Clin Oral Invest (2012) 16:727–734
DOI 10.1007/s00784-011-0554-7

ORIGINAL ARTICLE

Association between severity of body mass index and periodontal condition in women

André Luiz Pataro · Fernando Oliveira Costa · Sheila Cavalca Cortelli · José Roberto Cortelli · Mauro Henrique Nogueira Guimarães Abreu · José Eustáquio Costa

Received: 25 August 2010 / Accepted: 14 April 2011 / Published online: 10 May 2011

Abstract This study evaluated the association between body mass index (BMI) and periodontal condition in a population of Brazilian women. A hospital convenience sample of 594 eligible women was recruited from a women's health reference center of Belo Horizonte, Brazil. Four groups were formed considering BMI levels: BMI normal group (n=352), overweight (n=54), obesity level I (n=48), obesity level II (n=56), and obesity level III (n=74). Full-mouth periodontal examination was performed and biological, demographic, and behavioral risk variables were evaluated. Obese and overweight women showed statistically significant differences in bleeding on probing, probing depth and clinical attachment level ≥4 mm, and frequency of periodontitis (p<0.05) compared to women showing normal BMI. The final multivariate model for the occurrence of periodontitis revealed that obesity groups were significantly associated with periodontitis. In addition, age (25–45), smoking, diabetes, and hypertension remained significantly associated with the occurrence of periodontitis (p<0.05). Periodontitis was positively associated with obesity, and this association was more evident as obesity levels increases. These findings indicate the need for early diagnosis and the inclusion of periodontal care in health care programs for obese women.

Keywords Obesity · Body mass index · Periodontal disease · Risk factors

A. L. Pataro · F. O. Costa (✉) · J. E. Costa
Department of Periodontology, Dentistry School, Federal University of Minas Gerais,
Antonio Carlos Avenue, 6627—Pampulha, PO Box 359,
31270-901, Belo Horizonte, Minas Gerais, Brazil
e-mail: focperio@uol.com.br

S. C. Cortelli · J. R. Cortelli
Department of Dentistry, Periodontics Research Division,
University of Taubaté,
Taubaté, São Paulo, Brazil

M. H. N. G. Abreu
Department of Social and Preventive Dentistry,
Federal University of Minas Gerais,
Belo Horizonte, Brazil

Introduction

Obesity is a chronic, multifactorial disease which affects adults and children, being responsible for the increased risk of occurrence of different systemic disorders such as cardiovascular disease [1], diabetes [2], and other life-threatening diseases [3]. Today, obesity is considered the fastest growing health-related problem worldwide [4], both in developed and underdeveloped countries [5], with prevalence ranging from 9% to 35% [6, 7]. In 2005, the World Health Organization (WHO) [8] estimated that 1 billion people were overweight or obese and that, if this tendency continuous, this number could increase up to 1.5 billion by 2015.

Recent studies have shown that being overweight or obese has also been associated with an increased susceptibility to [9–18] and severity of periodontal disease [2, 5, 11, 13, 17, 18]. Some studies have demonstrated the important role of nutritional status in periodontal disease [19, 20]. Tomofuji et al. (2005) [20] related that a rich high-cholesterol diet was associated with junctional epithelium proliferation and with increasing bone resorption in rats. Moreover, obese individuals usually consume large amounts of caloric food containing saturated fat and low nutritional values, which could contribute to a poor oral health.

The biological plausibility of the association between obesity and periodontal diseases is based on the effect of pro-inflammatory cytokines released by adipose tissue,

suggesting that similar mechanisms are involved in obesity and periodontitis pathophysiologies, and that the secretions of these substances could induce a hyperinflammatory response on periodontal disease [19]. Various molecules released by adipose tissue, i.e., interleukin-6 and tumor necrosis factor-α affect the metabolism of the body and contribute to the development of a low grade systemic inflammation [21]. The levels of these pro-inflammatory cytokines were proportional to BMI, particularly on individuals presenting visceral obesity, since the increase in the body mass may induce a hyperinflammatory response on periodontal disease. Furthermore, obesity could affect host vascular and immune responses due to a decreased blood flow [22].

So far, there have been different studies on the connection between obesity and periodontitis still showing, however, conflicting data [4, 5, 10, 12, 15, 17, 18, 23, 24]. There is still a need, therefore, for more comprehensive research about this connection, specifically the one related to BMI severity, which had not been evaluated until now.

Therefore, this study investigated the association between obesity/overweight and periodontal status in a population of Brazilian women. Additionally, the study evaluated the influence of biological, demographic, and behavioral risk variables on the occurrence of periodontitis.

Material and methods

Participants

A hospital convenience sample consisting of 848 women, monitored in the program of women's health from the hospital of the Federal University of Minas Gerais, Brazil, were invited to participate in a cross-sectional study on their periodontal condition. Seven hundred and twenty women volunteers were eligible for the study and underwent a full-mouth periodontal examination and collection of risk variables of interest from March 2008 to March 2010. After adopting the following exclusion criteria: younger than 18 and older than 65, BMI <18 kg/m^2, less than 12 teeth present, pregnant, and antibiotic use within 3 months prior to periodontal examination, 126 women were excluded.

Therefore, the study population was composed of a total of 594 women belonging to mixed ethnic group and aged between 18 and 65 years (mean of 39.7±17.35). Participants were carefully informed about the objectives of the study and, after their approval, they signed a consent form. The present study was performed in accordance with the Helsinki declaration of human studies and received approval from the Federal University of Minas Gerais Research Committee (ETIC 578–07).

Social–demographic characteristics

Demographic data such as age, gender, household income, marital status, and years of education were collected from each participant. Individuals' medical histories were obtained from medical records focusing on history of diabetes, hypertension, and dyslipidemia. These same data were carefully obtained from controls by laboratory examination and blood pressure measurement. Smoking habits were categorized as: nonsmoker (people who never smoked or who stopped smoking more than 5 years prior to that date) or smoker/past smoker. Oral hygiene habits such as flossing, frequency of daily toothbrushing, and past professional cleaning were also collected.

Anthropometric measurements including weight (in kilograms), height (in meters), and BMI (in kilograms per square meter) were measured with subjects wearing light clothing and no shoes by a trained and calibrated professional nutritionist. Height was registered by a vertical metric ruler and weight by a scale (Welmy, Model 104A, Brazil), both checked and certified by INMETRO (Instituto Nacional de Metodologia, Normalização e Qualidade Industrial, Brazil.). Calculation of BMI was conducted for all participants. Measures of height and weight of 20 individuals were replicated to recalculation of BMI. Intra-examiner agreement for measurements of BMI showed reproducibility values by kappa test greater than 0.90. Based on WHO obesity guidelines, participants were categorized according to BMI as group normal BMI (GN)—20–24.99 kg/m^2 (n=352), overweight group (GOW)—BMI 25–29.99 kg/m^2 (n=54), obesity level I (GOI)—30–34.99 kg/m^2 (n=48), obesity level II (GOII)—35–39.99 kg/m^2 (n=56), and obesity level III (GOIII)—≥40 kg/m^2 (n=74) [25].

Periodontal clinical examination

For each participant, a full-mouth periodontal examination was performed in a hospital gurney with a photophore (Missouri-Luxo 70, Brazil).The hospital gurney was adjusted to the sitting position and teeth were cleaned with sterile gauze when necessary, after plaque index registration. Two periodontists (ALP and FOC) trained and calibrated in the beginning of the study measured probing depth (PD) and clinical attachment level (CAL). After 7 days, periodontal examinations of 10 women were repeated revealing intra- and inter-examiner reliability scores greater than 0.85 (pondered kappa test) for PD and CAL clinical parameters. Intraclass correlation tests presented scores greater than 0.90. The following periodontal parameters were registered: bleeding on probing (BOP), suppuration, PD, and CAL. PDs and CALs were recorded to the nearest millimeters with a manual periodontal probe

(North Carolina University model—UNC—#15, Hu-friedy; Chicago, USA) at six sites per tooth. PD was measured as the distance from the gingival margin to the bottom of the gingival crevice. CAL was determined by measuring the distance from the cement–enamel junction to the bottom of the gingival crevice. For this study, periodontitis was defined as the presence of proximal CAL (mesial or distal) ≥4 mm in two or more teeth, or proximal PD (mesial or distal) ≥5 mm in two or more teeth [26]. Oral hygiene was assessed using plaque index [27]. Additionally, the number of teeth was also recorded. Teeth with unscorable enamel–cement junction with caries cavities or teeth with iatrogenic fillings that made an adequate dental examination impossible were excluded from this study [28].

Statistical analysis

Statistical analyses included the descriptive characterization of the control, overweight, and obese groups, which were initially assessed in terms of all variables of interest including periodontal disease. Characteristics of individuals' variables were described using frequency distribution for categorical variables and median, mean, and standard deviation for continuous variables. Normality data distribution was verified by Kolmogorov–Smirnov test, finding a normal distribution. Each independent variable was subjected to analysis using the χ^2 test, Mann–Whitney, and *t* Student when appropriate to assess the association between the groups, including or not the dependent variable "periodontitis." Subsequently, in order to control the effect of potential confounders [29], all variables were tested step by step and separately, and those showing a p value <0.20, along with their interactions, were introduced into the logistic regression analysis. During the following steps, nonsignificant variables ($p>0.05$) were manually removed, and new variables were added to reach the final adjusted full model. All variables included in the final logistic regression model were determined to be independent, assessing the colinearity. Odds ratio (OR) and their 95% confidence intervals were calculated. Interaction effects between BMI and periodontitis occurrence for interest variables were calculated. All statistical tests were performed using a software SSPS (Statistical Package for Social Sciences, version 16 for Windows; Chicago, USA) and considered significant for p values <0.05.

Results

Table 1 shows the periodontal condition of the study sample. Obese (GOI, GOII, and GOIII) and overweight women (GOW) showed statistically significant differences of BOP, PD, and CAL ≥4 mm, as well as periodontitis

Table 1 Periodontal status of the sample according to BMI

Variables	Obesity, $n=178$			Overweight, $n=54$	Normal weight, $n=352$
	GOI	GOII	GOIII		
Sample, *n* (%)	48 (26.9%)	56 (31.5%)	74 (41.6%)	54 (100%)	352 (100%)
Plaque index	0.81 (±0.34) $p=0.065$	0.87 (±0.42) $p=0.057$	0.92 (±0.45) $p=0.032$	0.84 (±0.47) $p=0.073$	0.76(±0.32)
Bleeding on probing sites	29.6 (23.4–33.8) $p<0.001$	31.4 (26.8–31.9) $p<0.001$	34.8 (27.3–36.7) $p<0.001$	24.6 (14.3–25.2) $p<0.005$	17.4 (14.1–19.8)
Mean of sites with PD and CAL ≥4 mm[a]	12.34 (8.23–12.45) $p<0.001$	11.71 (8.71–13.27) $p<0.001$	14.57 (9.98–18.50) $p=0.003$	10.78 (7.89–13.42) $p<0.005$	8.4 (7.8–9.2)
Mean of sites with PD and CAL ≥5 mm[a]	6.45 (4.98–7.29) $p=0.021$	6.89 (3.91–7.84) $p=0.010$	7.41 (3.98–8.97) $p=0.035$	6.23 (2.98–9.37) $p=0.023$	4.1 (3.15–7.11)
Mean of sites with PD and CAL ≥7 mm[a]	0.44 (0.18–0.77) $p=0.527$	0.38 (0.11–0.81) $p=0.381$	0.57 (0.32–0.89) $p=0.523$	0.39 (0.23–1.1) $p=0.078$	0.41 (0.14–0.80)
Periodontitis, *n* (%)	35 (72.9) $p=0.005$	37 (66.0) $p=0.007$	50 (67.6) $p=0.005$	35 (64.8) $p=0.008$	152 (43.1%)

GOI obesity level I group (30–34.99 kg/m^2), *GOII* obesity level II group (35–39.99 kg/m^2) *GOIII* obesity level III group (>40 kg/m^2), *CAL* clinical attachment level, *PD* probing depth

[a] Same site (*t* Student test)

frequencies compared to normal group (GN, $p<0.05$). BOP was even more prevalent in the obese women group III (34.8%, $p<0.001$). This same group (GOIII) showed statistically significant differences for plaque index compared to normal group (GN, $p=0.032$). The occurrence of periodontitis for the groups GOW, GOI, GOII, and GOIII were respectively 64.8%, 72.9%, 66.0%, and 67.6%. All of them presented higher prevalence of periodontitis than GN (43.1%).

Table 2 shows the distribution of demographic, behavioral, and medical variables for the sample studied. It is noteworthy that overweight and obese groups did not differ from the GN group in relation to familiar income, past professional dental cleaning, and oral hygiene habits. However, GOW, GOI, GOII, and GOIII differed from the GN in the relation to age, years of education, marital status, hypertension, dyslipidemia, diabetes, and smoking ($p<0.005$).

The final multivariate logistic regression model for the occurrence of periodontitis is presented in Table 3. It is observed that obesity I, II, and III were significantly associated with periodontitis, presenting respectively OR 1.46 (1.02–3.89), 1.78 (1.08–4.21), and 2.31 (1.42–5.91). In addition, age (25–45 years), smoking, diabetes, and hypertension remained significantly associated with periodontitis (Table 3).

Table 4 shows two adjusted logistic regression models for the association between BMI and periodontitis, initially with adjustment for age and then for age, years of education, marital status, smoking, diabetes, hypertension, and dyslipidemia. The groups GOI, GOII, and GOIII showed significant association with periodontitis, and the odds ratio for age ranged from 1.26 to 2.12 and when adjusted for other variables ranged from 1.17 to 1.89.

Finally, it is important to consider that some interaction effects in the periodontitis occurrence could be found.

Table 2 Characteristics of the study sample according to BMI

Variables	Overweight n=54 (%)	GOI n=48(%)	GOII n=56(%)	GOIII n=74(%)	Normal Weight N=352 (%)	p value
Years of education						
<12	10 (18.7)	8 (4.4)	13 (7.3)	11 (6.2)	116 (33.1)	0.031
>12	44 (81.3)	31 (17.5)	51 (28.7)	64 (35.9)	236 (66.9)	
Age (years)						
<25	10 (18.7)	13 (7.3)	18 (10.1)	14 (7.8)	61 (17.3)	0.001
25–45	30 (55.9)	27 (15.2)	42 (23.6)	26 (14.6)	155 (44.1)	
>45	14 (25.4)	10 (5.7)	16 (9.0)	12 (6.7)	136 (38.6)	
Familiar income						
<5 MS	38 (69.5)	52 (29.3)	37 (20.7)	44 (24.7)	242 (68.6)	NS
>5 MS[a]	16 (30.5)	11 (6.2)	19 (10.7)	15 (8.4)	110 (31.4)	NS
Marital status						
Companion	33 (61)	34 (19.1)	51 (28.7)	48 (27)	223 (63.2)	
Without companion	21 (39)	18 (10.1)	15 (8.4)	12 (6.8)	129 (36.8)	0.001
Hypertension	6 (11.1)	10 (5.6)	16 (9.0)	27 (15.1)	17 (4.8)	0.002
Diabetes mellitus	2 (3.7)	5 (2.8)	8 (4.5)	9 (5.1)	11 (3.1)	0.031
Dyslipidemia	3 (5.6)	2 (1.2)	6 (3.3)	8(4.4)	15 (4.2)	0.014
Smoking						
Yes	6 (11.2)	7 (3.9)	10 (5.6)	11 (6.2)	29 (8.3)	<0.001
No	38 (70.3)	36 (20.3)	43 (24.2)	31 (17.4)	306 (86.9)	
Former	10 (18.5)	13 (7.3)	11 (6.2)	16 (9.0)	17 (4.8)	
Professional cleaning						
<6 m	30 (55.6)	35 (19.6)	32 (18)	24 (13.5)	155 (43.9)	NS
>6 m	24 (44.4)	31(17.4)	26 (14.6)	30 (16.9)	197 (56.1)	NS
Daily dental cleaning						
<1 per day	3 (5.6)	3 (1.7)	5 (2.8)	4 (2.3)	17 (4.7)	NS
1–3 per day	38 (70.4)	37 (20.8)	45 (25.3)	55 (30.9)	283 (80.4)	NS
>3 times a day	11 (20.0)	13 (7.3)	9 (5.1)	5 (2.8)	52 (14.9)	NS
Flossing						
Yes	37 (68.5)	42 (23.6)	61 (34.3)	32 (18)	258 (73.2)	NS
No	18 (31.5)	22 (6.8)	17 (9.6)	14 (7.9)	94 (26.8)	NS

NS no significance

[a] R$415.00, corresponding approximately to US $220.00

Table 3 Multivariate logistic regression model for the periodontitis

Variables	Odds ratio (95% CI)	*p* value
Normal weight	1.0 (reference)	–
Overweight	1.20 (0.59–2.70)	NS
Obesity (GOI)	1.46 (1.02–3.89)	*
Obesity (GOII)	1.78 (1.08–4.21)	**
Obesity (GOIII)	2.31 (1.42–5.91)	**
Age (25–45 years)	3.2 2 (1.57–9.14)	**
Marital status (without companion)	1.43 (0.28–2.17)	NS
Smoking	3.89 (1.22–9.73)	**
Diabetes	1.21 (1.01–3.74)	*
Dyslipidemia	1.71 (0.32–3.05)	NS
Hypertension	2.76 (1.32–4.47)	*

GOI obesity level I group (30–34.99 kg/m^2), *GOII* obesity level II group (35–39.99 kg/m^2), *GOIII* obesity level III group (>40 kg/m^2), *NS* no significance

*$p<0.05$; **$p<0.01$

BMI >30 kg/m^2 interacted with age (10.25), hypertension (15.70), diabetes (4.03), and also with smoking (15.79, $p<0.03$). Significant interaction effect for overweight individuals was not observed.

Discussion

This study showed that periodontitis was associated with different levels of obesity and that the chance of obese women to present periodontitis increased proportionally to BMI levels. Recent studies also showed an association between obesity and periodontitis [2, 11, 12, 18, 29, 30]. Similarly to this study, high prevalence of periodontitis was reported, ranging from 52.9% [17] to 70% [29]. Additionally, positive OR values between obesity and periodontitis, ranging from 1.48 to 8.6, were also observed [5, 9, 12, 13, 17, 18].

Table 4 Final multivariate logistic regression model for the association between BMI and periodontitis

BMI	OR (95% IC)[a]	OR (95% IC)[b]
Normal weight	1.00 (reference)	1.00 (reference)
Overweight	1.17 (0.67–1.36)	1.03 (0.52–1.23)
Obesity (GOI)	**1.26 (1.02–2.78)**	**1.17 (1.01–2.11)**
Obesity (GOII)	**1.64 (1.02–3.86)**	**1.23 (1.03–3.10)**
Obesity (GOIII)	**2.12 (1.23–4.62)**	**1.89 (1.07–3.19)**

Significant values in bold ($p<0.05$)

GOI obesity level I group (30–34.99 kg/m^2), *GOII* obesity level II group (35–39.99 kg/m^2), *GOIII* obesity level III group (>40 kg/m^2)

[a] Adjusted for age

[b] Adjusted for age, education, marital status, smoking, diabetes, hypertension, and dyslipidemia

However, Ylöstalo et al. (2008) [31] in a subpopulation of 2,841 nondiabetic individuals aged 30–49 found a weak but positive association between BMI and periodontitis after adjusting for potential confounders in a multivariate statistical model, perhaps influenced by the cutoff criteria used to periodontitis. Kongstad et al. (2009) [24] found an inverse association between BMI and CAL, but positively associated with bleeding on probing.

Bleeding on probing is an important indicator of periodontal disease and although it does not represent severity, it may indicate a higher prevalence and risk for clinical attachment loss [28]. In this study, high frequencies of bleeding on probing was observed, especially in obesity group III—GOIII—(34.8% $p<0.001$), showing hypothetically that the activity of periodontal disease could be incrementally associated with increased BMI. In addition, in this study, GOIII had higher levels of plaque index, which was statistically significant compared to women with normal BMI ($p=0.032$).

A higher prevalence of obesity in women than in men has been observed worldwide [6, 7]. Moreover, high prevalence of periodontitis in non obese women has also been reported. Dalla Vecchia et al. (2005) [12], in a study involving 706 Brazilian individuals, showed that obese women had 80% more chance to show periodontitis than normal weight women. Haffajee and Socransky (2009) [19] also reported that young, overweight or obese women showed a higher risk of periodontitis than individuals with normal BMI. In the present study, men were excluded due to his absence at the center searched. Thus, our results cannot make inferences about the association between obesity and periodontitis according to gender. Due to clinical significance and possible interactions, other factors related to individuals' systemic health status like diabetes, dyslipidemia, and hypertension were also analyzed in this study.

Diabetes mellitus is a known risk factor for periodontal disease [2, 14], and obesity is a risk factor for insulin resistance [32]. Therefore, diabetes could be considered a confounder according to Dalla Vecchia et al. (2005) [12] when studying the connection between obesity and periodontitis. The presence of diabetes in the multivariate model for the occurrence of periodontitis resulted in an OR of 1.21 (1.01–3.74), i.e., diabetic women had 1.2 times higher chance of showing periodontitis. Previous studies reported similar findings [2, 14].

It is well known that nutrition has influence on the immune system [32]. Cell membranes of defense cells, such as polymorphonuclear leukocytes, are primarily composed of phospholipids, and their turnover is influenced by the type and amount of ingested lipids. Thus, it has been reported that an excessive diet of lipids or fatty acids can depress immune function, in addition to decreased blood flow, affecting the vascular and immune response [23].

Additionally, dyslipidemia has been associated with periodontal disease, and it has been considered a risk factor variable for periodontitis [23, 33] However, in our final model, dyslipidemia was not significantly associated with the occurrence of periodontitis. Katz et al. (2002) [34] observed in a case–control study that total cholesterol levels, low density lipoproteins, and triglycerides were significantly higher in individuals with periodontal disease. Moreover, it has also been suggested that periodontal disease can be a risk factor for hyperlipidemia [33]. Hyperlipidemia causes hyperactivity on white blood cells increasing the production of free radicals, which are frequently associated with progression of periodontitis in adults [35]. Today, it is not clear enough if periodontal disease affects the lipid metabolism or if the abnormalities or conditions related to dyslipidemia were capable of causing periodontal tissue destruction [2, 36]. Nevertheless, the relation that seems to exist between these two variables requires further studies.

In the present study, hypertension showed an OR of 2.76 for occurrence of periodontitis. These results confirm previous studies reporting a positive association between hypertension and periodontal disease [37, 38]. Overweight and obesity are recognized as important determinants for high blood pressure levels. It is well known that weight gain is strongly associated with increased blood pressure, independent of changes in sodium intake [39].

An epidemiological study in Sweden involving more than 4,000 individuals showed an increased prevalence of hypertension in patients presenting periodontitis [37]. It has been found that hypertension is more prevalent in individuals with advanced alveolar bone loss [38], and that hypertension occurs significantly more frequently in patients with periodontitis, compared to populations with little or no periodontal disease [38, 39]. However, the question of whether hypertension is a risk factor for periodontitis is still inconclusive.

Smoking is considered a risk factor for the development and progression of periodontitis since it affects cellular and immune functions [12, 14, 15]. In this study, obese smoking women showed 3.89 times more chance of showing periodontitis. Our results corroborate the findings published by Nishida et al. (2005) [15] who also observed a significant correlation between BMI, periodontitis, and annual smoking in a group of 372 Japanese individuals. It has been shown that smokers tend to present lower BMI than never smokers, and that former smokers tend to gain weight after they stop smoking. Additionally, heavy smokers frequently show unhealthy habits such as alcohol consumption, saturated fat intake, and reduced physical activity [14, 39].

Age has been considered an indicator of risk, but also a confounder in studies of periodontal disease [26, 28]. In this study, individuals aged 25–45 were more likely to show periodontitis (OR=3.22). Although a greater proportion of the sample was in this age group (53.4% of obese people and 44.1% with normal BMI), it is recognized that clinical attachment loss can be cumulative. However, it should be noted that the periodontitis case definition criterion used in the present study includes both changes in the clinical parameters probing depth and clinical attachment loss [26].

In addition, it should be emphasized that the statistical treatment minimized the possible effects of confusion. Even after adjustments for age and other variables, a significant OR for the association between obesity and periodontitis was observed, and these odds ratios increased proportionally to BMI levels. Additionally, an interaction effect between age and medical conditions (hypertension and diabetes) as well as smoking showed OR raging from 4.03 to 15.79, revealing that the effect of BMI on the occurrence of periodontitis was powered by the action of these covariates.

Thus, studies have suggested a possible causal association between obesity and periodontitis, i.e., the presence of inflammation in periodontal tissue may represent a predisposing factor for obesity [24]. Also, a tooth loss caused by periodontal disease could contribute to obesity development. After the tooth loss, the individual will progressively show difficulty in chewing. Subsequently, with an inefficient chewing, together with inadequate swallowing, resulting both from reduced production of saliva as well as from the use of ill-fitting dentures, the patients are eventually lead to choosing foods that are easy to chew, such as carbohydrates, sugars, and fats, all rich in calories [17]. Additionally, such individuals are not able to eat foods such as: (1) proteins, which are fundamental to the maintenance of periodontal tissues; (2) fruits and vegetables, which are rich in fiber, reduce fat absorption, and promote satiety; and (3) antioxidant vitamins and minerals, essential in combating free radicals [39].

The majority of studies on the association between periodontitis and obesity are cross-sectional studies which do not stratified or analyzed the association between periodontitis and obesity levels statistically [3–5, 10, 12, 18, 24]. The present study design revealed that the chance of occurrence of periodontitis increased proportionally to the increase in BMI, however this type of study is still limited because it reflects a specific point in time. In epidemiological studies, BMI is the easiest and probably the most common index used to classify obesity [23]. However, it may involve an erroneous categorization of the sample population due to individual variations in the distribution of adipose tissue and muscle mass [31]. Therefore, the use of BMI classification may be misleading to the interpretation of the effect of weight on periodontitis [30].

Although recent studies have shown some association between obesity and periodontitis, the biological mechanism for this correlation is not well known. Nevertheless, it is shown elsewhere that cytokines and hormones, such as adipokines or adipocytokines derived from lipid tissue, also known as an endocrine tissue, may play a key role [21]. Until now, the available evidence is not enough to sustain a direct relation between obesity and periodontitis. Therefore, this association still needs further investigations by different study designs, mainly longitudinal and/or interventional research. Additionally, these studies should investigate the association between periodontitis and systemic inflammatory markers including samples of saliva, serum, and gingival fluid.

In conclusion, this study revealed a high prevalence of periodontitis in women exhibiting different levels of obesity, whereas individuals with higher BMI showed a greater chance of having periodontitis. This condition was influenced by biological and behavioral variables, especially systemic conditions such as diabetes and hypertension, and smoking. Although the relationship obesity/periodontal disease is not yet clear, there seems to be a set of factors that can impact the development and evolution of these two conditions. Thus, particular attention through preventive programs for oral health and periodontal care should be implemented and targeted for obese individuals.

Acknowledgments Financial support was obtained from the Research Support Foundation of Minas Gerais (FAPEMIG/15354), Coordination for the Improvement of Higher Education Personnel (CAPES), and the National Council for Scientific and Technological Development (CNPq).

Conflict of interest Authors declare that they have no conflict of interests.

References

1. Wilson PW, Bozeman SR, Burton TM, Hoaglin DC, Ben-Joseph R, Pashos CL (2008) Prediction of first events of coronary heart disease and stroke with consideration of adiposity. Circulation 118:124–130
2. Saito T, Shimazaki Y (2007) Metabolic disorders related to obesity and periodontal disease. Periodontol 2000 43:254–266
3. Reeves G, Pirie K, Beral V, Green J, Spencer E, Bull D (2007) Cancer incidence and mortality in relation to body mass index in the Million Women Study: cohort study. BMJ 335:1134
4. Haenle M, Brockmann S, Kron M, Bertling U, Mason R, Steinbach G (2006) Overweight, physical activity, tobacco and alcohol consumption in a cross-sectional random sample of German adults. BMC Public Health 6:233
5. Linden G, Patterson C, Evans A, Kee F (2007) Obesity and periodontitis in 60–70-year-old men. J Clin Periodontol 34:461–466
6. IBGE—Instituto Brasileiro de Geografia e Estatística (2004) Pesquisa de orçamentos familiares, POF 2002–2003. http://www.ibge.gov.br/home/presidencia/noticias/noticia_impressao.php?id_noticia=278. Accessed 20 May 2009
7. Flegal KM, Carroll MD, Ogden CL, Curtin LR (2010) Prevalence and trends in obesity among US adults, 1999–2008. JAMA 303:275–276
8. WHO—World Health Organization (2005) The World Health Organization warns of the rising threat of heart disease and stroke as overweight and obesity rapidly increase. http://www.who.int/mediacentre/news/releases/2005/pr44/en/index.html. Accessed 19 April 2007
9. Saito T, Shimazaki Y, Koga T, Tsuzuki M, Ohshima A (2001) Relationship between upper body obesity and periodontitis. J Dent Res 80:1631–1636
10. Al-Zahrani MS, Bissada NF, Borawski E (2003) Obesity and periodontal disease in young, middle-aged and older adults. J Periodontol 74:610–615
11. Al-Emadi A, Bissada N, Farah C, Siegel B, Al-Zaharani M (2006) Systemic diseases among patients with and without alveolar bone loss. Quintessence Int 37:761–765
12. Dall Vecchia CF, Susin C, Rosing CK, Oppermann RV, Albandar JM (2005) Overweight and obesity as risk indicators for periodontitis in adults. J Periodontol 76:1721–1728
13. Wood N, Johnson RB, Streckfus CF (2003) Comparison of body composition and periodontal disease using nutritional assessment techniques: Third National Health and Nutrition Examination Survey (NHANES III). J Clin Periodontol 30:321–327
14. Genco RJ, Grossi SG, Ho A, Nishimura F, Murayama Y (2005) A proposed model linking inflammation to obesity, diabetes, and periodontal infections. J Periodontol 76:2075–2084
15. Nishida N, Tanaka M, Hayashi N, Nagata H, Takeshita T, Nakayama K, Morimoto K, Shizukuishi S (2005) Determination of smoking and obesity as periodontitis risks using the classification and regression tree method. J Periodontol 76:923–928
16. Reeves AF, Rees JM, Schiff M, Hujoel P (2006) Total body weight and waist circumference associated with chronic periodontitis among adolescents in the United States. Arch Pediatr Adolesc Med 160:894–899
17. Ritchie CS (2007) Obesity and periodontal disease. Periodontol 2000 44:154–163
18. Khader YS, Bawadi HA, Haroun TF, Alomari M, Tayyemm RF (2009) The association between periodontal disease and obesity among adults in Jordan. J Clin Periodontol 36:18–24
19. Haffajee AD, Socransky SS (2009) Relation of body mass index, periodontitis and *Tannerella forsythia*. J Clin Periodontol 36:89–99
20. Ritchie S, Kinane F (2003) Nutrition, inflammation, and periodontal disease. Nutr J 19:475–476
21. Tomofuji T, Yamamoto T, Tamaki N, Ekuni D, Azuma T, Sanbe T, Irie K, Kasuyama K, Umakoshi M, Murakami J, Kokeguchi S, Morita M (2009) Effects of obesity on gingival oxidative stress in a rat model. J Periodontol 80:1324–1329
22. Shuldiner AR, Yang R, Gong DW (2001) Resistin, obesity and insulin resistance—the emerging role of the adipocyte as an endocrine organ. New Engl J Med 345:1345–1346
23. Cutler CW, Iacopino AM (2003) Periodontal disease: links with serum lipid/triglyceride levels? Review and new data. J Int Acad Periodontol 5:47–51
24. Kongstad J, Hvidtfeldt UA, Grønbaek M, Stoltze K, Holmstrup P (2009) The relationship between body mass index and periodontitis in the Copenhagen City Heart Study. J Periodontol 80:1246–1253
25. WHO—World Health Organization (1997) Obesity: preventing and managing the global epidemic—report of a WHO consultation on obesity.
26. Page RC, Eke PI (2007) Case definitions for use in population-based surveillance of periodontitis. J Periodontol 78:1387–1399

27. Silness J, Löe H (1964) Periodontal disease in pregnancy. II. Correlation between oral hygiene and periodontal condition. Acta Odontol Scand 22:121–135
28. Costa FO, Guimarães AN, Cota LOM, Pataro AL, Segundo TK, Cortelli SC, Costa JE (2009) Impact of different periodontitis case definitions on periodontal research. J Oral Sci 51:199–206
29. Hyman J (2006) The importance of assessing confounding and effect modification in research involving periodontal disease and systemic diseases. J Clin Periodontol 33:102–103
30. Socransky SS, Haffajee AD (2005) Periodontal microbial ecology. Periodontol 2000 38:135–187
31. Ylöstalo P, Suominen-Taipale L, Reunanen A, Knuuttila M (2008) Association between body weight and periodontal infection. J Clin Periodontol 35:297–304
32. Marti A, Marcos A, Martinez JA (2001) Obesity and immune function relationships. Obes Rev 2:131–140
33. Noack B, Jachmann I, Roscher S, Sieber L, Kopprasch S, Luck C, Hanefeld M, Hoffmann T (2000) Metabolic diseases and their possible link to risk indicators of periodontitis. J Periodontol 71:898–903
34. Katz J, Flugelman MY, Goldberg A, Heft M (2007) Association between periodontal pockets and elevated cholesterol and low density lipoprotein cholesterol levels. J Periodontol 73:494–500
35. Uchiumi D, Kobayashi M, Tachikawa T, Hasegawa K (2004) Subcutaneous and continuous administration of lipopolysaccharide increases serum levels of triglyceride and monocyte chemoattractant protein-1 in rats. J Periodontal Res 39:120–128
36. Fentoğlu O, Oz G, Taşdelen P, Uskun E, Aykaç Y, Bozkurt FY (2009) Periodontal status in subjects with hyperlipidemia. J Periodontol 80:1610–1615
37. Holmlund A, Holm G, Lind L (2006) Severity of periodontal disease and number of remaining teeth are related to the prevalence of myocardial infarction and hypertension in a study based on 4,254 subjects. J Periodontol 77:1173–1178
38. Engström S, Gahnberg L, Högberg H, Svärdsudd K (2007) Association between high blood pressure and deep periodontal pockets: a nested case-referent study. Ups J Med Sci 112:95–103
39. Pischon N, Heng N, Bernimoulin JP, Kleber BM, Willich SN, Pischon T (2007) Obesity, inflammation, and periodontal disease. J Dent Res 86:400–409