

# Comparison of Periodontal and Socioeconomic Status Between Subjects With Type 2 Diabetes Mellitus and Non-Diabetic Controls

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**Background:** The association among periodontal conditions, socioeconomic status (SES), and diabetes has been reported. However, there is a lack of published data comparing periodontal conditions among individuals with poorly controlled type 2 diabetes mellitus (T2D). The aim of the present study was to compare the periodontal conditions and SES between subjects with T2D and non-diabetic controls.

**Methods:** A total of 75 (31 males and 44 females) individuals with T2D (62 poorly controlled and 13 well-controlled) and 99 non-diabetic patients (healthy controls; 51 males and 48 females) participated in the study. Plaque index (PI), bleeding on probing (BOP), and probing depth (PD) were investigated. Random blood glucose level was recorded. Premolar and molar marginal bone loss (MBL) was measured digitally on scanned orthopantomograms.

**Results:** Individuals with poorly controlled T2D had increased MBL in molars and maxillary premolars ( $P < 0.05$ ) compared to individuals with well-controlled T2D. PI, BOP, and PD of 4 to  $< 6$  mm were increased in individuals with poorly controlled T2D compared to those with well-controlled T2D ( $P < 0.001$ ). There was no difference between the diabetic groups when PD was  $\geq 6$  mm. Individuals with poorly controlled T2D had a lower SES compared to patients with well-controlled T2D ( $P < 0.05$ ). Illiteracy and the number of missing teeth were not different between the groups.

**Conclusions:** Radiologic and clinical indicators of periodontal destruction were increased in individuals with poorly controlled T2D. Low SES aggravated the periodontal condition in individuals with T2D. *J Periodontol* 2007;78:2112-2119.

## KEY WORDS

**Bleeding; bone loss; smoking; socioeconomic status; type 2 diabetes.**

Periodontal disease is three times more prevalent in individuals with type 2 diabetes mellitus (T2D)<sup>1</sup> and is recognized as the “sixth complication of diabetes.”<sup>2</sup> Individuals with diabetes also may experience periodontal conditions at earlier ages compared to non-diabetic individuals.<sup>2</sup> Case reports<sup>3</sup> showed that there is an increased prevalence and severity of periodontal conditions in individuals with poorly controlled diabetes. Marginal bone loss (MBL) is four times greater in individuals with T2D compared to non-diabetic individuals.<sup>4</sup> Furthermore, increased MBL and probing depth (PD)  $\geq 4$  mm have been associated with poor control of diabetes.<sup>3,5</sup> As a result, periodontal treatment by a specialist may be unable to halt the progression of periodontal disease if the diabetes is poorly controlled.<sup>3</sup> In contrast, an individual with well-controlled diabetes may have better periodontal health.<sup>5</sup> Therefore, regular metabolic control of diabetes is essential.

The population of Pakistan is estimated to be 158 million, and ~10% of adults have T2D<sup>6</sup> compared to Sweden, where 3% to 4% individuals are estimated to be diabetic, the majority of whom have T2D.<sup>7</sup> Poor education and a low socioeconomic status (SES) have been linked with a high prevalence of T2D.<sup>8</sup> These factors may compel individuals to use

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inexpensive and non-conventional modes of treatment for their medical disorders, including T2D. There is a scarcity of published data on periodontal conditions in southeastern Asian countries, including Pakistan.<sup>9</sup>

The aim of the current study was to compare the periodontal conditions and SES between subjects with T2D and non-diabetic controls.

## MATERIALS AND METHODS

### Interview Questionnaire

Between July and December of 2004, a questionnaire was distributed to 1,000 adults aged 45 to 64 years, residing in the Punjab colony, an underprivileged area of Karachi, Pakistan. Participants were given written information (a consent form), which also was translated into Urdu, the native language of Pakistan. Individuals were asked if they had diabetes and were questioned about their SES, education level, and smoking habits. Individuals were requested to present their medical records and/or medical prescriptions, which confirmed the presence or absence of T2D.

The monthly income was recorded in Pakistani currency (rupees), which was converted into United States dollars. One United States dollar was equivalent to 60.97 Pakistani rupees. Smokers were defined as individuals who had been smoking at least one cigarette daily for  $\geq 1$  year. Individuals who had never smoked were classified as non-smokers.

From the 1,000 individuals interviewed, every tenth non-diabetic subject was taken as a control to represent the non-diabetic individuals in this population. Consenting diabetic and non-diabetic individuals were invited to an oral health care center for a clinical examination and radiographs followed by the measurement of a random blood glucose level (RBGL). Edentulous individuals were excluded from the study.

### Treatment for T2D and RBGL

Diabetic treatments were classified as conventional (use of medically approved hypoglycemic agents, such as sulfonylureas and metformin) and non-conventional treatments (use of traditional folk remedies, such as herbalism, homeopathy, and spiritual treatments).<sup>10,11</sup>

T2D was categorized as well-controlled (RBGL  $< 11.1$  mmol/l [ $< 200$  mg/dl]) or poorly controlled (RBGL  $\geq 11.1$  mmol/l [ $\geq 200$  mg/dl]).<sup>12</sup> RBGL was recorded for all individuals using a glucometer.<sup>¶</sup> The individuals were instructed not to eat, drink, or smoke for  $\geq 2$  hours prior to the recording of RBGL.

### Periodontal Examination

All examiners were trained and calibrated before measuring plaque index (PI), bleeding on probing (BOP), and PD. A full-mouth PI<sup>13</sup> was determined, and BOP<sup>14</sup> and PD were measured at four sites (mesial, distal, buccal, and lingual/palatal) on all maxillary and man-

dibular teeth (excluding third molars). PD was measured to the nearest millimeter with a graded probe.<sup>¶</sup> Periodontal pockets of 4 to  $< 6$  mm and  $\geq 6$  mm and the number of missing teeth were recorded. A tooth with embedded root remnants was considered missing.

### Radiographs

Extraoral panoramic radiographs<sup>#</sup> were taken with a panoramic tomography machine<sup>\*\*</sup> and scanned<sup>††</sup> for further analysis. Radiographs were viewed on a computer monitor.<sup>‡‡</sup> Vertical distance was measured from the cemento-enamel junction (CEJ) to the most apical part of marginal bone. MBL was gauged in pixels<sup>§§</sup> on the scanned radiographs. Premolars and molars (excluding third molars) from both arches were assessed. One pixel was calibrated as 0.387 mm.

Tooth surfaces at which the CEJ and/or the bone crest were not evident because of technical reasons (dental caries, overlapping, restorations, and/or poor quality of radiograph) were not measured. Similarly, if the bony landmarks were not identifiable on the mesial or distal aspect, the tooth surface also was excluded. In individuals with well-controlled or poorly controlled T2D, 64 (4.2%) and 170 (11%) teeth were excluded, respectively. Among the controls, 278 (11.3%) teeth were excluded. Overall, most of the excluded teeth were maxillary premolars.

### Ethical Requirements

The ethical committees of Karolinska Institutet and Altamash Institute of Dental Medicine approved the study design in accordance with the Helsinki Declaration of 1975, as revised in 2000.

### Statistics

All statistical analyses were performed using a software program.<sup>|||</sup> MBL on the mesial and distal aspects of premolars and molars from both arches was used as an independent variable and was expressed as a mean with a 95% confidence interval. The association between mean MBL as a dependent variable and the variables of diabetes (yes/no), diabetic status (poorly controlled/well-controlled), PD, PI, BOP, age, gender, smoking (yes/no), and missing teeth as predictors were assessed using one-way analysis of variance (ANOVA). For multiple comparisons, the Bonferroni adjustment post hoc test was performed. T2D and smoking were statistically significant predictors.

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§§ Image Tool 3.0 Program, Department of Dental Diagnostic Science, University of Texas Health Science Center, San Antonio, TX.

||| STATISTICA v. 6.0, Statsoft, Tulsa, OK.

**RESULTS**

**Interview Questionnaire**

Of the 1,000 individuals interviewed, 83 individuals (8.3%) reported that they had medically diagnosed T2D. Seventy-nine of these individuals volunteered to visit the oral health care center. Four edentulous males with diabetes and one non-diabetic female were excluded. Of the 100 self-reported non-diabetic individuals, one edentulous individual (age 62 years) was excluded at the time of RBGL measurement for being unaware of her diabetic status (16.1 mmol/l or 321 mg/dl). Therefore, the study sample consisted of 75 individuals with T2D (31 males and 44 females) and 99 (51 males and 48 females) controls.

**RBGL**

Among individuals with diabetes, 82.7% (27 males and 35 females) had poorly controlled T2D (mean RBGL, 16.3 mmol/l [326 mg/dl]; range: 13.5 to 23.3 mmol/l [126-466 mg/dl]) and 17.3% (four males and nine females) had well-controlled T2D (mean RBGL, 6.5 mmol/l [130 mg/dl]; range: 6.2 to 7.5 mmol/l [136 to 192 mg/dl]). RBGL was <11.1 mmol/l (200 mg/dl) in all self-reported non-diabetic individuals, with a mean RBGL of 6.6 mmol/l (132 mg/dl) and range of 4.5 to 8.6 mmol/l (90 to 172 mg/dl).

Fifty-two individuals with diabetes (69.3%) reported using conventional treatments, and 23 (30.7%) were using non-conventional medications for diabetes. All individuals using non-conventional treatments had poorly controlled T2D.

**MBL in Relation to Age, Gender, and RBGL**

Individuals with T2D had a significantly higher MBL in both arches (mean, 7.4 mm; range: 5.8 to 9.3 mm) compared to controls (mean, 3.7 mm; range 2.8 to 4.2 mm) (*P* <0.0001). In individuals with poorly controlled T2D, MBL was significantly higher in the maxillary molars (#2 and #3), premolars (#12 and #13) (*P* <0.01), and mandibular molars (#30 and #31) compared to individuals with well-controlled T2D (*P* <0.05). These results are shown in Table 1. In both arches, MBL was increased in individuals with poorly controlled T2D (mean, 8.7 mm; range: 3.4 to 11.1 mm) compared to well-controlled T2D (mean, 6.3 mm; range: 2.4 to 9.1 mm) (*P* <0.01).

MBL was notably lower in controls between 45 and 49 years of age (mean age, 47 years; mean bone loss, 1.1 mm; range: 0.5 to 1.7 mm) compared to controls between 60 and 64 years of age (mean age, 62 years; mean bone loss, 4 mm; range: 1.6 to 8 mm) (*P* <0.001). There was no statistically significant effect of age and gender on bone loss in individuals with T2D.

**Number of Missing Teeth, PI, BOP, PD, Smoking, and Age**

Individuals with T2D had more missing teeth (mean, 18.6; range: 14 to 23) compared to controls (mean, 11.2; range: 7 to 14) (*P* <0.01). There was no difference in the number of premolars and molars in individuals with poorly or well-controlled T2D (Table 2). PI (*P* <0.01), BOP (*P* <0.05), and PD (4 to <6 mm; *P* <0.01) were increased in controls aged 60 to

**Table 1.**  
**MBL (mean ± SD) in Maxillary and Mandibular Premolars and Molars in Study Subjects**

	Non-Diabetic Individuals		Individuals With T2D	
	All Individuals (n = 99)	All Individuals (n = 75)	Poorly Controlled T2D (n = 62)	Well-Controlled T2D (n = 13)
<b>Maxilla</b>				
Molars (#2 and #3)	1.68 ± 0.17*	5.26 ± 0.51*	8.03 ± 1.45†	3.26 ± 0.77
Premolars (#4 and #5)	1.17 ± 0.28‡	6.47 ± 0.49‡	4.48 ± 1.05	2.66 ± 0.85
Molars (#14 and #15)	1.91 ± 0.17*	5.84 ± 0.13*	7.89 ± 0.88	6.45 ± 0.49
Premolars (#12 and #13)	1.21 ± 0.47*	5.17 ± 0.20*	8.35 ± 1.11†	3.87 ± 1.47
<b>Mandible</b>				
Molars (#19 and #18)	2.22 ± 0.14*	8.60 ± 0.20*	10.36 ± 2.62	6.50 ± 2.12
Premolars (#21 and #20)	1.78 ± 0.73*	9.31 ± 0.39*	8.50 ± 0.63	10.00 ± 1.11
Molars (#30 and #31)	2.42 ± 0.93*	5.90 ± 0.58*	8.36 ± 1.95†	3.25 ± 1.63
Premolars (#28 and #29)	1.84 ± 0.13*	8.42 ± 0.35*	7.42 ± 0.95	9.43 ± 0.90

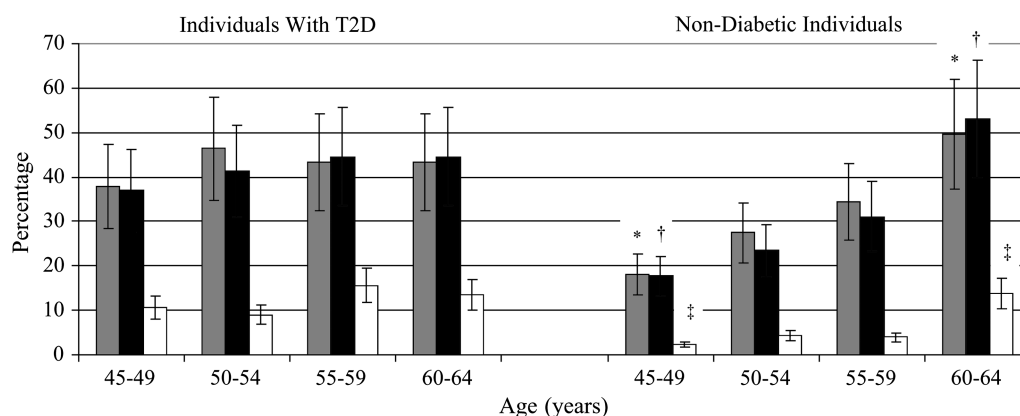
Differences in MBL between controls and individuals with T2D were tested using one-way ANOVA. Differences in MBL between individuals with poorly controlled and well-controlled T2D were tested using one-way ANOVA. For multiple comparisons, the Bonferroni adjustment post hoc test was performed.  
 \* *P* <0.0001.  
 † *P* <0.01 compared to individuals with well-controlled T2D.  
 ‡ *P* <0.001.

**Table 2.**  
**Missing Premolars and Molars (mean  $\pm$  SD) in Study Subjects**

	Missing Teeth in Controls (n)	Missing Teeth in Individuals With T2D (n)		
	All Individuals (n = 99)	All Individuals (n = 75)	Poorly Controlled T2D (n = 62)	Well-Controlled T2D (n = 13)
<b>Maxilla</b>				
Molars (#2 and #3)	0.20 $\pm$ 0.04*	0.61 $\pm$ 0.18*	0.83 $\pm$ 0.23	0.92 $\pm$ 0.31
Premolars (#4 and #5)	0.13 $\pm$ 0.03*	0.37 $\pm$ 0.07*	0.77 $\pm$ 0.18	0.61 $\pm$ 0.17
Molars (#14 and #15)	0.12 $\pm$ 0.04*	0.26 $\pm$ 0.07*	0.40 $\pm$ 0.11	0.30 $\pm$ 0.05
Premolars (#12 and #13)	0.17 $\pm$ 0.06*	0.40 $\pm$ 0.12*	0.59 $\pm$ 0.19	0.32 $\pm$ 0.10
<b>Mandible</b>				
Molars (#19 and #18)	0.09 $\pm$ 0.03*	0.33 $\pm$ 0.09*	0.58 $\pm$ 0.17	0.41 $\pm$ 0.11
Premolars (#21 and #20)	0.06 $\pm$ 0.03*	0.16 $\pm$ 0.08*	0.25 $\pm$ 0.14	0.20 $\pm$ 0.09
Molars (#30 and #31)	0.11 $\pm$ 0.17*	0.32 $\pm$ 0.14*	0.53 $\pm$ 0.18	0.39 $\pm$ 0.17
Premolars (#28 and #29)	0.08 $\pm$ 0.12*	0.25 $\pm$ 0.12*	0.43 $\pm$ 0.10	0.29 $\pm$ 0.10

Differences in the number of missing teeth between controls and individuals with T2D were tested using one-way ANOVA.

\*  $P < 0.01$ .



**Figure 1.**

PI (gray bars), BOP (black bars), and PD (4 to <6 mm) (white bars) in type 2 diabetic individuals and controls in relation to age. There was a statistically significant difference in PI, BOP, and PD (4 to <6 mm) among non-diabetic individuals aged 45 to 49 and 60 to 64 years. There was no statistical significance for periodontal conditions between non-diabetic individuals aged 45 to 49 and 50 to 54 years. There was no statistical significance for periodontal conditions between non-diabetic individuals aged 50 to 54 and 55 to 59 years. There was no statistical significance for periodontal conditions between non-diabetic individuals aged 55 to 59 and 60 to 64 years. Periodontal conditions were not related to age among individuals with T2D. \* $P < 0.01$ ; † $P < 0.01$ ; ‡ $P < 0.05$ .

64 years (mean, 62 years) compared to controls aged 45 to 49 years (mean, 46.6 years). There was no significant effect of age on PI, BOP, and PD (4 to <6 mm) in individuals with T2D (Fig. 1).

PI, BOP, and PD were increased in individuals with T2D compared to controls ( $P < 0.0001$ ; Table 3). PI, BOP, and PD of 4 to <6 mm also were higher in individuals with poorly controlled T2D compared to individuals with well-controlled T2D ( $P < 0.05$ ). PD of 4 to <6 mm was increased in individuals with poorly controlled T2D compared to individuals with well-controlled T2D ( $P < 0.05$ ). There was no difference between the diabetic groups when PD was  $\geq 6$  mm.

All self-reported smokers were males (mean ages of 51 and 49.3 years among individuals with T2D and controls, respectively). Smoking habits were self-reported by 15.2% of controls and 6.7% of individuals with T2D. The type 2 diabetic and non-diabetic smokers had been smoking for an average of 19.5 and 20.4 years, respectively (range, 8 to 25 years). Bone loss was significantly higher in non-diabetic smokers (mean, 5.8 mm; range: 2.6 to 10 mm) compared to non-smokers (mean, 3.4 mm; range: 2.5 to 4.7 mm) ( $P < 0.001$ ). There was no significant difference in MBL, PI, BOP, PD, and missing teeth between type 2 diabetic smokers and non-smokers (data not shown).

**Table 3.**  
**PI, BOP, and PD in Study Subjects**

	Controls (mean ± SD)	Individuals With T2D (mean ± SD)		
	All Individuals (n = 99)	All Individuals (n = 75)	Poorly Controlled T2D (n = 62)	Well-Controlled T2D (n = 13)
PI (%)	32.5 ± 18.86*	63.1 ± 11.76*	66.9 ± 33.87†	44.9 ± 25.75
BOP (%)	28.9 ± 22.68*	65.4 ± 15.99*	70.5 ± 35.17‡	40.6 ± 23.26
PD (4 to <6 mm)	0.7 ± 0.93*	2.2 ± 1.15*	2.4 ± 1.18§	0.3 ± 0.10
PD (≥6 mm)	0.06 ± 0.08*	0.2 ± 0*	0.2 ± 0.12	0.2 ± 0.12

Differences between controls and individuals with T2D were tested using one-way ANOVA. Differences between individuals with poorly controlled T2D and well-controlled T2D were tested using one-way ANOVA. For multiple comparisons, the Bonferroni adjustment post hoc test was performed.

\*  $P < 0.0001$ .

†  $P < 0.05$  compared to subjects with well-controlled T2D.

‡  $P < 0.01$  compared to subjects with well-controlled T2D.

§  $P < 0.0001$  compared to subjects with well-controlled T2D.

**Table 4.**  
**PI, BOP, PD, and MBL in Controls**

	Non-Diabetic Smokers	Non-Diabetic Non-Smokers
% individuals	15.2	84.8
PI (% sites)	60.9*	26.9
BOP (% sites)	15.6†	30.5
Pockets with PD (4 to <6 mm; n)	13.8‡	7.1
Pockets with PD (≥6 mm; n)	1	2
MBL (mm)	5.8‡	3.4
Missing teeth (n)	4.5*	1.6

Differences between PI, BOP, PD, MBL, and number of missing teeth between non-diabetic smokers and non-smokers were tested using one-way ANOVA.

\*  $P < 0.001$  compared to non-smokers.

†  $P < 0.01$  compared to non-smokers.

‡  $P < 0.05$  compared to non-smokers.

PI was increased in non-diabetic smokers (60.9%) compared to non-smokers (26.9%) in the same group ( $P < 0.01$ ). Non-smokers in the control group had increased BOP (30.5%) compared to smokers (15.6%) in the same group ( $P < 0.05$ ). The number of sites with PD 4 to <6 mm was significantly greater in non-diabetic smokers (mean, 13.8) compared to non-smokers (mean, 7.1;  $P < 0.05$ ). There was no difference in the number of sites with PD ≥6 mm in non-diabetic smokers (mean age, 51 years) and non-smokers (mean age, 50 years). Notably, more teeth were missing in non-diabetic smokers (mean, 4.5; range: 2 to 11) compared to non-smokers (mean,

1.6; range: 0 to 9 teeth) in the same group ( $P < 0.001$ ). These results are shown in Table 4.

**Education and SES**

Illiteracy was significantly higher among individuals with T2D (30.7%) compared to controls (9.1%;  $P < 0.05$ ). There was no significant difference in the illiteracy rate between the individuals with poorly controlled T2D (36.6%) and those with well-controlled T2D (24.8%).

Among the controls, 61% voluntarily provided their monthly income compared to 48% of individuals with T2D. The monthly income of controls (mean, \$227.9; range: \$125 to \$310) was significantly higher compared to individuals with T2D (mean, \$100; range: \$50 to \$158.3;  $P < 0.05$ ). Individuals with well-controlled T2D (mean, \$154.6; range: \$80 to \$200) also had a significantly higher SES compared to individuals with poorly controlled T2D (mean, \$54.6; range: \$50 to \$133.3;  $P < 0.05$ ).

**DISCUSSION**

Diabetes is an important risk factor for periodontal disease.<sup>15,16</sup> However, in individuals with diabetes, the severity of periodontal conditions may vary depending on the maintenance of blood glucose level. A clinical study<sup>3</sup> showed that periodontal inflammation was increased in patients with poorly controlled T2D. One possible biologic explanation for this finding is the accumulation of glucose-mediated advanced glycation end products (AGEs) that impair the chemotactic and phagocytic function of polymorphonuclear leukocytes and produce proinflammatory cytokines, thereby leading to periodontal inflammation and bone loss in individuals with diabetes.<sup>17</sup>

Treatment of T2D is associated strongly with SES. In the current study, all individuals on non-conventional treatments had low standards of living. An underprivileged lifestyle may compel individuals with diabetes to neglect their medically prescribed antidiabetic therapies and use inexpensive remedies, including herbalism, homeopathy, and spiritual treatments. In the current study, periodontal conditions and RBGL were increased in all individuals on non-conventional remedies for T2D. A study<sup>18</sup> conducted in an urban area of Karachi, Pakistan reported that ~38% of individuals with diabetes had received educational information about diabetes care. However, in rural areas where education is less readily accessible, the individuals may have poor diabetes perception and control. Oral health care providers should be aware of this issue. Individuals with diabetes should be informed that traditional folk remedies lead to poor glycemic control that increases the severity of periodontal conditions.

In prediagnosed patients with diabetes, RBGL measurement is a practical method to check the blood glucose levels.<sup>19</sup> Although glucometers can be used effectively for screening the blood glucose level in prediagnosed cases, the diabetic status (poorly controlled or well-controlled) may be a short-term finding. Measurement of glycated hemoglobin A<sub>1c</sub> is a long-term indicator of diabetes control.<sup>20</sup>

In poorly controlled diabetes, increased salivary glucose levels may assist the survival of plaque bacteria that may elevate inflammation levels, increase periodontal pockets, and lead to bone loss.<sup>21</sup> Simultaneously, a decreased salivary flow rate in individuals with diabetes also may help these bacteria to adhere to tooth surfaces. The present study demonstrated increased PI, BOP, and PD of 4 to <6 mm in individuals with poorly controlled T2D.

An association exists between bone loss, as observed on panoramic radiographs, and clinical periodontal evaluation.<sup>22</sup> Therefore, panoramic radiographs can be substituted for full-mouth intraoral radiographs.<sup>23</sup> Computer-based measurements for MBL may be advantageous compared to the use of handheld instruments, such as a metric rule or caliper. Additionally, image-processing programs may be used to improve the visual quality of the radiograph, enhance a selected region on the panoramic radiograph, and visualize bone height in different contrasts. Such features of computer-based imaging may assist in the detection of minor bony changes, which may otherwise be overlooked.<sup>24</sup> A study<sup>25</sup> also reported a 40% improvement in results with the use of computer-based methods compared to manual measurement. Based on these advantages, we chose computer-based programs for measuring the MBL. In the present study, MBL was 1.6 times greater in the premolar and

molar regions in poorly controlled T2D compared to well-controlled T2D. In a prospective, longitudinal study,<sup>26</sup> the maxillary premolars (#12 and #13) and molars (#2 and #3) showed significant MBL over a period of 17 years. The present study supported these results; however, we also observed a higher MBL in mandibular molars (#30 and #31).

It has been reported that the risk for periodontal bone loss increases with age.<sup>2,27</sup> Our results showed that PI, BOP, and PD (4 to <6 mm) also increased with age. However, individuals with T2D did not show any significant relationship between these variables with age. It may be hypothesized that in individuals with T2D, the pathogenesis of the disease makes PI, BOP, and PD (4 to <6 mm) independent of age. Further studies on individuals with poorly and well-controlled T2D may help to predict the relationship of these variables to age. MBL was 2.4 times higher in individuals with TD2, with an age range of 45 to 49 years, compared to controls in the same age group. This is in accordance with earlier results.<sup>28</sup> The present study showed that age does not play a dominant role in influencing periodontal bone loss in individuals with T2D. The pathophysiology may be explained by the glucose-mediated AGEs that encourage the production of interleukin-1 and tumor necrosis factor-alpha.<sup>17,29</sup> These proinflammatory cytokines play an essential role in the pathogenesis of diabetic complications, including periodontal disease.<sup>17,29</sup> Therefore, it may be postulated that poorly controlled T2D induces the production of proinflammatory cytokines and exposes individuals with diabetes to periodontal conditions at earlier ages compared to non-diabetic individuals. There was no association between gender and periodontal conditions in individuals with T2D and controls, which is in accordance with a recent study.<sup>30</sup>

Smoking masks the signs of inflammation and tissue damage by suppressing gingival bleeding.<sup>31</sup> An experimental study<sup>32</sup> demonstrated that nicotine causes an acute vasoconstriction in the skin, and the same mechanism is assumed to take place in gingival tissues as well. In Pakistan, the prevalence of smoking was reported to be 16.1% and is dominant among males between 45 and 64 years of age.<sup>33</sup> The present study showed similar results; 15.2% of controls (all males) were habitual smokers. We observed a significantly high number of sites with PD 4 to <6 mm with reduced BOP in non-diabetic smokers as reported earlier.<sup>14,34,35</sup>

Periodontal disease has been associated with low SES.<sup>23,36</sup> In low-income countries like Pakistan, where the majority of inhabitants strive to meet a basic living standard, oral health care may not be a priority and may be considered more of a luxury, rather than an obligation. A study on Pakistani immigrants in

Norway also concluded that living standards and SES are strong predictors of periodontal disease in these individuals.<sup>37</sup> An improved SES may be associated with better diabetes control.<sup>38</sup> In the current study, a superior SES in individuals with well-controlled T2D may have permitted them to use conventional treatments for diabetes and to maintain their oral health compared to individuals with poorly controlled T2D. We expected to observe a difference in the number of teeth present between individuals with poorly and well-controlled T2D, but the results did not show any statistical significance. However, it remains to be established if both groups of individuals with a shorter duration of the disease show a difference in the number of missing teeth. A study<sup>29</sup> also reported that periodontal therapy in conjunction with systemic antimicrobial treatment reduced the severity of periodontal conditions in individuals with poorly controlled T2D.

## CONCLUSIONS

Individuals with T2D experience increased MBL, BOP, and PD of 4 to <6 mm compared to controls. Low SES is the major contributing factor in the progression of periodontal conditions in T2D.

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## REFERENCES

- Emrich LJ, Shlossman M, Genco RJ. Periodontal disease in non-insulin-dependent diabetes mellitus. *J Periodontol* 1991;62:123-131.
- Löe H. Periodontal disease. The sixth complication of diabetes mellitus. *Diabetes Care* 1993;16:329-334.
- Ainamo J, Lahtinen A, Uitto VJ. Rapid periodontal destruction in adult humans with poorly controlled diabetes. *J Clin Periodontol* 1990;17:22-28.
- Taylor GW, Burt BA, Becker MP, Genco RJ, Shlossman M. Glycemic control and alveolar bone loss progression in type 2 diabetes. *Ann Periodontol* 1998;3:30-39.
- Tervonen T, Oliver RC. Long-term control of diabetes mellitus and periodontitis. *J Clin Periodontol* 1993;20:431-435.
- Nishtar S. Prevention of non-communicable diseases in Pakistan: An integrated partnership-based model. *Health Res Policy Syst* 2004;2:7.
- Berger B, Stenstrom G, Chang YF, Sundkvist G. The prevalence of diabetes in a Swedish population of 280,411 inhabitants. A report from the Skaraborg Diabetes Registry. *Diabetes Care* 1998;21:546-548.
- Larrañaga I, Arteagoitia JM, Rodriguez JL, et al. Socioeconomic inequalities in the prevalence of type 2 diabetes, cardiovascular risk factors and chronic diabetic complications in the Basque Country, Spain. *Diabet Med* 2005;22:1047-1053.
- Corbet EF, Zee KY, Lo ECM. Periodontal diseases in Asia and Oceania. *Periodontol 2000* 2002;29:122-152.
- Gerich JE. Redefining the clinical management of type 2 diabetes: Matching therapy to pathophysiology. *Eur J Clin Invest* 2002;32:46-53.
- Lee MS, Lee MS, Lim HJ, Moon SR. Survey of the use of complementary and alternative medicine among Korean diabetes mellitus patients. *Pharmacoepidemiol Drug Saf* 2004;13:167-171.
- American Diabetes Association. Diagnosis and classification of diabetes mellitus. *Diabetes Care* 2005;28:S37-S42.
- Ainamo J, Bay I. Problems and proposals for recording gingivitis and plaque. *Int Dent J* 1975;25:229-235.
- Mühlemann HR, Son S. Gingival sulcus bleeding – A leading symptom in initial gingivitis. *Helv Odontol Acta* 1971;15:107-113.
- Paulander J, Wennström JL, Axelsson P, Lindhe J. Some risk factors for periodontal bone loss in 50-year-old individuals. *J Clin Periodontol* 2004;31:489-496.
- Campus G, Salem A, Uzzau S, Baldoni E, Tonolo G. Diabetes and periodontal disease: A case-control study. *J Periodontol* 2005;76:418-425.
- Liu R, Bal HS, Desta T, et al. Diabetes enhances periodontal bone loss through enhanced resorption and diminishes bone formation. *J Dent Res* 2006;85:510-514.
- Rafique G, Azam SI, White F. Diabetes knowledge, beliefs and practices among people with diabetes attending a university hospital in Karachi, Pakistan. *East Mediterr Health J* 2006;12:590-598.
- Lambert TJ, Chapman LH. Consensus Working Group. Diabetic, psychotic disorders and antipsychotic therapy: A consensus statement. *Med J Aust* 2004;181:544-548.
- Bennett CM, Guo M, Dharmage SC. HbA1c as a screening tool for detection of type 2 diabetes: A systematic review. *Diabet Med* 2007;24:333-343.
- American Dental Association. Diabetes and oral health. *J Am Dent Assoc* 2002;133:1299.
- Walsh TF, Al-Hokail OS, Fosam EB. The relationship of bone loss observed on panoramic radiographs with clinical periodontal screening. *J Clin Periodontol* 1997;24:153-157.
- Persson RE, Tzannetou S, Feloutzis AG, Brägger U, Persson GR, Lang NP. Comparison between panoramic and intra-oral radiographs for the assessment of alveolar bone levels in a periodontal maintenance population. *J Clin Periodontol* 2003;30:833-839.
- Mol A. Imaging methods in periodontology. *Periodontol 2000* 2004;34:34-48.
- Hildebolt CF, Brunnsden B, Yokoyama-Crothers N, et al. Comparison of reliability of manual and computer-intensive methods for radiodensity measures of alveolar bone loss. *Dentomaxillofac Radiol* 1998;27:245-250.
- Airila-Månsson S, Söder B, Klinge B. Bone height changes in individuals with periodontal disease: A 17-year prospective longitudinal study. *J Clin Periodontol* 2005;32:822-827.
- Norderyd O, Hugoson A. Risk of severe periodontal disease in a Swedish adult population. *J Clin Periodontol* 1998;25:1022-1028.

28. Thorstensson H, Hugoson A. Periodontal disease experience in adult long-duration insulin-dependent diabetes. *J Clin Periodontol* 1993;20:352-358.
29. Promsudthi A, Pimapansri S, Deerochanawong C, Kanchanasita W. The effect of periodontal therapy on poorly controlled type 2 diabetes mellitus in older individuals. *Oral Dis* 2005;11:293-298.
30. Bakhshandeh S, Murtomaa H, Mofid R, Vehkalahti MM, Suomalainen K. Periodontal treatment needs of diabetic adults. *J Clin Periodontol* 2007;34:53-57.
31. Dietrich T, Bernimoulin JP, Glynn RJ. The effect of cigarette smoking on gingival bleeding. *J Periodontol* 2004;75:16-22.
32. Black CE, Huang N, Neligan PC, et al. Effect of nicotine on vasoconstrictor and vasodilator responses in human skin vasculature. *Am J Physiol Regul Integr Comp Physiol* 2001;281:R1097-R1104.
33. Nasir K, Rehan N. Epidemiology of cigarette smoking in Pakistan. *Addiction* 2001;96:1847-1854.
34. Natto S, Baljoon M, Bergström J. Tobacco smoking and periodontal bone height in a Saudi Arabian population. *J Clin Periodontol* 2005;32:1000-1006.
35. Baharin B, Palmer PM, Coward P, Wilson RF. Investigation of periodontal destruction patterns in smokers and non-smokers. *J Clin Periodontol* 2006;33:485-490.
36. Klinge B, Norlund A. A socioeconomic perspective on periodontal diseases – A systematic review. *J Clin Periodontol* 2005;32:314-325.
37. Selikowitz HS. The relationship between periodontal conditions and perceptions of periodontal health among Pakistani immigrants in Norway. *J Clin Periodontol* 1987;14:340-344.
38. Mainous AG, King DE, Garr DR, Pearson WS. Race, rural residence, and control of diabetes and hypertension. *Ann Fam Med* 2004;2:563-568.

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