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# Pathologies of the oral cavity in patients with non-controlled diabetes type 1 and type 2 — analysis of periodontal status and periodontal treatment needs

## Patologie jamy ustnej u pacjentów z niewyrównaną cukrzycą typu 1 i typu 2 — analiza stanu przyzębia i periodontologicznych potrzeb leczniczych

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**Abstract**

**Introduction:** Decompensated diabetes is a major risk factor in the development of periodontal diseases. This leads to disturbances of carbohydrates, protein, and fat and morphological changes in many organs. These changes also apply to the masticatory system, especially periodontal tissues. The aim of the study was to assess the periodontal status and periodontal treatment needs in patients with non-controlled diabetes type 1 and type 2 ($HbA_{1c} > 7\%$), and to compare the results with the data obtained in a group of generally healthy patients.
**Material and methods:** The study included 275 patients, 155 of them were patients with non-controlled diabetes during hospitalisation (study group), while 120 subjects constituted the control group of healthy people. The study excluded edentulous people. CPITN index (according to Ainamo et al.) was used to assess the periodontal state and periodontal treatment needs.
**Results:** The average level of glycated haemoglobin $HbA_{1C}$ among patients in the study group was 9.43% in women and slightly more at 9.57% in men. The periodontal status in healthy people was satisfactory, dominated by the maximum values of CPITN = 0, CPITN = 1, and CPITN = 2. The study group more frequently revealed the maximum values of CPITN = 3 and CPITN = 4. This shows the more advanced periodontal changes in this group. Due to the bad condition of the periodontium, the periodontal treatment needs proved to be far greater in the study group and related primarily to comprehensive specialist treatment (TN3).
**Conclusions:** Decompensated diabetes may be an important cause of changes in periodontal tissues and may cause a significant loss of masticatory function in patients. **(Endokrynol Pol 2015; 66 (5): 428–433)**

***Key words:*** *oral cavity; periodontium; non-controlled diabetes*

**Streszczenie**

**Wstęp:** Niewyrównana cukrzyca jest jednym z głównych czynników ryzyka w powstawaniu i rozwoju schorzeń tkanek przyzębia. Prowadzi do zaburzeń gospodarki węglowodanowej, białkowej i tłuszczowej oraz zmian morfologicznych w wielu narządach. Zmiany te dotyczą również narządu żucia a przede wszystkim tkanek przyzębia. Celem pracy była ocena stanu przyzębia i periodontologicznych potrzeb leczniczych u pacjentów z niewyrównaną cukrzycą typu 1 i typu 2 ( $HbA_{1c} > 7\%$ ) oraz porównanie wyników badań z danymi otrzymanymi w grupie osób ogólnie zdrowych.
**Materiał i metody:** Badaniem objęto 275 pacjentów, 155 z nich to chorzy z niewyrównaną cukrzycą w trakcie hospitalizacji (grupa badana), zaś 120 osób stanowiło grupę porównawczą ludzi zdrowych. Z badania wykluczono osoby z bezzębiem. Do oceny stanu przyzębia i periodontologicznych potrzeb leczniczych zastosowano wskaźnik CPITN (według Ainamo i wsp.).
**Wyniki:** Przeciętny poziom hemoglobiny glikowanej $HbA_{1C}$ wśród pacjentów z grupy badanej wynosił ogółem 9,43% u kobiet, a u mężczyzn nieznacznie więcej bo 9,57%. Stan przyzębia u osób ogólnie zdrowych był zadowalający, przeważały maksymalne wartości CPITN = 0, CPITN = 1 i CPITN = 2. W grupie badanej natomiast częściej występowały maksymalne wartości CPITN = 3 oraz CPITN = 4, co świadczy o bardziej zaawansowanych zmianach w przyzębiu w tej grupie. Wynikające ze złego stanu przyzębia potrzeby lecznicze okazały się zdecydowanie większe w grupie badanej i dotyczyły przede wszystkim kompleksowego leczenia specjalistycznego (TN3).
**Wnioski:** Niewyrównana cukrzyca może stanowić istotną przyczynę zmian w obrębie tkanek przyzębia i wpływać na znaczną utratę funkcji narządu żucia u osób chorych. **(Endokrynol Pol 2015; 66 (5): 428–433)**

***Słowa kluczowe:*** *jama ustna; przyzębie; niewyrównana cukrzyca*

## Introduction

Among the systemic metabolic diseases affecting the periodontal tissues, diabetes is the most common, especially non-controlled [1].

Diabetes (diabetes mellitus) is considered to be a social disease. According to epidemiological data, from 3 to 6 per cent of the population suffers from it [2]. The World Health Organisation predicts that by 2025 the number of people with diabetes will increase

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to 5.4%, and up to 25% in the population between 65 and 74 years of age [3–5]. The long-term consequences of the disease significantly affect overall morbidity and mortality, and shorten life expectancy by about 30% [2, 6].

Diabetes can be defined as a systemic disease characterised by hyperglycaemia resulting from the defect of insulin secretion or action [6].

There are the following types of diabetes: type 1 diabetes (autoimmunological or idiopathic aetiology), type 2 diabetes and other specific types (e.g. in the course of infectious diseases, chronic pancreatitis, endocrinopathies, genetic diseases, associated with taking certain medications), and gestational diabetes [4, 6].

This disease is characterised by the risk of complications, both acute and chronic [7]. All chronic diabetic complications are likely to have a vascular background. They can be divided into non-specific macroangiopathy and specific microangiopathy. Analysing the vascular changes in diabetes, their impact on the periodontal tissues seems to be indisputable: the narrowing of capillaries and precapillaries hinders the transport of nutrients [1, 8]. The impairment of oxygen diffusion and the elimination of unnecessary metabolites lead to the physiological imbalance and increase the sensitivity to the periodontal damage [9]. According to Matthews [10], chronic complications of diabetes are associated with persistent hyperglycaemia, which results in the formation of advanced glycation end products (AGEs). They act by "arming" the endothelial cells and monocytes, making them more sensitive to stimuli that induce inflammatory mediators. Because, as a result of non-controlled diabetes, AGEs accumulate in plasma and tissues, it can be assumed that the periodontal tissues with the high levels of AGEs have greater vascular permeability, increased loss of collagen fibres, and exhibit accelerated destruction of non-mineralised connective tissue and bone.

In the recent years, more and more importance is attributed to the inflammatory process in the development of chronic complications of diabetes. It is connected with the activation of defence mechanisms — referred to as the acute phase response [11, 12].

Both diabetes and diseases (mainly inflammatory) of the periodontal tissues are chronic disorders of multifactorial aetiology, which play an increasingly important role in the ageing of society and significantly lower the quality of patients' life [8, 11].

The aim of the study was to assess the periodontal status and periodontal treatment needs in patients with non-controlled diabetes type 1 and type 2 ($HbA_{1c} > 7\%$), and to compare the results with the data obtained in a group of generally healthy people.

## Material and methods

The study included 275 patients, 155 of them were patients with non-controlled type 1 diabetes (subgroup I) and type 2 (subgroup II) during hospitalisation — the study group. The control group of healthy people consisted of 120 patients without diabetes or clinically overt comorbidities. Edentulous people were excluded. Patients declared their consent in writing. The study obtained the consent of the Bioethics Committee of the Medical University of Białystok (Nr R-I-002/47/2008). The study was conducted in a dental surgery, under artificial light, and using a dental set and standardised probe (periodondometer WHO). The obtained data was applied to specially designed examination cards. The CPITN index, proposed by Ainamo et al., was used to assess the periodontal status and periodontal treatment needs [13–15). The evaluation was done on a scale of five graded criteria as follows:

0 = healthy periodontium, the patient does not require treatment — TN0

1 = bleeding after gentle probing, oral hygiene instruction is indicated — TN1

2 = tartar (over or subgingival), the additional removal of tartar/scaling is necessary — TN2

3 = depth of the gingival pockets from 3.5 to 5.5 mm, also needs from the point TN2

4 = deepened pockets depth > 5.5 mm, the additional need for a comprehensive periodontal procedures, including surgery — TN3.

The index was evaluated in the six following oral sections (sextets):

17–14, 13–23, 24–27

47–44, 43–33, 34–37

The results were statistically analysed. The study hypotheses were verified using the *t*-Student test for two means and "u" test for two frequencies. Differences for which $p < 0.05$ were statistically significant.

## Results

The structure of both groups is presented in Table I, while the number and percentage of edentulous patients (excluded from the study) is shown in Table II. The average level of glycated $HbA_{1C}$ among patients in the study group was 9.43% in women and slightly higher in men at 9.57% (Table III). In subgroup I it reached an average level of 9.47%, and it was slightly higher in women (9.51%) than in men (9.43%). In subgroup II it was 9.53%; 9.36% in women and 9.68% in men, respectively.

The smallest proportion of patients in the study group, at only 3.6%, showed the maximum value of index CPITN = 0, i.e. healthy periodontium (Table IV).

**Table I.** *The age and gender in the study group and the control group*
**Tabela I.** *Wiek i płeć w badanej grupie i grupie kontrolnej*

| Patient groups | Age | Gender | | | |
|---|---|---|---|---|---|
| | | M | % | F | % |
| Subgroup I | 20–35 years old | 9 | 26.4 | 12 | 33.3 |
| | 36–51 years old | 14 | 41.2 | 10 | 27.8 |
| | 52–67 years old | 11 | 32.4 | 14 | 38.9 |
| Total: | – | 34 | 100 | 36 | 100 |
| Subgroup II | 20–35 years old | 2 | 4.3 | 4 | 10.2 |
| | 36–51 years old | 20 | 43.5 | 17 | 43.6 |
| | 52–67 years old | 24 | 52.2 | 18 | 46.2 |
| Total: | – | 46 | 100 | 39 | 100 |
| Control group | 20–35 years old | 12 | 20.0 | 15 | 25.0 |
| | 36–51 years old | 22 | 36.7 | 23 | 38.3 |
| | 52–67 years old | 26 | 43.3 | 22 | 36.7 |
| Total | – | 60 | 100 | 60 | 100 |

**Table II.** *The number and percentage of toothless individuals in the study group and the control group*
**Tabela II.** *Liczba i odsetek osób bezzębnych w grupie badanej i w grupie kontrolnej*

| Toothlessness | | Subgroup I | | | Subgroup II | | | Control group | | | Study group (total) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | M | F | Total | M | F | Total | M | F | Total | M | F | Total |
| Incidence | n | 10 | 8 | 18 | 16 | 10 | 26 | 9 | 6 | 15 | 26 | 18 | 44 |
| | % | 29.4 | 22.2 | 25.7 | 34.8 | 25.6 | 30.6 | 15.0 | 10.0 | 12.5 | 32.5 | 24.0 | 28.4 |
| None | n | 24 | 28 | 52 | 30 | 29 | 59 | 51 | 54 | 105 | 54 | 57 | 111 |
| | % | 70.6 | 77.8 | 74.3 | 65.2 | 74.4 | 69.4 | 85.0 | 90.0 | 87.5 | 67.5 | 76.0 | 71.6 |
| Total | n | 34 | 36 | 70 | 46 | 39 | 85 | 60 | 60 | 120 | 80 | 75 | 155 |
| | % | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 |
| | | | | | | Statistical analysis: | | | | $p < 0.0015$ | | | |

**Table III.** *The average level of glycated haemoglobin $HbA_{1c}$ in the study group (depending on gender)*
**Tabela III.** *Przeciętne stężenie hemoglobiny glikowanej $HBA_{1c}$ w grupie badanej (w zależności od płci)*

| $HBA_{1c}$ | Subgroup I | | | Subgroup II | | | Study group (total) | | |
|---|---|---|---|---|---|---|---|---|---|
| | M | F | Total | M | F | Total | M | F | Total |
| Number | 34 | 36 | 70 | 46 | 39 | 85 | 80 | 75 | 155 |
| Standard deviation | 1.15 | 1.58 | 1.38 | 1.86 | 1.3 | 1.63 | 1.49 | 1.44 | 1.52 |
| Median | 9.7 | 9.8 | 9.8 | 9.7 | 9.6 | 9.6 | 9.7 | 9.6 | 9.6 |
| Mean value | 9.43 | 9.51 | 9.47 | 9.68 | 9.36 | 9.53 | 9.57 | 9.43 | 9.5 |

The proportion of these patients was similar in subgroup I — 3.8% and subgroup II — 3.4%. In the control group, theses patients accounted for 17.1%. The maximum value of CPITN = 1 was found in 11.7% of patients with non-controlled diabetes (total) and in 23.8% of cases in the control group.

Subgroup I contained 19.2% of such patients and subgroup II far fewer at only 5.1%. People with CPITN

**Table IV.** *The number and percentage of patients with maximum values of CPITN index in the study group and the control group*
**Tabela IV.** *Liczba i odsetek osób z maksymalnymi wartościami wskaźnika CPITN w grupie badanej i w grupie kontrolnej*

| Max. CPITN | | Subgroup I | | | Subgroup II | | | Control group | | | Study group (total) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | M | F | Total | M | F | Total | M | F | Total | M | F | Total |
| 0 | n | 1 | 1 | 2 | 0.0 | 2 | 2 | 4 | 14 | 18 | 1 | 3 | 4 |
| | % | 4.2 | 3.6 | 3.8 | 0.0 | 6.9 | 3.4 | 7.8 | 25.9 | 17.1 | 1.9 | 5.3 | 3.6 |
| 1 | n | 5 | 5 | 10 | 0.0 | 3 | 3 | 13 | 12 | 25 | 5 | 8 | 13 |
| | % | 20.8 | 17.9 | 19.2 | 0.0 | 10.3 | 5.1 | 25.5 | 22.2 | 23.8 | 9.3 | 14.0 | 11.7 |
| 2 | n | 5 | 8 | 13 | 1 | 4 | 5 | 16 | 7 | 23 | 6 | 12 | 18 |
| | % | 20.8 | 28.6 | 25.0 | 3.3 | 13.8 | 8.5 | 31.4 | 13.0 | 21.9 | 11.1 | 21.1 | 16.2 |
| 3 | n | 8 | 7 | 15 | 13 | 12 | 25 | 10 | 15 | 25 | 21 | 19 | 40 |
| | % | 33.3 | 25.0 | 28.8 | 43.3 | 41.4 | 42.4 | 19.6 | 27.8 | 23.8 | 38.9 | 33.3 | 36.0 |
| 4 | n | 5 | 7 | 12 | 16 | 8 | 24 | 8 | 6 | 14 | 21 | 15 | 36 |
| | % | 20.8 | 25.0 | 23.1 | 53.3 | 27.6 | 40.7 | 15.7 | 11.1 | 13.3 | 38.9 | 26.3 | 32.4 |
| Total: | n | 24 | 28 | 52 | 30 | 29 | 59 | 51 | 54 | 105 | 54 | 57 | 111 |
| | % | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 |

**Table V.** *The needs for periodontal treatment in the study group and the control group*
**Tabela V.** *Potrzeby lecznicze przyzębia w grupie badanej i w grupie kontrolnej*

| Code of needs | | Subgroup I | | | Subgroup II | | | Control group | | | Study group (total) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | M | F | Total | M | F | Total | M | F | Total | M | F | Total |
| TN0 | n | 1 | 1 | 2 | 0.0 | 2 | 2 | 4 | 14 | 18 | 1 | 3 | 4 |
| | % | 4.2 | 3.6 | 3.8 | 0.0 | 6.9 | 3.4 | 7.8 | 25.9 | 17.1 | 1.9 | 5,3 | 3.6 |
| TN1 | n | 5 | 5 | 10 | 0.0 | 3 | 3 | 13 | 12 | 25 | 5 | 8 | 13 |
| | % | 20.8 | 17.9 | 19.2 | 0.0 | 10.3 | 5.1 | 25.5 | 22.2 | 23.8 | 9.3 | 14.0 | 11.7 |
| TN2 | n | 13 | 15 | 28 | 14 | 16 | 30 | 26 | 22 | 48 | 27 | 31 | 58 |
| | % | 54.2 | 53.6 | 53.8 | 46.7 | 55.2 | 50.8 | 51.0 | 40.7 | 45.7 | 50.0 | 54.4 | 52.3 |
| TN3 | n | 5 | 7 | 12 | 16 | 8 | 24 | 8 | 6 | 14 | 21 | 15 | 36 |
| | % | 20.8 | 25.0 | 23.1 | 53.3 | 27.6 | 40.7 | 15.7 | 11.1 | 13.3 | 38.9 | 26.3 | 32.4 |
| Total: | n | 24 | 28 | 52 | 30 | 29 | 59 | 51 | 54 | 105 | 54 | 57 | 111 |
| | % | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 |
| | | | | | | | | | TN0 | 0.0010 | | | |
| | | | | | Statistical analysis: | | | | TN1 | 0.0196 | | | |
| | | | | | | | | | TN3 | 0.0009 | | | |

= 2 accounted for 16.2% of the study group and 21.9% of the control group. Subgroup I contained 25% of such patients and subgroup II 8.5%. The maximum value of CPITN = 3 was found in 36% of patients in the study group (total) and in 23.8% of the control group. Subgroup I contained 28.8% of such patients and subgroup II much more at 42.4%. CPITN index = 4 occurred in as many as 32.4% of patients in the study group (in total) and in 13.3% of patients in the control group. Subgroup I accounted for 23.1% and subgroup II — 40.7%. The highest detected value of the index among all evaluated groups of teeth determined the choice of a specific category of treatment needs (Table V). It may be noted that only 3.6% of patients in the study group (total) did not require treatment (TN0), including subgroup I — 3.8% and subgroup II — 3.4%. In the control group the percentage of these subjects was 17.1% and the difference showed statistical significance ($p < 0.0010$) in relation to the study group. The category of TN1 treatment needs was found in the study group in 11.7%, while in the control group in 23.8% ($p < 0.0196$). This category of needs occurred three times more often in

subgroup I than II and amounted to 19.2% and 5.1%, respectively. The second category of treatment needs (TN2) concerned the greatest number of people, both in the study group (total) — 52.3% and in the control group — 45.7%. The removal of dental deposits was required by a similar proportion of patients in both subgroups. The comprehensive treatment of the periodontium (TN3) was needed by as many as 32.4% of patients in the study group, and in 13.3% ($p < 0.0009$) of the generally healthy subjects. The number of such patients was almost twice as high in subgroup II — 40.7% than in subgroup I — 23.1%.

## Discussion

Many cross-sectional, long-term studies and illustrative works describe the adverse effects of diabetes on the development and severity of periodontal diseases [16]. These studies were carried out, however, in terms of narrowly formulated problems. The selection of indicators to assess the periodontal status also had a diverse nature. According to Soskolne et al. [17], the dynamics of periodontal pathologies in diabetes depends on many factors, primarily on the degree of diabetic control and the presence of vascular complications. Long-term measure of diabetes control is via the determination of the glycated haemoglobin concentration. Numerous studies indicate a significant correlation between the parameters of diabetic metabolic control and the development of chronic microvascular and macrovascular complications [9, 18, 19]. The severity of periodontal diseases in diabetes may be caused by an increase of collagenase activity, which leads to the prevalence of gingival destruction, immune disorders, and local and general intensive development of oral bacterial flora, particularly Gram-negative bacteria of the genus: Bacteroides, Fusobacterium, and Capnocytophaga. These bacteria may secondarily reduce the function of neutrophils by producing leucotoxins and thereby cause inflammation [1, 2, 19]. These factors probably all contribute to a more advanced and rapidly extending periodontal disease in people with non-controlled diabetes compared to healthy subjects [20, 21]. In addition, microangiopathy of the gingival blood vessels impairs the distribution of oxygen, the removal of waste products, and the leukocyte migration. Thus, it can be assumed that these factors impair the possibilities to repair and regenerate periodontal tissues in patients with diabetes.

Many publications describe the presence of the pathological periodontal pockets in patients with diabetes mellitus and the frequent loss of epithelial attachment compared to the healthy population [10]. According to Iacopino [22], this causes a reduction in the periodontal adaptability, creating conditions for more frequent infections, both specific and non-specific. The above changes are favoured by local metabolic disorders and abnormal impaired macrophage chemotaxis of neutrophils. A significantly greater increase in periodontal diseases was observed in patients with diabetes type 1 compared with type 2 [17, 18] although Saito et al. [23] demonstrated a greater risk factor for these diseases in diabetes type 2. The assessment of the periodontal therapeutic needs of patients with diabetes suggests that the range is much greater than in generally healthy people, as shown by the values of CPITN index. This is confirmed by other authors. Rodrigues et al. [24] believe that the scope and nature of the needs in the treatment of periodontitis significantly distinguished patients with diabetes compared with generally healthy people, especially in terms of the removal of dental deposits. Other authors have shown instead a relatively frequent need for the surgical treatment of periodontal disease in diabetic patients [25, 26]. They stress the need to maintain constant metabolic control and absolute oral hygiene. Many researchers point out the great need for comprehensive treatment of periodontal disease in diabetics, including surgery — category TN3 [26–28]. The results of most authors are therefore consistent with the results of our studies and confirm the previous suggestions about the close relationship between non-controlled diabetes and inflammatory processes of the periodontal tissues.

## Conclusions

1. Patients with uncontrolled diabetes had significantly higher maximum value of CPITN = 3 and CPITN = 4. This was confirmed by the more advanced periodontal changes in this group, often requiring complex periodontal procedures together with surgery (category TN3).
2. Decompensated diabetes may be an important cause of changes in periodontal tissues and may cause a significant loss in masticatory function.
3. There is a need for close cooperation between diabetologists and dentists in order to use the most appropriate means and methods of treatment and significantly improve the quality of patient's life.

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