

Oral Disease Burden in Northern Manhattan Patients With Diabetes Mellitus

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Periodontal diseases are bacterially induced chronic inflammatory diseases affecting the tissues surrounding and supporting the teeth. The lesion begins as gingivitis, an inflammation of the gingival tissues only, and may progress to periodontitis, where destruction of connective tissue attachment and alveolar bone can eventually lead to tooth loss. In 1993, periodontitis was referred to as the sixth complication of diabetes mellitus¹; in the 1997 report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus, it was cited as one of the pathological conditions often found in patients with diabetes.² Indeed, multiple studies have provided conclusive evidence that the prevalence, severity, and progression of periodontal disease are significantly increased in patients with diabetes.³ Other oral complications have been reported in patients with diabetes, such as caries, xerostomia, and mucosal lesions. However these associations are weaker, and conflicting data have been reported.^{4–8}

If left untreated, periodontitis can lead to tooth loss, thereby compromising a patient's ability to maintain a proper diet and affecting the quality of life. Furthermore, longitudinal studies have reported that severe periodontal disease in diabetic patients at baseline is associated with poor metabolic control and other diabetic complications at follow-up.^{9,10} There also has been a suggestion in the literature that mechanical periodontal therapy in conjunction with systemic antibiotics may result in improved metabolic control in some patients with diabetes, especially those with poor metabolic control and severe periodontitis at baseline.¹¹

As previous studies have reported an overall high degree of agreement between radiographic and clinical assessments of destructive periodontal disease,¹² we explored the association between oral/periodontal disease and diabetes using dental and radiographic records of patients seen at the Comprehen-

Objectives. We explored the association between diabetes mellitus and oral disease in a low-socioeconomic-status urban population.

Methods. Dental records of 150 adults with diabetes and 150 nondiabetic controls from the dental clinic at Columbia University in Northern Manhattan matched by age and gender were studied.

Results. There was a 50% increase in alveolar bone loss in diabetic patients compared with nondiabetic controls. Diabetes, increasing age, male gender, and use of tobacco products had a statistically significant effect on bone loss.

Conclusions. Our findings provide evidence that diabetes is an added risk for oral disease in this low-income, underserved population of Northern Manhattan. Oral disease prevention and treatment programs may need to be part of the standards of continuing care for patients with diabetes (*Am J Public Health*. 2004;94:755–758)

sive Care Clinic at Columbia University School of Dental and Oral Surgery. Most individuals served by this clinic, and included in this retrospective case–control study, reside in Northern Manhattan. The Northern Manhattan communities of Washington Heights/Inwood and Harlem had a population of 500 000 in 2000; residents' incomes were among the lowest in New York City.¹³ An estimated 34% of this population was living at or below the federal poverty level as of 1990, and Northern Manhattan is identified as a Medical and Dental Health Manpower Shortage Area by the Health Resources Services Administration of the Department of Health and Human Services. Forty-nine percent of the residents are Hispanic (mostly of Dominican origin), 44% are African American, and the balance represents other ethnic/racial groups.¹⁴

METHODS

Study Population

Data on 300 dentate adults were included in this study. Dental records for 150 people aged older than 18 with diabetes mellitus seen at the Comprehensive Care Clinic at Columbia University School of Dental and Oral Surgery in Northern Manhattan were selected

at random. Dental records with a full-mouth series of intraoral radiographs taken during a 3-year period (1999–2001) were used. The selection process involved reviewing the recorded medical history to identify a positive history for diabetes. The age range of the diabetic patients was 20 to 88 years. The records of a control group of 150 nondiabetic patients were then chosen. Control subjects were matched by gender and age (± 5 years) to the case group. The age range of the nondiabetic subjects was 18 to 90 years.

The following general patient information was identified from the chart and recorded: age at the time of the radiographic examination, gender, and ethnicity (Hispanic or non-Hispanic). No information on race was available in the dental records. The subjects' tobacco use habits and pregnancy status also were recorded. In addition, for the diabetic group, type of diabetes (type 1, type 2, or unknown) and mode of therapy (insulin, oral hypoglycemic agent, both, or unknown) were noted.

Radiographic Examination and Assessments

Full-mouth periapical and posterior bite-wing radiographs for all patients were evaluated by a single examiner (D.B.P.). All linear

measurements, performed with a ruler, were rounded to the nearest whole millimeter. Sites where excessive radiographic distortion existed or where either the cemento-enamel junction (the junction of the crown and root of the tooth) or alveolar bone crest were unidentifiable were recorded as “nonreadable” and excluded from the analysis. The following parameters were determined:

- *Missing teeth*: the number of missing teeth based on a complete dentition of 32 teeth.
- *Alveolar bone level*: the distance in millimeters from the cemento-enamel junction to the most coronal level along the distal and mesial root surface at which the periodontal ligament space was considered to have a normal width.¹⁵ Thus, an increased value translates into increased alveolar bone loss.
- *Root length*: the distance from the cemento-enamel junction to the radiographic apex of the tooth along the distal and mesial root surface of the tooth.
- *Proportional bone loss*: the ratio of the alveolar bone level minus 2 to the root length minus 2 was calculated for the distal and mesial surface of each tooth (in healthy periodontal tissues, the alveolar bone crest is approximately 2 mm apical to the cemento-enamel junction).
- *Furcation involvement*: the number of multirooted teeth with radiolucency between the roots, suggesting interradiolar bone destruction.
- *Cariou lesions*: the number of teeth with radiolucencies extending into the dentin.
- *Periapical radiolucencies*: the number of teeth with radiolucencies around the apex, indicative of necrosis of pulpal tissue and an inflammatory response at the root tip.
- *Endodontic treatment*: the number of teeth with radiographic evidence of endodontic treatment (obturation of the root canal with radio-opaque material).
- *Restorations and fixed prostheses*: the number of teeth with radiographic evidence of amalgam restorations and crowns, respectively.

Error of the Method

The error inherent in the linear measurements was evaluated by repeated measurements. Specifically, in a subsample of 100 individuals, 50 with diabetes and 50 nondiabetic, measurements of alveolar bone level

were repeated on a second occasion by the same examiner. The mean difference between the first and second measurement for alveolar bone level was 0.6 mm (SD=0.7). At 44.4% of the sites, the double measurements were identical. Reproducibility within 1 mm was 88.4%; within 2 mm it was 98.1%, and within 3 mm it was 99.3%.

The error inherent in the method by which the radiographs were obtained and evaluated also was assessed through the root length measurements as follows: for all teeth, except molars, the 2 root length measurements were averaged and used in the calculation of the mean root length per tooth type. These values were compared with published root length data obtained from measurements on extracted teeth. The radiographically assessed root length was similar to the data reported by Wheeler¹⁶ on extracted teeth: for 14 of the 18 measurements, the difference was less than 1 mm. The biggest differences were noted in maxillary second molar and mandibular premolar measurements, similar to what has been reported previously¹⁷; they seem to be mostly related to the technique used to obtain dental radiographs.

Statistical Analysis

The Statistical Analysis System package (SAS Institute Inc, Cary, NC) was used for calculating mean values, standard deviations, and frequencies, as well as for performing Student *t* tests and multiple regression analyses. *P* values of less than .05 were considered statistically significant.

RESULTS

The case and control group each consisted of 93 females (62%) and 57 males (38%). None of the females in either group was pregnant. In the diabetic group, 103 subjects (69%) were Hispanic and 47 (31%) were non-Hispanic. In the control group, 90 subjects (60%) were Hispanic and 60 (40%) were non-Hispanic. The mean age in the diabetic group was 56.1 ±13.1 years versus 55 ±14.2 years in the control group. Twenty-three (15%) of the cases, versus 25 (17%) of the controls, had reported that they were cigarette smokers, were using some other tobacco product, or both.

Of the 150 patients with diabetes, 23 (15%) were type 1, 103 (69%) were type 2, and 24 (16%) did not know their type and we were unable to retrieve this information from other chart entries. Forty-two (28%) of the diabetic patients were on insulin, 94 were (63%) on 1 or more oral hypoglycemic agents, 10 (7%) were on both, and 5 (3%) were on a diet/exercise regimen only. There was no relevant information for 10 (7%) of the diabetic individuals in the group.

Table 1 shows the radiographic findings in our study population. The mean number of missing teeth per patient was 10 ±6.6 in the control group and 11.5 ±6.8 in the diabetic group. This difference approached, but did not reach, statistical significance (*P*=.06). However, alveolar bone loss was significantly greater in the diabetic group than in the control group (mean alveolar bone level=4.0 ±1.9 mm and 3.1 ±1.4 mm, respectively; *P*=

TABLE 1—Radiographic Findings in Study Population of Diabetic Cases and Nondiabetic Controls in Northern Manhattan

	Nondiabetic (n = 150)	Diabetic (n = 150)	<i>P</i>
Missing teeth	10 (6.6)	11.5 (6.8)	.06
Mean alveolar bone level, mm	3.1 (1.4)	4.0 (1.9)	.0001
Mean proportional bone loss, %	6.0 (5.0)	9.0 (7.0)	.0001
Teeth with furcation involvement	0.5 (1.3)	0.5 (1.1)	.9999
Cariou teeth	2.2 (2.2)	2.4 (2.4)	.4
Teeth with periapical pathology	0.4 (1.0)	0.4 (0.7)	.6
Teeth with restorations and fixed prostheses	8.5 (5.4)	6.7 (5.4)	.005
Teeth with endodontic treatment	1.0 (1.8)	0.6 (1.2)	.02

Note. Values given are means and standard deviations (in parentheses). *P* values in boldface are statistically significant.

.0001). Proportional bone loss was 50% higher in the diabetic group (0.09 ± 0.07) than in the control group (0.06 ± 0.05 ; $P = .0001$). The mean number of teeth with radiographic evidence of furcation involvement per subject in both the control and the diabetic group was 0.5 (± 1.3 and ± 1.1 , respectively; $P = .9999$).

Interestingly, although the mean number of teeth with carious lesions was similar in controls and cases (2.2 ± 2.2 and 2.4 ± 2.4 , respectively; $P = .4$), the control group had significantly more teeth with restorations and fixed prostheses than the diabetic group (8.5 ± 5.4 and 6.7 ± 5.4 , respectively; $P = .005$). Similarly, although the mean number of teeth with periapical radiolucencies per patient in the control group (0.4 ± 1.0) was comparable to that in the diabetic group (0.4 ± 0.7 ; $P = .6$), the control group had significantly more endodontically treated teeth than the diabetic group (1.0 ± 1.8 and 0.6 ± 1.2 , respectively; $P = .02$).

To identify some of the determinants of alveolar bone destruction in our study population (both cases and controls), a multiple regression model using diagnosis of diabetes, age, gender, and cigarette smoking/tobacco product use as the independent variables was constructed. Of particular significance, the model revealed that, in this population, diabetes, increasing age, male gender, and smoking/use of tobacco products had a statistically significant effect on bone loss, with age and diabetes being the most important determinants (Table 2). Multiple regression for alveolar bone destruction in the cases included (in addition to the variables above) only type of diabetes and type of diabetes regimen (insulin vs oral agent) as independent variables. This model also revealed increasing age, male gender, and smoking as statistically significant determinants of bone loss.

DISCUSSION

Our findings in this Northern Manhattan population confirm previous evidence that diabetes mellitus is associated with increased severity of periodontal destruction. Our study cohort represents a low-income, underserved, mostly Hispanic population. Recent studies in children and seniors from this population

TABLE 2—Multiple Regression Analysis of Study Population of Diabetic Cases and Nondiabetic Controls in Northern Manhattan

Independent Variable	Regression Coefficient	P	Squared Part Correlation
Diabetes ^a	0.8930	.0001	0.073
Age	0.0447	.0001	0.107
Gender ^b	-0.5604	.0021	0.034
Smoking/tobacco product use ^c	0.8845	.0003	0.035

Note. The dependent variable was alveolar bone level. $R^2 = 0.25$.

^a0 = no, 1 = yes.

^b0 = male, 1 = female.

^c0 = no, 1 = yes

have reported that, compared with national standards, the oral disease burden in Northern Manhattan is high.^{18–20} In such communities, inability to afford care and limited access to dental services are likely to lead to high levels of oral disease. Diabetes then becomes an added risk in a population already at risk for oral disease. Indeed, although evidence of periodontal destruction and tooth loss was present in the control group of nondiabetic individuals, diabetes clearly conferred an increased risk.

In our multiple regression model for the whole study population, diabetes, age, male gender, and use of tobacco products were identified as significant determinants of bone destruction. For the diabetic group only, the same 3 variables also had a statistically significant effect on bone loss, which is in agreement with what is well established for periodontal destruction in the general population.²¹ Furthermore, there was a trend for an increased number of missing teeth in the individuals with diabetes, but the difference only approached statistical significance ($P = .06$). It is important to remember that tooth loss reflects not only a history of severe periodontal destruction but also the accumulated effects of advanced caries and endodontic infections. In patients with limited resources living in underserved areas, teeth with even moderately advanced dental problems are often extracted rather than restored or endodontically treated.

Of importance is the finding that although the caries rate and presence of periapical pathology were similar in the case and control groups, diabetic patients had fewer teeth previously treated for these conditions. In a recent report,²² patients with diabetes were less likely than those without diabetes to have seen a dentist within the past year; this difference was statistically significant even after age, race/ethnicity, education, income, and dental insurance coverage were adjusted for. Interestingly, the primary reason for not seeing a dentist given in that study was lack of a perceived need.

The etiopathogenesis of periodontitis is complex, and evidence is accumulating that a wide range of factors are probably responsible for the increased risk of periodontal disease observed in diabetes. Impaired recruitment and function of neutrophils, upregulated pro-inflammatory monocyte response to pathogenic bacteria, impaired collagen synthesis, exaggerated collagenolytic activity, and genetic predisposition are all factors that have been implicated.^{23–25} Work from our group has suggested a role for RAGE (receptor of advanced glycation end products) activation in this setting. Blockade of RAGE resulted in suppression of alveolar bone loss and of markers of inflammation/tissue destruction in diabetic mice infected with a periodontal pathogen, providing novel insights into the mechanisms underlying the association between diabetes and oral disease.²⁶

Importantly, evidence of an effect of oral/periodontal infections on systemic health has accumulated in recent years. This includes an effect on the level of metabolic control in diabetic individuals¹¹ and an increased risk for cardiovascular and cerebrovascular events.^{27,28} In combination with the increased risk of vascular disease associated with diabetes mellitus, this highlights another reason for referral of patients with diabetes for dental evaluation and treatment.

Currently, treatment guidelines of the Centers for Disease Control and Prevention recommend that people with diabetes see a dentist at least every 6 months, and more frequently if they are diagnosed with periodontal disease. However, in the American Diabetes Association position statement on standards of care for diabetic patients, an oral examination is sug-

gested as part of the initial evaluation but not as a standard of continuing care.²⁹

Taken together, our findings provide additional evidence that diabetes is associated with an added risk for periodontal destruction, even in a population already at increased risk for oral disease. Further, our findings corroborate the importance of including oral health information in educational materials and promoting oral prevention/treatment programs for patients with diabetes. ■

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Contributors

E. Lalla synthesized the analyses and led the writing of the article. D.B. Park collected the data and performed all radiographic measurements. P.N. Papapanou assisted with the study and conducted the data analyses. I.B. Lamster conceived the study and supervised its implementation. All authors helped to interpret findings and reviewed drafts of the article.

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Human Participant Protection

No protocol approval was needed for this study at the time it was conducted.

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