Compare and Correlate the Presence of Bacterial Pathogen Concentrations and Glycaemic Status in Type 2 Diabetic and Non-Diabetic Patients with Chronic Periodontitis Following Phase 1 Therapy

Amit Lakhani[1], Ena Sharma[2], Surinder Sachdeva[3], Supreet Thind[4], Karun Chaudhary[5], Savita Kapila[6]

[1]Department of Orthopaedics, Maharishi Markandeshwar Medical College and Hospital Kumarhatti, Solan, Himachal Pradesh, India. [2]Department of Periodontics, Maharishi Markandeshwar College of Dental Sciences and Research, Mullana, Haryana, India. [3]Department of Periodontics, Maharishi Markandeshwar College of Dental Sciences and Research, Mullana, Haryana, India. [4]Department of Periodontics, Maharishi Markandeshwar College of Dental Sciences and Research, Mullana, Haryana, India. [5]Department of Periodontics, Maharishi Markandeshwar College of Dental Sciences and Research, Mullana, Haryana, India. [6]Department of Medicine, Maharishi Markandeshwar Institute of Medical Sciences and Research, Mullana, Haryana, India.

ABSTRACT

BACKGROUND

At present, a bidirectional pathway has been suggested between DM and periodontitis, since patients with DM are more predisposed to periodontal diseases, the established periodontitis may simultaneously impair adequate glycaemic control. We wanted to study the change in bacterial pathogen concentrations in plaque as confirmed polymerize chain reaction (PCR) in patients with and without type-2 diabetes before and after phase-1 therapy.

METHODS

Subgingival plaque samples were collected from the deepest part of the pocket by using a sterile curette in 2 different Eppendorf tubes containing tris-EDTA buffer. The plaque samples were then subjected to PCR for detecting bacteria (*P. gingivalis*, *T. forsythia*, *P. intermedia*).

RESULTS

In-vitro microbiological assessment (PCR analysis) showed significant reduction of bacteria within both diabetic and non-diabetic groups. In contrast, the intergroup comparison revealed no significant differences in the microbial counts in both the diabetic and non-diabetic groups thus suggesting that phase-1 therapy may not alter the pathogenic microflora in chronic periodontitis patients with or without diabetes.

CONCLUSIONS

In-vitro microbiological assessment (PCR analysis) showed significant reduction of bacteria within both diabetic and non-diabetic groups. In contrast, the intergroup comparison revealed no significant differences in the microbial counts in both the diabetic and non-diabetic groups thus suggesting that phase-1 therapy may not alter the pathogenic microflora in chronic periodontitis patients with or without diabetes.

KEY WORDS

Chronic Periodontitis, PCR, Oral Pathogens, Diabetes

BACKGROUND

Periodontal disease is a microbial infection involving a variety of microbes that trigger inflammation, loss of connective tissue attachment and alveolar bone around the teeth. The primary etiologic factor of periodontitis is bacterial plaque. The bacteria involved are mainly of gram-negative species, express pathogenic factors that elicit host defence responses resulting in inflammation and tissue destruction. In fact, the propensity of periodontitis to proceed with periods of exacerbation and remission could suggest that the presence of other organisms contributes in the disease progression.[1] The development of periodontitis may depend upon cooperative interactions among specific pathogenic bacteria and tissue destructive inflammatory mediators. Various evidences has shown relationship between type 2 diabetes and periodontal disease.[2] However, the effect of periodontal intervention on these elements is yet to be ascertained.

Diabetes is a group of metabolic diseases characterized by hyperglycaemia resulting from defects in insulin secretion, insulin action, or both. The chronic hyperglycaemia of diabetes is associated with long-term damage, dysfunction, and failure of different organs, especially the eyes, kidneys, nerves, heart, and blood vessels. Several pathogenic processes are involved in the development of diabetes. These range from autoimmune destruction of the β-cells of the pancreas with consequent insulin deficiency to abnormalities that result in resistance to insulin action. Deficient insulin action results from inadequate insulin secretion and/or diminished tissue responses to insulin at one or more points in the complex pathways of hormone action. Impairment of insulin secretion and defects in insulin action frequently coexist in the same patient, and it is often unclear which abnormality, if either alone, is the primary cause of the hyperglycaemia.

At present, a bidirectional pathway has been suggested between DM and periodontitis, since patients with DM are more predisposed to periodontal diseases, the established periodontitis may simultaneously impair adequate glycaemic control.[3] Moreover, poorly controlled diabetic subjects, presenting elevated levels of glycosylated haemoglobin (HbA1c), show higher attachment and alveolar bone loss and local inflammatory cytokines than well controlled patients[3,4] Exacerbated local inflammation in diabetic subjects with inadequate glycaemic control could modify the subgingival environment, and consequently, the subgingival microbial profile. Evidence has shown variations in the micro flora in non-diabetic and diabetic individuals; and also, within the diabetic individuals as well, based on their glycaemic control.[5,6]

We wanted to assess, compare and correlate the change in bacterial pathogen concentrations in plaque as confirmed polymerize chain reaction (PCR) in patients with and without type-2 diabetes before and after phase-1 therapy in patients with chronic periodontitis.

METHODS

This study was conducted among 120 patients of chronic periodontitis visiting the Department of Periodontics, Maharishi Markandeshwar College of Dental Sciences and Research, Mullana, Ambala and Department of Medicine, Maharishi Markandeshwar Medical College, Mullana Ambala. Ethical approval was obtained.

Method of Collection of Data
In this study, which was of 4 months duration, following initial screening, 120 patients with chronic periodontitis (Mild to moderate) were divided into 2 main groups based on their glycaemic status. Glycated haemoglobin levels were assessed using standardized assay- High Pressure Liquid Chromatography (HPLC).

Group-1-Control (n=30)
    30 non-diabetic patients with chronic periodontitis.

Group-2-Test: (n=9 0)
These 90 diabetic patients were further subdivided into 3 subgroups-
- 2 (A)- 30 diabetic patients with chronic periodontitis. (Good control (HbA1c<5%))
- 2 (B)- 30 diabetic patients with chronic periodontitis. (Moderate control (HbA1c<7%))
- 2 (C)- 30 diabetic patients with chronic periodontitis. (Poor control (HbA1c>8%))

Inclusion Criteria
- Patients with chronic periodontitis (mild to moderate with up to 4 mm (CAL) clinical attachment loss and with the presence of true periodontal pocket (up to 6 mm) in ≥30% of sites in the mouth.
- Systemically healthy patients (for Group 1).
- Diabetic patients having chronic periodontitis with varied levels of glycaemic control classified as good, moderate and poor (Group 2).

Exclusion Criteria
- Patients with any systemic diseases (Group 1)
- Presence of any systemic or debilitating diseases apart from diabetes in group.

The following parameters were assessed in all the patients at baseline (0), 1 months and 4 months
1) Clinical Parameters
    1. Gingival index (Loe and Silness).(1963)(GI)
    2. Plaque index (Silness and Loe).(1967)(PI)
    3. Probing Pocket Depth (PPD) and relative Attachment Level (RAL)

2) Microbiological Parameters
    1. Subgingival plaque samples were collected from the deepest part of the pocket by using a sterile curette in 2 different Eppendorf tubes containing tris-EDTA buffer.
    2. The plaque samples were then sent for detecting bacteria (*P. gingivalis*, *T. forsythia*, *P. intermedia*,)

Treatment Procedure

Clinical Aspect- All the 120 patients were subjected to phase 1 therapy. After assessing the clinical parameters ultrasonic scaling was done followed by thorough root planing at baseline. Patients were recalled after 1 month and 4 months during which time, plaque samples were collected, and clinical parameters were recorded.

Microbiological Aspect- The plaque samples were subjected to PCR. The main components of the PCR Master Mix were HemoklenTaq buffer 5x, dNTP (2.5 mM), HaemoklenTaq enzyme and PCR water. Additives like DMSO, glycerol and formamide were also added for getting better results.

PCR Protocol

For PCR, bacterial DNA from the plaque samples was amplified in 50 µl of a reaction mixture containing 3 µl of the DNA sample, 1X PCR buffer (50 mMKCl, 1.5 mMMgClz, 10 mM Tris-HCl, pH 9.0), 2.5 mM deoxyribonucleotide mixture 1 µl of each primer, and 0.5-1 µl of haemoklenTaq DNA polymerase (NAB) in a thermal cycler for 30 cycles. After amplification, 20 µl of the PCR product was analyzed by agarose gel electrophoresis by incorporating propidium iodide on 1% gel. The newly synthesized DNA fragments were then visualized under ultraviolet light. The size of the PCR products was estimated from the electrophoretic migration of products relative to a 1 kb ladder marker. (Thermoscientific).

Statistical Analysis

All the clinical, microbiological and biochemical parameters recorded were statistically analysed using SPSS software version 13, and Paired t test.

R E S U L T S

P. gingivalis (PG)- Intra Group (Table 1A)

Group-1 (Control)- The mean difference in *P. gingivalis* counts between baseline and 1 month was 4926000000.0$\pm$ 5081385637.78 and baseline and 4 months was 5010000000.0$\pm$ 5069972454.52 and 1 month and 4 months was 84000000.0$\pm$ 179031879.27. This difference was not found to be statistically significant (p <0.05) at all-time intervals i.e. from baseline to 1 month, baseline to 4 months & from 1 month to 4 months.

Group-2A (Test)- The mean difference in *P. gingivalis* counts between baseline and 1 month was 4286666666.67$\pm$ 4992665885.44 and baseline and 4 months was 4312000000.0$\pm$ 5016730904.23 and 1 month and 4 months was 25333333.33$\pm$ 43607444.27. This difference was found to be statistically significant (p <0.05) at all-time intervals i.e. from baseline to 1 month, baseline to 4 months & from 1 month to 4 months.

Group-2B (Test)- The mean difference in *P. gingivalis* counts between baseline and 1 month was 4677000000.00$\pm$4902599697.42 and baseline and 4 months was 4763666666.67$\pm$ 4978032074.36 and 1 month and 4 months was 86666666.67 $\pm$ 238346796.50. This difference

was found to be statistically significant (p <0.05) at all-time intervals i.e. from baseline to 1 month, baseline to 4 months & from 1 month to 4 months.

Groups	Time intervals	Mean	Std. Deviation	Sig.	Inference
Group 1	Baseline - 1 month	4926000000.0000000	5081385637.7960530	.000	S
	Baseline – 4 months	5010000000.0000000	5069972454.5262740	.000	S
	1 month- 4 months	84000000.000000	179031879.264849	.000	S
Group-2A	Baseline – 1 month	4286666666.6666665	4992665885.4441830	.000	S
	Baseline – 4 months	4312000000.0000000	5016730904.2360690	.000	S
	1 month – 4 months	25333333.333333	43607444.265885	.000	S
Group-2B	Baseline – 1 month	4677000000.0000000	4902599697.4159990	.000	S
	Baseline – 4 months	4763666666.6666670	4978032074.3576310	.000	S
	1 month – 4 months	86666666.666667	238346796.501021	.05	S
Group-2C	Baseline – 1 month	6844333333.3333330	4733278140.3613910	.000	S
	Baseline – 4 months	6731000000.0000000	4727797001.0099430	.000	S
	1 month – 4 months	-113333333.333333	503363400.182888	.227	N.S
Table 1A. Inter Group Comparison of P. gingivalis					

Group-2C (Test): The mean difference in *P. gingivalis* counts between baseline and 1 month was 6844333333.33$\pm$ 4733278140.37 and baseline and 4 months was 6731000000.00$\pm$ 4727797001.00 and 1 month and 4 months was -113333333.33$\pm$503363400.19. This difference was found to be statistically significant (p <0.05) from baseline to 1 month and baseline to 4 months but not from 1 month to 4 months, (p>0.05).

P. gingivalis (PG)- Inter Group (Table 1B)

Intergroup comparisons of Pg counts at the end of 1 month showed no significant differences between group 1 and 2A, 2B and 2C and between groups 2A and 2B and 2A and 2C and also between 2B and 2C, (p >0.05). In contrast, at the end of 4 months, significant reduction was observed in group 1 when compared with group 2C, group 2A in comparison with group 2C and group 2B when compared with group 2C (p <0.05) However, no significant differences in Pg counts were observed between group 1 and 2A, group 1 and 2B and also between group 2A and 2B (p >0.05) at the end of 4 months.

Dependent Variable	Groups	Groups	Mean Difference	Std. Error	Sig.	Inference
GI 1 month	1	2A	40666666.666667	59457412.557611	1.000	NS[1 v/s 2A]
		2B	-5666666.666667	59457412.557611	1.000	NS[1 v/s 2B]
		2C	-68333333.333333	59457412.557611	1.000	NS[1 v/s 2C]
	2A	2B	-46333333.333333	59457412.557611	1.000	NS[2Av/s2B]
		2C	109000000.000000	59457412.557611	.416	NS[2A v/s 2C]
	2B	2C	-62666666.666667	59457412.557611	1.000	NS[2B v/s 2C]
GI 4ᵗʰ month	1	2A	-18000000.0000	58166329.0838	1.000	NS[1 v/s 2A]
		2B	-3000000.0000	58166329.0838	1.000	NS[1 v/s 2B]
		2C	-265666666.6667*	58166329.0838	.000	S[1 v/s 2C]
	2A	2B	15000000.0000	58166329.0838	1.000	NS[2Av/s2B]
		2C	-247666666.6667*	58166329.0838	.000	S[2A v/s 2C]
	2B	2C	-262666666.6667*	58166329.0838	.000	S[2B v/s 2C]
Table 1b. Inter Group Comparison of P. gingivalis						

T. forsythia (TF)- Intra Group (Table 2A)
Group-1 (Control): The mean difference in *T. forsythia* scores between baseline and 1 month was 70000000.00$\pm$255653321.30, at baseline and 4 months was 76333333.33$\pm$252675112.83 and 1 month and 4 months was 6333333.33$\pm$24280449.54. This difference was not found to be statistically significant (p>0.05) at all time intervals i.e. from baseline to 1 month, baseline to 4 months and 1 month and 4 months.

Group-2A (Test): The mean difference in *T. forsythia* scores between baseline and 1 month was 1995333333.33$\pm$4042906715.80, at baseline and 4 months was 1696551724.13 $\pm$3768050154.98 and 1 month and 4 months was -23103448.28$\pm$170589386.89. This difference was found to be statistically significant (p <0.05) from baseline to 1 month and baseline to 4 months but not from 1 month to 4 months, (p>0.05).

Group-2B (Test): The mean difference in *T. forsythia* scores between baseline and 1 month was 1626000000.00$\pm$3617409627.60, at baseline and 4 months was 1663333333.33$\pm$3699998446.72 and 1 month and 4 months was 37333333.33$\pm$182736541.71. This difference was found to be statistically significant (p <0.05) from baseline to 1 month and baseline to 4 months but not from 1 month to 4 months, (p>0.05).

Group-2C (Test): The mean difference in *T. forsythia* scores between baseline and 1 month was 1099633333.33$\pm$3020280170.64, at baseline and 4 months was 1044000000.00 $\pm$3052032222.90 and 1 month and 4 months was -55633333.33$\pm$243356483.70. This difference was found to be statistically significant (p <0.05) at baseline to 1 month but not from baseline to 4 months & from 1 month to 4 months, (p>0.05).

Groups	Time intervals	Mean	Std. Deviation	Sig.	Inference
Group 1	Baseline - 1 month	70000000.0000000	255653321.2959597	.144	NS
	Baseline - 4 months	76333333.3333333	252675112.8300493	.109	NS
	1 month – 4 months	6333333.333333	24280449.540424	.164	NS
Group-2A	Baseline - 1 month	1995333333.3333335	4042906715.7978890	.011	S
	Baseline – 4 months	1696551724.1379310	3768050154.9798074	.022	S
	1 month – 4 months	-23103448.275862	170589386.880474	.472	NS
Group-2B	Baseline – 1 month	1626000000.0000000	3617409627.5916977	.020	S
	Baseline – 4 months	1663333333.3333333	3699998446.7222586	.020	S
	1 month – 4 months	37333333.333333	182736541.715555	.272	NS
Group-2C	Baseline – 1 month	1099633333.3333333	3020280170.6456640	.056	S
	Baseline – 4 months	1044000000.0000000	3052032222.9057760	.071	NS
	1 month – 4 months	-55633333.333333	243356483.704297	.221	N.S

Table 2a. Intra Group Comparison of T. forsythia

T. forsythia (TF)- Inter Group (Table 2B)
Inter group comparisons of Tf counts at the end of 1 month showed no significant differences between group 1 and 2A, 2b and 2C respectively and also between group 2A and 2B and 2A and 2C and also between groups 2B and 2C. (p>0.05). Similarly, no significant differences were found between group 1 and 2A and 2B respectively, and group 2A and 2B and 2A and 2C and also group 2B and 2C at the end of 4 months, (p>0.05).In contrast, significant difference was found in the T.F counts at the end of 4 months between group 1 and group 2C (p<0.05)

Dependent Variable	Groups	Groups	Mean Difference	Std. Error	Sig.	Inference
GI 1 month	1	2A	6000000.000000	33543266.792703	1.000	NS[1 v/s 2A]
		2B	-56666666.666667	33543266.792703	.563	NS[1 v/s 2B]
		2C	2966666.666667	33543266.792703	1.000	NS[1 v/s 2C]
	2A	2B	-62666666.666667	33543266.792703	.386	NS[2Av/s2B]
		2C	-3033333.333333	33543266.792703	1.000	NS[2A v/s 2C]
	2B	2C	59633333.333333	33543266.792703	.468	NS[2B v/s 2C]
GI 4th month	1	2A	-23482758.620690	47492484.013034	1.000	NS[1 v/s 2A]
		2B	-25666666.666667	47088285.309193	1.000	NS[1 v/s 2B]
		2C	-59000000.000000	47088285.309193	1.000	S[1 v/s 2C]
	2A	2B	-2183908.045977	47492484.013034	1.000	NS[2Av/s2B]
		2C	-35517241.379310	47492484.013034	1.000	NS[2A v/s 2C]
	2B	2C	-33333333.333333	47088285.309193	1.000	NS[2B v/s 2C]

Table 2b. Inter Group Comparison of T. forsythia

P. intermedia (PI)- Intra Group (Table 3A, Graph 3A)
Group-1 (control): The mean difference in *P. intermedia* counts between baseline and 1 month was 1755333333.33$\pm$3731564092.27, at baseline and 4 months was 1775000000.00$\pm$3750355155.60 and 1 month and 4 months was 19666666.67$\pm$37644739.82. This difference was found to be statistically significant (p<0.05) at all time intervals i.e. from baseline to 1 month, baseline to 4 months & from 1 month to 4 months.

Group-2A (Test): The mean difference in *P. intermedia* counts between baseline and 1 month was 1821333333.33$\pm$3716936756.33, at baseline and 4 months was 1799666666.67$\pm$3652283634.53 and 1 month and 4 months was -21666666.67$\pm$310428883.90. This difference was found to be statistically significant (p<0.05) at baseline to 1 month and baseline to 4 months but not from 1 month to 4 months, (p>0.05).

Group-2B (Test): The mean difference in *P. intermedia* counts between baseline and 1 month was 2124666666.67$\pm$3927763014.60, at baseline and 4 months was 2169000000.00$\pm$3988301297.71 and 1 month and 4 months was 44333333.33$\pm$184216576.69. This difference was found to be statistically significant (p<0.05) at baseline to 1 month, baseline to 4 months but not from 1 month to 4 months, (p>0.05).

Group-2C (Test): The mean difference in *P. intermedia* counts between baseline and 1 month was 287266670.30$\pm$421938784.74, at baseline and 4 months was 292633336.97$\pm$501342091.10 and 1 month and 4 months was 5366666.67$\pm$370736404.49. This difference was found to be statistically significant (p <0.05) at baseline

to 1 month, baseline to 4 months but not from 1 month to 4 months, (p>0.05).

P. intermedia (PI)- Inter Group (Table 3B, Graph 3B)

Inter group comparisons of Pi counts revealed no significant differences between all the four groups at the end of 1 month and 4 months respectively,(p>0.05).

Groups	Time intervals	Mean	Std. Deviation	Sig.	Inference
Group1	Baseline – 1 month	1755333333.3333333	3731564092.2691710	.015	S
	Baseline – 4 months	1775000000.0000000	3750355155.5957274	.015	S
	1 month- 4 months	19666666.666667	37644739.828847	.008	S
Group-2A	Baseline – 1 month	1821333333.3333333	3716936756.3323855	.012	S
	Baseline – 4 months	1799666666.6666667	3652283634.5396880	.011	S
	1 month- 4 months	-21666666.666667	310428883.891340	.705	NS
Group-2B	Baseline – 1 month	2124666666.6666665	3927763014.5988407	.006	S
	Baseline – 4 months	2169000000.0000000	3988301297.7180290	.006	S
	1 month- 4 months	44333333.333333	184216576.687433	.198	NS
Group-2C	Baseline – 1 month	287266670.3000000	421938784.7430390	.001	S
	Baseline – 4 months	292633336.9666666	501342091.9915900	.003	S
	1 month- 4 months	5366666.666667	370736404.483287	.937	N.S.
Table 3A. Intra Group Comparison					

Dependent Variable	Groups	Groups	Mean Difference	Std. Error	Sig.	Inference
GI 1 month	1	2A	-24000000.000000	46950335.706878	1.000	NS[1 v/s 2A]
		2B	-28333333.333333	46950335.706878	1.000	NS[1 v/s 2B]
		2C	-51400000.000000	46950335.706878	1.000	NS[1 v/s 2C]
	2A	2B	-4333333.333333	46950335.706878	1.000	NS[2Av/s2B]
		2C	-27400000.000000	46950335.706878	1.000	NS[2A v/s 2C]
	2B	2C	-23066666.666667	46950335.706878	1.000	NS[2B v/s 2C]
GI 4th month	1	2A	-65333333.333333	46424079.867789	.972	NS[1 v/s 2A]
		2B	-3666666.666667	46424079.867789	1.000	NS[1 v/s 2B]
		2C	-65700000.000000	46424079.867789	.958	NS[1 v/s 2C]
	2A	2B	61666666.666667	46424079.867789	1.000	NS[2Av/s2B]
		2C	-366666.666667	46424079.867789	1.000	NS[2A v/s 2C]
	2B	2C	-62033333.333333	46424079.867789	1.000	NS[2B v/s 2C]
Table 3B. Inter Group Comparison						

DISCUSSION

Periodontitis usually results from the complex interactions of the microbial communities with the host elements in the gingival sulcus that leads to inflammation, loss of connective tissue attachment and alveolar bone around the teeth.[6]

Diabetes mellitus (DM) is well recognized as a risk factor for periodontal disease, while periodontitis is thought to influence the systemic inflammatory condition, lipid and glucose metabolism and insulin resistance.[7] Diabetes mellitus, especially when uncontrolled, appears to be an important risk factor for periodontal destruction, since an alteration in host-response and microbiological aspects occurs in diabetic subjects.[8] Periodontal disease leads to an increase in insulin resistance, which could impair glycaemic control. Thus, the control of periodontal disease is necessary for better systemic health in these individuals. Periodontal treatment relies on biofilm disruption and plaque control to prevent recolonization and recurrence of the disease.[8]

The relationship between plaque accumulation and HbA₁c levels remains controversial.[9-11] Some reports have observed an improved compliance with oral hygiene amongst diabetic subjects with better glycaemic control, while others have not found this association.[12-14] Some studies have shown the impact of type 1 and type 2 DM on tooth loss,[15] but the association between metabolic control and the number of remaining teeth has not been considered. Previous studies have shown that the microbiota associated with diabetes does not appear different from the microbiota of non-diabetic patients.[15-19] Although the microbiology of periodontal disease in DM is fairly well understood. The sample size of 120 patients evaluated in this study was arrived at following consultation with the statistician which was in accordance with the vast majority of clinical periodontal treatment studies in humans.[20] In order to understand this phenomenon with better sensitivity and specificity, it was decided to carry out the study by dividing the 120 patients into 4 groups based on the level of glycaemic control.

The clinical parameters (GI, PI, PPD, RAL) were assessed to evaluate the disease severity at regular intervals and also to understand whether phase-1 therapy has an effect on patients with chronic periodontitis with and without diabetes.

The gold standard for recording changes in periodontal status is the longitudinal measurements with relative attachment level (RAL) from CEJ to the base of the pocket. Due to the relative inconsistencies in determining CEJ accurately at the selected sites, it was decided to use a customized acrylic stent and use the base of the stent as the fixed reference point and evaluate relative attachment level (RAL).

In addition, the microbiological analysis with the help of multiplex PCR technique was done to evaluate the effect of phase-1 therapy on bacterial concentrations in patients with chronic periodontitis with and without diabetes. Single-stage PCR was used to detect *P. gingivalis*, *P. intermedia*, and *T. forsythia*. It has been suggested that pooling of plaque samples increases the probability of detecting existing pathogens.[21]

Intergroup comparison revealed, a significant improvement in glycated Hb levels in the non-diabetic group as compared to the diabetic groups at 1 month following phase-1 therapy. Additionally significant improvement was also observed in the well-controlled and moderately controlled diabetic group when compared with the poorly controlled group. This is in accordance with Taylor et al 2008,[22] Shlossman 1990[23] who concluded that phase-1 therapy improves the glycaemic status of diabetic patients with chronic periodontitis.

With regard to the microbiologic parameters, intra group comparison revealed a significant improvement in Pg counts, Tf count, Pi count, count with in both the test (Diabetic) and control (non-diabetic) group from baseline to 1 month and 4 months except in poorly controlled diabetic group at 1 month and 4 months. This is in accordance with the findings of Sandholm et al 1989[24], Contreras at al 1999[25], Saygun et al

2008[26] who concluded that phase-1 therapy results in reduction of bacterial counts in patients with chronic periodontitis and diabetes and also with Hintao et al 2007[27,] IbrahimuMdala 2013[28] who concluded that phase-1 therapy results in reduction of bacterial counts in patients with chronic periodontitis

Inter group comparison revealed significant reduction in Pg counts, Tf count, Pi count, in the non-diabetic group compared to the poorly controlled diabetic group and well controlled diabetic group compared to poorly-controlled diabetic group at the end of 4 months. But interestingly no such differences were observed at 1 month. This is accordance with Makiura et al 2008[29] who concluded that Pg counts were reduced in the patients with diabetes following phase-1 therapy.

CONCLUSIONS

In-vitro microbiological assessment (PCR analysis) showed significant reduction of bacteria within both diabetic and non-diabetic groups. In contrast, the intergroup comparison revealed no significant difference in the microbial counts in both the diabetic and non-diabetic groups thus suggesting that phase-1 therapy may not alter the pathogenic microflora in chronic periodontitis patients with or without diabetes. Correlation assessments of clinical parameters with the microbiological and biochemical parameters showed significant correlation at various time intervals again suggesting that phase 1 therapy results in improved clinical and microbiologic status and glycaemic status in chronic periodontitis patients with and without diabetes.

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