the Journal of CONTEMPORARY DENTAL PRACTICE at www.thejcdp.com
Volume 9 Number 5 July 1, 2008

Alterations in HbA1c Following Minimal or Enhanced Non-surgical, Non-antibiotic Treatment of Gingivitis or Mild Periodontitis in Type 2 Diabetic Patients: A Pilot Trial

Theresa E. Madden, DDS, MS, PhD, FACD; Brock Herriges, DMD;
Linda Boyd, RDH, MS, EdD; Gayle Laughlin, DDS, PhD;
Gary T. Chiodo, DMD, FACD; David I. Rosenstein, DMD, MPH

Abstract

Aim: The purpose of this pilot study was to determine and compare the effects of two protocols aimed at reducing periodontal inflammation, upon the metabolic control of the diabetic condition in subjects with elevated baseline glycosylated hemoglobin (HbA1c).

Methods and Materials: Forty-two non-smoking type 2 diabetes subjects with mildly elevated HbA1c (>7 but < 9%) and severely elevated (>9%) were randomized to one of two non-surgical periodontal therapy protocols. Patients in the "minimal therapy" (MT) group received scaling, root planning, and oral hygiene instructions on two occasions six months apart. Participants randomized to the "frequent therapy" (FT) protocol received scaling, root planing, and oral hygiene instructions at two-month intervals and were provided a 0.12% chlorhexidine rinse for home use twice daily. Neither systemic nor local antibiotics were provided to either group. Subjects were asked to report any changes in diabetic medications, nutrition, and physical activity. Data analyses (ANOVA, t-test, Mann-Whitney) grouped subjects according to baseline HbA1c (>7 and < 9%, or > 9%), treatment protocol (minimal or frequent), and +/- medication change.

Results: In both MT and FT groups the clinical attachment level (CAL) remained unchanged but the other measures [gingival index (GI) and pocket dept (PD)] of periodontal health improved. Mean reductions in plaque showed improvement but calculus was worse in the FT group, likely due to the use of chlorhexidine. At six months, the largest reduction of HbA1c was 3.7; experienced by a subject receiving FT but no changes in diabetic medication. Among the MT and no medication change subjects, the maximum reduction was 1.6.

Overall mean reduction in HbA1c of 27 subjects with baseline HbA1c >9.0 and no medication change was 0.6 with no statistical difference between the MT and FT groups. Among the medication-change subjects with baseline HbA1c >9.0, mean reduction of 1.38 was seen with FT compared to 1.10 with MT.

Conclusion: Overall, modest improvements in HbA1c were detected with a trend towards FT being better than MT. Although this pilot trial was under-powered to detect small between-group differences, the magnitude of our findings (0.6 mean improvement in HbA1c) matches closely findings from the only meta-analysis conducted on this topic to date. Larger scale studies must be undertaken on diabetic patients with periodontal problems.

Clinical Significance: Preventive periodontal regimens for diabetic patients should be sufficiently intense and sustained to eliminate periodontal inflammation and should be closely coordinated with the patient's overall clinical diabetic management.

Keywords: Diabetes, periodontal disease, HbA1c

Citation: Madden TE, Herriges B, Boyd L, Laughlin G, Chiodo GT, Rosenstein DI. Alterations in HbA1c Following Minimal or Enhanced Non-surgical, Non-antibiotic Treatment of Gingivitis or Mild Periodontitis in Type 2 Diabetic Patients: A Pilot Trial. J Contemp Dent Pract 2008 July; (9)5:009-016.

Introduction

Oral Health and Glycemic Control

For the diabetic patient, tremendous improvements in health and quality of life are the rewards of maintaining life-long normoglycemia.[1-2] The American Diabetes Association Standards of Medical Care (2006) recommend diabetic patients strive to maintain the HbA1c <7, ideally between 4 and 6.[3-8] While many diabetic patients have difficulty maintaining this level of glycemic control, those achieving intermediate and/or intermittent control experience far fewer complications such as retinopathy, nephropathy, neuropathy, fatigue, weakness, memory loss, cardiovascular disease, need for amputations, tooth loss, and periodontal infection.[1-5,9-28] Using a proportional hazards model and stringent controls of potential confounders, it has been shown the risk of diabetic nephropathy and cardiac mortality in Pima Indians is elevated 3.2 fold (95% CI 1.1-9.3) in those with severe periodontal disease.[23]

Excellent glycemic control is achieved with strict regimens of diet, exercise, weight loss, avoidance of infections, fastidious self-care, avoidance of tobacco, medication adjustments, frequent medical attention, and self-monitoring of blood glucose levels using home glucometers. Emerging evidence suggests the reduction of periodontal inflammation may be one additional strategy in reducing HbA1c.[29-31] When a series of studies of various sizes were subjected to a

systematic review and meta-analysis, periodontal intervention reduced HbA1c by 0.66 in type 2 diabetic patients.[31]

For this pilot study, a small number of diabetic patients with elevated HbA1c were recruited. Because subjects with moderate and severe periodontitis were excluded, the use of antibiotics during this short-term intervention was avoided. Chlorhexidine gluconate was the sole chemotherapeutic agents tested because it has been shown to be effective in managing gingivitis.[32-35]

Methods and Materials

With approval from the Oregon Health and Science University Institutional Review Board,

eligible subjects were recruited using flyers and advertisements in newspapers in the greater Portland area. A screening oral examination, medical history review, and blood sample to determine HbA1c were carried out to confirm eligibility (see Table 1 for inclusion and exclusion criteria) and to answer participant questions about the study. Fifty eligible, consenting subjects were randomized (by flip of a coin) to either the minimal therapy (MT) or frequent therapy (FT) groups. The treatment groups were balanced for gender only.

Following the pre-treatment assessment, all subjects were provided with oral home care instructions, oral prophylaxes including scaling, and root planing was limited to the inflamed periodontal pockets with clinical attachment loss. These services were provided in one appointment which varied in time between 60 and 90 minutes. Control subjects were recalled for oral hygiene instructions and oral prophylaxes with scaling and localized root planing at one six-month interval. FT subjects were recalled for oral prophylaxes with localized scaling. Localized root planing and oral hygiene instruction every two months (four sessions total) were provided at no cost and 0.12% chlorhexidine gluconate rinse (Peridex™, Zila Pharmaceuticals) was given for twice daily, 30 second, oral rinsing. All treatment provided in this protocol was delivered by the research dental hygienist, and all periodontal evaluations were performed by a graduate periodontology resident who was blinded to the subjects' group assignment.

The weight and medical history of each participant was updated at each appointment to identify factors that might impact glycemic control within the six weeks preceding the serological and periodontal data collection. These specific factors included: weight gain or loss, infections, illnesses, the use of antibiotics, steroids, or any other new medications that are known to interfere with blood glucose control. All subjects were also interviewed to gather information on exercise levels and frequency of glucose monitoring and medications. HbA1c was measured at baseline, six months, and eight months following randomization and represented the primary endpoint in this investigation.

The study also evaluated the severity of periodontal disease in subjects using several indices of oral health at baseline, six months, and eight months. Inflammation (GI), clinical attachment loss (CAL), probing depth (PD), plaque index (PI), and calculus index (CI) all were quantified. PI and CI scores were quantified using indices in which the four smooth surfaces of all existing teeth are scored and a percentage

Table 1. Inclusion and exclusion criteria.

Inclusion Criteria	Exclusion Criteria
• Mild or moderate gingivitis OR periodontitis • Elevated HbA1c (>7.0% but <13.11)[30] • History of elevated blood glucose for > one year • 15 or more natural teeth • 18 to 80 years of age	• Need for periodontal surgery • Need for antibiotic prophylaxis • Other conditions or medication interfering with diabetic control • Use of antibiotics, steroids, hydantoin, NSAIDS • Immunosuppression • Rheumatoid arthritis • HIV • Pregnancy • Tobacco use • Fixed orthodontic appliances • Inability to give consent • Unable or unwilling to remain enrolled for eight months

Table 2. Gingival Index.[36]

Löe and Silness Gingival Index (GI)	
0	Normal gingival
1	Mild inflammation – slight change in color, slight edema. No bleeding on probing.
2	Moderate inflammation – redness, edema, and glazing. Bleeding on probing.
3	Severe inflammation – marked redness and edema. Tendency for spontaneous bleeding.

of surfaces with plaque or calculus are recorded. The Löe and Silness GI[36] (Table 2) was used to assess inflammation of the gingiva on the mesial, distal, buccal, and lingual surfaces of the Ramfjord index teeth (#3, 9, 12, 19, 25, and 28).[37]

The GI procedure consisted of inserting a calibrated periodontal probe no more than 2 mm into the gingival sulcus, starting just distal to the midpoint of the buccal surface, then moving the probe tip gently into the mesial interproximal area. CAL and PD were measured at six sites for the index teeth using the Michigan Probe™ calibrated in 2 mm increments. Because PD recordings can vary significantly according to positions of the probe tip, all probing and recession measurements were completed and then repeated for a second set of measurements. Where there was a difference between these two readings, the two numbers were averaged.

In the FT group mouth rinse compliance was evaluated by questioning the participants and by measuring remaining rinse in the bottles returned at the follow-up appointments.

Analytical and Statistical Methods

Data from baseline, six month, and eight month follow-up evaluations were summarized for each oral health variable by calculating means and standard deviations. Significant differences in oral health indices between the FT, enhanced oral treatment regimen, and the control regimen over time were calculated by using the Student's t-test analysis for continuous data (PD, CAL) and the Mann-Whitney test for non-parametric data (PI, CI, GI, and HbA1c). The main point of interest, the influence of dental treatment on the metabolic indicator, was analyzed using a mixed between-within subjects analysis of variance (ANOVA) to test whether there were effects due to treatment group, time of examination, or interaction of these two independent variables.

Using the upper limit of the average HbA1c level in type 2 diabetes in the United States (9.0) as a defining value, subjects in each group were divided into those who had baseline levels above 9.0 from those below 9.0. This data was analyzed for improvement in HbA1c at six and eight months. Those subjects undergoing physician-advised diabetic medication changes during the study period were grouped separately. Obviously improvements in HbA1c cannot be attributed solely to the periodontal intervention in these subjects.

Results

The study enrolled 50 subjects who met the inclusion/exclusion criteria at baseline and randomized 25 to the control treatment group and 25 to the FT group. Two subjects passed-away for reasons unrelated to the study. Six subjects were withdrawn from the study due to violations in the inclusion/exclusion criteria, such as smoking. Of the 42 remaining subjects who completed the study, 15 had their diabetes medications changed at the advice of their physicians. There were 27 subjects who did not have diabetic medication changes. No subjects needed to be omitted from

the analysis because of significant changes in diet, weight, or exercise habits.

Figure 1 and Table 3 demonstrate the changes that were observed in the oral health variables. With the exception of CAL, all oral health measurements demonstrated significant ($p<.05$) improvement in both the MT and FT subjects at six months. FT subjects showed greater improvements in PI at eight months (FT 81% v. MT 74% less plaque), PD (FT 17% v. MT 16% shallower probing depths), and GI (FT 63% v. MT 57% less gingival inflammation). MT subjects demonstrated more improvement in CI (FT 67% v. MT 79% less calculus).

Tables 4 and 5 and Figures 2 and 3 show the changes observed in HbA1c over the course of the study. Because the groups were so small, we can assert no statistically significant main effects for either treatment group or time of examination.

Although the most dramatic HbA1c reductions (Table 5) of 1.38 (FT) and 1.10 (MT) were observed in subjects with HbA1c > 9, it must be assumed the medication change influenced these results. In the HbA1c > 9 subjects with no medication change about half as much improvement in HbA1c was seen at six months. Mean reductions of 0.58 (FT) and 0.64 (MT) were encouraging but do not indicate "more periodontal therapy is better" as had been expected.

Discussion

As expected, the periodontal health of all subjects improved; the FT subjects demonstrated greater improvements in PD, PI, and GI compared to

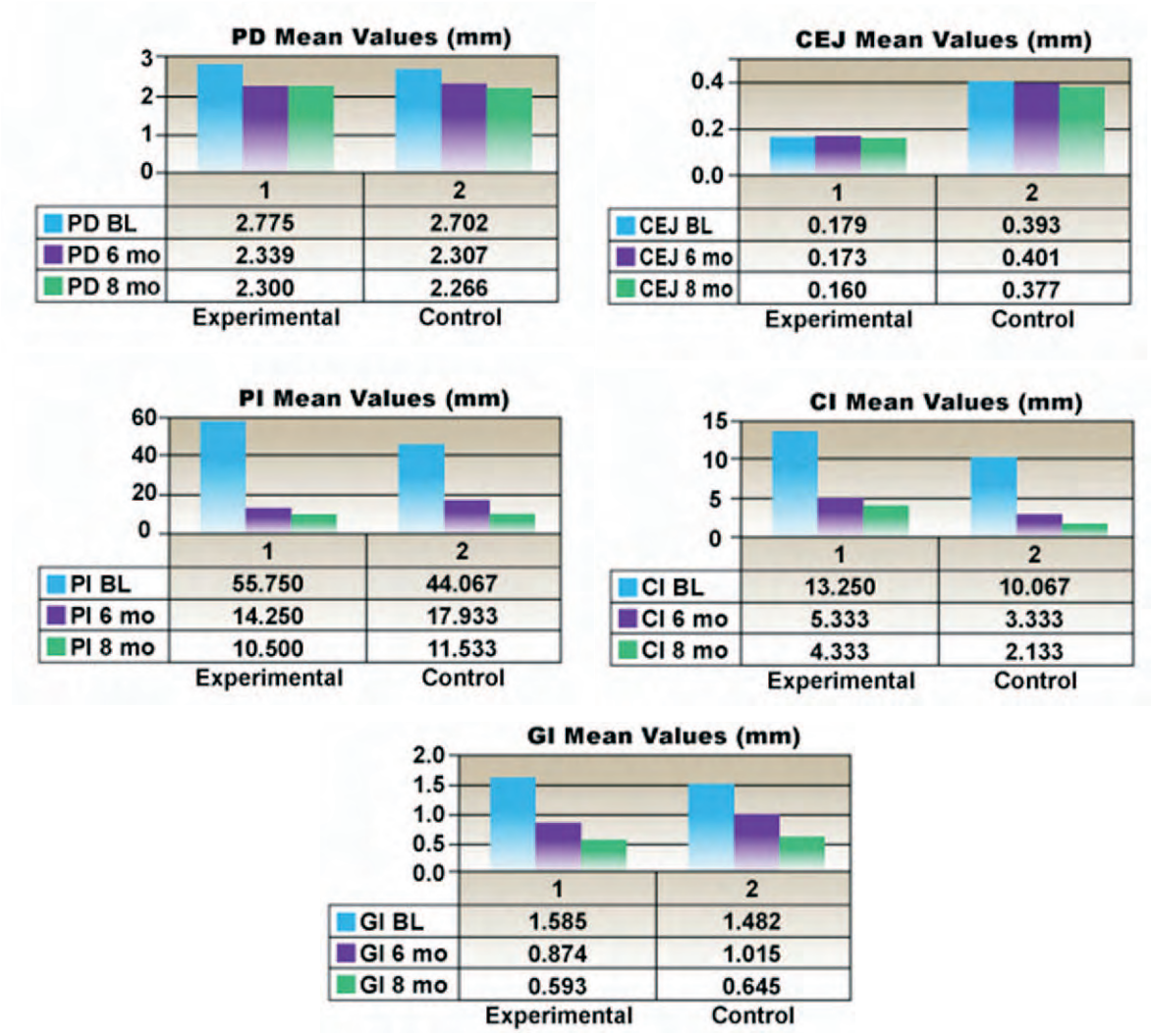

Figure 1. Observed changes in oral health variables.

Table 3. Oral health variables.

		Probing Depth (mm)	Loss of Attachment (mm)	Plaque Index (%)	Calculus Index (%)	Gingival Index (%)
Frequent Therapy n=12	Baseline	2.78 (0.17)	0.18 (0.19)	55.75 (23.02)	13.25 (7.80)	1.58 (0.22)
	6 month	2.34 (0.19)	0.17 (0.22)	14.25 (6.11)	5.33 (4.96)	0.87 (0.23)
	8month	2.30 (0.20)	0.16 (0.19)	10.5 (5.69)	4.33 (5.37)	0.59 (0.21)
	Overall Change	⬇0.48mm 17.3%	⬇0.02mm 11.1%	⬇45.25 81.2%	⬇8.92 63.7%	⬇0.99 62.7%
Control n=15	Baseline	2.70 (0.46)	0.39 (0.77)	44.07 (19.07)	10.07 (8.07)	1.48 (0.22)
	6 month	2.31 (0.26)	0.40 (0.74)	17.93 (10.29)	3.33 (3.63)	1.02 (0.29)
	8 month	2.27 (0.22)	0.38 (0.68)	11.53 (7.10)	2.13 (2.85)	0.64 (0.27)
	Overall Change	⬇0.43 15.9%	⬇0.01 2.5%	⬇32.54% 73.9%	⬇7.94% 78.8%	⬇0.84% 56.7%

Brackets indicate significant difference between group means ($p<.05$) of oral health variables. Standard deviations given in parentheses.

MT subjects, and no changes in CAL occurred because very few (n=3) baseline pocket depths were greater than 3 mm. Increased levels of calculus in the FT groups is certainly explained by their use of chlorhexidine.[35]

Diabetic subjects entering the study with HbA1c levels above 9.0 experienced a trend toward greater HbA1c reductions than those with levels below 9.0 at baseline, regardless of the study condition to which they were exposed. These subjects experienced 0.6 reduction in HbA1c, which is similar to other periodontal interventions of this nature.[38-40] However, because of the small samples sizes, the differences between groups did NOT reach statistical significance. At the eighth month follow-up, mean HbA1c in all groups gravitated toward return to baseline levels as has been seen in other periodontal and diabetes studies.[30,41,42]

Sample size presented the major flaw in this study and was exacerbated by the need to accommodate statistically for 15 subjects (nine in the FT and six in the MT groups) undergoing changes in their diabetes medications. In addition, as this study was being completed, results of a systematic review of like studies indicate much larger samples sizes are needed to determine what intensity and duration of periodontal therapy is needed to answer the question, particularly when the severity of periodontitis is mild.[31] Other modest limitations of this study include failure to have a non-treatment control group, failure to provide placebo rinse to the MT group, measuring GI and CAL only on the six Ramfjord

Table 4. Effect of periodontal treatment on HbA1c.
(Subjects with no medication change.)

Group	Time points			
	n	Baseline	6 Month (Final Treatment)	8 Months
Frequent Therapy Severe HbA1c (>9%) elevation	6	10.30	9.72 (⬇0.58)	10.32 (⬇0.02)
Frequent Therapy Mild HbA1c (<9%) elevation	6	8.00	8.20 (⬇0.20)	8.08 (⬇0.08)
Minimal Therapy Severe HbA1c (>9%) elevation	5	10.00	9.36 (⬇0.64)	9.84 (⬇0.16)
Minimal Therapy Mild HbA1c (>9%) elevation	10	8.07	8.30 (⬇0.23)	7.95 (⬇0.12)

Subjects with no medication change during trial. Largest decrease in hemoglobin levels occurred in groups whose baseline value was >9.

Table 5. Effect of periodontal treatment in subjects requiring medication change.

Group	Time Points			
	n	Baseline	6 Month (Final Treatment)	8 Month
Frequent Therapy Severe HbA1c (>9%) elevation	5	11.10	9.72 (⬇1.38)	10.45 (⬇0.65)
Frequent Therapy Mild HbA1c (<9%) elevation	4	7.78	7.95 (⬇0.17)	7.90 (⬇0.12)
Minimal Therapy Severe HbA1c (>9%) elevation	2	10.15	9.05 (⬇1.10)	8.50 (⬇1.65)
Minimal Therapy Mild HbA1c (>9%) elevation	4	7.65	7.15 (⬇0.50)	7.08 (⬇0.57)

Subjects excluded from study due to medication change during trial. Largest decrease in hemoglobin levels occurred in groups whose baseline value was >9.

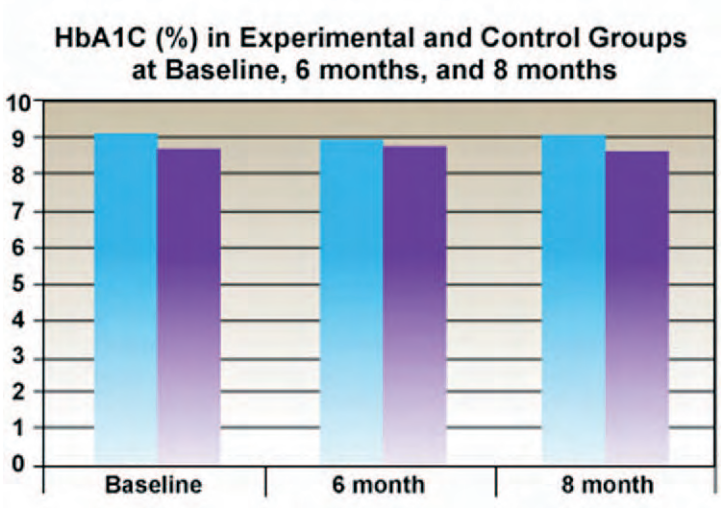

Figure 2. No significant difference was found between experimental and control HbA1C levels (p>.05) at baseline, six months, and eight months.

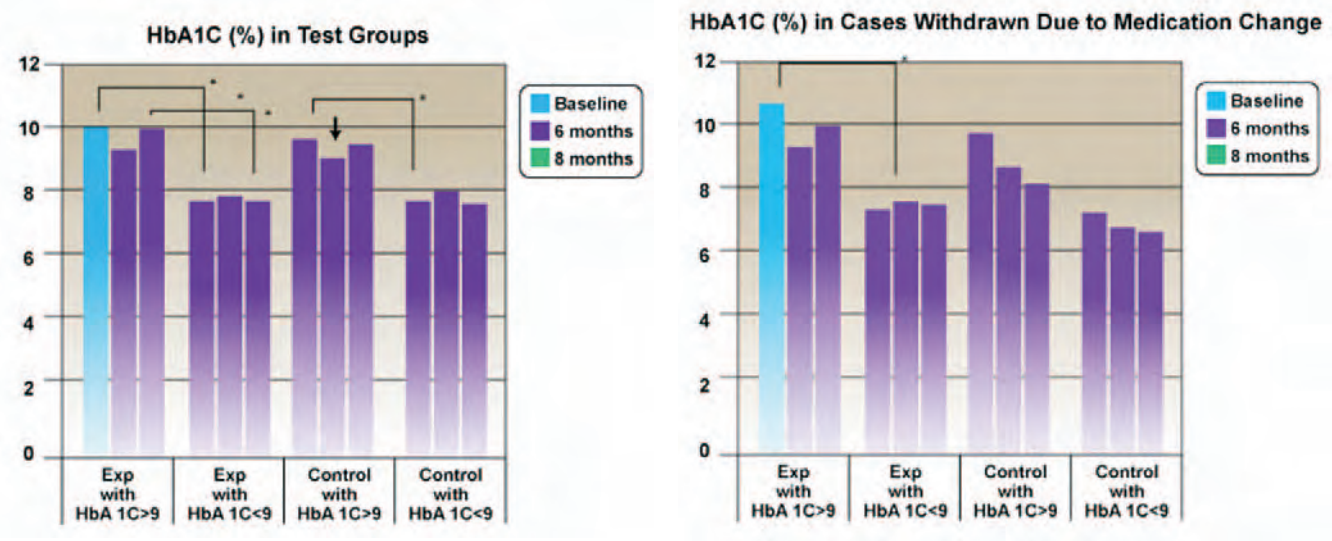

Figure 3. Comparison of changes in HbA1C over time for test subjects and those excluded because of medication change during trial period, grouped according to initial levels. There were no significant differences within each group over time but significant (p<.05) differences between subjects with <9% and >9% HbA1C entry levels are shown (*). The reduction in baseline to six months for the medication change groups >9% can be seen to be about twice that noted for the test groups >9% (downward arrows).

teeth[39] instead of the entire dentition, and inherent difficulties blinding an examiner when chlorhexidine stain is present. As in all university-based studies, the results may not be as generalizable to "real world patients" as would be the results from practice-based and community-based studies.

With the limitations noted, a reduction of 0.6 (or 6%) HbA1c should be considered clinically desirable in a given patient with mild periodontal inflammation. As little as a 1% decrease in HbA1c has been shown to reduce myocardial infarctions by 14%,[46] and a 1% elevation in HbA1c results in a 25% increase in complications.[45] Large scale (medical, non-dental) studies in the US and UK of intensive medical treatment regimens resulted in an average HbA1c reduction of 1.86 (or approximately 19%).[1-2,10,21] These reductions are comparable to periodontal intervention studies of

severe periodontitis patients which yield HbA1c reduction (up to 17.1%) when using antibiotics with special populations more severely affected by periodontitis and diabetes, such as Pima Indians and US Veterans.[23,30]

Clearly, the modest, short-term improvement in metabolic control achieved with a group of diabetic patients with fairly good oral health points to the need to perform a larger-scale, longer-term study with diabetic patients who have more serious inflammatory and infectious oral diseases. While it is likely practicing dentists and physicians are intuitively aware that alleviation of oral disease and the associated infection has a beneficial effect on metabolic control of diabetes, the magnitude of this effect and its long-term sequelae need additional documentation.

Conclusion

Overall, modest improvements in HbA1c were detected with a trend towards FT being better than MT. Although this pilot trial was under-powered to detect small between-group differences, the magnitude of our findings (0.6 mean improvement in HbA1c) matches closely findings from the only meta-analysis conducted on this topic to date. Larger scale studies must be undertaken on diabetic patients with periodontal problems.

Clinical Significance

Preventive periodontal regimens for diabetic patients should be sufficiently intense and sustained to eliminate periodontal inflammation and should be closely coordinated with the patient's overall clinical diabetic management.

References

1. Testa MA, Simonson DC. Health economic benefits and quality of life during improved glycemic control in patients with type 2 diabetes mellitus. JAMA. 1998; 280(17):1490-6.
2. UK Prospective Diabetes Study Group. Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). Lancet. 1998; 352(9131):837-53.
3. American Diabetes Association. Standards of Medical Care in Diabetes-2006. Diabetes Care. 2006; 29(Suppl 1):S4-S42.
4. The Diabetes Control and Complications Group. Retinopathy and nephropathy in patients with type 1 diabetes four years after a trial of intensive therapy. N Engl J Med. 2000; 342(6):381-9.
5. Reichard P, Nilsson BY, Rosenqvist U. The effect of long-term intensified insulin treatment on the development of microvascular complications of diabetes mellitus. N Engl J Med. 1993; 329:304-9.
6. Cohen MP. Non-enzymatic glycosylation: A central mechanism in diabetic microvasulopathy? J Diabet Complications. 1988; 2(4):214-7.
7. Fitzgibbons JF, Koler RD, Jones RT. Red cell age-related changes of hemoglobins A1a+b and A1c in normal and diabetic subjects. J Clin Invest. 1976; (58):820-4.
8. The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Report os the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Diab Care. 1998; 21(S1):s5-s19.
9. The Diabetes Control and Complications Group. Diabetes control and complications trial (DCCT): results of feasibility study. Diabetes Care. 1987; 10:1-19.
10. The Diabetes Control and Complications Trial Research Group. The effect of intensivetreatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. N Engl J Med. 1993; 329:977-86.
11. Tervonen T, Knuuttila M. Relation of diabetes control to periodontal pocketing and alveolar bone level. Oral Surg. 1986; 61:346-9.
12. Emrich LJ, Schlossman M, Genco RJ. Periodontal disease in non-insulin dependent diabetes mellitus. J Periodontol. 1991; 62:123-30.
13. Hugoson A, Thorstennson H, Falk J, Kuylenstierna J. Periodontal conditions in insulin dependent diabetes. J Clin Periodontol. 1989; 16:215-23.
14. Safkan-Seppala B, Ainamo J. Periodontal conditions in insulin dependent diabetes mellitus. J Clin Periodontol. 1992; 19:24-9.
15. Tervonen T, Oliver R. Long-term control of diabetes mellitus and periodontitis. J Clin Periodontol. 1993; 20:431-5.

16. Harrison R, Bowen WH. Periodontal health, dental caries, and metabolic control in insulin-dependent diabetic children and adolescents. Ped Dent. 1987; 9:283-6.
17. Gislen G, Nilsson KO, Matsson L. Gingival inflammation in diabetic children related to degree of metabolic control. Acta Odontologica Scand. 1980; 38:241-6.
18. Cohen DW, Friedman LA, Shapiro J, Kyle GC, Franklin S. Diabetes mellitus and periodontal disease: Two-year longitudinal observations, Part I. J Periodontol. 1970; 41:709-12.
19. National Institute for Dental Research. Oral health of United States Adults. The National Survey of Oral Health in U.S. Employed Adults and Senior: 1985-1986 National Findings. Bethesda, MD, U.S. Govt. Printing Office, 1987 (DHEW NIH publ. no. 87-2868).
20. Tervonen T, Karjalainen K, Knuuttila M, Huumonen S. Alveolar bone loss in type 1 diabetic subjects. J Clin Periodontol. 2000 Aug;27(8):567-1.
21. UK Prospective Diabetes Study Group. Effect of intensive blood-glucose control with metformin on complications in patients with type 2 diabetes (UKPDS 34). Lancet. 1998; 352(9131):654-65.
22. Finestone AJ, Boorujy SR. Diabetes mellitus and periodontal disease. Diabetes. 1967; 16:336-40.
23. Nelson RG, Shlossman M, Budding LM, Pettitt DJ, Saad MF, Genco RJ, Knowler WC. Periodontal disease and NIDDM in Pima Indians. Diabetes Care. 1990; 13(8):836-40.
24. Campbell D, Pollick HF, Lituri KM, Horowitz AM, Brown J, Janssen JA, Yoder K, Garcia RI, Deinard A, Hemphill S, de la Torre MA, Shrestha B, Vargas CM. Improving the oral health of Alaska natives. Am J Public Health. 2005; 95(5):769-73.
25. Jones JA, Miller DR, Wehler CJ, Rich S, Krall E, Christiansen CL, Rothendler JA, Garcia RI. Study design, recruitment, and baseline characteristics: the Department of Veterans Affairs Dental Diabetes Study. J Clin Periodontol. 2006 Oct 13;.[Epub ahead of print].
26. Ainamo J, Lahtinen A, Uitto VJ. Rapid periodontal destruction in adult humans with poorly controlled diabetes: a report of two cases. J Clin Periodontol. 1990; 17:22-8.
27. Shlossman M, Knowler WC, Pettitt D, Arevalo A, Genco RJ. Type II diabetes and periodontal disease (Abs). J Dent Res. 1987; 66:256.
28. Saremi A, Nelson RG, Tulloch-Reid M, Hanson RL, Sievers ML, Taylor GW, Shlossman M, Bennett PH, Genco R, Knowler WC. Periodontal disease and mortality in type 2 diabetes. Diabetes Care. 2005; 28(1):27-32.
29. Faria-Almeida R, Navarro A, Bascones A. Clinical and metabolic changes after conventional treatment of type 2 diabetic patients with chronic periodontitis. J Periodontol. 2006; 77(4):591-8.
30. Grossi SG, Skrepcinski FB, DeCaro T, Robertson DC, Ho AW, Dunford RG, Genco RJ. Treatment of periodontal disease in diabetics reduces glycated hemoglobin. J Periodontol. 1997; 68:713-9.
31. Janket SJ, Wightman A, Baird AE, Van Dyke TE, Jones JA. Does periodontal treatment improve glycemic control in diabetic patients? A meta-analysis of intervention studies. J Dent Res. 2005; 84(12):1154-19.
32. Lindhe J, Nyman S. Long-term maintenance of patients treated for advanced periodontal disease. J Clin Periodontol. 1984; 11:504-14.
33. Wennstrom J, Lindhe J. The effect of mouthrinses on parameters characterizing human periodontal disease. J Clin Periodontol. 1986; 13:86-93.
34. Schaeken MJ, Keltjens HM, Van der Hoeven JS. Effects of fluoride and chlorhexidine on the microflora of dental root surfaces and progression of root-surface caries. J Dent Res. 1991; 70:150-3.
35. Lang NP, Grec MC. Chlorhexidine digluconate - an agent for chemical plaque control and prevention of gingival inflammation. J Periodontal Res. 1986; suppl:74-89.
36. Loe H, Silness J. Periodontal disease in pregnancy. I - Prevalence and severity. Acta Odont Scand. 1963; 21:533-51.
37. Ramfjord, SP. Indices for prevalence and incidence of periodontal disease. J Peridontol. 1959; 30:51-9.
38. Iwamoto Y, Nishimura F, Nakagawa M, Sugimoto H, Shikata K, Makino H, Fukuda T, Tsuji T, Iwamoto M, Murayama Y. The effect of antimicrobial periodontal treatment on circulating tumor necrosis factor-alpha and glycated hemoglobin level in patients with type 2 diabetes. J Periodontol. 2001 Jun; 72(6):774-8.

39. Stewart JE, Wager KA, Friedlander AH, Zadeh HH. The effect of periodontal treatment on glycemic control in patients with type 2 diabetes mellitus. J Clin Periodontol. 2001 Apr; 28(4):306-10.
40. Seppala B, Seppala M, Ainamo J. A longitudinal study on insulin-dependent diabetes mellitus and periodontal disease. J Clin Periodontol 1993; 20:161-5.
41. Miller LS, Manwell MA, Newbold D, Reding ME. The relationship between reduction in periodontal inflammation and diabetes control: A report of 9 cases. J Periodontal. 1992; 63:843-8.
42. Sastrowijoto SH, van der Velden U, van Steenbergen TJM, Hillemans, P, Hart AAM, de Graaff J, Abraham-Inpijn L. Improved metabolic control, clinical periodontal status and subgingival microbiology in insulin-dependent diabetes mellitus: a prospective study. J Clin Periodontol. 1990; 17:233-242.
43. Beck JD, Caplan DJ, Preisser JS, Moss K. Reducing the bias of probing depth and attachment level estimates using random partial-mouth recording. Community Dent Oral Epi. 2006; 34(1):1-10.
44. Fisher M. Prevention of macrovascular complications. European Heart J Supplements. 2003; 5 (Suppl B):B21-B26.
45. Schellhase KG, Koepsell TD, Weiss NS. Glycemic control and the risk of multiple microvascular diabetic complications. Fam Med. 2005; 37(2):125-30.

About the Authors

Theresa E. Madden, DDS, MS, PhD, FACD

Dr. Madden is an Associate Professor of Public Health & Psychosocial Studies (Auckland University of Technology) and of Periodontics at Oregon Health and Science University School of Dentistry. Dr. Madden's research interests include periodontal-systemic disease interactions and global health. She serves on the executive committee of the Oregon Health and Science University Heart Research Center and is a consultant to the Virginia Garcia Memorial Health Centers. She is an avid sailor, bread-baker, and amateur vocalist.

e-mail: maddent@ohsu.edu

Brock Herriges, DMD

Dr. Herriges is a Periodontist in private practice in Portland, OR, USA. He received his Certificate in Periodontics at the Oregon Health and Science University School of Dentistry. He served in the US Navy at Norfolk, VA, USA.

Linda Boyd, RDH, MS, EdD

Linda Boyd is an Associate Professor and Director of the online Master of Science Degree in Dental Hygiene at Idaho State University (ISU). Dr. Boyd completed her MS degree in Nutrition Science and Policy from Tufts University, completed a Dietetic Internship at New England Medical Center in Boston, MA, USA and received her doctorate in Educational Leadership with a Specialization in postsecondary Adult and Continuing Education at Portland State University. Prior to entering education, she worked in general and periodontal practices for 20 years. She was an Assistant Professor at Oregon Health and Sciences University School of Dentistry for five years where she taught prevention and periodontics. She has been involved in clinical research addressing diabetes and complementary and alternative medicine (CAM) and periodontal disease. Linda served as Chair, Department of Dental Hygiene at Georgia Perimeter College for two years prior to arriving at ISU. Dr. Boyd has many publications and has presented continuing education courses on nutrition and oral health as well as critical thinking and adult learning topics.

e-mail: boydlind@isu.edu

Gayle Laughlin, DDS, PhD

Dr. Gayle Laughlin is an Assistant Professor of Restorative Dentistry at the Oregon Health & Science University in Portland, OR, USA.

e-mail: laughlig@ohsu.edu

Gary T. Chiodo, DMD, FACD

Dr. Chiodo is the Chief Integrity Officer of the Oregon Health & Science University in Portland, OR, USA. As such, he carries responsibility for all Integrity Programs at the University including corporate, research, environmental health and radiation safety, audit and advisory services, and information privacy and security. Dr. Chiodo is also a Professor of Community Dentistry and the Associate Director of the Center for Ethics in Health Care. During the first 21 years of his career, he practiced at the Russell Street Dental Clinic, which is a public health dental clinic in Portland, that treats the majority of identified HIV-positive persons in Oregon and southwest Washington. Dr. Chiodo received his Certificate in Health Care Ethics from the University of Washington School of Medicine in 1992. He has served on several state and national committees related to public health, ethics, and infectious diseases; has lectured internationally on these issues; and has published over 100 peer reviewed articles related to these topics. Dr. Chiodo is currently on the editorial board of "Medical Research Law and Policy" and chairs OHSU's Institutional Ethics Committee. Dr. Chiodo has been the principal investigator for an NIH R01 funded clinical AIDS study and an MRF funded diabetes study. In 1997 he received the Oregon Health Sciences University Distinguished Faculty Award in Recognition of Leadership.

e-mail: chiodoga@ohsu.edu

David I. Rosenstein, DMD, MPH

Dr. Rosenstein is a Professor Emeritus and former Chair of the Department of Public Health Dentistry at the Oregon Health Sciences University in Portland, OR, USA. He is the Director of the Russell Street Dental clinic that provides dental services to HIV seropositive dental patients.

Acknowledgement

The authors are indebted to the Medical Research Foundation of Oregon for a grant supporting this research.