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Cardiovascular risk in obese patients with chronic periodontitis. A clinical controlled study

Risco cardiovascular em pacientes obesos com periodontite crônica. Estudo clínico controlado

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Resumo

Introdução: Estudos têm demonstrado que a obesidade tem sido considerada um fator de risco para o desenvolvimento de doença periodontal e dos eventos cardiovasculares. Objetivo: Avaliar o risco às doenças cardiovasculares (DCVs) em pacientes obesos com e sem doença periodontal. Material e método: Participaram do estudo 100 pacientes, os quais foram divididos em quatro grupos: Grupo O - obeso sem periodontite crônica (n=25), Grupo OP - obeso com periodontite crônica (n=25), Grupo NO - não obeso sem periodontite crônica (n=25), Grupo NOP - não obeso com periodontite crônica (n=25). Foram avaliados dados demográficos e laboratoriais (colesterol total, lipoproteína de alta densidade - HDL e de baixa densidade - LDL, triglicérides e glicemia), dados antropométricos (índice de massa corporal - IMC; circunferência abdominal - CA; gordura corporal - GC), pressão arterial e parâmetros periodontais (sangramento à sondagem - SS, profundidade de sondagem - PS e nível de inserção clínico - NIC). O risco cardiovascular foi obtido baseado no escore de PROCAM. A correlação entre obesidade, doença periodontal e risco às DCVs foi verificado pelo teste de Spearman ($\alpha=0,05$). **Resultado:** O grupo OP apresentou estatisticamente maior quantidade de sítios com PS \geq 7 mm (11,2±2,03) quando comparado aos outros grupos ($p \leq 0,05$). Os níveis de triglicérides, colesterol total e LDL foram estatisticamente maiores no grupo OP. O risco às DCVs foi estatisticamente maior no grupo OP (28,1±3,3) quando comparado ao grupo O (16,5±3,5), grupo NOP (12,8±3,9) e grupo NO (7,7±0,9). A obesidade e a doença periodontal estão diretamente relacionadas com um aumento moderado do risco DCVs (r=0.53; p<0.0001 e r=0.62; p<0.0001, respectivamente). **Conclusão:** Conclui-se que a obesidade e a doença periodontal aumentam o risco à eventos cardiovasculares.

Descritores: Doenças periodontais; periodontite crônica; obesidade; doenças cardiovasculares.

Abstract

Introduction: Studies have shown that obesity is considered a risk factor for the development of periodontal disease and cardiovascular events. Objective: The objective was to evaluate the risk for cardiovascular diseases (CVDs) in obese patients with and without periodontal. Material and method: One hundred patients were divided into four groups: Group O - obese without chronic periodontitis (n=25); Group OP - obese with chronic periodontitis (n=25); Group NO - non-obese without chronic periodontitis (n=25); and Group NOP - non-obese with chronic periodontitis (n=25). Demographic and laboratorial data (total cholesterol, high-density lipoprotein - HDL and low-density lipoprotein - LDL, triglycerides, and glucose); anthropometric measurements (body mass index - BMI; waist circumference - WC; body fat - BF); blood pressure; and periodontal parameters (bleeding on probing - BOP, periodontal probing depth - PPD, and the clinical attachment level - CAL) were evaluated. Cardiovascular risk was obtained according to the PROCAM's score. The correlation between obesity, periodontal disease and risk for CVD was verified by Spearman's test ($\alpha = 0.05$). **Result:** The group OP showed a statistically higher rate of PPD \geq 7 mm (11.2 ± 2.03) when compared with other groups, as well as higher levels of triglycerides, total cholesterol, and LDL ($p \le 0.05$). The risk for CVD was statistically higher in the group OP (28.1 ± 3.3) when compared with group O (16.5 ± 3.5) , group NOP (12.8 ± 3.9) , and group NO (7.7 ± 0.9) . Obesity and periodontal disease are directly related to a moderate increase in CVD risk (r = 0.53, p < 0.0001 and r = 0.62, p < 0.0001, respectively). Conclusion: It was concluded that obesity and periodontal disease increases the risk to cardiovascular events.

Descriptors: Periodontal diseases; chronic periodontitis; obesity; cardiovascular diseases.

INTRODUCTION

Cardiovascular diseases (CVDs) are regarded as an alteration that changes the hemodynamics of the circulatory system,¹ and are responsible for 50% of deaths in Brazil². Among the different CVDs, arteriosclerosis is the most prevalent, and is characterized by atherosclerotic or atheromatous plaques that are deposited on the internal arterial walls^{2,3}.

Some factors that can exacerbate the risk for developing CVDs are: oxidation of low-density lipoprotein (LDL), dyslipidemia, hyperglycemia, release of free radicals due to smoking, ethnicity, psychosocial factors, arterial hypertension, a family history of CVD, diabetes mellitus, genetic alterations, being overweight or obese, sedentarism⁴, an increase in C-reactive protein, hyperhomocysteinemia, and the presence of periodontopathogenic microorganisms⁵⁻⁷. Some studies have been conducted with the ultimate aim of determining how to identify individuals who might be at a high-risk for developing CVDs, taking into account the existence of the strong correlation between the different high-risk factors associated with CVDs and the increased prevalence of cardiovascular diseases^{7.8}.

Approximately 20% of CVD cases are associated with being overweight or obese,⁹ as a higher body mass index (BMI) can be related to the elevation of triglycerides, glucose levels, and blood pressure, and a decrease in high-density lipoprotein (HDL) levels^{9,10-12}. Currently, obesity is considered one of the major public health problems, and it has also been considered as one of the main risk factors associated with CVDs¹³. Adipose tissue secretes several immunomodulatory factors,¹⁴ such as tumor necrosis factor- α (TNF- α), leptin, interleukin-1 (IL-1),^{15,16} interleukin 8 (IL-8)¹⁶ and interleukin 6 (IL-6)¹⁷. All of these cytokines exacerbates the inflammatory response, and also play a role in the regulation of vascular metabolism by mediating metabolic, immunological, and cardiovascular responses^{9,18}.

In addition to obesity, studies have reported that patients with periodontal disease have certain risk factors, which are similar to those related to cardiovascular disease, including older age, sex (predominately male), low social economic status, stress, and smoking¹⁹. The association between periodontal disease and CVD has been discussed in many studies,²⁰⁻²² however, some of these studies are primarily based on retrospective analyses,^{7,22} have methodological limitations.²¹ Other authors reported that individuals with CVDs, or who have suffered from an acute myocardial infarction or a cerebrovascular accident^{22,23} may demonstrate severe periodontal involvement characterized by tissue and bone loss, suppuration, excessive bleeding, a large accumulation of biofilm and calculus, and consequent tooth loss.

In a recent meta-analysis, researchers identified that patients with periodontitis presented a relative risk of 1.14 for the development of CVDs, among people with periodontal bone loss compared with the ones without bone loss, suggesting that a slight association exists between these two pathologies; however, causality remained uncertain²². Other meta-analyses also reported that periodontal diseases led to an increased risk of developing CVDs^{7,8}. Besides, some researchers postulated that

the relationship between periodontitis and CVDs occurs due to the colonization of periodontal microorganisms in atheromatous plaques^{7,8}.

Considering the common risk factors shared between periodontal diseases and CVDs, the prevention of these risk factors may result in lower morbidity and mortality⁵. To address this, several researchers have conducted studies with the ultimate aim of facilitating the early identification of individuals who might be at risk of developing future heart diseases^{24,25}. In this light, these studies have tried to identify at-risk individuals through the application of scores of different cardiac risk assessments, such as the Society of Cardiology Systematic Coronary Risk Evaluation (SCORE),25 the Cardiovascular Risk Management (CARRISMA) software,²⁶ and the Prospective Cardiovascular Münster (PROCAM) risk score²⁴. Each of these tools evaluates the probability that an individual will suffer from a cardiac event in the next ten years. In this same vein, the purpose of this controlled clinical study was to evaluate the risk for cardiovascular diseases (CVDs) in obese patients with and without periodontal.

MATERIAL AND METHOD

1. Population Screening

Approval to conduct this study was granted by the Human Research Ethics Committee (protocol number 01/08), which conforms to the ethical guidelines of the 1975 Declaration of Helsinki. Approximately 380 patients were recruited from March 2008 throughout June 2009. Of all these recruited patients, 260 obese (Body Mass Index - $BMI \ge 30 \text{ kg/m}^2$) and 120 non-obese (BMI between 18.5 to 24.9 kg/m²) patients underwent clinical screening. Of all patients screened, 123 (69 obese and 54 non obese) showed the following criteria: to be around 35 and 60 years old; to have at least 20 teeth (excluding third molars); to have a clinical diagnosis of generalized chronic periodontitis,27 with a minimum of 6 non-adjacent teeth with periodontal probing depths measuring \geq 5 mm, bleeding on probing and a clinical attachment loss \geq 4 mm. Besides, patients had to present a negative history of antibiotic therapy during the last 6 months and a negative history of anti-inflammatory treatment in the last 3 months; and they must have not received periodontal treatment in the last 6 months. Smokers were included in the study, but all patients reported that they have never smoked. Pregnant women and patients with diabetes were excluded from the study.

The measurement of systolic and diastolic blood pressure was performed, as well as the fulfilment of a specific questionnaire to check whether the patients are smokers, whether they perform physical activity and the frequency of it.

2. Obesity Status

Obesity was confirmed by Waist-to-Hip Ratio (WHR) measurements (≥ 0.85 for women and ≥ 0.9 for men),²⁸ Waist Circumference (WC) (> 88 cm for women and >102 cm for men); and percentage of Body Fat (BF), using the bio-impedance method (\geq 33% for women and \geq 25% for men)²⁹. Obese patients

presented with a BMI \ge 30 kg/m², while non-obese or normal weight patients presented with a BMI ranging from 18.5 to 24.9 kg/m². Non-obese or normal weight patients also presented with WHR, WC, and BF measurements that were less than the reference values obtained from obese patients.

3. Clinical Parameters

A single blind examiner for cardiovascular disease was trained to evaluate the clinical periodontal parameters (r = 0.962, p = 0.002, Kappa test). The clinical periodontal examination was performed using a manual periodontal probe (PCPN115BR, Hu-Friedy, Chicago, IL). Six sites per tooth were evaluated for periodontal probing depth (PPD), bleeding on probing (BOP), and clinical attachment level (CAL). Generalized Chronic Periodontitis was established when the patient showed a minimum of 6 non-adjacent teeth with periodontal probing depths measuring ≥ 5 mm, bleeding on probing and a clinical attachment loss measuring ≥ 4 mm²⁷.

4. Groups

Of all patients screened, 123 (69 obese and 54 non obese) showed the inclusion criteria, only 100 patients (50 obese and 50 non obese) participated in the study. During the study 19 patients were excluded from obese and 4 non-obese group. Among these patients, 5 had ingested drugs by medical advice, a patient became pregnant, 10 patients did not attend the regular return, and 8 dropped out of treatment.

Subjects were divided into four groups: Group O - obese patients without chronic periodontitis (n=25); Group OP - obese patients with chronic periodontitis (n=25); Group NO - non-obese patients without chronic periodontitis (n=25); and Group NOP - non-obese patients with chronic periodontitis (n=25).

The sample size was calculated in a previous pilot study, using the t test for independent means of the clinical attachment level in obese (3.8 ± 0.35 mm) and non-obese patients (4.1 ± 0.34 mm). The sample size was estimated into 24 patients per group, considering a power of 85% and α of 0.05 (*t* test).

5. Cardiovascular Risk

PROCAM score values were used to evaluate the cardiovascular risk indicators assessed in this study²⁴. Patients were evaluated for their risk of developing CVDs over the next ten years, and percentage values were calculated based on each risk factor. The risk factors for developing CVDs measured in this study included: age (years); high density lipoprotein level (HDL, mg/dL); low density lipoprotein level (LDL, mg/dL); level of triglycerides (mg/dL); smoking status (quantity and frequency over the last 12 months); having a history of diabetes mellitus; having a family history of acute myocardial infarctions; and systolic arterial blood pressure (mmHg) (Table 1).

6. Data Management and Statistical Analysis

The statistical analysis was performed using both the Kruskal-Wallis test and the Mann-Whitney's test for ordinal data. McNemar's test was used for nominal data. A probability level

Variables		Values
Age (years)	35-39	0
	40-44	6
	45-49	11
	50-54	16
	55-59	21
	60-65	26
	<100	0
	100-129	5
LDL cholesterol (mg/dL)	130-159	10
~ 0 * /	160-189	14
	≥190	20
	<35	11
HDL cholesterol	35-44	8
(mg/dL)	45-54	5
	≥55	0
	<100	0
Triglycerides	100-149	2
(mg/dL)	150-199	3
	≥200	4
Smoling	No	0
Smoking	Yes	8
Diabetes Mellitus	No	0
Diabetes Menitus	Yes	6
Family History	No	0
Family History	Yes	4
	<120	0
	120-129	2
Systolic arterial pressure (mmHg)	130-139	3
	140-159	5
	≥160	8

of $p \le 0.05$ was regarded as statistically significant. Spearman test was used to verify possible correlation between obesity, periodontal disease and cardiovascular risk ($\alpha = 0.05$).

Since this is a controlled study, an ad hoc test of statistical power was performed. LDL data was explored to assess the independent means between obese patients (Group O: 108.1 ± 13.8) and non-obese patients (group NO: 98.7 ± 10.9). The power analysis was performed to ensure that the sample used was large enough to uncover statistically significant differences. The power was calculated by a post-hoc t test (G Power[®] 3.0.10, Faul et al.³⁰ (2007), Bonn, Germany), and it was estimated to be 0.91 with an error of $\alpha = 0.05$.

RESULT

When examining the factors associated with obesity in each group, patients' weight, BMI, BF, and WC were significantly higher in the obese groups (O and OP) (Table 2).

The frequency of periodontal parameters, BOP and CAL of 4-6 mm and \geq 7 mm in the groups of patients with periodontal disease (OP and NOP) was statistically higher when compared with groups without periodontal disease (O and NO). It is worth noting that participants in Group OP showed higher frequency of PPD \geq 7mm (11.2 ± 2.03) than Groups O (0.11 ± 0.3), NOP (6.97 ± 1.46), and NO (0.0) (Table 3).

Table 4 illustrates the biological and behavioral factors that **were** used in determining the degree of patients' risk in developing CVDs according to the PROCAM score. Each group of patients was compared with each of these measures. In terms of the biological factors associated with an increased risk of developing

CVDs, Group NO showed significantly lower triglycerides levels when compared with the levels found in the other groups. In contrast, members of Group OP scored significantly higher in terms of their triglycerides levels (106.6 \pm 6.7 mg/dL), total cholesterol levels (206.4 \pm 10.4 mg/dL), and LDL levels (139.2 \pm 7.9 mg/dL) than the other groups. In addition, patients in Group OP showed higher frequency of family history of acute myocardial infarctions (30.43%) when compared with the other groups (group O: 20%; group NO: 15.8% and group NOP: 18%). However, group OP showed a significant lower prevalence of members who practice physical activity.

It was observed that 34% of patients in Group NOP, 18.75% of patients in Group O, and 52.4% of patients in Group OP showed a low level of risk for developing CVD in the next ten years. Among the group of patients who presented obesity and periodontal disease (Group OP), 18.9% of patients exhibited a moderate risk for developing CVD in the next 10 years. Moreover, 4.7% of

Table 2. Demographic and anthropometric data of the study population

Characteristics —	Groups			
	Group O	Group OP	Group NO	Group NOP
Men (%)	12.5	8.7	21	18.2
Women (%)	87.5	91.2	79	81.8
Age (years)	37.2 ± 2.07^{a}	$41.5\pm5.4^{\rm a}$	38.2 ± 3.6^{a}	$42.9\pm3.5^{\rm a}$
Weight (Kg)	93.5 ± 17.4^{a}	88.7 ± 9.0^{a}	$59.3 \pm 7.5^{\mathrm{b}}$	$56.5 \pm 7.2^{\mathrm{b}}$
Height (m)	1.6 ± 0.1^{a}	$1.59\pm0.07^{\rm a}$	$1.66 \pm 0.06^{\text{a}}$	1.59 ± 0.06^{a}
Body Mass Index (kg/m ²)	35.4 ± 6.6^{a}	35.3 ± 4.8^{a}	$22.09\pm2.3^{\rm b}$	$23.03\pm2.1^{\rm b}$
Body Fat (%)	$0.4\pm0.06^{\rm a}$	$0.41\pm0.05^{\rm a}$	$0.24\pm0.04^{\rm b}$	$0.30\pm0.05^{\rm b}$
Waist-Hip Ratio	0.9 ± 0.06^{a}	$0.89\pm0.07^{\rm a}$	0.85 ± 0.1^{a}	$0.86\pm0.08^{\rm a}$
Waist Circumference (cm)	108.5 ± 13.2^{a}	107.3 ± 10.5^{a}	$79.3 \pm 7.5^{\rm b}$	83.2 ± 7.2^{b}

^{a,b}Different letters in the lines represent statistically significant differences between the groups, Kruskal-Wallis and Mann-Whitney's test, $p \le 0.05$. Obese: $\ge 30 \text{ Kg/m}^2$; Non-obese: $< 25 \text{ Kg/m}^2$.

Table 3. Average and Standard Deviation of clinical periodontal parameters

	Groups			
Clinical Parameters —	Group O	Group OP	Group NO	Group NOP
Number of teeth	24.3 ± 2.1^{a}	$22.9\pm2.7^{\mathrm{a}}$	23.85 ± 2.85^{a}	23.7 ± 1.9^{a}
Number of sites	154 ± 12.2^{a}	137.4 ± 16.3^{a}	144.6 ± 14.5^{a}	134 ± 10.2^{a}
BOP (%)	3.6 ± 2.5^{a}	$63.7\pm9.4^{\rm b}$	3.2 ± 2.9^{a}	$61.4 \pm 4.6^{\mathrm{b}}$
PD ≤ 3 mm (%)	96.4 ± 4.6^{a}	$61.9\pm5.9^{\mathrm{b}}$	98.9 ± 2.07^{a}	$58.5\pm4.15^{\rm b}$
PD 4-6 mm (%)	$3.5 \pm 4.7^{\text{a}}$	$34.8\pm4.0^{\rm b}$	2.5 ± 1.9^{a}	$37.6 \pm 6.9^{\mathrm{b}}$
PD ≥ 7 mm (%)	$0.11 \pm 0.3^{\text{a}}$	$11.2 \pm 2.03^{\mathrm{b}}$	0.0°	$6.97 \pm 1.46^{\rm d}$
CAL ≤ 3 mm (%)	$94.2\pm6.5^{\rm a}$	47.2 ± 7.9^{b}	95.2 ± 6.5^{a}	$42.5\pm7.8^{\rm b}$
CAL 4-6 mm (%)	5.6 ± 3.2^{a}	$35.9\pm5.8^{\mathrm{b}}$	4.6 ± 1.9^{a}	$44.8\pm3.3^{\rm b}$
CAL ≥ 7 mm (%)	$0.5\pm0.35^{\mathrm{a}}$	$16.9 \pm 7.7^{\rm b}$	$0.28\pm0.46^{\rm a}$	$12.7 \pm 2.02^{\rm b}$

^{a,b,c}Different letters in the lines represent statistically significant differences among the groups, Kruskal-Wallis and Mann-Whitney's test, for ordinal data and McNemar's test for nominal data being $p \le 0.05$. PPD: periodontal probing depth; VPI: visible plaque index; GBI: gingival bleeding index; BOP: bleeding on probing; CAL: clinical attachment level.

patients in Group OP showed a high level of risk for developing CVD in the next 10 years. Besides, the control group (i.e., non-obese, non-PD: group NO) would have had an even lower risk of developing CVD in 10 years than the NOP group.

Through the Spearman correlation test, it was found that cardiovascular risk was positively correlated to obesity (r = 0.53; p < 0.0001) and to periodontal disease (r = 0.62; p < 0.0001). It means that obesity and periodontal conditions increases moderately the risk of cardiovascular disease.

DISCUSSION

Some authors have found that changes in WC and WHR are related to an elevation in the risk for developing coronary disease,³¹ however, BMI is the recommended anthropometric index upon which the clinical analysis of longitudinal follow-up

after cardiology intervention¹¹. BMI has been linked with CVD, especially when accompanied by diabetes mellitus,^{4,29} yet changes in WC and WHR are often overlooked in at-risk patient populations.

Overall, in the present study, the obese patients showed the greatest cardiovascular risk values (Group O: 16.5 ± 3.5 and Group OP: 28.1 ± 3.3) when compared with the non-obese patients (Group NO: 7.7 ± 0.9 and Group NOP: 12.8 ± 3.9); however, only the obese with PD group showed a significant statistical difference (Table 5). In accordance with our findings, Bogers et al.¹¹ (2007) found that weight gain represents a 45% increase in the risk of developing cardiovascular disease in the future. Changes in obese patients such as arterial hypertension, lipid profile (elevated total cholesterol, LDL, triglycerides, and lower HDL), elevated glucose, salty food consumption, smoking, and lack of physical activity³¹ can be predictive factors for cardiovascular risk. Furthermore,

Table 4. Average and Standard Deviation of the risk factors

Coronary Disk Factors* -	Groups				
Coronary Risk Factors* –	Group O	Group OP	Group NO	Group NOP	
Age (years)	37.2 ± 2.07^{a}	$41.5\pm5.4^{\rm a}$	38.2 ± 3.6^{a}	$42.9\pm3.5^{\rm a}$	
Smoking (%)	0.0^{a}	0.0ª	0.0ª	0.0ª	
Systolic Pressure (mmHg)	120 ± 15.7^{a}	129 ± 25^{a}	$109.7 \pm 10.0^{\rm a}$	125 ± 22.0^{a}	
Diastolic Pressure (mmHg)	$78.6\pm10.5^{\rm a}$	82 ± 12^{a}	72.06 ± 6.7^{a}	78.2 ± 8.3^{a}	
Triglycerides (mg/dL)	83.8 ± 12.8^{a}	$106.6\pm6.7^{\rm b}$	$72.6 \pm 8.7^{\circ}$	84.4 ± 11.0^{a}	
Total Cholesterol (mg/dL)	161.7 ± 13.8^{a}	$206.4\pm10.4^{\rm b}$	$159.5 \pm 17,1^{a}$	170.7 ± 8.3^{a}	
LDL (mg/dL)	$108.1 \pm 13.8^{\rm a}$	$139.2\pm7.9^{\rm b}$	$98.7 \pm 10.9^{\rm a}$	109.5 ± 8.1^{a}	
HDL (mg/dL)	48.8 ± 3.4^{a}	50.4 ± 4.0^{a}	51.7 ± 3.77^{a}	49.7 ± 3.4^{a}	
Glucose (mg/dL)	$84.8\pm7.9^{\rm a}$	$96.7\pm8.9^{\rm a}$	$78.9\pm9.08^{\rm a}$	$83.3\pm6.3^{\rm a}$	
Family History (%)**	20.0ª	30.43 ^b	15.78ª	18.0^{a}	
Physical Activity (%)	7.6ª	$0.0^{\rm b}$	20.0°	18.2°	
PROCAM score	16.5 ± 3.5^{a}	$28.1\pm3.3^{\rm b}$	$7.7\pm0.9^{\circ}$	12.8 ± 3.9^{a}	

^{a,b,c} Different letters in the lines represent statistically significant differences between the groups, Kruskal-Wallis and Mann-Whitney's test for ordinal data and McNemar test for nominal data being $p \le 0.05$. *PROCAM score²⁴.**Family History of MI – family history of Acute Myocardial Infarct.

Points	PROCAM risk (%)* —	Groups			
		Group O	Group OP	Group NO	Group NOP
0-20	<1%	81.25ª	28.7 ^b	100 ^c	66.00 ^d
21-28	1-1.99	12.5 ª	38.1 ^b	0.0 ^c	9.0 ^d
29-37	2-4.99	6.25 ^a	14.3 ^b	0.0 ^c	25^{d}
38-44	5-9.99	0.0ª	4.7 ^b	0.0^{a}	0.0ª
45-53	10-19.99	0.0ª	9.5 ^b	0.0ª	0.0ª
54-61	20-39.99	0.0ª	4.7 ^b	0.0ª	0.0ª
>61	>40	0.0 ^a	0.0^{a}	0.0^{a}	0.0 ^a

^{a,b,c,d}Different letters in the columns represent statistically significant differences between the groups, Kruskal-Wallis test, being p≤0.05. *low risk (< 10 %), moderate risk (10% to < 20 %) and high risk (≥ 20 %)²⁴.

the authors showed that sedentary individuals had twice the risk for developing a coronary event when compared with physically active individuals. These results corroborated the findings of this study, in which obese patients with PD (Group OP) showed a statistically lower prevalence for physical activity, which suggests an unhealthy lifestyle.

Additionally, researchers³² reported that engagement in physical exercise is associated with a lower risk for developing periodontitis, a fact which can be observed through the greater prevalence of non-obese patients without PD who are physically active (Group NO; 20%) when compared with non-obese patients with periodontitis (Group NOP; 18.2%). It should be noted, however, that no statistically significant difference in CVD risk was found between these two groups.

Obesity and periodontitis have been associated with changes in lipid metabolism, thus potentially increasing cardiac risk among patients presenting one or both factors^{6,8}. Those authors report the existence of a dose-dependent relationship among metabolic serum markers, elevated white blood cell count, and a higher severity of periodontal disease. According to Morita et al.³³ (2011) the dependence between BMI and periodontal disease when observed by the authors showed that 36.8% of men and 28.3% of women developed bone loss of more than 3 mm in a 5-year period follow-up. Moreover, in the present study, it was noted that obese patients with PD (Group OP) had elevated LDL and an increased cardiovascular risk; these findings may be reinforced by Barter et al.12 (2007) study, which suggested that high LDL serum levels and low HDL levels are predictive of the development of cardiovascular disease, thus representing important risk factors for heart disease.

Some authors have explained that the severity of periodontal disease observed in patients with obesity is associated with hyper-stimulation of the immune system present in obesity,²⁸ and with an increase in hormone secretion, cytokines, and in the inflammatory mediators that are involved in the pathogenic process of periodontal disease³⁴. These results can be confirmed by another study³⁵ that verified the positive association between periodontal disease and obesity on cardiovascular events. Additionally, when considering adipose tissue, some authors

believe that the involvement of periodontitis in the pathogenesis of atheroma formation relates to the release of cytokines and inflammatory mediators¹⁵⁻¹⁷.

Therefore the present study did not analyzed the impact of smoking on patients, in which it is important to note that smoking is one of the most important lifestyle-related risk factors for periodontitis,³⁶ for obesity¹⁹ and for cardiovascular disease⁴⁻⁷. A previous study also found out that smoking and obesity are independent risk indicators for periodontitis and exhibited a clear dose-response relationship³⁷.

It can be surmised that the public rise in both obesity and periodontal disease significantly increases patient morbidity. Moreover, preventive public health strategies, which include measures to reduce excessive weight gain, to prevent periodontal disease, and to increase nutritional education and the engagement in physical exercise should be adopted.

It also needs to be kept in mind that this study may be subject to several limitations. First, this study was observational from a sample that was evaluated in just a moment. Second, periodontal parameters were used to measure periodontal status and anthropometric data was used to determine the obesity. Thus, even though an association between poor periodontal status and elevated risk factors for CVD was observed. Likewise, obesity was associated with increased cardiovascular risk.

CONCLUSION

Within the limits of this study, it can be concluded that obesity and periodontal disease increased the risk to cardiovascular events.

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CONFLICTS OF INTERESTS

The authors declare no conflicts of interest.

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