Glycemic Control and Alveolar Bone Loss Progression in Type 2 Diabetes

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Abstract

This study tested the hypothesis that the risk for alveolar bone loss is greater, and bone loss progression more severe, for subjects with poorly controlled (PC) type 2 diabetes mellitus (type 2 DM) compared to those without type 2 DM or with better controlled (BC) type 2 DM. The PC group had glycosylated hemoglobin (HbA_1) ≥ 9%; the BC group had HbA_1 < 9%. Data from the longitudinal study of the oral health of residents of the Gila River Indian Community were analyzed. Of the 359 subjects, aged 15 to 57 with less than 25% radiographic bone loss at baseline, 338 did not have type 2 DM, 14 were BC, and 7 were PC. Panoramic radiographs were used to assess interproximal bone level. Bone scores (scale 0–4) corresponding to bone loss of 0%, 1% to 24%, 25% to 49%, 50% to 74%, or ≥ 75% were used to identify the worst bone score (WBS) in the dentition. Change in worst bone score at follow-up, the outcome, was specified on a 4-category ordinal scale as no change, or a 1-, 2-, 3-, or 4-category increase over baseline WBS (WBS1). Poorly controlled diabetes, age, calculus, time to follow-up examination, and WBS1 were statistically significant explanatory variables in ordinal logistic regression models. Poorly controlled type 2 DM was positively associated with greater risk for a change in bone score (compared to subjects without type 2 DM) when the covariates were included in the model. The cumulative odds ratio (COR) at each threshold of the ordered response was 11.4 (95% CI = 2.5, 53.3). When contrasted with subjects with BC type 2 DM, the COR for those in the PC group was 5.3 (95% CI = 0.8, 53.3). The COR for subjects with BC type 2 DM was 2.2 (95% CI = 0.7, 6.5), when contrasted to those without type 2 DM. These results suggest that poorer glycemic control leads to both an increased risk for alveolar bone loss and more severe progression over those without type 2 DM, and that there may be a gradient, with the risk for bone loss progression for those with better controlled type 2 DM intermediate to the other 2 groups. Ann Periodontol 1998;3:30–39.

Key Words: Alveolar bone loss; diabetes mellitus, non–insulin-dependent; hyperglycemia; Indians, North American; periodontal diseases/epidemiology.

INTRODUCTION

Diabetes mellitus is a heterogeneous group of disorders with different causes but all characterized by hyperglycemia, absolute or relative insulin deficiency or resistance to the action of insulin, and the tendency to develop certain long-term complications. The commonly recognized complications include accelerated atherosclerosis (macrovascular disease), diabetic retinopathy, diabetic nephropathy, and diabetic neuropathy.[1]

There are 4 major types of diabetes, with type 2 diabetes mellitus (formerly designated non–insulin-dependent diabetes mellitus) most prevalent. Approximately 90% to 95% of the estimated 7.8 million people in the United States diagnosed with diabetes have type 2 diabetes (type 2 DM),[2] and virtually all people with diabetes aged > 45 years have type 2 DM.[3] It is also estimated that for every diagnosed case of type 2 DM, there is approximately 1 undiagnosed case.[3] The next most common type is type 1 diabetes mellitus (type 1 DM, formerly designated as insulin-dependent diabetes mellitus), occurring in 5% to 10% of diagnosed cases of diabetes. Diabetes associated with certain systemic conditions or syndromes comprises approximately 2% of all diagnosed cases of diabetes. These conditions include pancreatic disease, disease of hormonal etiology, drug- or chemical-induced conditions, abnormalities of insulin or its receptors, certain genetic syndromes, and other miscellaneous conditions.[4] Approximately 2% to 5% of all pregnancies have associated gestational diabetes mellitus.[5]

The complications of diabetes, found in both type 2 DM and type 1 DM, result from structural and functional changes in susceptible tissues. These complications rarely occur in individuals without the metabolic abnormalities, hyperglycemia and insulin deficiency (relative or absolute), that characterize diabetes mellitus. There has been extensive research to characterize the mechanisms responsible for the pathogenesis of both the microvascular and macrovascular complications, and several mechanisms have been hypothesized. These mechanisms include altered myoinositol metabolism,[6] non-enzymatic glycosylation,[7] altered hemodynamics,[8] and genetic factors.[9-12] However, it is uncertain which of the hypothesized mechanisms, or combinations of mechanisms, is directly responsible in the target tissues for the pathogenesis of complications. It is also undetermined whether different mechanisms are operative in different tissues. It is known that not all people with diabetes experience these complications and that there is variability in the rate of development and severity of complications.

Strong evidence exists to support the notion that people with diabetes have increased risk for periodontitis. Indeed, periodontal disease has been recognized as another complication of diabetes.[13] Many studies have reported an association between poor glycemic control and increased occurrence of periodontitis,[14-29] although a number have also reported no association.[30-42] Most of these studies involve type 1 DM or do not specify the diabetes type; most are cross-sectional, thus unable to provide an estimate of glycemic control-related risk for poorer periodontal health; few use multivariate analysis; and none using sites or teeth as units of analysis accounts for extravariation (correlated observation) in the statistical analyses. The extensive variations in the design, conduct, and analyses of this set of studies contribute to inconsistencies in the findings. Consequently, no firm conclusions can be drawn from this collective body of literature. There are no previous reports that model risk for alveolar bone loss progression related to level of glycemic control of type 2 DM.

The purpose of this study was to test the hypothesis that the risk for alveolar bone loss is greater, and bone loss progression more severe, for subjects with poorly controlled type 2 DM compared to those without type 2 DM or with better controlled type 2 DM.

MATERIALS AND METHODS

Subjects for these analyses were 359 individuals, aged 15 to 57, who were part of a longitudinal study of type 2 DM and perio-

dontal disease in the Gila River Indian Community. Full details of this study have been presented elsewhere.[43-45] There were 338 subjects free of diabetes at baseline who did not develop type 2 DM during the follow-up period. The other 21 had type 2 DM (14 with better controlled DM and 7 with poorly controlled DM) at baseline. Type 2 DM was defined as having a plasma glucose concentration ≥ 200 mg/dL 2 hours after a 75 g oral glucose load following an overnight fast. Poorly controlled was defined as glycosylated hemoglobin (HbA_1) values ≥ 9% and better controlled as HbA_1 < 9%. Subjects selected were those who had 20 or more teeth, lost no teeth during the follow-up period, and had less than 25% radiographic bone loss at baseline. The median time to follow-up was 2.3 years; the minimum and maximum follow-up periods were 1.2 and 6.9 years, respectively.

The response variable for these analyses was change in radiographic bone score, as determined from panoramic radiographs. The baseline worst bone score (WBS1) for the mesial and distal of each tooth was assessed on a 0 to 4 ordinal scale, using a modified Schei ruler[45] with the score corresponding to percentage of root length not supported by interproximal bone. One value for each tooth, the greater of the mesial and distal measurements, was recorded. Bone scores corresponded to bone loss of 0%, 1% to 24%, 25% to 49%, 50% to 74%, or ≥ 75%. Change in bone score category (WBSch), the outcome of interest in this analysis, was computed as the greatest difference between baseline worst bone score (WBS1) and worst bone score at follow-up (WBS2). This outcome was specified as a 4-category ordinal scale representing no change, a 1-category increase, 2-category increase, or 3- or 4-category increase over WBS1.

Glycemic control status, the principal exposure in these analyses, was specified as 2 indicator variables, better control (HbA_1 < 9%) and poorer control (HbA_1 ≥ 9%). Additional dental, behavioral, medical, and demographic variables were evaluated at their baseline values for confounding and effect modification. Age, calculus index,[46] plaque index,[47] gingival index,[48] and time to follow-up examination were used in the models as continuous covariates. Indicator variables were defined for WBS1 (0% or 1% to 24%), self-reported alcohol consumption (≥ 3 drinks/day), smoking (any smoking in the year preceding baseline), obesity (body mass index > 27 kg/m^2), coronary artery disease, and gender.

Statistical analyses consisted of contingency table analysis and regression modeling. Contingency tables were used to assess relationships among variables for sparseness and patterns of possible confounding or effect modification. Regression modeling consisted of developing ordinal logistic regression models with cumulative logits,[49] using an ordinal specification for WBSch, to test the effects of other covariates on the WBSch–glycemic control association. Use of cumulative logits to analyze the ordinal response provided a way to analyze both the incidence of any change of periodontal status over time, as well as the severity of change, where change in periodontal status in subjects was ordinally defined at follow-up as no change, or a 1-, 2-, 3-, or 4-category increase. Parameter estimates obtained from this approach also allowed estimating the probability of making any change, as well as a 1-category leap, 2-category leap, and so on, over the follow-up period.

The regression models were developed in stages. First, using a forward stepwise selection procedure,‖ all the dental, behavioral, medical, and demographic variables considered to be important risk factors or confounders were included in a model, with the 2 indicator variables for glycemic control forced to remain in each model tested (the reference category included all subjects without type 2 DM). This initial selection step resulted in a preliminary candidate model. Next, each of the variables that had been previously eliminated in the stepwise selection procedure was separately retested in this candidate model and retained if it attained a level of significance with P value < 0.05. Following testing for main effects, first-order interaction terms were tested in the candidate model for relations between baseline age or glycemic

‖Proc Logistic, SAS Institute Inc., Cary, NC.

Table 1. Baseline descriptors*

Selected Characteristics	No Diabetes n = 338	Diabetes $HbA_1 < 9$ n = 14	Diabetes $HbA_1 \geq 9$ n = 7
Gender: Male	138 (40.8)	3 (21.4)	4 (57.1)
Female	200 (59.2)	11 (78.6)	3 (42.9)
Age (median years)	21	27	26
Age categories			
15–19	114 (33.7)	1 (7.1)	1 (14.3)
20–34	194 (57.4)	10 (71.4)	5 (71.4)
35–49	30 (8.9)	3 (21.4)	1 (14.3)
Smoker (current)			
No	220 (65.5)	10 (71.4)	2 (28.6)
Yes	116 (34.5)	4 (28.6)	5 (71.4)
Number of teeth (median)	28	28	27
Worst bone score			
0% bone loss	189 (55.9)	4 (28.6)	3 (42.9)
1–24% bone loss	149 (44.3)	10 (71.4)	4 (57.1)
Calculus index (median)	.28	.31	.39
Gingival index (median)	1.61	1.67	1.39
Plaque index (median)	1.17	1.28	1.22

*Percentage of subjects shown in parentheses.

control and all the main effects variables and for relations between baseline periodontal status and selected covariates. Quadratic terms were tested for all continuous covariates. Using likelihood ratio tests for the ordinal logistic regression models, terms found not to be statistically significant at the 0.05 level or important as effect modifiers or confounders in the modeled relationships were eliminated. Parameter estimate interpretation incorporated the point estimate as well as the 95% confidence interval in testing the ability to reject the null hypothesis. After obtaining a parsimonious model, the model fit was evaluated by using the likelihood ratio chi-squared test statistic, score test for the proportional odds assumption, and residual analysis.

RESULTS

Table 1 presents selected descriptive baseline statistics of the subjects without type 2 DM contrasted with 21 subjects with type 2 DM classified by level of glycemic control (i.e., better controlled and poorly controlled). Among subjects with better control, a notably higher proportion was female, while among those with poorer control, a somewhat higher proportion was male. The median ages for subjects with better control and for those with poorer control were similar, although both groups were slightly older than the group without type 2 DM. The proportion of current smokers was slightly higher in the group without type 2 DM compared to the group with type 2 DM who were better controlled, but there was a notably higher proportion of current smokers in the group with poorly controlled type 2 DM compared to both of the other 2 groups. (Data on smoking status were missing for 2 subjects in the "No Diabetes" group.) The median number of teeth was equivalent for the 3 groups, and the calculus and plaque indices were similar. The gingival index was similar for those without type 2 DM and better controlled groups but slightly lower in the poorly controlled group.

Table 2 shows the bivariate relationships between degree of WBSch, glycemic control status, and other selected baseline characteristics. There was a notable trend for the proportion of subjects experiencing no WBSch to diminish as glycemic control status worsened, with 59.5% of those without type 2 DM, 42.9% of the better controlled, and only 1 (14.3%) of the poorly controlled subjects having no WBSch at follow-up. Although the number of subjects with type 2 DM was small in the last 2 categories of

Table 2. Change in worst bone score by selected baseline characteristics (percentage of subjects in each row of each variable is shown in parentheses for the categories of change in worst bone score)

Baseline Characteristics	Degree of Change in Worst Bone Score at Follow-Up				
	None	1	2	3–4	N
Diabetes					
No diabetes	201 (59.5)	126 (37.3)	7 (2.1)	4 (1.2)	338
$HbA_1 < 9\%$	6 (42.9)	6 (42.9)	2 (14.3)	0	14
$HbA_1 \geq 9\%$	1 (14.3)	4 (57.1)	1 (14.3)	1 (14.3)	7
Gender					
Male	75 (51.7)	64 (44.1)	3 (2.1)	3 (2.1)	145
Female	133 (62.1)	72 (33.6)	7 (3.3)	2 (0.9)	214
Age					
15–19	83 (71.5)	31 (26.7)	2 (1.7)	0	116
20–34	114 (54.5)	87 (41.6)	5 (2.4)	3 (1.4)	209
35–57	11 (32.3)	18 (52.9)	3 (8.8)	2 (5.9)	34
Smoker					
No	134 (57.8)	89 (38.4)	7 (3.0)	2 (0.9)	232
Yes	72 (57.6)	47 (37.6)	3 (2.4)	3 (2.4)	125
Worst bone score					
0%	101 (51.5)	84 (42.9)	8 (4.1)	3 (1.5)	196
1–24%	107 (65.6)	52 (31.9)	2 (1.2)	2 (1.2)	163
Calculus index					
Lower 50th^{pctile}	117 (62.6)	63 (33.7)	4 (2.1)	3 (1.6)	187
Upper 50th^{pctile}	91 (52.9)	73 (42.4)	6 (3.5)	2 (1.2)	172

Table 3. Cumulative logistic regression model* (No Diabetes is the referent group)

Covariate	Beta	Standard Error	*P* Value	Odds Ratio (95% CI)
Main Effects				
Intercept 1	−3.385	.513	.0001	NA
Intercept 2	−6.699	.647	.0001	NA
Intercept 3	−7.886	.753	.0001	NA
$HbA_1 < 9\%$	0.769	.567	.1750	2.2 (0.7, 6.5)
$HbA_1 \geq 9\%$	2.438	.785	.0019	11.4 (2.5, 53.3)
No diabetes	NA	NA	NA	1.0 (ref. group)
Age	0.104	.018	.0001	1.1 (1.1, 1.2)
Time to follow-up	0.306	.106	.0038	1.4 (1.1, 1.7)
WBS1	−1.749	.292	.0001	0.2 (0.1, 0.3)
Calculus index	1.087	.281	.0001	3.0 (1.7, 5.1)

*Model evaluation statistics: likelihood ratio chi-squared statistic = 79.4 (6 degrees of freedom), $P = .0001$; score test for the proportional odds assumption = 14.3 (12 degrees of freedom), $P = .2841$.

WBSch, there was a tendency for the subjects with poorer control to have a higher proportion with more severe bone loss (i.e., a 1-, 2-, 3-, or 4-category change). The 3 age groups in Table 2 reflected a pattern of increasing severity of WBSch as age increased. The pattern of bone loss for smokers and nonsmokers was equivalent, except possibly the most severe category. Subjects with some evidence of radiographic bone loss at baseline (WBS1 of 1% to 24%), also shown in Table 2, tended to have less severe WBSch at follow-up (i.e., higher proportion with no change at follow-up and lower proportions in each of the other WBSch categories). Finally, subjects with a calculus index score greater than the median generally exhibited greater progression of bone loss.

Table 3 shows the results of the final ordinal logistic regression model used to test the hypothesis that the risk for alveolar bone loss is greater, and bone loss progression more severe, for subjects with poorly controlled type 2 DM than for those without type 2 DM (the referent group in this model). The coefficient estimates, *P* values, odds ratios

(with 95% confidence limits), and model evaluation statistics are shown. This model suggests that subjects with better controlled type 2 DM may not have a statistically significantly greater risk for alveolar bone loss progression than subjects without type 2 DM, as evidenced by the *P* value of 0.1750 for the coefficient for better control term ($HbA_1 < 9$); the odds ratio of 2.2 includes 1 in its 95% confidence interval (0.7, 6.5). The model estimates that subjects with poorly controlled type 2 DM ($HbA_1 \geq 9\%$) have a significantly higher risk for alveolar bone loss, as well as more severe bone loss progression at follow-up, than subjects without type 2 DM; the odds ratio is 11.4 (95% CI = 2.5, 53.3). This final model also estimated significant effects for time to follow-up and baseline values for age, calculus, and WBS. There was no significant risk associated with alcohol consumption, smoking, obesity, systolic blood pressure, coronary artery disease, gender, or number of teeth in the final model.

Table 4 shows the results of the final ordinal logistic regression model used to test the hypothesis that the risk for alveolar bone loss over 2 years is greater, and bone loss progression more severe, for subjects with poorly controlled type 2 DM than for those with better controlled type 2 DM. To test this hypothesis, the model is specified with indicator variables for subjects with poorly controlled type 2 DM ($HbA_1 \geq 9\%$) and for those without type 2 DM. In this table, the referent group consists of those subjects with better controlled type 2 DM ($HbA_1 < 9\%$). This model shows that subjects with poorly controlled type 2 DM may not have a statistically significantly greater risk for alveolar bone loss progression than subjects with better control, as evidenced by the *P* value of 0.0751 for the coefficient for the poorer control term ($HbA_1 \geq 9\%$); the odds ratio of 5.3 includes 1 in its 95% confidence interval (0.8, 33.4). This interpretation is made with caution because the size of the odds ratio and wide confidence interval having the lower bound at 0.8 suggest that there may indeed be a difference between those with poorer control and those with better control, although not as strong as the contrast between those with poorer control and those without type 2 DM. As expected, the remainder of the parameters estimated by this model mirrors those shown in Table 3.

Figure 1 contrasts the probabilities of change in bone score category estimated by the ordinal logistic regression model (using the parameter estimates of Table 3). It shows that the probability of a 1-, 2-, 3-, or 4-category change in radiographic worst bone score was greatest for subjects with poorly controlled type 2 DM (e.g., subjects with poorly controlled type 2 DM had a probability of 0.29 for a 2-category change, while the probability for those with better control was 0.16, and for those without diabetes 0.09). The figure also shows a gradient in the effect of type 2 DM on the probability of bone loss progression, with risk increasing as glycemic control worsens.

DISCUSSION

The results from these analyses provide strong evidence to support the hypothesis that the risk for alveolar bone loss over 2 years is greater, and bone loss progression more severe, for subjects with poorly controlled type 2 DM than those without type 2 DM. The results provide somewhat more equivocal evidence in testing the hypothesis that the risk for alveolar bone loss and its severity is greater for those with poorer control than those with better control. The effect parameter for those with poorly controlled type 2 DM ($HbA_1 \geq 9\%$), estimated in the model with better control ($HbA_1 < 9\%$) as the referent group in Table 4, is not statistically significant when a *P* value < 0.05 is used as the cut-off. However, the size of the effect (odds ratio = 5.3), width of the confidence interval (.8, 33.4), and nearness of the lower boundary of the 95% confidence interval to 1, as shown in Table 4, suggest that those with poorer control may have greater risk than those with better control. The absence of statistical significance in this case may be due to the small numbers of subjects with better control and poorer control, leading to reduced power to detect significant differences in these analyses. The same may hold for the absence of statistical significance in contrasting the risk for those with better control with

Table 4. Cumulative logistic regression model* (HbA_1 < 9% is the referent group)

Covariate	Beta	Standard Error	*P* Value	Odds Ratio (95% CI)
Main Effects				
Intercept 1	−7.118	.936	.0001	NA
Intercept 2	−5.931	.856	.0001	NA
Intercept 3	−2.616	.777	.0008	NA
HbA_1 < 9%	NA	NA	NA	1.0 (ref. group)
HbA_1 ≥ 9%	1.669	.938	.0751	5.3 (0.8, 33.4)
No diabetes	−0.769	.567	.1750	0.5 (0.1, 1.4)
Age	0.104	.018	.0001	1.1 (1.1, 1.2)
Time to follow-up	0.306	.106	.0038	1.4 (1.1, 1.7)
WBS1	−1.749	.292	.0001	0.2 (0.1, 0.3)
Calculus index	1.087	.281	.0001	3.0 (1.7, 5.1)

*Model evaluation statistics: likelihood ratio chi-squared = 79.4 (6 DF), *P* = .0001; score test for the proportional odds assumption = 14.3 (12 DF), *P* = .2841.

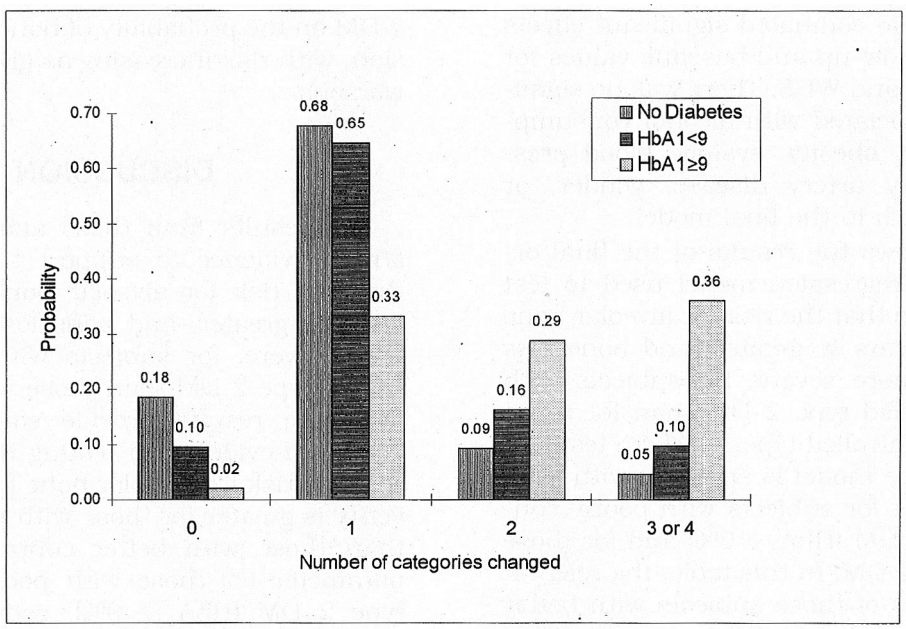

Figure 1. Probabilities of change in worst bone score category.

those who do not have type 2 DM. Given the small sample sizes in the poorly controlled and better controlled groups, the size and statistical significance of the effect estimated for the poorly controlled group, and the presence in the models of other covariates known to be associated with progression of periodontal diseases, the evidence is strengthened for the contrast in risk for those with poorly controlled type 2 DM over those without type 2 DM. Further, the results from these analyses suggest that there may be a gradient in risk for alveolar bone loss and severity of its progression, as illustrated in Figure 1, with the risk for poorer control > risk for better control > risk for those without type 2 DM.

In general, the direction of the effects for the other covariates in the logistic regression models is as expected, except for WBS1. The models suggest that there may be an inverse relationship between WBS1 and WBSch at follow-up; i.e., there was a higher risk of WBSch in subjects with no measurable radiographic bone loss (maximum WBS = 0%) than in subjects with maximum WBS of 1% to 24% at baseline. Papapanou and Wennstrom[50] also found an inverse relationship between baseline radiographic bone level and

amount of bone loss at follow-up. However, Albandar[51] reported a direct relationship between degree of bone loss at baseline and at follow-up. Consideration of the unit of analysis may provide resolution of these seemingly divergent findings. As was the case with Papapanou and Wennstrom,[50] the individual in the present study is the analytic unit rather than the tooth or site, as was the case in Albandar.[51]

The stringent subject selection criteria employed in these analyses resulted in a considerable reduction in the number of subjects with type 2 DM and potentially weakened statistical power to detect significant differences between the groups. The motivation for choosing subjects who had 20 or more teeth at baseline, lost no teeth during the follow-up period, had WBS1 ≤ 24%, and whose diabetes status did not change during the follow-up period was to reduce the extraneous effects of other factors as much as possible. These strict selection criteria could have led to a selection bias. However, the selection bias in these analyses, if present, would be towards selecting the "healthiest" of the type 2 DM subjects, and hence would bias results against rejecting the null hypothesis. Since the analyses resulted in a well-fitting model that permitted rejecting the null hypothesis for contrasting WBSch in the poorly controlled versus non-type 2 DM groups, the potential selection bias actually provides more confidence in the conclusions reached.

Prior to accepting the results from the models presented, several tests were performed to assess the effects of the stringent selection criteria. Separate data sets with larger numbers were created to test the consistency of the effect of poorer control where WBS1 was ≤ 2 (0% to 49%) instead of ≤ 1 (0% to 24%), where minimum number of teeth at baseline was 12 rather than 20, and where subjects with tooth loss were not excluded. The results (not shown) of models incorporating these relaxed inclusion criteria were not superior to those presented here. The supplemental model that included subjects who lost teeth during the follow-up period (but otherwise met the inclusion criteria) had 401 observations and estimated an improvement in the statistical significance for the effect of being in the better controlled group, with an odds ratio of 2.8 (95% CI = 0.99, 7.8; P value = 0.052). This result further supports our suggestion of a gradient of risk related to glycemic control. However, this supplemental ordinal logistic regression model did not fit quite as well (score test P value = 0.0678) as the final model presented. This worsening of the model fit, combined with the relaxed inclusion criteria, lessened our confidence in using this model as our final model. Hence, the results of the more conservative analyses are presented in the tables.

The principal exposure in these analyses was glycemic control status, formed by dichotomizing the continuous values for glycosylated hemoglobin (HbA_1). The cut point of 9% or more, used to distinguish those with poorer control from those with better control, was chosen because it is near the HbA_1 antimode in this population.[52] A potential limitation in these analyses is choosing a cut point for the HbA_1 measures. By dichotomizing baseline HbA_1, subjects with values slightly above or below the cut point would tend to be similar with respect to glycemic control status, although classified as having a different exposure in the analysis. This imposed dichotomous classification of better versus poorer control could weaken the power to detect an effect on alveolar bone loss severity by assigning subjects with similar baseline glycemic control characteristics to different exposure categories. To address this issue, we conducted a supplemental analysis by creating a "buffer zone" to identify and exclude subjects who had HbA_1 values from 8% to 9%. This supplemental analysis excluded 3 subjects, all in the group with better controlled type 2 DM. Even with the loss of 3 of the better controlled subjects, we observed minimal changes in the results of the logistic regression model that was otherwise identical to the model presented in Table 4. Hence, it is not likely that misclassification of glycemic control status had a substantial role in these analyses.

These results support and extend other reports in the literature describing an association between poor glycemic control and periodontal disease. This study has evaluated the effects of glycemic control status on both

the risk for, as well as severity of, periodontal destruction over time. The temporal sequence specified in these longitudinal analyses provides evidence to support a cause-effect relationship. Subjects with poorer glycemic control had significantly greater risk for alveolar bone loss progression, and the progression was more severe than in subjects without type 2 DM. Additionally, these analyses suggest that there may be a gradient in risk for any alveolar bone loss, as well as severity of progression of alveolar bone loss, with poorly controlled > better controlled > no type 2 DM.

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