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DIABETES AND PERIODONTAL HEALTH

Dental Science

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ABSTRACT

Periodontitis is a common chronic inflammatory disease characterized by destruction of the supporting structures of the teeth. The management of diabetes is complex and the prevention of cardiovascular and microvascular disease, through early detection and management of complications, which are the key components.

KEYWORDS

Diabetes mellitus, periodontitis, wound healing

What is periodontal disease?
It can be defined as the destruction of the tissues that support the tooth by accumulation and maturation of oral bacteria on the teeth.

Two major entities can be seen in a periodontal disease (gingivitis and periodontitis). Gingivitis is characterized by irreversible inflammation of periodontal tissues whereas periodontitis refers to destruction of periodontium leading to tooth loss.[1,2] the pockets deepen as a result of destruction of fibers of periodontal ligament (attachment loss) [fig 1].

Diabetes mellitus (DM) is a term referred to a heterogeneous group of disorders sharing the characteristic of altered glucose tolerance/ impaired lipid and carbohydrate metabolism. It is divided into two types: Type 1 (insulin dependent) and Type 2 (non-insulin dependent). Type 1 is caused by destruction of insulin producing B-cells of pancreas. Type 2 is due to impaired insulin function rather than deficiency.[3]

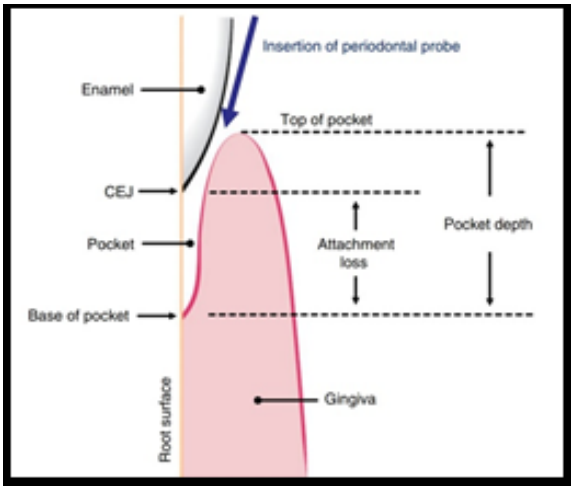

Fig 1: Periodontal pocket in periodontitis

Etiology and pathogenesis
The main etiological factors comprises of microorganisms in combination with individual host susceptibility and environmental factors.

Accumulation of plaque on teeth produces gingivitis, but the host susceptibility defines the degree of inflammation and destruction of the alveolar bone supporting teeth.[4]

Various studies have shown how gingival inflammation can be modulated by numerous conditions such as systemic diseases, steroid hormones variations, nutritional deficiency, drug intake, diabetes, tobacco smoking, resulting in an increased response to bacterial plaque accumulation.[5]

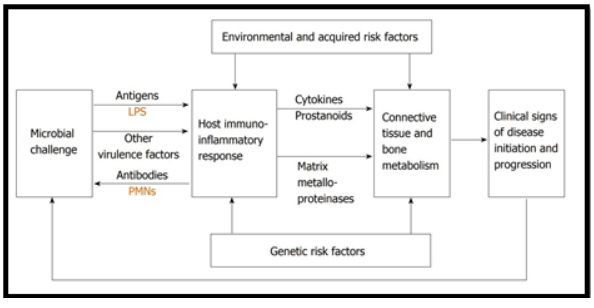

Fig 2: Etiology and pathogenesis[6]

Table 1: Diabetes associated changes in periodontal tissues

	Type 1 diabetes mellitus	Type 2 diabetes mellitus
Loss of attachment	Present	Present
Bone loss	Increased alveolar bone loss	Increased alveolar bone loss
Neutrophils	Phagocytic activity decreased Chemotaxic activity decreased	Apoptosis delayed Chemotaxic activity decreased
Macrophages	Increased	Increased
Matrix metalloproteinases	Matrix metalloproteinases-8, -9 increased	Matrix metalloproteinases-8, -9 increased
Cytokines	Interleukin-beta, tumor necrosis factor-alpha increased	Interleukin-beta; interleukin -4, -6, -8, -10, -17; interferon gamma, interleukin-8; tumor necrosis factor-alpha increased.
Prostaglandins	Prostaglandin E2 increased	Prostaglandin E2 increased

CLINICAL MANIFESTATIONS [7]

A. Clinical features of gingival inflammation(gingivitis):
i. Enlarged gingival contours (due to edema/fibrosis)
ii. Color transition (red/bluish red hue)
iii. Elevated sulcular temperature
iv. Bleeding on probing increased gingival exudate.
B. Clinical features of periodontitis:
i. Clinical attachment loss
ii. Alveolar bone loss
iii. Periodontal pocket formation
iv. Gingival inflammation
v. Gingival hyperplasia
vi. Gingival recession
vii. Mobility of tooth

DIAGNOSIS OF PERIODONTAL DISEASE [8]

Clinical assessment requires periodontal probing to evaluate:

1. Probing depth: distance a periodontal probe penetrates into a periodontal pocket from gingival margin to it's bottom.
2. Clinical attachment level: distance from CEJ to bottom of the pocket.
3. Bleeding on probing: bleeding on insertion of probe to evaluate subgingival inflammation.
4. Tooth mobility

Radiographic assessment shows alveolar bone loss. In a healthy person, 1-2mm bone is available below the tooth crown.

INTER-RELATIONSHIP BETWEEN PERIODONTITIS AND DIABETES

Investigations have suggested associations between periodontitis and various systemic diseases [1][17,18] such as cardiovascular disorders,[9,10] respiratory disorders,[11,12] osteoporosis,[13,14] immunodeficiencies[15] and diabetes mellitus.[16]

Various authors have demonstrated a two-way relationship between diabetes and periodontitis, with more severe periodontal tissue destruction in diabetic individual with poor glycemic control and periodontal disease.[17,18]

DIABETES ASSOCIATED FACTORS THAT INCREASES THE SEVERITY OF PERIODONTITIS

Effect of diabetes on periodontal flora

Insulin dependent diabetic patients with periodontitis have been reported to have subgingival flora consisting of mainly capnocytophaga, anaerobic vibrios and actinomyces species whereas, porphyromonas gingivalis, prevotella intermedia and actinomycetemcomitans seen in a healthy individual are low in numbers in diabetics. Increased level of glucose and decreased level of cAMP in gingival fluid could be a potential cause of periodontal disease.[19]

Defect in host response

Impairment of neutrophil may lead to increased susceptibility to periodontitis in diabetics. On the other hand hyperglycemia decreases chemotaxis, phagocytosis and intercellular bacterial activity in diabetics. Oliver et al.[20] found that poly morphonuclear cells were present in large number in inflamed gingival crevices of a poorly controlled diabetic individual. Hawley [21] showed that saturated fatty acid produced in high concentration causes inhibition of chemotaxis.

Inflammatory response

Significantly more amount of prostaglandins E2 are secreted from peripheral blood monocytes of periodontally compromised diabetic patient. In patients with early onset of the disease Shapira et al.[22] observed hypersecretory response of prostaglandin E2 to lipopolysaccharides in monocytes.

Collagen defect

Collagen from diabetic patients has been found to be more insoluble and resistant to digestion, causing a defect in degradation and remodeling. Increased collagenolytic activity and decreased synthesis of collagen in gingival crevicular fluid is reported in diabetic patients. Insulin administration prevents the onset and corrects the defective collagen production.

Defective phagocytosis

Neutrophil phagocytosis impairment is seen in poorly controlled diabetics. PMNs isolated from diabetic patient with ketoacidosis have demonstrated poorly ingested staphylococci, which may be the cause of bacterial infection. Possible causes are decreased antibody production, interference with bactericidal action of blood and presence of lipid deposits resulting vascular insufficiency in small blood vessels.

Vascular changes

Vascular changes are subject to genetics, long duration and uncontrolled hyperglycemia. The gingival capillaries of diabetic patients have greater basement membrane thickness. Campbell [2][12][23] reported the width of basement membrane to be associated to the age of the patient.

Impaired wound healing

Delayed wound healing has been found to be the most common finding in diabetes, characterized by a decrease in the amount of wound collagen and lowered tensile strength. It is due to non-enzymatic glycosylation of collagen and other proteins during the period of hyperglycemia. Besides, impaired growth factor secretion may be a key mechanism for impaired wound healing process. The principal cell involved in wound debridement and growth factor secretion is the monocyte. The conversion of monocyte from a reparative regenerative cell to an inflammatory phenotype may be the underlying cause mechanism for impaired wound healing and exaggerated inflammatory response in diabetics.

Dental management in diabetic patient

It has been observed that the chances of complications are more in uncontrolled diabetic individuals. Hyperglycemia triggers a chain of events which results in the risk of infection and delayed wound healing. Uncontrolled diabetics, has more bone loss, severe bleeding and severe periodontal disease than seen controlled patient.

Miller[19] in his study reported the effect of periodontal therapy in diabetic patients and found that there was reduction in glyHbAc in patients who underwent periodontal therapy with systemic doxycycline and chlorhexidine rinse. This study showed that the removal of pathogens by treatment leads to decrease in inflammation, which in turn reduces insulin resistance, therefore reducing sugar level. In addition to it, reduced inflammation leads to degraded level of adrenalin, which subsequently leads to reduced sugar level. The following factors, together reduces the dosage of insulin or oral hypoglycemic drugs.

Relationship between diabetes and periodontal health shows a two-way pathway, therefore systemic condition is directly associated with oral health and vice versa.

PERIODONTAL TREATMENT IS ASSOCIATED WITH IMPROVED GLYCAEMIC CONTROL

Several meta-analyses have confirmed that effective periodontal therapy can result in reduced HbA1c. The first reported on ten interventional studies with a combined population of 456 patients; the authors identified a weighted mean reduction in HbA1c of 0.66% as a result of periodontal therapy (though this failed to achieve statistical significance).[25] In 2008, a meta-analysis of nine studies involving 485 patients reported a significant reduction of HbA1c of 0.46% following periodontal treatment.[26] In 2010, a meta-analysis of five studies involving 371 patients also reported a significant weighted mean reduction in HbA1c of 0.40% over a follow-up period of 3–9 months after periodontal therapy .[27] The authors of these meta-analyses all commented on the heterogeneity of the data, different methodologies having being used in the different studies. Most recently, the Cochrane Collaboration has reported on studies that investigated the relationship between periodontal treatment and glycaemic control in people with diabetes .[28]Three studies were included in this meta-analysis which reported a significant reduction in HbA1c of 0.40% 3–4 months after conventional periodontal therapy (Fig. 5). The findings of these meta analyses are supported a recent population-based study of over 5,000 individuals with diabetes [29] reporting that patients who received at least one episode of periodontal surgery (an intense form of periodontal treatment, not routinely undertaken in all patients with periodontitis) had HbA1c levels that were 0.25% lower than patients who did not undergo periodontal surgery.[30-32] Taken collectively, the evidence supports the notion that improvements in metabolic control can be anticipated following effective treatment of periodontitis (although there are few studies available, and some studies lack power). The mechanisms by which this occurs are not yet clear, but probably relate to reduced systemic inflammation (e.g. reduced serum levels of mediators such as TNF-α and IL-6) following the treatment and resolution of periodontal inflammation. Larger randomised trials are warranted to investigate this further. These observations are important because reductions in HbA1c are associated with a reduced risk of diabetes complications. For example, each 1% reduction in HbA1c has been associated with reductions in risk of 21% for any endpoint related to diabetes, 21% for deaths related to diabetes, 14% for myocardial infarction and 37% for microvascular complications .[33]

CONCLUSION

Diabetes and periodontal condition go hand in hand, as diabetes is a systemic disease which is a serious oral co-morbidity.[2]Poor metabolic control, periodontal disease, dental caries, xerostomia and fungal infections are inter-related. However, it has to be kept in mind that the

level of metabolic control and duration of diabetes appear to influence the risk for periodontal disease, with a significant heterogeneity among diabetic individuals. Hence, there is a need for oral health education for diabetic individuals for their proper management. Closer collaboration between medical and dental clinical teams is necessary for the joint management of people with diabetes and periodontitis, contact with dentist is important after the diagnosis of diabetes.

DECLARATION OF PATIENT CONCENT

The authors certify that they have obtained all the appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their/images and other clinical information to be reported in the journal. The patient(s) understand that their name and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be granted.

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CONFLICTS OF INTEREST

There are no conflicts of interest.

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