

## To Study the Effect of Non-Surgical Periodontal Therapy in Type-2 Diabetes Patients with Chronic Periodontitis with Evaluation of Hba1c- in Vivo Study

Neha Waman Dhadse<sup>1</sup>, Pradeep Kumar<sup>2</sup>, Rohan Gupta<sup>3</sup>, Sonia Godara<sup>4</sup>, Rajendra Yadav<sup>5</sup>, Mayank Gupta<sup>6</sup>

<sup>1</sup>MDS Resident, Department of periodontics, Rajasthan Dental College and Hospital, Jaipur, Rajasthan, India

<sup>2</sup>Professor and Head, Department of periodontics, Rajasthan Dental College and Hospital, Jaipur, Rajasthan, India

<sup>3</sup>Associate Professor, Department of periodontics, Rajasthan Dental College and Hospital, Jaipur, Rajasthan, India

<sup>4</sup>Assistant Professor, Department of periodontics, Rajasthan Dental College and Hospital, Jaipur, Rajasthan, India

<sup>5</sup>Associate Professor, Department of periodontics, Rajasthan Dental College and Hospital, Jaipur, Rajasthan, India

<sup>6</sup>PG Resident, Department of periodontics, Rajasthan Dental College and Hospital, Jaipur, Rajasthan, India

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Corresponding author: Dr. Rajendra Yadav

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### Abstract

**Background:** This study was performed to study the effect of non-surgical periodontal therapy in type-2 diabetes patients with chronic periodontitis with evaluation of hba1c.

**Materials and Methods:** The study planned on 50 subjects with type 2 diabetes mellitus (HbA1c values 6-8%) within a period of 7 years with moderate to severe periodontitis were selected. Medical records and data regarding HbA1c level, type of medications, and changes in medication or dosages was collected. The clinical parameters (PI, GI, SBI) and HbA1c were recorded at baseline & as well as 6 months post periodontal treatment. Data was analysed using SPSS software version 22. Descriptive frequencies for gender and age were calculated. The comparison between values of all the clinical and metabolic parameters at the baseline and 6 months was done by Z-test. The correlation between the clinical parameters with HbA1c at the baseline and 6 months was done by Karl Pearson coefficient test. The correlation between the differences in mean values of PI, GI, and SBI with HbA1c from the baseline to 6 months was done by t-test.

**Results:** Reduction in all the clinical parameters were observed and were found to be statistically significant. The mean HbA1c value at baseline was  $8.146 \pm 1.75$ , which reduced to  $7.11 \pm 1.80$  at 6 months. The mean percentage difference was 12.8%.

**Conclusion:** The study showed that the Periodontal Therapy significantly contributed to glycemic control in T2DM patients.

**Keywords:** HbA1c, Chronic Periodontitis, SRP, T2DM

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## Introduction

Diabetes mellitus (DM) is a systemic disease with several major complications affecting both the quality and length of life. One of these complications is periodontal disease (periodontitis). Current evidence suggests that periodontitis is more than a localized oral infection. [1] The chronic gram-negative infection of periodontal origin may be considered a potential focus of infection that aggravates metabolic control in patients who have diabetes. [2]

Periodontitis is a chronic and progressive inflammatory disease predominantly caused by the interaction of microorganisms and host immune response and is an important cause of tooth loss among adults. [3] Chronic periodontitis (CP) not only progressively and painlessly destroys the supporting tissues around the teeth but also has an influence on the systemic health of the individual. [4]

It has been extensively studied that in the presence of CP, patients with DM exhibit worsening of their glycemic control. The release of endotoxins by periodontal pathogens, and secretion of proinflammatory cytokines by the affected gingival tissue results in worsening of insulin resistance and consequent hyperglycemia. Various studies have reported that patients with diabetes have a higher prevalence of CP. Furthermore, patients with poorly controlled diabetes have a higher risk (2.9 times) of having severe periodontitis compared to the nondiabetic participants. Therefore, it is imperative to consider periodontal disease as an important complication of diabetes. [5]

Both diabetes and periodontal infection are complicating and enhancing each other's magnitude and severity; therefore, it is suggested that along with the medications for diabetes, the control of active periodontal infection should also be aimed to achieve a long-term glycemic control in

these patients. [5] Nonsurgical periodontal therapy (NSPT) is the initial step in the management of periodontal diseases. The target of NSPT is to modulate or abolish the microbes and other causative factors implicated in gingival and periodontal diseases. [6] Hyperglycemia is known to favor pro-inflammatory priming of periodontal tissues thus contributing to the earlier onset of gingivitis, the initial and reversible stage of periodontitis, in patients with diabetes. Persistent hyperglycemic state and advanced glycation end product besides causing the macrovascular and microvascular complications of diabetes also result in the impaired gingival fibroblast synthesis, defective phagocytic activity of mononuclear, and polymorphonuclear cells. [6]

This results in the loss of periodontal fibers and supporting alveolar bone. These findings led to consider periodontitis as the sixth complication of diabetes mellitus. Importantly, the relationship between diabetes and periodontitis is bidirectional. [7]

## Materials and methods

The present study was carried out in the Department of Periodontology and Oral Implantology, Rajasthan Dental College and Hospital, Jaipur. The subjects of this study were selected from the Outpatient Department of Periodontology and Oral Implantology, Rajasthan Dental College and Hospital, Jaipur. All patients were selected on the basis of the following inclusion and exclusion criteria.

### Selection criteria

The following were the inclusion criteria

- The age of the subjects ranged from 40 years to 70 years.
- The mean duration of diabetes was  $3.3 \pm 1.6$  years.
- Probing pocket depth of  $\geq 5$  mm in minimum five teeth.

- Clinical attachment loss of 3-4 mm in a minimum of eight teeth.

The following were the exclusion criteria

- Radiographic evidence of periapical pathology.
- Current smokers.
- Those who were on antibiotics for the last 4 months.
- Physically or mentally disabled, pregnant patients.
- And patients with major diabetic complications such as retinopathy, nephropathy, cardiopathy, and cerebrovascular changes.
- Patients undergone any periodontal treatment 4 months prior to the study.

### Study design

A prospective, interventional, comparative, clinico biochemical study was planned. Medical records that of preoperative and postoperative to periodontal treatment were reviewed and data regarding HbA1c level, type of medications, and changes in medication or dosages was collected. 50 subjects with type 2 DM (HbA1c values 6-8%) within a period of 7 years with moderate generalized chronic periodontitis were recruited with their informed consent. Out of the 50 subjects, there were 31 (62%) males and 19 (38%) females.

### Procedure

Glucose control was determined by HbA1c. The patients underwent: general medical history, radiographic examination, determination of HbA1c and glucose in blood, periodontal examination, provision of information on periodontal disease and oral hygiene.

After all the examinations the patients underwent supragingival prophylaxis. The clinical and biochemical parameters were recorded at the baseline (day 0) and 6 months after the periodontal therapy. The participants were instructed to continue with their medical management of DM

without any modification during the study period.

Recordings of the clinical parameters for periodontal status were recorded using:

- Plaque index (PI) (Silness P and Loe H 1967)[8]
- Gingival index (GI) (Loe H and Silness J 1963)[8]
- Sulcus bleeding index (SBI) (Muhlemann HR and Son S 1971)[8]

Probing pocket depth (PPD) — The recordings were done on all the four sites (buccal, lingual, mesial, and distal) of each tooth. Clinical attachment level (CAL) — The recordings were done on all the four sites (buccal, lingual, mesial, and distal) of each tooth. It was measured from the cemento-enamel junction (CEJ) to the base of the pocket using UNC-15 probe. [8]

For the metabolic assessment, 3-4 mL of venous blood sample was withdrawn and analyzed for HbA1c (Glycohemoglobin analyzer, D-10 Hemoglobin Testing System, Bio Rad Laboratories, Inc.)

All the participants received oral hygiene instructions before the first session of full-mouth scaling and root planing (SRP). Modified Bass method of brushing was instituted. The treatment period for scaling and root planing session was 1 week. There were four sessions and each session lasted for 1 h. The sessions were performed by the same investigator using an ultrasonic device (Guilin Woodpecker Medical Instrument Co., Ltd. Information Industrial Park, Guilin National High Tech Zone, Guilin, Guangxi, China) Root planing was performed with Gracey curettes (Hu- Friedy Mfg. Co. LLC. 3232N, Rockwell St., Chicago, IL 60618, USA) using 12-14 strokes per site until a smooth, hard, and polished surface was obtained.

Patients were asked to visit at a 1-month interval to assess the efficiency of their oral hygiene methods and reinstructed, if required, during the follow-up of 6

months. Any alteration in diabetes control or antibiotic use was recorded during their visits.

### Results

The study was done on 50 subjects (men and women) between the ages 40-60 years. Out of 50 diabetic subjects in the study population, frequency of female patients is 19 (38%) and frequency of male population is 31 (62%). Mean age of the patients was  $51.84 \pm 7.055$  years.

The mean HbA1c value at baseline was  $8.146 \pm 1.75$ , which reduced to  $7.11 \pm 1.80$  at 6 months. The mean percentage difference is 12.8%. The difference in the mean values was 1.036, which was statistically significant ( $P < 0.05$ ). The mean plaque index was  $2.37 \pm 0.38$  at the baseline, which declined to  $0.47 \pm 0.21$  at 6 months after the periodontal therapy. The mean percentage difference is 80.2%. The mean difference was 1.9, which was statistically significant ( $P < 0.05$ ). Following the treatment, GI dropped from  $2.59 \pm 0.33$  at the baseline to  $0.60 \pm 0.29$  after 6 months. The mean difference was

1.99, The mean percentage difference is 76.9% which was statistically significant ( $P < 0.05$ ). The mean SBI index was  $3.22 \pm 0.46$  at the baseline, which declined to  $0.42 \pm 0.64$  at 6 months after the periodontal therapy. The difference in the mean values was 2.8 The mean percentage difference is 87% which was statistically significant ( $P < 0.05$ )

The correlation between PI and HbA1c was  $r = 0.63$  at the baseline and 0.34 at the 6 month which mean that the values of PI and HbA1c from the baseline to 6 months was statistically significant  $r = 0.34$ ,  $P < 0.05$  The correlation between GI and HbA1c both at the baseline  $r = 0.57$  and after 6 months,  $r = 0.42$ . The correlation between the differences in mean value of GI and HbA1c from the baseline to 6 months was statistically significant ( $P < 0.05$ ). The correlation between SBI and HbA1c both at the baseline  $r = 0.64$  and after 6 months,  $r = 0.38$ . The correlation between the differences in mean value of SBI and HbA1c from the baseline to 6 months was statistically significant ( $P < 0.05$ ).

**Table 1: The study was done on 50 subjects (men and women) between the ages 40-60 years**

| Gender | Frequency | Percent |
|--------|-----------|---------|
| Female | 19        | 38.0    |
| Male   | 31        | 62.0    |
| Total  | 50        | 100.0   |

**Table 2: Diabetic subjects in the study population, frequency of female as well as male population and Mean age of the patients**

| Age (years) | Frequency | Percent | Mean $\pm$ SD     |
|-------------|-----------|---------|-------------------|
| 40          | 3         | 6.0     | 51.84 $\pm$ 7.055 |
| 41          | 1         | 2.0     |                   |
| 42          | 2         | 4.0     |                   |
| 44          | 1         | 2.0     |                   |
| 45          | 4         | 8.0     |                   |
| 46          | 4         | 8.0     |                   |
| 47          | 4         | 8.0     |                   |
| 48          | 2         | 4.0     |                   |
| 49          | 1         | 2.0     |                   |
| 50          | 3         | 6.0     |                   |

|       |    |       |  |
|-------|----|-------|--|
| 51    | 1  | 2.0   |  |
| 52    | 1  | 2.0   |  |
| 54    | 1  | 2.0   |  |
| 55    | 1  | 2.0   |  |
| 58    | 6  | 12.0  |  |
| 59    | 4  | 8.0   |  |
| 60    | 11 | 22.0  |  |
| Total | 50 | 100.0 |  |

**Table 3: The mean baseline, six months, Z value and P value of HbA1c value, Plaque index, Gingival index and Sulcus bleeding index**

|                       | Mean±SD<br>baseline | Mean ± SD<br>Six months | Z value | P value |
|-----------------------|---------------------|-------------------------|---------|---------|
| HbA1c                 | 8.146 ±1.75         | 7.11±1.80               | 32.82   | 0.00    |
| Plaque index          | 2.37±0.38           | 0.47±0.21               | 43.80   | 0.00    |
| Gingival index        | 2.59±0.33           | 0.60±0.29               | 54.32   | 0.00    |
| Sulcus bleeding index | 3.22±0.46           | 0.42±0.64               | 27.81   | 0.00    |

**Table 4: The baseline, six months and difference of HbA1c value, Plaque index, Gingival index and Sulcus bleeding index**

| PARAMETERS            | BASELINE     | 6 MONTHS    | % DIFFERENCE |
|-----------------------|--------------|-------------|--------------|
| HbA1c                 | 8.146 ± 1.75 | 7.11 ± 1.80 | 12.8         |
| Plaque Index          | 2.37 ± 0.38  | 0.47 ± 0.38 | 80.2         |
| Gingival index        | 2.59 ± 0.33  | 0.60 ± 0.29 | 76.9         |
| Sulcus Bleeding Index | 3.22 ± 0.46  | 0.42 ± 0.64 | 87           |

**Table 5: The Baseline comparison in mean, STD, T value, R value and P value of HbA1c's Plaque index, Gingival index and Sulcus bleeding index**

| Baseline comparison            | Mean | Std. Deviation | T value | R value | P value |
|--------------------------------|------|----------------|---------|---------|---------|
| HbA1cb - Plaque index          | 5.77 | 1.54           | 26.474  | 0.63    | 0.000   |
| HbA1cb - Gingival index        | 5.55 | 1.58           | 24.799  | 0.57    | 0.000   |
| HbA1cb - Sulcus bleeding index | 4.92 | 1.49           | 23.286  | 0.64    | 0.000   |

**Table 6: The Baseline comparison in mean, STD, T value, R value and P value of HbA1c's Plaque index, Gingival index and Sulcus bleeding index**

| Baseline comparison           | Mean | Std. Deviation | T value | R value | P value |
|-------------------------------|------|----------------|---------|---------|---------|
| HbA1c - Plaque index          | 6.64 | 1.74           | 26.916  | 0.34    | 0.000   |
| HbA1c - Gingival index        | 6.50 | 1.70           | 26.946  | 0.42    | 0.000   |
| HbA1c - Sulcus bleeding index | 6.69 | 1.66           | 28.487  | 0.38    | 0.000   |

## Discussion

The question of whether periodontal treatment has an effect on metabolic control of diabetes is not new or recent. Intuitively, this question has been

addressed from the standpoint of degree of diabetes severity control. [9] The escalating human and economic burden of diabetes requires a multidisciplinary approach for the prevention, diagnosis and management of the disease and its

complications including periodontitis. Regarding increased susceptibility to periodontitis in people with diabetes, this has a number of implications for dental management. Firstly, it is important that diabetic patients are aware of the potential impact their condition may have on their oral and periodontal health. Patients who are newly diagnosed with diabetes should be told about this impact and we should continue to urge our medical colleagues to recommend a dental examination to their patients. Periodontitis is a progressive inflammatory disease of the periodontal tissues, namely gingiva, periodontal ligament, and alveolar bone (tooth-supporting bone). It begins with gingivitis in response to the microorganisms in dental plaque and progresses to periodontitis characterized by soft-tissue inflammation, loss of attachment between the periodontal tissues and teeth, and alveolar bone recession. [10] The loss of tissue attachment and bone resorption in the periodontal tissues is orchestrated through collagen breakdown by the reactive oxygen molecules, proteolytic enzymes, and matrix metalloproteinases synthesized by the neutrophils in response to inflammation. [11]. Inability to achieve glycemic control in T2DM leads to impaired function of neutrophils, accumulation of advanced glycation end products, and oxidative stress pathways, thereby contributing to an increased risk for developing periodontitis. [12]

The peculiar aspect of periodontitis in diabetic patients lies in the fact that the body's immune system, instead of completely eliminating the source of inflammation (microorganisms), keeps the inflammatory process continuously activated, leading to a chronic inflammatory reaction. [11] This chronic inflammatory response leads to systemic upregulation of pro-inflammatory cytokines such as interleukins (IL-1 $\beta$ , 4, 6, 8, and 10) and tumor necrosis factor- $\alpha$ . [13,1] These pro-inflammatory

cytokines have been reported to play a role in inducing insulin resistance, initiating pancreatic beta-cell destruction, and altering lipid metabolism, leading to hyperlipidemia with low-density lipoproteins and triglycerides. [1] Based on a long-term prospective study among nondiabetic patients, Saito *et al.* [14] reported that patients with periodontitis had significantly higher frequency of impaired glucose tolerance in comparison to patients without periodontitis. All of these findings make it alluring to hypothesize a causative role for periodontal inflammation, resulting in poor glycemic control.

The potential implications of the present study were aimed toward periodontal health promotion as an adjunct to achieve better glycemic control among T2DM patients. Unlike prevention of tooth decay, dental public health activities do not offer primary focus toward prevention of PDs, recognizing populations at risk and mitigating its prevalence. [15] The present study results imply the importance of routine screening for PD among diabetic patients as they not only belong to a specific population at risk for developing periodontitis, but also alleviation of periodontal inflammation might help them achieve good glycemic control. Although it may be alluring to hypothesize and recommend that treating periodontitis among T2DM patients can help achieve glycemic control (HbA1c <7%), further multicenter, controlled clinical trials in diverse populations are required to definitively establish this causal relationship. [16]

### Conclusion

The data in the current study have been interpreted to suggest that Non-Surgical Periodontal therapy is associated with improvement in the periodontal status along with the improvement in the glycemic control in patients with T2DM. The clinical improvement obtained was accompanied by a significant reduction in

HbA1c values in type II Diabetes Mellitus patients, confirming the existing interrelationship between DM and periodontal disease. Therefore, periodontal treatment should be included in preventive measures for treating Type II Diabetes Mellitus.

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