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Research Article

Impact of Periodontal Therapy on Disease Activity in Rheumatoid Arthritis: A Prospective Clinical Study

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ABSTRACT

Introduction: Periodontal disease and rheumatoid arthritis (RA) are chronic inflammatory conditions that share pathogenic mechanisms involving immune activation and connective tissue degradation. Both disorders have demonstrated epidemiological and biological links, suggesting a bidirectional influence between oral and systemic inflammation**.**

Objective: This prospective clinical study aimed to evaluate the impact of non-surgical periodontal therapy on systemic inflammatory markers and functional outcomes in patients diagnosed with both RA and periodontitis.

Methodology: Adult patients clinically diagnosed with RA as per 1988 American Rheumatology Association criteria and moderate to severe periodontal disease were recruited from clinical settings in Bengaluru. Forty-two participants were randomly allocated to two groups: Group A received supragingival scaling and oral hygiene instruction, while Group B underwent scaling and root planing. Clinical periodontal parameters (gingival bleeding index, plaque index, probing pocket depth, clinical attachment level), systemic markers (erythrocyte sedimentation rate [ESR], rheumatoid factor [RF], C-reactive protein [CRP]), and functional disability (Stanford Health Assessment Questionnaire) were assessed at baseline, 1 month, and 3 months post-intervention. Statistical analysis included Mann-Whitney U and Wilcoxon signed-rank tests.

Results: Thirty-six female participants completed the study. Both groups showed significant improvements in bleeding on probing and plaque scores at 1 and 3 months ($p < 0.05$). Group B exhibited greater reductions in deep periodontal pockets and clinical attachment loss. ESR levels significantly declined in Group B after periodontal therapy, whereas both RF and CRP levels decreased in both groups without reaching statistical significance. Functional disability improved modestly in the intervention group. No strong correlation was found between changes in periodontal and systemic parameters**.**

Conclusion: Non-surgical periodontal therapy, especially scaling and root planing, improved local periodontal health and led to decreases in systemic inflammatory markers in RA patients. These findings support periodontal therapy as a potential adjunct in managing RA, warranting further research for long-term systemic benefits.

Keywords*: Rheumatoid arthritis, Non-surgical Periodontal therapy, Systemic inflammation, Biochemical markers, Periodontal status.*

INTRODUCTION

Periodontal disease comprises a heterogeneous group of chronic infectious conditions resulting from the intricate interplay between microbial pathogens, host immune responses, genetic predispositions, and environmental factors.[1] While the immune system's primary role is to localize and contain bacterial invasion, an excessive and persistent microbial stimulus can shift the host response from protective to destructive, leading to breakdown of the periodontal connective tissues. This pathological process is mediated by complex cellular and cytokine interactions that release proteolytic enzymes and bone-resorbing factors, driving chronic destruction of both soft and hard tissues in the periodontium.[2]

Rheumatoid arthritis (RA) is a prevalent systemic autoimmune disorder principally involving the joints, characterized by chronic synovial inflammation, accumulation of lymphocytes and monocytes, proliferation of endothelial cells, tissue edema, and progressive degradation of the extracellular matrix.[3] The etiology of RA points towards microbial exposure or the presence of autoantigens as possible triggers of the destructive immune response, which is further modified by systemic, genetic, and environmental influences.[4] Similar to periodontitis, RA leads to the activation of complement pathways and production of a range of inflammatory mediators, culminating in persistent inflammation and tissue destruction in regions with connective tissue and bone.[5]

Recent epidemiological and experimental studies have established associations between periodontal disease and several systemic conditions, such as pre-term birth, coronary heart disease, thromboembolic events including myocardial infarction and stroke, and type 1 diabetes mellitus.[6–11] Both periodontal disease and RA share a pathogenic pattern involving chronic immune activation, complement activation, cytokine release, and connective tissue/bone degradation.[12] Rheumatoid factors, autoantibodies of the IgG, IgM, IgA, and IgE classes found in blood and synovial fluid are hallmarks of RA and reflect chronic antigenic stimulation. The most common form, IgM rheumatoid factor, reacts with multiple epitopes on Fc fragments of human and animal IgG and is detected in over two-thirds of adult RA patients.[12]

Comprehensive assessment of RA and periodontal disease relies on a combination of clinical and laboratory markers, with laboratory indicators such as rheumatoid factors, C-reactive protein, erythrocyte sedimentation rate, and prostaglandins serving as valuable measures of inflammatory activity.[13] Numerous studies have examined the periodontal status of RA patients, reporting variable results with most indicating a detrimental influence of RA on periodontal health.[12–16] However, the evidence regarding the impact of periodontal treatment on the progression or severity of RA remains limited and inconclusive.

Given the marked similarities in the pathophysiological mechanisms underlying periodontal disease and RA, studying their coexistence presents an important opportunity to assess whether controlling periodontal inflammation might have favorable systemic effects on RA progression. Mechanical debridement, a cornerstone of non-surgical periodontal therapy, is well established in local inflammation control.[17] The present study aims to determine the effect of non-surgical periodontal therapy on systemic inflammatory parameters in patients with rheumatoid arthritis.

METHODOLOGY

This study employed a prospective clinical trial design to assess the impact of periodontal therapy on inflammatory and functional outcomes in individuals with rheumatoid arthritis and periodontitis. The research was conducted at the Department of Periodontics, M. S. Ramaiah Dental College, Bengaluru, with patient referrals originating from the Immunology Clinic of M. S. Ramaiah Memorial Hospital.The target population included adult patients who were clinically diagnosed with both rheumatoid arthritis and periodontal disease, while attending the Immunology Clinic and subsequently referred to the dental college.

Those who were diagnosed of rheumatoid arthritis in accordance with the 1988 revised criteria of the American Rheumatology Association, having at least two teeth, at least two sites exhibiting probing pocket depth ≥5 mm or clinical attachment loss ≥6 mm at baseline were included in the study. Those diagnosed with xerostomia, pregnant or lactating individuals, co-morbid systemic conditions influencing the progression or treatment of periodontitis, those using antibiotics in the last six months, and current smokers were excluded from this study.

The study enrolled forty-two participants who visited the study setting during the study duration met the eligibility criteria . Random allocation to the intervention and control groups was achieved via simple coin-toss randomization, producing two groups A and B for comparative intervention.

DATA COLLECTION METHODS

Clinical Examination

Full-mouth periodontal examination was performed under appropriate illumination using a mouth mirror and UNC-15 probe. The following indices and measures were recorded:
Plaque Index (Silness and Löe, 1963), Gingival Bleeding Index (Ainamo and Bay, 1975), Probing Pocket Depth (PPD), Clinical Attachment Level (CAL). Acrylic stents were employed to ensure reproducibility of probe placement at each measurement point.

Rheumatoid Arthritis Assessment

Functional disability was measured using the Stanford Health Assessment Questionnaire (HAQ) Disability Index, which surveys eight functional domains and produces an average score ranging from 0 (no disability) to 3 (complete disability).[18]

Venous blood was collected at baseline and three months after treatment. One sample was used to measure Erythrocyte Sedimentation Rate (ESR) using the Westergren method; another sample was processed for serum analysis of

Rheumatoid Factor (IgM-RF) and C-Reactive Protein (CRP) via immunoturbidimetric assay using an automated biochemical analyzer (Mindray, China).

Intervention

On the first visit, detailed case history including clinical parameters and serum ESR, and patient's disability status was recorded. Serum samples were made and preserved for estimation of RF and CRP levels. This was followed by a phase I therapy which included patient education and motivation and supragingival scaling in group A and scaling, root-planing in group-B which was performed in two or three sittings.The patients were given oral hygiene instructions and advised meticulous home care. Following this, the patient was recalled at one month and periodontal clinical parameters, ESR and disability were further recorded. Group A received reinforcement of oral hygiene instructions and supragingival scaling. Group B received scaling and root planning if necessary. Periodontal clinical parameters, ESR and disability were again recorded 3 months after baseline. Serum samples were collected at 3–months post-treatment also and all samples including those preserved at baseline were subjected to immunoturbidimetric assay for estimation of Rheumatoid Factor and C-reactive protein. The rheumatoid arthritis patient's medical prescriptions were not altered during the period of the study. All participants received standardized oral hygiene instruction and reinforcement at follow-up. Follow-up assessments (clinical and biochemical) were conducted at post baseline, one and three months.

Ethical considerations

The study was conducted following all the ethical principles. Privacy and confidentiality were assured. The study commenced only after obtaining the informed consent from the participants. They were free to withdraw from their study at any point in time. Ethical clearance was obtained from the Instituitional Ethics Committee.

Data Management and Analysis

All clinical, functional, and biochemical data were recorded using standardized forms and securely stored. Statistical analysis was conducted to compare within- and between-group changes from baseline to three months, considering both clinical and laboratory outcomes.

The normality of the data was checked using Anderson -Darling test. Since the data was found to be non-normal in distribution, Mann-Whitney U Test was used to compare the efficacy of treatment in Groups A and B, and Wilcoxon sign rank test to assess the intragroup comparisons at different timeline.

RESULTS

A total of forty-two patients, 40 females and two males were included in this study of which only thirty-six female patients completed the study. The biochemical results and clinical periodontal finding of nineteen patients in Group A was compared with the17 patients in Group B.

Age ranged from 21-68 years, and the mean age was 42.58 (11.03) in group A and 47.12 (10.48) in Group B. The mean years of Rheumatoid Arthritis were 4.95 (2.20) in Group A and 6.59 (3.20) for Group B.

All the thirty-six participants returned regularly for their maintenance programme. None of them underwent any change in their anti-rheumatoid drug therapy during the 3-month study period. All the clinical periodontal parameters and ESR were assessed at baseline, 1 month and 3 months and Rheumatoid Factor and CRP values at baseline and 3-month of completion of respective treatment in Group-A and Group B.

Periodontal Parameters

The percentage of sites with bleeding on probing was averaged at baseline, 1 month and 3month and was 55.19(16.17), 36.43(14.86) and 28.21(11.62) % respectively in Group A and 62.42(15.24), 42.32(14.47) and 28.84(10.48) % respectively in Group-B. There was a statistically significant reduction ($p<0.05$) in BOP assessed by gingival bleeding index in both groups at 1 month and 3 months compared to baseline. Intergroup comparison showed that there was no significant difference between Group-A and Group-B at the end of 3 months after treatment (Table 1).

The plaque control in all patients was satisfactory after the treatment provided. The plaque mean scores in baseline, 1 month and 3 month in Group-A and Group-B were 2.09(0.48), 1.50 (0.38) and 1.21 (0.45) and 2.09 (0.48), 1.50 (0.38), 1.21 (0.45) respectively. There was significant improvement in the plaque score at 1- month and 3-month compared to baseline in both treatment groups. (Table 1)

Periodontal pockets vary in their location and depth, hence changes in their mean probing depths for entire mouth may not provide realistic information. Hence % of sites < 4mm, 4-6 mm and > 6 mm probing pocket depth was calculated at baseline, 1 month and 3-month after treatment in both groups.

The mean % of sites with probing pocket depth < 4mm, 4-6 mm and > 6 mm at baseline was 67.86 (15.81), 27.59(10.90) and 4.39(6.12) in Group A which became 70.20 (15.97), 24.35(10.20), 4.98 (6.62) 1 month after treatment and 72.63(15.04), 21.22(8.89) and 6.14 (7.31) respectively, 3-month after treatment. There was a statistically significant decrease in the percentage of sites with probing pocket depth 4-6mm at 3-months post treatment in group-A compared to baseline

(p< 0.05). However, there was an increase in % of sites with PPD > 6mm after 3- months compared to baseline, though not significant. In Group B the mean % of sites with probing pocket depth < 4mm, 4-6 mm and > 6 mm at baseline was 63.93(17.03), 28.93(11.05), 7.14(7.62), 1-month after treatment was found to be 72.15 (15.05), 25.38(13.30) and 2.47(2.63) and at end of 3 month was 77.34 (13.89), 21.92 (13.15), 0.73(1.41).). There was a statistically significant increase in % of sites with PPD <4 mm after 3- months compared to baseline (p<0.05). Also there was a statistically significant decrease in % of sites with PPD > 6mm after1 month and 3- month compared baseline(Table 2) .Inter-group comparison at 3 month showed no significant differences between the treatment groups, though the mean values were lesser in group-B compared to group-A.

The mean % of sites with clinical attachment level < 4mm, 4-6 mm and > 6 mm at baseline was 62.96(17.98), 29.64(±12.1) and 7.89(±8.6) in Group A which became 66.86(±17.48), 25.59(±10.4), 7.95(±8.37), 1 month after treatment and 67.1(±16.75), 23.66(±9.22) and 8.77(±8.61) respectively, 3-month after treatment. There was no statistically significant change in the percentage of sites with different clinical attachment levels at 1 month and 3 months compared to baseline. The mean % of sites with clinical attachment level < 4mm, 4-6 mm and > 6 mm at baseline was 55.27±19.19, 31.44±12.40 and 13.29±10 in Group B which became 63.42 (17.32), 28.61 (13.50), 7.97 (8.77) 1 month after treatment and 67.19 (17.03), 26.51 (12.32) and 6.27 (7.27) respectively, 3-month after treatment. There was a statistically significant decrease in % of sites with CAL > 6 mm after 3- months compared to baseline. (Table-2). Inter-group comparison at 3 months showed no significant differences between the treatment groups, though the mean values were lower in group-B compared to group-A.

Biochemical Parameters

The mean ESR values at baseline in group A and Group B were 42.58 (22.8) and 38.47 (13.85) respectively. After treatment this reduced to 38.74 (17.89) and 26.59 (15.40) at 1 month and 37.21 (13.04) and 26.47 (14.94) at 3 months in group-A and group-B, respectively. Mean ESR levels in group-A showed slight reduction post-treatment at 1-month and 3-month . Median values were observed to be 33, 35, 35 at baseline, 1-month, and 3-month, with no statistically significant difference. Mean ESR levels in group-B showed significant reduction post-treatment at 1-month and 3-month. Median values were observed to be 31,22,22 at baseline, 1-month, and 3-month. There was statistically significant change at 1month compared to baseline (p<0.05) and 3-month compared to baseline (p<0.05) (Table 3). Inter-group analysis showed significant reduction at 3-month in group-B compared to group-A. (Table-3).

Mean Rheumatoid Factor values for Group A at baseline and 3 months were 45.26 (69.93) and 39.76 (61.81) and in Group-B it was 23.89 (19.21) and 16.19 (16.98) at baseline and 3 months, respectively. The RF values in 3 months showed a reduction in both groups A and B compared to the baseline values though not statistically significant. Inter – group comparison at 3- month post-treatment showed no significant difference between both treatment groups.(Table-3). Odds ratio of group-B to Group-A was found to be 2.15.

Mean C-reactive protein values for Group A at baseline and 3 months were 8.61 (15.48) and 8.63 (16.55) and in Group-B were 4.13 (3.04) and 2.74 (2.33) at baseline and 3 months, respectively. The CRP values in 3 months showed a reduction in both group-A and B compared to the baseline values though not statistically significant. (Table-8). Inter-group comparison at 3- month post-treatment showed no significant difference between both treatment groups. (Table-3).

Stanford Health Assessment Questionnaire Disability Index

Patients' Disability status measured by HAQ improved in both groups at the end of 3 months. When three patients showed severe disability, ten showed moderate disability and six showed mild disability at baseline in group-A, it became 3,8 and 8 respectively at 1 month and 3,9,7 at 3 months of treatment. In Group B the number of patients with severe, moderate, and mild disability was 3,11,3 at baseline, which reduced to 2, 8, and 7 at 1 month and 2, 7 and 8 at end of 3 months of treatment. (Fig 1,2)

Spearman correlation test showed no statistically significant correlation between changes in periodontal parameters and biochemical parameters at different periods. There was no correlation within biochemical parameters also (RF and C-reactive protein)

Table 1: Intragroup comparisons of bleeding on probing and plaque index using Wilcoxon sign rank test

Parameter		**Group A**		**Group B**	
		Mean ±SD	**P value**	**Mean ±SD**	**P value**
Bleeding on probing (% sites)	Baseline	55.19±16.17	BL &1 M* BL & 3 M*	62.42±15.24	BL &1 M* BL & 3 M*
	1 month	36.43±14.86		42.32±14.47	
	3 month	28.21±11.62		28.84±12.38	

Plaque Index	Baseline	1.98±0.52	BL &1 M* BL & 3 M*	2.09±0.48	BL &1 M* BL & 3 M*
	1 month	1.47±0.15		1.50±0.38	
	3 month	1.25±0.3		1.21±0.45	
*- Statistically Significant- p< 0.05; BL - Baseline, 1 M - 1 Month, 3M - 3 Month					

Table 2: Intragroup comparisons of bleeding on pocket probing depth and clinical attachment level using Wilcoxon sign rank test

Parameter		**Group A**		**Group B**	
		Mean % ±SD		**Mean % ±SD**	**P value**
Probing Pocket Depth (mm)	Baseline-<4	67.86±15.81		63.93±17.03	BL & 3M*
	1Month -<4	70.2±15.97		72.15±15.05	
	3Month-<4	72.63±15.04		77.34±13.89	
	Baseline -4-6	27.59±10.9	BL & 3M*	28.93±11.05	
	1Month -4-6	24.35±10.2		25.38±13.3	
	3Month- 4-6	21.22±8.89		21.92±13.15	
	Baseline >6	4.39±6.12		7.14±7.62	BL & 3M*
	1Month ->6	4.98±6.62		2.47±2.63	
	3Month >6 mm	6.14±7.31		0.73±1.41	1M& 3M*
Clinical Attachment Level (mm)	Baseline -<4	62.96±17.98		55.27±19.19	
	1Month -<4	66.86±17.48		63.42±17.32	
	3Month -<4	67.1±16.75		67.19±17.03	
	Baseline -4-6	29.64±12.1		31.44±12.4	
	1Month -4-6	25.59±10.4		28.61±13.5	
	3Month -4-6	23.66±9.22		26.51±12.32	
	Baseline ->6	7.89±8.6		13.29±10.56	BL & 3M*
	1Month -->6	7.95±8.37		7.97±8.77	
	3Month -->6	8.77±8.61		6.27±7.27	
*- Statistically Significant- p< 0.05; BL - Baseline, 1 M - 1 Month, 3M - 3 Month					

Table 3: Intragroup comparisons of biochemical parameters Wilcoxon sign rank test

Study Parameter	Group	Treatment effect on study parameters			
		Baseline	1 Month	3 Month	p value
		Mean SD	Mean SD	Mean SD	
ESR	A	42.58 22.80	38.74 17.89	37.21 13.04	0.763
	B	38.47 13.85	26.59 15.40	26.4714.95	<0.05*
RF	A	45.26 69.93	----	39.76 61.8	0.821
	B	23.89 19.21	----	16.19 16.98	0.614
CRP	A	8.61 15.48	----	8.63 16.55	0.712
	B	4.13 3.04	----	2.74 2.33	0.893

ESR- Erythrocyte Sedimentation Rate; RF- Rheumatoid factor; CRP- C-reactive protein; *- statistically significant.

Fig 1: Disability at different timelines in Group A assessed using Stanford Health Assessment Questionnaire

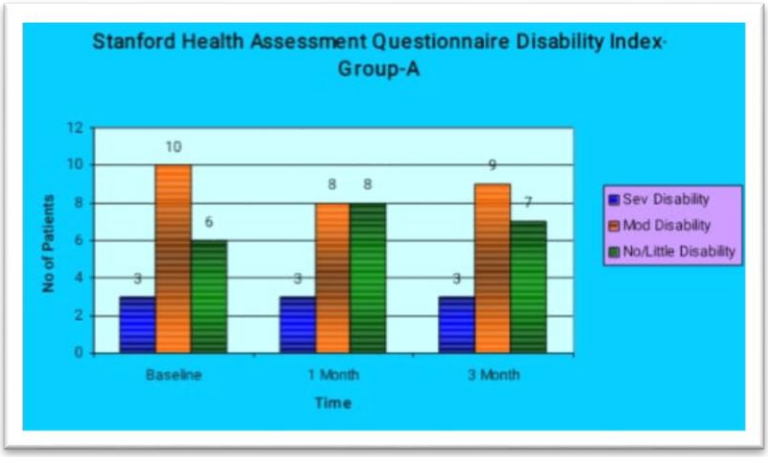
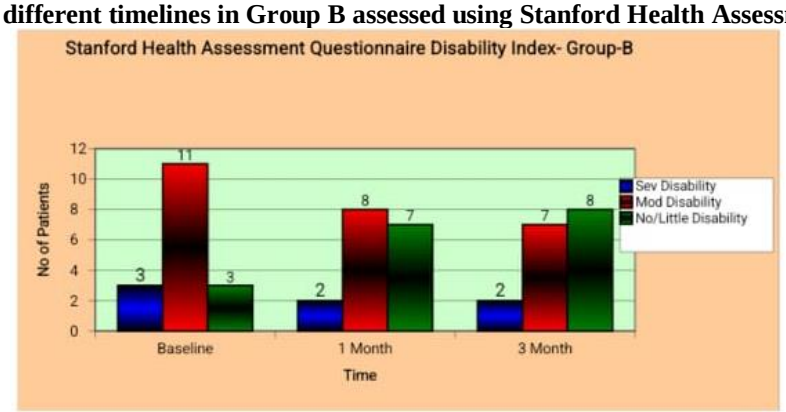


Fig 2: Disability at different timelines in Group B assessed using Stanford Health Assessment Questionnaire



DISCUSSION

Periodontal medicine is an advanced subspecialty of Periodontology that explores and substantiates the significant link between periodontal health and systemic diseases. Contemporary research has robustly documented associations between periodontitis and conditions such as coronary heart disease, myocardial infarction, stroke, type 1 diabetes mellitus, and pre-term low birth weight. A growing body of evidence also points to a link between periodontal disease and rheumatoid

arthritis (RA). While their etiologies are distinct, both diseases involve similar immunoinflammatory and pathogenic mechanisms. This supports the hypothesis that individuals presenting with both RA and periodontitis may experience a systemic dysregulation of inflammatory responses.

In this study, 42 RA patients were recruited, but after two male participants dropped out, thirty-six female patients completed the study. This pronounced female predominance reflects the epidemiology of RA, as previously documented by Arnett et al. and Harris, who noted a 3:1 female-to-male ratio and peak disease onset for women in their forties and fifties. Baseline clinical periodontal evaluations showed both groups had comparable degrees of disease, predominantly mild in severity, with few deep periodontal pockets. This may be attributed to widespread use of non-steroidal anti-inflammatory drugs (NSAIDs) among participants, which are known to reduce oral inflammation and modify periodontal tissue response.

The effectiveness of periodontal treatment was assessed by comparing supragingival scaling and oral health education (Group A) with scaling and root planning at multiple visits (SRP, Group B). Both groups demonstrated significant improvements in bleeding on probing and plaque index post-treatment. Notably, SRP led to a more pronounced reduction in deep periodontal pockets and clinical attachment loss than scaling alone, in line with previous research.[19,20]Consistent with earlier studies, this research found that scaling and root planing (SRP) significantly improved local periodontal parameters such as bleeding on probing, plaque index, pocket depth, and clinical attachment levels.[13,21] Microbiological assessments indicated that SRP shifted the subgingival ecosystem from predominantly gram-negative bacteria toward gram-positive flora, which are associated with periodontal health.[22] These studies similarly observed microbial shifts to a healthier subgingival flora post-treatment, reinforcing the established role of SRP in managing periodontitis in systemic disease contexts.

Systemic inflammatory markers were also evaluated. Erythrocyte sedimentation rate (ESR) showed a significant decline in the SRP group after periodontal therapy, reflecting reduced systemic inflammation following improved periodontal health. Rheumatoid factor (RF) and C-reactive protein (CRP) levels decreased in both groups in three months, although these changes were not statistically significant. These findings are in accordance with a randomized control trial in Vietnam, China, and at Bhopal in India.[23–25] These findings support the concept that periodontal pathogens may stimulate systemic auto-antibody production, and their reduction via periodontal therapy can mitigate systemic inflammation, even if not reaching statistical significance within the study's time frame.
The trend toward reduction in rheumatoid factor (RF) and C-reactive protein (CRP) levels observed in the present study, despite not reaching statistical significance, echoes mixed evidence in the literature.A Systematic review and meta-analysis by Kaur *et al.*,[26] concluded in favour of reduction of rheumatoid disease activity after periodontal therapy. However, other authors reported no changes in the disease activity of RA after periodontal therapyreflecting variability due to study design differences and follow-up durations.[27]

Functional disability, assessed via the Stanford Health Assessment Questionnaire (HAQ), improved modestly in the SRP group, and remained unchanged in the scaling-only group. However, given that RA is a chronic condition and the study follow-up was limited to three months, more extended observation may be necessary to detect statistically significant changes in functional status.

The study found no strong correlation between the severity of RA (as measured by biochemical markers and HAQ scores) and changes in periodontal clinical parameters. This could be due to the mild periodontal disease at baseline, the modest sample size, or the short duration of observation. Periodontal therapy induced clear improvements in local clinical parameters and showed trends toward systemic benefit.

This study's strengths include its focus on a well-defined RA population with clinically documented mild to moderate periodontitis and its comparison of two common periodontal treatment modalities. It incorporated comprehensive clinical and biochemical outcome measures relevant to both periodontal and RA disease activity. However, limitations include a small sample size and a short follow-up period of only three months, which may be insufficient to observe longer-term system or functional changes. The lack of a randomized controlled design, potential confounding by ongoing RA pharmacotherapy, and absence of blinding may affect internal validity. Moreover, the predominance of female subjects and exclusion of males' limit generalizability. Future research should include larger, randomized, and longer-duration trials, with standardized RA and periodontal disease criteria, to better elucidate the impact of periodontal therapy on RA progression.

Limitations noted in this study mirror challenges in existing literature, including small sample sizes, short follow-ups, and confounding effects of RA medications, which often complicate establishment of definitive causal links. Moreover, the female predominance in both this study and other RA cohorts emphasizes the need for more inclusive designs.

Overall, this study coherently supports the hypothesis that periodontal therapy can modulate systemic inflammation and potentially improve RA disease activity, in line with the evidence base, while underscoring the necessity for larger, well-controlled, and longer-term trials to confirm and elaborate these associations.

CONCLUSION

This study concludes that periodontal therapy, particularly scaling and root planing (SRP), significantly improves local periodontal health in patients with rheumatoid arthritis (RA) and mild to moderate periodontitis. The reduction in periodontal inflammation corresponded with a decrease in systemic inflammatory markers such as erythrocyte sedimentation rate (ESR), supporting the concept that controlling periodontal infection can positively influence systemic inflammation in RA. Although reductions in rheumatoid factor (RF) and C-reactive protein (CRP) levels, as well as improvements in functional disability measured by the Health Assessment Questionnaire (HAQ), did not reach statistical significance within the three-month follow-up, trends suggest potential systemic benefits with more intensive periodontal care.

Periodontal therapy is a promising adjunctive approach in the management of RA with concomitant periodontitis, capable of improving oral and systemic inflammatory parameters and potentially contributing to better overall disease control. Integration of periodontal assessment and treatment into RA management protocols is advisable, with further research needed to optimize treatment strategies and evaluate long-term outcomes.

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