

# Periodontal Disease Status of Pregnant Women with Diabetes Mellitus

Adrian Kasaj, Gregor Georg Zafiroopoulos, Haki Tekyatan, Alex Pistorius and Brita Willershausen

Department of Operative Dentistry, »Johannes Gutenberg« University, Mainz, Germany

## ABSTRACT

*The aim of the present study was to evaluate the association between type I diabetes mellitus (DM) and periodontal disease in pregnant women. Fifty-two pregnant women aged 27.9±6.9 years with type I DM participated in the present study. Forty-two non-pregnant type I female diabetics (mean age: 27.9±6.1 years) and 121 healthy non-pregnant women (mean age: 29.1±5.7 years) without diabetes formed the control group. All subjects were given a clinical periodontal examination including probing pocket depth (PPD), probing attachment level (PAL), assessment of plaque and gingivitis scores (SBI). Blood parameters included levels of hemoglobin, glycosylated hemoglobin, total cholesterol, triglyceride and leukocytes. The pregnant diabetic subjects showed despite a good metabolic control significantly higher values for the SBI compared to the controls. Pregnant diabetic subjects displayed a significant correlation between the dose of insulin per day and PPD ( $p \leq 0.05$ ) as well as the PAL ( $p \leq 0.05$ ). In conclusion, the results of the study indicate that pregnant diabetics demonstrate a higher degree of periodontal inflammation and destruction compared to non-pregnant diabetics and healthy non-pregnant patients.*

**Key words:** diabetes mellitus, periodontal disease, pregnancy in diabetes, oral health

## Introduction

Diabetes mellitus (DM) is a chronic metabolic disorder with more than 100 million people affected worldwide<sup>1</sup>. Uncontrolled or poorly controlled diabetes is associated with a multitude of complications, including retinopathy, nephropathy, neuropathy, macrovascular disease, impaired wound healing, delayed wound healing and increased susceptibility to oral infections, including periodontitis<sup>2–4</sup>. Numerous controlled studies have demonstrated significant associations between diabetes and periodontal disease, which is valid for both type I DM and type II DM<sup>5–10</sup>. In an initial study by Cianciola et al. (1982)<sup>11</sup>, investigating the relationship between periodontal disease and type I DM (formerly »insulin-dependent diabetes mellitus«) in a population aged 4–33 years, the prevalence of periodontitis among diabetics 13 to 18 years old was 13.6%. Individuals 19–32 years old had a prevalence of 39% as compared to 2.5% in the non-diabetic controls. In addition, the authors found a correlation between the duration of diabetes and the severity of periodontal disease. Already in young diabetics the prevalence of periodontal disease is higher than in non-diabetic children of the same age<sup>9,12</sup>. The glycemic status of these young dia-

betics affects the periodontal probing depths, salivary pH, buffering capacity and peroxidase activity<sup>13</sup>. In an epidemiologic study among 1.342 Pima Indians subjects with type II DM (»formerly non insulin dependent diabetes«) were 2.8 times more likely to have periodontal disease as defined by clinical attachment loss compared to non-diabetic individuals, and 3.4 times more as defined by radiographic bone loss<sup>14</sup>. The increased risk of developing periodontal disease in this population could not be explained on the basis of age, gender or oral hygiene.

The incidence of periodontitis increases among diabetics as the population ages and is more frequent in individuals with more advanced systemic complications<sup>5</sup>. However, the increased susceptibility does not correlate with increased levels of plaque and calculus, and there are no differences between diabetic and control patients in prevalence and quantity of periodontal pathogens in periodontal pockets<sup>15,16</sup>.

Pregnancy is associated with an increased inflammatory response of the gingiva due to changes in hormone levels (androgens, estrogens and progesterone) and should

be considered as a modifying factor for the development of gingival disease. The prevalence of pregnancy gingivitis ranges from 35 to 100%, occurring first in the second and third month of pregnancy<sup>17–19</sup>. Pregnant women often show hyperplastic changes of the marginal gingiva with increases in parameters such as gingival probing depths, bleeding on probing and crevicular fluid flow<sup>20–22</sup>. Pregnant diabetics have to be considered as a high risk group within pregnant patients due to a complex metabolic disease combined with hormonal changes.

The aim of the present study was to compare the periodontal status of pregnant type I diabetics with non-pregnant female type I diabetics and a healthy control.

## Materials and Methods

Fifty-two pregnant women with type I DM that were being treated for diabetes at a General Hospital (Munich-Schwabing), Division of Diabetes, participated in the present study. Individuals with a history of infectious disease and patients subjected to immunosuppressive therapy were omitted from the study. Patients taking cyclosporin, calcium channel blockers or antibiotics within the last six-months were also excluded from the study. The mean age of the pregnant diabetics was 27.9±6.9 years and the mean duration of gestation was 20±9.7 weeks, respectively. The mean time span since onset of diabetes was 10.0 years. Forty-two non-pregnant female type I diabetics (mean age: 27.9±6.1 years) and 121 healthy non-pregnant female patients (mean age: 29.1±5.7 years) without diabetes formed the control group. A written informed consent was obtained prior to the study from the patients and control subjects. At the time of enrollment, the following parameters were assessed for each patient: age, height, weight, duration of gestation, blood pressure (hypertension was defined according to WHO guidelines), type of diabetes (type I, type II) and diabetes therapy (insulin, oral agent, diet). Furthermore the values for hemoglobin, glycosylated hemoglobin (HbA1c), total cholesterol, triglyceride, leukocytes and the albumin excretion rate were obtained from the patients' medical history. A previously calibrated examiner performed the periodontal examinations. All subjects received a clinical dental examination to determine caries frequency (DMF-T value), probing pocket depth (PPD), probing attachment level (PAL), assessment of plaque (approximal plaque index API, according to Lange et al. 1974)<sup>23</sup> and gingivitis scores (sulcus bleeding index SBI, according to Lange et al. 1977)<sup>24</sup>. The periodontal measurements were recorded at 6 sites on each of the 6 Ramfjord teeth (16, 21, 24, 36, 41, 44) using a pressure sensitive probe.

All statistical analyses were performed using SPSS (11.0 for windows). For the clinical parameters DMF-T, PPD, PAL, API and SBI data were expressed as mean values ± standard deviation. For the comparison between the groups the paired t-test was used. P-values <0.05 were considered as statistically significant.

## Results

The results of the oral hygiene evaluation demonstrated a significantly higher mean approximal plaque index in pregnant diabetic subjects compared to the controls: 82±13.4% versus 53±15.2% ( $p<0.001$ ). However, no significant differences in approximal plaque index were observed between pregnant diabetics and non-pregnant diabetics (Figure 1). No significant differences between the three groups were shown regarding the frequency of caries (DMF-T values). The mean DMF-T for the controls was 16.4±4.4, for the non-pregnant diabetics 15.5±5.3, and for the pregnant diabetics 16.9±4.7, moreover no statistical difference between the groups was observed in terms of the number of missing teeth. Concerning periodontal inflammation, statistically significant differences were found between the three groups. The mean sulcus bleeding index for the healthy control group was 43.8±28.7% versus 48.1±29.2% in non-pregnant diabetics and 73.5±25.8% in pregnant diabetic subjects. The sulcus bleeding index was found to be significantly higher ( $p<0.001$ ) in pregnant diabetic subjects than in non-pregnant diabetics and the controls (Figure 2). Mean probing depths for pregnant diabetics (4.1±0.9 mm) were significantly ( $p<0.001$ ) greater compared to that of non-pregnant diabetics (3.2±0.9 mm) and healthy controls (2.9±0.4 mm). In terms of clinical attachment loss no significant differences were obtained for the three groups (pregnant type I diabetics: 1.1±0.8 mm, non-pregnant type I diabetics: 1.2±0.9 mm, healthy controls: 0.8±0.5 mm). The pregnant diabetic subjects were all under medical surveillance and demonstrated good metabolic control

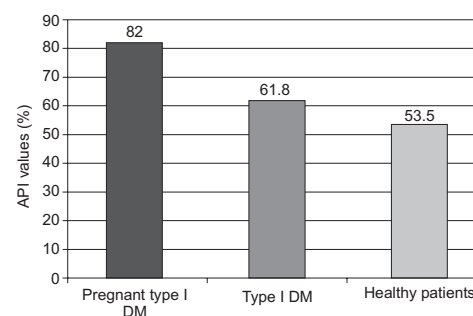


Fig. 1. Mean approximal plaque index (API) of the pregnant diabetics and control groups. DM – Diabetes mellitus.

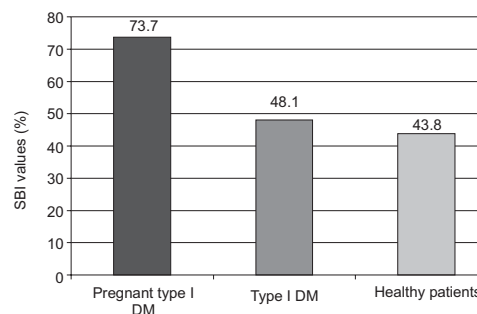


Fig. 2. Mean sulcus bleeding index (SBI) of the pregnant diabetics and control groups. DM – Diabetes mellitus.

**TABLE 1**  
ANAMNESTIC AND MEDICAL PARAMETERS OF  
NON-PREGNANT TYPE I DIABETICS (n=42) AND PREGNANT  
TYPE I DIABETICS (n=52)

	Type I DM		Pregnant Type I DM	
	x	±	x	±
Age (years)	27.5	9.3	27.9	4.3
Duration of diabetes (years)	7.9	8.8	9.8	7.4
Dose of insulin/day	44.6	20.6	55.9	25
Hemoglobin	–	–	12.8	1.2
HbA1c (%)	9.8	2.6	7.8	1.5
Glucose (mg/dL)	226.4	66.3	160	80.5
Creatinine (mg/dL)	1.2	1.3	0.8	0.8

DM – Diabetes mellitus, HbA1c – glycosylated hemoglobin

(HbA1c<8%) for blood glucose and HbA1c. Mean HbA1c values in pregnant diabetic subjects were noticeably lower than in non-pregnant diabetics. In metabolically well controlled pregnant diabetics, no relationship was detected between diabetic status and clinical periodontal parameters. However, in poorly controlled diabetic subjects (HbA1c>8%) a significant association between HbA1c values and the sulcus bleeding index could be observed. Table 1 displays further blood parameters for the pregnant and non-pregnant type I diabetics. The mean time since onset of the disease for the diabetic subjects was not directly associated with the occurrence of periodontal disease. In contrast to that the pregnant diabetic subjects displayed a significant correlation between the dose of insulin per day and probing pocket depth ( $p \leq 0.05$ ) as well as the loss of attachment ( $p \leq 0.05$ ). The average dosis of insulin per day was significantly lower ( $p \leq 0.05$ ) in diabetic subjects with mean probing pocket depths  $\leq 3$

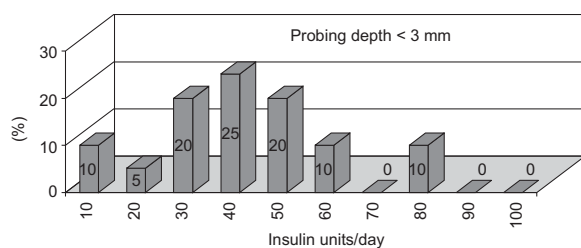


Fig. 3. Association between insulin doses per day and sites with probing pocket depth < 3 mm.

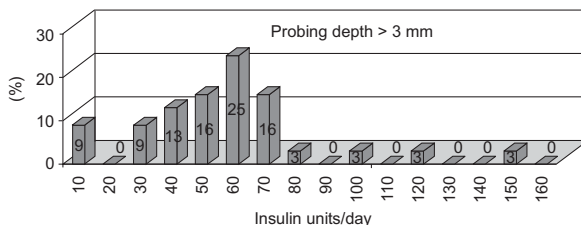


Fig. 4. Association between insulin doses per day and sites with probing pocket depth > 3 mm.

mm (45.5 dose of insulin/day, Figure 3) compared to diabetics with mean probing pocket depths  $\geq 3$  mm (59.8 dose of insulin/day, Figure 4). For all other clinical diabetes parameters no significant interaction with the periodontal condition was found.

## Discussion

In the present study pregnant diabetic subjects exhibited significantly higher plaque scores compared to the non-diabetic control group, which is in accordance to observations of Aren et al. (2003)<sup>13</sup> and Campus et al. (2005)<sup>8</sup>. This can be explained by the fact that in diabetics excess glucose enters the oral cavity through saliva and gingival crevicular fluid, which is responsible for a polysaccharide formation and an enhanced plaque growth<sup>13</sup>. Another related factor for higher plaque index values in this group might be a lack of knowledge about oral hygiene procedures in these patients. In terms of caries frequency no significant differences were found between pregnant diabetics, non-pregnant diabetics and the healthy control group, although a low caries prevalence in children and adolescents with type I DM has been reported due to a sucrose restricted diet<sup>25,26</sup>. In the present study pregnant diabetics exhibited with a sulcus bleeding index value of 73.5% greater gingival inflammation compared to non-diabetics and the healthy control group despite a good metabolic control. In a longitudinal study of 132 pregnant diabetic women under care Albrecht et al. (1987)<sup>27</sup> revealed even a 96.2% prevalence of gingivitis which was most pronounced between weeks 11 to 15 and 24 to 26 of pregnancy. Gislén et al. (1980)<sup>28</sup> demonstrated that diabetic children with poor metabolic control showed higher tendencies towards gingivitis than non-diabetic children.

However, in the present study a significant correlation between gingival inflammation and the severity of diabetes was only found for the poorly controlled non-pregnant diabetic subjects. This finding is consistent with studies suggesting that poorly controlled diabetic subjects experience more often periodontal disease over the long term<sup>29,30</sup>. Furthermore a significant correlation between severity of diabetes and attachment loss despite a good metabolic control was only found in the pregnant diabetics. A previous study by Safkan-Seppälä & Ainamo (1992)<sup>31</sup> has already shown that more attachment loss and approximal bone loss can be found in poorly controlled than in well controlled type I DM subjects, whereas the influence of diabetes duration is still discussed controversially<sup>32,33</sup>. A higher degree of gingival inflammation and enhanced periodontal destruction manifested by greater attachment loss in pregnant diabetics compared to non-diabetics was already reported by Guthmiller et al. (2001)<sup>34</sup>. There are several autoimmune factors and mechanisms that may explain the increased severity of periodontitis in diabetics compared to non-diabetics. In numerous studies diminished polymorphnuclear leukocyte (PMN) functions such as chemotaxis, adherence and phagocytosis have been demonstrated<sup>35,36</sup>. Reduced PMN function in diabetics might lead to impaired

host resistance and progression of infection<sup>37</sup>. Diabetic patients with periodontitis exhibit significantly higher levels of PGE<sub>2</sub>, IL-1 $\beta$  and TNF- $\alpha$  in crevicular fluid compared to non-diabetic controls, which might explain why diabetic subjects compared to non-diabetics react with a higher degree of inflammation to an equivalent bacterial load<sup>38</sup>. Other factors for the increased risk for infections in diabetics are vascular changes in the form of thickened basement membranes and changes in the physical properties of the gingival capillaries<sup>39,40</sup>.

Diabetes in pregnant women is associated with an increased risk for maternal and neonatal morbidity. As the

diabetic control can be complicated by periodontitis and hormonal changes lead to an exacerbation of periodontal inflammation this has an important influence on maternal and fetal outcomes<sup>41</sup>. In the present study a significantly increased gingival inflammation and periodontal destruction was found in pregnant type I diabetic subjects despite a good metabolic control compared to non-pregnant diabetics with poor metabolic control. Periodontal therapy might influence the diabetic control and thus maternal and fetal outcomes. The effect of periodontal therapy prior to pregnancy should therefore be examined in further studies.

## REFERENCES

- HARRIS MI, NIH Publication, 95 (1995) 1468. — 2. TEUSCHER A, EGGER M, HERMAN JB, Arch Intern Med, 149 (1989) 1942. — 3. LALLA E, LAMSTER IB, DRURY S, FU C, SCHMIDT AM, Periodontol 2000, 23 (2000) 50. — 4. LÖE H, Diabetes Care, 16 (1993) 329. — 5. HUGOSON A, THORSTENSSON H, FALK H, KUYLENSTIERNA J, J Clin Periodontol, 16 (1989) 215. — 6. CUTLER CW, MACHEN RL, JOTWANI R, IACOPANI AM, J Periodontol, 70 (1999) 1313. — 7. THORSTENSSON H, HUGOSON A, J Clin Periodontol, 20 (1993) 352. — 8. CAMPUS G, SALEM A, UZZAU S, BALDONI E, TONOLO G, J Periodontol, 76 (2005) 418. — 9. RYLANDER H, RAMBERG P, BLOHME G, LINDHE J, J Clin Periodontol, 14 (1986) 38. — 10. MOORE PA, WEYANT RJ, MONGELUZZO MB, MYERS DE, ROSSIE K, GUGGENHEIMER J, BLOCK HM, HUBER H, ORCHARD T, J Periodontol, 70 (1999) 409. — 11. CIANCIOLO LJ, PARK BH, BRUCK E, MOSOVICH L, GENCO RJ, J Am Dent Assoc, 104 (1982) 653. — 12. PINSON M, HOFFMAN WH, GARNICK JJ, LITAKER MS, J Clin Periodontol, 22 (1995) 118. — 13. AREN G, SEPET E, OZDEMIR D, DINCAG N, GUVENER B, FIRATLI E, J Periodontol, 74 (2003) 1789. — 14. EMRICH LJ, SHLOSSMAN M, GENCO RJ, J Periodontol, 62 (1991) 123. — 15. ROSENTHAL JM, ABRAMS H, KOPCYK A, J Clin Periodontol, 15 (1988) 425. — 16. COLLIN HL, UUSITUPA M, NISKANEN L, KONTTURI-NARHI V, MARKKANEN H, KOIVISTO AM, MEURMAN JH, J Periodontol, 69 (1998) 962. — 17. LUNDGREN D, MAGNUSSEN B, LINDHE J, Odontol Revy, 24 (1973) 49. — 18. JENSEN J, LILJENMARK W, BLOOMQUIST C, J Periodontol, 52 (1981) 599. — 19. LÖE H, J Periodontol, 36 (1965) 209. — 20. HUGOSON A, J Periodontol Res, 5 (1970) 1. — 21. MIYAZAKI H, YAMASHITA Y, SHIRAMA R, GOTO-KIMURA K, SHIMADA N, SOGAME A, TAKEHARA T, J Clin Periodontol, 18 (1991) 751. — 22. OJANATKO-HARRI AO, HARRI MP, HURTIA HM, SEWON LA, J Clin Periodontol, 18 (1991) 262. — 23. LANGE DE, LÜBBERT H, ALAI-OMID W, Dtsch Zahnarztl Z, 28 (1974) 1239. — 24. LANGE DE, PLAGMANN HC, EENBOOM A, PROMESBERGER A, Dtsch Zahnarztl Z, 32 (1977) 44. — 25. ALBRECHT M, BANOCZY J, GYULA T, Community Dent Oral Epidemiol, 16 (1988) 378. — 26. TWETMAN S, JOHANSSON I, BIRKHED D, NEDERFORS T, Caries Res, 36 (2002) 31. — 27. ALBRECHT M, BANOCZY J, BARANYI E, TAMAS G, SZALAY J, EGYED J, SIMON G, EMBER G, Acta Diabetol Lat, 24 (1987) 1. — 28. GISLEN G, NILSSON KO, MATSSON L, Acta Odontol Scand 38 (1980) 241. — 29. SEPPÄLÄ B, SEPPÄLÄ M, AINAMO J, J Clin Periodontol, 20 (1993) 161. — 30. OLIVER RC, TERVONEN T, J Periodontol, 65 (1994) 530. — 31. SAFKAN-SEPPÄLÄ B, AINAMO J, J Clin Periodontol, 19 (1992) 24. — 32. ERVASTI T, KNUUTTILA M, POHJAMO L, HAUKIPURO K, J Periodontol 56 (1985) 154. — 33. CERDA J, DE LA TORRE CV, MALACARA JM, NAVA LE, J Periodontol 65 (1994) 991. — 34. GUTHMILLER JM, HASSEBROEK-JOHNSON JR, WEENIG DR, JOHNSON GK, KIRCHNER HL, KOHOUT FJ, HUNTER SK, J Periodontol, 72 (2001) 1485. — 35. LEEPER SH, KALKWARF KL, STROM EA, J Oral Med 40 (1985) 127. — 36. BAGDADE JD, STEWART M, WALTERS E, Diabetes, 27 (1978) 677. — 37. UETA E, OSAKI T, YONEDA K, YAMAMOTO T, J Oral Pathos Med, 22 (1993) 168. — 38. SALVI GE, BECK JD, OFFENBACHER S, Ann Periodontol, 3 (1998) 40. — 39. BROWNLEE M, CERAMI A, VLASSARA H, Diabetes Metab Rev, 4 (1988) 437. — 40. SASTROWIJOTO SH, HILLEMANN S, VAN STEENBERGEN TJM, ABRAHAM-INPIJN L, DE GRAAFF J, J Clin Periodontol, 16 (1989) 316. — 41. TAYLOR GW, BURT BA, BECKER MP, GENCO RJ, SHLOSSMAN M, KNOWLER WC, PETTIT DJ, J Periodontol, 67 (1996) 1085.

B. Willershausen

Department for Operative Dentistry, Johannes Gutenberg University, Augustusplatz 2, 55131 Mainz, Germany  
e-mail: willersh@uni-mainz.de

## STATUS PARODONTNE BOLESTI U TRUDNICA SA DIABETES MELITUSOM

### SAŽETAK

Svrha ove studije bila je vrednovanje povezanosti između tipa I diabetes melitusa (DM) i parodontne bolesti u trudnica. Pedeset dvije trudne žene u dobi od 27,9±6,9 godina sa DM tipa I sudjelovale su u ovoj studiji. Četrdeset i dvije žene sa DM tipa I (prosječna dob: 27,9±6,1 godina) i 121 zdrava žena (prosječna dob: 29,1±5,7 godina) bez dijabetesa tvorile su kontrolnu grupu. Svi subjekti bili su obrađeni prema kliničkim parodontološkim parametrima uključujući probnu dubinu džepa, probnu razinu pričvrstka, prisutnost plaka, vrijednosti gingivitisa. Krvni parametri uključivali su razinu hemoglobina, glikolizirani hemoglobin, ukupni kolesterol, trigliceride i leukocite. Trudni dijabetički subjekti pokazali su unatoč dobroj metaboličkoj kontroli statistički značajno veću vrijednost gingivitisa u usporedbi sa kontrolnom grupom. Trudni dijabetički subjekti pokazali su statistički značajnu korelaciju između doze inzulina na dan i probne dubine džepa (p<0,05) kao i gubitka pričvrstka (p<0,05). Zaključno, rezultati ove studije pokazuju kod trudnica sa dijabetesom veći stupanj parodontne upale i destrukcije u usporedbi sa diabetičarkama ne trudnicama i zdravim pacijenticama.