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The effect of improved periodontal health on metabolic control in type 2 diabetes mellitus

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Abstract

Objectives: The aim of the present study was to investigate the effect of improved periodontal health on metabolic control in type 2 diabetes mellitus (DM) patients.

Material and Methods: Fourty-four patients with type 2 DM were selected. Subjects were randomly assigned into two groups.

Data collection: Plaque index (PI), gingival index (GI), probing pocket depth (PPD), clinical attachment levels (CALs), gingival recession (GR) and bleeding on probing (BOP) were recorded at baseline at 1st and 3rd months.

Fasting plasma glucose (FPG), 2-h post-prandial glucose (PPG), glycated haemoglobin (HbA1c), total cholesterol (TC), triglyceride (TG), HDL-cholesterol, LDL-cholesterol and microalbuminure were analysed at baseline, 3 months following the periodontal therapy. The treatment group received full-mouth scaling and root planing whereas the control group received no periodontal treatment.

Results: A statistically significant effect could be demonstrated for PI, GI, PPD, CAL and BOP for the treatment group. HbA1c levels in the treatment group decreased significantly whereas the control group showed a slight but insignificant increase for this parameter.

Conclusions: The results of our study showed that non-surgical periodontal treatment is associated with improved glycaemic control in type 2 patients and could be undertaken along with the standard measures for the diabetic patient care.

Key words: metabolic control; periodontal therapy; type 2 diabetes mellitus

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Diabetes mellitus (DM) and chronic periodontitis are common chronic diseases in adults in the world population. DM is a complex disease with both metabolic and vascular components, characterized by hyperglycaemia due to defects in insulin secretion, insulin action or both. Dysregulation of protein and lipid metabolism also occurs (Expert Committee on the Diagnosis and Classification of Diabetes Mellitus 2000). Patients suffering from DM are known to have increased susceptibility to certain infections. Infections, as they lead to poor metabolic control in diabetes, are of great concern since it has been shown that hyperglycaemia and poor metabolic control result in increased diabetic complications of the eye, kidney and nerves (Clark & Lee 1995).

The interrelationships between periodontitis and diabetes provide an example of systemic disease predisposing to oral infection, and once that infection is established, the oral infection exacerbates systemic disease.

The prevalence of periodontal disease among individuals with inadequately controlled type 2 diabetes is generally higher than that of people free of systemic disorder (Ueta et al. 1993, Shlossman 1990).

The diabetic state impairs the gingival fibroblast synthesis of collagen and glycosaminoglycan, enhances crevicular fluid collagenolytic activity, results in the loss of periodontal fibres, loss of the alveolar supporting bone, loosening and finally exfoliation of the teeth (Iacono et al. 1985, Collin et al. 1998).

Diabetes-induced changes in immune cell function produce an inflammatory immune cell phenotype (upregulation of proinflammatory cytokines from monocytes/polymorphonuclear leucocytes and downregulation of growth factors from macrophages). This predisposes to chronic inflammation, progressive tissue breakdown and diminished tissue repair capacity (Iacopino 2001). Recent studies demonstrate that hyperlipidaemia may be one of the factors associated with diabetes-induced immune cell alterations (Salvi et al. 1997). These human studies have established a relationship between high-serum lipid levels and

periodontitis (Cutler et al. 1999a, b). Clinical and epidemiological evidence demonstrates that individuals with diabetes tend to have a higher prevalence and more severe periodontitis than non-diabetics (Cianciola et al. 1982, Safkan-Seppala & Ainamo 1992). Furthermore, patients with poor control of diabetes experience more periodontitis than well-controlled diabetics (Tervonen & Knuuttila 1986, Seppala & Ainamo 1994).

Recent studies illustrated the synergistic relationship between diabetes and periodontitis. Severe periodontitis was associated with poor glycaemic control and exacerbated diabetes-induced hyperglycaemia (Taylor et al. 1996). Although it has been reported that improved metabolic control may lead to improved periodontal health (Sastrowijoto et al. 1990), it is still unclear whether the control of periodontal infections may improve the metabolic control of diabetes.

The aim of the present study was to investigate the effect of improved periodontal health on metabolic control in type 2 DM patients.

Material and Methods

Subjects

This clinical study was carried out as a joint collaboration between Department of Metabolic Diseases and Endocrinology of Ankara University, Faculty of Medicine and Department of Periodontology of Ankara University, Faculty of Dentistry. The study was reviewed and approved by the Ethical Committee of Ankara University, Faculty of Dentistry. Subjects were selected randomly from the pool of treated and maintained patients in the Department of Metabolic Diseases and Endocrinology of Ankara University, Faculty of Medicine.

The criteria for inclusion in the study were:

(1) patients with type 2 DM with glycated haemoglobin (HbA1c) values: 6%–8%;
(2) creatinine values < 1.4 mg/dl;
(3) liver function tests were not up to three times the normal range;
(4) no major diabetic complications;
(5) no history of systemic antibiotic administration within the last 3 months;
(6) no periodontal treatment 6 months prior to the study.

Fourty-four subjects fulfilling these criteria signed an informed consent form. These subjects were 26 (59%) women and 18 (41%) men, with the mean age of 54.39 ± 11.72 years. Subjects were randomly assigned into two groups as treatment and non-treatment (control) groups. The treatment group consisted of 12 (55%) women and 10 (45%) men and the age range of 31–79 (mean age 55.95 ± 11.21) years. For the treatment group, the mean diabetes duration was 9.32 ± 8.36 years. And the control group consisted of 14 (64%) women and eight (36%) men with the age range of 31–79 (mean age 52.82 ± 12.27) years. For the control group, the mean diabetes duration was 8.05 ± 5.90 years.

Data related to brushing habits, duration of their diabetes, denture usage, number of missing teeth are recorded. Nine patients in the treatment and seven patients in the control group had fixed crown and bridge restorations. Seven patients in the treatment and 10 patients in the control group were not using any dentures. Only three patients in the treatment and two patients in the control group had removable partial dentures.

Five patients in the treatment and two patients in the control group were smokers. The mean tooth loss for both groups was 13.

The clinical characteristics of the study population are shown in Table 1.

For assessing the effect of the periodontal treatments on metabolic control, no change in the medication or diet was made for both groups during the study period. None of the groups received any additional guidance in managing their diabetic status. The subjects of both groups were analysed according to the methods used to control hyperglycaemia. The percentage of patients treated by these regimes is presented in Table 2.

Periodontal treatment

All subjects underwent periodontal examination by a single examiner. The patients received oral hygiene instructions and full-mouth scaling and root planing performed under local anaesthesia.

Five patients in the control group had five teeth with periapical lesions where they received root canal treatment. In the treatment group nine patients had nine teeth with periapical lesions. four teeth were extracted and five had root canal treatment. Periodontal parameters were recorded following the extractions.

All subjects underwent periodontal examination by a single examiner. The patients received oral hygiene instructions and full-mouth scaling and root planing performed under local anaesthesia.

The control group received no periodontal treatment during the study period. After completion of the study, these patients were given a full non-surgical and supportive periodontal treatment if needed.

Table 1. Patient characteristics of the study and control subjects

	Age (mean ± SD)	Male/female (%)	Diabetes uration (years) (mean ± SD)	Daily brushing habit (times/day) (%)	Smoking (*n*/day)	Denture (*n*)	Missing tooth (mean ± SD)
Treatment ($n = 22$)	55.95 ± 11.21	46/54	9.32 ± 8.36	**0** 27.2 **1** 36.4 **2** 36.4	5	15	13.23 ± 6.73
Control ($n = 22$)	52.82 ± 12.27	36/64	8.05 ± 5.90	**0** 50 **1** 27.3 **2** 22.7	2	12	12.86 ± 6.78
Total ($n = 44$)	54.39 ± 11.27	41/59	8.68 ± 7.18	**0** 38.6 **1** 31.9 **2** 29.5	7	27	13.5 ± 6.76

Table 2. Distribution of methods used to control hyperglycaemia

Group	Diet only (%)	Sulphanylurea (%)	Biguanides (%)	Acarbose (%)	Oral hypoglycaemic combinations (%)	Insulin (%)	Oral hypoglycaemic combinations+insulin (%)
Treatment	9.1	4.4	9.1	9.1	40.9	9.1	18.3
Control	4.5	13.5	18.3	0	40.9	9.1	13.7
Total	6.8	9.1	13.7	4.5	40.9	9.1	15.9

Data collection

The periodontal parameters were recorded at baseline (day 0) and at 1st and 3rd months following the periodontal treatment in both groups. The first month following root planing, periodontal parameters were recorded to identify the surgical treatment needs of the study groups. The data concerning the group of patients who had surgical treatment were excluded in the statistical analysis. Periodontal measurements were performed by a single examiner. The examining investigator was unaware of the group assignments.

Recordings were made from the buccal, lingual and two interproximal surfaces of each tooth.

The parameters recorded were:

(1) Plaque index (PI): Recordings for plaque were made for each tooth according to the criteria for the PI (Silness and Löe 1964).
(2) Gingival index (GI): Recordings for gingival status were made for each tooth according to the criteria for the GI (Löe and Silness 1963).
(3) Probing pocket depth (PPD), clinical attachment level (CAL) and
(4) gingival recession (GR): Recordings for PPD, CAL and GR were measured with a Williams's periodontal probe using customized occlusal acrylic stents.
(5) Bleeding on probing (BOP): The % of bleeding sites was assessed by gentle probing of the bottom of the pockets using a periodontal probe.

For the metabolic assessment, venous blood samples were taken from each patient and analysed for fasting plasma glucose (FPG), 2-h post-prandial glucose (PPG), glycated haemoglobin (HbA1c), total cholesterol (TC), triglyceride (TG), HDL-cholesterol (HDL), LDL-cholesterol (LDL) and microalbuminurea analysis.

Metabolic measurements were performed in baseline and at 3rd month following the periodontal treatment in the treatment group and in baseline, 1st and the third months in the control group. Metabolic laboratory assessments were performed in Endocrinology and Central Laboratories of Ankara University, Faculty of Medicine.

Statistical analysis

The statistical analysis was performed using SPSS software program.

The Student *t*-test was used to test the differences of age, sex, diabetic control methods, tooth brushing frequency, number of missing teeth and the duration of denture use between the treatment and control groups. The changes of PI, GI, PPD, CAL, GR and BOP values from baseline to 3rd month within both groups were compared using Wilcoxon signed ranks test.

The evaluation of differences for periodontal parameters in time for the treatment and the control groups was compared by using the Mann–Whitney *U*-test and Student *t*-test. The significance of the metabolic parameters within the groups was assessed by Wilcoxon signed ranks test. Non-parametric Mann–Whitney *U*-test was used to compare the changes in metabolic parameters between the treatment and the control groups. In the treatment and the control groups, correlations between HbA1c and BOP changes were assessed using Pearson's correlation coefficient.

Results

Fourty-four subjects comprised our study sample. The clinical characteristics of the study population are shown in Table 1.

At baseline, treatment and control groups had similar mean values for age, sex, medications, duration of diabetes, tooth-brushing frequency, smoking habits, denture usage and number of missing teeth.

Periodontal parameters

Following the treatment the PI dropped significantly from 1.60 ± 0.63 to 0.29 ± 0.17, and the GI dropped from 0.94 ± 0.47 to 0.26 ± 0.18 in the treatment group.

The PI dropped from 1.63 ± 0.84 to 1.54 ± 0.88 and the GI dropped from 0.87 ± 0.87 to 0.84 ± 0.52 in the control group. Although there was an increase in these parameters in the 3rd month measurements, they were not found to be statistically significant.

For the pocket depths at baseline in both patient groups, the examined sites showed 3–4 mm of pocket depth. After scaling and root planing, a statistically significant improvement was observed in the treatment group whereas minor changes in PPD categories were found for the control group. Statistically significant differences between both groups for two examination time points are shown in Table 3.

Assessment of CAL in the two groups revealed a significant attachment gain of 0.39 mm for the treatment group and 0.05 mm attachment gain for the control group at the end of 3 months. There were no statistically significant differences between the two groups regarding the change in the attachment level during the observation period.

BOP revealed periodontal inflammation at baseline in 54% of treatment and 50% of the control group. Non-surgical periodontal treatment caused a significant decrease in the treatment group whereas BOP values were found to increase slightly for the control group.

Metabolic parameters

Table 4 shows the metabolic data for the treatment and the control groups. There were no statistically significant differences between the two groups associated with all metabolic parameters at baseline.

The baseline mean FPG was 132.82 ± 31.85 for the treatment and 139.55 ± 35.33 for the control group.

Table 3. Clinical periodontal status in treatment and control groups at baseline and 3rd month

	PI (mean ± SD)	GI (mean ± SD)	PPD (mm) (mean ± SD)	CAL (mm) (mean ± SD)	GR (mm) (mean ± SD)	BOP (%) (mean ± SD)
Treatment						
Baseline	1.60 ± 0.63	0.94 ± 0.47	2.29 ± 0.49	3.19 ± 1.13	0.94 ± 0.79	54.38 ± 18.75
3rd month	0.29 ± 0.17	0.26 ± 0.18	1.80 ± 0.25	2.80 ± 1.03	1.04 ± 0.95	23.90 ± 12.73
Δ	− 1.31	− 0.68	− 0.49	− 0.39	0.10	− 30.48
p^*	0.000	0.000	0.000	0.000	0.339	0.000
Control						
Baseline	1.63 ± 0.84	0.87 ± 0.47	2.24 ± 0.70	2.92 ± 1.10	0.73 ± 0.60	50.48 ± 26.1
3rd month	1.54 ± 0.88	0.84 ± 0.51	2.26 ± 0.63	2.87 ± 1.03	0.70 ± 0.56	51.91 ± 27.38
Δ	− 0.9	− 0.03	0.02	− 0.05	− 0.03	1.43
p^*	0.414	0.372	0.794	0.381	0.436	0.330
p^{**}	0.000	0.000	0.005	0.742	0.291	0.000

Δ: changes in pre- and post-treatment.
p^*: comparison of baseline and 3rd month data.
p^{**}: comparison of the changes in periodontal data between treatment and control groups.
PI, plaque index; GI, gingival index; PPD, probing pocket depth; CAL, clinical attachment level; GR, gingival recession; BOP, bleeding on probing.

Table 4. Mean (± SD) metabolic data for treatment and control groups

	Glycated haemoglobin (HbA1c) (%)	Fasting plasma glucose (mg/dl) (mean ± SD)	2-h post-prandial glucose (mg/dl) (mean ± SD)	Total cholesterol (mg/dl) (mean ± SD)	Triglyceride (mg/dl) (mean ± SD)	HDL-cholesterol (mg/dl) (mean ± SD)	LDL-cholesterol (mg/dl) (mean ± SD)
Treatment							
Baseline	7.31 ± 0.74	132.82 ± 31.85	168.95 ± 42.20	187.14 ± 38.39	136.68 ± 98.17	51.59 ± 13.94	113.64 ± 24.22
3rd month	6.51 ± 0.80	128.86 ± 29.13	145.36 ± 52.92	183.14 ± 31.13	122.77 ± 55.87	53.00 ± 15.37	110.68 ± 31.19
Δ	− 0.86	− 3.96	− 23.6	− 4.00	− 13.91	1.41	− 2.96
$p*$	0.000	0.284	0.027	0.157	0.297	0.151	0.794
Control							
Baseline	7.00 ± 0.72	139.55 ± 35.33	162.0 ± 55.77	179.09 ± 35.01	130.68 ± 68.51	45.64 ± 13.16	107.27 ± 32.58
3rd month	7.31 ± 2.08	140.77 ± 39.33	164.18 ± 75.54	190.32 ± 37.22	165.09 ± 107.18	51.27 ± 14.15	106.86 ± 38.94
Δ	0.31	1.22	1.5	0.72	34.41	5.63	− 0.41
p^*	0.684	0.884	0.614	0.498	0.092	0.019	0.730
p^{**}	0.033	0.481	0.067	0.963	0.033	0.345	0.548

Δ: changes in pre- and post-treatment.
p^*: comparison of baseline and 3rd month data.
p^{**}: comparison of the changes in metabolic data between treatment and control groups.

There was a tendency towards a decrease in the treatment group (128.86 ± 29.13) whereas this change was not found to be statistically significant although FPG remained unchanged for the control group. There was no statistically significant difference between these two groups.

Two hour PPG levels decreased in the treatment group from 168.95 ± 42.20 to 145.36 ± 52.92 whereas this parameter remained unchanged for the control group.

The normal range for the HbA1c subjects without diabetes is 4.5–6.0%. In our study, population the mean standardized HbA1c values were 7.31 ± 0.74 for the treatment and 7.00 ± 0.72 for the control group. Both groups showed moderate metabolic control at baseline. The treatment group showed a reduction in HbA1c to 6.51 ± 0.80. This reduction equates to a level approximately 10.94% of the baseline HbA1c level. The control group showed 4.42% increases in levels of HbA1c compared with baseline whereas this was not found to be statistically and clinically significant. In other words, the reductions in the HbA1c levels in the treatment group did last beyond the 3-month period.

There was a slight decrease in the cholesterol levels in the TC levels in the test group (187.14 ± 38.94 to 183.14 ± 31.13) that was not statistically significant. The TC level increased slightly in the control group from 170.09 ± 35.01 to 190.32 ± 37.22 ($p > 0.01$).

The TG levels in the treatment group decreased from 136.68 ± 98.16 to 122.77 ± 55.87 following the completition of the periodontal treatment. Although not statistically significant, the TG levels in the control group increased from 130.68 ± 68.51 to 165.09 ± 107.18. There was a significant difference between the study groups because of a slight decrease in the treatment group and a slight increase in the control group.

The treatment group showed a slight increase in the HDL cholesterol levels compared with baseline (51.59 ± 13.94–53.00 ± 15.37). On the contrary, there was a slight decrease in the LDL cholesterol level (113.64 ± 24.22–110.68 ± 31.19). The changes from the baseline were not found to be statistically significant.

The control group showed a slight increase in the HDL cholesterol level (45.64 ± 13.16–51.27 ± 14.15) and a slight decrease in the LDL cholesterol level (107.27 ± 32.58–106.86 ± 38.94).

No significant association was found between these parameters for different time periods.

The microalbuminurea level, which is the initial indicator of the renal pathology, was positive in 14 subjects (63.6%) in the treatment group and in 16 subjects (72.7%) in the control group at baseline. Third month microalbuminurea levels were positive for 15 subjects (68.2%) in the treatment and 18 subjects (81.8%) in the control group. Although a slight increase was observed for the control group this did not reach a significant level within or between the two groups.

Discussion

The influence of diabetes on periodontal health has been discussed widely in the dental literature (Cohen et al. 1970, Cianciola et al. 1982, Rylander et al. 1987, Nelson et al. 1990, Shlossman et al. 1990, Emrich et al. 1991, Novaes Junior et al. 1991, de Pommereau et al. 1992, Thorstensson & Hugoson 1993, Bridges et al. 1996, Fıratlı et al. 1996, Fıratlı 1997, Tervonen & Karjalainen 1997, Taylor et al. 1998, Tervonen et al. 2000). A number of studies reported a high incidence and severity of periodontal disease in diabetic patients as compared with non-diabetic controls (Cianciola et al. 1982, Emrich et al. 1991, Hugoson et al. 1989, Nelson et al. 1990, Taylor et al. 1998).

There is substantial evidence to support considering diabetes as a risk factor for poor periodontal health, there is also evidence for periodontal infection adversely effecting glycaemic control in diabetes where this has been less extensively studied (see the review of Taylor 2001). These studies lead to a hypothesis that successful management of periodontal infection will lead to a reduction of the local symptoms of the disease and control the glucose metabolism.

More direct evidence regarding the effects of periodontal infection on glycaemic control in diabetes comes from treatment studies. There is evidence to support periodontal infection having adverse effect on glycaemic control (Williams & Mahan 1960, Tervonen et al. 1991, Tervonen & Oliver 1993, Miller et al. 1992, Aldridge et al. 1995, Smith et al. 1996, Westfelt et al. 1996, Grossi et al. 1997, Christgau et al. 1998, Iwamoto et al. 2001, Stewart et al. 2001, Al-Mubarak et al. 2002). However, not all investigations report on improvement in glycaemic control after periodontal treatment (Seppala & Ainamo 1994, Aldridge et al. 1995, Smith et al. 1996, Westfelt et al. 1996).

Results of this study suggest that following periodontal therapy there was a marked improvement in glycaemic control in individuals with type 2 DM when compared with a non-treatment control group. At baseline, both age, metabolic level-matched patients in the test and control groups showed similar levels of plaque accumulation, gingival and periodontal inflammation (PI, GI, BOP) as well as of periodontal breakdown (PPD, PAL).

The healing results of supragingival therapy were assessed after 1st and 3rd months following the periodontal treatment. There are contradictory opinions in the literature concerning the appropriate time for assessing the healing response to non-surgical subgingival therapy. Morrison et al. (1980) and Lowenguth & Greenstein (1995) suggested a period of 1 month, Badersten et al. (1981) found that in periodontal pockets of 4–7 mm depth most changes occur in the first 4–5 months while the deep pockets up to 12 mm a gradual improvement takes place over a period of 12 months. In our study the response to subgingival therapy was evaluated after 3 months as the majority of the examined sites had a PPD up to 3 mm.

The good healing response of diabetics to non-surgical therapy in the present study confirms the result of previous investigations (Westfelt et al. 1996, Tervonen et al. 1991).

Analysis of our data concerning the pocket depths showed similar distribution of PPD values. As PPD from all sites were recorded, the mean PPD for the treatment group was 2.29 ± 0.49 and 2.24 ± 0.70 mm for the control group. Eleven patients in the control and seven patients in the treatment group showed few sites with a probing depth of 5–7 mm. Regarding the moderate pocket depths, this limited us to make a comment that the HbA1c values may differ between those with more severe or less severe periodontitis. The improvement in the HbA1c values is possibly due to the reduction in the GI and BOP values. Considering that the patients did not have any deep pockets, the effect on the metabolic control is actually a consequence of a decrease in gingivitis.

As with other complications of diabetes, current evidence supports poorer glycaemic control contributing to poorer periodontal health. Primary research reports in the literature investigating the relationship between glycaemic control level and periodontal disease have been studied where subjects either had type 1 diabetes, a combination of both type 1 and type 2 diabetes or where the diabetes type was not specified (Sastrowijoto et al. 1989, de Pommereau et al. 1992, Seppala et al. 1993, Unal et al. 1993, Seppala & Ainamo 1994, Tervonen & Karjalainen 1997, Fıratlı 1997, Taylor et al. 1998, Tervonen et al. 2000).

The significant finding of this study is that the data supported the clinical improvement and significant reductions in levels of HbA1c in type 2 diabetes patients following mechanical subgingival treatment only. Our clinical trial provided the evidence that elimination of periodontal infection and improvement of periodontal inflammation significantly reduced the HbA1c in the short term, this improving diabetes metabolic control.

On the contrary, previous studies involving periodontal treatment alone reported improvement in periodontal status only (Seppala & Ainamo 1994, Aldridge et al. 1995, Smith et al. 1996, Westfelt et al. 1996) whereas studies including systemic antibiotics accompanying mechanical therapy reported an improvement in both periodontal status and glycaemic control (Miller et al. 1992, Grossi et al. 1997, Iwamoto et al. 2001).

Despite the variations in the design of their study, Stewart et al. (2001) reported a decrease in the level of HbA1c following the non-surgical periodontal treatment in patients with type 2 DM. As stated in their study nothing was known regarding the dental status of the control group and their medication was changed during the study period. Because of these factors we may think that this study protocol may have limitations in evaluating the effect of treatment on HbA1c values. Neither periodontal nor metabolic treatment was given to our control group for 3 months. By controlling this parameter, the effects of these variables are excluded.

The blood urea nitrate (BUN), creatinin and microalbuminurea levels were within the normal range during the

study period both in test and control groups. Christgau et al. (1998), who assessed their diabetic patients by evaluating creatinin, TG and TC levels, found only a minor influence on this medical data following their treatment.

TC, TG, LDL levels decreased in our study group whereas these values increased slightly in the control group. With the improvement in the metabolic control, the decrease in the TG levels is an expected outcome.

Because of the high vascularity of the inflamed periodontium, this inflamed tissue may serve as an endocrine-like source for tumour necrosis factor-α (TNF-α) and other inflammatory mediators. These mediators have been shown to have important effects on glucose and lipid metabolism (Iacopino & Cutler 2000, Loesche et al. 2000). Because of this fact there had been studies evaluating the relation of the state of gingiva with metabolic parameters. Ringelberg et al. (1977), Rylander (1987), Sandholm et al. (1989), Karjalainen & Knuuttila (1996) noted a weak correlation between these parameters whereas Hayden & Buckley (1989) and de Pommereau et al. (1992) found a negative correlation. We found a statistically significant differences between the treatment and control groups for both parameters. Also, we observed an improvement in both the state of gingiva and metabolic parameters in the treatment group. However, there was no statistically significant correlation between these two parameters, which may be attributed to the limited number of the study population.

Conclusion

As periodontitis is a complex multifactorial disease and similarly DM is a complex metabolic syndrome, diabetes has long been identified as a complicating factor in the periodontal therapy by the periodontists.

Our results showed that non-surgical periodontal treatment is associated with improved glycaemic control in type 2 DM patients. Considering our study population with moderate pocket depths, the effect on the metabolic control is a consequence of a decrease in gingivitis. This treatment could be undertaken along with the standard measures for the diabetic patient care. Prevention and control of periodontal disease must be considered an integral part of diabetes control.

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