Diabetes mellitus & apical periodontitis

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ABSTRACT
Diabetes mellitus is a chronic disease with serious health consequences. Diabetics are associated with depression of natural defence against infection, increases the risk of periodontal and periradicular diseases. However the effect of diabetes on periradicular tissues which differs structurally than periodontal tissuees are not discussed much. In this review, diabetes mellitus and its effect on pulp and periradicular tissues and its effect on treatment outcome are discussed. Some other studies suggests that some bacterial species are more prevalent in diabetics than non diabetics patients. Here diabetics is considered as a potential modulating factor for endodontic pathosis and alterations in the treatment pattern needed for the same are discussed.

Key words: Diabetes mellitus, periradicular disease, apical periodontitis

Introduction
Diabetes mellitus is a complex metabolic disorder, a syndrome characterized by abnormalities in carbohydrate, lipid & protein metabolism that result either from profound or absolute deficiency of insulin or from target tissue resistance to its cellular metabolic effects. The prevalence of diabetes is rapidly rising all over the globe at alarming rate. Type 2 diabetes mellitus is the commonest form of diabetes & its prevalence is 2.4% in rural population & 11.6% in urban population. The primary driver of the epidemic of diabetes is the rapid epidemiological transition associated with changes in dietary patterns & decreased physical activity as evident from the higher prevalence of diabetes in the urban populations. The most disturbing trend is the shift in age of onset of diabetes to a younger age in recent years. Type 1 diabetes results from cellular-mediated autoimmune destruction of pancreatic Î²-cells, which usually leads to total loss of insulin secretion; in contrast, type 2 diabetes is caused by resistance to insulin combined with a failure to produce enough additional insulin to compensate for the resistance. Type 2 diabetes is commonly linked to obesity, which contributes to insulin resistance through elevation of circulating levels of free fatty acids derived from the adipocytes; these free fatty acids inhibit glucose uptake, glycogen synthesis and glycolysis.

Oral Complications
The oral complications of uncontrolled diabetes mellitus include xerostomia, infection, poor healing, increased incidence & severity of caries, candidiasis, gingivitis, periodontal diseases & burning mouth syndromes. In the two last decades several epidemiological studies have investigated the association between systemic...
health and PD. Thus, PD has been associated to diabetes mellitus (DM)\textsuperscript{5,6}.

Poorly controlled insulin dependent diabetes (PIDD) had more gingivitis & attachment loss & gingival recession than controlled insulin dependent diabetes (CIDD) with a mean HBAI C of 9.2%\textsuperscript{7}. Higher incidence of attachment loss had been described among diabetics. Increased susceptibility to periodontal diseases due altered host response, subgingival microflora, collagen metabolism, vascularity\textsuperscript{8}.

The possible connection between chronic oral inflammatory processes of infectious origin, i.e. chronic apical periodontitis and periodontal disease (PD), and systemic health is one of the most interesting aspects faced by the medical and dental scientific community\textsuperscript{5,6}.

Although some differences are evident between chronic periodontal and periapical inflammatory processes, both show three important similitudes: 1) both are chronic infections of the oral cavity, 2) both are polymicrobial infections sharing a common microbiota with predominance of Gram-negative anaerobic bacteria and 3) elevated cytokine levels may be released systemically from acute and chronic manifestations of both disease processes\textsuperscript{9,10}. Likewise, it can be assumed that Apical periodontitis (AP), an inflammatory process around the apex of the tooth is the primary sequelae to microbial infection of pulp space of the teeth and is a remarkably wide spread clinical problem linked with the same systemic disorders associated to periodontal diseases\textsuperscript{11}. Consequently, numerous investigations have been conducted to study the relationship between AP and DM. In this paper, the current status of knowledge regarding the relationship between diabetes mellitus and apical periodontitis and effect of diabetes mellitus on tooth pulp and periapical area are reviewed.

**Pulp in diabetes mellitus:**

Histopathologic pulp observations in human diabetics are limited. Russel et al showed that there is angiopathies and thickened basement membrane of large and small blood vessels which impairs the leukocytic response and failure to deliver the humoral and cellular components of immune system. Vascular problems associated with diabetes also cause an increase in anaerobic infection, which may be attributed to reduced oxygen diffusion across the capillary wall. Infection become more severe and last longer because of neutrophil microbicidal suppression and synergism of aerobic and anaerobic bacteria, due to anoxia\textsuperscript{12}.

Pulp of patients who suffer from diabetes tend to age more rapidly because of obliterative endartities\textsuperscript{12}. The histological changes in pulpal & periapical tissues after pulpal exposure in streptozotocin induced diabetic rats showed inflammation in apical periodontal ligament, root resorption & alveolar bone resorption were more severe in diabetic rats than that in control rats\textsuperscript{13}. Nayak et al showed higher incidence of pulpal calcification in type II diabetes and other systemic disease. Though pulpal calcifications were found in both diabetics and other systemic disease, in type 2 diabetics these calcifications were more frequent and often sickle shaped. Bissada and Sharawy group also found amorphous calcified bodies in the pulp of diabetics\textsuperscript{14}. 
Microbial findings in endodontic infection of diabetic patients

The root canal system of diabetic patients colonized with different microbial profiles from non diabetic, make them more susceptible to severe periradicular diseases. Significant increase in *Prevotella Intermedia* in the deepest probing site and *Porphyromonas Gingivalis* were recovered from insulin dependent diabetes. Fouad et al, conducted a microbial study by collecting samples aseptically from necrotic pulp of twenty-four patients including six diabetics undergoing endodontic treatment. DNA was extracted from the specimens and subjected to PCR amplification with universal bacterial primers for the 16S rRNA gene. The results showed that *F. nucleatum*, *P. micros*, and *Streptococcus* spp. were the most prevalent of the microorganisms examined. There were highly significant associations between *Streptococcus* species and the combination of *Streptococcus* spp. and *F. nucleatum* and preoperative pain. With respect to diabetes, there was a positive, yet nonsignificant, association between diabetes and the presence of *P. endodontalis* and *P. gingivalis*.

Effect of diabetes mellitus on apical periodontitis:

Numerous reports documenting that diabetes mellitus, especially poorly controlled is associated with significant periodontal disease and tooth loss. Individual study had demonstrated the prevalence of apical periodontitis amongst type 2 diabetic patients. They recorded full mouth radiographic survey of 38 subjects with diabetes. Periapical status was assessed using periapical index system. The results showed apical periodontitis in 81.3 % in diabetic patients & 58% of control subjects & concluded that type 2 diabetes mellitus is significantly associated with an increased prevalence of apical periodontitis. Similar study by Britto found no significant difference in the prevalence of apical periodontitis between diabetic and non diabetic group. Falk et al found that a group of individuals amongst patients with diabetes who had more periradicular lesions than did those without diabetes. Long duration diabetics exhibited teeth with periapical lesions to a greater extent than short duration diabetics. Diabetics & non diabetics exhibited a similar caries frequency, but among the diabetics there was a group of individuals who had more periapical lesions than non diabetics.

There is a trend towards increased symptomatic periradicular disease in patients with diabetes who received insulin as well as flare ups in all patients with patients with diabetes. They should be treated with effective antimicrobial root canal regimens, particularly in cases with preoperative lesions.

Endodontic treatment outcome in diabetics:

The clinical management of apical periodontitis involves infection control by root canal treatment. Success rate of treatment has been generally regarded as high, of the order of 87%. However, when the failure rate is measured relative to the prevalence of root canal treatment; the full dimension of the problem becomes apparent with a conservative failure rate of 13% for root canal treatment. Several factors affect the higher failure rate of endodontic treatment and diabetic patients had almost threefold increase in their failure rate.
Prevalence of radiographic periradicular radiolucencies in endodontically treated & untreated teeth in patients with & without diabetes showed that type 2 diabetes to be associated with an increased risk of ill response by the periradicular tissues to odontogenic pathogens. We conducted a study to evaluate single visit endodontic treatment in type 2 diabetic patients with irreversible pulpitis and apical periodontitis over a period of 6 months and found diabetic subjects showed delayed healing with only 90% compared to normal group. A significant difference were seen from 2 weeks to 6 months, lesions <1 mm and 1 mm healed within 1 month and 2 months. Poor controlled diabetics showed delayed healing compared to fair and good controlled(fig :1).

Histologic analysis showed larger periradicular lesions & severe alveolar bone resorption in diabetic rats suggesting that the metabolic conditions produced by the diabetes enhance the development of periradicular lesions in rats. Fouad et al (2002) showed a more severe outcome in a type 1 diabetic mouse model after inoculation of the exposed pulp with a mixture of facultative & anaerobic bacteria, compared to normal mice.

DM affects many functions of the immune system and is associated with delayed healing and compromised immune responses. DM-induced changes in immune cell function produce an inflammatory immune cell phenotype (up-regulation of pro-inflammatory cytokines from monocytes / polymorphonuclear leukocytes and down-regulation of growth factors from macrophages). This predisposes to chronic inflammation, progressive tissue breakdown, and diminished tissue repair capacity.

Diabetics who present for endodontic treatment, particularly those with periradicular pathosis may have increased preoperative symptoms and should be treated with effective
antimicrobial root canal regimens. Cases with preoperative periradicular pathosis are less likely to be determined successful two years or longer postoperatively, if the patient reports a history of diabetes.\(^\text{15}\)

Researches suggested the relationship between diabetes and endodontic problems. Vitamin D has important biologic effects on glucose homeostasis, insulin release and response, and is considered to play a role in the pathogenesis of diabetes. Vitamin D can also influence the alveolar bone formation and inflammatory reactions in periradicular tissues. Hence they hypothesised that intake of Vit D may help in treating apical periodontitis in diabetic patients.\(^\text{24}\)

**Effect of apical periodontitis on diabetes mellitus**

It has been stated that PD can have a significant impact on the metabolic state in diabetes. The presence of periodontitis increases the risk of worsening of glycemic control over time.\(^\text{25}\) Several biologically plausible mechanisms could be proposed to explain the interactions between diabetes and PD. As it has been exposed previously, there are important similitudes between PD and AP. So, it could be hypothesized that chronic periapical inflammatory processes can also contribute to the pathogenesis of DM, being a risk factor for worsening glycemia control among diabetic patients. The activation of the inflammatory pathway 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. Bender et al\(^\text{26}\) reported that inflammatory periapical reactions are greater in diabetic states, and the increased local inflammation causes an intensification of diabetes with a rise in blood glucose, placing the patient in an uncontrolled diabetic state. This often requires an increase in insulin dosage or therapeutic adjustment. Removal of the inflammatory state usually creates a need for a lesser amount of insulin for diabetic control. Thus, it becomes axiomatic to remove all infections including those of the dental pulps.

**Conclusion**

The information available on pathogenesis, progression and healing of pulpal and periradicular pathosis in diabetic patients remains in its infancy. There is evidence from the literature 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. The prognosis for root filled teeth is worse in diabetics, showing a higher rate of root canal treatment failure with increased prevalence of persistent chronic apical periodontitis. A number of important unanswered questions should be addressed in future research in this area. Ultimately these should determine effective antimicrobial agents and treatment strategies for diabetic patients and help them to preserve a healthy dentition throughout life.

**References**


