Journal of Periodontal Medicine & Clinical Practice

Vol- 1, Issue - 1, Jan - Apr 2014

www.jpmcp.com

Original Research

Outcome Of Non Surgical Periodontal Therapy On Glycemic Control In Patients With Type 2 Diabetes Mellitus

Dr. Yashbir Singh Raghav[1], Dr. Yash Paul Dev[2], Dr. Priyanka Singla[1], Dr. Dinesh Duhan[1]

Raghav YS, Dev YP, Singla P, Duhan D. **Outcome of non surgical periodontal therapy on glycemic control in patients with type 2 diabetes mellitus.** J Periodontal Med Clin Pract 2014;01:53-60

Affiliation:

1. Post Graduate student, Department of Pseriodontology, Swami Devi Dyal Hospital and Dental College, Barwala, Panchkula, Haryana, India.
2. Professor & Head, Department of Periodontology, Swami Devi Dyal Hospital and Dental College, Barwala, Panchkula, Haryana, India.

Corresponding author:-

Dr. Yashbir Singh Raghav
Post Graduate Student, Department of Periodontology,
Swami Devi Dyal Hospital and Dental College,
Barwala, Panchkula, Haryana, India
Email: yashbir.rr@gmail.com

Abstract:-

Background: The literature suggests that an alteration in glucose metabolism occurs as a result of antibacterial therapy. The objective of this study was to monitor the effect of nonsurgical periodontal therapy on glycemic control of patients with type 2 diabetes mellitus (DM).

Method: Thirty type 2 DM subjects with periodontitis were randomly divided into two groups. Group 1, 15 patients received one stage full mouth scaling and root planing (FMSRP)along with amoxicillin/clavulanic 625 mg and Group 2, 15 patients received only (FMSRP). At baseline and at 3 months, the glycated hemoglobin HbA1c, fasting glucose and clinical parametes are recorded.

Result: After 3 months, both groups show clinical improvements. Group 1 shown a mean probing depth reduction from 6.2 ± 1.03 mm to 6.1 ± 0.83 mm and Group 2 from 6.1 ± 0.8 mm to 5.1 ± 0.83 mm. however, the difference between the group were not statistically significantly ($p \leq 0.05$).

Conclusion: Periodontal therapy improve glycemic control in patients with type 2 DM in both groups however, the reduction in HbA1c values reached statistical significance only in the group receiving scaling and root planing with amoxicillin/ clavulanic acid 625 mg.

KEY WORDS: Glycated hemoglobin ($HbA1_c$), non insulin dependent diabetes mellitus (NIDDM)

INTRODUCTION

Type 2 diabetes mellitus results from defect in the insulin molecule, altered insulin cell receptor or insulin resistance rather than deficiency. This metabolic problem results in hyperglycemia, the non- specific glycosylation of proteins and microangiopathy. Onset of symptoms is generally gradual. According to Mealey B(1999)Patients are often obese and their glucose tolerance can be typically improved with diet and body weight control.[1]

The classic signs of diabetes include polydipsia (excessive thirst), polyuria (excessive urination) and polyphagia (excessive hunger). If the patient has any of these signs or

symptoms, further investigation with laboratory studies and physician consultation is indicated.

The association between periodontal diseases and diabetes mellitus has been demonstrated in several studies over the years. According to Loe H (1991)Periodontal diseases are recognized as " the sixth complication of diabetes mellitus".[2]In 1990, epidemiological studies in a population presenting an extremely high prevalence of type 2 diabetes mellitus revealed that this population also had a high prevalence for periodontal diseases. Novaes AB Jr(1991) divided the diabetic group into controlled, moderately and poorly controlled diabetes mellitus, there was significant difference in periodontal status among the poorly controlled diabetics and control group suggesting that DM affects the evolution of periodontal diseases.[3,4,5]

Grossi and Genco (1998)[6]proposed the model for two way relationship between periodontal diseases and DM. The biochemical basis whereby hyperglycemia may lead to the microvascular complications seen in DM is increased accumulation of advanced glycation end products(AGEs).[7] Cristgau et al[8] reported that mechanical therapy had no effect on the levels of glycated hemoglobin when looking at poorly controlled diabetes. Stewart et al (2001)[10] suggested in a retrospective study that there was a marked improvement in glycemic control in individual with type 2 DM following periodontal therapy.

Mongardini C(1999)[11,12] have reported that there was significant improvement up to 8 months, both from clinical and microbiological point of view, when the full mouth approach was compared to the conventional treatment .

Successful elimination of periodontal infection with systemic antibiotics would significantly reduce the systemic bacterial challenge with a concomitant reduction in the secretion of inflammatory mediators and improve metabolic control of type 2 DM .In this study, a treatment protocol was designed to manage periodontal diseases associated with DM and to compare the changes in glycemic control in a group of patients with type 2 DM following one stage full- mouth scaling and root planing alone or combination with amoxicillin/clavulanic acid.[15]

MATERIALS AND METHODS

This study included 30 patients of type 2 Diabetes mellitus having chronic periodontitis.

Inclusion Criteria[16]

- At least one site with probing depth ≥5mm.
- Two teeth with clinical attachment loss ≥6mm.

Exclusion Criteria

- Any form of periodontal treatment in the previous 6 months.
- Insulin utilization.
- Smoking, pregnancy and DM diagnosis within the past 5 years.

CLINICAL PARAMETERS:-

At the time of periodontal examination, the following clinical parameters were recorded at six sites per tooth at baseline and 3 months following treatment.[17]

• Glycated	Pair 1
• Fasting glucose level	Pair 2
• Bleeding on probing.(BOP)	Pair 3
	Pair 4
• Suppuration.(S)	Pair 5
• Probing depth.(PD)	Pair 6
• Clinical attachment	Pair 7
CLINICAL PARAMETERS	

Probing depth and clinical attachment level was recorded with a probe at six sites per tooth using customized acrylic stent as a reference to determine the site and angle of the measurements. Biofilm, bleeding on probing and suppuration were recorded as absent(0) or present(1). Blood samples were taken at baseline and at the 3 month recall visit to monitor glycated hemoglobin and fasting glucose levels.

METHODOLOGY

The subjects were randomly distributed into two groups:-

Group 1(G 1):- Full mouth scaling and root planing only(15 patients)

Group 2 (G 2):- Full mouth scaling and root planing in combination with amoxicillin/clavulanic acid(15 patients)

All patients received standard oral hygiene instructions before the first session of scaling and root planing without the use of chlorohexidine.[18] Oral hygiene control and reinstruction were reviewed two times monthly, followed by prophylaxis. Group 2 received systemic amoxicillin/clavulanic acid 625mg twice daily for 2 weeks after therapy. At third month visit, changes in clinical parameters along with HbA_1clevel and fasting glucose level of both the groups are compared.

STATISTICALANALYSIS

The mean and standard deviation values per site of the clinical parameters were calculated. The change in probing depth, relative attachment level, percentage of surfaces exhibiting biofilm, bleeding on probing and suppuration between baseline and the 3 month visit were tested within the treatment groups using the wilcoxon test. The differences in changes in HbA_1clevels between groups were assessed using analysis of covariance (ANCOVA) to eliminate the influence of the baseline variables.

RESULTS

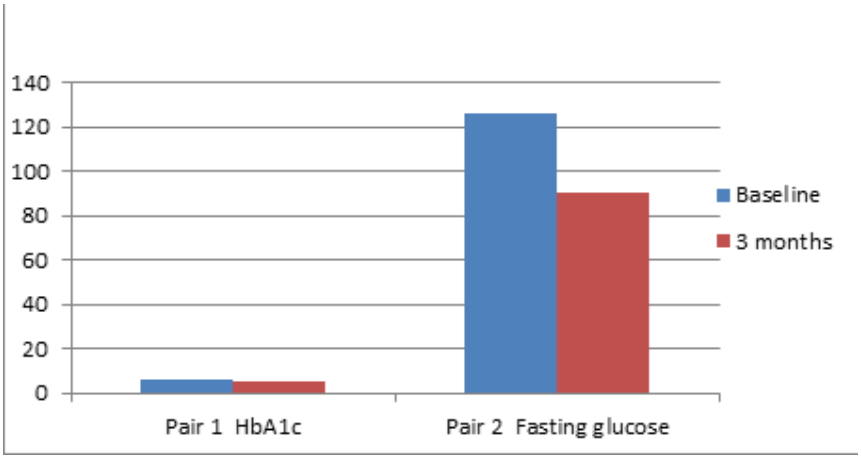
The HbA_1cvalues were reduced for both groups, although only the changes in group 2 were statistically significant ($p<.05$).(Graph 3) The baseline mean fasting glucose level and at third month visit, results for both the groups were statistically significant($p<.05$).(Graph 1 &3) Both the therapies resulted in significant in periodontal parameters at third month visit, except clinical attachment loss in group 2 (Graph 2 &4).

Both treatment groups showed similar mean HbA_{1C} level and periodontal parameters at baseline. The fasting glucose level between the two groups was statistically significant.($p<.05$)

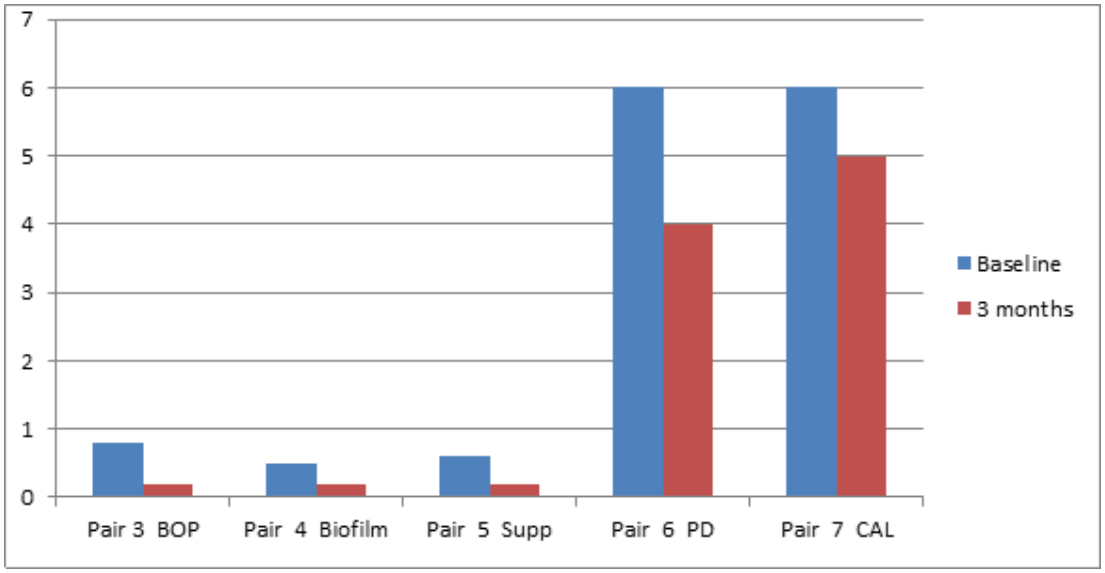
Both the groups showed reduction inHbA_1clevels. The change inHbA_1c levels was .8% in Group1 and 2.8% in Group 2 with statistically difference between groups by ANCOVA. (Table 1 & 3)

Table 1: Clinical parameters at baseline and at third month of group 1

Clinical parameters		N	Mean±Std. Deviation	
Pair 1	(Baseline)	15	6.0000±	1.30931
	(3 months)	15	5.2000±	.69591
Pair 2	(Baseline)	15	126.600±	8.30490
	(3 months)	15	90.8667±	7.21968
Pair 3	(Baseline)	15	.8000±	.41404
	(3 months)	15	.2667±	.45774
Pair 4	(Baseline)	15	.5333±	.51640
	(3 months)	15	.2000±	.41404
Pair 5	(Baseline)	15	.6667±	.48795
	(3 months)	15	.2667±	.45774
Pair 6	(Baseline)	15	6.2667±	1.03280
	(3 months)	15	4.4667±	.83381
Pair 7	(Baseline)	15	6.5333±	.51640
	(3 months)	15	5.4667±	.51640



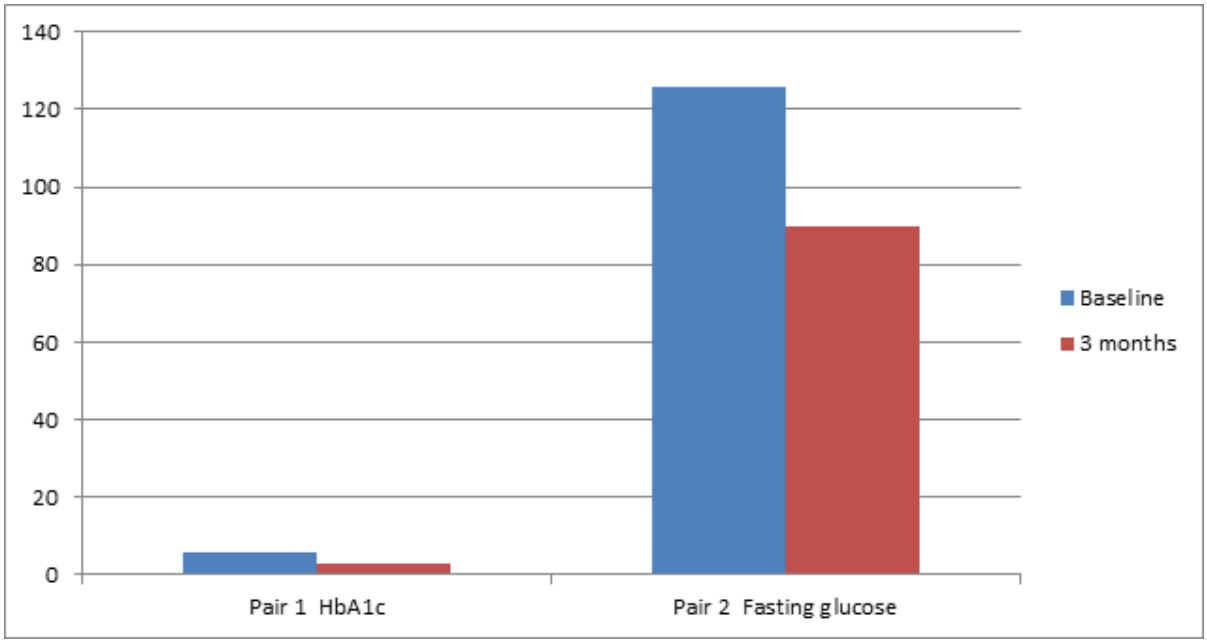
Graph 1:- HbA1c and fasting glucose at baseline and at 3 month in group 1



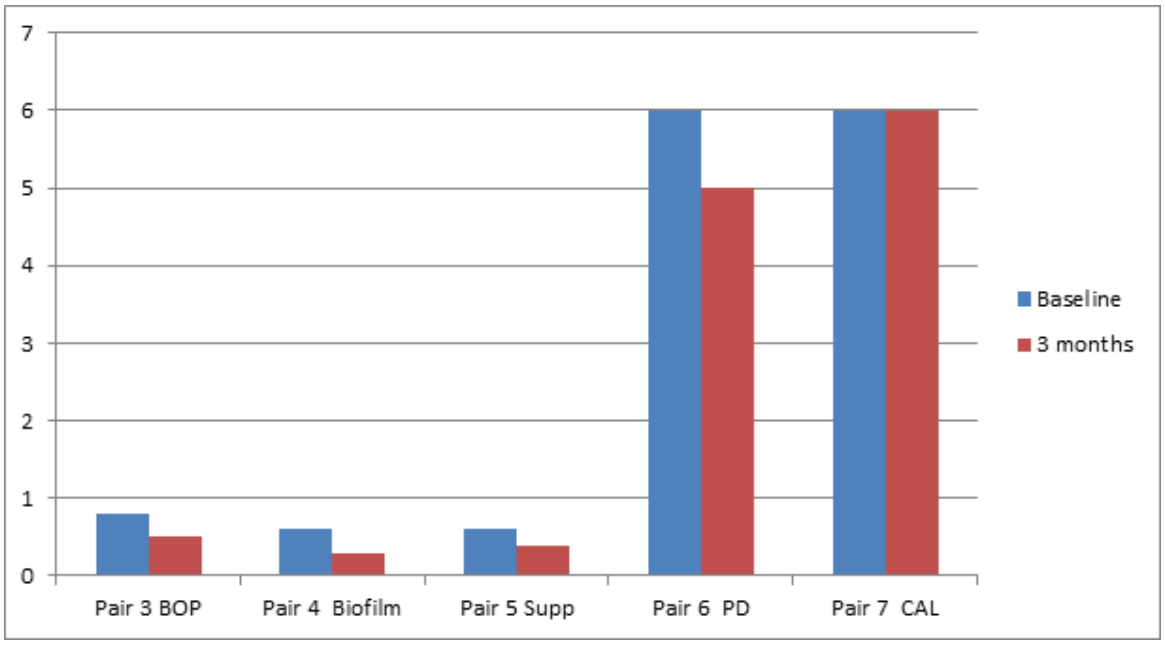
Graph 2:- Clinical parameters at baseline and at 3 month in group 1

Clinical parameters		N	Mean	Std. Deviation
Pair1	(Baseline)	15	6.0000±	1.30931
	(3 months)	15	3.8667±	1.30201
Pair 2	(Baseline)	15	126.60±	8.30490
	(3 months)	15	90.000±	6.55744
Pair 3	(Baseline)	15	.8000±	.41404
	(3 months)	15	.5333±	.51640
Pair 4	(Baseline)	15	.6000±	.50709
	(3 months)	15	.3333±	.48795
Pair 5	(Baseline)	15	.6667±	.48795
	(3 months)	15	.4000±	.50709
Pair 6	(Baseline)	15	6.1333±	.83381
	(3 months)	15	5.1333±	.83381
Pair 7	(Baseline)	15	6.6667±	.48795
	(3 months)	15	6.1333±	.63994

Table 2: Clinical parameters at baseline and at third month of group 2



Graph 3:- HbA1c and fasting glucose at baseline and at 3 month in group 2



Graph 4:- Clinical parameters at baseline and at 3 month in group 2

Table 3: Inter group comparison of group 1 and group 2

Scaling-(Group 1- G1) Scaling+ antibiotics-(Group 2-G2)		N	Mean	Std. Deviation	P value
(Baseline) Pair 1	G1	15	6.0000±	1.30931	1.000
	G2	15	6.0000±	1.30931	1.000
Pair 2	G1	15	126.6000±	8.30490	1.000
	G2	15	126.6000±	8.30490	1.000
Pair 3	G1	15	.8000±	.41404	1.000
	G2	15	.8000±	.41404	1.000
Pair 4	G1	15	.5333±	.51640	1.000
	G2	15	.6000±	.50709	.724
Pair 5	G1	15	.6667±	.48795	.724
	G2	15	.6667±	.48795	1.000
Pair 6	G1	15	6.2667±	1.03280	1.000
	G2	15	6.1333±	.83381	.700
Pair 7	G1	15	6.5333±	.51640	.700
	G2	15	6.6667±	.48795	.473
(3 months) Pair 1	G1	15	5.2000±	.69591	.473
	G2	15	3.8667±	1.30201	.002
Pair 2	G1	15	90.8667±	7.21968	.002
	G2	15	90.0000±	6.55744	.733
Pair 3	G1	15	.2667±	.45774	.733
	G2	15	.5333±	.51640	.146
Pair 4	G1	15	.2000±	.41404	.146
	G2	15	.3333±	.48795	.426
Pair 5	G1	15	.2667±	.45774	.427
	G2	15	.4000±	.50709	.456
Pair 6	G1	15	4.4667±	.83381	.456
	G2	15	5.1333±	.83381	.037
Pair 7	G1	15	5.4667±	.51640	.037
	G2	15	6.1333±	.63994	.004

The baseline mean fasting glucose levels for both groups were not statistically different ($p<0.05$); Group 1 126±8.3 mg/dl and Group 2 126±8.3mg/dl. At the 3 month visit, fasting glucose level were Group 1 90± 7.2mg/dl and Group 2 90± 6.5 mg/dl respectively, however the changes were not statistically significant($p>0.05$).(Table 1& 3)

Both therapies resulted in significant improvement in periodontal parameter over the experimental period. At the 3 month examination, a statistically significant improvement in probing depth was recorded treatment groups. Group 1 shown a mean depth reduction from 6.2 ± 1.03 mm to 6.1 ± 0.83 mm and Group 2 from 6.1 ± 0.8 mm to 5.1 ± 0.83 mm. however the difference between the group were notstatistically significantly ($p \leq 0.05$).(Table 2)

Group 1 shows a mean relative attachment level of 6.5± mm and 5.4± mm and group 2 of 6.6± .48mm and 6.1 ±.63 mm at baseline and post treatment respectively. The mean between groups differ significantly.($p \leq 0.05$)(Table 2)

Both groups presented and improvement in bleeding on probing, with a significant reduction in the mean percentage of sites with bleeding on probing at 3- months visit. ($p< 0.05$). (Table 2 &4)

There was a significant reduction in the mean percentage of sites with suppuration in both groups ($p <0.05$), but no detectable difference between groups over study period. ($p> 0.05$).(Table 2 & 4)

Both groups presented a statistically significant changes in mean percentage of sites with Biofilm ($p < 0.05$)but no detectable difference between groups over the study period ($p > 0.05$).(Table 2 & 4)

DISCUSSION

The two groups studied showed an improvement in clinical periodontal conditions i.e., a reduction in probing depth and percentage of sites with bleeding and suppuration. The metabolic control of diabetes also was influenced; there was reduction in HbA_1clevels, although the Group 2, which received periodontal therapy plus antibiotics, presented a less favorable response. Having shown that the presence of infection, mainly periodontal diseases, can influence an individual's systemic conditions,[19] a series of studied have attempted to verify the effects of periodontal therapy in the control of other pathologies.[20]

The periodontal inflammation has a significant impact on various systemic diseases with high prevalence, incidence, morbidity, and mortality; e.g., diabetes mellitus (DM) and cardio vascular disease (CVD).[21]

Thus, preexisting periodontitis influences glycemic control and complication outcomes in patients with DM. Conversely, extensive epidemiologic studies have shown that DM increases the risk of periodontal tissue damage.

Glycemic control and disease duration are important variables in the relationship between DM and periodontal diseases. Most previous investigations suggest that long-lasting DM and especially poor metabolic control are risk factors for severe periodontitis, but there is significant heterogeneity among individuals with DM .[22]

It is assumed that the etiologic mechanisms by which DM influences periodontitis are similar to the pathways of the other known complications. Although the level of glycemic control and enhanced inflammation may play the central role with respect to periodontitis, other risk modifiers, such as cigarette smoking or genetic background, in combination with DM may contribute to cumulative risks.

CONCLUSION

The two groups studied shown an improvement in clinical periodontal conditions' a reduction in probing depth and percentage of sites with bleeding and suppuration. The metabolic control of diabetes also was influenced; there was a reduction in HbA_1clevels,although G2 group presented a less favorable response. One stage non-surgical periodontal therapy results in additional clinical and microbiological benefits, demonstrating clinical improvement superior to conventional periodontal therapy, mainly by preventing bacterial recolonization of the sites by microorganism that remained in the non treated area.

The diabetic patient is considered to be at high risk for infection due to vascular alteration and a deficient healing response, the one stage therapy helps minimize possible reinfection of the treated areas as it reduce edema which is responsible for maintaining high level of proinflammatory cytokines.

REFERENCES

1. Mealey B. Diabetes and periodontol diseases. J Periodontol 1999;70:935-949.
2. Loe H. Periodontal diseases: The sixth complication of diabetes mellitus. Diabetes Care 1993;16:329-334
3. Emrich LJ, Shlossman M, Genco RJ. Periodontol diseases in non-insulin dependent diabetes mellitus. J Periodontol 1991;62:123-130
4. Novaes AB Jr, Silva MAP, Batista EL Jr, Anjos BA, Novaes AB, Pereira ALA. Manifestation of insulin dependent diabetes mellitus in the periodontium of

young brazillian patients. J Periodontol 1991;62:116-122
5. Novaes AB Jr, Silva MAP, Batista EL Jr, Anjos BA, Novaes AB, Pereira ALA. Manifestation of insulin dependent diabetes mellitus in the periodontium of young brazillian patients.A 10 year follow up study. J Periodontol 1997;68:328-334
6. Grossi SG, Genco R. Periodontol diseases and diabetes mellitus: A two way relationship. Ann Periodontol 1998;3:51-61
7. Schmidt AM, Weidman E, Lalla E. Advanced glycation end products(AGEs) induce oxidant stress in the gingiva: A potential mechanism underlying accelerated periodontal diseases associated with diabetes. J Periodont Res 1996;31:508-515
8. Christagau M, Palitzch KD, Kreiner SG, Frenzel S. Healing response to non surgical periodontal therapy in patient with diabetes mellitus: Clinical, microbiological and immunological results. J Clin Periodontol 1998;25:112-124
9. Westfelt E, Rylander H, Blohme G, Jonasson P, Lindhi J. The effect of periodontal therapy in diabetes. Result after 5 years. J Clin Periodontol 1996;23:92-100
10. Stewart JE, Wager KA, Friedlander AH, Zadeh HH. The effect of periodontal treatment on glycemic control in patient with type 2 diabetes mellitus. J Clin Periodontol 2001;28:306-310
11. Mongardini C, Van steenberghe D, Dekseyser C, Quirynen M. One stage full versus partial mouth disinfection in the treatment of chronic adult or generalized early onset periodontitis. J Periodontol 1999;70:632-645
12. Quirynen M, Mongardini C, Pauwels M, Bollen C, van Eldere J, Van Steenberghe D. One stage full versus partial mouth disinfection in the treatment of chronic adult or generalized early onset periodontitis. J Periodontol 1999;70:646-656
13. Grossi SG, Skrepcinski FB, De Caro T. Treatment of periodontal diseases in diabetes reduce glycated hemoglobin. J Periodontol 1997;68:713-719
14. Williams R, Mahan C. Periodontal diseases and diabetes in young adults. JAMA 1960;172:776-778
15. Walker CB, Gordon JM, Magnusson l, Clark WB. A role for antibiotics in the treatment of refectory periodontitis. J Periodontol 1993;64:772-781
16. Machtei EE, Christersson LA, Grossi SG, Dunford R, Zambon JJ, Genco RJ. Clinical criteria for the definition of "Established periodontitis". J Periodontol 1992;63:207-215
17. Loe H. Gingival index, plaque index and the retention index systems. J Periodontol 1967;38:610-616
18. Quirynen M, Mongardini C, Pauwels M, Bollen C, Van Eldere J, Van Steenberghe D. One stage full versus partial mouth disinfection in the treatment of chronic adult or generalized early onset periodontitis.Long term clinical and microbiological observation. J Periodontol 2000;27:578-589
19. Greenstein G, Lamster I. Changing periodontal paradigms: Therapeutic implications. Int J Periodontics Restorative Dent 2000;20:337-357
20. Iancopino AM, Cutler CW. Pathophysiological relationship between periodontitis and systemic diseases: Recent concepts involving serum lipids. J Periodontol 2000;71:1375-1384
21. Ramirez-Tortosa MC, Quiles JL, Battino M, et al. Peri-odontitis is associated with altered plasma fatty acids and cardiovascular risk markers. Nutr Metab Cardiovasc Dis 2010;20:133-139.
22. Lalla E. Periodontal infections and diabetes mellitus: When will the puzzle be complete?J Clin Periodontol 2007;34:913-916.

Competing interest / Conflict of interest The author(s) have no competing interests for financial support, publication of this research, patents and royalties through this collaborative research. All authors were equally involved in discussed research work. There is no financial conflict with the subject matter discussed in the manuscript.
Source of support: NIL