Diabetes and periodontal disease

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ABSTRACT

Diabetes mellitus is a systemic disease characterized by increased blood glucose levels and abnormalities of lipid metabolism due to absence or decreased level of insulin. It affects all the body organs and their functions either directly or indirectly. Every dentist should have a basic understanding of the etiopathogenesis, oral and systemic manifestations of this disease. The periodontal diseases are a consequence of extension of the gingival inflammation into the underlying supporting structures of the periodontium, initiated by the presence of plaque and its products on the surfaces of the teeth and the adjoining structures. The progression of periodontal disease is influenced by variety of factors like microorganisms, host response, systemic background, and genetic makeup of the host. Amongst them, diabetes mellitus tops the list. Diabetes and periodontitis influence the clinical outcome of each other and control of both influences the clinical improvement of each.

KEY WORDS: Diabetes mellitus, periodontitis, vascular changes

Diabetes mellitus (DM) is a term applied to a heterogeneous group of disorders that share the characteristic of altered glucose tolerance (or) impaired lipid and carbohydrate metabolism. It develops as a result of either deficient production of insulin or impaired use of insulin. It can be divided into two types: Type I or insulin-dependent DM and Type II or non–insulin-dependent DM. Type I is caused by the destruction of insulin producing B-cells of pancreas. Type II is due to impaired insulin function rather than deficiency.[1]

Type I usually develops before the age of 30 and the patient is totally dependent on the supply of insulin. Type II has its onset later in life and is usually managed through diet modification in combination with hypoglycemic agents. The general signs and symptoms of diabetes are a direct result of hyperglycemia, and the signs and symptoms are polyuria, polydipsia, and polyphagia, weakness, and fatigue with pruritus. The signs and symptoms may be reversible with early diagnosis and therapy.

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History

Description of DM dates back to 1500 years BC by the Egyptians. In 400 BC, Sushrutha described it as honey urine. In 1869, Langerhans discovered the islets in pancreatic tissue. In 1921, Banting and Best described hypoglycemic substances isolated from pancreas. In 1955, oral hypoglycemic agents were introduced. Gruner first reported the association between diabetes and periodontal disease. In 1928, Williams stated that gingivitis and periodontitis among diabetic patients are modified. He termed it "diabetic periodontoclasia." Glickman in 1946 found in an experimental animal study that periodontal disease in diabetic animals was not different histologically and was thus not a unique clinical entity.

Relationship of Diabetes and Periodontitis

Periodontitis is stated to be the sixth complication of diabetes.[2] Prevalence of severe periodontitis in diabetics as compared to non-diabetics has been found to be 59.6%:39%.[3] Majority of well-controlled studies show a higher prevalence and severity of periodontal disease in diabetics than in non-diabetics with similar local irritation including greater loss of attachment, greater alveolar bone loss, increased bleeding on probing, and increased tooth mobility resulting in tooth loss.

The probable influence of diabetes on the onset and development of periodontal disease has been studied by

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many investigators.[4] Ray and Orban[5] observed that the basic structural changes in the diabetic periodontium are the degeneration of tissues and the presence of calcified bodies in and around small blood vessels of the gingiva.

Most striking changes in uncontrolled diabetes are reduction in defense mechanism and increased susceptibility to infection leading to destructive periodontal disease. According to the opinion of many clinicians, periodontal disease in diabetics follows no consistent pattern. Very severe gingival inflammation, deep periodontal pockets, rapid bone loss, and frequent periodontal abscess often occur in diabetic patients with poor oral hygiene. Numerous studies have shown increased prevalence and severity of periodontal disease in Type I diabetics.[6] Diabetes has impaired defense mechanisms involving micro- and macro-vasculatures. The increased susceptibility to infection and reduced healing capacity with altered collagen metabolism may explain the increased level of periodontal destruction.

Diabetes Associated Factors that Increase the Severity of Periodontitis

Effect of diabetes on periodontal flora

Insulin-dependent diabetic patients with periodontitis have been reported to have subgingival flora composed mainly of capnocytophaga, anaerobic vibrios, and *Actinomyces* species. *Porphyromonas gingivalis*, *Prevotella intermedia*, and *actinomycetemcomitans* present in non-diabetics are present in low numbers in diabetics. Increased glucose level in crevicular fluid in diabetics may favor the growth of some microbial species. Gingival fluid in diabetics contains reduced level of cyclic adenosine monophosphate (cAMP) compared to that in non-diabetics. Decreased level of cAMP could be another potential mechanism in modifying the severity of periodontal disease.[7]

Defect in host response

Neutrophil impairment leads to increased susceptibility to periodontitis in diabetics. Hyperglycemia induces decreased chemotaxis, phagocytosis, and intracellular bacterial activity in diabetics.

Oliver *et al.*[8] showed that large number of Poly morpho nuclear cells were present in inflamed gingival crevices of poorly controlled diabetics.

In juvenile diabetes, decreased responsiveness of PMNs to chemotactic stimuli has been demonstrated. Impaired chemotaxis can probably develop as a result of numerous biochemical alterations in the body mechanism. Hawley[9] found that saturated fatty acid produced in high concentration caused inhibition of chemotaxis. Diabetics with severe periodontitis have significant impairment of PMN chemotaxis, which possibly facilitates the development of periodontal disease.

Defective phagocytosis

Impairment of neutrophil phagocytosis is observed in poorly controlled diabetics. PMNs isolated from diabetic patients with ketoacidosis have shown poorly ingested staphylococci. Reduced neutrophil hydrophobicity has been shown to be accompanied by decreased phagocytic activity.

Defects in PMN function have been considered as a potential cause of bacterial infection in diabetic individuals. Speculative possibilities are decreased antibody production, interference with bactericidal action of blood, and presence of lipid deposits causing vascular insufficiency in small blood vessels.

Inflammatory response

Peripheral blood monocytes isolated from diabetics with periodontal disease secrete significantly more prostaglandin E2 than those without the disease. Shapira *et al.*[10] observed a prostaglandin E2 hypersecretory response to lipopolysaccharides in monocytes from patients with early onset form of the disease.

Collagen defect

Golub *et al.*[11] found in experimentally induced diabetes, impaired production of bone matrix component by osteoblasts, decreased collagen synthesis by gingiva and periodontal ligament fibroblasts, and increased collagenase activity. Collagen from diabetic patients has been reported to be more insoluble and resistant to digestion, directly impairing degradation and remodeling. Increased crevicular fluid collagenolytic activity and decreased synthesis of collagen by gingival fibroblasts was found in diabetic patients. Administration of insulin prevents the onset and corrects the defective collagen production.

Vascular changes

Vascular changes have generally been associated with genetic predisposition, long duration, and poor control of hyperglycemia. The basement membrane protein undergoes non-enzymatic glycosylation when subjected to hyperglycemic condition. The gingival capillaries of diabetic patients have greater basement membrane thickness. Campbell[12] found the width of basement membrane to be related to age of the patient.

Impaired wound healing

Poor wound healing has been a common finding in diabetics and is characterized by a decrease in the amount of wound collagen and lowered tensile strength. Decreased granular tissue and reduced amount of protein and DNA have also been reported in diabetic rat. The defective wound healing may be due to non-enzymatic glycosylation of collagen and other proteins during the period of hyperglycemia. Impaired growth factor secretion may be a key mechanism for impaired

wound healing in diabetics. The monocyte is the principal cell involved in wound debridement and growth factor secretion. The shift in monocyte from a reparative regenerative cell to an inflammatory phenotype may represent a common underlying mechanism for both impaired wound healing and exaggerated inflammatory response in diabetics.

Dental management of diabetic patients

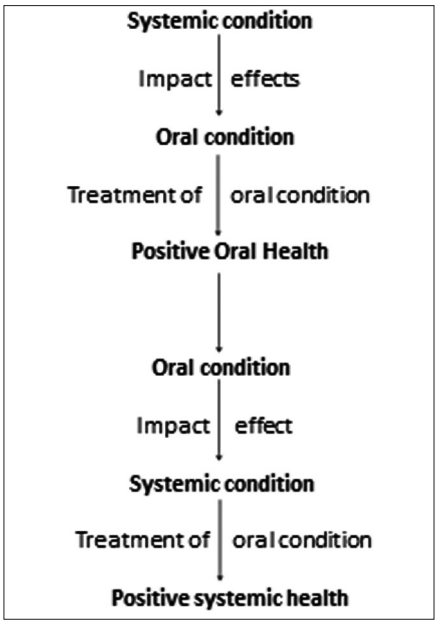
The chances of complications are more in poorly controlled or uncontrolled diabetics than in controlled diabetes. It has been observed that contain granulocyte abnormalities like phagocytosis improved with better diabetes control. It has been shown that glycemic control reduces the incidence of complications. There are more chances of infection in uncontrolled diabetics than in controlled diabetics. Hyperglycemia triggers a chain of events which result in the risk of infection and delayed healing. It has been shown that uncontrolled diabetics have more bone loss, severe bleeding, and severe periodontal disease than controlled individuals.

Miller[7] studied the effect of periodontal therapy in diabetics and found that there was reduction in glyHbAc in patients who underwent periodontal therapy with systemic doxycycline and chlorhexidine rinse. The removal of pathogens by treatment leads to a decrease of inflammation, which in turn reduces insulin resistance; this in turn reduces the sugar level. The absence of inflammation causes a decrease in adrenaline level, which in turn regulates anti-insulin action, leading to decrease in sugar level. These facts together lead to an overall reduction in the dosage of insulin or oral hypoglycemic drugs.

Conclusion

Diabetes is a systemic disease which is a serious oral co-morbidity.[2] Most oral complications occur in uncontrolled diabetics, involving the periodontium, the calcified tissue, and the oral mucosa. Therefore, poor metabolic control, periodontal disease, dental caries, xerostomia, and fungal infections go hand in hand. Hence, there is a need for appropriate health education as good oral health is important for diabetic individuals.

The relationship between diabetes and periodontitis creates the following paradigm.



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