Dietary deficiencies in patients with periodontal disease – preliminary data

Niedobory żywieniowe u pacjentów ze zdiagnozowaną chorobą przyzębia – badania wstępne

Małgorzata Woźniewicz [1/], Joanna Bajerska [1/], Anna Kurhańska-Flisykowska [2/], Janina Stopa [2/], Jan Jeszka [1/], Anna Kowalska [3/]

[1/] Katedra Higieny Żywienia Człowieka, Uniwersytet Przyrodniczy w Poznaniu
[2/] Katedra Stomatologii Zachowawczej i Periodontologii, Uniwersytet Medyczny im. Karola Marcinkowskiego w Poznaniu
[3/] Instytut Genetyki Człowieka, Polska Akademia Nauk, Poznań

Cel badań. Identyfikacja czynników żywieniowych sprzyjających powstawaniu chorób przyzębia.

Materiał i metody. W badaniach wzięło udział 10 pacjentów ze zdiagnozowaną (wg kryteriów IWC, 1999) chorobą przyzębia – P (w wieku 40±15 lat). U dwóch trzecich badanych rozpoznano chroniczną postać periodontozy, natomiast u jednej trzeciej obserwowano agresywną postać tego schorzenia. Pozytywną grupę kontrolną (K) stanowiło 10 pacjentów w wieku 37±11 lat, leczonych zachowawczo w działającej przy Centrum Stomatologii Uniwersytetu Medycznego w Poznaniu Poradni Stomatologicznej. Negatywna grupa kontrolna (NK) obejmowała 10 ogólnie zdrowych osób nie leczonych stomatologicznie w wieku 41±16 lat. Oceny sposobu żywienia pacjentów dokonano za pomocą wywiadu z ostatnich 24 h. Zdolność racji pokarmowej do przeciwdziałania procesom wolnorodnikowym wyrażono w jednostkach ORAC (oxygen radical absorbance capacity).

Wyniki. Istotnie niższą podaż witaminy C jak i kwasu foliowego oraz potencjał antyoksydacyjny racji pokarmowych obserwowano w grupie P w porównaniu z nie leczoną stomatologicznie grupą kontrolną. Różnice te nie ujawniły się w przypadku grupy pacjentów leczonych zachowawczo. W grupie pacjentów cierpiących na choroby przyzębia obserwowano istotnie wyższy odsetek osób nie spełniających zaleceń żywieniowych odnośnie podaży w diecie wapnia oraz witaminy B_1.

Wniosek. Stan zapalny tkanek przyzębia może dodatkowo pogłębiać niedobory pokarmowe w aspekcie istotnych dla rozwoju tej choroby składników pokarmowych.

Słowa kluczowe: *choroby przyzębia, składniki odżywcze, pojemność antyoksydacyjna*

Aim. To determine the dietary factors associated specifically with periodontal disease.

Material & Methods. Ten clinical patients (aged 40±15 y) with periodontal disease (PP), diagnosed according to the criteria of the IWC (1999), were recruited to the study. Two thirds of the PP group had the chronic type of periodontal disease, and one third had the aggressive type. As a positive control group (CDT), 10 patients, aged 37±11 y, undergoing another dental treatment were recruited randomly. The negative control group (CwDT) included 10 healthy subjects without dental treatment, aged 41±16 y. Food consumption data were collected by 24-hour recall and on this basis nutrient intakes and the antioxidant capacity of the diets (ORAC score) were calculated.

Results. The vitamin C and folate intake, as well as the total antioxidant capacity of the diet, was significantly lower in the PP group than in the CwDT individuals, although not significantly lower than among the CDT patients. There was also a higher number of PP subjects who did not meet dietary recommendations on the intake of calcium and vitamin B_1. This insufficiency of nutrients was more pronounced in PP patients than in CDT patients.

Conclusion. The nutrient supply in the PP group differs significantly from that of the CwDT individuals, but not from other dental patients (CDT), although periodontal patients appear to be more susceptible to nutritional insufficiency.

Key words: *periodontal disease, nutrients, antioxidant capacity*

© **Probl Hig Epidemiol 2012, 93(2): 445-451**

www.phie.pl

Nadesłano: 15.04.2012
Zakwalifikowano do druku: 21.05.2012

Adres do korespondencji / Address for correspondence
Dr hab. Anna Kowalska
Instytut Genetyki Człowieka PAN
32 Strzeszyńska, 60-479 Poznań
tel. 61 65 79 217, e-mail: annkowal@rose.man.poznan.pl

Introduction

Periodontal disease (periodontitis) is a complex chronic inflammatory disease of the periodontium, which results in the loss of supporting connective tissue and alveolar bone. Advanced forms of periodontal disease are characterized by severe inflammation extending deep into the tissues of the periodontium. The process eventually leads to the loss of teeth. Periodontopathies are highly prevalent and, according to the WHO, affect human populations worldwide at prevalence rates of up to 20% [1,2,3]. Aggressive periodontitis (AgP) is the most severe form of

periodontopathy, characterized by a rapid progression and a particularly early age of onset (generally 35 years). Chronic periodontitis (CP) is one of the most common forms of periodontopathy, and the major cause of tooth loss in adults over 40 years of age [4]. Periodontopathies generally have been shown in epidemiological studies to have co-morbidity with coronary heart disease, diabetes, renal disease and dementia [5,6,7]. There is increasing evidence of a relationship between periodontitis and several nutrition-linked chronic conditions, such as obesity, dyslipidaemia and metabolic syndrome [8].

Major periodontal pathogens (*Porphyromonas gingivalis* and *Aggregatibacter actinomycetemcomitans*) induce a cascade of proinflammatory alterations leading to the development of chronic systemic inflammation which is considered to be involved in the initiation and progression, e.g. atherogenesis [9,10] and even possible neurodegeneration [11,12,13]. Periodontal disease is caused by an unbalanced reaction of the immune system to environmental and behavioral factors [3]. Pathogenic oral microflora is the major agent for the development of periodontitis, but poor daily hygiene, smoking, and an inadequate diet are the most important factors strongly contributing to the risk of oral disease [3,14].

There is some evidence to suggest that the periodontal disease progresses more rapidly in malnourished populations [14]. Malnutrition exacerbates the severity of oral infections (e.g. acute necrotizing ulcerative gingivitis) [15]. Nutrition is an important factor in maintaining an adequate host immune response, which can be disturbed in chronic systemic inflammation [5]. Periodontal disease is also associated with an increased production of reactive oxygen species which, if not buffered sufficiently, may cause damage to the host cells and tissues [2]. Antioxidant nutrients, e.g. ascorbic acid, beta-carotene and alpha-tocopherol, are well-known scavengers of reactive oxygen species and could play a certain role in the prevention of periodontitis and in restraining its progress [2]. Some studies also have reported that the decreased intake of vitamin C and calcium is linked to periodontal disease [16,17,18,19], and that the consumption of whole-grain foods, as well as foods containing lactic acid, has a prophylactic effect on periodontal disease [17].

The etiology of periodontal disease is complex, and many environmental and genetic or epigenetic factors are involved in the pathogenesis of both aggressive and chronic forms of periodontiotis. Although the role of the diet and nutritional factors in pathogenesis of periodontiotis is still unclear, it is known that the defense mechanisms of the gingival tissues and saliva can be affected by nutritional status.

Aim

The aim of this study is to recognize the dietary factors specifically associated with periodontal disease. We hypothesize that there are some specific nutrient deficiencies in periodontal patients' diets, and that their dietary intake of some nutrients differs from non-periodontal patients and healthy individuals. The results of this preliminary study should offer tentative information on the possible role of nutritional deficiencies in developing further more advanced investigations on the dietary background to the periodontal disease risk.

Material and Methods

Subjects characteristics

Patients with dental diseases were recruited from the Department of Conservative Dentistry and Periodontology at the Poznań University of Medical Sciences. The study was approved by the Ethics Committee of the Poznań University of Medical Sciences. The patients were diagnosed according to the criteria of the IWC 1999 International Workshop for a Classification of Periodontal Diseases and Conditions [4]. Detailed medical, oral, and family histories of each patient were taken, followed by a complete periodontal examination and radiographs. A clinical diagnosis of chronic periodontal disease (PP) was made for 10 patients (6 females and 4 males) with average age of 40±15 years (Tab. I). Two thirds of the periodontal patients (73%) had the chronic type of periodontal disease, and one third (27%) had the aggressive type.

A positive control group was used, consisting of 10 unrelated healthy individuals, 6 females and 4 males, 37±11 years of age, who lacked symptoms of periodontal disease and had been recruited from among the patients undergoing other dental treatment. The subjects for sampling were selected at random from among the individuals scheduled for a routine oral examination. A negative control

Table I. Background data for subjects in PP, CDT and CwDT groups *

Parameter	Periodontitis patients (PP)	Control group with dental treatment (CDT)	Control group without dental treatment (CwDT)
Female	6	6	6
Male	4	4	4
Age [y]	40±15	37±11	41±16
Height [cm]	167±4	174±13	174±13
Body weight [kg]	64.5±9.2	69.0±12.6	69.0±12.6
Body mass index [kg/m^2]	23.2±2.6	22.8±3.4	22.8±3.4
Chronic type [%]	73	–	–
Aggressive type [%]	27	–	–

* Data are mean ± SD

group included 10 healthy subjects not undergoing dental treatment (CwDT): 6 females and 4 males of ages 41±16 years. These individuals were recruited randomly from among those who responded to a media advertisement, having excluded those with dental diseases by means of an oral examination.

The data concerning body weight, height, tobacco use, and consumption of sweetened drinks was obtained by self-report. The prevalence of smoking was 20%, 10%, and 30% in PP, CDT, and CwDT groups, respectively. Respectively 60%, 50%, and 40% of individuals from the PP, CDT, and CwDT groups stated that they consumed sweetened drinks at least once a day. All participants of the study were inhabitants of Poznań.

Dietary intake

The evaluation of dietary status was carried out by the 24-hour dietary recall method. Participants were asked to remember and report all food and beverages consumed in the previous 24 hours. The interviewer questioned in detail about each item reported, the place and time of consumption, method of preparation, amount consumed, recipes used, and whether the food was a take-away or some other food eaten outside home. During the interview the participants were shown a photographic album of food products and meals as a visual aid in estimating the amount consumed. The amounts were measured using household units (glasses, cups, tablespoons, slices, etc.). The energy content, as well as the nutritional value of daily food rations, were calculated on the basis of food composition tables [20] using the computer software package „Dietetyk 2" (Food and Nutrition Institute & A. Mięgoć, 1997/2001). The daily dietary nutrient intake was compared with the recommendations of the Food and Nutrition Institute of 2008 [21]. The calculated energy and nutritional contents were reduced by 10%, with the exception of vitamins A, B_1, B_2, vitamins C, which were reduced by 30%, 25%, 20%, and 50%, respectively. Food intakes were recorded for three days each week (two weekdays and one weekend day).

Antioxidant capacity of the diets

To determine the antioxidant capacity of the diets, the ORAC (Oxygen radical absorbance capacity) score method was used [22]. In this method, the total ORAC values reported in micromole Trolox equivalents (TE) per 100 gram sample for plant foods (fruits, vegetables, nuts, seeds, spices, grains, etc.) [23] were summed up. The recommendation for ORAC unit ingestion is about 5,000 units per day [23].

Statistical Methods

The obtained results, expressed as mean ± standard deviation (SD), were analyzed statistically using the Statistica Version 8.0 software. The statistical analysis was performed using a one-way ANOVA test with post-hoc Tukey tests for pairwise comparisons between groups. When a significant F ratio was obtained, Tukey's HSD was used to locate the differences between the means. The values used in the statistical analysis were age and gender-adjusted. The chi-square analyses were calculated on the differences in the distribution of the number of patients with the adequate and inadequate nutrient intake levels. The differences were considered significant at $p<0.05$.

Results

The results of our preliminary studies on a small sample of periodontitis patients (mostly with the chronic type) were compared to other dental patients from the same dental clinic, and to a healthy group of individuals (Tab. II). Generally, in all the examined groups the diet compositions were inadequate, and did not meet nutritional guidelines. This manifested as an excess of energy obtained from fat, SFAs, and saccharose in more than half the subjects, while adequate PUFA intake was found in only 30-40% of the participants. Nearly 90% of the subjects received too little vitamin D (from 2.8 to 3.6 μg/day) from their daily meals. A low intake of dietary fiber was observed in the majority (60%) of patients from the control group with dental treatment (CDT).

The periodontitis patients (PP) received on the average less calcium (682±236 mg/day) than the patients from the control groups with dental treatment (CDT: 737±233 mg/day) and without dental treatment (CwDT: 890±218 mg/day), but the differences between the means were not significant. However, all the PP individuals manifested calcium intakes below the recommended level, in comparison to the CDT (70%) and CwDT (60%) groups; the significance of the difference in the distribution of the results was confirmed by the chi square test ($p<0.05$). The between-group diversity was also indicated in the case of vitamin B_1, folate, and vitamin C. Although the mean level of vitamin B_1 intake (1.1±0.5 mg/day) was sufficient for the PP patients, 80% of them did not meet the recommended intake in comparison to 50% and 10% in CDT and CwDT, respectively, and in consequence it was demonstrated by a significant ($p<0.05$) difference in the distribution of the results between groups. The diets of both the PP and CDT group provided especially low amounts of folate (162±37 and 158±32 μg/day, respectively), but in the CwDT group, the intake of this vitamin (212±62 μg) was significantly higher ($p<0.05$).

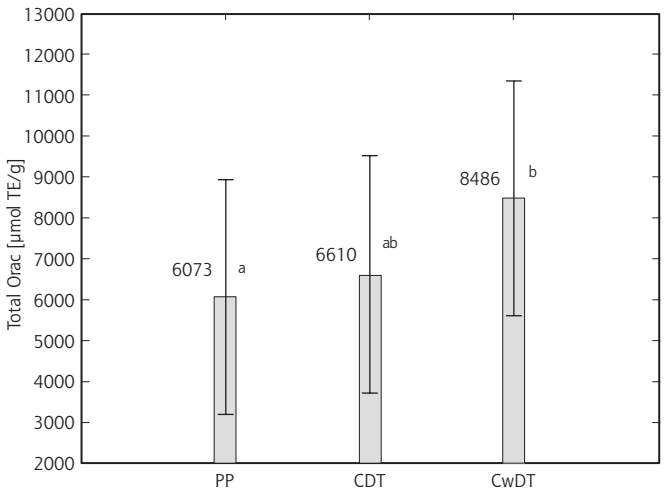
Table II. Nutrient intake in the daily diets of periodontitis and control subjects

Dietary components	Periodontitis patients (PP) N=10		Control group with dental treatment (CDT) N=10		Control group without dental treatment (CwDT) N=10	
	intake[1]	number below/above[2] recommendation	intake[1]	number below/above[2] recommendation	intake[1]	number below/above[2] recommendation
Energy (kcal)	1961±282	6	1780±373	8	2156±388	7
(MJ)	8.2±1.2	–	7.5±1.6	–	7.5±1.6	–
Protein [g]	69±12	2	66±16	2	76±17	2
Fat [g]	69±16	4	67±20	5	84±25	4
Carbohydrate [g]	266±59[ab 6]	–	227±68[a]	–	297±68[b]	–
Energy from [%]:						
Protein	14±2	0	15±2	0	14±2	0
Fat	32±7	6	34±8	6	34±6	7
SFAs[3]	12±3	6	14±3	8	13±3	8
PUFAs[4]	6±2	6	5±1	7	5±1	7
Carbohydrate	54±8	1	51±9	2	56±7	0
Saccharose	12±4	7	11±5	6	13±5	6
Cholesterol [mg]	218±104	2	197±80	2	270±122	6
Dietary fiber [g]	2±4	2	20±4	6	24±6	3
Minerals						
Ca [mg]	682±236	10*	737±233	7*	890±218	6*
P [mg]	1230±164	0	1292±197	0	1378±270	0
Mg [mg]	293±58	7	322±112	4	333±67	4
Fe [mg]	12±6	4	10±3	5	12±3	5
Zn [mg]	11±2	1	11±2	1	12±2	0
Vitamins						
A [µg][5]	1055±704	3	837±522	6	1012±746	6
D [µg]	3.4±0.8	9	2.8±1.4	9	3.6±1.4	9
E [mg]	9.2±2.4	3	6.8±1.2	8	9.2±3.4	5
B_1 [mg]	1.1±0.5	8*	1.0±0.3	5*	1.4±0.3	1*
B_2 [mg]	1.5±0.9	1	1.2±0.2	3	1.8±1.1	1
PP [mg]	16.4±4.7	2	13.4±4.7	6	17.0±7.3	4
B_6 [mg]	1.8±0.4[ab]	2	1.5±0.4[a]	2	2.0±0.4[b]	0
Folate [µg]	162±32[a]	10	158±37[a]	10	212±62[b]	10
B_{12} [µg]	2.7±1.3	4	2.5±0.5	3	2.7±1.1	4
C [mg]	27±17[a]	10*	48±35[ab]	8*	62±32[b]	5*

[1] Data are means±SD (range); [2] number of patients with nutrient intake below the nutritional recommendation – except in the case of cholesterol, percentage of energy from fat, saccharose and SFAs, for which number of patients with excessive intake is given; [3] saturated fatty acids; [4] PUFAs – polyunsaturated fatty acids; [5] vitamin A as retinol equivalents; [6] means for subgroups not sharing a common letter are significantly different at the $p<0.05$ level; * differences in chi-square distribution (the number of patients with adequate/inadequate nutrient intake level) between subgroups are significant at the $p<0.05$ level.

Despite this, none of subjects in any of the examined groups received adequate levels (400 µg/day) of folate. Moreover, the supply of vitamin C in the PP and CDT groups was also insufficient, with average intakes ranging from 27 to 48 mg/day, yet only in PP patients was the level significantly lower ($p<0.05$) than in the CwDT group (62±32 mg). None of the periodontal patients met the recommended intake for this vitamin, and in two control groups 50-80% of the subjects had deficient vitamin C intakes, which gave significant ($p<0.05$) differences in the distribution of the results between the groups.

The antioxidant capacities of the subjects' diets are presented in Figure 1. The mean antioxidant capacity of daily diets was 6073±3604, 6610±3023, and 8486±6058 µmol TE/g for PP, CDT and CwDT groups, respectively. A significant difference in the antioxidant capacity was observed only between the diets of the PP and CwDT groups ($p<0.05$). There was also a significant difference in the distribution of the number of individuals showing inadequate antioxidant supply in the examined diet, as verified by the chi square test ($\chi^2=11.27$, $p<0.01$).



Data are means ± SD (range); means for subgroups not sharing a common letter are significantly different on the level of $p<0.05$; The numbers of patients with ORAC score intake below the recommendation level were 6, 0, 4, in the PP, CDT and CwDT groups, respectively; the differences in chi-square distribution (the number of patients with adequate/inadequate ORAC level) between subgroups are significant at the $p<0.01$ level (chi-square = 11.27).

Fig. 1. Antioxidant capacity of subjects' diets, expressed as total ORAC value [µmol TE/g]

Discussion

Poor diet can act as a factor negatively influencing oral health – and inversely, the conditions connected with oral diseases, such as oral pain syndromes, periodontal inflammation, and oral mucosal diseases, can have an impact on nutrition and dietary pattern [4]. In our study, three groups of patients – periodontal patients, patients with other dental treatment, and healthy individuals – were examined in order to find nutritional deficiencies. There was no significant difference in energy intake, energy supply from the main macronutrients, level of dietary cholesterol or fiber between the examined groups. The diets of the majority of individuals, independently of oral health status, were deficient in vitamin D and dietary fiber, with a high percentage of energy from SFAs and saccharose, and a low percentage of energy from PUFAs. The between-group diversity in dietary intake concerns calcium and micronutrients.

All the PP patients, but only 60-70% of CDT and CwDT individuals, failed to meet the dietary calcium recommendation. The PP patients' diets were also more frequently deficient in vitamins B_1 and C. Furthermore, the level of folate supplied in the diets was decreased in both the PP and CDT groups. However, the current model of our preliminary study does not give a clear answer to the question of whether poor nutrition is one of the causes of periodontitis, or if oral ailments (oral pain syndromes) are responsible for the patients avoiding some foods (for example: fresh fruit and vegetables, whole grain bread etc.), and in consequence receiving inadequate nutrition. Nevertheless, deficient nutrition would be a factor accelerating the disease progress. There are some data indicating the positive influence of certain nutrients on oral health. Morillo et al. [8] suggest that omega-3 fatty acids (ω-3 PUFAs), vitamin C, and a diet rich in vegetables and fresh food appear to be favorable to better periodontal health, whereas diets rich in fat, especially in saturated fatty acids (SFAs) may be harmful to the periodontal tissue condition. It has been found that omega-3 PUFAs also inhibit lipid mediators of inflammation (such as prostaglandin E_2, arachidonic acid, 5-lipoxygenase and cyclooxygenase), modulate lymphokine production, and increase antioxidant capacity [24, 25, 26], and are also reported to decrease osteoclast activity [27]. Moreover, Garcia et al. [28] have shown that one year of calcium ($\geqslant$1000 IU/day) and vitamin D supplementation ($\geqslant$400 IU/day) had a modest positive effect on periodontal health in patients of dental clinics. Neiva et al. [29] indicated some benefits from vitamin-B complex supplementation (including vitamin B_1) on periodontal wound healing, but there are no reports on potential relation between vitamin B_1 intake, as an individual nutrient, and periodontitis. A protective effect against dental disease is also attributed to foods which stimulate salivary flow, such as wholegrain foods and peanuts [2]. Those foods are often good sources of polyphenols, another factor which may determine the intensity of development of periodontal disease [30]. The polyphenols have been shown to possess a significant antioxidant activity towards free radicals – a factor increasing oxidative stress.

A disequilibrium between oxidative stress and the antioxidant capacity of the organism leads to oxidative injury of both cells and tissues. Chronic inflammatory processes involved in periodontitis induce oxidative stress, escalated by the low antioxidant capacity of the organism [31]. The latter condition may be caused by a number of factors, including smoking and poor nutritional status related to the dietary supply of antioxidants. Therefore, diets rich in antioxidants (such as vitamins C and E, as well as other bioactive components such as polyphenols) might inhibit periodontal disease development and progression, particularly in subjects exposed to other external factors such as smoking, diabetes, obesity, metabolic syndrome, and dietary sources of oxidative stress [30].

In our preliminary study, we observed a highly insufficient supply of vitamin C in dental patients' diets, but only in patients with periodontal disease (PP) this level was considerably lower than in the control group of healthy subjects (CwDT). Vitamin C can be found in plant foods also rich in other bioactive

components (e.g. polyphenols and carotenoids) – the most important sources of antioxidants. It has been suggested that polyphenols (from apples, tea and wine catechins, and cranberries) interfere with various activities (including biofilm formation and adhesion) of *Porphyromonas gingivalis*, the main etiologic agent in chronic periodontitis [30]. The antioxidant capacities of the diet could be expressed by the total ORAC value, calculated as a sum of the lipid soluble (e.g. carotenoid) and water-soluble (e.g. phenolic) antioxidant fractions. In our preliminary study, the total ORAC values of the diets were the lowest in periodontal patients in comparison with two control groups, especially in comparison to subjects without dental problems (CwDT). Our results are consistent with previous observations. Chapple et al. [31] indicated an inverse relationship between reduced concentrations of plasma total antioxidants, vitamin C level, and the prevalence of periodontitis. Similar findings with regard to serum vitamin C were reported by Amarasena et al. [32] and by Nishida et al. [33] in case of dietary vitamin C supply, at least up to an intake of 180 mg daily. A recent study [34] of patients with metabolic syndrome has provided early indications of the potential of antioxidants found naturally in foods to reduce periodontal inflammation at clinical and biomarker levels. Also, deficiencies of folate and zinc may increase the permeability of gingival tissue, making patients deficient in these more susceptible to the bacterial plaque that causes periodontal disease. Earlier studies have shown that folic acid supplements in the diet may increase the resistance of gingival tissue to local irritants, and thus lead to a reduction in inflammation [35]. Our results concerning low folate supply in the diets of both dental groups (PP and CDT) indirectly confirm the important role of this vitamin in oral health. Smoking is one factor, which, like a poor diet, also decreases the serum folic acid concentration [36]. The prevalence of smoking in the current study was 20%, 10%, and 30% in the PP, CDT, and CwDT groups, respectively, and so folate status in smokers could be deteriorated to an even greater extent. Moreover, the literature cites several clinical studies which indicate the promising benefits of nutritional supplements including multiple vitamins – such as vitamins A, C, E, the B-complex vitamins, as well as green tea catechins – in periodontally compromised patients [37,38]. It is common knowledge that the bioavailability of most of vitamins and other bioactive food components is much better from foods than from supplements, and thus it is crucial in recommendations for periodontal patients in terms of adequate diet composition.

The results obtained point to the possible significance of dietary background in the progress of periodontitis, but a larger sample of patients – one also including chronic and aggressive types of the disease, as well as control groups – should be examined in order to draw a reliable conclusion on the differences in nutrient supplies and dietary patterns. To explain the etiology of periodontal disease, it would be useful to examine the dietary history, and the interaction between genetic, dietary, and other environmental factors.

Conclusions

Our preliminary study has indicated that the nutrient supply in periodontal patients differs significantly from that of healthy individuals, but not from other dental patients in terms of vitamin C and folate intake, and also of the total antioxidant capacity of the diet. There was also a higher number of periodontal subjects not meeting dietary recommendations for calcium and vitamin B_1, in comparison to the other groups. The nutritional insufficiency was more pronounced in periodontal patients than in patients suffering from other dental disorders. Additionally, the negative effects of these nutrient deficiencies could be strengthened by a low vitamin D intake, inadequate dietary lipid levels, and an excessively high saccharose intake, which were quite common in the examined population as a whole. These findings indicate that the coexistence of several dietary factors could be relevant to the progress of periodontal disease.

Piśmiennictwo / References

1. Albandar JM, Brunelle JA, Kingman A. Destructive periodontal disease in adults 30 years of age and older in the United States, 1988-1994. J Periodontal 1999, 70(1): 13-29.
2. Moynihan P. The interrelationship between diet and oral health. Proc Nutr Soc 2005, 64(4): 571-580.
3. Schaefer AS, Richter GM, Nothnagel M, et al. A 3' UTR transition within DEFB1 is associated with chronic and aggressive periodontitis. Genes Immun 2010, 11(1): 45-54.
4. Armitage GC. Development of a classification system for periodontal diseases and conditions. Ann Periodontol 1999, 4(1): 1-6.
5. Jepsen R, Kuchel GA. Nutrition and inflammation: the missing link between periodontal disease and systemic health in the frail elderly? J Clin Periodontol 2006, 33(5): 309-311.
6. Ziętek M. Schorzenia przyzębia jako przyczyny chorób ogólnoustrojowych. Przew Lek 2009, 1(12): 235-237.

7. Zaremba M, Górska R. Choroba przyzębia jako potencjalny czynnik ryzyka chorób sercowo-naczyniowych. Kardiol Pol 2008, 66: 1102-1106.
8. Morillo JM, Bullon P, Ramirez-Tortosa MC, et al. Nutrition-linked chronic disease and periodontitis: are they the two faces of the same coin? Mediterr J Nutr Metab 2009, 2: 103-109.
9. Fong IW. Infections and their role in atherosclerotic vascular disease. J Am Dent Assoc 2002, 133 Suppl: 7-13.
10. Pussinen PJ, Alfthan G, Rissanen H, et al. Antibodies to periodontal pathogens and stroke risk. Stroke 2004, 35(9): 2020-2023.
11. Griffin WS. Inflammation and neurodegenerative diseases. Am J Clin Nutr 2006, 83(2): 470-474.
12. Stein P, Scheff S, Dawson DR. Alzheimer's disease and periodontal disease: mechanisms underlying a potential bidirectional relationship. Grand Rounds Oral Systemic Med 2006, 1(3): 14-24.
13. Stewart R, Sabbah W, Tsakos G, et al. Oral health and cognitive function in the Third National Health and Nutrition Examination Survey (NHANES III). Psychosom Med 2008, 8: 936-941.
14. Enwonwu CO, Ritchie CS. Nutrition and inflammatory markers. J Am Dent Assoc 2007, 138(1): 70-73.
15. Enwonwu CO. Interface of malnutrition and periodontal diseases. Am J Clin Nutr 1995, 61(2): 430-436.
16. Enwonwu CO, Phillips RS, Falkler WA Jr. Nutrition and oral infectious diseases: state of the science. Compend Contin Educ Dent 2002, 23(5): 431-434.
17. Schifferle RE. Periodontal disease and nutrition: separating the evidence from current fads. Periodontol 2000. 2009, 50: 78-89.
18. Nishida M, Grossi SG, Dunford RG, et al. Calcium and the risk for periodontal disease. J Periodontol 2000, 71(7): 1057-1066.
19. Dixon D, Hildebolt CF, Miley DD, et al. Calcium and vitamin D use among adults in periodontal disease maintenance programmes. Br Dent J 2009, 206(12): 627-631.
20. Kunachowicz H, Nadolna I, Przygoda B, Iwanow K. Tabele składu i wartości odżywczej żywności. PZWL, Warszawa 2005.
21. Jarosz M, Bułhak-Jachymczyk B. Normy żywienia człowieka. Podstawy prewencji otyłości i chorób niezakaźnych. PZWL, Warszawa 2008.
22. Cronin JR. Comparing antioxidant values with the ORAC method. Alternative and Complementary Therapies 2004, 10: 167-170. http://www.chiroonline.net/_fileCabinet/orac_method.pdf
23. Nutrient Data Laboratory, Agriculture Research Service, US Department of Agriculture, USDA Database for the Oxygen Radical Absorbance Capacity (ORAC) of Selected Foods, Release 2. 2010. http://www.ars.usda.gov/SP2UserFiles/Place/12354500/Data/ORAC/ORAC_R2. pdf
24. Alam SQ, Bergens BM, Alam BS. Arachidonic acid, prostaglandin E2 and leukotriene C4 levels in gingiva and submandibular salivary glands of rats fed diets containing n-3 fatty acids. Lipids 1991, 26(11): 895-900.
25. Blok WL, Vogels MT, Curfs JH, et al. Dietary fish-oil supplementation in experimental gram-negative infection and in cerebral malaria in mice. J Infect Dis 1992, 165(5): 898-903.
26. Fernandes G, Venkataraman J. Role of omega-3 fatty acids in health and disease. Nutr Res 1993, 13(1): 19-45.
27. Campan P, Planchand PO, Duran D. Pilot study on n-3 polyunsaturated fatty acids in the treatment of human experimental gingivitis. J Clin Periodontol 1997, 24(12): 907-913.
28. Garcia MN, Hildebolt CF, Miley DD, et al. One-year Effects of Vitamin D and Calcium Supplementation on Chronic Periodontitis. J Periodontol 2010 Sep 1.
29. Neiva RF, Al-Shammari K, Nociti FH Jr, et al. Effects of vitamin-B complex supplementation on periodontal wound healing. J Periodontol 2005, 76(7): 1084-1091.
30. Petti S, Scully C. Polyphenols, oral health and disease: A review. J Dent 2009, 37(6): 413-423.
31. Chapple IL, Milward MR, Dietrich T. The prevalence of inflammatory periodontitis is negatively associated with serum antioxidant concentrations. J Nutr 2007, 137(3): 657-664.
32. Amarasena N, Ogawa H, Yoshihara A, et al. Serum vitamin C-periodontal relationship in community-dwelling elderly Japanese. J Clin Periodontol 2005, 32(1): 93-97.
33. Nishida M, Grossi SG, Dunford RG, et al. Dietary vitamin C and the risk for periodontal disease. J Periodontol 2000, 71(8): 1215-1223.
34. Jenzsch A, Eick S, Rassoul F, et al Nutritional intervention in patients with periodontal disease: clinical, immunological and microbiological variables during 12 months. Br J Nutr 2009, 101(6): 879-885.
35. Vogel RI, Fink RA, Schneider LC, et al. The effect of folic acid on gingival health. J Periodontol 1976, 47(11): 667-668.
36. Erdemir EO, Bergstrom J. Relationship between smoking and folic acid, vitamin B_{12} and some haematological variables in patients with chronic periodontal disease. J Clin Periodontol 2006, 33(12): 878-884.
37. Muñoz CA, Kiger RD, Stephens JA, et al. Effects of a nutritional supplement on periodontal status. Compend Contin Educ Dent 2001, 22(5): 425-428.
38. Hirasawa M, Takada K, Makimura M, et al. Improvement of periodontal status by green tea catechin using a local delivery system: a clinical pilot study. J Periodontal Res 2002, 37(6): 433-438.