mediated autoimmune destruction of the insulin producing beta cells of the islets of Langerhans in the pancreas, which result in insulin deficiency. Type 1 diabetes accounts for 5 -10% of all diabetes and most occurs in children and young adults. This types of diabetes results from a lack of insulin production and is very unstable and difficult to control. Type 2 diabetes mellitus, formerly non-insulin dependent diabetes mellitus, is caused by peripheral resistance to insulin action, impaired insulin secretion, and increased glucose production in the liver. The insulin producing beta cells in the pancreas are not destroyed by cell mediated autoimmune reaction. Type 2 diabetes mellitus is the most common form of diabetes, accounting for 90 - 95% of all cases and usually has an adult onset. 5-10

Periodontal infection represents a complication that may be involved in altering systemic physiology in diabetic patients. Studies suggest that as an infectious process with a prominent inflammatory component, periodontal disease can adversely affect the metabolic control of diabetes by worsening glycemic control over time compared to diabetic subjects without periodontitis. Diabetic patients with the periodontal disease may have an increased risk of diabetic complications. <sup>2</sup>

### **PATHOGENESIS**

A number of clinical studies have shown a significant relationship between diabetes and periodontal disease. Despite their being extensive research, the mechanism underlying the association of periodontitis and diabetes mellitus is not clear. Evidence has consistently indicated that diabetes is a risk factor for increased severity of gingivitis and periodontitis. As with other systemic conditions associated with periodontitis, diabetes mellitus does not cause gingivitis or periodontitis, but evidence indicates that it alters the response of the periodontal tissue to local factors.

However, while investigating the mechanism relating the link between the two chronic diseases, several studies have been focused on the microbial flora of the dental plaque which is the primary etiologic agent of periodontal disease. Anerobic gram-negative pathogens: Actinobacillus actinomycetemcomitans, Bacteroides forsynthus, Porphyromonas gingivali, Prevotella intermedia, Treponema denticola, and Eikenella corrodens are found to be associated with development and progression of periodontal disease.

Certain bacterial strains are found to be of producing proteolytic capable enzymes or leukotoxins, which facilitate the invasion into host tissues. A. actinomycetemcomitans and P. gingivalis produce proteases and metabolic by-products that can degrade surrounding tissue, and it has also been suggested that bacterial lipopolysaccharide can induce the bone resorption. Matrix metalloproteinases (MMPs) like collagenases, gelatinases, and elastiases of periodontal tissue plays a role in collagen degradation of osseous and connective tissue. Bacterial toxins, endoand cell membrane products toxins challenge the host thereby activating an inflammatory cascade with the synthesis of some effective mediators such as TNF alpha, IL-6, and IL-1 beta.<sup>2</sup>

Due to the accumulation of dental plaque, an inflammatory reaction occurs in the gingiva. In susceptible individuals, as the plaque matures, clinical attachment loss, gingival enlargement or recession, loss of alveolar bone, periodontal pocket formation, or bleeding gums ultimately results in tooth loss if it remains untreated. In case of diabetic patients, concentrations of oral microbial flora are increased due to higher concentrations of glucose in saliva and crevicular fluid.<sup>2</sup>

The increased glucose in the gingival fluid and blood of diabetic patients could change the environment of the microflora, inducing qualitative changes in bacteria that could contribute to the severity of periodontal disease observed in those with poorly controlled diabetes.<sup>5</sup> Both diabetes and periodontitis are chronic diseases. Diabetes has many adverse effects on the periodontium, including decreased collagen turnover, impaired neutrophil function, and increased periodontal destruction. Periodontitis can alter systemic physiology in diabetic patients. The effect of periodontitis on diabetes mellitus is believed to result from the nature of the inflammatory response in periodontal tissues.<sup>11</sup>

The function of inflammatory cells, such as neutrophils, monocytes, and macrophages, is altered in diabetic patients. Chemotaxis, adherence, and phagocytosis of neutrophils are impaired. The impairment of the neutrophil function may disturb host defense activity, thereby leading to periodontal destruction. In the presence of periodontal pathogens, macrophages, and monocytes exhibit an elevated production of cytokines, such as the tumor necrosis factor (TNF)- $\alpha$ , which may result

in further host tissue destruction. 1,4,7

Another hypothesis proposed is that in diabetes, hyperglycemia is associated with disturbances in carbohydrates, fat and protein metabolism; and persistent hyperglycemia which result in the alteration of circulating and immobilized proteins. Exposure of proteins (collagen) and lipids to the aldose sugars leads to non-enzymatic glycation and oxidation of proteins and lipids and the subsequent formation of advanced glycation endproducts (AGEs), which have the tendency to accumulate in the plasma and tissues. Chronic hyperglycemia adversely affects the synthesis, maturation, and maintenance of collagen and extracellular matrix. In the hyperglycemic state, numerous proteins and matrix molecules undergo a nonenzymatic glycosylation, resulting in accumulated AGEs. The formation of AGEs occurs at normal glucose levels as well, but in hypergylicemia environments, AGEs formation is excessive. 2,5,9

Accumulation of AGEs, as a result of the chronic hyperglycemic state or diabetes, coupled with the presence of infection and an exaggerated host response, may provide a viable explanation for the clinical outcomes observed in diabetic patients with periodontal disease. Evidence has accumulated supporting a role for AGEs in exacerbating diabetic systemic complications and periodontal disease severity associated with a chronic and intense inflammatory response. <sup>5,9</sup> Collagen is crosslinked by AGE formation, making it less soluble and less likely to be normally repaired or replaced. Cellular migration through cross-linked collagen is impeded, and perhaps more importantly, tissue integrity is impaired as a result of damaged collagen remaining in tissues for longer periods (i.e collagen is not renewed at a normal rate). As a result, collagen in the tissues of patients with poorly controlled diabetes is aged and more susceptible to breakdown (i.e less resistant to destruction by periodontal infections).<sup>5,11</sup>

Hyperglycemia results in an imbalance in lipid metabolism generally characterized by increased low density lipoproteins, triglycerides, and fatty acids in diabetic patients. Changes in lipid metabolism are correlated with impaired function of monocytes and/or macrophages in successive in vitro and in vivo studies ultimately leading to the overproduction of inflammatory cytokines. Several researchers have reported decreased functions of polymorphonuclear leukocyte (PMN) such as chemotaxis and phagocytosis in patients with periodontal disease. Along with inflammatory cytokines (TNF-α, interleukin/IL-1β, and IL-6), C-reactive protein (CRP) levels are also found to be raised in periodontal patients with diabetes mellitus.<sup>2</sup>

# INTERVENTION AND ITS IMPACT ON DIABETIC PATIENTS

Treatment attempts to reduce the number of pathogens and produce a peridontium that is conducive for health. Mechanical therapy remains the treatment of choice. Traditional therapy includes nonsurgical treatment by scaling and root planning. Mechanical therapy is generally used to disrupt the biofilm thereby reducing the virulence of pathogens and allowing the host to reestablish its periodontal health. Adjunctive antibiotics are being used wherever necessary but are used only after mechanical disruption of the biofilm.

Diabetic patients with periodontitis present increased serum levels of IL-6, TNF-α, and CRP, and are often found to have poor glycemic control. Periodontal treatment decreases local inflammation and a consequence, decreases chemical mediators involved in inflammation, among them IL-6 and CRP, positively contributing to proper glycemic control.4

Several studies have shown that scaling and root planing combined with the systemic administration of doxycycline can improve glycemic control. As cited by Deshapande from Hence et al. who conducted the study in metabolically controlled type 2 diabetic patients resulted in a significant improvement in the diabetic status and a reduction of its complications. <sup>2,8</sup> As

cited by Rosa and Ruben from Auito, that tissue insulin demand in type I diabetic patients decreases after periodontal treatment including scaling, root planning, curettage, gingivectomies, and selective extractions, in addition to the use of antibiotics such as penicillin and streptomycin.<sup>4</sup>

As cited by Abhijit & Varsha from Al Mubarak et al, scaling and root planning with adjunctive therapy may be of value in establishing a healthy periodontium in diabetic patients. As cited by Deshapande from Grosi et al, 113 native Americans who were treated by ultrasonic scaling with systemic doxycycline and irrigation with water, chlorhexidine, or povidone iodine showed a significant reduction in glycated hemoglobin, probing depth and subgingival microbial infection.

### **CONCLUSION**

Diabetes mellitus is a disease of which the general public, practicing dentists, and dental hygienists should be aware. Treatment of periodontal disease in diabetic patients may reduce the insulin requirements and improve the metabolic balance.

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