

The role of obesity in modifying the course of periodontal diseases

Walis M., Kłosek S.*

The Department of Periodontology and Oral Mucosal Diseases at the Chair of Oral Surgery and Periodontology of the Medical University in Lodz, Poland

ABSTRACT

Overweight and obesity constitute a serious social problem. They are considered hazards of developed countries. Overweight and obesity affect both adults and children. Numerous researches on the negative impact of obesity on the condition of oral cavity have been conducted. The purpose of this paper is to provide a review of the most recent data published and noted in PubMed database between 2007 and 2014, on the association between overweight and obesity and oral diseases, including

caries and periodontal diseases, as well as to discuss their mechanisms. The majority of studies discussed in this paper demonstrate the existence of an association between obesity and the health of the oral cavity. An association between overweight and obesity expressed as various anthropometric indices, and oral cavity conditions should be the subject of further prospective studies.

Key words: Overweight, obesity, periodontal diseases, oral cavity.

***Corresponding author**

Zakład Periodontologii i Chorób Błony Śluzowej Jamy Ustnej
ul. Pomorska 251
92-213 Łódź, Poland
e-mail:sebastian.klosek@umed.lodz.pl

Received: 30.03.2014

Accepted: 22.04.2014

Progress in Health Sciences

Vol. 4(1) 2014 pp 195-199

© Medical University of Białystok, Poland

INTRODUCTION

Obesity has been a serious social problem since 1980 [1]. According to recent WHO reports, the number of obese people in the world has doubled since then. In 2008, 35 percent of adults over the age of 20 were overweight, and 11 percent were obese. As much as 65 percent of the world's population live in countries where overweight and obesity cause more fatalities than underweight. In addition, the problem increasingly affects children.

In 2011, over 40 million children under 5 years of age were overweight. Over 30 million overweight children live in developing countries and 10 million live in developed countries. Moreover, overweight and obesity are the fifth-leading cause of death worldwide. At least 2.8 million people die because of excessive weight and obesity every year [2].

According to WHO, obesity and overweight are defined as excessive accumulation of adipose tissue, resulting in unfavourable health effects [2]. Obesity is a risk factor for hypertension, hypercholesterolemia, type 2 diabetes, periodontal diseases, heart problems, myocardial infarction and some types of cancer [3].

Indices of obesity

Obesity may be determined using several anthropometric indices. They are: body mass index (BMI), waist circumference (WC) and waist-to-height ratio (WHR) [4]. BMI is the most popular one. It is a ratio of body weight to squared body height, expressed by the formula: kg/m^2 [5]. Overweight is defined as $\text{BMI} \geq 25 \text{ kg/m}^2$, and obesity as $\text{BMI} \geq 30 \text{ kg/m}^2$ [2].

The WC index is suitable for the evaluation of visceral fat content. This index has proved significant in the evaluation of mortality related to diabetes and coronary disease. As the discussed indices do not consider differences in height, the WHR index has been introduced. The level of abdominal adipose tissue may be variable within a very narrow BMI range, so the WHR index may be used to identify individuals with an increased risk of metabolic diseases, even if they remain in the normal weight category according to BMI.

Gorman et al. [6] demonstrated that "each unit increase in BMI was associated with a 5 percent increase in the risk of experiencing an alveolar bone loss (ABL) progression. An increase of 1 cm in waist circumference was associated with a 1 to 2 percent increase in the risk of experiencing probing pocket depth (PPD) and clinical attachment loss (CAL) progression, and each 1 percent increment in baseline WHR was associated with a 3 percent increase in experiencing periodontal disease progression events as defined by all three periodontal disease indicators".

Obesity and condition of the oral cavity

Poor condition of the oral cavity may indirectly contribute to obesity. Infectious diseases of the oral cavity, including caries, gingivitis and periapical changes, cause impairment of chewing ability and a tendency for exchanging hard and nutritious foods into soft ones, containing carbohydrates and unsaturated fatty acids. That in turn results in obesity. And conversely, obesity – being often the result of an unbalanced diet rich in carbohydrates stimulating development of *Lactobacillus sp.* and *Streptococcus* bacteria – favours the development of caries [7]. The study by Modéer et al. [8] on a group of youths, with BMI as a measure of obesity, demonstrated that obese individuals had a significantly higher number of carietic defects, and the stimulated saliva flow was 1.2 ml/min, whereas in a group of children with normal body weight, the corresponding value was 2.0ml/min. Another study confirming the existence of a correlation between obesity and caries was completed by Costa et al. [9] on a group of children from families with low income, with a mean age of 6 years. Over 50 percent of the study participants had caries, and 25 percent of these children were obese. However, the level of family wealth was the strongest factor in determining the existence of caries.

Adipose tissue metabolism and inflammatory markers

In order to better understand an association between obesity and periodontitis, the structure and role of the adipose tissue should be explained. It is a loose tissue made of adipocytes. Its main role involves isolation, amortisation and nutritional reserve. Adipose tissue is a metabolically active organ secreting over 50 bioactive substances, including pro-inflammatory cytokines, such as tumour necrosis factor (TNF- α) and interleukin-6 (IL-6). Both cytokines initiate production of pro-inflammatory proteins, including CRP. Additionally, adipocytes produce hormones: resistin that stimulates lipid production, and leptin – a protein responsible for appetite reduction. However, there are studies suggesting pro-inflammatory properties of leptin. Pro-inflammatory effect of TNF- α and IL-6 leads to the destruction of collagen contained in the gingival connective tissue, that is to the destruction of the connective tissue attachment, and activation of osteoclasts, leading to the destruction of alveolar processes [3,10].

Obesity and diseases of the periodontium

Studies on the association between diseases and the periodontium and obesity have been conducted for years. Periodontitis can be defined as a chronic, bacterial disease affecting tissues that support and surround teeth. It is one of

the most common human diseases affecting approximately 50 percent of the population over 30 years of age in the USA [11]. In Poland, studies on people aged 35-44 demonstrated that only 1.1 percent of subjects were free from periodontium diseases [12]. Inflammation of periodontal tissue leads to progressive destruction of periodontium and alveolar process bone, and to the formation of periodontal pockets [13]. Risk factors of periodontium diseases include: gender, addictions (tobacco smoking and alcohol consumption), and diabetes, obesity, osteoporosis, vitamin D deficiency, as well as stress, immunological factors: interleukin-1 (IL-1), tumor necrosis factor (TNF- α), and genetic factors [1,14].

To confirm the validity of the above-mentioned associations, Lissner et al. conducted a study determining the level of adipocytokines TNF- α and IL-6 in people with normal body weight and in obese patients with chronic periodontitis. Cytokine levels were determined in blood serum and periodontal pocket fluid. It was demonstrated that periodontitis influences mostly the level of resistin and adiponectin produced by adipocytes, and both obesity and periodontitis cause an increased leptin level. Moreover, obesity also causes an increased TNF- α level [15]. Whereas CRP is an acute phase protein, ORP (Orosomucoid) is a chronic phase protein synthesized in the liver under the influence of IL-1, IL-6 and TNF- α . Studies on ORP have demonstrated that in the case of morbidly obese people, periodontitis was closely correlated with the level of the protein. Moreover, among various studied adipocytokines and inflammatory markers, only the level of ORP was associated with severity of periodontitis [16]. Results of studies on rats with periodontitis experimentally provoked by gingival-irritating ligatures for 30 days were published in 2012. Obesity was also induced in part of the study on animals. The purpose of the study was to determine the effect of obesity on the loss of alveolar processes. The study demonstrated that a tooth-associated bone loss was much more pronounced in the maxilla, than in the jaw; there was no significant difference in alveolar process bone loss between obese and normal animals [17]. Further studies on Wistar rats demonstrated that, regardless of obesity, alveolar process loss was more pronounced in the group with artificially provoked periodontitis [18]. Different conclusions may be drawn following the analysis of human studies. Prpić et al. [7] studied a correlation between obesity (measured by BMI) and general condition of the oral cavity, in a group of men and women aged 31-60 years. The study demonstrated that obese subjects' oral cavities were in poorer condition. This manifested itself by a higher number of lost teeth, an increased number of carietic defects and an intensity of periodontium diseases. One of the most

extensive studies focused on an association between obesity and the condition of the oral cavity, and verification if a high BMI at the age of 21 determined poor periodontium condition in later life. This study was conducted by Linden et al. on a group of men aged 60 -70 years. The first phase of the study was conducted in 1991-1994 on a group of 2748 subjects aged 50 - 60 years; the second phase was conducted in 2001-2003 on 1400 subjects from the group studied in 1990s. Periodontic evaluations involving determination of depth of periodontal pockets and the level of connective tissue attachment were completed for facial and lingual, mesial and distal surfaces of all teeth, except for third molars. During the 10-year study period, subjects' BMI increased on average by 1.4 kg/m², and the number of obese men doubled. Subjects with higher BMI had lost more teeth, had more periodontal pockets ≥ 5 mm, compared to subjects with low BMI. Obesity increased the chance of periodontium diseases by as much as 77 percent. The problem affected only subjects with the highest BMI, and overweight men had periodontologic results comparable to those with normal BMI. Additionally, some of the subjects retrospectively diagnosed obesity at the age of 21. However, the study demonstrated that the frequency of periodontium diseases was not associated with the youth BMI value [19]. Another study on young adults in Brazil demonstrated that obese people had more often ≥ 2 teeth with gingival bleeding at probing. Moreover, subjects with at least one episode of obesity between the ages of 15 and 24 years had a 13 percent higher risk of tartar development. The study demonstrated no association between obesity and the development of periodontal pockets [20]. Another study was performed by Gorman et al. on a group of 1038 healthy men, examined from 1969 until 1996. The study was designed to evaluate the risk of periodontium diseases in overweight or obese subjects. Progressive disease was defined as a ≥ 40 percent loss of the alveolar process in at least two teeth, presence of periodontal pockets ≥ 5 mm deep and a loss of connective tissue attachment ≥ 5 mm. During the follow-up period, the BMI value increased by 1.6 kg/m², and the WHR by 4.5 percent; during the same period, the number of teeth free from periodontium diseases was more significantly reduced in the case of obese men. The risk of development of periodontal pockets, loss of the connective tissue attachment and alveolar process bone loss was higher by 40 percent, 52 percent and 60 percent, respectively, in the case of obese men (BMI ≥ 30 kg/m²). A 41 percent increase of risk of the connective tissue attachment loss of ≥ 5 mm was estimated in the case of men with visible abdominal obesity [6]. Jimenez et al. [21] in their twenty-year observation of a group of 36,910 people initially free from periodontium

diseases demonstrated that obesity measured by BMI, WHR and WC was indeed associated with an increased risk of periodontium diseases. Interestingly, they have acquired evidence suggesting a more significant correlation between diseases of the periodontium and BMI in obese dentists compared to obese people of other professions. Similarly, in obese diabetics, the risk of diseases of periodontium was as much as 93 percent higher, compared to people with normal weight. Obesity is an element of metabolic syndrome, constituting an important risk factor of arterial atheromatosis. Other components of the syndrome are hypertension, hyperglycaemia or pharmacologically controlled type 2 diabetes, and dyslipidemia. Presence of three of the above-mentioned criteria is necessary for the diagnosis of metabolic syndrome [22]. The study by Rousseau et al. on children of both sexes, aged 8-10 years, with a diagnosed metabolic syndrome, demonstrated increased TNF- α level in periodontal pocket fluid and bleeding gums. Those factors were particularly strong in obese boys with arterial hypertension and dyslipidemia [23]. A study on the effect of each component of metabolic syndrome on the periodontium was performed in a group of middle-aged and elderly women in Japan. Women were divided into 4 groups: with periodontal pockets < 2.0 mm and \geq 2.0 mm, and with connective tissue attachment loss of < 3.0 and \geq 3.0. The study demonstrated that obese women had periodontal pockets \geq 2.0 mm, but without a loss of the connective tissue attachment. Similarly, hyperglycemia and arterial hypertension increased the risk of development of periodontium diseases. However, among all components of metabolic syndrome, it was low HDL-cholesterol level that had the most pronounced effect on development of periodontium diseases [24]. On the other hand, the study published in 2013 by Tu et al. [25] demonstrated that people suffering from periodontitis and gingivitis were more commonly diagnosed with metabolic syndrome, compared to people free from periodontium diseases.

CONCLUSIONS

The studies discussed above suggest the existence of an association between biological and anthropometric indices defining overweight and obesity, and poor conditions of the oral cavity. The correlation is manifested by an increased frequency of oral dryness, numerous carietic defects and periodontitis. However, despite many years of studies, determining a direct causal relationship between obesity and condition of the oral cavity remains unsuccessful. The association may still be coincidental. Despite the results, further studies on human populations of various ages are necessary to provide a better understanding of those relations

and their mechanisms. The results presented above should induce a co-operation between MDs and DDSs aimed at increasing health awareness and improving the oral cavity conditions in overweight and obese patients.

Conflicts of interest

The authors declare that they have no conflicts of interest in the publication of the manuscript.

Financial disclosure/funding

The project has been funded by statutory resources of the Department of Periodontology and Oral Mucosal Diseases at the Chair of Oral Surgery and Periodontology of the Medical University in Lodz.

Authors' contributions

M.W - literature search, analysis, and writing the paper. S.K – concept, analysis, supervision, correction.

REFERENCES

1. Genco RJ, Borgnakke WS. Risk factors for periodontal disease. *Periodontol* 2000. 2013 Jun;62(1):59–94.
2. WHO Global InfoBase [Internet]. [cited 2014 Mar 13] Available from: <http://www.who.int/mediacentre/factsheets/fs311/en/>
3. Karels AJ, Cooper BR. Obesity and its role in oral health. *JAHSP* [Internet]. 2007; 5(1). [cited 2014 January 23] Available from: <http://ijahsp.nova.edu/articles/vol5num1/toc.htm>
4. Kaur P, Radhakrishnan E, Sankarasubbaiyan S, Rao SR, Kondalsamy-Chennakesavan S, Rao TV, Gupte MD. A comparison of anthropometric indices for predicting hypertension and type 2 diabetes in a male industrial population of Chennai, South India. *Ethn Dis*. 2008 Winter; 18(1):31-6.
5. Suvan J, Petrie A, Moles DR, Nibali L, Patel K, Darbar U, Donos N, Tonetti M, D'Aiuto F. Body mass index as a predictive factor of periodontal therapy outcomes. *J Dent Res*. 2014 Jan; 93(1):49-54.
6. Gorman A, Kaye EK, Apovian C, Fung TT, Nunn M, Garcia RI. Overweight and Obesity Predict Time to Periodontal Disease Progression in Men. *J Clin Periodontol*. 2012 Feb;39(2):107–14.
7. Prpić J, Kuis D, Pezelj-Ribarić S. Obesity and oral health--is there an association? *Coll Antropol*. 2012 Sep;36(3):755-9.
8. Modéer T, Blomberg CC, Wondimu B, Julihn A, Marcus C. Association between obesity, flow rate of whole saliva and dental caries in adolescents. *Obesity*. 2010 Dec;18(12):2367–73.
9. Costa LR, Daher A, Queiroz MG. Early childhood caries and body mass index in young

- children from low income families. *Int J Environ Res Public Health*. 2013 Mar;10(3):867-78.
10. Pischon N, Heng N, Bernimoulin JP, Kleber BM, Willich SN, Pischon T. Obesity, Inflammation and Periodontal Disease. *J Dent Res*. 2007 May; 86(5):400-9.
 11. Linden GJ, Lyons A, Scannapieco FA. Periodontal systemic associations: review of the evidence. *J Periodontol*. 2013 Apr; 84(4 Suppl.):S8-S19.
 12. Górska R, Pietruska M, Dembowska E, Wysokińska-Miszczuk J, Włosowicz M, Konopka M. Częstość występowania chorób przyzębia u osób w wieku 35–44 lat w populacji dużych aglomeracji miejskich. *Dent Med Probl*. 2012; 49(1):19–27 (Polish).
 13. Jańczuk Z. Praktyczna periodontologia kliniczna. Warszawa: Wyd. Kwintesencja, 2004: p.94-5 (Polish).
 14. Palle AR, Reddy CM, Shankar BS, Gelli V, Sudhakar J, Reddy KK. Association between Obesity and Chronic Periodontitis: A Cross-sectional Study. *J Contemp Dent Pract*. 2013 Mar 1; 14(2):168-73.
 15. Lissner L, Visscher TL, Rissanen A, Heitmann BL. Monitoring the obesity epidemic into the 21st century--weighing the evidence. *Obes Facts*. 2013; 6(6):561-5.
 16. Rangé H, Poitou C, Boillot A, Ciangura C, Katsahian S, Lacorte JM, Czernichow S, Meilhac O, Bouchard P, Chaussain C. Orosomucoid, a new biomarker in the association between obesity and periodontitis. *PLoS One*. 2013; 8(3):e57645.
 17. Verzeletti GN, Gaio EJ, Linhares DS, Kuchenbecker Rösing C. Effect of obesity on alveolar bone loss in experimental periodontitis in Wistar rats. *J Appl Oral Sci*. 2012 Mar-Apr; 20(2): 218-21.
 18. do Nascimento CM, Cassol T, da Silva FS, Bonfleur ML, Nassar CA, Nassar PO. Radiographic evaluation of the effect of obesity on alveolar bone in rats with ligature-induced periodontal disease. *Diabetes Metab Syndr Obes*. 2013 Oct 7;6:365-70.
 19. Linden G, Patterson C, Evans A, Kee F. Obesity and periodontitis in 60–70-year-old men. *J Clin Periodontol*. 2007 Jun;34(6):461–6.
 20. de Castilhos ED, Horta BL, Gigante DP, Demarco FF, Peres KG, Peres MA. Association between obesity and periodontal disease in young adults: a population-based birth cohort. *J Clin Periodontol*. 2012 Aug; 39(8):717–24.
 21. Jimenez M, Hu FB, Marino M, Li Y, Joshipura KJ. Prospective associations between measures of adiposity and periodontal disease. *Obesity (Silver Spring)*. 2012 Aug; 20(8):1718-25.
 22. Szczeklik A. Choroby wewnętrzne. Stan wiedzy na rok 2013. Kraków: Med Prakt. 2013: p.1283-4 (Polish).
 23. Ka K, Rousseau M-C, Lambert M, Tremblay A, Tran SD, Henderson M, Nicolau B. Metabolic syndrome and gingival inflammation in Caucasian children with a family history of obesity. *J Clin Periodontol*. 2013 Nov;40(11):986–93.
 24. Shimazaki Y, Saito T, Yonemoto K, Kiyohara Y, Iida M, Yamashita Y. Relationship of Metabolic Syndrome to Periodontal Disease in Japanese Women: The Hisayama Study. *J Dent Res*. 2007 Nov; 86(3):271-5.
 25. Tu Y-K, D' Aiuto F, Lin H-J, Chen Y-W, Chien K-L. Relationship between metabolic syndrome and diagnoses of periodontal diseases among participants in a large Taiwanese cohort. *J Clin Periodontol*. 2013 Nov; 40(11):994–1000.