REVIEW ARTICLE

Diabetes mellitus: An endodontic perspective

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ABSTRACT

Diabetes mellitus (DM) is a complex metabolic disease, characterised by hyperglycaemia resulting from defects in insulin secretion, insulin action or both. In patients with DM, several aspects of the immune system are compromised and wound healing is impaired. Studies indicate increased prevalence, severity of periapical lesions and a decreased success rate of endodontic treatment in diabetics, suggesting that diabetes may serve as a disease modifier of periapical lesions. A reciprocal relationship exists between glycaemic control and chronic periapical lesions. Treating infections of pulp and periodontium will improve glycaemic control and help in healing of lesions similar to non-diabetics. To provide competent care to patients with DM, dental clinicians must understand the disease, its treatment, and its impact on the patients' ability to undergo and respond to endodontic treatment. This review article is a detailed assessment of the literature on DM and its implication on pulp and periapical diseases, and their treatment outcome.

Key words
Diabetes mellitus, endodontics, periapical lesion

INTRODUCTION

Diabetes mellitus (DM) is a complex metabolic disease, characterised by hyperglycaemia resulting from defects in insulin secretion (type-1) or insulin action (type-2).[1] Diabetes is a major public health concern and the number of people suffering from diabetes is rapidly increasing.[2] It is likely that patients with known and unknown diabetes requiring endodontic treatment will be increasingly common. Diabetic patients usually have concomitant oral manifestations.[3] This review article is a detailed assessment of the literature on DM and its implication on pulp and periapical diseases, and their treatment outcome.

PREVALENCE OF PERIAPICAL LESIONS IN DIABETICS

Segura *et al.*[4] found higher prevalence of apical periodontitis at 81.3% in diabetics compared with 58% in control subjects. Among diabetic patients, 7% of teeth had apical periodontitis, whereas in control subjects 4% of teeth were affected. Similar results of increased prevalence of periapical lesions in patients with diabetes were reported by other studies[5-7] suggesting that diabetes may serve as a disease modifier of periapical lesions.

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PATHOGENESIS OF PERIAPICAL LESIONS IN DIABETICS

Numerous studies have been done on laboratory animals and humans to study diabetes. Two experimental rodent models are commonly used for study of type-1 diabetes.[8] The first one is rats in which diabetes is induced by injecting streptozotocin (SZT) that is selectively cytotoxic to the islet of pancreas. The second model involves use of non-obese diabetic mice, which are closer model to human type-1 DM. In non-obese diabetic mice, onset of diabetes is marked by an increase in autoantibodies to insulin and glutamic acid dehydrogenase. Effects of type-2 DM are often studied in Goto–Kakizaki rats that are one of the most reliable models for type-2 DM.[9] Goto–Kakizaki rats are non-obese Wistar sub-strain that develop type-2 DM early in life.

Several aspects of the immune system are compromised and wound healing is impaired in DM.[10] Foremost among those aspects is impaired function of leukocytes.[10]

During an inflammatory response, leukocytes adhere to endothelial cells due to the presence of adhesion receptors on leukocytes and endothelial cells. The leukocytes eventually become firmly attached to the vascular wall before migrating into tissues.[11] Accordingly, one possible explanation for the abnormal leukocyte function in DM might be a downregulation of adhesion molecules leading to decreased leukocyte–endothelial cell interactions and a reduced number of leukocytes in inflammatory lesions. Treatment of diabetic rats with insulin potentiates the expression of adhesion molecules, suggesting that upregulation of adhesion molecules might be associated with the circulating levels of this hormone.[12] Iwama *et al.*,[13] reported decreased chemotaxis of leukocytes and increased detection of obligate anaerobic bacteria in the pulp of Goto–Kakizaki rats (type-2 DM rats) on a 30% sucrose solution diet than in control rats or Goto–Kakizaki rats on a normal diet and water.[13]

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.[14] AGEs interact with their receptors (RAGEs) on endothelial cells, smooth cells and infiltrating mononuclear 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 adhesion molecules on endothelial cells, attraction and activation of macrophages, impaired collagen synthesis, and impaired leukocyte function.[15] 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.

The polyol pathway is another metabolic route by which hyperglycaemia is linked to leukocyte dysfunction.[16] Under physiological conditions, glucose is converted to glucose-6-phosphate by hexokinase. When in excess, because the hexokinase pathway is saturated, glucose is converted to sorbitol by aldose reductase, a rate-limiting enzyme of the polyol pathway. Abnormalities in neutrophil functions have been shown to be associated with the polyol pathway. Inhibition of the polyol pathway corrects the defective leukocyte–endothelial interaction found in experimental diabetes and may have a similar effect in diabetic patients.[16]

Besides increased polyol pathway flux, the major metabolic change caused by hyperglycaemia is an increase in oxygen-free radical formation that decreases resistance to oxidative stress.[17] Diabetes results in hyperglycaemia, inducing the formation of superoxides that contribute to the pathogenesis of microvascular and macrovascular complications. The first defence action against these superoxides is the enzymatic action of superoxide dismutase, catalase and glutathione peroxidise and peroxidise. Diabetes caused a reduction in superoxide dismutase, glutathione peroxidise and reductase activity in the dental pulp of alloxan-induced diabetic rats.[18] The second defence mechanism against superoxides is by the action of dietary derivatives such as vitamins C and E, and carotenes.[19] There was a reduction in pulpal blood flow in SZT-induced diabetic rats that improved by supplementation with vitamin C.[20] Astaxanthin, a carotenoid antioxidant, partially improved diabetic complications in the dental pulp of alloxan-induced diabetic rats.[18] The third mechanism is by sialic acid that acts as hydrogen peroxide scavenger and antioxidant agent. Both free and total sialic acid concentrations are lower in the dental pulps of SZT-induced diabetic rats compared with control rats.[18]

Armada *et al.*,[21] in their study found that periradicular lesions were larger and inflammatory response was more severe in SZT-induced diabetic rats compared with normal rats. So was in the study by Kohsaka *et al.*,[22] where root and alveolar bone resorption were more severe in SZT-induced diabetic rats than in control rats. Similar results had been reported in type-2 diabetic rats too.[23] Increased morbidity was noticed in non-obese diabetic rats than non-diabetic BALB/c mice when endodontic infection was induced.[24]

Diabetics are particularly prone to bacterial or opportunistic infections, which can be attributed to a generalized circulatory disorder whereby blood vessels are damaged by accumulation of atheromatous deposits in the tissues of the blood vessel lumen. In addition, blood vessels, particularly capillaries, develop a thickened basement membrane that impairs the leukotactic response, and there is a decrease in leukocyte microbicidal ability and failure to deliver the humoral and cellular components of the immune system.[25] Long-standing diabetes may result in angiopathy and thickening of the basement membrane in dental pulp vessels, affecting pulpal blood flow.[26] Dental pulp has limited or no collateral circulation, therefore it is more prone to be at risk of infection especially spread of periodontal infection to the pulp via the periapical route.[27] In diabetic patients, the circadian rhythm of pulp sensibility is altered than that of healthy elderly. This is of value in diagnosis of dental pulp diseases and deciding the suitable time for treatment of diabetic elderly.[28]

Garber *et al.*,[29] reported decreased dentin bridge formation and increased inflammation in the pulps of SZT-induced diabetic rats when compared with controls rats on pulp capping with a mineral trioxide aggregate.

EFFECT OF DIABETES ON ENDODONTIC TREATMENT OUTCOME

Lopez-Lopez *et al.*,[6] reported that 46% of root-filled teeth in patients with type-2 diabetes had apical periodontitis compared with 24% in non-diabetics. Bender *et al.*,[30] 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.*,[31] found that men with type-2 diabetes who had endodontic treatment were more likely to have residual lesions after treatment. The study by Fouad and Burlesson[32] suggests that success of endodontic treatment is reduced in diabetic patients having pre-operative periapical lesions. Decreased success rate of endodontic treatment[33,34] and increased prevalence of periapical lesions[35] in root-filled teeth has been reported in diabetics. Cheraskin *et al.*,[36] in their study monitored the radiographic healing of periapical lesions following endodontic treatment in 12 patients with low plasma glucose (70-89 mg/dl) and 13 patients with high plasma glucose (90-110 mg/dl). None of the patients in either group were known diabetics. At the end of the endodontic treatment visit, blood glucose measurement was done 2 h postprandially. After 30 weeks, the periapical radiolucencies of the low glucose levels were reduced by 74% compared with a reduction of 48% in the high-glucose group. Fouad *et al.*,[37] investigated the root canal microbiota of diabetics and non-diabetics using polymerase, chain reaction and reported that there was a positive, yet no significant, association between diabetes and presence of *Porphyromonas endodontalis* and *Porphyromonas gingivalis*. The number of different microorganisms detected per specimen was, on average, higher in diabetics than in non-diabetics, but the differences were not statistically significant.[37] In another study, *Eubacterium infirmum* was significantly more in diabetics than non-diabetics.[38] It can be inferred from the above polymerase chain reaction studies that there is a positive correlation between increased prevalence of some virulent endodontic microorganisms and a more pronounced periapical infection in diabetics compared with non-diabetics. Patients with diabetes have increased periodontal disease in teeth involved endodontically.[32]

EFFECT OF PERIAPICAL LESIONS ON GLYCAEMIC CONTROL OF DIABETICS

Chronic periapical lesions are similar to periodontal disease, as they share common characteristics such as chronic infections, predominantly caused by Gram-negative anaerobic microorganisms, and demonstration of increased levels of inflammatory mediators that may have an impact on systemic levels.[39] The presence of periodontal disease worsens glycaemic control.[40] So it can be hypothesized that periapical lesions being similar to periodontal disease can lead to increase insulin resistance and poor glycaemic control.[39] Schulze A *et al.*,[41] reported a case in which insulin demand increased following exacerbation of an endo-perio lesion. Insulin dosage was brought to the level of 4 weeks prior to root canal treatment when the lesion was treated endodontically and systemic antibiotics were administered. From this it can be hypothesised that inflammation contributes to the pathogenesis of diabetes, and a reciprocal relationship exists between diabetes and chronic periodontitis. Greater inflammatory reactions in diabetic states cause an increase in blood glucose level, leading to an uncontrolled diabetic condition.[42] This often requires therapeutic adjustment or increase in the dosage of insulin. Treating infections of pulp and periodontium will improve glycaemic control and help in healing of lesions similar to non-diabetics.[25]

CONSIDERATIONS FOR ENDODONTIC THERAPY IN DIABETICS

To provide competent care to patients with DM, dental clinicians must understand the disease, its treatment, and its impact on the patients' ability to undergo and respond to dental care. To minimise the risk of an intra-operative emergency, clinicians need to consider some issues before initiating dental treatment. Medical history is very important. When reviewing medical histories, a clinician should be aware of the cardinal signs of DM, such as polydypsia, polyphagia, polyuria, weight loss and weakness, and should refer to a physician for diagnosis and treatment.[43] In diabetic patients, clinicians should ascertain how well controlled the diabetic condition may be.[44] This should include detail information regarding most recent test results (e.g., glycosylated haemoglobin and postprandial blood glucose levels), frequency of hypoglycaemic episodes, medication, dosage and timing.[45]

Patients with diabetes who are well controlled medically and free of complications, such as renal disease, hypertension or coronary atherosclerotic disease, are candidate for endodontic treatment[46] like any other indicated dental treatment.[47] However, in the presence of acute infections, special considerations are to be given. Non-insulin controlled patients may require insulin, or the insulin dose for some insulin-dependent patients may have to be increased.[46] Acute infections in diabetic patients should be managed using incision and drainage, pulpectomy, antibiotics and warm rinses.[46]

For all endodontic treatments in well-controlled diabetics, appointments should be scheduled at early morning since endogenous cortisol levels are generally higher at this time (cortisol increases blood sugar levels). For patients receiving insulin therapy, appointments should be scheduled so that they do not coincide with peaks of insulin activity, since this is the period of maximal risk of developing hypoglycaemia.[45]

Before the procedure it has to be ensured that the patient has eaten normally and taken medication as usual.[43] Emotional and physical stress increases the amount of cortisol and epinephrine secretion that induces hyperglycaemia. Therefore, if the patient is very apprehensive, pre-treatment sedation should be contemplated.[45]

It is always advisable to consult a patient's physician before any surgical procedure to consider adjustment of the patient's insulin and dosage, antibiotic coverage, and dietary needs during the postoperative period.[46] Prophylactic antibiotic are not indicated for endodontic surgery in well-controlled diabetics as they are at no greater risk of postoperative infection than the non-diabetics.[48] Whereas when endodontic surgery is required in a poorly controlled diabetic, prophylactic antibiotic should be considered due to altered function of neutrophils. Surgery may also increase insulin resistance such that a diabetic may become hyperglycaemic in the postoperative period, requiring prescription of antibiotic.[44] Delayed alveolar healing following dentoalveolar surgery should raise the suspicion of osteomyelitis for which prompt surgical consultation has to be arranged. Lengthy appointments should be tried to be avoided. If a lengthy, especially surgical, procedure is to be undertaken, the patient's physician should be consulted. Blood glucose level should be constantly monitored during a lengthy surgical procedure. Hypoglycaemia is a common complication during dental treatment in diabetic patients. Symptoms of hypoglycaemia may range from mild, such as anxiety, sweating and tachycardia, to severe, such as mental status changes, seizure and coma. Severe hypoglycaemic episodes are medical emergencies and should be treated promptly with 15 g of oral carbohydrate such as 6 oz orange juice or 3-4 tea spoons of table sugar. If the patient is unable to cooperate or swallow, 1 mg of glucagon may be administered by subcutaneous or intramuscular injection.[44]

Many studies have reported a high incidence of failed endodontic treatment in diabetics.[30-34] Such cases may have to be referred to an endodontist for alternative treatment options such as orthograde retreatment or surgical retreatment.

CONCLUSION

DM affects people of all age groups, and its prevalence has been increasing. Therefore more and more diabetic patients will be requiring endodontic treatment. Research indicates increased prevalence of periapical lesions in diabetics, with decreased success rate of endodontic treatment. A reciprocal relationship exists between glycaemic control and chronic periapical lesions. Hence it is very vital for dental clinicians to understand the disease process of DM, its effect on pulp and periapical diseases, and their treatment outcome to provide competent endodontic treatment to patients with DM.

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