Difusão do Conhecimento Através das Diferentes Áreas da Medicina 2

Lais Daiene Cosmoski
(Organizadora)

Atena Editora
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contato@atenaeditora.com.br

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APRESENTAÇÃO

Cada vez mais percebemos, que no mundo da ciência, principalmente da área da saúde, nenhuma profissão trabalha sozinha, é necessário que vários profissionais estão envolvidos e engajados em conjunto, prezando pela, prevenção, diagnóstico e tratamento de diversas patologias, visando sempre a qualidade de vida da população em geral.

A Coletânea Nacional “Difusão do Conhecimento Através das Diferentes Áreas da Medicina” é um *e-book* composto por 4 volumes artigos científicos, que abordam relatos de caso, avaliações e pesquisas sobre doenças já conhecidas da sociedade, trata ainda de casos conforme a região demográfica, onde os locais de realização dos estudos estão localizados em nosso país, trata também do desenvolvimento de novas tecnologias para prevenção, diagnóstico e tratamento de algumas patologias.

Abordamos também o lado pessoal e psicológico dos envolvidos nos cuidados dos indivíduos, mostrando que além dos acometidos pelas doenças, aqueles que os cuidam também merecem atenção.

Os artigos elencados neste *e-book* contribuirão para esclarecer que ambas as profissões desempenham papel fundamental e conjunto para manutenção da saúde da população e caminham em paralelo para que a para que a ciência continue evoluindo para estas áreas de conhecimento.

Desejo a todos uma excelente leitura!

Lais Daiene Cosmoski

SUMÁRIO

CAPÍTULO 6

SMOKING INCREASES PREVALENCE OF CHRONIC PERIODONTITIS IN INDIVIDUALSWITH CHRONIC KIDNEY DISEASE

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Cristiane Oliveira de Souza
Program in Brazilian Health, and Voluntary Dentist of the Interdisciplinary Nucleus of Studies, Research, and Treatment in Nephrology (NIEPEN) of the Federal University of Juiz de Fora (UFJF), Juiz de Fora, MG, Brazil

Rogério Baumgratz de Paula
MD,PhD
Department of Clinical Medicine and researcher at NIEPEN, UFJF, Juiz de Fora, MG, Brazil

Isabel Cristina Gonçalves Leite
D.H.Sc
Department of Public Health UFJF, Juiz de Fora, MG, Brazil

Letícia Martins de Paiva
Faculty of Odontology - UFJF

Giovanna César Caruso
Faculty of Odontology - UFJF

Júlia Azevedo Bahia
MDS
Department of Dentistry, UFJF

Jessica do Amaral Bastos
D.H.Sc
Dentistry of the Interdisciplinary Nucleus of Studies, Research, and Treatment in Nephrology (NIEPEN) of the Federal University of Juiz de Fora (UFJF), Juiz de Fora, MG, Brazil

ABSTRACT: Chronic periodontitis (CP) is an infectious inflammatory disease, which can be increased by the systemic inflammatory response in patients with chronic kidney disease (CKD). The aim of this study was to evaluate the prevalence of CP in smokers in the pre-dialysis stages of CKD. Subjects and Methods: Ninety-four individuals were and divided into two groups; group 1 was composed of smokers with CKD and group 2 was composed of non-smokers with CKD. Results: The probing depth (PD) was significantly elevated in group 1 than in group 2 (2.28 ± 0.52 mm vs. 1.89 ± 0.47 mm, respectively; $P < .001$.It was found that CP was more prevalent in group 1, with a total of 41 patients (87%), compared to group 2 in which 33 patients were affected by the disease (70%). Conclusion: Smoking appears to increase the prevalence of CP in patients with CKD.

KEYWORDS: chronic renal insufficiency, periodontitis, smoking

PREVALÊNCIA DA PERIODONTITE CRÔNICA EM PACIENTES RENAIS CRÔNICOS TABAGISTAS

1 I INTRODUCTION

Chronic kidney disease (CKD) is considered a global public health problem[1] In Brazil, the prevalence of CKD is 40.5/100,000, which is lower than the prevalence of CKD in the USA and Japan.[2] Chronic renal disease has a high mortality rate. Cardiovascular risk factors, such as diabetes mellitus (DM), high blood pressure (HBD), dyslipidemia, and smoking, are often noted in this population.[3,4] Additionally, some non-traditional risk factors, such as inflammation, oxidative stress, uremic toxins, anemia, and hyperparathyroidism, can contribute to high morbidity and mortality rates among patients with cardiovascular disease (CVD).[5]

In recent years, chronic periodontitis (CP) has been considered a cardiovascular risk factor.[6] The dissemination of the Gram-negative bacteria present in patients with CP destroys the supporting tissues of the teeth, which in turn induces local inflammation, and is associated with a systemic inflammatory and immunological response; this results in the production of cytokines and prostaglandins.[7] These alter the homeostatic properties of the endothelium and its permeability, thereby increasing its adhesiveness to leukocytes and platelets, modifying the anticoagulant properties of the procoagulants, and resulting in endothelial dysfunction[7,8] Thus, CP is considered a subclinical disease associated with atherosclerosis[8].

Chronic periodontal inflammation may contribute to a systemic inflammatory overload in patients with CKD, which can lead to a decrease in the estimated glomerular filtration rate (eGFR).[9,10] Some published studies have demonstrated the bidirectional relationship between CP and CKD.[11-13] Concordantly, the data from previous study of our group found that CP is more severe in patients with CKD compared to systemically healthy individuals. The findings of this study suggest that hypovitaminosis D leads to a decrease in the immune response in patients with CKD, thereby leading to a more severe form of the disease in these patients.[14]

Recently, some studies have demonstrated a significant association between smoking and CP,[15, 16] which could contribute to an increased risk of CVD, as well as an increased loss of renal function in patients with CKD.[17] However, there is currently a lack of information regarding the prevalence of CP in smokers with CKD. This study aimed to evaluate the prevalence of CP in smokers with CKD.

2 I SUBJECTS AND METHODS:

This is a cross-sectional study according to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Initiative. The participants of this study were found using a convenience sampling method amongst patients of a secondary healthcare service in Juiz de Fora, Minas Gerais, and Brazil. A total of

1,167 patients with CKD, secondary to HBD and DM, were being managed under conservative treatment at the healthcare service between 2014 and 2016. Of these, 94 subjects, 47 smokers (known as group 1) and 47 non-smokers (known as group 2) were considered eligible for this study (Figure 1). All subjects were aged above 18 years and had an eGFR of < 60 mL / min / 1.73 m². Patients who were taking anti-inflammatory drugs and antibiotics were excluded during the last four weeks of the dental evaluation. Furthermore non-smokers with < 10 years of smoking cessation; pregnant women; patients diagnosed with HIV, hepatitis, neoplasms, or other infections; patients with any other infection or a fever of unknown origin ; those who were treated for periodontitis in the last six months; and those who declined to provide written consent were also excluded.

The CKD diagnosis and the disease stage assessment were made following the criteria proposed by the National Kidney Foundation (K / DOQI, 2013)[18] and the eGFR was calculated based on the serum creatinine levels using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation.[19] The clinical and laboratory data collected included the following: gender, age, ethnicity, household income, educational, body mass index (BMI), stage and etiology of the CKD, previous diagnoses of CVD, alcohol consumption, systolic/diastolic blood pressure, DM diagnosis, HBD diagnosis, total cholesterol level, high density lipoprotein (HDL) level, serum creatinine level, and fasting blood glucose level. Only data from patients who had laboratory tests from a maximum of three months prior to clinical evaluation to three months after clinical evaluation were considered in this study.

A periodontal examination was conducted by two qualified examiners. The data obtained from the interclass correlation coefficient test during the inter-examiner calibration was 0.88 and 0.81 from the intra-examiner calibration. All of the participant's teeth, except the third molars, were examined. The probing depth (PD) and gingival recession were measured at six sites per tooth (mesiobuccal, buccal, distobuccal, mesiolingual, lingual, distolingual) using a computerized probe (Florida Probe Corp., USA). The clinical attachment level (CAL) was calculated using the distance between the cementum-enamel junction (CEJ) and the base of the probable pocket. The PD was considered as the distance of the gingival margin to the apical limit of the periodontal pocket, according to the software used (Florida Probe Corp., USA), the number of sites with bleeding on probing (BOP) was evaluated. The severity of CP was classified as mild, moderate, and severe according to the classification suggested by Eke et al (2012),[20] severe CP involved the presence of two or more interproximal sites with a CAL ≥ 6 mm (not in the same tooth) and one or more interproximal sites with a PD ≥ 5 mm; moderate CP comprised of the presence of two or more interproximal sites with a CAL ≥ 4 mm (not in the same tooth) and one or more interproximal sites with a PD ≥ 5 mm (not in the same tooth); mild CP

involved the presence of two or more interproximal sites with a CAL ≥ 3 mm and two interproximal sites with a PD ≥ 4 mm (not in the same tooth) or a site with a PD ≥ 5 mm. CP was determined as generalized when more than 30% of the sites had a PD > 4 mm. Patients who were diagnosed with CP were referred to the Faculty of Dentistry at the Federal University of Juiz de Fora (UFJF), for specialized treatment.

Information regarding smoking was collected, such as number of cigarettes smoked per day, if they were considered light smokers (< 10 cigarettes / day) or heavy smokers (≥ 10 cigarettes/day), how long they had been addicted, and an evaluation of the degree of smoking dependence using a test developed by Fargestrom, the Fagerstrom Tolerance Questionnaire (FTQ,1978)[21] and its revised version, the Fagerstrom Test for Nicotine Dependence (FTND,1991).[22] These tests assess the degree of nicotine dependence of a smoker. In Brazil, this scale was validated by Carmo and Pueyo in 2002,[23] and is composed of questions that focus on the reason for the addiction, the ability to quit smoking, and the consumption of tobacco.

The scale is based on 6 questions that indicate the individual's dependence: the number of cigarettes smoked per day; the amount of nicotine present in each cigarette; the effectiveness of the drug; how long it takes to smoke the first cigarette in the morning; if it is linked to the relief from nicotine withdrawal syndrome; the greater control of internal stimuli compared to external leading to the consumption of tobacco. Scale scores range from 0 to 10, with the response being the sum of all values; high dependence is characterized by a score ≥ 6, and a score of < 3 indicates low nicotine dependence. In this study, we considered high dependence ≥ 5 and low dependence < 5. This project was approved by the Ethics in Research Committee through the Brazilian Platform (CAAE: 36698614.0.0000.5147).

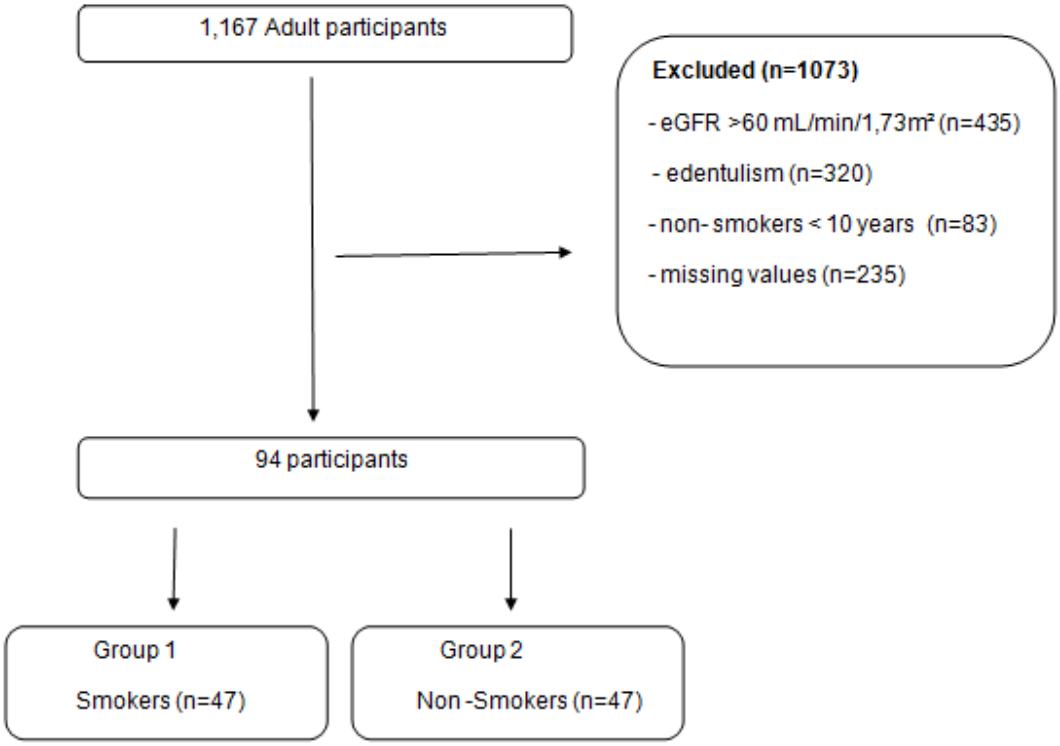

Figure 1. Participant´s flow chart.

3 I STATISTICAL ANALYSIS

The data were evaluated by the Statistical Package for the Social Sciences (SPSS), version 21.0. The quantitative variables were expressed as mean and standard deviation, for the data that presented as normal distribution, was analyzed using the Student's t-test. The qualitative variables were represented by relative and absolute frequencies and analyzed using the chi-squared test with Fisher's exact test. Statistical difference was considered for *P* values < .05.

4 I RESULTS

Patients in group 1 (n = 47; 33 males; mean age = 60 years) and group 2 (n = 47; 33 males; mean age = 60 years) were paired for demographic, clinical, and laboratory data. It was noted that gender, age, ethnicity, education, HBD, DM status, stage of CKD, etiology of CKD, history of CVD, and values for fasting glucose levels, total cholesterol, serum creatinine and eGFR did not differ statistically between groups, indicating homogeneity (Tables 1 and 2). In this study, the groups were predominantly men with an average age of 60 years and a low education and low household income, who were the usual users of the public health system, and so had restricted access to dental services. The population analyzed in this

study was composed of individuals with CKD, secondary to HBD and DM, which are responsible for about 70% of the cases of CKD worldwide.

TABLE 1. Demographics and clinicals characteristics of study population by groups

Characteristics	Group 1 n=47 (%)	Group 2 n=47 (%)	*P (value)*
Age, years			
< 60	24 (51,1%)	21 (44,7%)	0,340
>60	23 (48,9%)	26 (55,3%)	
Gender			
Men	33 (70,2%)	33 (70,2%)	0,589
Women	14 (29,8%)	14 (29,8%)	
Ethnicity			
White	20 (42,6%)	28 (59,6%)	0,074
Non- White	27 (57,4%)	19 (40,4%)	
Education			
Less than high school	28 (59,8%)	34 (72,4%)	
High school	16 (34,0%)	12 (25,5%)	0,452
More than high school	3 (6,4%)	1 (2,1%)	
Lower income			
Yes	46 (97,8%)	39 (83,0%)	0,038
No	1 (2,1%)	8 (17,0%)	
Hypertensive			
Yes	46 (97,9%)	46 (97,9%)	0,753
No	1 (2,1%)	1 (2,1%)	
Diabetic			
Yes	23 (49,8%)	28 (59,6%)	0,117
No	24 (51,1%)	19 (40,4%)	
Stage of CKD			
3 A /3 B	33 (70,2%)	26 (55,3%)	0,204
4	10 (21,3%)	19 (40,4%)	
5	4 (8,5%)	2 (4,3%)	
Etiology of CKD			
Hypertensive nephropaty	23 (48,9%)	23 (48,9%)	0,313
Diabetic nephropaty	13 (27,7%)	18 (38,3%)	
Chronic glomerulonephritis	5 (10,6%)	1 (2,1%)	
Other and unspecified	6 (12,8%)	5 (10,7%)	
Previous diagnoses of CVD			0,144
Yes	15 (31,9%)	21 (44,4%)	
No	32 (68,1%)	26 (55,5%)	
Alcohol consumption			
Yes	21 (44,7%)	14 (29,8%)	0,011
No	26 (55,3%)	33 (70,2%)	
BMI (kg/m²)			
<18,5	3 (6,4%)		
18,5-24,99	22 (46,8%)	10 (21,3%)	0,005
25-29,99	14 (29,8%)	17 (36,2%)	
>30,00	8 (17%)	20 (42,6%)	

p≤0,05; CKD= chronic kidney disease; CVD= cardiovascular disease; BMI:= body mass index

TABLE 2. Clinical and Laboratory data of study population divided by groups

Variables	Group 1 n=47 (Mean ± SD)	Group 2 n=47 (Mean ± SD)	P (value)
Fasting blood glucose (mg/dL)	104 ± 46	115 ± 37	0,736
Total cholesterol (mg/dL)	194 ± 52	180 ± 38	0,069
HDL cholesterol (mg/dL)	49 ± 17	41 ± 9	0,001
Creatinine (mg/dL)	2,2 ± 1,1	2,3 ± 1,0	0,789
eGFR (mL/min/1,73m²)	36 ± 13	33 ± 12	0,503
SBP (mmHg)	134 ± 21	140 ± 16	0,080
DBP (mmHg)	72 ± 21	72 ± 23	0,591

p≤0,05*; SD = standard deviation; HDL = high density lipoprotein ; eGFR= estimated glomerular filtration rate; SBP = systolic blood pressure; DBP = diastolic blood pressure.

This study found that CP was significantly more prevalent in group 1, in which 41 patients (87%) were diagnosed with CP, compared to the 33 patients (70%) in group 2 with CKD (*P* = .035) (Figure 2). During the analysis of periodontal clinical parameters, the mean number of similar teeth was noted, with significantly higher levels of PD in smokers when compared to non-smokers (2.28 ± 0.52 mm compared to 1.89 ± 0.47 mm, respectively; *P* = .001).The CAL and BOP were 3.97 ± 1.23 mm and 21 ± 25 sites, respectively, in group 1 and 4.11 ± 1.16 mm and 30 ± 27 sites, respectively, in group 2. Regarding the extent of CP, it was considered generalized in 37 smokers and 33 non-smokers (Table 3).

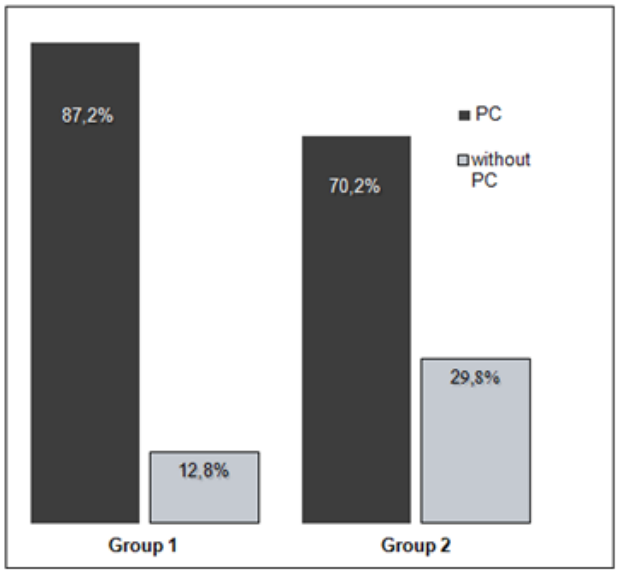

Figure 2. Prevalence of CP and without CP in patients divided by Group 1 and Group 2; CP = chronic periodontitis $P = .035$

TABLE 3. Evaluation of clinical periodontal paramenter divided by groups

Variables	Group 1 n=47 (Mean ± SD)	Group 2 n=47 (Mean ± SD)	*P (value)*
N° of theeth	15 ± 7,6	14 ± 7,4	0,955
Probing depth (mm)	2,28 ± 0,52	1,89 ± 0,47	0,001
Clinical attachment level (mm)	3,97 ± 1,23	4,11 ± 1,16	0,463
Bleeding on probing (sites)	21 ± 25	30 ± 27	0,247
Generalized CP (%)	78,7%	70,2%	0,239
Severe CP (%)	48,9%	44,7%	0,418

p≤0,05*; SD = standard deviation; CP = chronic periodontitis

The smoking history of group 1 indicated that the mean cigarette consumption was 11 ± 8 cigarettes /day and the mean duration of active smoking was 43 ± 12 years. When individuals were subdivided into light smokers (< 10 cigarettes / day) and heavy smokers (≥ 10 cigarettes / day), no difference was observed in the prevalence of CP among the two subgroups. On the other hand, smokers with higher scores in the FTND (n = 21), who were considered to be highly dependent, presented with significant values for PD, CAL, and BOP when compared to those

with low dependence (n = 26) (Table 4).

TABLE 4. Fagerström Test for Nicotine Dependence (FTND) and clinical periodontal paramenter

Variables	Highly Dependence (n=21) (Mean ± SD)	Low Dependence (n= 26) (Mean ± SD)	*P (value)*
N° of theeth	15 ± 6,9	14 ± 8,1	0,182
Probing depth (mm)	2,17 ± 0,33	2,37 ± 0,63	0,013
Clinical attachment level (mm)	4,12 ± 0,9	3,84 ± 1,4	0,049
Bleeding on probing (sites)	18 ± 18	23 ± 29	0,047
Generalized CP (%)	81%	76,9%	0,512

5 I DISCUSSION

In the present study, a higher prevalence of CP was observed in smokers with CKD compared to non-smokers. In the smoking group, the participants had a low dependence on nicotine, i.e., an average consumption of 11 cigarettes per day and an average time of exposure to tobacco of 43 years, which may be related to a higher prevalence of mild/moderate CP in this group.

CP is a chronic and inflammatory disease of infectious origin that has been associated with inflammatory factors in hemodialysis patients[24] and an increased risk of cardiovascular death in patients with CKD.[7,25] In a subpopulation of the NHANES III study, 13,748 individuals, 861 of whom were undergoing conservative CKD treatment, were followed up for a period of 14 years. At the end of this study, the authors observed that CP was associated with increased all-cause and, specifically, CVD mortality rates; the presence of CP in these individuals was an additional risk factor alongside other traditional risk factors for mortality, such as DM, hypertension, and smoking. [25]

In a cross-sectional study previously carried out by our group, the severity of CP in patients with CKD was assessed among four groups: systemically healthy subjects (with and without CP) were compared to patients with CKD (with and without CP. Periodontal clinical parameters, such as PD and CAL, were increased

in the CKD with CP group when compared to the systemically healthy groups. The percentage of sites with PD > 5 mm was similar in both groups with and without CKD, but the CAL was higher in the CKD with CP group when compared to the systemically healthy with CP group (32 ± 18 and 18 ± 16, respectively; $P < .05$). The same significant trend was observed in relation to the percentage of BOP, patients with CKD with CP who had more severe cases of periodontal disease with a higher prevalence of inflammation compared to systemically healthy individuals with CP (51 ± 32 and 23 ± 2, respectively; $P < .05$). With regards to systemic inflammation, the level of the inflammatory marker interleukin-6 (IL-6) was measured in the serum of the study subjects, and it was observed that the CKD with CP group had significantly higher levels of systemic inflammation when compared to the systemically healthy with CP group (14.3 ± 10.8 pg / mL and 5.9 ± 3.4 pg / mL, respectively; $P < .05$).[26]

The prevalence of CP in the general population is associated with local, systemic, and genetic risk factors, such as poor oral hygiene, systemic conditions such as DM and/or HIV infection, stress, socioeconomic level, and smoking status.[27] Several studies have shown a strong association between smoking and CP in the general population, [28,29] however, this association has not been evaluated in patients with CKD. This is the first study to evaluate the prevalence of CP in smokers with CKD.

In this study, a higher prevalence of CP was observed in smokers with CKD compared to non-smokers. This is consistent with a study by Shabrukh (2016), [30] in which it was observed that among 443 Pakistani smokers and non-smokers, there was a higher prevalence of CP in the smoking group (81.6%). Additionally, there was a greater chance of developing CP among heavy smokers (≥ 5 cigarettes / day) compared to moderate/ light smokers (< 5 cigarettes / day) and nonsmokers. In the current study, smokers consumed more cigarettes (11 cigarettes / day) and the average length of time of tobacco exposure was 43 years. An increased prevalence of CP among smokers with CKD (87%) was observed when compared to other studies focusing on systemically healthy smokers[9,10, 24]

Smoking is a key risk factor for the development of CP, which can be seen through the host's response, resulting in alterations in neutrophil function, antibody production, fibroblastic activities, vascular factors, and the production of inflammatory mediators, according to the studies analyzed by Javed (2014).[31]In a study by Nile et al. (2013),[32] tumor necrosis factor alpha (TNFα) and receptor activator of nuclear factor kappa-B ligand (RANKL) were shown to be good systemic indicators of the inflammatory process, which can in turn lead to osteoclastogenesis through an increase in RANKL expression and a decrease in osteoprotegerin (OPG) levels in osteoblasts. Under ideal physiological conditions, there is a balance between the reabsorption and bone formation, which is dependent on the receptor activator of

nuclear factor kappa-B (RANK)-RANKL-OPG axis. In patients with CP, there is a breakdown of this axis with a decrease in the OPG levels or an increase in the expression of RANKL, thereby leading to bone destruction.

In this study sample, smokers had significantly higher PD than non-smokers, although the values were not significant in terms of CAL. However, in other studies previously conducted by our group, patients with CKD and CP had similar PD values and increased CAL values when compared to those with CP who were systemically healthy.[14] This may be explained by the fact that this study involved patients with CKD in both groups (smokers and non-smokers), that the mean age of the patients was 60 years, and that the groups were paired in relation to their demographic, clinical, and laboratory data. This would explain why CAL is similar between the groups, as this represents bone loss over time due to the association of CKD in periodontal tissues. In contrast, smokers with CKD had increased PD compared to non-smokers. This could be because smoking contributes to an increase in the levels of inflammatory mediators.[33] On the other hand; an increase in local inflammation can be masked by the decrease in gingival bleeding. The data from this study is in agreement with the fact that of other similar study, [34, 35] in which BOP values were lower in smokers, although not significantly in comparison to those in the control group. The reduction of gingival bleeding in smokers may be associated with gingival vasoconstriction, secondary to the release of catecholamine, a hormone released by the nicotine present in tobacco.[35] In a study conducted by Calsina (2002),[36] an analysis of the effect of smoking on periodontal tissues among healthy individuals revealed significantly higher values for CAL and a lower number of sites with BOP in smokers compared to non-smokers, although there was no significant difference in terms of PD between the groups. In addition, the authors indicated that the values for BOP decreased and the PD, CAL, and gingival recession (RG) increased significantly after more than 10 years of smoking.

Tobacco consumption results in higher PD and greater loss of both clinical attachment and alveolar bone; consequently, smoking results in greater tooth loss.[15] In the present study, when analyzing the bone loss using CAL, no clinical or statistical relevance was found between the groups studied; clinical measures of PD and CAL are important for CP classification. These results are consistent with a study by Perrson et al. (2005)[37] in which smoking was not identified as a clinically significant risk factor for vertical bone loss. Using periapical radiographic analysis, the authors observed the long-term effect of all CP risk factors in the alveolar bone with the minimum history of smoking of 30 year.

The analyzed groups in this study had a similar mean number of teeth, which did not appear to be the main risk factor for dental loss in this population, when other probable causes for the event were taken into consideration, such as untreated

periodontal disease, restricted access to dental services, non-preventive dental treatments and an unfavorable socioeconomic status. In the study by Perrson et al. (2005)[37], a greater dental loss was noted in heavy smokers when compared to light and non-smokers, all were individuals over 60 years old, and, as in this study, the identification of the possible causes related to the lower number of teeth became impossible to be evaluated. In this study, the smoking group there was no association between the mean number of cigarettes/day, mean smoking time, and CP severity. The prevalence was similar between light (<10 cigarettes/day) and heavy (≥ 10 cigarettes/day) smokers. In the study by Jang et al. (2016),[38] the association between smoking status and CP in healthy adult Koreans was analyzed, and it was found that smoking was associated with a high risk for CP, with a mean consumption of ≥ 10 cigarettes/day and a time of exposure to smoking of ≥ 20 years. Systemically healthy patients who smoked from 1 to 30 cigarettes/day had a 2 to 4 times greater chance of developing CP compared to non-smokers, and the risk increased to 12 times for those who consumed more than 30 cigarettes/ day.

A study conducted in the Brazilian population found a strong association between smoking and the loss of clinical attachment in patients with severe CP, especially among heavy smokers and those aged 30–39 years compared to individuals over 50 years of age.[39] As in this study, most studies use only the tobacco history of the participants to find the association between smoking and CP. In this study, the FTND was used to assess the degree of nicotine dependence; 44.7% of patients in this study were considered highly dependent (≥ 5 points) and presented lower mean values of PD and less sites with BOP than those with low nicotine dependence with a higher CAL, along with the generalized extension of their periodontal disease. Thus, it seems that the use of the FTND in this study, in addition to the smoking history, is suggestive of a higher specificity in the evaluation of the degree of nicotine dependence of smokers, which demonstrates significant associations with the periodontal disease clinical parameters.

6 I CONCLUSION

The present study has some limitations. CP was evaluated in a single center and the sample was selected using a convenience method. In conclusion, the data obtained in this study suggests that smoking constitutes a risk factor for the development of CP in patients with CKD.

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