International Journal for Multidisciplinary Research (IJFMR)
E-ISSN: 2582-2160 ● Website: www.ijfmr.com ● Email: editor@ijfmr.com

Outcomes of Periodontal Treatment on Patients with Type 2 Diabetes Mellitus and Periodontal Disease's Metabolic Control

Shabana Tabbassum

Graduate Student, Career Institute of Dental Sciences and Hospital, Lucknow, Uttar Pradesh, India.

Abstract

Introduction: Periodontal disease (PD) is a chronic inflammatory disease that destroys the gingiva or tooth-supporting tissues.

Methodology: 260 patients from the diabetes clinic were referred to the dental clinic for treatment. 92 patients, or 35% of the type 2 DM patients who received contact letters, began dental treatment. 169 people were left, and 65% of them didn't make a dental appointment. The therapeutic goal of dental therapy was to get rid of infections in the periapical areas of teeth and from periodontal disorders**.**

Results: The frequency distribution of the percent pre/post changes in HbA1C in the control and treatment groups is compared in Table 3 between the two groups. Ten control individuals and five treatment subjects, out of each group's 36 participants, showed essentially no change in HgA1c levels (o5%) during the investigation's 10-month timeframe. 10 control subjects and 4 treatment subjects experienced worsening glucose control during this time. In contrast, glucose control significantly got better in 27 treatment patients and 16 control subjects.

Conclusion: We interpret the data in the study to suggest that periodontal therapy was associated with improved glycemic control in persons with type 2 DM.

Keywords: Type 2 Diabetes Mellitus, Hba1c, Periodontal Therapy

Introduction

Periodontal disease (PD) is a chronic inflammatory disease that destroys the gingiva or tooth-supporting tissues. It is one of the most prevalent chronic infections in adults between ages 30 and 90 years in the United States and the most prevalent dental disease in people with diabetes, affecting up to 22% of diabetic patients.[1] According to reports, PD affects 90% of individuals worldwide.[2] Bacterial plaque causes the two main types of PD, gingivitis and periodontitis, that eventually lead to the destruction of gingival tissue and periodontal attachment structures.[3]

Although tooth-adherent microbes cause PD to start, the illness's chronic characteristic and final tissue disintegration are caused by an individual's inflammatory response and coexisting persistent illnesses such diabetes, coronary artery disease, and chronic pulmonary problems. These circumstances imply that

T2DM, or type 2 diabetes mellitus, is a risk factor for periodontitis.[1] Individuals with T2DM have an elevated incidence, prevalence, and severity of periodontitis, according to epidemiological investigations conducted over decades.[4] Incidence of periodontitis increases as diabetic patients age and is both more frequent and more severe in T2DM patients with advanced systemic complications.[5]

Investigations involving PD patients with diabetes have investigated a variety of periodontitis and gingivitis treatments that aim to lessen mouth bacteria and related calculus. Patients with gingivitis who don't also have a condition that affects their oral health may benefit from just improving their own plaque control utilising a combination of mechanical and sanitary procedures.[6] However, self-care alone is uneven and rarely maintains plaque-free state, hence it is typically advisable to seek expert reinforcement.[6]

The goal of treating periodontitis is to eradicate harmful bacteria and prevent their recolonization, as well as to address modifiable risk factors like stress, alcoholism, smoking, and drug abuse, as well as to manage systemic illnesses like autoimmune disorders, diabetes, cardiovascular disease, lung disorders, and osteoporosis.[7]

There have been numerous studies that have found a positive relationship between poor glycemic control in persons with type 2 DM and increased periodontitis (Bissada et al. 1982, Sorsa et al. 1992, Tervonent & Oliver 1993, Mendieta & Reeva 1993, Saflam-Seppala & Ainamo 1992, Loe 1993, Finestone & Boorujy 1967, Bacic et al. 1988, Hove & Stallard 1970, Wolf 1977, Rayfield et al. 1982, Wheat 1980). Conversely, limited data is available suggesting that periodontitis affects glycemic control in individuals with type 2 DM.

Insulin resistance in the tissues is a well-known co-morbidity of infections (Vki-Jarien 1989). According to a recent investigation, insulin resistance rose by 33% during the acute stage of a bacterial infection and by 28% throughout the convalescence period (Sammalkorpi 1989). Furthermore, according to Grossi et al. (1996), diabetic patients may develop insulin resistance and poor metabolic regulation as a result of ongoing Gram-negative infections and persistent endotoxemia, such those found in periodontal conditions. These data have led to the hypothesis that reducing periodontal infections helps with diabetes metabolic management. In the present investigation, we compared the changes in glycemic control between a group of type 2 DM patients who had received periodontal treatment and a type 2 DM control group who had not.

Methodology

The Department of Periodontology at the Career Institute of Dental Sciences and Hospital in Lucknow conducted the investigation. In addition, 260 patients from the diabetes clinic were referred to the dental clinic for treatment. 92 patients, or 35% of the type 2 DM patients who received contact letters, began dental treatment. 169 people were left, and 65% of them didn't make a dental appointment. The therapeutic goal of dental therapy was to get rid of infections in the periapical areas of teeth and from periodontal disorders. By April 2022, 36 patients with type 2 diabetes had completed the recommended extractions and root planing procedures as part of phase 1 periodontal therapy. Haemoglobin A1C (HbA1C) values taken prior to and after dental procedures were separated by an average of ten months. As a result of their failure to respond to the initial appointment mailings, a pool of patients was employed to choose the control

group.

Data collection

When the medical records of this cohort were examined, it was shown that 65 patients had similar (8–10 month) intervals between two HbA1C readings. A random selection of 36 of these patients made up the control group, which received no dental care. Next, medical records for both groups were examined to look for information on HbA1C levels, medication types, dose variations, trips to the diabetes clinic, fluctuations in body weight, and histories of visits to healthcare professionals other than dentists for infections that weren't dental-related. HbA1C, expressed in percentage, and assessed by high-pressure liquid chromatography were used to calculate the glucose control. Clinical information, such as carious lesions, missing teeth, periodontal probing depths, gingival hemorrhage, and calculus accumulation was documented for the patients who presented to the dental clinic (treatment group). A treatment strategy was developed that included treating periodontitis and extracting teeth with extreme alveolar bone loss or periapical infections.

All patients got advice on maintaining good dental health as well as root planning, subgingival curettage, and full mouth scaling under local anaesthesia. Teeth that had enough periodontal damage and periapical radiolucencies to be regarded unsalvageable were extracted. 25% of the extracted teeth showed periapical lesions from caries, and 75% of the teeth had severe periodontitis. Because of the periapical involvement, all of the teeth recommended for extraction also had moderate to severe periodontal disease. 34 of the 81 initial patients who began dental therapy and underwent all recommended extraction and periodontal procedures were successful. Concerning the dental health of the control group, nothing was known.

Characteristics of the subjects

A comparison of the treatment and control groups is presented in Table 1. For initial HbA1C values, visits to the diabetic clinic during the investigational period, the interval among the first and second HbA1C levels, the percentage of patients who received diet control, oral hypoglycemic medication, or insulin, and the distribution of ethnic groups, these 2 groups were closely comparable. The age of the control individuals (67.3o10.8) compared to that of the treatment patients (62.4o8.4) was the only statistical difference between these groups. This discovery led us to investigate the connection between age and HbA1C levels. We tested this link using a factorial ANOVA and an age stratification. Age and initial HbA1C levels did not significantly differ between or among these groups, according to statistics. Thus, in subsequent analyses, age was not included as a covariate.

Results

After finishing dental work, the HbA1C values in the treatment group dropped from 9.5 to 7.6 (17.1%). The HbA1C levels in the control group likewise dropped from 8.5 to 7.7 (6.7%) during the same time period, and both decreases were statistically significant (p 120.0001 and p 120.02, respectively) (Table 2). Additionally, there was a statistically significant difference in changes between the two groups (F 125.57, p 120.02). The frequency distribution of the percent pre/post changes in HbA1C in the control and treatment groups is compared in Table 3 between the two groups. Ten control individuals and five treatment subjects, out of each group's 36 participants, showed essentially no change in HgA1c levels (o5%) during the investigation's 10-month timeframe. 10 control subjects and 4 treatment subjects

experienced worsening glucose control during this time. In contrast, glucose control significantly got better in 27 treatment patients and 16 control subjects.

Effects of the Method of Hyperglycemic Control

The participants in both groups were then divided into groups based on how they were managing their hyperglycemia (i.e., using insulin, diet, or oral hypoglycemic drugs). Table 4 displays the total number of patients subjected to each of these three regimes. For the diet, oral hypoglycemic, and insulin treatment groups, the HbA1C values dropped from 7.6 to 6.6 (16%), 9.4 to 7.7 (18%), and 10.3 to 8.4 (18%), correspondingly. Subjects taking oral hypoglycemics (p 120.0005) and insulin (p 12 0.003) in the therapy group were the only changes that were statistically significant.

The HbA1C levels for the same groups in the control category reduced from 9.5 to 8.5 (10%), from 8.1 to 7.4 (8.7%), and from 7.7 to 6.8 (13.7%). Statistics showed that the changes in the control group were considerable. We investigated 3 potential modifiers of HbA1C variations between groups:
(1) Treatment of non-dental infections.
(2) Changes in medication to improve glycemic control.
(3) Changes in body weight.

No participants from either group sought treatment for infections from any non-dental healthcare professionals throughout the time period under consideration, according to an analysis of their medical records. An increase in oral drugs was seen in 8 of 19 and in 10 of 22 patients. 20 members of the therapy group put on weight, 2 remained the same, and 12 people lost weight. Between-group variations in weight changes were not statistically significant (c 2123.62, p 120.16). Additionally, weight changes did not mitigate the drop in HbA1C after dental work (F 1–20.6, p 1–20.55).

Table 1: Clinical Characteristics of the Study Subjects

Factor	n	M∫SD	M∫SD	n
Age (years)*		67.0∫10.8	62.4∫8.4	
Starting HbA1C		8.5∫2.1	9.2∫2.2	
Visits to Diabetic Clinic		3.6∫1.5	4.0∫2.1	
D time 1st and 2nd HbA1C (months)		11.7∫5.8	10.0∫3.3	
Diabetic Control Diet	6			4
Oral Medications	19			22
Insulin Ethnicity	11			10
African American	11			13
Asian	3			2
Caucasian	8			6
Hispanic	14			15

* pΩ 0.05

Table 2: Pre/post Changes in HgA_{1C}

	Initial HbA_{1C}		Final HbA_{1C}		HbA_{1C}*			
Group	**R**	**M∫SD**	**R**	**M∫SD**	**R**	**M∫CI**	**p value****	**p value*****
Control	5.6–14.6	8.5∫2.1	3.5–11.8	7.7∫1.4	[a]3.4–6.4	0.8∫0.6	0.02	0002
Treatment	5.1–15.9	9.5∫2.2	5.0–11.5	7.6∫1.4	[a]2.1–6.9	1.9∫0.3	0.0001	

RΩ range; CIΩ 95% confidence interval; MΩ mean ∫; SDΩ standard deviation.
* % change of the initial to the final HbA_{1C}.
** Comparison of the initial to the final HbA_{1C}.
*** Comparison of the % changes in HbA_{1C} in the control and the treatment groups.

Table 3: Frequency Distributions in Pre-post Changes in HbA_{1C}

% Change	Control	Treatment HbA_{1C}
([a]45)–([a]36)	2	
([a]35)–([a]26)		1
([a]25)–([a]16)	3	
([a]6)–(15)	5	3
([a]5)–(π5)	10	5
(6)–(15)	9	4
(16)–(25)	2	9
(26)–(35)	2	6
(36)–(45)	2	5
(46)–(55)	1	

Table 4: Changes in Hb_{A1C} between the Control and Treatment Groups Stratified by Glycemic Control

	Glycemic	Control HbA_{1C}					Treatment HbA_{1C}			
N	**Control**	**Initial**	**Final**	**D**	**p-value**	**n**	**Initial**	**Final**	**D**	**p-value**
6	Diet	7.7∫1.5	6.8∫1.3	0.9	0.16	4	7.6∫2.2	6.6∫0.9	1.0	0.5
19	Oral	8.1∫2.0	7.4∫1.6	0.6	0.18	22	9.4∫2.4	7.7∫1.2	2.0	0.0005
11	Insulin	9.5∫2.2	8.5∫1.3	1.0	0.17	10	10.3	8.4∫1.5	1.9	0.003

Mean∫SD

Discussion

The findings of this research indicate that when compared to a non-treatment control group, people with type 2 DM showed a substantial rise in glycemic control after receiving treatment for periodontal disease. It appears that these differences represented broad changes among the groups and were not caused by significant changes among a small number of participants when the frequency distribution of the pre/post

variations in glucose control were analysed among subjects in the two groups. It's interesting to note that 7 out of 9 patients who received periodontal care had lower insulin needs, according to Williams & Mahan (1960)[8], in a non-controlled trial. In a different uncontrolled research, the treatment of gingival inflammation improved the glycemic control in nine individuals.

Two recent investigations have used experimental and quasi-experimental approaches to investigate the connection between periodontitis and glycemic control in the Pima Indian community, a population previously known to have a high prevalence of type 2 diabetes. In the first trial, 105 participants with HgA1C levels of 9% between the ages of 18 and 65 were chosen. This group of people underwent periodontal examinations, were categorised as having severe periodontal disease or not, and were then observed for two years. At follow-up visits, there was a higher risk of poor glycemic control for patients with severe periodontitis at baseline. Other variables that had an impact on this outcome included:
(1) baseline age,
(2) level of glycemic control at baseline,
(3) duration of diabetes,
(4) smoking and
(5) an interaction between age and periodontal disease status (Nelson et al. 1990).[9]

The effectiveness of systemic doxycycline and topical antimicrobial therapy on periodontal disease and glycemic control was examined in the second research, which included 85 patients with severe periodontal disease who were randomly assigned to one of four groups (Grossi et al. 1996).[10] At 3 months, the HbA1C levels in the groups treated with doxycycline significantly decreased, whereas the levels in the control group remained same. The degree of diabetic metabolic control was assessed by the authors to be negatively impacted by periodontal diseases. There were no statistically significant differences when we looked at weight and medication changes as mediators of glycemic control. The number of participants in these groups, however, was insufficient to generate the statistical power required to reliably assess these variables.

The research's overall 6.7% improvement in glucose management in the control group was an intriguing discovery. There's no clear explanation for this. Changes in the Diabetic Clinic's regimen for managing type 2 DM could be one reason. About the same time the trial started, healthcare professionals in this clinic started using HbA1C results as a reference for medication adjustments. All patients' medication adjustments up until this point had been based on their fasting blood glucose readings. Therefore, the enhancement seen in the control group may signify improved diabetic treatment. It's likely that people in the therapy group will also benefit from this improvement in diabetes management. Nevertheless, compared to the control group, the treatment group saw a statistically significant 17% improvement in glucose management. Only the participants receiving oral hypoglycemics and insulin in the experimental group showed reductions in HbA 1C that were statistically significant when both groups were stratified based on diabetic treatment. Perhaps those with worse glycemic control show the most benefit after periodontal treatment. Among the treatment and control groups, Table 1 demonstrates a good match for the majority of variables. However, this study did not look into the dental health of the control group. Furthermore, we do not know whether or not individuals in the control group sought dental care elsewhere

during the study period despite receiving contact letters from the dental clinic. It is doubtful that many individuals sought dental care elsewhere given that this group received free dental care at the clinic.

Due to its quasi-experimental and retrospective form, this study's limitations pose risks to internal validity. Additionally, the small sample size made it impossible to establish the statistical power required to accurately assess the potential moderating effects of drug modifications and body weight on glucose management. The results of this investigation, in our opinion, call for additional research on the part of patients with type 2 diabetes using a randomised prospective design and a larger sample size.

Conclusion

We interpret the data in the study to suggest that periodontal therapy was associated with improved glycemic control in persons with type 2 DM.

References

1. Albandar JM, Brunelle JA, Kingman A. Destructive periodontal disease in adults 30 years of age and older in the United States 1988–1994. J Periodontol. 1999;70:13–29.
2. Cullinan MP, Ford PJ, Seymour GJ. Periodontal disease and systemic health: current status. Aust Dent J. 2009;54:1S62–9.
3. American Academy of Periodontology. The pathogenesis of periodontal diseases. Periodontol. 1999;70:457–70.
4. Loe H. Periodontal disease: the sixth complication of diabetes mellitus. Diabetes Care. 1993;16:329–34.
5. Mealey BL, Oates TW. Diabetes mellitus and periodontal diseases. J Periodontol. 2006;77:1289–1303.
6. American Academy of Periodontology Research Therapy Committee. Treatment of plaque-induced gingivitis, chronic periodontitis, and other clinical conditions. Pediatr Dent. 2004;35:13–14.
7. Serio FG, Duncan TB. The pathogenesis and treatment of periodontal disease. 2010. http://www.ineedce.com
8. Williams, R. C. & Mahan, C. J. (1960) Periodontal disease and diabetes in young adults. JAMA 172, 776–778.
9. Nelson, R., Shlossman, M., Budding, L., Pettitt, D., Saad, M., Genco, R. & Knowler, W. (1990) Periodontal disease and NIDDM in Pima Indians. Diabetes Care 13, 836–40.
10. Grossi, S. G., Skrepcinski, F.B., DeCaro, T., Zambon, J.J., Cummin, D. & Genco, R. J. (1996) Response to Periodontal Therapy in Diabetics and Smokers. J Periodontol 67, 1094–1102.