Periodontal Changes in Children and Adolescents With Diabetes

A case-control study

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OBJECTIVE — To evaluate the level of oral disease in children and adolescents with diabetes.

RESEARCH DESIGN AND METHODS — Dental caries and periodontal disease were clinically assessed in 182 children and adolescents (6–18 years of age) with diabetes and 160 nondiabetic control subjects.

RESULTS — There were no differences between case and control subjects with respect to dental caries. Children with diabetes had significantly higher plaque and gingival inflammation levels compared with control subjects. The number of teeth with evidence of attachment loss (the hallmark of periodontal disease) was significantly greater in children with diabetes (5.79 ± 5.34 vs. 1.53 ± 3.05 in control subjects, unadjusted $P < 0.001$). When controlling for age, sex, ethnicity, gingival bleeding, and frequency of dental visits, diabetes remained a highly significant correlate of periodontitis, especially in the 12- to 18-year-old subgroup. In the case group, BMI was significantly correlated with destruction of connective tissue attachment and bone, but duration of diabetes and mean HbA_{1c} were not.

CONCLUSIONS — Our findings suggest that periodontal destruction can start very early in life in diabetes and becomes more prominent as children become adolescents. Programs designed to promote periodontal disease prevention and treatment should be provided to young patients with diabetes.

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Periodontal diseases are bacterial infections of the tissues surrounding and supporting the teeth. Gingivitis, an inflammation of the soft tissues only, can progress to periodontitis, where destruction of connective tissue attachment and alveolar bone can eventually lead to tooth loss. The prevalence of severe periodontitis is 10–15% in most populations (1). In 1993, periodontal disease was identified as the sixth complication of diabetes (2), and in the 1997 report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus, periodontal disease was referred to as one of the pathologic conditions often found in adults with diabetes (3).

The association between diabetes and periodontal diseases has been recognized in the dental literature for many decades. Indeed, multiple studies have demonstrated that the prevalence, severity, and progression of periodontal disease are significantly increased in patients with diabetes (4). Furthermore, longitudinal studies have shown that severe periodontal disease in diabetic patients at baseline is associated with poor metabolic control and other diabetes complications at follow-up (5,6). A number of reports on the relationship between diabetes and periodontal disease have included children and adolescents; however, these studies are limited with respect to the depth of data collected and analyses presented. Manouchehr-Pour and Bissada (7) reviewed periodontal conditions in individuals with diabetes and reported that in patients with childhood-onset diabetes, periodontitis seems to ensue around puberty and to progress with age. The goal of the present study was to define the oral disease burden in young children and adolescents with diabetes.

RESEARCH DESIGN AND METHODS

— The study protocol was approved by the Columbia University Medical Center Institutional Review Board. Parents/legal guardians of participants signed a consent form.

One hundred eighty-two patients with diabetes, 6–18 years of age, were recruited from among the patients followed at the Naomi Berrie Diabetes Center at the Columbia University Medical Center. One hundred and sixty 6–18 year old subjects seen at the pediatric dental clinic at the Columbia University School of Dental and Oral Surgery who denied history of diabetes served as control subjects. Children in both groups were excluded if they were undergoing active orthodontic therapy.

Oral examination protocol

Participants and/or their guardians responded to questions concerning the participants' dental history. All assessments were performed by one of two calibrated examiners (S.L. and S.T.). The oral examination included the following: *1)* Dental examination: recording of missing teeth, existing carious lesions, and dental restorations, based on clinical observation using a dental mirror and explorer. 2) Periodontal examination of one randomly assigned maxillary and the diagonally opposite mandibular quadrant. For primary, erupting, and fully erupted permanent teeth (third molars excluded), the following were recorded for four sites per tooth (mesiobuccal, distobuccal, me-

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A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

siolingual, and distolingual) using a manual periodontal probe: *A*) Plaque index: each site was given a score from 0 to 3, as described by Silness and Löe (8). *B*) Gingival index: each site was given a score from 0 to 3, according to Löe and Silness (9). In this index, a gingival index score of 2 or 3 denotes a bleeding site. For fully erupted permanent teeth, two additional parameters were evaluated at four sites per tooth, using a periodontal probe: *C*) Probing depth, defined as the distance between the gingival margin and the bottom of the probeable pocket to the nearest whole millimeter. *D*) Location of the gingival margin, the distance between the cementoenamel junction and the gingival margin to the nearest whole millimeter. The distance was deemed nonreadable whenever the cementoenamel junction was obscured by dental restorations or was impossible to identify. The two parameters above were used to compute clinical attachment level. Any loss of attachment represents destruction of periodontal support around teeth.

Table 1—*Demographic, dental, and periodontal characteristics of study population

	Control subjects	Case subjects	*P* value
Age (years)	10.9 ± 2.6	11.9 ± 3.3	0.001
Sex (female)	80 (50)	83 (46)	0.42
Ethnicity			
Hispanic	134 (84)	75 (41)	0.001
Non-Hispanic	26 (16)	101 (56)	
Medical insurance, with coverage	150 (94)	172 (95)	0.77
Reported frequency of dental visits (per year)	1.5 ± 1.4	1.5 ± 0.9	0.65
Reported age at first dental visit (years)	5.0 ± 2.4	4.3 ± 2.3	0.006
Reported ever had red/inflamed gums	21 (13)	29 (17)	0.37
Reported ever had bleeding gums	43 (27)	65 (38)	0.04
Number of carious and restored surfaces	3.4 ± 4.5	3.2 ± 4.8	0.60
Number of carious and restored teeth	2.5 ± 2.7	2.4 ± 3.1	0.62
Plaque index	1.1 ± 0.3	1.2 ± 0.4	<0.001
Percent of sites with plaque	18.2 ± 16.4	28.1 ± 24.1	<0.001
Gingival index	1.0 ± 0.3	1.2 ± 0.3	<0.001
Percent of bleeding sites	10.2 ± 13.6	23.6 ± 23.9	<0.001
Mean attachment loss (mm)	0.8 ± 0.9	1.8 ± 1.1	<0.001
Number of affected teeth†	1.5 ± 3.1	5.8 ± 5.3	<0.001

Data are mean ± SD or *n* (%). *Study population included 160 control and 182 case subjects. †At least one site with >2 mm of attachment loss.

Diabetes-related variables

The following information related to the cases' diabetic state was collected from medical records: *1*) Type of diabetes, duration (years since diagnosis), and age at diagnosis. 2) Height and body weight, for calculation of BMI. In addition, BMI-for-age and percentile ranks, signifying nutritional status (10), were calculated based on Centers for Disease Control and Prevention age- and sex-specific growth charts (11). *3*) Insulin regimen (multiple daily insulin injections or continuous subcutaneous insulin infusion), oral hypoglycemic medications, and any other medications. *4*) Laboratory data, including measurements of HbA_{1c} (A1C) and lipid profiles.

Data and statistical analysis

Analyses were performed using SAS, version 9.1 (SAS Institute, Cary, NC), and the R statistical software, version 2.0.0. The case-control analysis focused on the association between periodontitis and diabetes. First, we directly compared case and control subjects using unadjusted Student's *t* and χ^2 tests. Then, we performed formal analyses using logistic regression with presence of periodontitis as the dependent variable. As there is no universal definition of the extent/severity of attachment loss necessary for clinically significant periodontal destruction, periodontitis was defined as the presence of at least one site with attachment loss >2 mm on at least two teeth. This definition is appropriate, considering the age of our subjects and the fact that the periodontal examination was performed for half of the mouth. Periodontal destruction has been shown to occur in a symmetric pattern in the left and right side of the jaws (12), and the above definition of disease probably translates into four affected teeth for the whole mouth. Covariates included disease-related variables such as presence or absence of diabetes, age-subgroup (<12 and ≥12 years of age), proportion of sites with gingival bleeding (square root transformed to achieve a better fit), and adjusting variables such as sex, ethnicity, frequency of dental visits (log transformed to achieve a better fit), and dental examiner.

We did not include plaque index as one of the adjusting variables, as it is highly correlated with the proportion of sites with bleeding. To avoid multicollinearity, of the two we chose the latter because it is not only a measure of gingival inflammation and a precursor of periodontal destruction but also an index of oral hygiene (affected by the level of plaque control over a longer period of time rather than just the examination day).

The case-only analysis investigated the association between periodontitis and certain diabetes-related variables, using an adjusted linear regression model. The dependent variable was number of affected teeth (those having at least one site with >2 mm of attachment loss). Independent variables included: A1C (mean value of all tests performed in the 2 years before the dental examination, excluding any tests that were within 3 months of diagnosis of diabetes), BMI, duration of diabetes, age-subgroup, proportion of sites with gingival bleeding, and adjusting variables: sex, ethnicity, frequency of dental visits, and dental examiner. $P < 0.05$ (two sided) was considered statistically significant for all analyses.

RESULTS

Case-control analysis

Demographic, dental, and periodontal characteristics of the study population are presented in Table 1.

For each subject, we computed the number of carious (decayed) and restored (treated for decay) surfaces and teeth based on the full mouth examination. There were no significant differences between case and control subjects in regard to caries experience.

Children with diabetes had significantly more dental plaque than nondiabetic control children (plaque index 1.2 vs. 1.1, respectively; unadjusted $P < 0.001$). Similarly, when we calculated the percentage of sites that had visible plaque

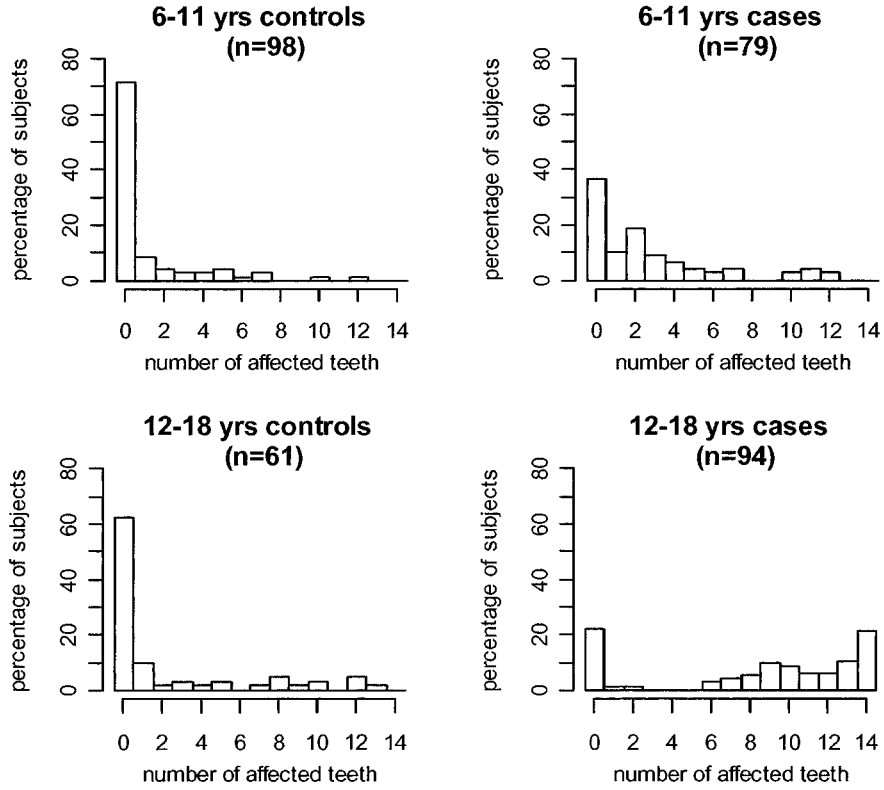

Figure 1—*Distribution of periodontal destruction in the study population. The number of affected teeth (those having at least one site with >2 mm of attachment loss) was significantly higher in diabetic children versus control subjects (χ^2 tests comparing presence of ≥2 vs. <2 affected teeth between cases and control subjects yielded* P *values <0.001 for both age-subgroups).*

(plaque index ≥2) there was again a significant difference (28.1 vs 18.2%, respectively). Children with diabetes also had significantly more gingival inflammation than control children (gingival index 1.2 vs. 1.0 and percentage of sites that bled upon examination: 23.6 vs. 10.2%, respectively). These differences in gingival inflammation remained significant after adjustment for its main determinant, dental plaque ($P < 0.001$, adjusted for plaque index).

Attachment loss, the hallmark of periodontitis, calculated as either a subject-based mean or as the number of affected teeth (having at least one site with >2 mm of attachment loss) was also significantly higher in diabetic children compared with nondiabetic control subjects (1.8 vs. 0.8 and 5.8 vs. 1.5, respectively).

As shown in Fig. 1, the number of affected teeth was significantly higher in diabetic children versus control children in both age-subgroups, <12 years of age or ≥12 year of age (χ^2 tests comparing presence of ≥2 vs. <2 affected teeth between case and control subjects yielded *P* values <0.001 for both the younger and older children).

Examination of the distribution of subjects with attachment loss in our study population revealed differences depending on the age-subgroup. This was supported by the logistic regression analysis (with presence of periodontitis as the dependent variable) adjusting for several relevant variables, which revealed diabetes and age as the two statistically significant correlates of periodontitis. Results for the whole population and by age-subgroup are summarized in Table 2. When we used different definitions of periodontal disease to allow for even more extensive periodontal destruction, the effect of diabetes remained significant for the older but not for the younger group of patients (data not shown).

Case-only analysis

Diabetes-related variables in our cases (for all subjects and by age-subgroup) are presented in Table 3. Ninety-four percent of all children had type 1 diabetes; 11% in the older subgroup had type 2 diabetes. Mean A1C over the 2 years before the examination was 8.4 ± 1.7% and similar in the two subgroups ($P = 0.09$). However, the percentage of children with poor metabolic control was significantly higher in the older group ($P = 0.01$). BMI-for-age percentiles were similar in the two groups, but actual BMI was significantly higher in the older group (25.0 ± 7.5 kg/m^2) compared with 19.2 ± 4.4 kg/m^2 in the younger group ($P < 0.001$). Further, a higher percentage of the older children had a poor nutritional status indicator, based on their BMI for age ($P = 0.04$).

Table 4 summarizes results from the linear regression analysis. The focus here was to investigate the relationship between periodontal destruction and diabetes-related variables. The association between number of affected teeth and mean A1C or duration of diabetes was not statistically significant. However, there was a positive and statistically significant association between the number of affected teeth and BMI (regression coefficient 0.12, $P = 0.03$). One of the adjusting variables, frequency of dental visits, also reached significance in this model (regression coefficient 1.01, $P = 0.046$).

Table 2—*Estimated odds ratios (and 95% CIs) from logistic regression models* for periodontitis†*

	Odds ratio (95% CI)	*P* value
All		
Diabetes	5.23 (2.41–11.35)	<0.001
12–18 years age-group	4.80 (2.17–10.63)	<0.001
Proportion of bleeding sites‡	1.96 (0.40–9.35)	0.40
6–11 years		
Diabetes	3.44 (1.31–9.02)	0.01
Proportion of bleeding sites‡	1.34 (0.17–10.31)	0.77
12–18 years		
Diabetes	20.29 (3.82–107.76)	<0.001
Proportion of bleeding sites‡	10.80 (0.43–270.17)	0.15

Analysis is for all subjects and by age-subgroup. *Regression models also adjusted for sex, ethnicity, frequency of dental visits, and dental examiner. †Defined as at least one site with attachment loss >2 mm on at least two teeth. ‡Square root transformation performed to achieve a better fit.

Table 3—*Diabetes-related variables for the case group**

	All	6–11 years	12–18 years
Type 1 diabetes	170 (94.4)	87 (100)	83 (89.3)
Duration (years)	4.5 ± 8.0	3.3 ± 2.5	5.6 ± 10.7
Age at diagnosis (years)	7.8 ± 4.0	5.7 ± 2.6	9.9 ± 3.9
Treated with insulin o:p	174 (96.1)	87 (100)	87 (92.6)
Multiple daily injections	130 (74.7)	67 (77.0)	63 (72.4)
Continuous subcutaneous infusion	44 (25.3)	20 (23.0)	24 (27.6)
Mean A1C over past 2 years (%)	8.4 ± 1.7	8.2 ± 1.4	8.6 ± 1.9
< 7.5%	55 (32.2)	26 (31.7)	29 (32.6)
7.5–9.5%	80 (46.8)	46 (56.1)	34 (38.2)
> 9.5%	36 (21.0)	10 (12.2)	26 (29.2)
Total cholesterol (mg/dl)†	171.1 ± 37.9	164.7 ± 31.7	176 ± 41.5
HDL cholesterol (mg/dl)	56.6 ± 15.6	56.9 ± 14.2	56.4 ± 16.8
LDL cholesterol (mg/dl)	94.7 ± 30.6	89.6 ± 24.5	98.6 ± 34.3
Triglycerides (mg/dl)	99.5 ± 65.1	91.1 ± 46.3	106.1 ± 76.2
BMI (kg/m^2)	22.2 ± 6.9	19.2 ± 4.4	25.0 ± 7.5
BMI for age (percentile)	74.4 ± 24.4	73.5 ± 24.1	75.2 ± 24.8
BMI-for-age–based nutritional status indicator			
At risk of overweight (85th-94th percentile)	38 (20.9)	22 (25.3)	16 (16.8)
Overweight (≥95th percentile)	46 (25.3)	15 (17.2)	31 (32.6)

Data are mean ± SD or *n* (%). **n* = 182; 87 age 6–11 years and 95 age 12–18 years. †Lipid data available for *n* = 123; 53 age 6–11 years and 70 age 12–18 years.

CONCLUSIONS — Previous studies in children with diabetes have indicated that gingival inflammation is significantly increased compared with nondiabetic control subjects, even after adjusting for oral hygiene levels (13–15). Findings from the present study indicate that periodontal destruction is increased in children and adolescents with diabetes and, importantly, that this starts earlier in life than formerly recognized (7).

Our nondiabetic control subjects represent an underserved, mostly Hispanic population, and recent studies in this population have reported that the oral disease burden is high compared with national standards (16,17). In this study, evidence of periodontal destruction was indeed present in the control group. However, presence of diabetes clearly conferred a significant risk. Regression analysis revealed that diabetes was a statistically significant correlate of periodontitis, even in the 6- to 11-year-old group. This relationship became more pronounced after the age of 12.

The etiopathogenesis of both diabetes and periodontitis is complex, and identifying the mechanisms underlying this association was beyond the scope of this study. However, evidence suggests that mechanisms that account for the development of other diabetes complications might be operating in the pathogenesis of accelerated periodontitis in diabetes as well (18).

Our finding in this study, that duration of diabetes, and especially mean A1C, were not significantly correlated with the number of affected teeth was intriguing. It appears to be in contrast to previous evidence suggesting that individuals with diabetes and poor metabolic control are at a higher risk for suffering from more severe periodontitis (4). However, not all studies have reported such an association, and there are conflicting data between prospective follow-up studies that have in their majority suggested the former and many cross-sectional studies like ours that have shown no correlation. Interestingly, in the case group, BMI displayed a modest but statistically significant correlation with the number of affected teeth. This finding, although preliminary, is consistent with the current understanding that obesity is associated with the development of a systemic inflammatory state and the recent reports on a significant correlation between periodontitis and BMI in adults (19,20). Further studies including larger numbers of children and adolescents are under way; these will shed more light into these associations, other factors involved, as well as the natural history of the development of periodontal changes in diabetes.

Dental caries, xerostomia, and mucosal lesions have been reported in patients with diabetes, but the data are conflicting (21,22). Most recent studies suggest that young people with diabetes exhibit similar levels of caries to systemically healthy individuals (23,24). In this study we found no differences in clinical caries experience between children with diabetes and nondiabetic control children, and the presence of other mucosal lesions was very rare in both groups.

It has been reported that patients with diabetes are significantly less likely than those without diabetes to have seen a dentist within the past year, even after adjusting for age, race/ethnicity, education, income, and dental insurance coverage (25). Interestingly, the primary reason for not seeing a dentist given in that study was lack of a perceived need. As periodontal diseases are largely preventable and progression of destruction can be best arrested when identified in early stages, screening for periodontal changes and implementing prevention and treatment programs should be considered as a standard of care for young patients with diabetes. This becomes even more important in the light of the emerging view that control of periodontal infections in adults with diabetes can further have a positive effect on the level of metabolic control in these individuals (26,27).

Table 4—*Estimated regression coefficients (and 95% CIs) from linear regression model* for number of affected teeth† among case subjects*

	Regression coefficient (95% CI)	*P* value
Mean A1C	0.12 (−0.25 to 0.49)	0.51
Duration of diabetes	−0.02 (−0.20 to 0.17)	0.87
Proportion of bleeding sites‡	1.72 (−0.92 to 4.36)	0.20
12–18 years age-group	5.17 (3.80–6.54)	<0.001
BMI	0.12 (0.02–0.23)	0.03

*Regression model also adjusted for sex, ethnicity, frequency of dental visits, and dental examiner. †Having at least one site with >2 mm of attachment loss. ‡Square root transformation performed to achieve a better fit.

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