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Diabetes And Periodontal Diseases

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ABSTRACT:

Periodontitis is a chronic inflammatory disease affecting the tooth supporting structures, which requires life-long management involving self-care by the affected individuals, and professionally delivered care from dental professionals. Diabetes is a major risk factor for the development of periodontal disease in certain populations. Type 1 (formerly insulin-dependent diabetes mellitus) and type 2 diabetes have been shown to be major risk factors for the development of periodontal disease in certain populations. Type 2 diabetes is usually diagnosed after the age of 30 years and is observed frequently as part of a multifaceted syndrome that includes obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. This report is to describe the clinical characteristics of periodontal disease and the impact of type 2 diabetes on these parameters.

KEY WORDS: Oral hygiene, Diabetes mellitus, Periodontal disease

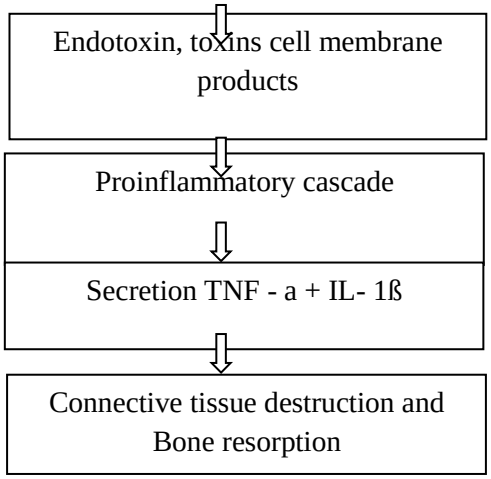
INTRODUCTION:

Diabetes mellitus (DM), a chronic metabolic disease characterized by hyperglycemia, is often attributed to environmental and genetic factors. The prevalence of DM has risen dramatically in recent years, resulting in a rapid increase of diabetic patients. Asia in particular has the highest prevalence of diabetes in the world. Countries exhibiting the fastest rate in diabetic population growth include India and China, among many other developing countries. Periodontal diseases can be divided into gingivitis and periodontitis[1]. Periodontitis is known as a chronic infectious disease of tissues surrounding the teeth which is induced by microorganisms. Periodontitis is a disease characterized by periodontal pocket formation, loss of connective tissue attachment, alveolar bone resorption, and gingival inflammation, ultimately resulting in tooth loss[1]. Diabetes is a major risk factor for the development of periodontal disease in certain populations[4]. Diabetes and periodontitis are complex chronic diseases with an established bidirectional relationship[2]. However, convincing evidence is still lacking on whether the therapeutic remedy for periodontal disease (such as antibiotic treatment) may achieve optimal glycemic control in diabetic patients[1].

Periodontal Infection:

Infections of periodontal origin are chronic, mostly Gram-negative, not only af fecting the tissues surrounding the tooth, but also constituting a systemic challenge to immunocompetent cells and cells active in the inflammatory cascade. Periodontopathic organisms exhibit a number of virulence factors that enable them to evade neutrophil clearance and establish themselves as chronic subgingival inhabitants[3]. Among these, the most relevant factors are bacterial lipopolysaccharide (LPS, endotoxin), lipoteichoic acids, toxins, proteinases, and short-chain fatty acid, capsule and cell membrane products. Interaction of any of these bacterial products with mononuclear phagocytic cells

results in activation of a catabolic inflammatory cascade, with synthesis and secretion of mediators including primarily IL-lß, PGE2, TNF-a, and IL-6. The connective tissue destruction and alveolar bone resorption occurring in periodontal disease result mostly from an "infection-mediated" pathway of cytokine upregulation[3].



Schematic model of the pathogenesis of periodontal disease: "infection-mediated." (Adapted from Grossi et al, 1998)

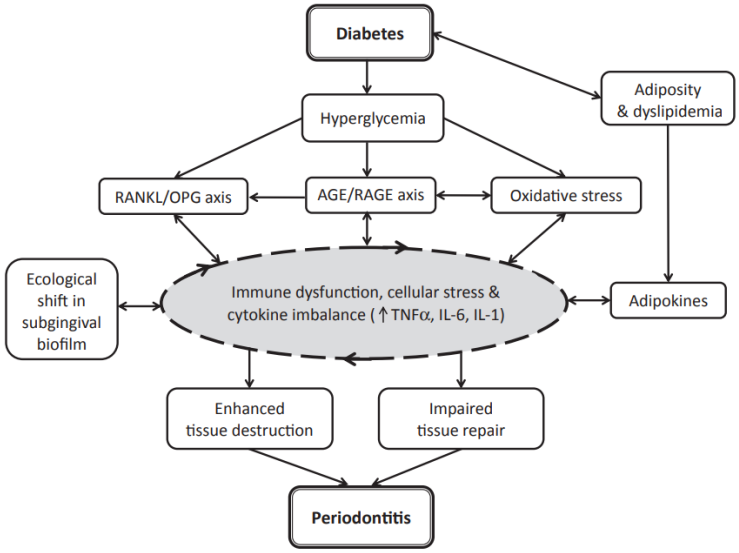
Infections And Diabetes Mellitus:

Acute infections have been reported to alter the endocrinologic-metabolic status of the host, thus leading to difficulty in controlling blood sugar levels. Bacterial infections decrease insulin-mediated glucose uptake by skeletal muscle and produce whole-body insulin resistance. Acute endotoxemia and cytokine production, mostly TNF-a and IL-lß, induce insulin resistance and decreased insulin action[3]. TNF-a has been suggested as the mediator of insulin resistance in infection by suppressing insulin induced tyrosine phosphorylation of insulin receptor substrate-1 (IRS-1), thus impairing insulin action[6]. Infections, as they lead to insulin resistance and poor metabolic control in diabetes, are of great concern since it häs been shown that hyperglycemia and poor metabolic control result in increased diabetic complications such as retinopathy, nephropathy, and neuropathy[7]. Periodontal disease, a chronic Gram-negative infection, has been shown to be associated with poor glycemic control in a longitudinal study of diabetic Pirna Indians,suggesting that periodontal infections may indeed, as do other infections, complicate diabetes status[8]. Gram-negative infection of periodontal origin may induce insulin resistance, hence contributing to hyperglycemia and complicating the metabolic control of diabetes[3].

Mechanism Involved In The Pathogenesis Of Periodontitis In Diabetes:

There are promising data on certain mediators such as IL-1b, IL-6, TNFa and emerging data on RANKL and OPG; these are likely to have a central role in the pathogenesis of periodontitis in diabetic patients. A major challenge will be to gain a holistic understanding of the role of the numerous molecules with action relevant to disease pathogenesis[5]. Diabetes is the pathological consequence of a number of physiological changes and the resulting metabolic dysregulation, hyperglycaemia and chronic inflammation potentially impact on tissue integrity and repair[5].The hyperglycaemic state that characterizes diabetes has several deleterious effects. It drives the formation of irreversible advanced glycation end-products (AGEs) and the expression of their chief signalling receptor RAGE. This interaction, in turn, leads to immune cell dysfunction, alters phenotype and function of other key cells in the periodontium, and contributes to cytokine imbalance with increased generation of certain pro-inflammatory cytokines. Hyperglycaemia also contributes to enhanced levels of reactive oxygen species (ROS) and a state of oxidative stress, both directly and indirectly through

the AGE/RAGE axis, promoting quantitative and qualitative shifts in cytokine profiles[5]. Finally, hyperglycaemia modulates the RANKL/OPG ratio, again directly and indirectly via the AGE/RAGE axis, tipping the balance towards enhanced inflammation and destruction. All the above, complemented by the effects of ecological shifts in the subgingival biofilm and the circulating adipokines generated due to diabetes-associated adiposity and dyslipidaemia, drive this vicious cycle of cellular dysfunction and inflammation. The end result is a loss of equilibrium where enhanced periodontal tissue destruction and impaired repair ensue, leading to accelerated and severe periodontitis[5].



Network of potential mechanisms involved in the pathogenesis of periodontitis in diabetes. (Adapted from Taylor et al, 2013)[5]

Hyperglycemia And Complications Of Diabetes Mellitus:

Prolonged hyperglycemia is the primary factor for the development of diabetic complications including retinopathy, nephropathy, and neuropathy[9]. The most common biochemical reaction is hyperglycemia-mediated formation of non enzymatic advanced glycation end products (AGEs) on macromolecules. AGEs are known to be glucose-derived, chemically irreversible, compounds that form slowly as a function of blood glucose concentration[9]. AGEs accumulate in diabetic tissues and recently have been demonstrated as it accumulated in gingiva of diabetic patients. The chronic stimulus for matrix proliferation is increased local production of growth factors such as TNF-a, IL-1, and platelet-derived growth factor (PDGF). Macrophages have high-affinity receptors for the structural element on AGE modified proteins. Binding of the AGE modified protein to the macrophage receptor (RAGE)[10] initiates a cycle of cytokine up regulation, with synthesis mostly of IL-1 and TNFa. These cytokines have the ability to bind to a number of cells active in normal tissue remodeling. However, if synthesis and secretion are increased, a degradative cascade is triggered, resulting in connective tissue degradation, proliferation, and focal thrombosis[9].

Effect Of Periodontal Treatment On Control Of Diabetes Mellitus:

Studies shows that periodontal treatment would improve diabetes control if an individual had poorly controlled diabetes and conversely some studies states it would have little or no effect if an individual had well controlled diabetes. A recent review of available literature concluded that "periodontal therapy may not be associated with improved glycemic control in diabetic patients who are relatively well controlled, but may result in improved metabolic control in some individuals with poorly

controlled diabetes[3]. On conclusion the effect of periodontal treatment on diabetes metabolic control is dependent on the mode of therapy: mechanical versus mechanical combined with systemic antibiotics. When mechanical periodontal treatment alone is provided, regardless of the severity of periodontal disease or degree of diabetes control, the treatment outcome is strictly improvement in periodontal status or a local effect. On the contrary, when systemic antibiotics are incorporated with mechanical therapy, a systemic effect is seen as well, that is there is an improvement in diabetes control measured as a reduction in glycated hemoglobin or reduction in insulin requirements[3].

Systemic Effect Of Periodontal Treatment On Diabetes:

The reduction in HbAlc following doxycycline treatment48'49 and reduction in insulin requirement after penicillin administration are likely the result of the antimicrobial effect of these drugs[11]. It has been reported that tetracycline caused the retention of membrane-associated TNF-a, thereby preventing the release of TNF-a from the monocyte membrane[12]. A potential therapeutic role for tetracyclines and their derivatives has been proposed in the treatment of Periodontitis by inhibiting tissue-destructive enzymes[12].

CONCLUSION:

Periodontal disease contain several chronic bacteria which complicates diabetes mellitus. The severity of Periodontitis was predominantly higher in diabetic patient than non diabetic. A better understanding of this bidirectional relationship provides more appropriate treatment for these patient. From the available preliminary evidence, it is recommended that control of the chronic Gram-negative periodontal infection with systemic antibiotics as an adjunct to mechanical periodontal therapy should be part of the standard care of the diabetic patient who has Periodontitis.

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