Healing of Apical Periodontitis after Nonsurgical Treatment in Patients with Type 2 Diabetes

Suman Arya, MDS, Jigyasa Duhan, MDS,* Sanjay Tewari, MDS,* Pankaj Sangwan, MDS,* Veena Ghalaut, MD,† and Sameer Aggarwal, MD‡*

Abstract

Introduction: The purpose of this prospective study was to compare the success of primary root canal treatment between type 2 diabetic and nondiabetic patients and to investigate the effect of periapical healing on glycated hemoglobin (HbA1c) in type 2 diabetic patients with apical periodontitis. **Methods:** Sixty mandibular molars with necrotic pulps and apical radiolucency (size $\geq$ 2 mm $\times$ 2 mm) were included in the study. Based on the HbA1c levels, patients were divided into 2 groups: type 2 diabetic (HbA1c $\geq$6.5%) and nondiabetic (HbA1c <6.5%). Forty-six teeth were evaluated at the 12-month follow-up time period. The primary outcome measure was the change in apical bone density as determined by the periapical index. **Results:** Both the diabetic and nondiabetic group depicted a significant reduction in the periapical score after endodontic treatment at the 12-month follow-up ($P < .05$). Significantly less periapical healing was observed in the diabetic group (43%) compared with the nondiabetic group (80%) at the 12-month follow-up ($P < .05$). HbA1c levels in the diabetic group increased at each follow-up after endodontic treatment. **Conclusions:** Diabetes mellitus may have a negative impact on the outcome of endodontic treatment in terms of periapical healing. Nonsurgical endodontic treatment did not improve HbA1c levels in patients with type 2 diabetes. *(J Endod 2017;■:1–5)*

Key Words

Apical periodontitis, diabetes mellitus, glycated hemoglobin

Significance

Patients with diabetes might respond to nonsurgical root canal treatment in a similar way as healthy controls. Healing is delayed in these patients but not compromised; thus, periodical follow-up and evaluation to monitor healing in patients with diabetes is mandatory.

Diabetes mellitus (DM) is a clinically and genetically heterogeneous group of disorders affecting the metabolism of carbohydrates, lipids, and proteins in which hyperglycemia is the main feature. These disorders are either caused by insulin deficiency related to autoimmune destruction of pancreatic beta cells (type 1) or target tissue resistance to its cellular metabolic effects (type 2) (1). DM affects more than 9% of the adult population (2). It affects the immune cell function, up-regulates cytokines from monocytes or leukocytes, and down-regulates the various growth factors predisposing to chronic inflammation, progressive tissue breakdown, and reduced tissue repair capacity (3). This immune phenotype leads to the oral complications of DM, mainly gingivitis, periodontal disease (PD), and apical periodontitis (AP). Evidence has consistently indicated that DM is a risk factor for increased severity of PD including gingivitis and periodontitis (4, 5).

AP is an acute or chronic inflammation of the periodontium located at the root apex caused by bacterial infection of the pulp canal system (6). Chronic PD and AP have similarity in terms of chronicity, polymicrobial infections with common microbiota, and elevated cytokines and inflammatory mediator levels; thus, there appears to be a biologic basis that suggests an association between DM and a high prevalence of periapical lesions and its response to endodontic treatment (7). Polymicrobial irritants like lipopolysaccharides from gram-negative bacteria provoke a periapical inflammatory response by activating the innate immunity. The integrity of the nonspecific immune system is a significant predictor for primary endodontic treatment and retreatment outcome (8). The nonspecific immune system has an impact on the healing of periapical tissues. Thus, the proinflammatory status and impaired immune response associated with systemic diseases can affect the reparative response of the dental pulp and periapical healing (9). A disproportionately high percentage of clinically severe pulpal or periodontal infections in patients with DM have been reported (10). Segura Egea et al (11) found a higher prevalence of AP in type 2 diabetic patients. However, Britto et al (12) found no significant differences in the prevalence of AP between diabetics and controls but reported that men with type 2 diabetes who had endodontic treatments were more likely to have residual lesions after treatment.

Traditionally, DM was diagnosed by either fasting or postprandial blood glucose, but since 2010, the American Diabetes Association has recommended the mean glycated hemoglobin (HbA1c) as the diagnostic criterion of diabetes with its threshold

From the *Department of Conservative Dentistry and Endodontics, Post Graduate Institute of Dental Sciences; and Departments of †Biochemistry and ‡Medicine/Endocrinology, Post Graduate Institute of Medical Sciences, Rohtak, India.

Address requests for reprints to Dr Jigyasa Duhan, Department of Conservative Dentistry and Endodontics, Post Graduate Institute of Dental Sciences, Rohtak 124001, India. E-mail address: prashitiduhan@rediffmail.com

0099-2399/$ - see front matter

http://dx.doi.org/10.1016/j.joen.2017.05.013

level of 6.5%. HbA1c is a resulting product of hemoglobin and glucose interaction, and its elevated level indirectly reflects the hyperglycemic condition (ie, DM). Cintra et al (13) investigated the effect of AP and PD on long-term blood glucose concentrations and HbA1c levels, concluding that oral infections affect glycemic conditions in diabetic rats and increase HbA1c levels in normoglycemic and diabetic rats. A significantly higher prevalence of AP has been found in type 2 diabetics with HbA1c levels ≥6.5% (14).

The evidence available from the existing literature regarding the relationship among DM, apical periodontitis, and endodontic outcome is unclear. On searching the literature, we found a few studies discussing the relationship between diabetes and AP (11, 12, 15–19). Most of these studies are cross-sectional studies (11, 12, 15, 17–19) with a low level of evidence and have mainly investigated the prevalence of AP in diabetic patients and not the endodontic success. Thus, the present study was designed in a prospective manner, with the objective to investigate the success rate of primary nonsurgical root canal treatment (RCT) in type 2 diabetic patients and its effect on HbA1c level.

Materials and Methods

Approval for the study was provided by the Institutional Ethical Committee of the Post Graduate Institute of Dental Sciences (PGIDS/IEC/2014/112), Rohtak, India. Patients for the study were recruited from the pool of patients referred to the Department of Conservative Dentistry and Endodontic at the Post Graduate Institute of Dental Sciences for initial nonsurgical RCT. Patients with a history of type 2 DM (with HbA1c ≥6.5%) between the ages of 30 and 65 years with mature permanent mandibular molars with pulpal necrosis as confirmed by a negative response to cold and electric tests and radiographic evidence of AP (minimum size ≥2 mm × 2 mm) were included in the study. Nondiabetic patients were enrolled in the control group.

Patients with PD or systemic disorders other than DM, patients taking steroids, pregnant patients, patients with cracks/fractures, patients who underwent a procedural error, smokers, and patients with a history of antibiotic intake in the preceding month were excluded from the study. After fulfilling the eligibility criteria, patients were informed about the study, and written consent to participate in the study was acquired from each patient. The diabetic patients were instructed to continue with their medical treatment of DM (oral hypoglycemic agents), diet, and lifestyle without modifications during the study period.

Sample Size Calculation

With an assumption of normal sample distribution, the Karlsson (20) method was used for sample size determination. To show an adequate difference with a power of 0.90, $P < .05$, and standard deviation ±0.50 U and the minimum clinically significant mean difference between 2 groups set at 0.50 U while using the periapical index (PAI), a minimum sample size of 21 was calculated, but keeping in mind the patient dropout rate, a total of 30 patients per group were recruited.

Glycemic Control Assessment

To determine the metabolic control status of diabetic patients, HbA1c was registered on the day of treatment initiation. Adequate glycemic control was defined according to the American Association of Clinical Endocrinologists as HbA1c <6.5% (21). Based on these criteria, the subjects for the study were divided into 2 groups: type 2 diabetic patients and nondiabetic patients.

Clinical Procedure

The endodontic treatment was performed by a single operator (S.A.) following a standardized protocol. After local anesthesia administration (2% xylocaine with 1:80,000 epinephrine), the access cavity was prepared using a sterile carbide bur under rubber dam isolation. After pulp chamber debridement and canal orifice identification, coronal shaping and enlargement were performed with low-speed Gates Glidden drills (Dentsply Maillefer, Tulsa, OK) to obtain straight-line access to the apical third of each root canal. The working length was determined with the help of a Root ZX apex locator (J Morita, Tokyo, Japan) and verified radiographically. Canals were prepared using the crown-down technique with rotary ProTaper (Dentsply Maillefer) instruments. The finishing of the apical third was done with either an F2 or F3 file at the working length depending on the size of apical preparation after gauzing with a 2% tapered hand instrument. Canals were irrigated with 5 mL 5.25% sodium hypochlorite (NaOCl) (NeoDent, New Delhi, India) subsequent to changing each instrument. After complete canal instrumentation, the canals were irrigated with 5.0 mL 17% EDTA (Prevest Denpro, Jammu, India) for 1 minute followed by a final irrigation with 5.0 mL 5.25% NaOCl. Canals were filled with a paste of calcium hydroxide (Prevest Denpro), and the access cavity was sealed temporarily with Intermediate Restorative Material (Dentsply Ltd, Weybridge, UK). Patients were scheduled for a second visit after 1 week. At the next appointment, the paste was removed with Hedstrom files (Mani Inc, Brussels, Germany) followed by copious irrigation with 5.25% NaOCl. Canals were obturated using the lateral condensation technique. After obturation, the cavity was permanently restored with composite restoration. An immediate postoperative radiograph was taken using preset exposure parameters with a Rinn paralleling device (XCP Instruments, Elgin, IL) and processed manually. Follow-up clinical and radiographic examinations were performed at every 3-month interval up to 12 months using the same parameters of the initial examination.

Outcome Variable Assessment

The primary outcome measure was the change in apical bone density (PAI score) at 3, 6, 9, and 12 months. The secondary outcome measure was the presence of clinical symptoms or abnormal findings at 12 months (ie, spontaneous pain, presence of sinus tract, swelling, mobility, periodontal probing depth greater than baseline measurements, or sensitivity to percussion and palpation). Clinical findings were recorded and compared with preoperative diagnostic records. Immediate postobturation and follow-up radiographs were examined by 2 separate independent observers blinded to the diabetic status of the patients according to the PAI scoring system (22). The worst outcome of an individual root decided the overall outcome for the tooth. In case of any disagreement, the 2 observers met to discuss their findings and came to an agreement. The teeth were further categorized as healed (PAI ≤2) or improved (decreased PAI score) (23). HbA1c measurement was done at baseline, 6 months, and 12 months to assess the glycemic status.

Statistical Analysis

The Kolmogorov-Smirnov test for normality was used to assess data distribution, and because data were not normally distributed, nonparametric tests were applied. The Mann-Whitney *U* test and chi-square test were used to evaluate the PAI score change at baseline and subsequent time intervals for ordinal data and dichotomized data, respectively, between both groups. The change in the PAI score for the individual groups was analyzed using the Wilcoxon signed rank test.