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# Periodontitis and Rheumatoid Arthritis: Their Bidirectional Outcome of Treatment : A Narrative Review

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## Abstract

***Rheumatoid arthritis (RA) and periodontitis(Pd) share numerous*** biological and immunological interactions, making them closely linked across multiple pathophysiological aspects. The severity and activity of each disease have a tangible impact on the other. Accordingly, the treatment of one condition should also have a positive effect on the other. This review aims to illustrate the outcomes of treating Pd in rheumatoid arthritis and vice versa, given the bidirectional relationship between the two conditions. Method: a recent systematic review and studies involving the treatment outcome of the two conditions on each other have been evaluated. Result: regardless of the type of treatment used, it has been shown that treating periodontitis can decrease the activity of RA, and on the same manner treating RA can improve the periodontal breakdown. Conclusion: it is advised that periodontal assessment be performed for all RA patient for an early diagnosis and treatment of any periodontal problems, similarly, for periodontal patients who have RA the activity of RA should be investigated and managed to control periodontal distraction.

**Keywords:** periodontitis, rheumatoid arthritis, disease modifying antirheumatoid drugs(DMARD).

## Introduction

Periodontitis(Pd) implies complicated host-microbe interactions that stimulate inflammatory mediators, leading to destruction of the periodontium and risk of systemic inflammation (1,2) Systemically spreading inflammatory mediators act as a potential pathway linking oral to systemic illness (3). Recent research (4) has proved that periodontitis is associated with several systemic disorders, such as rheumatic disease (5). The common biological, immunological, environmental, and genetic backgrounds of rheumatoid arthritis(RA) and Pd make them strongly associated (6). They share numerous physiopathological connections, as both induce persistent inflammation that leads to the breakdown of host tissues (7). Epidemiologically, Pd is found with greater prevalence in individuals afflicted with RA and a positive rheumatoid factor (RF) as well as antibodies to cyclic citrullinated proteins(CCP) was observed (8), also periodontal inflammation

may promote RA activity via systemic mediators (i.e., anti-citrullinated protein antibodies ACPAS, RF, and cytokines) (6,9). The TNF-α level in plasma is correlated with the intensity of systemic inflammation and likely promotes Pd in RA patients (9). Moreover, the severity of Pd was the third in ranked among all identified predictors of RA following female gender and smoking(10). Microbiologically; P. gingivalis prevalence is increased in individuals with newly diagnosed and untreated RA compared to those with established RA or healthy controls (11). Furthermore, periodontal pathogens particularly, *P. gingivalis*, *A. actinomycetemcomitans*, *Tannerella forsythia*, and *Fusobacterium nucleatum* have been shown to play a role in the initiation of the disease, through interaction with citrullinated bacterial and host antigens and/or by causing a loss of immunotolerance to citrullinated proteins (12). Individuals with long-standing periodontitis have higher levels of CCP within the periodontium (8). Which has a correlation with Pd, characterized by more sever Pd compared to healthy subjects and worsen periodontal parameters, in terms of mean plaque index(PI), mean clinical attachment level(CAL), and number of pockets > 5 mm, were reported markedly linked to both positive and elevated titters of anti-CCP antibodies (6). Moreover, Alveolar bone loss is substantially connected to antibodies to CCP titers and disease activity in terms of disease activity score(DAS28) of RA (14).
This review attempts to line up the outcome of treating periodontitis and RA on each other based on the available research.

## Treatment outcome

Based on the mutual relation between both conditions, the chronic inflammation, and the main pathogenic components in both, it is logical to assume that a notable effect on the periodontal status of patients with RA could be exerted by treatment of RA (15). Similarly, it is reasonable that periodontal therapy influence on both clinical and biochemical expression of RA (16).

## Impact of Treating Rheumatoid Arthritis On Periodontitis

The most recent guidelines for RA treatment by the European Alliance of Associations for Rheumatology (EULAR task force of 2013) suggest a “treat-to-target” concept utilizing non-steroidal anti-inflammatory drugs (NSAIDs), immunosuppressive glucocorticoids (GC), and disease-modifying anti-rheumatic drugs (DMARDs). The research on the impact of various RA drugs on periodontal status demonstrated that these medications inhibit periodontitis regardless of its severity (15) Glucocorticoids (GC), such as prednisone are used at small doses in preliminary stages to achieve a swift clinical improvement and arrest articular erosion, awaiting for the DMARDs to have the desired effects (17). By adjusting cytokine levels, DMARDs can halt the advancement of joint degeneration (18). DMARDs are classified into 3 main categories (19), their types, indication and effect on periodontitis are listed on table.1 . sDMARDs can be used in single therapy or together with other sDMARDs or with a bDMARD (17). Various medications exert no differences among their effects (20).
TNF-α inhibitors are the most frequently consumed as bDMARAD they reduce pro-inflammatory cytokines and osseous resorption (26). While non TNF inhibitors enhance periodontal indices (27), this enhancement could be related to decrease in serum inflammatory mediators, specifically TNF-α, total immunoglobulin G, and serum amyloid A (28,29), But 6 months treatment is a prerequisite to reduce PPD (21).

**Table 1:** effect of common anti rheumatoid medications on periodontitis. BOP bleeding on probing, PPD probing pocket depth, CAL clinical attachment loss, GI gingival index, TNF tumour necrosis factor, GCF gingival crevicular fluid.

| category | Indication | Common Items | | Effect on periodontitis |
|---|---|---|---|---|
| Synthetic disease-modifying anti-rheumatic drugs(sDMARDs) | Early stages combined with GCs | Methotrexatei(MTX) Hydroxychloroquine(HCQ) Leflunomidei(LFN) Sulfasalazine (SSZ) | | Decrease in BOP &PPD with enhancement of CAL(20) |
| Target disease-modifying anti-rheumatic drugs(tDMARDs) | Moderate-to-severe RA | Baricitinib Tofacitinib Oral Janus-activated kinase (JAK)-dependent cytokine signaling inhibitors | | Improved GI, BOP, and sites with PPD ≥ 4 mm after 24 hours(21). |
| Biologic disease-modifying anti-rheumatic drugs(bDMARDs) | Severe RA | TNF-α inhibitors | Adalimumabi Certolizumab pegol Golimumab | 1.Reduce pro-inflammatory cytokines, GI, BOP, PPD and osseous resorption 2.Enhance extent and severity of periodontitis(22). |
| | | | Etanercept | 1. ↓BOP, GI, and TNF-α levels in GCF 2.Enhance extent and severity of periodontitis(22,23) |
| | | | Infliximab | ↓BOP, PPD, CAL and TNF-α level in GCF(24). |
| | | T-costimulatory cell inhibitor | Abatacept | 1. Decrease salivary levels of IL-8 and monocyte chemoattractant protein-1 2.GCF levels of TNF-α, IL- 1β, IL-8. 3.Significant reduction in GCF volume(17,25) |
| | | Anti-B-cell agent | Rituximab | |
| | | IL-6 receptor blocking monoclonal antibody | Tocilizumab | |
| | | IL-1 inhibitor | Anakinra | |

Conversely, some researchers rebut the positive effects of DMARDs on Pd or, reported a declined periodontal activity following anti-RA therapy. This controversy could be linked to the immune-suppressant therapy which put RA patients at more risk of having infections, including periodontitis. In addition to the drawbacks of such studies, including fewer samples, follow-up variability, the diversity criteria, and the few number of randomized clinical trials. Furthermore, periodontitis may impact the potency of DMARDs, as the inflamed periodontal tissue act as an extra supplier for pro-inflammatory cytokines(30), and progression of systemic inflammation may delay treatment outcome in RA patients (31). Previously, it was thought that immunosuppressive medications are among the risk factors for periodontitis, as with other types of infection, but this was refuted by the newest scientific evidence (32). Eventually,

severe periodontitis was found to be associated with higher activity in RA independent of anti RA therapy (33,34).

## Periodontal Treatment Impact on Rheumatoid Arthritis

Various modalities of non-surgical periodontal therapies (NSPT), namely, scaling and root planning(SRP) alone or combined with mouthwashes and photodynamic approaches, have been investigated for RA patients who have periodontitis (32). Most have a positive impact on RA prognosis. To the best of our knowledge, no studies were performed for the effect of surgical periodontal therapy on RA patients.

A notable reduction in anti-CCP and anti-P. gingivalis antibody and other systemic inflammatory markers levels 6 months after nonsurgical periodontal therapy(NSPT) to periodontitis patients with RA, indicating a possible positive impact on the activity of rheumatoid arthritis and is even more beneficial during early-phase RA (35). This benefit rely on the pre-treatment Pd severity and the degree of periodontal response to treatment (36).

The effect of NSPT on clinical and biochemical parameters associated with RA activity encompasses: a significant decrease in the erythrocyte sedimentation rate(ESR), with a reduction in RF, TNF-α levels, and 28-joint disease activity score with ESR (37,38). In contrast, the levels of C-reactive protein(CRP), ACPA, or IL-6 level had no substantial reduction (39). Other studies reported a notable reduction in DAS28 with ESR and CRP levels 6 months following NSPT (40,41). Discrepancies among the studies are due to variation in baseline patient characteristics and constraint methodologies. The effect of periodontal therapy on biomarkers of inflammation of RA is illustrated on table 2.

**Table. 2:** biomarkers that are decreased in level after NSPT in different body fluids of RA patients

| **Body fluid** | **Decreased Biomarkers** |
|---|---|
| serum | TNFa-α, IL-6, receptors activator of nuclear factor-KB ligand (RANKL), carbamylated protein (CarP), neutrophil extracellular traps (NETs), the apoptosis inhibitor survivin, and the neuroendocrine hormone prolactin(42–44). |
| GCF | TNF-α, IL-1β, IL-6, matrix metalloproteinase-8 (MMP-8), prostaglandin E2 (PGE2), tissue plasminogen activator (t-PA), plasminogen activator inhibitor-2 (PAI-2), and prolactin(44)(45). |
| synovial fluid | prolactin(45) |
| Saliva | RANKL(42) |

Other periodontal treatment modalities, such as mouthwash containing clove oil, eucalyptol, thymol, and tea tree, together with SRP, have been demonstrated to be more beneficial in lowering ESR, CRP, ACPAs, and RF after 6 weeks than only NSPT or NSPT with a chlorhexidine mouthwash (46). The in site implementation of curcumin is an effective adjuvant for RA patients with periodontitis.

Methylene blue-based photosensitizer and diode laser application inside periodontal pockets with SRP greatly lowers IL-6 and TNF-α levels in individuals with Pd and RA at six weeks more than only SCR (47). But does not affect RF level in GCF.

The outcome of SRP in decreasing local inflammation in RA patients was improved by photodynamic laser therapy due to triggering a toxic photo reaction that permit fastidious damage of pathological tissues (32).

Regarding periodontitis treatment in RA patient and non RA patient, RA does not influence the effectiveness of SRP in periodontitis (48). The mechanism by which periodontal treatment lower the clinical and biochemical parameters of RA is probably related to interfering with the pathogenesis (49) as demonstrated in figure 1.

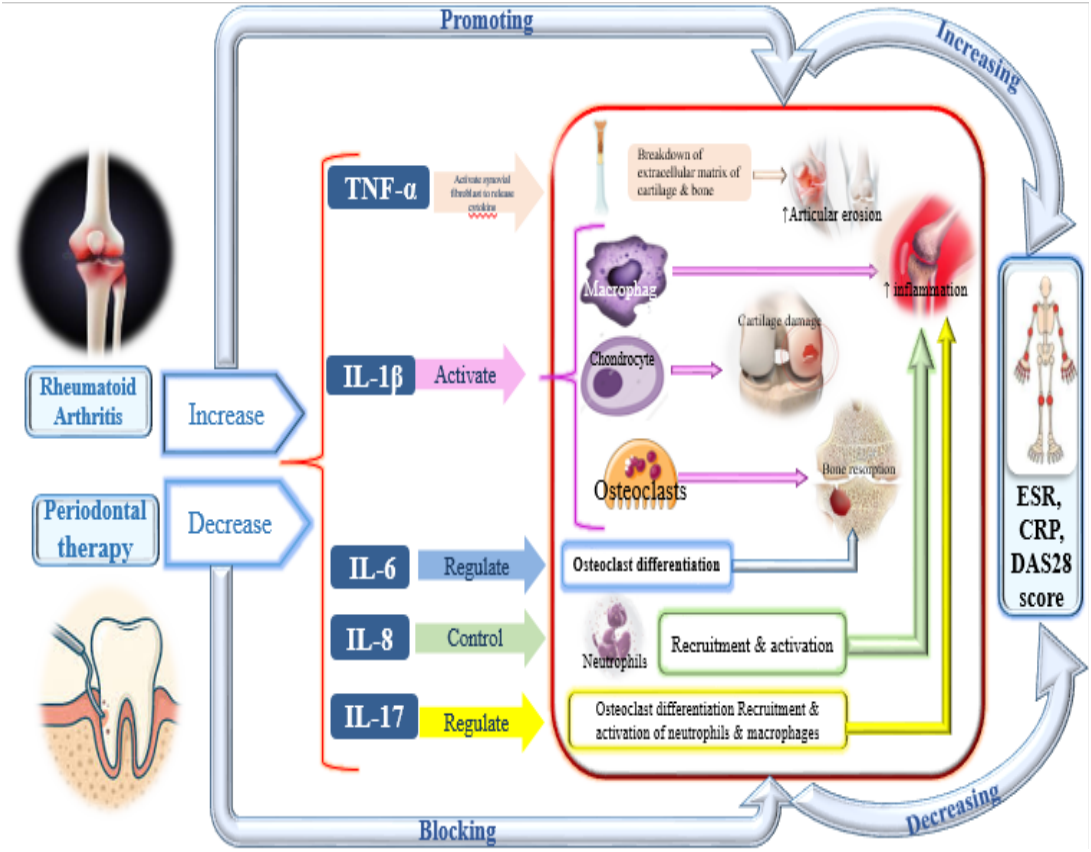


Figure 1: The influence of periodontal treatment on RA patient with periodontitis

## Implications for Prevention and Therapy

Since citrullination , which is key step in RA pathogenesis (8) is greatly influenced by periodontal inflammation and the abundance of periodontal pathogens (7), prevention and treatment to reduce both inflammation and pathogens are essential in the management of periodontitis patients with RA, they have to be considered prior to RA treatment (51). Accordingly, both the rheumatologist and the periodontist should recognize the significance of periodontal health in RA patients(16).

## Conclusion

According to previous publications the periodontal evaluation and management must be integrated in the management of RA and called "treat-to-target". Additionally, periodontal assessment must be constantly incorporated within the guidelines of RA therapy to diagnose and treat periodontitis as early as possible.

## Declarations

**Acknowledgment**
None

**Ethics statement**
Ethics statement: The authors declare that this study was conducted in accordance with the ethical standards and guidelines outlined in the journal's "Ethics Approval" section of the author guidelines. As this work is a narrative review, and the formal ethical approval was not required.

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**Author contributions**
NH and MSM provided the concept, data analysis, and writing of the manuscript; NH and worked with data collection and analysis; MSM revised the manuscript and analyzed the data. NH and MSM revised and approved the final version.

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