ABSTRACT

Periodontitis is usually initiated and propagated by bacteria and host factors. It is a multifactorial, irreversible condition. Diabetes mellitus is a complicated metabolic disorder and is leading cause of morbidity and mortality globally. Both these diseases are polygenic diseases with some extent of immune-regulatory dysfunction. Various studies have shown a higher incidence of periodontitis in diabetes patients as compared to healthy controls. The relationship between diabetes and periodontitis appears to be bi-directional with the appearance of the one condition favors another and also the management of the one condition may assist in the treatment of the other. Thus it is important that every dental practitioner should know this inter-relationship. The present article focusses on the relationship of the diabetes and periodontitis.

KEYWORDS: Diabetes, Metabolic disease, Periodontitis.
INTRODUCTION

Periodontal diseases are one of the most commonly occurring inflammatory and chronic infectious diseases in the world. Periodontal diseases have two important aspects in their pathogenesis: microorganisms and host response. Interactions between these two components play a decisive role in the initiation and progression of the periodontal diseases.\(^1\)

About 150 various species of microorganisms can be present in subgingival plaque and more than 500 different species of microorganisms can colonize the oral biofilm. Various systemic diseases and conditions can affect the initiation and progression of the periodontal disease and vice versa.\(^1\)

The biologic plausibility supporting such interaction is based on the fact that inflammatory periodontal disease due to reaction to pathogenic biofilm can stimulates a chronic systemic inflammation and thus contributes to the cumulative inflammatory burden in the host. Also in old age patients and in the indigenous people; the periodontitis prevalence is even higher.\(^2\)

Diabetes is a complex metabolic disease which affects quality of life with some major complications. It is caused either by deficiency in insulin production or an impaired utilization of insulin. There are two types of diabetes- Type 1 and Type 2. Type 1 has etiology of progressive autoimmune destruction of pancreatic insulin-producing \(\beta\) cells. Type 2 diabetes mellitus is a metabolic disorder of multiple etiology and characterized by chronic hyperglycemia with disturbances of carbohydrate, fat, and protein metabolism resulting from defects in insulin secretion, insulin action or both.\(^1,3\)

HISTORY

Gruner first reported the inter-relation between diabetes and periodontal disease. Williams in 1928 stated that, gingivitis and periodontitis among diabetic patients are modified. He termed it “diabetic periodontoclasia.” In 1946, Glickman found in an experimental animal study that periodontal disease in diabetic animals was not different histologically and was thus not a unique clinical entity.\(^4\)

Relation between diabetes and periodontitis

Diabetes and periodontal disease have a number of pathways common in their pathogenesis. These diseases are polygenic disorders with some degree of immunoregulatory dysfunction.\(^2\)

The association between diabetes and periodontal disease has been reported for more than 40
years but reverse has not been the focus of researchers until recently. Studies have suggested a bi-directional relationship between periodontal disease and glycaemic control with each disease having a potential impact on the other.\[^{3}\]

**Effect of diabetes on periodontitis**

Clinical studies have demonstrated a higher prevalence of periodontitis in diabetic patients. Periodontitis is now referred to as the sixth most common complication of diabetes.\[^{3,5}\]

Concentration of oral microbial flora is found to be increased due to higher concentration of glucose in saliva and crevicular fluid.\[^{6}\]

Diabetes-associated susceptibility traits for periodontal disease include defective function of immune cells, neutrophil dysfunction, defective secretion of growth factors, cytokines, abnormal cross-linking and glycosylation of collagen and subsequent impaired healing.\[^{1,7,8}\]

Neutrophil adherence, chemotaxis, and phagocytosis are often impaired, which may inhibit bacterial killing in the periodontal pocket and significantly increase periodontal destruction.\[^{3,7,8}\]

Macrophages and monocytes often exhibit elevated production of proinflammatory cytokines and mediators such as tumour necrosis factor (TNF-) in response to periodontal pathogens, which may increase host tissue destruction.\[^{3,4}\]

Altered wound healing is also major problem in people with diabetes. The primary reparative cell in the periodontium, the fibroblast, does not function properly in high-glucose environments. Furthermore; the collagen that is produced by these fibroblasts is susceptible to rapid degradation by matrix metalloproteinase enzymes.\[^{3}\]

The production of this enzyme is elevated in diabetes. Thus; periodontal wound healing responses to chronic microbial insult may be altered in those with sustained hyperglycaemia. The result an increased bone loss and attachment loss.\[^{3,4}\]

Reactive oxygen species also have a role in both periodontal disease and diabetes. Prolonged inflammation, such as periodontitis, is a constant source of reactive oxygen species and can compromise the antioxidant capacity of serum and tissues.\[^{1}\]

In addition; the level of glycemic control can play a role in the gingival response to microbial plaque in patients with diabetes.\[^{7}\]

People with diabetes, especially those with poor glycemic control, accumulate high levels of irreversibly glycated proteins called advanced glycation end products (AGEs) in the tissues,
including the periodontium. These advanced glycation end products are a primary link between numerous diabetic complications, because they induce marked changes in cells and extracellular matrix components. These changes, including abnormal endothelial cell function, capillary growth and vessel proliferation, also occur in the periodontium of some people with diabetes.[3, 8]

The effect of periodontitis on diabetes
All chronic infections usually affect the glycemic control in diabetics. Chronic inflammatory diseases lead to impairment in cell-mediated immunity such as neutrophil (polymorphonuclear leukocyte) chemotaxis, macrophage function and vascular disease. Cytokines which control humoral and cellular response are produced. These changes in cellular and hormonal response affect the release of insulin and affect glycemic control.[3, 8]

Periodontitis is one such chronic infection which affects the glycemic control in a similar manner. Previous studies indicated a six fold increase in risk of worsening glycemic control in diabetic patients having severe periodontitis compared with subjects with diabetics having no periodontitis.[3, 8]

Recently, the mechanisms by which periodontitis may affect the diabetic state have been elucidated. Both periodontitis and diabetes, especially type 2 diabetes, have major inflammatory components. Chronic periodontitis also have the potential to exacerbate insulin resistance and worsen glycemic control.[3] Periodontitis may initiate or propagate insulin resistance in a manner similar to that of obesity, by enhancing activation of the overall systemic immune response initiated by cytokines. Also, periodontal treatment that decreases inflammation may help diminish insulin resistance.[3, 9, 10]

CONCLUSION
Both periodontitis and diabetes have some common pathogenesis. Diabetes can initiate and worsen the periodontitis and also periodontitis can have effect on the pathogenesis of the diabetes. Thus understanding the pathogenesis of both the conditions and its effect on each other is important for the proper management.

REFERENCES