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Dr. R Madhumala
Professor and Head of the Department, Department of Periodontics, Vinayaka Missions Sankarachariyar Dental College, Salem, Tamil Nadu, India

Dr. Sajini Raj C
Post Graduate, Department of Periodontics, Vinayaka Missions Sankarachariyar Dental College, Salem, Tamil Nadu, India

Correspondence
Dr. R Madhumala
Professor and Head of the Department, Department of Periodontics, Vinayaka Missions Sankarachariyar Dental College, Salem, Tamil Nadu, India

Diabetes: A slow poison to periodontia

Dr. R Madhumala and Dr. Sajini Raj C

Abstract
Periodontitis is a common chronic inflammatory disease characterised by destruction of the supporting structures of the teeth, the periodontal ligament and alveolar bone. Epidemiological data confirm that diabetes is a major risk factor for periodontitis; susceptibility to periodontitis is increased by approximately threefold in people with diabetes. There is a clear relationship between degree of hyperglycaemia and severity of periodontitis. There is emerging evidence to support the existence of a two-way relationship between diabetes and periodontitis, with diabetes increasing the risk for periodontitis, and periodontal inflammation negatively affecting glycaemic control. Incidences of macro albuminuria and end-stage renal disease are increased two-fold and threefold, respectively, in diabetic individuals who also have severe periodontitis compared to diabetic individuals without severe periodontitis. Treatment of periodontitis is associated with HbA1c reductions of approximately 0.4%. Oral and periodontal health should be promoted as integral components of diabetes management.

Keywords: Diabetes, hyperglycemia, inflammation, periodontitis

Introduction
Periodontal diseases are a group of inflammatory diseases of the gingiva, which includes gingivitis in which the inflammation is confined to the gingiva, and is reversible and periodontitis in which the inflammation extends to the deeper tooth-supporting tissues and results in tissue destruction and alveolar bone resorption [1]. Periodontitis may also lead to tooth loss. Tissue destruction in periodontitis results in breakdown of the collagen fibres of the periodontal ligament, resulting in the formation of a periodontal pocket between the gingiva and the tooth. 'Pocketing' is not evident on simple visual inspection, and assessment using a periodontal probe is essential. Periodontitis is a slowly progressing disease but the tissue destruction that occurs is largely irreversible. In the early stages, the condition is typically asymptomatic; it is not usually painful, and many patients are unaware until the condition has progressed enough to result in tooth mobility. Advanced periodontitis is characterised by gingival erythema and oedema, gingival bleeding, gingival recession, tooth mobility, drifting of teeth, suppuration from periodontal pockets, and tooth loss.
Periodontitis is therefore a highly prevalent, but largely hidden, chronic inflammatory disease. Furthermore, it has negative and profound impacts on many aspects of daily living and quality of life, affecting confidence, social interactions and food choices [2]. Smoking is a major risk factor; it significantly increases risk for periodontitis and severity of the condition [3, 4]. Other risk factors for periodontal diseases include diabetes, conditions associated with compromised immune responses (e.g. HIV), nutritional defects, osteoporosis, medications that cause drug induced gingival overgrowth (e.g. some calcium channel blockers, phenytoin, cyclosporin), genetic factors (as yet poorly defined), and local factors (e.g. anatomical deficiencies in the alveolar bone) [3].

Associations between Diabetes and Periodontitis
Diabetes has been unequivocally confirmed as a major risk factor for periodontitis [5, 6]. The risk of periodontitis is increased by approximately threefold in diabetic individuals compared with non-diabetic individuals [7]. The level of glycaemic control is of key importance in determining increased risk. The importance of diabetes as a major risk factor for periodontitis became apparent in the 1990s in a number of cross-sectional and longitudinal studies investigating the Pima Indian population. The prevalence and incidence of periodontitis were greater in Pima Indians who had type 2 diabetes mellitus compared with those who did not[8, 9], with an approximately threefold increased risk for periodontitis [10].

The majority of research has focused on type 2 diabetes mellitus as a risk factor for periodontitis, probably because both diseases have historically tended to develop in patients in their 40s and 50s. However, type 1 diabetes mellitus also increases the risk of periodontitis, and all patients with diabetes (including children and young adults) should be considered to be at increased risk of periodontitis.

Pathophysiology

Periodontal disease is a microbe-initiated chronic inflammatory disease, in which dysregulated immune inflammatory processes are responsible for host tissue destruction, and ultimately tooth loss. This bacteria-initiated immune response stimulates the production of secondary mediators, which amplify the inflammatory response. The presence of these cytokines reduces the ability to repair damaged tissue by cells, and the bacterial products and this inflammatory cascade stimulate osteo clastogenesis, leading to alveolar bone destruction.

The oral cavity provides a nidus for bacterial dispersion into the blood stream. In most instances, these bacteremias are short-lived and transient, and are managed by the innate body defenses. However, on occasions they may lead to the seeding of organisms in different organs, leading to chronic infections. Toxins secreted by the pathogenic oral bacteria may either provoke direct damage to the host tissues or may cause indirect damage by activating host defenses. These bacterial toxins may cause systemic effects as well as local cytotoxic effects at the periodontal tissues.

The inflammatory response is characterized by secretion of host-derived mediators of inflammation and tissue breakdown. Interleukin-1β, interleukin-6, prostaglandin E2 (PGE2), tumor necrosis factor alpha (TNF-α), receptor activator of nuclear factor κB ligand (RANKL), and matrix metalloproteinases (MMP-8, MMP-9, and MMP-13) are the most commonly implicated mediators of inflammation [8]. Oral bacteria can damage periodontal tissues through the action of matrix-degrading enzymes. Tissue destruction occurs in periodontitis in the form of breakdown of the collagen fibers of the periodontal ligament, resulting in the formation of a periodontal pocket between the gingiva and the tooth. The pockets deepen as a result of the further destruction of fibers and the resorption of the alveolar bone also progresses with the progressing attachment loss.

Hyperglycemia increases the concentration of glucose in the saliva and the gingival crevicular fluid. This leads to proliferation of bacteria in the oral cavity. Hyperglycemia itself has also an indirect adverse effect, stimulating the immune system's cells to release inflammatory cytokines. Elevated levels of proinflammatory mediators in the periodontal pockets result in osteoclastic destruction. Diabetic microangiopathy, impaired immune response, and a lower resistance to infections contribute to the development of periodontitis in poorly controlled diabetics.

The continuous exposure of collagen fibers in the supporting periodontal ligaments to aldose sugars induces their nonenzymatic glycation and oxidation. This glycation leads to changes in the physical properties of these molecules, reducing collagen solubility and increasing the degradation of connective tissues. This results in accelerated degradation of both connective tissue and bone

Effect of Periodontitis on Diabetes Control

Diabetes and periodontitis are complex chronic diseases with an established bidirectional relationship. There is a long-established evidence that hyperglycemia in diabetes is associated with adverse periodontal outcomes. [9, 10]. Periodontitis may stimulate systemic inflammation, the oral cavity providing a means for the entry of periodontal organisms and their virulence factors into the circulation.

Inflammation-mediated oxidative-stress pathways and advanced glycation end products (AGEs)-receptor for AGEs (RAGE) interactions provide plausible links in the periodontitis to diabetes direction. The periodontium is a highly vascular tissue and any inflammation there may serve as a gateway to the systemic circulation for bacterial products and locally produced inflammatory mediators. The infectious challenge contributes to insulin resistance by modification of insulin receptor substrate-1 by serine phosphorylation, altered adipocyte function with increased production of free fatty acids, and diminution of endothelial nitric oxide production. The process may further alter pancreatic β-cell function, either acting directly or through stimulation of free fatty acid production.

Severe periodontitis is associated with increased hemoglobinA1c (HbA1C) in individuals with type 2 diabetes [11]. The treatment of periodontitis reduces the inflammation burden and may result in better glycemic management [12]. However, there have been conflicting views with a recent trial demonstrating little effect of nonsurgical management on improvement of HbA1c [13]. The effect of periodontitis management may lead to minor reductions in glycated hemoglobin but the effects are not maintained over a longer duration [14].

Conclusion

Diabetes and periodontitis are the two most commonly encountered chronic diseases today. Controlling diabetes (i.e. improving glycaemic control) is likely to reduce the risk and severity of periodontitis. Furthermore, evidence suggests that resolution of periodontal inflammation can improve metabolic control (with reported HbA1c reductions of approximately 0.4%), though large, multi-centre, randomised controlled trials are needed to further validate these findings. Thus, a routine oral examination should be included for every diabetic patient.

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