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Association Between Rheumatoid Arthritis and Periodontitis: Stage Specific Treatment Protocol and Standardised Collaborative Model Between Dentistry and Rheumatoid Arthritis: A Case Control Study

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ABSTRACT

Aim: To investigate the correlation between periodontitis and rheumatoid arthritis (RA), and to evaluate periodontitis (PA) as a potential contributor for the development and progression of RA and generate model between RA and PA.

Materials and Methods: A matched case-control analysis was conducted with 170 patients diagnosed with RA (case group) and 170 non-RA individuals (control group), all recruited from the same cohort. The matching of cases and controls was done at a one to one ratio based on age, gender, socioeconomic status. Random sampling method was employed. The periodontal status of participants were assessed following the 2017 World Workshop criteria for Classifying Periodontal and Peri-Implant Diseases and Conditions. Diagnosis of RA and assessment of its severity was measured using the Disease Activity Score (DAS-28). Statistical analysis included multivariate logistic regression, odds ratio (OR) estimation, and significance testing with a confidence interval of 95%.

Result: The RA group exhibited significantly higher periodontal parameters—probing pocket depth (PPD) and clinical attachment loss (CAL)—and systemic inflammation, indicated by elevated erythrocyte sedimentation rate (ESR) values ($p < 0.05$). Mild periodontitis was more prevalent in the RA group (39.4%) than among controls (29.4%), with adjusted odds ratios ranging between 4.1 and 4.5. Binary logistic regression identified age, DAS-28 score, and ESR as significant predictors of RA, while educational status exerted a protective effect. Rheumatoid disease activity (DAS-28) correlated positively with the severity of periodontitis, with higher activity levels noted in patients with moderate to severe periodontal disease ($p = 0.004$). Overall, individuals with periodontitis had nearly threefold increased odds of RA (OR = 2.986; 95% CI: 1.871–4.765; $p < 0.05$). The association remained statistically strong after adjustment.

Conclusion: The findings demonstrate a significant link between periodontitis and RA. Individuals with periodontitis demonstrated a higher risk of developing RA, and RA patients with co-existing periodontitis, tends to have more severe disease activity.

Keywords: Periodontitis, Rheumatoid arthritis, Case-Control Study, Disease Activity Score (DAS-28)

Introduction

Rheumatoid arthritis (RA) is a chronic autoimmune disorder characterised by ongoing synovial inflammation, which results into gradual joint destruction, pain, and long-term functional impairment [1]. The worldwide prevalence of RA is estimated at 0.5–1%, with onset most commonly in middle age, although its precise etiology remains multifactorial—arising from underlying

genetic risk, environmental triggers, and a abnormal immune system activation [2]. Among environmental risk factors, periodontitis—a common long-standing condition involving the periodontal tissues—has garnered increasing attention due to its epidemiological association and shared pathogenic mechanisms with RA [3].

Growing evidence highlights that RA and periodontitis are linked not only epidemiologically but also through overlapping processes such as chronic systemic inflammation, immune

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dysregulation, and genetic predispositions [4]. The oral pathogen *Porphyromonas gingivalis*, which evades immune surveillance and promotes a pro-inflammatory microenvironment, plays a key role in protein citrullination via its unique peptidylarginine deiminase (PAD) enzymes. This process leads to the production of anti-citrullinated protein antibodies—a key feature in RA pathogenesis—thus providing a plausible mechanistic bridge between gum disease and joint autoimmunity [5].

Recent meta-analyses and case-control studies demonstrate that individuals with RA frequently exhibit increased severity of periodontitis compared to healthy controls, underscoring the bidirectional and possibly exacerbate nature of this association [6]. Key pro-inflammatory molecules tumour necrosis factor- α , interleukin-1 β , and interleukin-6 contribute to tissue destruction in both conditions and perpetuate a vicious cycle of systemic inflammation. Interventional studies further suggest that effective periodontal therapy can reduce RA disease activity, highlighting the need for the coordinated management strategies that includes both oral and rheumatological management [7-10].

Despite substantial progress in understanding this relationship, significant research gaps remain. These include a lack of longitudinal studies to clarify the temporal and causal nature of the RA-periodontitis link, incomplete profiling of oral microbiomes beyond classical pathogens, and an insufficient insight of the molecular, genetic, and immunological mechanisms involved. Furthermore, the formation of personalized, stage-specific treatment protocols and standardized collaborative care models between dentistry and rheumatology are yet to be fully explored [11,12].

In light of these unresolved issues, the present case-control study aims to evaluate the strength of the link between periodontitis and RA and to examine if periodontitis independently contributes to RA onset and progression. Addressing these questions may pave the way for improved mechanistic insights and optimized therapeutic strategies beneficial for both diseases.

Materials and Methods

Study Design and Setting

This analytical case-control investigation was conducted at the Rheumatology Clinic, Post Graduate Institute of Medical Sciences, over a 12-month period. Data collection involved structured, interview-based questionnaires as well as clinical examinations. Participants were recruited from both rheumatology and outpatient clinics.

Participants and Sampling

- Inclusion:** Diagnosed rheumatoid arthritis (RA) patients (cases), and non-RA controls recruited from general outpatient clinics. RA was diagnosed using the American Rheumatism Association's Revised Criteria [13].
- Exclusion:** Pregnant or lactating women; individuals requiring chronic antibiotic prophylaxis; those diagnosed with diabetes, hypertension, blood disorders, or coronary heart disease; smokers and those on immunosuppressants; and patients with recent periodontal therapy within the past 6 months.

Sampling and Recruitment

A random sampling approach was used, with selection based on willingness and diagnostic criteria. Matched sampling was implemented to control for demographic confounders including details such as age, sex, occupation, and socioeconomic status (evaluating using the BG Prasad scale) [14]. Investigators were recruited from Dental department, PGIMS, and trained prior to data collection. Intra-examiner reliability was ensured (kappa coefficient ≥ 0.80 for categorical data; ICC ≥ 0.80 for continuous data).

Sample Size Determination

Sample size was calculated for 99% confidence and 90% power, requiring 162 subjects per group. The final sample comprised 170 cases and 170 controls (total n=340), with an anticipated 5% drop-out rate, ensuring sufficient power for statistical analysis.

Data Collection Tools and Procedures

Clinical Assessment

Clinical assessment included structured interviews and oral examinations. The main survey instrument was a questionnaire developed through literature review, expert consultations, focus group discussions, and pilot-testing on 20 RA patients. After pilot testing, modifications for clarity and reliability were made; reliability (test-retest, Cronbach's alpha) reached 0.83.

Periodontal examination

Periodontitis is currently classified and evaluated based on the 2017 World Workshop criteria's for classifying Periodontal and Peri-Implant Diseases and Conditions, which established a widely accepted staging and grading system [15]. This framework focuses on disease severity and complexity, while also considering how quickly it progresses and the presence of contributing risk factors.

Staging is based on clinical attachment loss (CAL), radiographic bone loss, probing depth (PD), and tooth loss, categorized as:

- Stage I (initial periodontitis)
- Stage II (moderate periodontitis)
- Stage III (severe periodontitis with tooth loss risk)
- Stage IV (advanced, extensive tooth loss)

Clinical periodontal examination typically involves probing six sites per tooth, recording PD and CAL, and assessing bleeding on probing (BOP) as a marker of inflammation. Radiographs are used to quantify bone loss. Systemic modifiers like smoking and diabetes are incorporated into the grading process.

Rheumatoid Arthritis Evaluation

RA disease activity in the case group was assessed with the 28 joint Disease Activity Score (DAS 28), incorporating:

- Tender joint count (TJC28)
- Swollen joint count (SJC28)
- Erythrocyte Sedimentation Rate (ESR)
- Patient general health (GH) via 100-mm visual analog scale.

Ethics and Confidentiality

The study was approved by the Institutional Ethics Committee (PGIDS/IEC/2016/67) and complied with the principles of the Declaration of Helsinki as well as STROBE guidelines. All participants provided written informed consent. Strict

confidentiality was maintained by anonymizing all data and limiting access to the research team only.

Reduction of Bias

The cohort was randomized, selection bias was minimized by recruiting both cases and controls from the same clinics and matching demographic characteristics (age ± 2 years, gender, occupation, socioeconomic status). Any observed drop-outs (approx. 5%) were analyzed and found similar in characteristics to included participants.

Data Management and Statistical Analysis

Data were recorded in Microsoft Excel and subsequently analyzed using SPSS software (version 21). Logistic regression (unconditional and conditional), odds ratios, and 99% confidence intervals were calculated. The threshold for statistical significance was defined as $p \leq 0.05$. Confounders such as oral hygiene habits, socioeconomic status, and demographics were matched or statistically controlled wherever possible [16,17].

Results

Demographic Characteristics

The demographic characteristics of the case (RA) and control groups are summarized in Table 1. The mean age of participants in both groups was similar (cases: 39.58 ± 6.74 years; controls: 39.52 ± 6.779 years), with no statistically significant difference ($p = 0.729$). The gender distribution was identical in both groups, with a predominance of females (92.4%) over males (7.6%), and no significant difference between groups ($p > 0.05$). Socioeconomic status and occupational classification were also comparable between groups ($p > 0.05$). However, a

higher proportion of RA patients resided in rural areas (92.9%) compared to controls (80.6%).

Table 1: Demographic Characteristics of Cases and Control

Parameter	Cases	Control	P value
Age (years)	39.58 \pm 6.74	39.52 \pm 6.779	0.729
Gender			>0.05
Male	13(7.6%)	13(7.6%)	
Female	157(92.4%)	157(92.4%)	
Socioeconomic status			
Class I	18	17	
Class II	32	32	> 0.05
Class III	44	41	
Class IV	70	74	
Class V	6	6	
Location			
Rural	158(92.9%)	137(80.6%)	
Urban	12(7.1%)	33(19.4%)	
Occupation			Unadjusted odds ratio (95%CI)
Up to semiskilled	160(94.7%)	168(98.8%)	1.0
Up to clerical	6(3.6%)	1(1.6%)	0.2 (0.02-1.3)
Semi-professional and above	4(1.8%)	1(1.6%)	0.3 (0.03-3.1)

Table 2: Strength Of Association of Periodontitis Severity with Rheumatoid Arthritis in Case Group and Control Group

	Case Group (With RA)		Control Group (Without RA)		Total		Unadjusted odds ratio (95%CI)	Adjusted* odds ratio (95%CI)	Adjusted† odds ratio (95%CI)
	N	%	N	%	N	%			
Healthy	2(1.2)	1.2	7	4.1	9	2.6	1.0	1.0	1.0
Gingivitis	37(21.8)	21.8	73	42.9	110	32.4	1.0	1.0	1.0
Mild	67(39.4)	39.4	50	29.4	117	34.4	4.3(1.9-10.0)	4.3(1.9-10.0)	4.1(1.6-10.3)
Moderate	43(25.3)	25.3	30	17.6	73	21.5	1.6(0.7-3.6)	1.8(0.7-4.3)	1.5(0.6-3.7)
Severe	21(12.4)	12.4	10	5.9	31	9.1	1.5(0.6-3.5)	1.5(0.6-3.8)	1.6(0.6-4.2)
Total	170	100	170	100	340	100			

$\chi^2 = 23.248$ df = 1 $p < 0.05$ significant

Association of Periodontitis Severity with Rheumatoid Arthritis

Table 2 shows the distribution of periodontal disease severity between RA cases and controls. Mild periodontitis was significantly more prevalent in the RA group (39.4%) compared to controls (29.4%). The unadjusted odds ratio for RA in patients with mild periodontitis was 4.3 (95% CI: 1.9–10.0), and the adjusted odds ratio remained significant even after controlling for demographic variables (OR: 4.5, 95% CI: 1.9–10.9) and oral hygiene practices (OR: 4.1, 95% CI: 1.6–10.3). A statistically significant difference was observed ($\chi^2 = 23.248$, $p < 0.05$).

*Adjusted for demographics age, gender, location, education, occupation and socioeconomic status

†Adjusted for oral hygiene practices

RA- Rheumatoid arthritis

The forest plot (Figure 1) visually represents the strength of the association between different severities of periodontitis and rheumatoid arthritis (RA). It shows unadjusted odds ratios (ORs) alongside two adjusted ORs accounting for demographic factors

(age, gender, location, education, occupation, socioeconomic status) and oral hygiene practices. Mild periodontitis exhibits the strongest association with RA, with an unadjusted OR of 4.3 (95% CI: 1.9–10.0), which remains significant after adjustment (adjusted OR* 4.5, 95% CI: 1.9–10.9; adjusted OR† 4.1, 95% CI: 1.6–10.3). Moderate and severe periodontitis show weaker, statistically non-significant associations with RA, with ORs close to 1.5 and wide confidence intervals overlapping 1. Healthy and gingivitis categories serve as references with ORs set at 1. These findings indicate that mild periodontitis severity is a significant risk factor for RA, emphasizing the need for early periodontal assessment and intervention in RA patients.

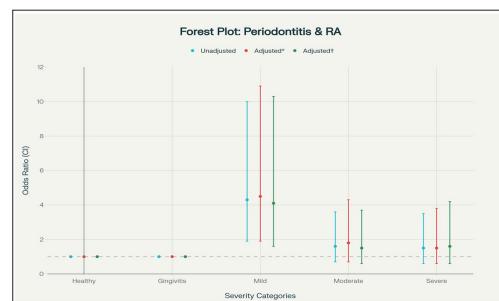


Figure 1: Forest Plot

Relationship Between Rheumatoid Disease Activity and Periodontal Staging

As presented in Table 3, there was a significant association between the severity of periodontal disease and rheumatoid arthritis disease activity ($p = 0.004$). RA patients with moderate to severe periodontitis showed higher frequencies of moderate (55.3%) and high disease activity (10%) as compared to those with gingivitis or healthy periodontium.

Table 3: Rheumatoid Diseases Activity Compared with the Severity of Periodontal Disease in Case Group(with RA)

	Healthy		Gingivitis		Mild		Moderate		Severe		Total	
	N	%	N	%	N	%	N	%	N	%	N	%
Low diseases activity	2	1.2	13	7.6	27	15.9	13	7.6	4	2.4	59	34.7
Moderate diseases activity	0	0	22	12.9	39	22.9	22	12.9	11	6.5	94	55.3
High diseases activity	0	0	2	1.2	1	0.6	8	4.7	6	3.5	17	10
Total	2	1.2	37	21.8	67	39.4	43	25.3	21	12.4	170	100

$\chi^2 = 22.541$ df = 8 $p = 0.004$ significant *

RA- Rheumatoid arthritis

Binary Logistic Regression Analysis

Binary logistic regression (Table 4) indicated that several variables were significantly associated with RA in individuals with periodontitis. Increased age (OR = 1.145, 95% CI: 1.097–1.194, $p < 0.001$), higher DAS scores (OR = 1.631, 95% CI: 1.337–1.989, $p < 0.001$), and elevated ESR (OR = 1.025, 95% CI: 1.008–1.042, $p = 0.004$) were significant predictors. Educational status demonstrated a protective effect (OR = 0.731, 95% CI: 0.591–0.859, $p < 0.001$). The spider plot (Figure 2) visually illustrates the relative strength of association of several variables with rheumatoid arthritis (RA) in patients evaluated via binary logistic regression. Among the factors analyzed, DAS score exhibits the strongest association (OR=1.631), followed by age (OR=1.145), duration of illness (OR=1.142), and erythrocyte sedimentation rate (ESR) (OR=1.025). Educational status is negatively associated with RA (OR=0.731), indicating a protective effect or lower odds in higher educational strata. The radial representation allows for immediate comparison of the magnitude and direction of each variable's influence, highlighting which factors contribute more prominently to RA risk in this cohort. This visual aid complements the tabular regression output by offering an intuitive, at-a-glance understanding of complex multivariable relationships in the data.

Table 4: Binary Logistic Regression (Forward Conditioning) Odds And B Coefficient for Association of Rheumatoid Arthritis with Periodontitis

Variable	β coefficient	Standard error	Wald ratio	Periodontitis status Odd's ratio	95%CI	p value
Age (years)	0.121	0.019	39.351	1.145	1.097-1.194	0.00*
Educational states	-0.333	0.080	17.22	0.731	0.591-0.859	0.00*
ESR	0.020	0.008	5.801	1.025	1.008-1.042	0.004*
Duration of illness	0.122	0.030	16.797	1.142	1.094-1.192	0.052
DAS score	0.492	0.113	18.865	1.631	1.337-1.989	0.00*

*Statistically significance

Multivariate Logistic Regression Analysis by Periodontitis Staging

In Table 5, age continued to be a significant determinant of RA, regardless of the degree of periodontal disease severity. Severe periodontitis had the strongest association with RA when adjusted for confounding factors (OR = 2.740, 95% CI: 1.526–4.921, $p = 0.001$). DAS score was also significantly associated with moderate (OR = 1.671, $p = 0.033$) and severe periodontitis (OR = 2.740,

$p = 0.001$). Educational status remained significantly protective only in the severe group (OR = 0.606, $p = 0.013$).



Figure 2: Radar Plot for Ra Odds Ratio with Other Factors

Table 5: Multiple Logistic Regression ORs And B Coefficient for Association of Rheumatoid Arthritis with Periodontitis with and Without Further Adjustments

Periodontal status	Variable	RA status Odd's ratio	95%CI	p value
Mild Periodontitis	Age (years)	1.085	1.039-1.134	0.00*
	Educational states	0.885	0.732-1.071	0.210
	ESR	0.985	0.958-1.012	0.268
	Duration of illness	1.088	1.088-1.004	0.039
	DAS score	1.329	0.864-2.043	0.195
Moderate periodontitis	Age (years)	1.170	1.106-1.237	0.00*
	Educational states	0.831	0.655-1.054	0.128
	ESR	0.996	0.966-1.026	0.778
	Duration of illness	1.079	0.989-1.177	0.086
	DAS score	1.671	1.041-2.681	0.033*
Severe periodontitis	Age (years)	1.243	1.139-1.356	0.00*
	Educational states	0.606	0.408-0.900	0.013*
	ESR	0.963	0.923-1.004	0.76
	Duration of illness	1.022	0.915-1.141	0.700
	DAS score	2.740	1.526-4.921	0.001*

ORs – odds ratio

RA- Rheumatoid arthritis

ESR- erythrocyte sedimentation rate

DAS- diseases activity severity

Overall Odds of RA in Periodontitis Patients

As summarized in Table 6, the odds of having RA were significantly higher in individuals with periodontitis (OR = 2.986, 95% CI: 1.871–4.765, $p < 0.05$). Among the RA group, 59.3% had periodontitis compared to 40.7% in the control group.

Table 6: Odds of Rheumatoid Arthritis in Periodontitis Patients

	Periodontitis present		Periodontitis Absent		Odds ratio Value (95% Confidence Interval)
	N	%	N	%	
Case Group (with RA)	131	59.3	39	32.8	2.986(1.871-4.765)
Control Group (Without RA)	90	40.7	80	67.2	
Total	170	100	170	100	
$\chi^2 = 21.732$ df = 1 $p < 0.05$ significant * likelihood ratio: 21.732					

Discussion

In this case-control investigation, individuals diagnosed with rheumatoid arthritis (RA) were paired with controls based on age, sex, education, occupation, and socioeconomic status to minimize confounding bias. The mean age distribution between groups was statistically similar ($p > 0.05$), aligning with previous findings that RA onset commonly occurs between 35 and 50 years of age. A large-scale Korean study by Lee KH et al [18], using national insurance data corroborates this age pattern. Additionally, a significantly higher prevalence of RA in females (92.4%) than males (7.6%) was observed ($p < 0.05$), supporting existing epidemiological evidence on gender distribution.

Our results further supported by prior studies, indicating significantly higher periodontal disease severity by PPD and CAL, and greater systemic inflammation marked by elevated ESR compared to controls. ($p < 0.05$). A nationwide study done by Rodríguez-Lozano et al [19], reported that 97.33% of RA patients had periodontitis compared to 66.24% of controls, with severe periodontitis significantly higher in RA (44.92% vs. 12.1%, $p < 0.001$). Increased clinical attachment loss and probing depth correlated with RA disease activity and inflammation markers like ESR in a hospital-based cohort and Wen SE et al [20], also highlighted a clear increase in RA activity among periodontitis patients in a recent meta-analysis. However, studies done by Sjostrom et al and Mobini et al suggests that there is no statistically meaningful association between RA status and the degree of periodontal disease. Our study demonstrates that Mild periodontitis was more prevalent in RA cases (39.4%) than controls (29.4%) and was strongly associated with RA, with adjusted ORs around 4.1 to 4.5 [21-23]. Rovas et al also found that significantly higher mean probing pocket depth in patients with both periodontitis and RA than those with periodontitis alone (mean PPD 2.81 mm vs. 2.58 mm, $p=0.009$; OR for association: 2.22, 95% CI: 1.16–4.31). Koziel J et al., Åyräväinen L et al. and Bingham CO III et al. also found similar results in their

epidemiological studies. However, Crisigiovanni AC et al. did not demonstrate a statistically significant relationship between periodontitis (including mild forms) and RA, suggesting that the link may be stronger for moderate to severe forms or influenced by other confounders [10,24-26].

We revealed in our study that RA disease activity (DAS-28) correlated significantly with periodontitis severity, with more active RA linked to moderate and severe periodontitis. Rodríguez-Lozano et al. supports these findings in their study as RA patients, moderate/high disease activity (by DAS-28) was directly linked to an increased occurrence and greater severity of periodontitis. Ordinal logistic regression showed severe periodontitis increased odds of active RA (OR 2.66, 95% CI: 1.24–5.74, $p=0.012$). Linear regression confirmed DAS-28 scores rose with increasing attachment loss ($p=0.002$ [27]. Varshney et al., Albrecht et al. found similar results [28]. On the other hand Kordtabar et al. and Khantisopon et al. found no similar correlation and this is all because of methodological difference [29,30]. Some case-control and cohort studies, such as those by Mobini et al., Khantisopon et al., and Kordtabar et al., have found no statistically significant association between periodontitis severity and DAS-28 scores—patients with and without severe gum disease did not differ in RA activity levels [22].

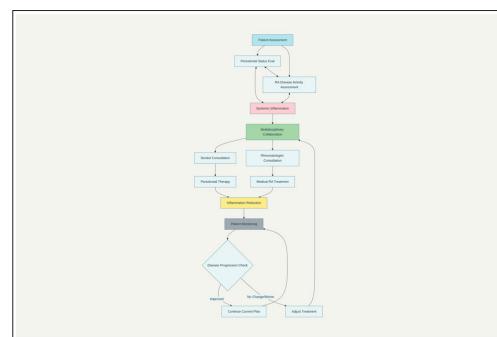
Our study indicates that individuals with periodontitis had approximately 3 times higher odds of RA than those without RA. Qiao et al. conducted a meta-analysis including 706,611 periodontitis patients and 349,983 controls reported a pooled OR for RA risk between periodontitis and controls of 1.69 (95% CI: 1.31–2.17; $p < 0.0001$); for longer RA duration (>5 years), the OR increased to 2.88 (95% CI: 0.66–12.62. A systematic review performed by de Oliveira Ferreira et al. most included studies report a significant association, with a pooled OR of 1.97 (95% CI: 1.68–2.31) for RA among those with periodontitis compared to controls. Bolstad et al. who had done a national study in Taiwan population found only a modest association (OR = 1.16; 95% CI: 1.12–1.20), much less than a threefold increase, though statistically significant [31-33]. The majority of meta-analyses and large-scale studies support a significant association between periodontitis and increased odds of RA—often reporting odds ratios near or above 2 (and sometimes as high as 3). Some studies—particularly those with strong control for confounding, specific populations, or differing access to care—report a weaker or no association, underscoring the importance of methodological rigor, accurate case definitions, and context.

Based on finding of these studies an integrated care model including routine periodontal screening, dental interventions, and multidisciplinary collaboration can be proposed which can play a crucial role in RA management to potentially slow disease progression or reduce RA development risk.

Conceptual RA Care Model with Integrated Dental Care

The proposed integrated care model for rheumatoid arthritis (RA) centers on routine periodontal screening and risk assessment, ensuring early identification of periodontitis in RA patients through clinical evaluation. Following risk assessment, early dental intervention—focusing on preventive care, plaque

control, scaling, root planning, and patient education—aims to reduce oral microbial and inflammatory burden. A key component of this model is ongoing multidisciplinary collaboration among rheumatologists, dentists, and primary care providers, fostering shared decision-making regarding treatment timing and regular patient monitoring. Systemic inflammation monitoring is also integrated, correlating RA disease activity (such as DAS-28 scores) with periodontal status, and utilizing biomarkers like ESR and CRP to track systemic immune activation. Targeted anti-inflammatory therapy involves combining dental interventions with pharmacological RA treatments and making real-time adjustments based on bidirectional feedback between oral and systemic disease indicators. Longitudinal follow-up and personalized care ensure that patients receive tailored interventions based on evolving disease severity and therapeutic responses. Evidence from this study demonstrates that periodontitis nearly triples the odds of RA and that moderate to severe periodontitis correlates with higher RA activity, supporting the view that periodontal health management can be a critical element in RA care. This comprehensive approach not only holds potential to delay RA onset and mitigate disease flares but also promises improved quality of life through interdisciplinary care—though further validation in longitudinal and interventional studies, with attention to confounders like smoking and comorbidities, is warranted.



This study may be affected by surveillance bias, unmeasured confounders (like BMI and lifestyle), and the inherent inability of observational case-control designs to establish causality. Channeling bias could have led to exclusion of more severe cases, and single-center, tertiary-care recruitment limits the generalizability of results, as the sample may not reflect the general population. The most important limitation is that findings from a single center may not be broadly applicable.

A major strength of this study is the inclusion of a relatively large sample size ($n = 340$), which provides enhanced statistical power and reliability in detecting associations. The use of validated disease activity measures such as DAS-28 and standard periodontitis classification (AAP criteria) ensures robust assessment of both RA and periodontal status. Internal validity is further improved by accounting for potential comorbid conditions, including diabetes, and considering patient medication profiles as part of the analysis. Finally, the identification of an age-related increase in periodontitis reinforces the significance of periodontal health in the natural history and progression of rheumatoid arthritis.

Conclusion

This study highlights a significant association between periodontitis and rheumatoid arthritis (RA), with individuals suffering from mild to severe periodontal disease showing higher likelihood of developing RA compared to healthy or gingivitis-only individuals. Notably, the odds increased with advancing periodontal severity and were significantly influenced by age, disease activity (DAS28), and educational status. The findings reinforce the systemic interplay between periodontal and rheumatic inflammation, suggesting shared immunological pathways. Although some studies report inconsistent associations, likely due to population diversity and differing diagnostic criteria, our findings are consistent with the concept that periodontitis could represent a modifiable risk factor for RA.

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