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Relationship between Diabetes Mellitus and Pulpal Infection & Periapical Diseases

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Abstract:

Diabetes mellitus is a metabolic disorder characterized by relative or absolute insufficiency of insulin and also disturbances of carbohydrate,fat and protein metabolism. Diabetes is fast gaining the status of a potential epidemic in India with more than 62 million diabetic individuals currently diagnosed with the disease. In 2000, India (31.7 million) topped the world with the highest number of people with diabetes mellitus followed by China (20.8 million) with the United States (17.7 million) in second and third place respectively. It is predicted that by 2030 diabetes mellitus may afflict up to 79.4 million individuals in India. The prevalence and the chance of developing periapical lesions were found to be higher in patients with diabetes compared to healthy controls. This article aims to review and increase an awareness of the relationship between pulpal infection; periapical diseases and diabetes mellitus to stimulate research on the subject.

Keywords: Diabetes Mellitus, insulin,periapical lesions,pulpal infection,periapical disease.

INTRODUCTION:

Diabetes mellitus can be broadly classified into Type1 and Type2 diabetes. Type 1 diabetes is characterized by beta cell destruction and absolute insulin destruction. It accounts for only 5-10% of those with diabetes. Type 2 diabetes is also called as insulin resistance diabetes or non-insulin dependent diabetes characterized by insulin deficiency. It accounts for ~90-95% of those with diabetes[1].Type 2 diabetes is linked with obesity:If the circulating level of free fatty acid from adipocytes increases, it inhibits the glucose uptake followed by reduction of glycogen synthesis and glycolysis.This contributes to insulin resistance[2].Diabetes affects our body's immune system thereby causing poor healing and disturbing the mutual immune responses[3].

The oral complications of diabetes mellitus includes: xerostomia, infection, poor healing, increased incidence & severity of caries, candidiasis, gingivitis, periodontal diseases & burning mouth syndromes[4].The patients with diabetes mellitus have a rapidly ageing pulp due to obliterative endartities[5].Many oral diseases have been associated with hyperglycemia such as periodontitis,dental caries etc.

DIABETES MELLITUS IN IMMUNE RESPONSE:

Glucose or its analogues interact with proteins and lipids.The end products of these non-enzymatically catalysed reactions,termed advanced glycation end products(AGE),have been linked to long-term complications of Diabetes.AGEs interact with their receptors(RAGEs) on endothelial cells,smooth cells and in monomolecular phagocytes.In normal states,RAGEs are at a low level,but in hyperglycaemia the expression of RAGEs on critical target cells is significantly enhanced.Accumulation of AGEs and their interaction with RAGEs alter the ability to respond to infection.The alterations include increased vascular permeability,enhanced expression of adhersion molecules on endothelial cells,attraction and activation of macrophages,impaired collagen synthesis,and impaired leukocyte function.From the above it can be inferred that if a periradicular infection occurs in an AGE-enriched environment,accelerated and excessive tissue destruction may be observed

Iwana et al.,(2002) reported decreased chemotaxis of leukocytes and increased detection of obligate anaerobic bacteria in the pulp of Goto-Kakizaki rats(Type-2 Diabetes Mellitus rats) on a 30% sucrose solution diet than in control rats or Goto-Kakizaki rats on a normal diet and water(6).

Periodontitis could initiate or propagate insulin resistance in a similar manner to that of obesity,by enhancing activation of the overall systemic immune response initiated by cytokines.

The mechanisms of the host-mediated response in Periodontitis involve activation of the broad axis of innate immunity,specifically by up-regulation of proinflammatory cytokines from monocytes and polymorphonuclear leukocytes.Thus,chronic gram-negative periodontal infections may induce or perpetuate an elevated chronic systemic inflammatory status,contributing to increased insulin resistance and poor glycemic control.

The lipopolysaccharide(LPS) from anaerobic gram-negative bacteria causing apical periodontitis activates intracellular pathways(nuclear factor kappa B,NF-kβ) on macrophages and neutrophils,upregulating proinflammatory cytokines such as IL-1β,IL-6,IL-8,tumour necrosis factor alpha(TNF-α) and prostaglandin E2(PGE2).These locally produced cytokines move into systemic circulation,where they interact with the free fatty acids and advanced products of glycosylation (AGEs), characteristic of type 2 DM. The activation of these in inflammatory pathways in immune cells (monocytes or macrophages), endothelium cells, adipocytes, hepatocytes and muscle cells could promote an increase in the overall

insulin resistance, altering the metabolic control in patients with both type 2 diabetes and chronic apical periodontitis(7).

DIABETES MELLITUS IN TOOTH DEVELOPMENT:

Hyperglycemia in a model of Type-1-Diabetes leads to altered amelogenesis,enamel biomineralization and predisposes teeth to excessive wearing and cavity formation.Elucidation of glucose-mediated effects on dental cells may provide therapeutic strategies for preserving enamel integrity and improving oral health in diabetic patients(4).

DIABETES MELLITUS AND PULPAL DISEASES:

Pulpitis is inflammation of dental pulp tissue. Dental pulpal infection is most commonly caused by extensive dental caries. The pulpal infection is most commonly caused by bacteria and it's furthur progress may also affect the periapical tissues of the involved tooth if not treated[8]. Bacterial invasion of dentinal tubules is commonly due to exposed dentin following a contravention in the integrity of the overlying enamel or cementum. Bacterial products passes through the dentinal tubule toward the pulp and provokes inflammatory changes in the pulpo-dentin complex. These may eliminate the bacterial insult and block the route of infection.If it is unchecked, invasion results in pulpitis and pulp necrosis, infection of the root canal system, and periapical disease[9]. Apical periodontitis (AP) is an acute or chronic inflammatory lesion around the apex of a tooth caused by bacterial infection of the pulp canal system. When apical periodontitis has occurred,restoring the periradicular tissues is necessary. Treatment is usually carried out by root canal treatment[7].

Akita mice were hyperglycemic (23.3mM) compared to normoglycemic(6.2mM) wild type controls. In Akita mice at 6 weeks, enamel was thin along cusp tips, less dense and showed significant wearing on x-ray compared to controls. This correlated with micro CT images of Akita mice teeth that showed decreased crown volume with rounded cusp tips, decreased enamel volume and mineral density. Histologic preparations of first molars showed intact dentin and pulp in wild type mice, whereas Akita mice showed pulpitis in the crown region. Gram-negative organisms were identified within the pulp and dentinal tubules and the adjacent tissue showed tertiary dentin formation. By 12 weeks, Akita mice showed severe pulpitis with destruction of periodontal structures and bone loss along tooth roots(10).

Pulps from patients with diabetes have the tendency to present limited dental collateral circulation, impaired immune response, increased risk of acquiring pulp infection (especially anaerobic ones) or necrosis, besides toothache and occasional tendency towards pulp necrosis caused by ischaemia.

Studies of rats with streptozotocin induced diabetes have been revealed a marked reduction in plasma blood flow in dental pulp.Because the dental pulp has limited or no collateral circulation,conditions induced by diabetes such as periodontitis can affect the dental pulp via periapical way(6).

Mesiodistal crown widths, crown and pulp widths at the cervix of first maxillary molars and heights of the mesial pulp horns of first mandibular molars in diabetics were greater than in non-diabetics but these are not important for clinical assessment.

Bissada and Sharawy observed calcified bodies in the pulp of diabetics. Similarly, there were more pulp stones in type I diabetics (35.6%) than in non-diabetics (10%) in first maxillary molars of the present study. Ranjitkar et al found that the prevalence of the pulp stones was similar in males and females.(11)

DIABETES MELLITUS AND PERIAPICAL DISEASES:

Diabetes Mellitus predisposes to oral infection and could also act as a risk factor for AP, increasing the rate of root canal treatment failure. Bender et al. reported that in inflammatory periapical reactions are greater in diabetic states, and the increased local in inflammation causes an intensication of diabetes with a rise in blood glucose, placing the patient in an uncontrolled diabetic state. Associating DM with higher prevalence of AP, greater size of the periapical osteolytic lesions, greater likelihood of asymptomatic periapical infections, and delay / arrest of periapical repair. Patients with diabetes presented increased risk of tooth extraction after nonsurgical root canal treatment by Wang et al(7).

DIABETES MELLITUS IN BONE RESORPTION:

Juan.J.et.al(2012) has found that alveolar bone resorption was most severe and the periradicular lesions were largest in diabetic rats given a sucrose solution, suggesting that the metabolic conditions produced by type 2 diabetes enhance the development of periradicular lesions .Bender & Bender found a high rate of asymptomatic tooth infections in diabetics exhibiting poor glycaemia levels of an unclear cause(7).

HEALING EFFECT OF DIABETES MELLITUS PATIENTS:

Hyperglycemia adversely affects pulpal healing in rats. Bender et al.(2003) reported that, in cases of poorly controlled DM, periapical radiolucencies tend to develop during treatment but, if DM is under therapeutic control, periapical lesions heal as readily as in non-diabetics. He reported that healing of periapical lesions is impaired in patients with uncontrolled diabetes and lesion size continues to increase despite endodontic treatment. Britto et al.,(2011) found that men with type-2 diabetes who had endodontic treatment were more likely to have residual lesions after treatment. Treating infections of pulp and periodontium will improve glycaemic control and help in healing of lesions similar to non-diabetics .Dentin bridge formation was inhibited in diabetic rats ($p = 0.029$) along with more inflammation in these pulps ($p = 0.005$). There was an inverse association between dentin bridge formation and inflammatory cell infiltration ($p = 0.001$). Based on these results, the authors conclude that it appears that hyperglycemia adversely affects pulpal healing in rats(12).

CONCLUSION:

It is hypothesized that vitamin D could improve the outcome of endodontic treatment in diabetic patients with clear effects over glucose homoeostasis, response and release of insulin and alveolar bone formation(13).Diabetes Mellitus is now prevelant among people of all ages because of its multiple causes of occurence.Dental Caries is the most common oral disease prevalent in developed and developing countries.So Dental Caries in Diabetic patients may lead to pulpal and periapical diseases.So this study is done to create awareness among public and dental practitioners about the relationship between diabetes mellitus and pulpal&periapical diseases.

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