

Association Between Rheumatoid Arthritis Disease Duration and Periodontal Health: A Retrospective Observational Study

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Abstract

Background

Rheumatoid arthritis (RA) is a chronic inflammatory disease that may be associated with alterations in periodontal health and disease. However, the relationship between RA duration and periodontal status remains incompletely characterized. This study evaluated the association between RA duration and periodontal disease.

Methodology

This retrospective observational study included 180 patients with RA. Patients were categorized according to RA duration as <5 years, 5-10 years, and >10 years, with 60 patients in each group. Periodontal parameters included the plaque index, bleeding on probing (BOP), probing pocket depth (PPD), clinical attachment level (CAL), and the number of missing teeth. Demographic and rheumatologic variables were also extracted. Between-group comparisons were performed using appropriate parametric and nonparametric tests. Multivariable linear regression was used to assess associations between RA duration and continuous periodontal outcomes, while multivariable logistic regression was used to evaluate severe periodontitis.

Results

Patients with RA for a duration of >10 years had significantly higher plaque index, BOP, PPD, and CAL than those with shorter disease duration. The mean plaque index increased from 0.99 ± 0.33 to 1.90 ± 0.29 , BOP from $21.49 \pm 9.23\%$ to $53.16 \pm 9.25\%$, PPD from 2.44 ± 0.48 mm to 4.11 ± 0.36 mm, and CAL from 1.67 ± 0.57 mm to 5.14 ± 0.85 mm (all $p < 0.001$). The number of missing teeth increased from 1.20 ± 1.10 to 4.60 ± 1.40 ($p < 0.001$). After adjustment for age, sex, smoking status, diabetes mellitus, body mass index, plaque index, and biologic disease-modifying antirheumatic drug use, RA duration remained significantly associated with CAL ($B = 0.158$, 95% confidence interval (CI): 0.130-0.185; $p < 0.001$) and PPD ($B = 0.073$, 95% CI: 0.056-0.090; $p < 0.001$). However, RA duration was not significantly associated with severe periodontitis (odds ratio = 1.01, 95% CI: 0.94-1.08; $p = 0.761$).

Conclusions

Longer RA duration was associated with progressively poorer periodontal health, particularly greater periodontal pocketing and attachment loss. Regular periodontal assessment may be warranted in patients with long-standing RA. These findings highlight the potential value of integrating periodontal evaluation into the routine management of patients with prolonged RA. Further longitudinal studies are needed to clarify the temporal relationship between RA duration and periodontal deterioration.

Categories: Dentistry

Keywords: clinical attachment loss, oral health, periodontal pocket, periodontitis, rheumatoid arthritis

Introduction

Rheumatoid arthritis (RA) is a chronic systemic autoimmune inflammatory disease characterized by persistent synovial inflammation, progressive joint damage, and functional impairment [1]. Although primarily considered a musculoskeletal disorder, RA is associated with a broad range of extra-articular manifestations and systemic inflammatory consequences [1]. Periodontitis is a chronic inflammatory disease of the supporting tissues of the teeth that can result in periodontal attachment loss, alveolar bone destruction, and tooth loss [2]. Both conditions share several biological features, including dysregulated

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immune responses, chronic inflammation, and overlapping environmental and host-related risk factors. Increasing evidence suggests a clinically relevant association between RA and periodontitis [3,4].

The potential relationship between RA and periodontitis may be explained by interactions between periodontal microbial dysbiosis and systemic immune dysregulation. Periodontal pathogens and their inflammatory products may contribute to systemic immune activation, whereas the persistent inflammatory state associated with RA may influence periodontal tissue susceptibility and disease progression [4,5]. Previous observational studies and systematic reviews have reported an association between the two diseases, although the magnitude and direction of this relationship have varied across studies [3-6]. Importantly, differences in smoking, metabolic conditions, disease activity, medication use, periodontal definitions, and study populations may contribute to the observed heterogeneity.

Periodontal disease severity is clinically assessed using parameters such as plaque accumulation, bleeding on probing (BOP), probing pocket depth (PPD), and clinical attachment level (CAL) [7]. The 2018 periodontal classification provides a standardized framework for staging periodontitis according to disease severity and complexity [8]. Although the association between RA and periodontal disease has been extensively investigated, relatively limited attention has been given to whether the duration of RA is associated with progressive differences in periodontal health [4,5]. Understanding this relationship may be clinically relevant because prolonged systemic inflammatory exposure and differences in disease activity or treatment over time could potentially coincide with changes in periodontal status [5,9]. However, such an association should be interpreted cautiously because the observational nature of available evidence does not establish causality.

Therefore, this retrospective observational cohort study aimed to evaluate the association between the duration of RA and periodontal health. The primary objective of this study was to assess the association between RA disease duration and periodontal health, including plaque index, BOP, PPD, CAL, and tooth loss. The secondary objective was to determine whether RA disease duration was associated with severe periodontitis and continuous periodontal outcomes after adjustment for potential confounding factors.

Materials And Methods

Study design and setting

This retrospective observational study was conducted in the Department of Periodontics, Kamineni Institute of Dental Sciences, Narketpally, India, from May to October 2025. Electronic medical records and dental case files from January 2021 to March 2025 were retrospectively reviewed. The study population was categorized according to the duration of RA at the time of the recorded periodontal examination, allowing comparison of periodontal health across different durations of systemic disease.

Ethical approval and confidentiality

The study protocol was reviewed and approved by the Institutional Ethics Committee of Kamineni Institute of Dental Sciences (IEC No. KIDS/IEC/2025/04/S12; approved on April 29, 2025). Written informed consent was obtained from the participants in accordance with the requirements of the Institutional Ethics Committee. Patient confidentiality was maintained throughout the study, and identifying information was not included in the study database.

Eligibility criteria

Eligible records were identified by systematic review of the institutional electronic health database and corresponding dental case files from January 2021 to March 2025. All patients meeting the eligibility criteria listed in Table 1 were extracted. RA duration was calculated as the exact interval (in years) between the documented date of RA diagnosis (from rheumatology records) according to the 2010 American College of Rheumatology/European League Against Rheumatism (ACR/EULAR) classification criteria [10] and the date of the most recent eligible periodontal examination (from dental records). Patients were then stratified into three pre-specified groups: Group I, RA duration <5 years; Group II, RA duration 5-10 years; and Group III, RA duration >10 years. Within each duration stratum, records were sorted by the date of periodontal examination (most recent first), and the first 60 consecutive eligible patients were included, yielding a final sample of 180 patients (60 per group). This consecutive, equal-allocation strategy was adopted to achieve the pre-calculated sample size while maintaining balanced group sizes for the primary comparative analyses.

Inclusion criteria	Exclusion criteria
Patients aged ≥ 18 years	Documented periodontal therapy within the preceding six months
Documented diagnosis of RA according to the 2010 ACR/EULAR classification criteria	Current orthodontic treatment
At least 20 natural teeth present.	Pregnancy or lactation
Complete periodontal examination record available	Previous head and neck radiotherapy
Panoramic or full-mouth intraoral radiographs are available	Another autoimmune or connective tissue disorder
Complete rheumatologic records documenting the date or duration of RA diagnosis	Incomplete clinical or radiographic information

TABLE 1: Eligibility criteria for study participants.

RA: rheumatoid arthritis; ACR/EULAR: American College of Rheumatology/European League Against Rheumatism

Sample size estimation

The sample size was calculated a priori using G*Power software version 3.1.9.7 (Heinrich-Heine-Universität Düsseldorf, Düsseldorf, Germany). A moderate effect size (Cohen's $f = 0.25$) was derived from a pilot study that assessed periodontal indices in patients with periodontitis and rheumatoid arthritis [11]. Using this effect size, along with 80% statistical power, a two-sided significance level of 5%, and three RA duration groups, the minimum required sample size was calculated to be 159 participants. To account for approximately 10% potential loss due to incomplete clinical or radiographic records, the required sample size was increased to approximately 177 participants. To facilitate equal allocation across the three study groups, the final sample was rounded to 180 patients (60 patients per group).

Patient selection and classification according to rheumatoid arthritis duration

Eligible records were identified from the institutional electronic health database and the corresponding dental records. The date of RA diagnosis was obtained from the rheumatology records, and the date of the most recent eligible periodontal examination was obtained from the dental records. RA duration was calculated as the interval between the documented date of diagnosis and the date of periodontal examination used for the study. Patients were subsequently categorized into three prespecified groups: Group I, RA duration < 5 years; Group II, RA duration 5-10 years; and Group III, RA duration > 10 years. An approximately equal number of eligible records were included in each group, with a final sample size of 60 patients per group. Smoking status was categorized as current smoker, former smoker, or never smoker for descriptive analyses. For multivariable regression analyses, smoking status was dichotomized as current smoker versus non-smoker, with former and never smokers combined into the non-smoker reference category.

Data collection

Two calibrated investigators (DS, KJ) independently reviewed the eligible clinical records and radiographs. Demographic variables included age, sex, body mass index (BMI), smoking status, and alcohol consumption. Rheumatologic variables included duration of RA, Disease Activity Score-28 (DAS28), rheumatoid factor (RF) status, anti-cyclic citrullinated peptide (anti-CCP) antibody status, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), current RA medications, and documented comorbidities. DAS28 is a validated composite measure that incorporates tender and swollen joint counts, an acute-phase inflammatory marker, and a patient-reported general health assessment [12]. DAS28 is an open-access, freely available source, and specific permission is not required. Medication records were categorized according to the documented use of disease-modifying antirheumatic drugs (DMARDs), biologic agents, and corticosteroids.

Periodontal assessment

The periodontal variables were obtained from the documented periodontal examinations performed during routine clinical care by an experienced periodontist (PG). The periodontal parameters included the plaque index, BOP, PPD, and CAL [13]. All examinations were performed using a calibrated UNC-15 periodontal probe by an experienced periodontist (PG), as documented in the clinical records. BOP was recorded as the presence or absence of bleeding following periodontal probing and was expressed as the percentage of examined sites demonstrating bleeding. PPD was recorded in millimeters as the distance from the gingival

margin to the base of the periodontal pocket. CAL was recorded in millimeters as the distance from the cemento-enamel junction to the base of the periodontal pocket and was selected as the primary periodontal outcome because it reflects cumulative periodontal attachment loss. The mean CAL is the primary outcome, and mean PPD, BOP, plaque index, number of missing teeth, and periodontitis stage are secondary outcomes.

Periodontitis classification

Periodontal diagnoses documented in the eligible records were classified according to the 2018 Classification of Periodontal and Peri-Implant Diseases and Conditions [14]. Staging was assigned retrospectively from the existing clinical and radiographic records using the 2018 criteria; no additional prospective examination or standardized staging protocol was applied. Patients were categorized as having periodontal health, gingivitis, Stage I periodontitis, Stage II periodontitis, Stage III periodontitis, or Stage IV periodontitis, according to the clinical and radiographic information available in their records [8]. For the secondary regression analysis, severe periodontitis was defined as Stage III or Stage IV disease.

Examiner calibration and reliability

To assess the reproducibility of retrospective data extraction, two investigators independently reviewed 20 randomly selected records that were not included in the final analysis. Calibration was performed before the extraction of the study dataset. Inter-examiner agreement for continuous variables was assessed using the intraclass correlation coefficient (ICC), while agreement for categorical variables was assessed using Cohen's kappa coefficient. The ICC values ranged from 0.86 to 0.94, while Cohen's kappa values ranged from 0.85 to 0.92, indicating excellent inter-examiner agreement. An ICC or kappa value of ≥ 0.80 was considered indicative of excellent reliability.

Statistical analysis

All statistical analyses were performed using SPSS Statistics version 29.0 (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for normality using the Shapiro-Wilk test and summarized as mean $\pm$ standard deviation (SD) when normally distributed and as median with interquartile range (IQR) when non-normally distributed. Categorical variables were summarized as frequencies and percentages. Differences among the three RA duration groups were assessed using one-way analysis of variance (ANOVA) for normally distributed continuous variables and the Kruskal-Wallis test for non-normally distributed continuous variables. For significant omnibus comparisons, pairwise independent-samples t-tests with Bonferroni adjustment were used for normally distributed variables, whereas pairwise Mann-Whitney U tests with Bonferroni adjustment were used for non-normally distributed variables. Categorical variables were compared using the chi-square test. Effect sizes were reported to quantify the magnitude of between-group differences, with eta squared (η^2) used for ANOVA and epsilon squared (ϵ^2) for Kruskal-Wallis analyses.

Multivariable linear regression analyses were performed to evaluate the association between RA disease duration and continuous periodontal outcomes, specifically CAL and PPD, after adjustment for potential confounding variables. Multivariable logistic regression was performed for the binary outcome of severe periodontitis, defined as stage III/IV disease. The regression models included age, sex, smoking status, diabetes mellitus, BMI, plaque index, and biologic DMARD use as covariates. Covariate selection was prespecified based on clinical relevance and the potential of each variable to confound the association between RA disease duration and periodontal outcomes. The models were designed to adjust for major demographic, behavioral, metabolic, and oral health-related confounders rather than for all rheumatologic disease characteristics. Consequently, RA disease activity (DAS28) and serological markers were not included as covariates in the primary models. Although disease activity may influence periodontal outcomes through systemic inflammatory pathways and residual confounding by DAS28 therefore cannot be excluded, the primary analysis was intentionally restricted to the pre-specified non-rheumatologic covariates. Regression coefficients (B), standard errors (SEs), 95% confidence intervals (CIs), and p-values were reported for the linear regression models, whereas odds ratios (ORs), 95% CIs, and p-values were reported for the logistic regression model. All statistical tests were two-sided, and a p-value < 0.05 was considered statistically significant.

Results

A total of 180 patients with RA were included and equally distributed across the three RA duration groups, with 60 (33.3%) patients in each group: Group I (< 5 years), Group II (5–10 years), and Group III (> 10 years). Patients in the > 10 -year group were older than those in the other groups ($p = 0.049$). Females predominated across all three groups, with 45 (75.0%), 39 (65.0%), and 40 (66.7%) patients, respectively, with no significant difference in sex distribution ($p = 0.448$). BMI, smoking status, and alcohol consumption did not differ significantly between the groups. In contrast, DAS28 scores increased progressively with RA duration ($p < 0.001$). RF positivity increased from 29 (48.3%) in Group I to 48 (80.0%) in Group III ($p = 0.001$), while anti-CCP positivity increased from 25 (41.7%) to 42 (70.0%) ($p = 0.007$). Comorbidities were present in 7 (11.7%), 17 (28.3%), and 17 (28.3%) patients, respectively ($p = 0.042$) (Table 2).

Variable		Group I (<5 years), n = 60	Group II (5-10 years), n = 60	Group III (>10 years), n = 60	Test statistic	P- value
Age (years)	Mean ± SD	47.49 ± 11.45	47.77 ± 7.96	51.79 ± 12.04	F = 3.06	0.049*
Sex, n (%)	Female	45 (75.0%)	39 (65.0%)	40 (66.7%)	$\chi^2 = 1.61$	0.448
	Male	15 (25.0%)	21 (35.0%)	20 (33.3%)		
BMI (kg/m ²)	Mean ± SD	25.93 ± 3.71	25.24 ± 3.14	25.28 ± 3.07	F = 0.8	0.451
Smoking status, n (%)	Current	8 (13.3%)	7 (11.7%)	8 (13.3%)	$\chi^2 = 0.59$	0.964
	Former	15 (25.0%)	13 (21.7%)	12 (20.0%)		
	Never	37 (61.7%)	40 (66.7%)	40 (66.7%)		
Alcohol consumption, n (%)	No	50 (83.3%)	45 (75.0%)	47 (78.3%)	$\chi^2 = 1.27$	0.531
	Yes	10 (16.7%)	15 (25.0%)	13 (21.7%)		
RA duration (years)	Mean ± SD	2.72 ± 1.04	7.37 ± 1.57	16.24 ± 3.19	F = 618.88	< 0.001*
DAS28 score	Mean ± SD	3.25 ± 0.66	4.05 ± 0.64	5.14 ± 0.59	F = 135.72	< 0.001*
Rheumatoid factor positivity, n (%)	Negative	31 (51.7%)	19 (31.7%)	12 (20.0%)	$\chi^2 = 13.63$	0.001*
	Positive	29 (48.3%)	41 (68.3%)	48 (80.0%)		
Anti-CCP positivity, n (%)	Negative	35 (58.3%)	28 (46.7%)	18 (30.0%)	$\chi^2 = 9.83$	0.007*
	Positive	25 (41.7%)	32 (53.3%)	42 (70.0%)		
Presence of comorbidities, n (%)	No	53 (88.3%)	43 (71.7%)	43 (71.7%)	$\chi^2 = 6.32$	0.042*
	Yes	7 (11.7%)	17 (28.3%)	17 (28.3%)		

TABLE 2: Demographic, clinical, and rheumatoid arthritis-related characteristics of the study participants according to rheumatoid arthritis disease duration.

Continuous variables are presented as mean ± standard deviation (SD), and categorical variables as frequency and percentage. Normally distributed continuous variables were compared using one-way analysis of variance (ANOVA), while non-normally distributed variables were compared using the Kruskal–Wallis test. Categorical variables were compared using the chi-square (χ^2) test.

*: P-values <0.05 were considered statistically significant.

BMI: body mass index; DAS28: Disease Activity Score-28; RA: rheumatoid arthritis; Anti-CCP: anti-cyclic citrullinated peptide

Periodontal parameters showed progressive deterioration with increasing RA duration. The mean plaque index increased from 0.99 ± 0.33 in Group I to 1.90 ± 0.29 in Group III ($p < 0.001$), and BOP increased from $21.49 \pm 9.23\%$ to $53.16 \pm 9.25\%$ ($p < 0.001$). The mean PPD increased from 2.44 ± 0.48 mm to 4.11 ± 0.36 mm ($p < 0.001$). The primary outcome, CAL, increased from 1.67 ± 0.57 mm in Group I to 5.14 ± 0.85 mm in Group III ($p < 0.001$). The number of missing teeth also increased from 1.20 ± 1.10 to 4.60 ± 1.40 ($p < 0.001$) (Table 3).

Periodontal parameter	Group I (<5 years), n = 60	Group II (5-10 years), n = 60	Group III (>10 years), n = 60	Test statistic	P- value	Effect size
Plaque index, mean $\pm$ SD	0.99 $\pm$ 0.33	1.41 $\pm$ 0.33	1.90 $\pm$ 0.29	F = 119.47	< 0.001*	η^2 = 0.574
Bleeding on probing (%), mean $\pm$ SD	21.49 $\pm$ 9.23	35.1 $\pm$ 9.17	53.16 $\pm$ 9.25	F = 178.28	< 0.001*	η^2 = 0.668
Probing pocket depth (mm), mean $\pm$ SD	2.44 $\pm$ 0.48	3.26 $\pm$ 0.51	4.11 $\pm$ 0.36	F = 201.53	< 0.001*	η^2 = 0.695
Clinical attachment level (mm), mean $\pm$ SD	1.67 $\pm$ 0.57	3.21 $\pm$ 0.72	5.14 $\pm$ 0.85	F = 348.26	< 0.001*	η^2 = 0.797
Number of missing teeth, mean $\pm$ SD	1.20 $\pm$ 1.10	2.80 $\pm$ 1.30	4.60 $\pm$ 1.40	H = 99.57	< 0.001*	ϵ^2 = 0.551

TABLE 3: Comparison of periodontal health parameters according to rheumatoid arthritis disease duration.

Continuous variables are presented as mean $\pm$ standard deviation (SD); normally distributed variables were compared using one-way analysis of variance (ANOVA), whereas non-normally distributed variables were compared using the Kruskal–Wallis test.

*: P-values <0.05 were considered statistically significant.

Post hoc analysis confirmed significant differences in the plaque index, BOP, PPD, and CAL among the three groups. The largest between-group difference was observed for CAL between Groups I and III (Cohen's d = -4.79), followed by PPD (Cohen's d = -3.92). The number of missing teeth also differed significantly between each pair of groups (all adjusted p < 0.001) (Table 4).

Parameter	Pairwise comparison	Test statistic	Adjusted p-value	Effect size
Plaque index	Group I vs. Group II	t = -6.90	< 0.001*	d = -1.26
	Group I vs. Group III	t = -15.72	< 0.001*	d = -2.87
	Group II vs. Group III	t = -8.47	< 0.001*	d = -1.55
Bleeding on probing (%)	Group I vs. Group II	t = -8.10	< 0.001*	d = -1.48
	Group I vs. Group III	t = -18.77	< 0.001*	d = -3.43
	Group II vs. Group III	t = -10.74	< 0.001*	d = -1.96
Probing pocket depth (mm)	Group I vs. Group II	t = -8.97	< 0.001*	d = -1.64
	Group I vs. Group III	t = -21.49	< 0.001*	d = -3.92
	Group II vs. Group III	t = -10.63	< 0.001*	d = -1.94
Clinical attachment level (mm) (primary outcome)	Group I vs. Group II	t = -13.10	< 0.001*	d = -2.39
	Group I vs. Group III	t = -26.26	< 0.001*	d = -4.79
	Group II vs. Group III	t = -13.38	< 0.001*	d = -2.44
Number of missing teeth	Group I vs. Group II	z = -5.98	< 0.001*	—
	Group I vs. Group III	z = -8.77	< 0.001*	—
	Group II vs. Group III	z = -5.99	< 0.001*	—

TABLE 4: Pairwise comparisons of periodontal health parameters between rheumatoid arthritis disease duration groups.

Pairwise comparisons for normally distributed variables were performed using Bonferroni-adjusted independent-samples t-tests with Cohen's d effect sizes, whereas non-normally distributed variables were analyzed using Mann–Whitney U tests with Bonferroni correction.

*: P-values <0.05 were considered statistically significant.

In multivariable linear regression, RA duration remained significantly associated with CAL after adjustment for age, sex, smoking, diabetes mellitus, BMI, plaque index, and biologic DMARD use (B = 0.158, 95% CI: 0.130-0.185; p < 0.001). The plaque index was also significantly associated with CAL (B = 1.036, p < 0.001), while the model explained 76.0% of the variance (adjusted R² = 0.749) (Table 5).

Predictor	B	Standard error	t value	95% CI	P-value
Constant	-0.693	0.567	-1.22	-1.812 to 0.427	0.224
RA duration (per year)	0.158	0.014	11.29	0.130 to 0.185	<0.001*
Age (per year)	0.011	0.006	1.83	-0.001 to 0.023	0.068
Sex (female vs. male) (reference: male)	-0.132	0.130	-1.02	-0.388 to 0.124	0.309
Current smoking (vs. non-smoker) (reference: non-smoker)	-0.275	0.180	-1.53	-0.630 to 0.080	0.128
Diabetes mellitus (yes vs. no) (reference: no)	-0.028	0.168	-0.17	-0.361 to 0.304	0.866
BMI (per kg/m ²)	0.027	0.018	1.47	-0.009 to 0.062	0.144
Plaque index (per unit score)	1.036	0.169	6.14	0.703 to 1.369	<0.001*
Biologic DMARD use (yes vs. no) (reference: no)	0.149	0.134	1.11	-0.115 to 0.413	0.267

TABLE 5: Multivariable linear regression analysis of factors associated with clinical attachment level.

The model was adjusted for age, sex, smoking status, diabetes mellitus, body mass index, plaque index, and biologic DMARD use.

*: P-values <0.05 were considered statistically significant.

B: unstandardized regression coefficient; BMI: body mass index; CI: confidence interval; DMARD: disease-modifying antirheumatic drug; RA: rheumatoid arthritis

Similarly, RA duration was significantly associated with PPD (B = 0.073, 95% CI: 0.056-0.090; p < 0.001), with the model explaining 64.6% of the variance (adjusted R² = 0.629) (Table 6).

Predictor	B	SE	t	95% CI	P-value
Constant	2.017	0.353	5.71	1.320 to 2.715	<0.001*
RA duration (per year)	0.073	0.009	8.11	0.056 to 0.090	<0.001*
Age (per year)	0.008	0.004	2.00	0.000–0.016	0.047 *
Sex (female vs. male) (reference: male)	0.038	0.081	0.47	-0.122 to 0.197	0.642
Current smoking (vs. non-smoker) (reference: non-smoker)	-0.114	0.112	-1.01	-0.335 to 0.107	0.312
Diabetes mellitus (yes vs. no) (reference: no)	0.047	0.105	0.45	-0.160 to 0.254	0.656
BMI (per kg/m ²)	-0.019	0.011	-1.72	-0.042 to 0.003	0.087
Plaque index (per unit score)	0.485	0.105	4.62	0.278 to 0.692	<0.001*
Biologic DMARD use (yes vs. no) (reference: no)	0.043	0.083	0.51	-0.122 to 0.207	0.607

TABLE 6: Multivariable linear regression analysis of factors associated with probing pocket depth.

The model was adjusted for age, sex, smoking status, diabetes mellitus, body mass index, plaque index, and biologic DMARD use.

*: P-values <0.05 were considered statistically significant.

B: unstandardized regression coefficient; BMI: body mass index; CI: confidence interval; DMARD: disease-modifying antirheumatic drug; RA: rheumatoid arthritis

For severe periodontitis (stage III/IV), multivariable logistic regression did not demonstrate a significant association with RA duration (OR = 1.01, 95% CI: 0.94-1.08; p = 0.761). None of the other variables included

in the model were significantly associated with severe periodontitis (Table 7).

Predictor	B	SE	Odds ratio (95% CI)	P-value
Constant	-1.080	1.500	—	0.471
RA duration (per year)	0.011	0.036	1.01 (0.94–1.08)	0.761
Age (per year)	0.017	0.015	1.02 (0.99–1.05)	0.256
Sex (female vs. male) (reference: male)	0.266	0.344	1.30 (0.66–2.56)	0.440
Current smoking (vs. non-smoker) (reference: Non-smoker)	0.682	0.480	1.98 (0.77–5.06)	0.155
Diabetes mellitus (yes vs. no) (reference: No)	0.029	0.435	1.03 (0.44–2.41)	0.946
BMI (per kg/m²)	-0.061	0.048	0.94 (0.86–1.03)	0.205
Plaque index (per unit score)	0.746	0.453	2.11 (0.87–5.13)	0.100
Biologic DMARD use (yes vs. no) (reference: No)	0.087	0.349	1.09 (0.55–2.16)	0.803

TABLE 7: Logistic regression analysis of factors associated with severe periodontitis.

The model was adjusted for age, sex, smoking status, diabetes mellitus, body mass index, plaque index, and biologic DMARD use.

*: P-values <0.05 were considered statistically significant.

B: unstandardized regression coefficient; BMI: body mass index; CI: confidence interval; DMARD: disease-modifying antirheumatic drug; RA: rheumatoid arthritis

Discussion

This study demonstrated a consistent association between longer RA duration and poorer periodontal health. Periodontal parameters, including plaque accumulation, BOP, PPD, CAL, and tooth loss, progressively deteriorated across the RA duration groups. These findings support the possibility that long-standing RA is associated with a greater periodontal disease burden. However, given the retrospective observational design, the findings should be interpreted as associations rather than evidence that an increase in RA duration directly causes periodontal deterioration.

The progressive increase in plaque accumulation and gingival bleeding with longer RA duration may reflect the interaction between systemic inflammation and oral health-related factors. Patients with longstanding RA may experience functional limitations involving the hands and upper extremities, which can make effective toothbrushing and interdental cleaning more difficult [15]. In addition, persistent inflammatory activity may contribute to an altered systemic inflammatory environment that could influence periodontal tissues [4]. Previous studies have reported a higher prevalence or severity of periodontal disease among individuals with RA, although the strength of this association has varied between studies [3-6]. A systematic review and meta-analysis reported evidence of a bidirectional relationship between RA and periodontitis while also emphasizing substantial heterogeneity among the available studies [4]. These observations support the need to consider periodontal health as part of the broader health assessment of patients with RA.

The deterioration in PPD and CAL with increasing RA duration is particularly relevant because these parameters reflect periodontal tissue destruction rather than inflammation alone. One possible explanation is the presence of overlapping inflammatory pathways in RA and periodontitis. Both diseases involve dysregulated innate and adaptive immune responses, production of proinflammatory mediators, connective tissue degradation, and osteoclast-mediated bone resorption [4,16]. The proposed relationship between periodontal infection and RA has also received considerable attention because periodontal pathogens, particularly Porphyromonas gingivalis, have been implicated in protein citrullination and the generation of anti-citrullinated protein antibodies [17]. Nevertheless, the biological relationship is complex, and the available evidence does not establish that periodontal infection is responsible for the development or progression of RA.

The observed increase in tooth loss among patients with longer disease duration indicates a cumulative oral health burden. Tooth loss may represent a long-term consequence of periodontal destruction, particularly when accompanied by increasing attachment loss and periodontal pocketing [18]. However, tooth loss is multifactorial and may result from dental caries, endodontic disease, trauma, or other reasons. Therefore,

the present findings should be interpreted as overall indicators of oral health deterioration rather than evidence that RA duration directly causes periodontal tooth loss.

Multivariate analyses provide additional support for an association between RA duration and periodontal tissue measurements after accounting for several recognized confounding factors. The persistence of the association after adjustment for age, sex, smoking, diabetes mellitus, BMI, oral hygiene status, and biologic DMARD use suggests that the observed relationship cannot be explained entirely by these measured variables. At the same time, the strong association between oral hygiene status and periodontal outcomes highlights the importance of modifiable local factors. This is clinically relevant because effective plaque control and regular professional periodontal care remain fundamental components of periodontal disease prevention and management, irrespective of the patient's systemic condition [19].

The finding that RA duration was not significantly associated with severe periodontitis when disease was categorized as stage III/IV is also important. This apparent difference from the continuous periodontal outcomes may partly reflect the loss of information that occurs when a continuous measure of periodontal destruction is converted into a categorical disease stage. Furthermore, periodontitis staging incorporates multiple clinical and radiographic considerations and is not determined solely by RA duration or a single periodontal measurement. Thus, longer RA duration should not be interpreted as an indication that an individual patient necessarily has severe periodontitis [8]. The large effect sizes observed for the continuous outcomes contrast with the nonsignificant logistic result. This discrepancy is expected because dichotomizing a continuous measure of attachment loss into Stage III/IV disease discards graded information, reduces statistical power, and incorporates additional staging criteria beyond CAL and PPD alone.

From a clinical perspective, these findings support closer collaboration between rheumatologists and dental professionals in the long-term care of patients with RA. Periodontal screening and regular periodontal maintenance may be particularly relevant for patients with long-standing disease. Early identification of gingival inflammation and periodontal attachment loss may allow appropriate preventive and therapeutic measures before substantial periodontal destruction occurs. However, the present findings should not be interpreted as evidence that periodontal treatment modifies RA progression; such a conclusion requires appropriately designed interventional or longitudinal studies. A systematic review and meta-analysis suggested potential interactions between periodontal treatment and systemic inflammatory activity in RA; however, the evidence remains heterogeneous [20].

Limitations

This study has several limitations. Its retrospective, single-center design limits causal inference and may restrict generalizability to other populations. Patient selection was restricted to individuals who had both complete rheumatologic records and a periodontal examination performed at the study center; consequently, the sample may over-represent patients with greater healthcare utilization or more clinically apparent oral symptoms. In addition, the decision to enrol exactly 60 patients in each duration group (rather than a consecutive series proportional to the natural prevalence of each duration) introduces a form of selection bias and means the relative sizes of the groups do not reflect the underlying distribution of RA duration in the source population. These factors limit the generalizability of the absolute prevalence estimates, although the within-study comparisons of periodontal parameters across duration groups remain valid.

The study relied on previously recorded clinical and radiographic information; therefore, some potentially relevant variables may not have been consistently documented. Periodontal staging was performed retrospectively by applying the 2018 classification criteria to the clinical and radiographic information already recorded in the charts; no prospective staging protocol was used. Likewise, the original periodontal examinations were conducted during routine clinical care by an experienced periodontist using a calibrated UNC-15 probe, but they were not performed under a uniform, prospectively defined research protocol with formal inter- or intra-examiner calibration of the clinical measurements themselves. Calibration was performed only for the subsequent retrospective data-extraction process.

Although the analyses adjusted for several important confounders, residual confounding from factors such as socioeconomic status, oral hygiene practices, dental attendance, dietary habits, detailed smoking exposure, and other medication-related factors cannot be excluded. Furthermore, although the number of missing teeth increased across the RA duration groups, the specific reasons for tooth loss, including periodontitis, dental caries, trauma, orthodontic treatment, or other causes, could not be consistently determined from the available records. Therefore, tooth loss should be interpreted as an indicator of cumulative oral health burden rather than being attributed exclusively to periodontal disease. In addition, conversion of continuous periodontal measurements into a binary severe periodontitis outcome reduced statistical power and may have contributed to the nonsignificant adjusted association with RA duration.

In particular, RA disease activity (DAS28) was not included in the multivariable models. Because DAS28 was strongly associated with disease duration and is a plausible determinant of periodontal tissue destruction, unmeasured confounding by disease activity remains possible. The absence of a non-RA control group also

prevented the determination of the extent to which periodontal findings differed from those in individuals without RA. In addition, radiographic assessment was based on available clinical records rather than a standardized prospective imaging protocol. Future multicenter longitudinal studies incorporating standardized periodontal examinations, appropriate control groups, detailed assessment of RA activity and treatment, documentation of the reasons for tooth loss, and repeated follow-up are needed to further clarify the temporal relationship between RA duration and periodontal health.

Conclusions

Longer RA disease duration was associated with progressively poorer periodontal health, reflected by greater plaque accumulation, gingival inflammation, PPD, CAL, and tooth loss. The associations of RA duration with CAL and PPD remained significant after adjustment for relevant demographic, behavioral, metabolic, and oral health-related confounders. These findings underscore the importance of regular periodontal evaluation and coordinated oral healthcare in patients with long-standing RA. Further longitudinal studies are needed to establish the temporal relationship and clarify whether prolonged RA contributes to the progression of periodontal disease.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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