The Relationship between Periodontal Disease and Glycemic Status of Type II Diabetic Patients in Indian Population

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Abstracts: Introduction: Diabetes mellitus is a clinically and genetically heterogeneous group of disorders affecting the metabolism of carbohydrates, lipids, and proteins. Diabetes mellitus (DM) and chronic periodontitis are common chronic diseases in adults in the world population. The association between periodontal disease and diabetes has long been hypothesized. Considering confirmation of treatment of periodontal disease positively influencing the glycemic control of Diabetes mellitus patients of great public importance because periodontal disease is both preventable and curable, the current study was planned. Improving periodontal health in a diabetic patient might improve their metabolic control and thereby decrease the associated morbidity and mortality. **Aims and Objectives:** To reveal whether the suggested association between periodontal disease and diabetes could be found in a Type 2 Diabetic Indian population, the present study was undertaken. **Materials and Methods:** This clinical study was carried out at the Department of Periodontology, Saraswati Dhanwantari Dental College and Hospital and Post-Graduate Research Institute, Parbhani. For assessing the effect of the periodontal treatments on metabolic control, no change in the medication or diet was made for the selected three groups during the study period. None of the groups received any additional guidance in managing their diabetic status. **Statistical Analysis:** The Student t-test was used to test the differences of age, sex and diabetic control methods between the treatment and control groups. The changes of PI, PPD, CAL and BOP values from baseline to 3rd month and 6th month within both groups were compared using unpaired t test. The significance of the metabolic parameters within the groups was assessed by unpaired t test. ANOVA were used to test changes from baseline and differences between the groups for any of the continuous variables assessed. **Results:** Results of this study showed that non-surgical periodontal treatment with and without antibiotic therapy (doxycycline) is associated with improved glycaemic control in type 2 DM patients and reduction of clinical parameters of periodontal infection, confirming the existing interrelationship between Diabetes mellitus and periodontal disease. **Conclusion:** 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. An improved communication between dentists / periodontists and physicians / endocrinologists is therefore warranted to work together to improve the management of Diabetic patients. [Joshi V NJIRM 2015; 6(1):7-16]

Key Words: Diabetes Mellitus, periodontitis, complications, periodontal therapy, antibiotic therapy.

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Introduction: Diabetes mellitus is a clinically andgenetically heterogeneous group of disorders affecting the metabolism of carbohydrates, lipids, and proteins. Diabetes mellitus (DM) and chronic periodontitis are common chronic diseases in adults in the world population.[1]

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.[1] 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.[2]

Diabetes and periodontal disease have a special two-way relationship.[3,4] Acute infections and inflammatory conditions lead to increases in glucose and insulin utilization and therefore complicate metabolic control in diabetes.[5] Grossi[6] suggested that chronic periodontal infection increases the severity of diabetes and complicates its control. However, Gustke[7] and Taylor[8] concluded that studies are currently insufficient to establish periodontal therapy

as having a positive influence on glycemic control in Type I or Type II diabetes.

Also a majority of studies published on this aspect have been carried out on men and women who may differ greatly from the average Indian adult with respect to race, socio-ecomomic status and access to health services in general. To reveal whether the suggested association between periodontal disease and diabetes could be found in a Type 2 Diabetic Indian population, the present study was undertaken.

A confirmation of treatment of periodontal disease positively influencing the glycemic control of Diabetes mellitus patients would be of great public importance because periodontal disease is both preventable and curable. Improving periodontal health in a diabetic patient may improve their metabolic control and thereby decreasing the associated morbidity and mortality.

Material and Method: This clinical study wascarried out at the Department of Periodontology, Saraswati Dhanwantari Dental College and Hospital, Parbhani. The study was reviewed and approved by the institutional ethical Committee.

The inclusion criteria for the study were patients with type 2 DM with glycated haemoglobin (HbA1c) values >6% and a least one tooth presenting with a probing pocket depth of > or = 4mm and CAL of > or = 4mm. Patients with type 2 DM with HbA1c value<6%, diabetic complications, history of systemic antibiotics within last 3 months and undergoing periodontal treatment 6 months prior to the study were excluded from this study. 75 patient subjects fulfilling these criteria signed an informed consent form. These subjects were 34 (45.3%) women and 41 (54.7%) men, with the mean age of 58.16 ± 8.585 years. Subjects were randomly assigned into three groups as:

GROUP I: Control group (No treatment)
GROUP II: Periodontal therapy only (Full mouthScaling and root planning)

GROUP III: Periodontal therapy (Full mouth Scalingand root planning) and antibiotic administration (Doxycyline 100mg prescribed for 14 days).

For assessing the effect of the periodontal treatments on metabolic control, no change in the medication or diet was made for all three groups during the study period. None of the groups received any additional guidance in managing their diabetic status. After the completion of the study the patients in the control group received full non-surgical therapy and supportive periodontal treatment if required.

1. Periodontal Examination: In the selected patients, the periodontal parameters were recorded at baseline (day 0) and at 1^{st}, 3^{rd}, and 6^{th} month following the periodontal treatment in both groups. The first month following treatment, periodontal parameters were recorded to identify the surgical treatment needs of the study groups. Periodontal measurements were recorded by a single examiner with the help of the Florida Probe.

The parameters recorded were:
Plaque Index (PI): The Plaque Index was recorded around all the teeth, on the facial/ buccal andlingual/palatal surfaces using the criteria proposed by Silness and Loe[9].

Clinical Attachment Level (CAL): For determiningthe level of attachment when the gingival margin was located on anatomic crowns, the level of attachment was determined by subtracting from the depth of the pocket the distance from the gingival margin to the cementoenamel junction (CEJ). If both were the same, the loss of attachment was zero. When the gingival margin coincided with the CEJ, the loss of attachment equaled the PD. When the gingival margin was located apical to CEJ, the loss of attachment was greater than the pocket depth, and therefore the distance between CEJ and the gingival margin was added to the PD. Measurements of CAL were made at six sites per tooth measured with the help of a Florida Probe.
Bleeding on Probing: Bleeding on probing was measured using the gingival bleeding index. Gingival crevice was gently probed with a periodontal probe; appearance of bleeding within 10 seconds indicated a positive score and was expressed as the percentage of the sites exhibiting this response.[10]

2. Metabolic Assessment: Collection Of Peripheral Venous Blood Sample: 10 ml of peripheral venousblood was collected by venipuncture. The blood samples were collected at baseline (0 month), 3 months and six months intervals and evaluated for fasting plasma glucose, post prandial glucose and

HbA1c. Metabolic measurements were performed at baseline (day 0), 3rd and 6th month in all three groups.

3. Periodontal Therapy: In Group I, the control group received no periodontal treatment duringthe study period. After completion of the study, these patients were given a full non-surgical and supportive periodontal treatment if needed. In Group II, the periodontal therapy consisted of two phases. A pretreatment phase in which patients received oral hygiene instructions, motivation, supragingival scaling, placement of emergency restorations, and removal of overhanging margins. A subsequent full mouth non-surgical periodontal therapy comprising subgingival scaling and root planning was performed under local anaesthesia. The periodontal parameters were re-evaluated at 1 month and 3 month. In Group III, the patients received oral hygiene instructions and full-mouth scaling and root planing performed under local anaesthesia. Adjunctive antibiotic therapy was also instituted on the first day of therapy. Doxycycline 100 mg twice was given orally STAT and then 100mg OD for 13 days. The periodontal parameters were re-evaluated at 1 month and if necessary oral prophylaxis re-instituted.

Statistical Analysis: The Student t-test was used totest the differences of age, sex, diabetic control methods, between the treatment and control groups. The changes of PI, PPD, CAL and BOP values from baseline to 3rd month and 6th month within both groups were compared using unpaired t test. The significance of the metabolic parameters within the groups was assessed by unpaired t test. ANOVA were used to test changes from baseline and differences between the groups for any of the continuous variables assessed. The SPSS software was used for the analysis.

Result: The study was carried out in 75 subjects (41 males and 34 females) divided into three groups. The age characteristic of the study population is shown in Table 1. At Baseline, control and both treatment groups had similar mean values for age. The mean age was 56.52 ± 6.571 years for group 1, 60.76 ± 7.688 years for group 2, and 57.20 ± 10.685 years for group 3. The age difference between the three groups was not statistically significant with a p value of 0.173. The average age of the study population was 58.16 ± 8.585 years.

At baseline, control and both treatment groups had similar mean values for duration of diabetes in

Table 1: Age and Duration of Diabetes

GROUP		AGE (Years)	DIABETES DURATION(Years)
1 (N: 25)	Mean	56.52	6.10
	SD	6.571	0.957
2 (N: 25)	Mean	60.76	6.84
	SD	7.688	1.526
3 (N: 25)	Mean	57.20	6.58
	SD	10.685	1.663
Total	Mean	58.16	6.51
	SD	8.585	1.430
Significance	p value	0.173	0.179

Years as shown in Table 1. The mean duration of diabetes was 6.10± 0.957 years for group 1, 6.84± 1.526 years for group 2, and 6.58± 1.663 years for group 3. The duration of diabetes difference between the three groups was not statistically significant with a p value of 0.179. The average duration of diabetes of the study population was 6.51 ± 1.430 years.

At baseline, control and both treatment groups had similar mean values for sex as shown in Table 2. Group 1 consisted of 15 men (60%) and 10 women (40%) with a male female ratio of 60/40. Group 2 consisted of 14 women (56%) and 11(44%) men, with a Male female ratio of 44/56 and Group 3 consisted of 15 (60%) males and 10 (40%) females with a Male female ratio of 60/40. The study population in total consisted of 34 (45.3%) females and 41 (54.7%) males.

Table 2: Sex Distribution

			GROUP			Total
			1	2	3	
SEX	F	Count	10	14	10	34
		%	40.0%	56.0%	40.0%	45.3%
	M	Count	15	11	15	41
		%	60.0%	44.0%	60.0%	54.7%
Total		Count	25	25	25	75
		%	100.0%	100.0%	100.0%	100.0%

At baseline, control and both treatment groups had similar values for method of hyperglycaemia control as shown in Table 3. Out of 25 patients in group 1, 19(76.0%) were being administered only oral hypoglycaemics and 6(24.0%) were taking a combination of oral hypoglycaemics and insulin. Out of 25 patients in Group 2, 17(68.0%) were being

administered only oral hypoglycaemics and 8(32.0%) were taking a combination of oral hypoglycaemics and insulin. Out of 25 patients in group 3, 18(72%) were being administered only oral hypoglycaemics and 7(28%) were taking a combination of oral hypoglycaemics and insulin. Thus in the study population a majority (72.0%) were taking only combinations of various oral hypoglycaemics as their method of controlling hyperglycaemia, while 28.0% were taking a combination of oral hypoglycaemics and insulin.

Table 3: Method of Control ofHyperglycaemia

			GROUP			Total
			1	2	3	
Method	OH	Count	19	17	18	54
		%	76.0%	68.0%	72.0%	72.0%
	OH +I	Count	6	8	7	21
		%	24.0%	32.0%	28.0%	28.0%
Total		Count	25	25	25	75
		%	100.0 %	100.0 %	100.0 %	100.0 %

OH: oral hypoglycaemic,
OH+I: oral hypoglycaemic + Insulin combination

Periodontal Parameters: The mean Plaque Index (PI) at baseline, 1, 3 and 6 months for all three groups is given in Table 4. The mean plaque index (PI) at baseline for Group 1 was 1.5836 ± 0.25934, at 3 months it was 1.5472 ± 0.16262 and at 6 months it was 1.5908 ± 0.14174. The PI in group 1 did not change significantly from baseline levels at 3 and 6 months. The PI at baseline for Group 2 was 1.5728 ± 0.22401 which dropped significantly at 3 months to 0.3336 ± 0.16262 and at 6 months it was 0.4416 ± 0.11097. The drop in PI from baseline was statistically significant at 1 month, 3 and at 6 months. The mean PI at baseline for Group 3 was 1.5964 ± 0.16598 which dropped significantly at 3 months to 0.2952 ± 0.07107 and at 6 months it was 0.4476 ± 0.11395. The drop in PI from baseline was statistically significant at 3 and at 6 months.

Table 4: Plaque Index

GROUP		PI Baseline	PI 3 Month	PI 6 Month
1 (N: 25)	Mean	1.5836	1.5472	1.5908
	SD	0.25934	0.16262	0.14174
2 (N: 25)	Mean	1.5728	0.3336	0.4416
	SD	0.22401	0.06626	0.11097
3(N: 25)	Mean	1.5964	0.2952	0.4476
	SD	0.16598	0.07107	0.11395
Total	Mean	1.5843	0.7253	0.8267
	SD	0.21706	0.59513	0.55734

PI: Plaque Index

The mean Probing pocket depth (PPD) at baseline, 3 and 6 months for all three groups is given in table 5. The mean PPD at baseline for Group 1 was 3.91241 ± 0.534360, at 3 months it was 3.91341 ± 0.534360 and at 6 months it was 3.91241 ±0.534360. The PPD in Group 1 did not change significantly from baseline levels at 3 and 6 months, as shown in Table 6. The PPD at baseline for Group 2 was 3.91965± 0.528867 which dropped significantly at 3 months to 3.33475 ± 0.397480 and at 6 months it was 3.25359 ± 0.299823. The drop in PPD from baseline was statistically significant at 3 and at 6 months, as shown in Table 6. The PPD at baseline for Group 3 was 3.89790 ± 0.538261 which dropped significantly at 3 months to 3.02842 ± 0.306210 and at 6 months it was 2.99283 ± 0.277104. The drop in PPD from baseline was statistically significant at 3 and at 6 months, as shown in Table 6.

Table 5: Mean Probing Depth

GROUP		PPD Baseline	PPD 3 Month	PPD 6 Month
1(N: 607)	Mean	3.91241	3.91341	3.91241
	SD	0.534360	0.534360	0.534360
2(N: 587)	Mean	3.91965	3.33475	3.25359
	SD	0.528867	0.397480	0.299823
3(N: 604)	Mean	3.89790	3.02842	2.99283
	SD	0.538261	0.306210	0.277104

PPD: Probing Pocket Depth

Table 6: Comparison Of Probing Pocket Depth Between Baseline And 3,6 Month Levels

GROUP		Significance (2-tailed)
GROUP 1	PPD_B - PPD3M	0.132
	PPD_B - PPD6M	0.132
GROUP 2	PPD_B - PPD3M	0.01
	PPD_B - PPD6M	0.01
GROUP3	PPD_B - PPD3M	0.000
	PPD_B - PPD6M	0.000

PPD: Probing Pocket Depth, B: Baseline, M: Month

The mean Clinical Attachment Level (CAL) at baseline, 3 and 6 months for all three groups is given in table 7. The mean CAL at baseline for Group 1 was 4.1301 ± 0.61509, at 3 months it was 4.1334 ± 0.61573 and at 6 months it was 4.1461 ± 0.61375. The CAL in Group 1 did not change significantly from baseline levels at 3 and 6 months, as shown in Table 10. The CAL at baseline for Group 2 was 4.1792 ± 0.62204 which dropped significantly at 3 months to 3.4429 ± 0.35564 and at 6 months it was 3.4199 ± 0.33323. The drop in CAL from baseline was statistically significant at 3 and at 6 months, as shown in Table 8. The CAL at baseline for Group 3 was 4.8882 ± 0.59438 which dropped significantly at 3 months to 3.4200 ± 0.55134 and at 6 months it was 3.1680 ± 3.90226. The drop in CAL from baseline was statistically significant at 3 and at 6 months, as shown in Table 8.

Table 7: Mean Clinical Attachment Level

GROUP		Baseline CAL	3 month CAL	6 month CAL
1(N: 607)	Mean	4.1301	4.1334	4.1461
	SD	0.61509	0.61573	0.61375
2(N: 587)	Mean	4.1792	3.4429	3.4199
	SD	0.62204	0.35564	0.33323
3(N: 604)	Mean	4.8882	3.4200	3.1680
	SD	0.59438	0.55134	3.90226

Table 8: Comparison of Clinical Attachment Levels between Baseline and 3, 6 Month Levels

GROUP		Significance (2-tailed)
GROUP 1	CAL_B-CAL3M	0.158
	CAL_B-CAL6M	0.835
GROUP 2	CAL_B-CAL3M	0.000
	CAL_B-CAL6M	0.222
GROUP3	CAL_B-CAL3M	0.000
	CAL_B-CAL6M	0.002

Table 9: Bleeding On Probing (%)

GROUP		BOP Baseline	BOP 3 Month	BOP 6 Month
1 (N: 25)	Mean	94.89	95.5988	95.4424
	SD	3.634	3.24877	2.80860
2 (N: 25)	Mean	94.58	64.2284	58.0692
	SD	4.169	5.51870	2.08244
3(N: 25)	Mean	94.26	68.1856	57.3816
	SD	4.105	3.51863	4.81685
Total	Mean	94.58	76.0043	70.2977
	SD	3.931	14.64679	18.22001

BOP: Bleeding on Probing

TABLE 10: MEAN FASTING PLASMA GLUCOSE (Mg/Dl)

GROUP		FPG Baseline	FPG 3 Month	FPG 6 Month
1 (N: 25)	Mean	135.32	130.04	136.36
	SD	21.925	17.698	15.408
2 (N: 25)	Mean	139.20	126.08	131.92
	SD	22.697	16.708	20.441
3(N: 25)	Mean	142.32	127.28	135.52
	SD	21.984	19.680	20.447

FPG: Fasting Plasma Glucose

The mean bleeding on Probing (BOP) at baseline, 3 and 6 months for all three groups is given in table 9. The mean BOP (%) at baseline for Group 1 was 94.89 ± 3.634 %, at 3 months it was 95.5988 ± 3.24877 %and at 6 months it was 95.4424 ± 2.80860 %. The mean BOP (%) at baseline for Group 2 was 94.58 ± 4.169 % which dropped significantly at 3 months to 64.2284 ± 5.51870 % and at 6 months it was 58.0692± 2.08244 %. The drop in BOP from baseline was statistically significant at 3 and at 6 months. The mean BOP (%) at baseline for Group 3 was 94.26± 4.105% which dropped significantly at 3 months to 68.1856 ± 3.51863% and at 6 months it was 57.3816 ± 4.81685 %. The drop in BOP from baseline was statistically significant at 3 and at 6 months.

Metabolic Parameters: Table 10, 14 and 16 shows there was no significant difference between the means of three groups associated with all the metabolic parameters at baseline.

TABLE 10: MEAN FASTING PLASMA GLUCOSE (Mg/Dl)

GROUP		FPG Baseline	FPG 3 Month	FPG 6 Month
1	Mean	135.32	130.04	136.36
	N	25	25	25
	SD	21.925	17.698	15.408
2	Mean	139.20	126.08	131.92
	N	25	25	25
	SD	22.697	16.708	20.441
3	Mean	142.32	127.28	135.52
	N	25	25	25
	SD	21.984	19.680	20.447

FPG: Fasting Plasma Glucose

Table 11: Comparison of Fasting Plasma Glucose between Baseline, 3 and 6 Months

GROUP		Significance (2-tailed)
GROUP 1	FPG_B - FPG3M	0.158
	FPG_B - FPG6M	0.835
GROUP 2	FPG_B - FPG3M	0.000
	FPG_B - FPG6M	0.222
GROUP3	FPG_B - FPG3M	0.000
	FPG_B - FPG6M	0.002

FPG: Fasting Plasma Glucose

Table 12: Mean 2-Hour Post Prandial Glucose (Mg/Dl)

GROUP		PPG Baseline	PPG 3 Month	PPG 6 Month
1	Mean	169.28	161.44	164.52
	N	25	25	25
	SD	17.674	17.215	16.566
2	Mean	174.04	150.12	158.32
	N	25	25	25
	SD	19.659	18.274	18.270
3	Mean	167.96	148.40	156.24
	N	25	25	25
	SD	20.695	17.127	17.089

PPG: 2 hours Post Prandial Glucose

Table 13: Comparison of 2-Hour Post Prandial Glucose (Mg/Dl) Between Baseline, 3 and 6 Months

GROUP		T	df	Significance (2-tailed)
GROUP 1	PPG_B – PPG3M	2.233	24	0.055
	PPG_B - PPG6M	1.178	24	0.250
GROUP 2	PPG_B – PPG 3M	6.376	24	0.000
	PPG_B - PPG6M	3.642	24	0.001
GROUP3	PPG_B – PPG3M	7.482	24	0.000
	PPG_B - PPG6M	4.659	24	0.000

PPG: 2 hours Post Prandial Glucose

Table 14: Mean HbA1c (%)

GROUP		HbA1c Baseline	HbA1c 3 Month	HbA1c 6 Month
1	Mean	7.932	8.016	8.032
	N	25	25	25
	SD	1.1338	1.0995	1.0094
2	Mean	8.280	7.444	7.916
	N	25	25	25
	SD	1.5452	1.2244	1.3437
3	Mean	8.100	7.252	7.604
	N	25	25	25
	SD	1.6000	1.2423	1.3801
Total	Mean	8.104	7.571	7.851
	N	75	75	75
	SD	1.4290	1.2189	1.2517

HbA1c: Glycated haemoglobin A1

Table 15: Comparison of HbA1c Values (%) Between Baseline, 3 and 6 Months

		t	df	Significance (2-tailed)
GROUP 1	HBA1C_B - HBA1C3M	-2.064	24	0.060
	HBA1C_B - HBA1C6M	-1.382	24	0.180
GROUP 2	HBA1C_B - HBA1C3M	9.855	24	0.000
	HBA1C_B - HBA1C6M	6.967	24	0.000
GROUP3	HBA1C_B - HBA1C3M	10.133	24	0.000
	HBA1C_B - HBA1C6M	7.327	24	0.000

The mean for Fasting Plasma Glucose (FPG) at baseline, 3 and 6 months for all three groups is given in table 10. The baseline mean for Fasting Plasma Glucose (FPG) was 135.32 ± 21.925 mg/dl for group 1, which was 130.04 ± 17.698 mg/dl at 3 months and 136.36 ± 15.408 mg/dl at 6 months. The baseline mean for FPG (mg/dl) was 139.20 ± 22.697 mg/dl for group 2, which dropped significantly to 126.08 ± 16.708 mg/dl at 3 months and 131.92 ± 20.441 mg/dl at 6 months. The baseline mean for FPG (mg/dl) was 142.32 ± 21.984 mg/dl for group 3, which dropped significantly to 127.28 ± 19.680 mg/dl at 3 months and 135.52 ± 20.447 mg/dl at 6 months.

The mean for 2 hour Post-Prandial Glucose (PPG) at baseline, 3 and 6 months for all three groups is given in table 12. The baseline mean for 2 hour Post-Prandial Glucose (PPG) was 169.28 ± 17.674mg/dl for group 1, which was 161.44 ± 17.215 mg/dl at 3

months and 164.52 ± 16.566mg/dl at 6 months. The baseline mean for PPG (mg/dl) was 174.04 ± 19.659mg/dl for group 2, which dropped significantly to 150.12 ± 18.274mg/dl at 3 months and 158.32 ± 18.270 mg/dl at 6 months. The baseline mean for PPG (mg/dl) was 167.96 ± 20.695 mg/dl for group 3, which dropped significantly to 148.40 ± 17.127 mg/dl at 3 months and 156.24 ± 17.089 mg/dl at 6 months.

The mean for HbA1c (%) at baseline, 3 and 6 months for all three groups is given in table 14. The baseline mean for HbA1c (%) was 7.932 ± 1.1338 mg/dl for group 1 which shows moderate metabolic control. At 3 month, HbA1c was 8.016 ± 1.0995 mg/dl and 8.032 ± 1.0094 mg/dl at 6 months. However the difference in HbA1c (%) at 3 month and 6 months from baseline was not statistically significant with a p value>0.05, as shown in table 17. The baseline mean for HbA1c (%) was 8.280 ± 1.5452 mg/dl for group, which dropped significantly to 7.444 ± 1.2244 mg/dl at 3 months and increased to 7.916 ± 1.3437 mg/dl at 6 months. However the difference in HbA1c (%) at 3 month and 6 months from baseline was statistically significant with a p value< 0.01, as shown in table 15. The baseline mean for HbA1c (%) was 8.100 ± 1.6000mg/dl for group 3, which dropped significantly to 7.252 ± 1.2423 mg/dl at 3 months and rose to 7.604 ± 1.3801mg/dl at 6 months. However the difference in HbA1c (%) at 3 month and 6 months from baseline was statistically significant with a p value< 0.01, as shown in table 15.

Discussion: Periodontal disease has been recognized as a complication of diabetes mellitus.[11] the presence of periodontal disease constitutes a serious health hazard for the diabetic individual. Once the periodontal disease is established, the chronic nature of this infection may contribute to the worsening of the diabetic status leading to more severe diabetes related complications.[3] Three independent studies examined the role of periodontal disease as a factor complicating the severity of diabetes.[12,13] Results from all three studies consistently indicate that diabetics with severe periodontal disease exhibit more diabetes complications compared to diabetics with no or mild periodontal disease, suggesting that presence of periodontal disease confers a significant risk for exhibiting other diabetes related complications.

Additional evidence for the role of periodontal disease complicating diabetes mellitus comes from studies examining the association between periodontal disease and diabetes metabolic control.[14] Mean glycated hemoglobin(HbA1c), a measure of long term glucose control, increased 0.5% in type 2 diabetics with severe periodontitis over an observation period of 2-3 years, whereas this measure of glucose control was reduced 0.9% in those with little or no periodontal disease, independent of the effect of diabetes medication.[14] This study concludes that periodontitis is not only common in type 2 diabetics, it can also worsen the metabolic control of diabetes. 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.

The present study was undertaken with the aim to investigate the effect of periodontal therapy on the glycemic control of type 2 Diabetes mellitus patients. Since the majority of studies published on this aspect have been carried out on men and women who may differ greatly from the average Indian adult with respect to race, socio-economic status and access to health services in general, this study was an attempt to reveal whether the suggested association between periodontal disease and diabetes could be found in a Type 2 Diabetic Indian population.

The present study was carried out in 75 subjects (41 males and 34 females) who were divided into three groups. At baseline, patients in all three groups showed similar levels of plaque accumulation, gingival and periodontal inflammation (PI, BOP) as well as of periodontal breakdown (PPD, CAL). The healing results of periodontal therapy were assessed after 1st and 3rd months following the periodontal treatment.

Considering the first objective of the study, the FPG was measured and compared at baseline, 3 and 6 months within all three groups. There was a significant decrease in FPG from 139.20 ± 22.697 to 126.08 ± 16.708 mg/dl at 3 months and 131.92 ± 20.441 mg/dl at 6 months for group 2. With respect to group 3, the FPG decreased from 142.32 ± 21.984 mg/dl to 127.28 ± 19.680 mg/dl at 3 months and 135.52 ± 20.447 mg/dl at 6 months. There was no significant change in the FPG from baseline in Group 1. Stewart JE et al in 2001[15] found similar decreases in FPG in the treatment group; however the differences did not reach statistical significance.

The second objective of the study was to measure and compare the change in 2-h PPG at baseline, 3 and 6 months. There was a significant decrease in 2-h PPG from 174.04 ± 19.659mg/dl to 150.12 ± 18.274mg/dl at 3 months and 158.32 ± 18.270 mg/dl at 6 months for group 2. With respect to group 3, the 2-h PPG decreased from 167.96 ± 20.695 mg/dl to 148.40 ± 17.127 mg/dl at 3 months and 156.24 ± 17.089 mg/dl at 6 months. There was no significant change in the 2-h PPG from baseline in Group 1. This finding is in accordance with Kiran M et al. in 2005[16] who found similar decreases in 2-h PPG in the treatment group, however the differences did not reach statistical significance.

Considering the final objective of the study, the HbA1c(%) was measured and compared at baseline, 3 and 6 months within all three groups There was a significant decrease in HbA1c (%) from 174.04 ± 19.659mg/dl to 150.12 ± 18.274mg/dl at 3 months and 158.32 ± 18.270 mg/dl at 6 months for group 2. This finding is in accordance with Kiran M et al in 2005[16] who found similar decreases in HbA1c (%) at the 3 month stage in the non-surgically treated treatment group. However, Smith et al 1996[17] and Christgau et al in 1998[18] found no significant change in the HbA1c (%) values after non-surgical periodontal therapy. Other studies involving periodontal treatment alone reported improvement in periodontal status only.[19,20,21]

With respect to group 3, the HbA1c (%) decreased from 167.96 ± 20.695 mg/dl to 148.40 ± 17.127 mg/dl at 3 months and 156.24 ± 17.089 mg/dl at 6 months. There was no significant change in the HbA1c (%) from baseline in Group 1. This finding is in accordance with Miller et al in 1992,[22] Grossi SG et al in 1997[23] and Iwamoto Y et al in 2001[24] who found similar decreases in HbA1c (%) which were statistically significant in the non-surgically treated treatment group who were also given systemic doxycycline. Also Group 3 showed a tendency towards greater decrease in HbA1c levels as compared to Group 2, however this difference was not statistically significant.

Two conclusions can be drawn from these results. One, since the decrease in metabolic parameters were seen only in the groups which were given periodontal treatment alone and periodontal treatment combined with antibiotics, and no change was seen in the control group – it can be inferred that the decrease was due to the decrease/ elimination of periodontal disease. The reduction in glycated hemoglobin observed in our study treated groups was independent of current treatment for diabetes, and could not be explained by a change in treatment regimen; i.e., insulin versus oral hypoglicemics or dose of diabetes medication. Therefore, it is likely the result of periodontal treatment. This study provides the evidence that elimination of periodontal infection and improvement of periodontal inflammation significantly reduced the HbA1c in the short term, thus improving diabetes metabolic control.

Second, the administration of systemic doxycycline provides an additional benefit beyond the strictly antimicrobial use. It has been demonstrated that tetracyclines and their chemically modified derivatives have, independent of their antimicrobial effect, a modulatory effect on the host response by suppressing or inhibiting collagenolytic processes and increasing protein synthesis and secretion.Ryan et al in 1998[25] reported a decrease in the level of glycated hemoglobin and collagen degradation in diabetic rats following administration of doxycycline or chemically-modified tetracycline. The authors proposed that extracellular glycation of proteins in diabetes are inhibited by tetracycline via a non-anti-collagenase mechanism. Overall, this evidence has provided the basis for a therapeutic approach to controlling periodontal disease in individuals with diabetes, by tetracyclines and their derivatives. It is proposed that the reduction in HbA1c seen at 3 months and 6 months in the doxycycline-treated group is the combined result of the antimicrobial effect and possibly a doxycycline-mediated inhibition of the glycation process. Therefore, the use of doxycycline as an adjunct to antiinfective periodontal therapy in diabetics may have a dual benefit. First, as a broad spectrum antibiotic effective against most periodontal pathogens, doxycycline reaches concentrations in the gingival fluid 7-10 fold over serum levels, thus providing an important adjunct in the reduction of periodontal pathogens.[6] Second, as a potent modulator of the diabetic patient's host response to the periodontal infection, doxycycline inhibits non-enzymatic glycation of extracellular proteins and possibly has a similar effect on glycation of hemoglobin as well.[6]

Results of this study suggest that following periodontal therapy alone and periodontal therapy in combination with doxycycline therapy, there was a

marked improvement in glycaemic control in individuals with type 2 DM when compared with a non-treatment control group. Although the pathogenesis is poorly understood, it is generally accepted that infection results in a state of insulin resistance and that bacterial LPS has a significant effect on insulin sensitivity.[6] The release of IL-1β and TNF-α in response to bacteremia/endotoxemia has numerous metabolic effects in addition to hyperlipidemia. Elevated levels of IL-1β are thought to play a role in the development of type1 diabetes.[6] TNF-α has been implicated as a causative factor in insulin resistance and type 2 diabetes in animal models and in human stuies.[6] Thus, infection induced insulin resistance syndromes, if long standing and chronic, are considered to be precursors to active diabetes due to the pancreatic β-cell destruction that results from sustained elevation of IL-1β/TNF-α. Some investigators have recently observed that systemically healthy patients with moderate periodontitis demonstrated significantly higher blood glucose levels than patients without periodontitis. This observation may indicate that patients with periodontitis have impaired glycaemic control and are in a 'pre diabetic' state. These investigators suggest that proinflammatory cytokines such as IL-1β and TNF-α, produced as a systemic response to periodontal infection, are responsible for insulin resistance and subsequent poor glycaemic control in periodontitis patients.[6] Thus, the elimination of Periodontal infection will lead to a subsequent decrease in IL-1β and TNF-α, which would decrease the insulin resistance and better the metabolic control of the diabetic individual.

The results of this study suggest that following periodontal therapy alone and periodontal therapy in combination with doxycycline therapy, there was a marked improvement in glycaemic control in individuals with type 2 DM when compared with a non-treatment control group.

Conclusion: 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.

Results of this study showed that non-surgical periodontal treatment with and without antibiotic therapy (doxycycline) is associated with improved glycaemic control in type 2 DM patients and reduction of clinical parameters of periodontal infection, confirming the existing interrelationship between Diabetes mellitus and periodontal disease. An improved communication between dentists / periodontists and physicians/ endocrinologists is needed so as to work together to improve the management of Diabetic patients.

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Conflict of interest: None

Funding: None

Cite this article as: Joshi V, Chivte P, Patil K, Nayyar A. The Relationship between Periodontal Disease and Glycemic Status of Type Ii Diabetic Patients in Indian Population. Natl J Integr Res Med 2015; 6 (1): 7-17