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ARTICLE

PERIODONTITIS AND RHEUMATOID ARTHRITIS. A CASE-CONTROL STUDY

Periodontitis y Artritis Reumatoide. Un estudio de casos y controles

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ABSTRACT

Background: Periodontitis and Rheumatoid Arthritis (RA) are immunoinflammatory diseases that share etiological mechanisms in their onset and development. **Objective:** To evaluate the bidirectional relationship between them.

Material and Methods: In 150 participants, sociodemographic variables, clinical attachment loss (CAL), probing depth (PD), bleeding on probing (BoP), rheumatoid factor (RF), anti-citrullinated protein antibodies (ACPA), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) were assessed. Additionally, arthritis activity was evaluated using the Disease Activity Score 28 and the Health Assessment Questionnaire. Initially, we analyzed the characteristics of the sample and associated different oral health. Finally, a logistic regression model was developed with rheumatoid arthritis status as the primary outcome.

Results: It was observed that patients with RA had a higher prevalence of periodontitis (p= 0.026), fewer present teeth (p<0.001), a higher number of sites with BoP (p=0.02), and more sites with PD = 4 mm and CAL ≥3 mm (p<0.001) compared to the group without RA. Additionally, when comparing patients with and without periodontitis, the periodontitis patients had a higher occurrence of RA than those without periodontitis (73.3% *versus* 26.7%, p=0.026). In the multivariate regression model, sites with PD = 4 mm and CAL ≥ 3 mm (OR 1.08, p<0.001), age (OR 1.07, p< 0.001), and CRP (OR 1.07, p=0.031) remained significant for the presence of RA.

Conclusions: Our results show worse oral health in patients with RA. It is also concluded that periodontal indicators negatively impact RA activity.

Keywords: *Periodontitis; Rheumatoid arthritis, Oral health; Case-control study; Periodontal diseases; Brazil*

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RESUMEN

Antecedentes: La Periodontitis (P) y la Artritis Reumatoide (AR) son enfermedades inmunoinflamatorias que comparten mecanismos etiológicos. **Objetivo:** Evaluar la relación bidireccional entre ambas.

Material y Métodos: Un estudio de casos y controles con 150 participantes, se evaluaron variables sociodemográficas, pérdida de inserción (PI), profundidad de sondaje (PS), sangrado al sondaje (SS), factor reumatoide (FR), anticuerpos anti-péptidos citrulinados cíclicos (ACPA), velocidad de eritrosedimientación (VES), proteína C-reactiva (PCR), DAS 28 y el Cuestionario de Evaluación de la Salud (HAQ). Se caracterizó la muestra y se analizaron diferentes parámetros de salud oral. Finalmente, se aplicó un modelo de regresión logística con el estado de la AR como resultado principal.

Resultados: Los pacientes con AR tienen mayor prevalencia de periodontitis (p= 0.026), menos dientes presentes(p< 0.001), más sitios con SS (p= 0.02), y con PS = 4 mm y PI >3 mm (p<0.001) comparado con los controles. Además, los pacientes con periodontitis tienen una mayor prevalencia de AR que aquellos sin periodontitis (73.3% frente a 26.7%, p=0.026). En el modelo multivariado, los sitios con PS = 4 mm y PI >3 mm (OR 1.08, p<0.001), la edad (OR 1.07, p<0.001) y la proteína C-reactiva (OR 1.07, p= 0.031) permanecieron significativos para la presencia de AR.

Conclusión: Nuestros resultados revelan una pobre salud oral en los pacientes con AR y que los indicadores periodontales afectan negativamente la actividad de la AR.

Palabras clave: *Periodontitis; Artritis reumatoide; Salud bucal; Estudio de casos y controles; Enfermedades periodontales; Brasil*

INTRODUCTION

Periodontitis is a chronic immunoinflammatory and infectious disease that affects the tissues supporting the teeth.[1] In Uruguay, the prevalence of periodontitis is up to 30% of the adult population, with 9% experiencing the most severe forms.[2]

The bacteria, bacterial products, and inflammatory mediators linked to periodontitis may contribute to the overall impact of systemic inflammation, affecting the development, severity, and progression of other chronic inflammatory diseases (diabetes mellitus, cardiovascular diseases, and respiratory diseases).[3]

Rheumatoid arthritis (RA) is an autoimmune disease characterized by synovitis and irreversible joint damage,[4] with a prevalence of 0.46% in South America.[5] However, the complications extend to extra-articular manifestations, such as interstitial lung disease involvement.[6]

Despite its uncertain etiology, the occurrence of RA is influenced by the presence of genetic and environmental risk factors that contribute to the development of systemic immunization. Among environmental factors, respiratory tract inflammation and periodontitis are significantly important in the pathogenesis of RA.[7] Recently, antigens from cytomegalovirus have been isolated in the synovial fluid of pa-

tients with RA.[8] Other studies also suggest a higher risk of developing RA in the presence of Human Parvovirus B199 and Epstein-Barr virus infection.[9,10]

Furthermore, certain oral bacteria, such as Porphyromonas gingivalis present in sites with periodontitis, can trigger the modification of host proteins through a process called citrullination.[11] These modified proteins are then recognized by the immune system, leading to the development of an inflammatory process.[12,13] Thus, periodontitis and rheumatoid arthritis share several characteristics. However, the scientific literature is not conclusive regarding the relationship between these two diseases.[14] The aim of this case-control study is to assess the association between periodontitis and rheumatoid arthritis.

MATERIAL AND METHODS

Design

During the period from September 2017 to September 2018, a case-control study was conducted. The cases were selected from the follow-up program at the National Institute of Rheumatology, Uruguay.[15]

Individuals allocated to the control group, systemically healthy individuals, were randomly selected from the dental clinics of the Federal University of Minas Gerais, Brazil.

Ethical considerations

Before the beginning of the study, each participant read and signed the informed consent. This project was approved by the Ethics Committee of the Republic University of Uruguay (registration number 091900-000248-17) and by the Research Ethics Committee of the Federal University of Minas Gerais (CAAE 37241214.2.0000.5097), and conducted according to the Declaration of Helsinki on human experimentation.

Population

Participants with a diagnosis of RA, aged >18 years, and with a minimum of six teeth present were eligible for the study. Exclusion criteria included the use of antibiotics within 3 months prior to entering the study, receipt of periodontal treatment within the last 6 months, other osteoarticular diseases, and pregnancy.

Variables

After confirming the diagnosis of arthritis, rheumatological variables were collected from the case group through venipuncture in the antecubital fossa after fasting for at least 8 hours in the morning. The samples were immediately processed in a public laboratory to analyze rheumatoid factor (RF), anti-citrullinated protein antibodies (ACPAs), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP). Additionally, arthritis activity was evaluated using the Disease Activity Score 28 (DAS 28) and the Health Assessment Questionnaire (HAQ).

An interview was conducted to gather data on age and gender. Educational level was categorized as < 8 years *versus* > 8 years, while smoking and diabetes status were dichotomized as yes/no.

A periodontal clinical examination was then conducted. Three examiners recorded periodontal probing depth (PD), clinical attachment loss (CAL), and bleeding on probing (BOP). All teeth, except third molars, were evaluated using circumferential periodontal probing, and data were recorded at four sites (mesial, buccal, distal, and lingual/palatal). Measurements were taken with a manual UNC 15 mm periodontal probe and rounded to the nearest millimeter. BOP was recorded dichotomously (absent/present).

Prior to the study, the examiners were trained and calibrated using ten non-study patients to assess reproducibility for PD and

AL, achieving an intraclass correlation coefficient of 0.8. The definition and classification of periodontitis as a "case" were based on the criteria outlined by Tonetti *et al.*,[16] Additionally, the sample was evaluated based on probing depth and attachment loss: PD = 4 mm and CAL ≥ 3mm; PD ≥ 5 mm and CAL ≥ 3 mm.

Sample

The sample size was determined based on previously reported prevalence (54% for cases *vesus* 31% for controls)[12] with a loss rate of 10%, totaling 75 participants per group. Therefore, the first patients with RA who met the inclusion criteria and attended the Faculty of Dentistry were enrolled, forming the final study sample.

Statistic

Descriptive statistical analysis of the total sample and comparison between the groups was initially conducted for the collected variables. Normality was assessed using the Kolmogorov-Smirnov statistical test.

The treatment groups were compared regarding the variables of interest using the t-test, Mann-Whitney test, Pearson chi-square, or Fisher's exact test, as appropriate. Subsequently, the influence of biological, behavioral, and social variables on arthritis was analyzed using multiple logistic regression. In the univariate analysis, variables for regression were selected based on a *p*-value of less than 0.20.

Next, a stepwise removal process was applied, in which variables were removed from the model one by one, starting with the variable with the highest *p*-value. In logistic regression, results with a significance probability lower than 5% were considered statistically significant ($p< 0.05$).

RESULT

A total of 75 patients with RA and 75 healthy controls participated in the study. Table 1 presents the sociodemographic characteristics of the sample. Considering the entire cohort, the percentage of females was similar, with no significant differences between groups (52.5% *versus* 47.5%, $p= 0.209$). Significant differences were observed regarding the age of the participants. Patients with RA were older than the controls (53.6 ± 12.1 *versus* 43.7 ± 8.9, $p< 0.001$). Most individuals with RA were over 54 years old, while most of the control group were between 30 and 54 years old. Notably, in the RA group, there were more smokers compared to the control group (70.4% *versus* 29.6%, $p = 0.019$).

On the other hand, there were no significant differences between groups for self-reported diabetes mellitus and educational level ($p = 0.058$, $p = 0.744$). Finally, when analyzing the CRP levels, it was observed that the RA cases had higher CRP scores than the controls (9.08 ± 11.1 *versus* 4.01 ± 4.30, $p< 0.001$). Laboratory data for ACPAs, ESR, and RF were evaluated in relation to periodontal status. The results showed that no significant differences were found for any of the variables studied. Similarly, the indicators of severity and joint damage (DAS 28 and HAQ) also did not show significant differences between the groups (Table 2).

People with RA had worse periodontal conditions (Table 3). The prevalence of periodontitis was higher in the RA group than in the controls (56.7% *versus* 43.3%, $p= 0.026$), with no significant differences between the different stages ($p= 0.901$). Likewise, they had a greater number of sites with PD = 4 mm and CAL ≥ 3 mm (20.0 *versus* 6.0, $p< 0.001$). For sites with PD ≥ 5 mm and CAL ≥ 3 mm (%), there were no

Table 1
Characteristics of the sample

		Cases (Rheumatoid Arthritis) (%)	Controls (n=75) (%)	*p*-value (n=75)
Age—mean (SD)		53.6 (12.1)	43.7 (8.98)	*p*< 0.001*
Age—n (%)	30-44 years old	17 (22,6)	48 (64.0)	*p*< 0.001**
	45-54 years old	18 (24.0)	18 (24.0)	
	55-64 years old	26 (34.7)	07 (9.3)	
	≥ 65 years old	14 (18.7)	02 (2.7)	
Gender—n (%)	Female	64 (85.3)	58 (77.3)	*p*= 0.209**
	Male	11 (14.7)	17 (22.7)	
Schooling—n (%)	≤ 8 years	33 (50.8)	36 (48.0)	*p*= 0.744**
	> 8 years	32 (49.2)	39 (52.0)	
Smoking—n (%)		19 (25.3)	08 (10.7)	*p*=0.019**
Diabetes—n (%)		5 (6.7)	0 (0.00)	*p*=0.058***
C-Reactive protein—mean (SD)		9.08 (11.1)	4.01 (4.3)	*p*<0.001****
Pharmacological therapy—n (%)	bDMARD	13 (17.3)		
	sDMARD	74 (98.6)		
	NSAIDs	52 (69.3)		
	Opioids	3 (4)		
	Corticosteroids	22 (29.3)		

:** t test. *:** Chi.square test. *****:** Exact Fisher test. ******:** Mann-Whitney test. **bDMARD:** Biological disease-modifying antirheumatic drugs. **sDMARD:** Synthetic disease-modifying antirheumatic drugs. **NSAIDs:** Non-steroid anti-inflammatory drugs.

Table 2
Laboratory parameters in the cases

		Rheumatoid Arthritis		
		Periodontitis (n=55)	Non-Periodontitis (n=20)	*p*-value
ACPAs—mean (SD)		235.27 (408.31)	245.16 (480.82)	0.697*
ESR—mean (SD)		15.4 (18.8)	12.6 (14.2)	0.542*
RF—mean (SD)		155.7 (280.7)	84.6 (128.7)	0.895*
DAS28—mean (SD)		3.51 (1.35)	3.02 (0.95)	0.139**
DAS28 cat—n (%)	1	14 (82.4)	03 (17.6)	0.082***
	2	06 (46.2)	07 (53.8)	
	4	30 (75.0)	10 (25.0)	
	5	5 (100.0)	0 (0.0)	
HAQ—n (%)	0	05 (83.3)	01 (16.7)	0.496***
	1	21 (80.8)	05 (19.2)	
	2	19 (73.1)	07 (26.9)	
	3	10 (58.8)	07 (41.2)	

:** Mann-Whitney test. *:** Chi.square test. *****:** Fisher´s exact test. **SD:** standard deviation. **ACPAs:** Anti-citrullinated protein antibodies. **ESR:** Erythrocyte sedimentation rate. **RF:** Rheumatoid factor. **DAS28:** Disease Activity Score 28. **HAQ:** Health Assessment Questionnaire.

Table 3

Laboratory parameters in the cases

		Cases (Rheumatoid Arthritis) (n=75) (%)	Controls (n=75) (%)	*p*-value
Periodontitis – n (%)		55 (56.7)	42 (43.3)	0.026*
Stages – n (%)	Stage 1	01 (100.0)	00 (0.00)	0.901**
	Stage 2	06 (66.7)	03 (33.3)	
	Stage 3	30 (56.5)	23 (43.4)	
	Stage 4	18 (52.9)	16 (47.1)	
Number of tooth presents, mean (SD)		16.4 (6.2)	23.8 (4.2)	< 0.001***
Sites with BOP (%)		48.9	36.6	0.020****
Sites with PD = 4 mm and CAL ≥ 3 mm (%)		20.0	6.0	< 0.001***
Sites with PD ≥ 5 mm and CAL ≥ 3 mm (%)		15.6	12.4	0.178***

:** Chi-square test. *:** Exact Fisher test. *****:** Mann-Whitney Test. **SD:** Standard Deviation. **CAL:** Clinical attachment loss. **PD:** Probing depth.

Table 4

Characterization of the sample regarding the prevalence of Rheumatoid Arthritis, considering Periodontitis as the outcome

	Case (Periodontitis) (n=97)	Control (non-Periodontitis) (n=53)	*p*-value
Rheumatoid Arthritis	55 (73.3%)	20 (26.7%)	0.026*

***:** Chi-square test.

Table 5

Multiple logistic regression for rheumatoid arthritis

	Final logistic regression model			
Independent variables	**Odds Ratio**	**CI (95%)**	***p*-value**	***p*; R^2N**
Age	1.076	1.038-1.115	<0.001	*p*< 0.001; 0.400
Probing depth = 4 mm and CAL ≥ 3 mm	1.086	1.035-1.140	<0.001	
C-reactive protein	1.078	1.007-1.154	0.031	

CI: Confidence interval.

significant differences between groups (p= 0.178). On the other hand, the controls had a greater number of teeth compared to the patients with RA (23.8 ± 4.24 *versus* 16.4 ± 6.17, p< 0.001) (Table 3).

When the total sample was divided into groups based on periodontitis as the outcome, a significantly higher prevalence of arthritis was observed in the group of individuals with periodontitis compared to the group without periodontitis (Table 4).

Among the variables with a p-value of less than 0.20 in the univariate analysis, those that remained in the final logistic regression model were age, PD = 4 mm and CAL ≥ 3 mm, and C-reactive protein, showing a significant impact on the outcome (Table 5).

The regression analysis demonstrated that the odds of RA among individuals with 3 or 4 mm attachment loss were 1.086 times higher than those without attachment loss (95% CI [1.035 – 1.140]; p< 0.001).

DISCUSSION

This study aimed to analyze the relationship between periodontitis and RA, two immuno-inflammatory diseases. The similarities between these conditions highlight the potential impact and connection between them.

Our results show a higher prevalence of periodontitis in individuals with RA compared to systemically healthy individuals (56.7% *versus* 43.3%, p= 0.026).

These percentages are similar to those reported in a systematic review conducted by Tang *et al.*,[17] where prevalences ranging from 15% to 100% were found in individuals with RA, and from 10% to 82.1% in the general population. The underlying inflammatory process, characterized by elevated levels of matrix metalloproteinases in RA-P18, shared factors[19] and the involvement of the HLA-DR1 epitope in response to the presence of P. gingivalis (Pg), help explain these results.[20] A greater number of sites with PD ≥ 4 mm and CAL ≥ 3 mm were observed in RA patients compared to controls. Specifically, *Porphyromonas gingivalis* has been analyzed in sites with incipient PD.

Data from a longitudinal study conducted at Helsinki Hospital are consistent with our findings, reporting that subjects with RA harbor elevated counts of *P. gingivalis* in sites with PD ≥ 4 mm compared to healthy controls.[21] BOP is another important clinical parameter for monitoring periodontal inflammation.16 The case group exhibited a higher number of sites with BOP compared to the control group (48.9% *versus* 36.6%; p= 0.020). Moura *et al.*,[12] also reported a similar difference in a sample of patients from the Rheumatology Clinic of UFMG.

Another notable finding in oral health is that individuals with RA have fewer teeth than those in the control group (16.4 *versus* 23.8; p< 0.001). This poor oral condition aligns with findings from other studies.[12,22] A primary explanation lies in the fact that the RA population is generally older[23] and has a higher prevalence of smoking compared to the non-RA population.[24] However, tooth loss cannot be fully explained by these factors and may be a result of the specific impact of RA itself. This was demonstrated in a cross-sectional study with 607 individuals with RA from the National Health and Nutrition Examination Survey (2009–2014).

Specifically, Hussein *et al.*,[22] tested three models adjusted for variables such as age and smoking, finding a statistically significant association between functional and non-functional dentition in relation to RA status. It is important to note that the difference in the number of teeth between individuals with RA and those in the control group could explain

the lack of association between the severity of periodontitis and RA. It is likely that, had the two groups had a similar number of teeth, individuals with RA would have shown an even higher prevalence and greater severity of periodontitis.

To assess RA status, patients underwent analysis of RF, ACPAs, ESR, DAS 28, and HAQ. None of these indicators showed significant differences between the RA population based on periodontal status. Specifically, regarding ACPAs, Karapetsa *et al.*,[25] found that within the group of RA patients with periodontitis, there was a greater number of positive ACPA cases compared to those without periodontitis.

However, González-Febles *et al.*,[26] did not find a significant association between positive and negative ACPAs in people with periodontitis, regardless of the classification of periodontitis used, even in the most severe stages.[16] On the other hand, our results align with several authors who report no association between these parameters and RA status or periodontitis.[25] Methodological differences, such as the definition of "case," classification of periodontitis, and severity criteria, may explain these discrepancies.

The results of Karapetsa *et al.*,[25] regarding the link between periodontitis and DAS 28 are intriguing. Similar to our findings, the authors did not find a significant association with DAS 28 when it was considered as a primary outcome evaluated dichotomously.

However, they found an association when DAS 28 was evaluated as a mean, further confirming the importance of methodological aspects when linking RA parameters and periodontitis. Furthermore, our results confirm that participants with RA have elevated CRP values compared to systemically healthy individuals (9.08 *versus* 4.01, $p< 0.001$), reflecting the inflammatory process itself. This finding supports the use of CRP as a diagnostic and prognostic marker for RA.[27]

The possibility that periodontitis may act as a risk indicator for the development of RA is relatively recent. A systematic review has been conducted, reporting a potential worsening of RA clinical activity in patients with periodontitis.[28]

Specifically, three studies with 116 participants with RA were grouped for quantitative synthesis, finding a statistically significant association (CI, 95% 0.74 [0.25–1.24], $p< 0.001$).[28] Our results are consistent with those of Hussain *et al.*,[28] showing a higher occurrence of RA in patients with periodontitis compared to those without periodontitis (73.3% *versus* 26.7%, $p= 0.026$).

Finally, the sites with mild periodontitis (PD = 4 mm and CAL ≥ 3 mm) in our study remained significant in the final adjusted model for the presence of RA. In this regard, the literature reveals contradictory findings. Varshney *et al.*,[29] reported variable results depending on the periodontal parameters used. They found that those with clinical attachment loss >2 mm presented an odds ratio (OR) of 2.89 for having RA compared to those with 2 mm ($p= 0.01$).

On the other hand, Silvestre-Rangil *et al.*,[30] did not find an association between RA and cases with PD of 1–3 mm, PD of 4–5 mm, or clinical CAL. Several factors may explain these differences. Heterogeneity in the definitions used, as well as the thresholds applied, could impact the associations.[28]

Additionally, some authors found an association between periodontal severity indicators and RA activity but did not use adjusted models, which limits the ability to control for potential confounding factors.[25]

Limitations

Despite confirming a significant bidirectional association between RA and periodontitis, the case-control design of this study means

that causal relationships cannot be established. Moreover, the age difference between the groups is a limitation that should be considered when interpreting the results. Future studies evaluating the association between RA and periodontitis should consider matching the groups by age.

CONCLUSION

This case-control study demonstrated that individuals with RA have a higher prevalence of periodontitis, a higher percentage of BOP, and more sites with CAL and altered PD.

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CONFLICT OF INTERESTS

The authors declare no conflict of interest.

ETHICS APPROVAL

Study approved by the Ethics Committee of the Faculty of Dentistry, University of the Republic Registration number: 091900-000248-17; and Ethics Committee of the Faculty of Dentistry, Federal University of Minas Gerais, Registration number: CAAE 37241214.2.0000.5097

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