Oral hygiene: the third column of basal diabetes therapy

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Summary

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Purpose
Diabetes is associated with increased prevalence and severity of periodontal inflammations. The aim was to examine the interrelationship among medical/periodontal variables and oral hygiene behaviour of type 2 diabetic and non-diabetic subjects to identify factors that may be predictors for periodontal disease.

Methods:
517 subjects (346 type 2 diabetic, 171 non-diabetic) underwent a clinical periodontal examination and completed an oral hygiene self-questionnaire. Screening parameters included the gingivitis (GI), papillary bleeding (PBI) and visible plaque (VPI) index, periodontal screening and recording (PSR), probing pocket depth (PPD). Anthropometric and diabetes parameters were also measured.

Results
Periodontal health indicators were significantly better in non-diabetics. Diabetic patients had significantly fewer teeth (minus 9.8% or 12.5% in patients with oral or insulin therapy), worse oral hygiene and the GI and PSR scores were significantly higher (GI score: plus 33% and 42% in patients with oral or insulin therapy; PSR score: plus 35% in diabetic patients). The percentage of papillary bleeding was also higher in diabetic patients (58% and 63% in patients with oral or insulin therapy vs. 51% in non-diabetics). The visible plaque index was significantly correlated with periodontal parameters.

Conclusions:
Diabetes is related to increased levels of periodontal inflammation scores, which showed a strong correlation to the plaque. We suggest that the relation between periodontitis and diabetes may have an important link in a minor oral hygiene. Therefore, like diabetes type 2 itself, this relation mainly may be due to behaviour characteristics.

Key words: diabetes, periodontal disease, oral hygiene

Introduction

Commonly, periodontal disease is considered a complication of diabetes. There are however certain risk factors that contribute to the development of diabetes, and as such can be viewed as risk factors for periodontal disease.
Epidemiological studies revealed that obesity is an important risk factor for diabetes, periodontal disease, and cardiovascular disease. Obesity and type 2 diabetes mellitus are associated with many metabolic disorders including dyslipidemia, hypertension and arteriosclerosis [1]. Furthermore diabetes and obesity are presumed to be linked through proinflammatory cytokines, secreted by adipocytes [2]. Studies also indicate a correlation between periodontal inflammation on diabetic balance and insulin resistance syndrome [3-6]. Severe periodontal disease causes attachment, alveolar bone and tooth loss, extending beyond the local level to produce systemic effects. It exacerbates the inflammatory milieu, increases insulin resistance [7, 8], and potentially worsens cardiovascular disease [9,10]. Severe periodontitis is a risk factor for early death due to ischemic heart disease or renal dysfunction independent of diabetes, increasing the risk 3.2 fold as compared to those with none, mild, or moderate periodontitis [11].
Periodontal diseases are bacteria-induced infections that affect the periodontium and result in the loss of tooth attachment. Advanced tooth loss can be associated with a 70% increased odds of incident diabetes [12]. Once

established in the tissue, the periodontal infection may complicate diabetes control and increase the occurrence and severity of microvascular and macrovascular complications [13]. The periodontal pathogen bacteria cause a chronic local low-grade inflammation and contribute to systemic inflammation. It is assumed that higher circulating levels of inflammatory markers such as C-reactive protein, IL-6 and TNF-α, may result [4, 14].

The association between diabetes and periodontal disease may be due to numerous physiological phenomena seen in diabetes, such as impaired immune resistance, vascular changes, altered microflora, and abnormal collagen metabolism. There is a direct causal or modifying relationship in which poor glycemic control results in more severe periodontitis [15-19]. Diabetes mellitus determines changes in bacterial population and production of inflammatory mediators, and reduces the efficacy of the host response.

The aim of the present study was to compare the periodontal status and oral hygiene behaviour of diabetes patients who receive different medical treatments, to non-diabetic, obese and pre-diabetic subjects; and to assess if there is a direct causal relationship between HbA1c or BMI and periodontal inflammation.

Methods

Study population and clinical measurements

517 participants of the Outpatient Ambulance of Sports Medicine were analysed. Measurements of dental health and metabolic state were generally part of the baseline examination before exercise training. Patients were placed in five groups (non-diabetic, obese, pre-diabetic patients, diabetes patients with oral medication, and insulin regimen). All relevant patient data are given in Table 1. For biometry, body mass index (BMI: weight (kg) ÷ height^2 (m)) was categorized as normal (BMI 18.5-24.9), overweight (BMI 25-29), or obese (BMI ≥30) (WHO 1997). All subjects were non-smokers and had a clinical periodontal examination to measure the gingival index (GI), papillary bleeding index (PBI), visible plaque index (VPI), probing pocket depth (PPD), periodontal screening and recording (PSR), number of teeth, and tooth mobility. Subjects who participated in this study had a minimum of five teeth (average mean between 19 and 20). Diabetes due to pregnancy or pancreatitis was an exclusion criterion. The clinical dental examination was conducted by a single dentist to avoid differences in measurements. The depth of the periodontal pockets was measured by probing the sulcus (pocket) of each tooth using a periodontal probe with a colored strip (running from 3.5 to 5.5mm) and a 0.5mm ball at the tip. The probe was inserted into the crevice until resistance was met. Readings were taken at the mesiofacial, midfacial, and distofacial areas, as well as the corresponding lingual or palatal areas. The highest reading for each sextant was then recorded. Each sextant was rated according to the PSR scale of criteria: PSR code 0: health, code 1: gingivitis, code 2: calculus and gingivitis, code 3: chronic periodontitis with early or moderate attachment loss, code 4: chronic periodontitis with moderate attachment loss or a form of aggressive periodontitis. A PSR code 3 is determined when the coloured band of the probe remains partially visible (pockets = 4-5mm). A PSR code 4 implies that the coloured band is not visible (pockets ≥ 5.5mm). The periodontal status was assessed by means of the PSR scoring system. The GI score (Loe and Silness) describes the clinical severity of gingival inflammation as well as its location. The GI scale of criteria is: GI code 0: no bleeding and inflammation, code 1: slight change in color and mild edema with slight change in texture, mild inflammation, code 2: redness, hypertrophy, edema and glazing, bleeding on probing, moderate inflammation, code 3: marked redness, hypertrophy, edema, ulceration, spontaneous bleeding, severe inflammation.

Questionnaire

Data were collected from a self-questionnaire which consisted of 44 questions, using oral health behaviour items such as use of mouth rinses, frequency and time spent on tooth brushing, approximal cleaning, and dental visits.

Statistical analyses

All data are presented as means ± SD. Analysis of groups across time was done using the Mann-Whitney U test. A p-value <0.05 was considered to indicate significance, $p<0.01$ was accepted as very significant and $p<0.005$ as extremely significant.

Results

Baseline characteristics of the participants are shown in Table 1. The obese patients were significantly younger ($p<0.03$) than the patients in the other groups. Mean glycemia was markedly higher in type 2 diabetic subjects with insulin therapy compared to those receiving oral medication ($p<0.0001$). The type 2 diabetes patients had

significantly fewer teeth ($p<0.01$) than the three non-type 2 diabetes groups. Data are also given for cumulated groups (Table 2).

Descriptive analysis of baseline medical data for all groups

	non-diabetic patients	obese	pre-diabetic patients	oral therapy	insulin therapy
N	46	51	74	205	141
Age	59.5 ± 12.3	55.2 ± 9.7	59.1 ± 9.6	59.6 ± 9.5	60.7 ± 9.7
females	65.2%	78.4%	59.5%	52.7%	61.0%
weight	73.1 ± 10.2	103.2 ± 20.6	90.4 ± 16.6	96.8 ± 18.6	96.0 ± 21.0
height	1.67 ± 0.83	1.66 ± 0.76	1.69 ± 0.79	1.69 ± 0.81	1.68 ± 0.98
BMI	26.1 ± 2.4	37.8 ± 7.5	32.7 ± 6.0	33.9 ± 5.9	34.0 ± 6.7
HbA1c			5.82 ± 0.48	6.48 ± 0.80	7.04 ± 1.0
number of teeth	22.7 ± 6.3	22.8 ± 6.1	21.9 ± 6.6	20.2 ± 6.9	19.6 ± 7.6

Table 1. Gender, age (years), weight (kg), height (m), body mass index, percentage of glycated hemoglobin in the blood (HbA1c) (%), and number of teeth of all groups.

Descriptive analysis of baseline medical data for cumulated non-diabetic groups, and diabetic groups with oral respectively insulin therapy

	cumulated non-diabetic patients	oral therapy	insulin therapy
N	171	205	141
age	58.0 ± 10.5	59.6 ± 9.5	60.1 ± 9.7
females	67.0	52.7	61.0
weight	92.4 ± 20.7	96.8 ± 18.6	96.0 ± 21.0
height	1.67 ± 0.80	1.69 ± 0.81	1.68 ± 0.98
BMI	33.0 ± 7.4	33.9 ± 5.9	34.0 ± 6.7
HbA1c		6.48 ± 0.80	7.04 ± 1.0
number of teeth	22.4 ± 6.3	20.2 ± 6.9	19.6 ± 7.6

Table 2. Gender, age (years), weight (kg), height (m), body mass index (BMI), percentage of glycated hemoglobin in the blood (HbA1c) (%), and number of teeth of the cumulated non-diabetic patients, and patients with oral and insulin therapy.

The clinical measurements and the corresponding fraction of patients are given in Table 3. Differences in dental and periodontal parameters between obese, non- and pre-diabetic subjects were not significant, so the data were also given for cumulated non-diabetic groups. Diabetic subjects had significantly fewer teeth, higher GI and PSR score values, and a higher percentage of papillary bleeding than the cumulated non-diabetic group. The significances are given in Table 4. There were no significant differences in periodontal parameters between the diabetes groups despite of different medication or diabetes degree. No significance was found in GI between obese and non-diabetic or pre-diabetic patients.

Descriptive analysis of dental and periodontal parameters

	non-diabetic group n=46	obese group n=51	pre-diabetic group n=74	cumulated non-diabetic groups n=181	oral therapy n=205	insulin therapy n=141
number of teeth	22.7 ± 6.3	22.8 ± 6.1	21.9 ± 6.6	22.4 ± 6.3	20.2 ± 6.9	19.6 ± 7.6
gingival index	1.09 ± 1.07	1.18 ± 1.0	1.08 ± 1.08	1.11 ± 1.0	1.48 ± 1.06	1.58 ± 1.12
mean periodontal screening and recording	1.47 ± 1.84	1.67 ± 1.44	1.48 ± 1.38	1.48 ± 1.42	2.0 ± 1.46	2.01 ± 1.46
visible plaque index	1.61 ± 0.65	1.64 ± 0.75	1.80 ± 0.80	1.70 ± 0.74	2.04 ± 0.75	2.02 ± 0.79
number of mobile teeth	1.87 ± 3.23	2.35 ± 3.26	2.94 ± 4.16	2.49 ± 3.68	2.76 ± 3.61	2.81 ± 3.69
percentage of bleeding interdental sites	49.65±24.62	47.55±24.56	53.31±26.75	50.66±25.54	58.4±27.65	62.71±29.91
mean papillary bleeding code 2	5.38 ± 3.83	5.15 ± 4.29	5.54 ± 4.19	5.38 ± 4.10	6.25 ± 4.76	6.13 ± 4.71
mean papillary bleeding code 3	1.58 ± 2.27	2.17 ± 2.62	1.46 ± 2.40	1.70 ± 2.44	1.91 ± 2.53	2.44 ± 3.43
number of 5mm pockets	5.05 ± 8.29	3.77 ± 6.24	5.23 ± 11.47	4.75 ± 9.37	6.25±10.05	5.36 ± 8.31
number of 6mm pockets	0.95 ± 2.21	0.88 ± 2.04	0.97 ± 2.94	0.90 ± 2.47	1.48 ± 3.55	1.33 ± 2.83

Table 3. Descriptive analysis of dental and periodontal parameters: number of teeth, gingival index (GI), mean score of periodontal screening and recording (PSR), visible plaque index (VPI), number of mobile teeth, percentage of bleeding interdental sites (%), mean papillary bleeding code 2 and 3, number of sites with pocket depth of 5 and 6 mm of all groups.

Significances of periodontal parameters between non-diabetic and diabetic groups

	number of teeth	gingival index	mean periodontal screening and recording	percentage of bleeding interdental sites
oral therapy vs. non diabetic groups				
oral therapy	20.2 ± 6.9	1.48 ± 1.06	2.0 ± 1.46	58.4 ± 27.65
cumulated non-diabetic groups	22.4 ± 6.3	1.11 ± 1.0	1.48 ± 1.42	50.66 ± 25.54
P	p<0.001	p<0.001	p<0.003	p<0.007
insulin therapy vs. non diabetic groups				
insulin therapy	19.6 ± 7.6	1.58 ± 1.12	2.01 ± 1.46	62.71 ± 29.91
cumulated non-diabetic groups	22.4 ± 6.3	1.11 ± 1.0	1.48 ± 1.42	50.66 ± 25.54
P	p<0.001	p<0.0004	p<0.005	p<0.0004
insulin therapy vs. oral therapy				
P	n.s.	n.s.	n.s.	n.s.

Table 4. Significances in number of teeth, gingival index (GI), and mean score of periodontal screening and recording (PSR), and percentage of bleeding interdental sites (%) between cumulated non-diabetic patients and patients with oral or insulin therapy respectively.

Type 2 diabetic subjects had a significantly worse oral hygiene than non-diabetic subjects as measured by the average of visible plaque index (VPI), and interdental cleaning frequency per day (ICF). Diabetic subjects with oral therapy brushed less frequently than non-diabetic subjects. No difference in oral hygiene was found between diabetic subjects (Table 5).

Oral hygiene parameters and significances of non-diabetic and diabetic subjects

Groups	VPI	TBF	TBT	ICF
non-diabetic (n=171)	1.71 ± 0.73	1.93 ± 0.49	5.45 ± 2.37	0.81 ± 0.83
oral therapy (n=205)	2.04 ± 0.75	1.77 ± 0.67	5.18 ± 2.65	0.61 ± 0.8
insulin therapy (n=141)	2.02 ± 0.79	1.87 ± 0.57	5.54 ± 2.89	0.58 ± 0.78
ND vs. OAD	p<0.0001	p<0.05	n.s.	p<0.02
ND vs. insulin	p<0.0008	n.s.	n.s.	p<0.02
OAD vs. insulin	n.s.	n.s.	n.s.	n.s.

Table 5. Visible plaque index (VPI), tooth brushing frequency (TBF), tooth brushing time (TBT), and interdental cleaning frequency (ICF) and the differences between non-diabetic and diabetic subjects with oral or insulin therapy. ND= non-diabetic subjects, OAD= diabetic subjects with oral therapy

Correlation between plaque and periodontal indicators

Visible plaque index (VPI) was significantly correlated with the percentage of bleeding interdental sites (r=0.42, p<0.0001), gingival index (GI) (r= 0.61, p<0.0001), mean periodontal screening and recording (PSR) (r=0.47, p<0.0001), HbA1c (r=0.12, p<0.03), BMI (r= 0.11, p<0.03). No significant correlation was between HbA1c and GI, PSR or percentage of bleeding interdental sites.

Discussion

Numerous studies have demonstrated that individuals with diabetes tend to have a higher prevalence and severity of periodontal disease than non-diabetic subjects. Two recent studies [20,21] compared the periodontal health of non-diabetic and type 2 diabetic subjects. Gingival inflammation and attachment loss were significantly increased in the diabetic patients. To our knowledge this is the first study to no investigate the periodontitis in diabetes with respect to different medical treatments. For an additional evaluation of the inflammatory response in diabetes patients, we compared the periodontal and oral hygiene behaviour of five subgroups (obese, pre- and non-diabetic subjects, type 2 diabetic patients with oral medical treatment, and insulin regimen). GI and PSR values were significantly elevated in diabetic subjects. The major finding is that the diabetic patients had a significantly worse oral hygiene and higher percentage of bleeding interdental sites. The visible plaque index was significantly correlated with GI, PSR, PBT (p<0.0001), BMI and HbA1c (p<0.03). In contrast no correlation was found between HbA1c and periodontal parameters.

Investigations lead by Grossi et al. [5] and Taylor et al. [15] have demonstrated that periodontal disease is associated with impaired fasting glucose levels and an increased demand for insulin, as a consequence of insulin resistance. The metabolic stress of inflammation would tend to shift an euglycemic person toward a pre-diabetic state of type 2 diabetes mellitus. It has been suggested that this metabolic effect is a consequence of systemic LPS, TNF-α, IL-1β, and IL-6, which enhance insulin resistance. Inflammation and bacteria, even at a low systemic level, when repeated acutely and aggravated chronically over years, can theoretically provide severe cumulative damage to systemic health. The metabolic stress induced by inflammation or infection leads to an increase in blood lipid and blood glucose levels and induces a state of insulin resistance. Thus, a chronic inflammation like periodontitis may predispose an individual to increased risk for developing states of metabolic dysregulation and leading to type 2 diabetes. For individuals with diabetes mellitus, the metabolic stress

of periodontal inflammation would worsen glycemic control and increase the need for hypoglycemic agents.

Diabetes mellitus and periodontitis have a relatively high incidence in the general population as well as a number of common pathways in their pathogenesis (both are polygenic disorders with immunoregulatory dysfunction). The altered host inflammatory response in diabetes includes neutrophil dysfunction, abnormal cross-linking, defective secretion of growth factors and subsequent healing. In hyperglycemic states found in diabetes patients, a nonenzymatic glycation and oxidation of proteins and lipids occurs. As a result, advanced glycation end products (AGE´s) accumulate in the plasma and tissues of diabetic subjects. The receptor (RAGE), expressed by multiple cell types including endothelium and mononuclear phagocytes, plays a central role in oral infection, exaggerated inflammatory host responses, and destruction of alveolar bone in diabetes patients. Antagonists of RAGE might have an adjunctive therapeutic role for the management of periodontitis in diabetes patients [22,23]. Chronic sub-clinical inflammation has been declared part of the insulin resistance syndrome, as such inflammatory responses participate in the progression of metabolic disorders like type 2 diabetes [1]. The control of periodontal inflammation is essential in diabetic patients. Diabetes is a disease that frequently causes multiple comorbidities.

The present study has two major aspects:

1. The results confirm the known relationship between diabetes and gingival inflammation.
2. The results show a significant difference in dental hygiene behaviour between non-diabetic and diabetic subjects. The same difference is apparent in gingival inflammation indices. No relevant differences were seen in body weight.

These results strongly suggest that oral hygiene behaviour may be an independent variable of periodontal infection and, thereby, of permanent low grade systemic inflammation. Between the diabetic groups, there was a marked ($p<0.0001$) difference in HbA1c, but no relevant differences in gingival inflammation, so there is at least no marked impact of HbA1c on periodontitis in this study. Type 2 diabetes, on a hereditary basis, is highly influenced by the individual behaviour. Reduction of body weight, frequent training and maintenance of the muscle mass are important determinants of the basic therapy. A consequence of these behavioural components would be sufficient in about 80% of the cases to reduce blood glucose and Hba1c towards normal values. From the present results it strongly appears that oral hygiene is the third column of basic diabetes therapy besides weight management and frequent exercise.

Periodontal disease and other oral pathologies (gingivitis, candidiasis, oral malignancies etc.) are frequent complications of diabetes. An aggressive and lasting management of oral hygiene and health control including early bacterial detection may be an efficient way to decrease periodontal inflammation and positively affect diabetes management and control of periodontal disease with its adverse effects on insulin resistance through the generation of inflammatory cytokines. Because of this relationship, the diabetes specialist should put an emphasis on oral health and its control as an integral part of diabetes treatment. In fact it appears to be part of the diabetes specialist's obligation to monitor oral health. He or she should provide for the early diagnosis of periodontitis using simple screening methods suitable for the office. If a patient shows signs of periodontitis, a specialist for oral diseases, a periodontologist, should perform further diagnostics and take therapeutic steps. Almost all subjects with diabetes require oral and dental evaluation and many of them will develop an oral pathology in the course of their disease that requires intervention.

A close cooperation between the diabetes specialist and the periodontologist is vital to manage the patient's periodontal problems and diminish the inflammatory milieu's detrimental effects on diabetes control and cardiovascular health. Combined, these two disciplines have a greater success in the diagnosis and control of diabetes and periodontitis.

Conclusions

Periodontal indices are significantly related to diabetes and to the insufficient oral hygiene behaviour in diabetic patients. No relation was seen between periodontal indices and HbA1c.

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