Review Article

"The Periodontitis – Rheumatoid Arthritis Axis: Pathogenesis and Therapeutic Insights" – A Review

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ABSTRACT

Rheumatoid arthritis (RA) and Periodontal disease (PD) are chronic inflammatory diseases with significant morbidity and socioeconomic burden. RA is characterized by synovial inflammation, autoantibody production, progressive joint destruction, whereas PD involves dysbiotic microbial biofilms, host immune dysregulation, and alveolar bone loss. Both conditions share common immunopathogenic mechanisms, genetic susceptibility, and environmental risk factors, suggesting a potential bidirectional relationship. Recent evidence suggests that the oral microbiome may contribute to systemic immune modulation and autoimmunity, with periodontal pathogens such as *Porphyromonas gingivalis* implicated in protein citrullination and the generation of anti-citrullinated protein antibodies. Advances in genomics, proteomics, coupled with biomarker identification and microbial profiles, offer opportunities for early diagnosis and risk stratification. Therapeutically, periodontal intervention, biologic disease-modifying antirheumatic drugs and targeted anti-cytokine therapies may have synergistic benefits in controlling both periodontal and systemic inflammation. Personalized and multidisciplinary approaches integrating dental and rheumatologic care could improve patient outcomes.

Keywords: Anti citrullinated protein antibodies, periodontitis, *Porphyromonas gingivalis*, protein – arginine deiminases, rheumatoid arthritis

1. INTRODUCTION

Rheumatoid arthritis (RA) is a chronic, inflammatory, systemic autoimmune disease characterized by the accumulation and persistence of an inflammatory infiltrate in the synovial membrane, resulting in cartilage and bone devastation, affecting about 0.4–1.3% of the global population and more commonly women. It is also termed "immortal cancer," causing huge burdens on patients and society [1–3].

Periodontal disease (PD) is a chronic inflammatory condition of the supporting tissues (gingiva, periodontal ligament, and alveolar bone) around the teeth, caused by a dysbiotic microbial biofilm on the tooth surfaces. It is common in elderly patients and is a major cause of tooth loss. It may be a risk factor for the development of other systemic inflammatory disorders[3,4].

The connection between PD and RA has been extensively investigated, as the two conditions share common inflammatory pathways, histopathologic features, and genetic/ environmental risk factors [3, 5].

2. RHEUMATOID ARTHRITIS: OVERVIEW AND PATHOPHYSIOLOGY

RA initiates with inflammation of joint and synovial tissue proliferation, followed by massive cell infiltration and surplus formation of inflammatory mediators and production of autoantibodies. It is associated with significant comorbidities (cardiovascular illness; skeletal disorders such as periarticular bone loss, juxta-articular bone erosion, joint ankyloses, and fractures), socioeconomic burden, and mortality [3, 6]. The joints of the hands, wrists, and feet are

most commonly affected, with associated muscle spasms and ligament weakening [1].

To date, the exact etiopathology of RA remains largely elusive. As with many other autoimmune diseases, it is proposed that, in genetically predisposed individuals, various environmental factors may trigger a loss of immune tolerance in secondary lymphoid organs, leading to the production of multiple autoantibodies [2].

2.1. PATHOGENESIS OF RA

Immune dysregulation was first implicated in the pathogenesis of RA with the discovery of Rheumatoid factors (RF), an anti-immunoglobulin G (IgG) antibody, initially by Erik Waaler and later fully described by Rose [6].

Increase and activation of both macrophage-like synoviocytes (MLSs) and fibroblast- like synoviocytes (FLSs) lead to the production of diverse pro-inflammatory cytokines such as interleukins (IL-1, IL-2, IL-6, IL-17), tumor necrosis factor- α (TNF-α), and matrix metalloproteinases (MMP) [7, 8] . Infiltration of immune cells into the synovial sublining leads to hallmark "pannus" formation, which consists of macrophages, FLSs, dendritic or plasma cells, and mast cells, and it mediates damage and erosion formation [8] .The cartilage-pannus junction is the site of predominant cartilage destruction [7].

The T Cell-mediated immune response, particularly Th1 cells and Th17 cells, plays a crucial role by inducing the production of IL-17 ,TNF-α, IL-1β, and IL-6 and by stimulating the receptor activator of nuclear factor-kβ ligand (RANKL) expression, leading to osteoclastogenesis and increased inflammation [7, 8].

IL-1β and IL-6 reduce regulatory T cell (Treg) inhibitory function and induce T cell plasticity by downregulating forkhead box P3 (Foxp3) expression and contribute to immune dysregulation and autoimmunity [8].

The functions of B cells, specifically autoantibody production, antigen presentation, and cytokine secretion, are related to the pathogenesis of RA. Toll-like receptor (TLR)-driven autoreactive B cells generate autoantibodies, which include RF, anti-citrullinated protein antibodies (ACPA), anti-modified citrullinated vimentin antibody, anti-carbamylated protein antibody, anti-PAD-4-antibody, and anti-GPI antibody [8, 9].

Citrullination (deimination) is a post-translational enzymatic modification that converts arginine into citrulline by calcium dependent peptidyl arginine deiminases (PADs). In genetically susceptible (e.g., in shared epitope-positive) individuals, they can act as autoantigens, leading to the production of autoantibodies. To date, four major citrullinated autoantigens have been identified: collagen type II, fibrinogen, vimentin, and α- enolase, which form immune complexes and amplify inflammatory response [9, 10].

2.2. DIAGNOSIS OF RA

The diagnosis of RA is based on the 2010 European League Against Rheumatism (EULAR) classification criteria [1], clinical history, laboratory tests (presence of antibodies, acute phase reactants), and also on imaging methods, such as magnetic resonance imaging (MRI) and ultrasonography methods. The Disease Activity Score including the 28-joint count (DAS28) is also used in the clinical assessment of the disease activity [3].

3. PERIODONTAL DISEASE: OVERVIEW AND PATHOPHYSIOLOGY

The tissue destruction in PD results from the interaction between bacterial products, inflammatory cells, and mediators in disease-susceptible individuals, causing the shift from periodontal health to dysbiosis. This is characterized as a complex disease with multifactorial etiology, with both genetic and environmental factors [3, 4].

Clinically, it presents with gingival inflammation, spontaneous bleeding, bleeding on probing (BOP), gingival recession, increased probing pocket depth(PPD), clinical attachment loss (CAL), periodontal bone loss, increased tooth mobility, and eventual tooth loss [2, 3].

Three bacterial species are strongly correlated with PD: *Porphyromonas gingivalis*, *Treponema denticola*, and *Tannerella forsythia* are referred to as the "red complex".

3.1. PATHOGENESIS OF PD

PD results from polymicrobial dysbiosis due to a microbial shift from Gram-positive to Gram-negative microbiota, activating innate immune responses via TLR and disrupting the balance between pro- and anti-inflammatory mediators [9, 11].

It has been described that proinflammatory mediators such as interleukins, prostaglandin E2, MMP, and nitric oxide (NO) play a crucial role in the pathogenesis of PD. IL-10 and transforming growth factor-β1(TGF) were downregulated in active periodontal lesions.

IL-17 induces RANKL expression by stimulating MMP-1, MMP-3, IL-6, and IL-8 secretion from human gingival fibroblasts and TNF release from macrophages, resulting in osteoclast differentiation & periodontal destruction [3, 9].

3.2. DIAGNOSIS OF PD

Periodontal examination is mainly based on clinical and intra-oral radiographic examination involving BOP, PPD, CAL, tooth mobility, furcation involvement, and alveolar bone loss [3].

The current classification proposed by the World Workshop is based on the complexity/severity and progression of the disease represented by Stages I–IV and Grades A–C.

4. INTERRELATIONSHIP BETWEEN RA AND PD

The scientific concept claiming that oral infections can contribute to systemic inflammatory diseases, including arthritis, was first proposed in the 19th century and further developed in the 20th century. PD severity was identified as the third most potent predictor of RA, apart from female gender and smoking [9].

Potential mechanisms linking RA and PD, because of the numerous similarities in pathological and immunological characteristics [3, 11], include:

1. Increased infiltration of inflammatory and immune cells, including neutrophils, monocytes, and T and B lymphocytes.
2. Increased release of pro-inflammatory mediators such as TNF-α, IL-1β, IL-6, and matrix-degrading enzymes (MMPs, Cathepsin).
3. Increased activation of the RANKL pathway induced by inflammatory mediators with subsequent osteoclast differentiation and maturation.
4. Decreased levels of anti-inflammatory mediators, such as the IL-10 and TGF-β1.
5. The etiology of both diseases is multifactorial, which includes genetic and environmental components.

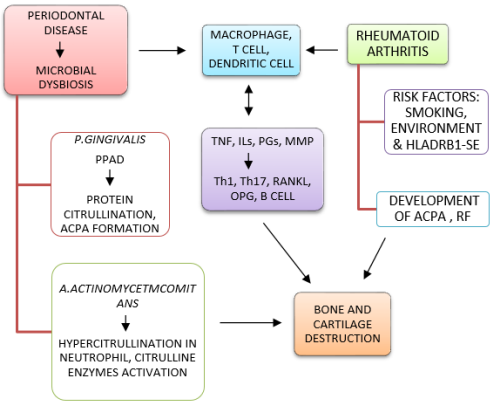

Figure 1 Possible Biological Interactions Between Rheumatoid Arthritis and Periodontal Disease

5. TWO HIT MODEL LINKING RA AND PD

The first hit is triggered by PAD-producing *P. gingivalis* in the periodontal microenvironment, leading to increased local citrullination of peptides and production of ACPAs. The second hit is represented by cross-reactivity of periodontal-generated ACPAs to antigens present in the joint microenvironment, further aggravating the inflammation associated with RA [3].

6. EPIDEMIOLOGICAL AND GENETIC ASSOCIATIONS

6.1 SHARED GENETIC SUSCEPTIBILITY

Certain genes have been found to linked with an increased risk of developing both ACPA-positive RA and PD. One potential genetic influence connecting RA and PD is the shared epitope (SE)-coding HLA-DRB1 allele [3, 11].

The HLADRB1 SE alleles have been associated with bone erosions in RA and alveolar bone destruction during PD progression [9]. Experimental studies in mice have shown that HLADRB1 may influence ACPA generation following *P.gingivalis* infection [3].

Other genetic variants of tyrosine phosphatase (PTPN22), interferon regulatory factor 5 (IRF5), and PR domain zinc finger protein 1 (PRDM1) contribute to a shared susceptibility between PD and RA [9].

6.2. SMOKING AS COMMON RISK FACTOR

Smoking is considered an environmental risk factor for both PD and RA. It stimulates peptide citrullination by PAD2 and PAD4, which play a role in development of ACPA-positive RA. Smoking is associated with an increased load of *T. forsythia*, *Peptostreptococcus micros*, *Fusobacterium nucleatum*, and *Campylobacter rectus* in subgingival dental plaque, contributing to periodontal inflammation [9].

7. ORAL MICROBIOME AND AUTOIMMUNE PATHOGENESIS

The most widely regarded mechanism by which periodontitis and periodontopathic bacteria may contribute to the development of RA is protein citrullination and subsequent ACPA production accelerated by the increased expression of neutrophil extracellular traps (NETs) , PAD-2, and PAD-4 in periodontitis patients [12].

Periodontal disease-associated oral mucosal breaks result in oral bacteremias that trigger innate and adaptive immune responses, which likely contribute to the pathogenesis of RA. Oral bacteria released into the blood may disseminate to the liver, spleen, joints, and gut [13]. Recent studies have reported an enrichment of oral *Streptococcus* species in the gut of RA patients.

Patients with RA exhibit higher serum and synovial fluid antibody levels against periodontal pathogens, including *P.gingivalis*, *Prevotella intermedia*, *Prevotella melaninogenica*, and *T.forsythia* [11].

In addition, periodontal bacterial DNA has also been detected in serum and synovial fluid, suggesting possible transport mechanisms via the bloodstream, viable cells, or phagocyte-mediated transport. Other periodontal bacteria play a role in initiating RA, but *P. gingivalis* and *Aggregatibacter actinomycetemcomitans* contribute at a later stage to maintain the disease [9, 14].

7.1. ROLE OF *P.GINGIVALIS*

P. gingivalis is the only known bacterium (wild type strain) expressing bacterial peptidyl arginine deiminase (PPAD), which citrullinates host and bacterial proteins, promoting ACPA production and RA development. Host PAD-mediated citrullination may be augmented by bacterial-derived PADs.

Gingipains, the most important virulence factors of *P. gingivalis* , generate arginine containing peptides that are citrullinated by PPAD, forming citrullinated proteins. PPAD and citrullinated PPAD peptide (CPP) act as autoantigens in RA [3, 11]**.** Anti-RgpB is elevated in pre-RA and established RA individuals [9]**.** *P. gingivalis* shows a significant correlation with RF-IgA, suggesting a potential association of this taxon with more severe RA.

In addition, *P. gingivalis* also induces the formation of pro-inflammatory cytokines (IL-6, IL-1β), stimulating a Th17 cell response that may accelerate inflammation of the gingival mucosa or the synovial membrane of the joint [3, 12].

7.2 ROLE OF *A. actinomycetemcomitans*

A.actinomycetemcomitans produces leukotoxin A (LtxA), a pore-forming RTX-family toxin that mainly binds to the leukocyte receptor LFA-1, leading to leukocyte (neutrophils) death, untamed bacterial growth, and potent immune activation [9]. This binding induces PADs to catalyze the production of autoantigens, which are released by lysing cells in the form of leukocyte toxicity hypercitrullination (LTH). This process releases autoantigens, which can trigger autoantibody production, contributing to RA [9, 11].

A recent study showed that the clinical symptoms of arthritis and ACPA levels were successfully diminished when the antibiotic treatment against *A. actinomycetemcomitans* was prescribed for the patient, who was presented with *A. actinomycetemcomitans* endocarditis [3].

8. MOLECULAR MECHANISMS LINKING RA AND PD

8.1. RHEUMATOID FACTOR AND AUTO ANTIBODY GENERATION

Gingipains from *P. gingivalis* are responsible for epitope exposure in RF-Fc region and have a key function in RF production [9]. Carbamylated proteins(CarP) and malondialdehyde–acetaldehyde (MAA)–modified proteins act as neoantigens and induce autoantibodies such as anti-CarP and anti-MAA, which are detected in both RA and inflamed periodontal tissues, suggesting a shared autoimmune response [9, 11]. Serum anti-*P. gingivalis*, anti-*P. intermedia*, and anti-*F. nucleatum* antibody concentrations also were positively associated with serum anti-MAA antibody concentrations, indicating a possible relationship between oral pathogens and anti-MAA antibodies [5, 9].

8.2. NEUTROPHIL EXTRACELLULAR TRAP (NETosis) IN RA

During NETosis, neutrophils release web-like structures called NET, which releases autoantigens and certain enzymes, including PADs, involved in the citrullination and autoantibody production [11] . The oral microbiota in PD is associated with the release of NETs . Elevated NET levels increases an individual's risk of developing RA (pre-RA individuals) and the progression of chronic RA [15]. Periodontitis-induced NETs also produce CarP and anti-CarP antibody, followed by joint inflammation and the development of RA [12].

8.3. MALADAPTIVE INNATE IMMUNITY TRAINING

PD induces the production of serum granulocyte-macrophage colony-stimulating factor(GM-CSF), which enters the bone marrow and stimulates the secretion of IL-1 by mature neutrophils, thereby facilitating trained myelopoiesis and exacerbating the systemic inflammatory response. Consequently, this process increases susceptibility to RA in experimental PD. Conversely, alterations in hematopoietic stem cells of experimental RA lead to maladaptive inflammatory phenotypes, which worsen PD susceptibility, indicating a bidirectional correlation [11].

8.4. INFLAMMATORY MEDIATORS

Inflammatory pathways may provide another mechanistic link between periodontitis and RA. In both diseases, inflammatory challenges may result in the secretion of excessive pro-inflammatory mediators, of which PGE2, IL-1, TNF-a, IL-6 , IL 17, RANKL, and OPG would appear to dominate, with key MMPs including MMP-1, MMP-3, MMP-9, and particularly MMP-8. Acute phase proteins such as ESR and CRP are also elevated in both conditions [4, 16]. Local immune activation resulting from periodontal disease may facilitate the migration, activation, and proliferation of immune cells via PTPRC gene, thereby potentiating the inflammation in RA [17]. Proinflammatory cytokines could stimulate STAT3 activation (Signal Transducer and Activator of Transcription 3), playing a key role in the pathogenesis of RA and PD [18]. Increased levels of periodontitis-associated mediators in oral fluids of the RA patients, particularly the neutrophil-associated mediators IL-8 and MMP-8 [19].

9. THERAPEUTIC INTERACTIONS BETWEEN RA AND PD

9.1 INFLUENCE OF ANTIRHEUMATIC THERAPY ON PERIODONTAL HEALTH

RA treatment involves the use of various pharmacological agents, including nonsteroidal anti-inflammatory drugs, glucocorticoids (GC), and synthetic and biological disease-modifying antirheumatic drugs (DMARDs) such as methotrexate, sulfasalazine, TNFα inhibitors, IL-1β monoclonal antibodies, and IL-1 receptor antagonists . These classes of drugs reduce pain and inflammation of the joint and therefore ameliorate the signs and symptoms of the disease. The TNF blockers used for the treatment of patients with RA resulted in a significant reduction of biochemical markers of PD, including IL-1β and IL-8, in the GCF of patients with established periodontitis. The treatment of RA patients with DMARDs and anti-TNF decreased the extent of CAL, BOP, and gingival index (GI) compared to patients without the treatment [3].

9.2. INFLUENCE OF PERIODONTAL TREATMENT ON RA ACTIVITY

The primary treatment for periodontitis is non-surgical periodontal therapy (NSPT), consisting of scaling and root planning to reduce microbial burden and inflammation and reestablish host–microbial homeostasis. NSPT in patients with both RA and PD offers benefits in terms of clinical activity, including improvement in DAS28 , tender joint counts (TJCs), swollen joint counts (SJCs), visual analogue scale (VAS) and reduction in inflammatory markers of RA such as CRP and ESR [3, 21]. The DAS28 score showed a tendency towards lessened signs and symptoms of disease, with a decrease in the number of painful joints and lesser morning stiffness and joint effusion, independently of the drugs used to treat RA [22]. Additionally, NSPT was also associated with decreased serum and salivary calprotectin levels, a key biomarker of RA activity [23].

10. EMERGING THERAPEUTIC TARGETS

In recent decades, understanding of immunopathological mechanisms has led to the development of treatments targeting specific components of the immune response. The IL-23/Th17/IL-17 axis represents a common immunopathogenic pathway in RA and PD, promoting bone and tissue destruction, and may serve as a potential therapeutic target in both conditions [20]. The emergence of the concept of osteoclast precursors (OCPs) subsets which are involved in inflammation provides new therapeutic opportunities for RA or periodontitis. Therapeutic approaches targeting, in particular, the OCPs that are promising in RA could be of potential interest in periodontitis due to the immunopathogenic similarities between the two diseases. Currently, cytotoxic T lymphocyte antigen-4-immunoglobulin (CTLA-4-Ig) and thiostrepton, which affect monocytic cells are the two therapies targeting OCPs in RA, showing promising results. No treatments targeting specific OCPs have been studied in periodontitis due to the lack of data. However, given the immunopathogenic similarities, these therapies could be interesting to explore once OCPs are characterized [24].

11. CONCLUSION

The accumulating evidence strongly supports a bidirectional relationship between RA and PD , driven by shared immunopathogenic mechanisms. Future longitudinal and interventional studies are needed to establish causal links and to evaluate periodontal therapy as a modifiable risk factor for RA. Advances in genomics, proteomics, and microbiome profiling may help identify susceptible individuals and biomarkers for early diagnosis. Integration of periodontal and rheumatologic care may represent a novel strategy to improve systemic and oral health outcomes.

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CONFLICT OF INTEREST

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