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Intricate relationship of diabetes and periodontitis

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Abstract

Periodontitis and diabetes mellitus, both are common, chronic diseases having two way relationships with each other. Diabetes mellitus is adversely affecting onset, progression and severity of periodontitis and severe periodontitis leads to compromised glycaemic control of diabetic patients. An attempt is made to know how diabetes mellitus and periodontitis affect each other.

Keywords: Diabetes mellitus, periodontitis

Introduction

Periodontitis is common oral health problem, caused predominately by gram negative anaerobic microorganisms and resulting in severe inflammation with potential for vascular dissemination of microorganisms and their products throughout the body.¹ The worldwide prevalence of periodontal disease ranges from 5 to 20% of adult population.² Diabetes mellitus is complex metabolic disease characterized by chronic hyperglycemia. It is also most prevalent disease affecting 245 million people in the world. Seven million people develop diabetes every year and it is predicted that 366 million patients will have diabetes worldwide by year 2030.³

People with diabetes have increased risk for periodontitis and risk increases with poor glycaemic control. Periodontitis also can have negative impact on glycaemic control. So it is necessary to detect and treat periodontal diseases in diabetic patients, otherwise

management of both conditions may be compromised.⁴

Periodontitis

Periodontitis is an inflammatory disease of supporting tissues of teeth caused by specific microorganisms, resulting in progressive destruction of periodontal ligament and alveolar bone with pocket formation, recession or both. Armitage GC in 1999 gave simplified classification of periodontitis: chronic periodontitis, aggressive periodontitis and periodontitis as a manifestation of systemic diseases.⁵

Chronic periodontitis: It is most common type of periodontitis and most prevalent in adults but can occur in children. It is characterized by frequently present sub gingival calculus, progression is slow to moderate with periods of rapid progression and can be

modified by some associated factors that are systemic diseases like diabetes mellitus and HIV infection.

Chronic periodontitis is further classified as:

Localized form: Less than 30% sites involved.

Generalized form: More than 30% sites involved.

Slight: Clinical attachment loss is of 1 to 2 mm.

Moderate: 3-4 mm attachment loss.

Severe: With attachment loss more or equal to 5mm.

Aggressive periodontitis: It is characterized by rapid attachment loss, bone destruction in clinically healthy patients. In this form disease severity is inconsistent with amount of microbial deposits. It is further sub classified into two forms:

Localized form: Onset of this form is circumpubertal. In this form localized involvement of first molar or incisor with proximal attachment loss on atleast two permanent teeth, including first molar as one of these two teeth, occurs and vigorous serum antibody response develops to infecting microorganisms.

Generalized form: It usually occurs in patients under 30 years but may occur in old age. It is characterized by generalized proximal attachment loss on atleast three teeth other than first molars and incisors. There is poor serum antibody response.

Periodontitis as a manifestation of systemic diseases: This type of periodontitis may be observed as a manifestation of some systemic diseases like acquired neutropenia, leukemia, familial and cyclic neutropenia, Down syndrome, Papillon Lefevre syndrome, histiocytosis, glycogen storage diseases, Cohen syndrome, hypophosphatasia etc.^{6,7,8}

Diabetes mellitus

Diabetes mellitus classified into two types based on underlying pathophysiology that are type 1 and type 2 diabetes.⁹ Type 1 diabetes constitutes about 5-10% of all cases and is due to autoimmune destruction of insulin producing cells in pancreas resulting in total loss of insulin. Insulin facilitates transfer of glucose from bloodstream to body tissues such as muscle, where glucose is utilized for energy production. Patient with type 1 diabetes has no endogenous insulin, glucose is unable to enter target cells, resulting in increased blood glucose level. Patient must take exogenous insulin for survival. Therefore this type of diabetes is called as insulin dependent diabetes.¹⁰

Type 2 diabetes constitutes about 85 to 90 % of all cases. It was previously also called non-insulin-dependent diabetes, or adult onset diabetes. It is due to insulin resistance that is reduced body cell responsiveness to insulin resulting in reduced capacity to transfer glucose from blood stream into cells and so hyperglycemia (elevated blood glucose levels) occurs. In the early stages of disease insulin secretion may be normal, but this can decrease over time, leading to insulin deficiency as well as insulin resistance. Type 2 diabetes is associated with some lifestyle factors such as overweight/obesity , lack of exercise, as well as genetic factors. The treatment of type 2 diabetes includes lifestyle change, weight loss, dietary modification, oral hypoglycemic drugs and, in severe cases, insulin injections. The age of onset of type 2 diabetes was previously typically considered to be in the 40s and 50s, but increasing numbers of cases in younger age groups are now being identified.¹¹

Diabetes and periodontal diseases

Various studies concluded that there is increased risk of periodontitis in diabetic patients. The increased risk of periodontitis is dependent on level of glycaemic control. Therefore in well controlled diabetes with HbA1c (glycated hemoglobin) of around 7% or lower, it has little effect on risk of developing periodontitis and risk increases exponentially as glycaemic control is diminished. Diabetes increases prevalence, extent and severity of periodontitis and net increase of risk for periodontitis is 2-3 times.^{12,13} A cross sectional study concluded that type 1 diabetes increases prevalence of periodontitis by five fold in teenagers¹⁴. Another study confirmed that attachment loss is more prevalent and extensive in diabetic children than in children without diabetes.¹⁵ Type 2 diabetic adult patients have four fold increased risk of progressive alveolar bone loss as compared to adults without diabetes.¹⁶ Another multivariate risk analysis reported that patients with type 2 diabetes have approximately threefold increased risk for developing periodontitis as compared to normal individuals after adjusting for confounding variables such as age, sex and oral hygiene measures.^{17,18}

Effect of diabetes on periodontal disease

Inflammatory response: Periodontal disease is infectious disease produced by microorganisms, initiating immune response, stimulate the production of secondary mediators, which amplify the inflammatory response. These cytokines diminish the ability to repair damaged tissue, and the bacterial

products and this inflammatory cascade stimulates osteoclastogenesis, resulting in alveolar bone destruction.¹⁹ The function of responding cells that are neutrophils, monocytes and macrophages, is altered in many patients with diabetes. The adherence, chemotaxis and phagocytosis of neutrophils are often impaired.²⁰ Neutrophils constitute the first line of host defence. Improper functioning of these cells may prevent destruction of bacteria in periodontal pocket, leading to increased periodontal destruction. In diabetic patients, macrophages and monocytes produce more amount of proinflammatory cytokines and mediators like tumor necrosis factor (TNF) in response to periodontal bacteria which may cause more host tissue destruction.^{21,22} Both blood and gingival crevicular fluid revealed, elevated levels of TNF-, representing both local and systemic hyperresponsiveness of these immune cells. Levels of interleukin 1 were twice in patients with HbA1c level greater than 8% as compared to patients having level of HbA1c less than or equal to 8%. This indicates that hyperglycemia contributes to increased inflammatory response and suggesting association between poor glycaemic control and periodontal destruction.²³ Formation of AGEs (advanced glycation end products) and their interaction with receptor in periodontal tissues, oxidative stress and accumulation of reactive oxygen species, all these also contribute to extensive periodontal inflammation in diabetic patients.⁴

Microflora

Studies have found a relation between glycaemic control and alterations in microflora which may increase the susceptibility of diabetic patients to periodontitis. Counts of Capnocytophaga species were significantly higher in periodontal pockets of diabetic patients as compared to periodontal pockets of healthy people.²⁴ Thorstenseon et al studied several bacterial species in subgingival microflora of type 1 diabetic patients and normal individuals. They found that *P. gingivalis* bacteria was detected in both shallow and deep pockets of diabetic individuals, whereas this bacteria was only present in deep pockets of non diabetic individuals.²⁵ Local environment changes due to salivary alteration and high levels of glucose in GCF (gingival crevicular fluid) may result in changed microflora.²⁶ But some studies reported that pathogens causing periodontitis are not greatly different in diabetic and non diabetic patients.²⁷ These different study results suggested that altered host response to existing periodontal pathogens may be responsible for

more aggressive damage of periodontium in diabetic patients.²⁸

Altered wound healing: Fibroblast is reparative cell in periodontium, does not function normally in high glucose environment.²⁹ In diabetic patients, there is increased production of matrix metalloproteinase enzymes and collagen produced by fibroblast is susceptible to rapid degradation by this enzyme.³⁰ Therefore healing of periodontal tissue in response to microbial destruction may be altered in patients with poor glycaemic control, resulting in increased attachment loss and bone loss.

Effect of periodontitis on diabetes:

Severe periodontitis was associated with high risk of poor glycaemic control, indicating that periodontitis may be compromising diabetic control.³¹ Higher prevalence of diabetic complications such as retinopathy, proteinuria and neuropathy, was found in individuals with advanced periodontitis.^{25,32,33} Chronic periodontal diseases have potential to aggravate insulin resistance and poor glycaemic control whereas periodontal treatment results in decreased inflammation and may decrease insulin resistance.³⁴ A longitudinal study reported that non diabetic individuals with advanced periodontitis at baseline have five times greater increase in HbA1c value over five years as compared to individuals with no periodontitis at baseline.³⁵ Bacteria and bacterial products such as LPs from periodontal pocket, and locally produced inflammatory mediators like TNF- and IL-6 enter into systemic circulation, these aggravate low grade systemic inflammation and worsen glycaemic control and increase development or progression of diabetic complications.^{26, 36,37} TNF- has many metabolic effects such as down regulation of genes related to normal insulin action and directly effect insulin signaling, glucose transport and pancreatic cells.³⁸ It was reported that IL-6 modulates TNF- production and also associated with insulin resistance. IL- participate in the regulation of glucose uptake.³⁹

Diabetes and periodontitis are inextricably linked and adversely affecting each other. Prevalence and severity of periodontitis becomes high in diabetic patients and glycaemic control is important determinant for this. Research reveals various mechanisms such as, alteration in microflora of periodontium, alteration in host response and change in wound healing which are responsible for more aggressive periodontitis in diabetic patients. Periodontitis can also worsen

glycaemic control and increase risk of developing diabetic complications, thus increasing mortality rate in diabetic patients. So oral health becomes more important in diabetic patients and periodontal assessment is also important in non diabetic individuals. Therefore these two diseases need collaborate management by medical and dental health professionals.

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