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Review Article

Connecting Rheumatoid Arthiritis and Periodontitis at Molecular and Cellular Level- Literature Evidence

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Abstract

Periodontitis and rheumatoid arthritis (RA) are both widespread chronic inflammatory diseases that destroy bones and supporting structures around teeth or joints, respectively, and can lead to permanent disability. These two inflammatory conditions share etiological, microbiological and pathogenic features, such as similar cytokine profiles, generation of anti-citrullinated protein antibodies (ACPAs) by involving Porphyromonas gingivalis and smoking as a major risk factor. Inflammatory systemic disease may increase the severity and incidence of periodontal disease. Periodontal pathogens and inflammation are the main culprit which linked directly etiopathogenesis of periodontitis and rheumatoid arthritis. Previous studies have proposed an association between RA and periodontitis, based on prevalence/incidence and severity of periodontitis although the strength and temporality of this association is still unclear. The following facts includes similar association between periodontitis and RA, the potential bacterial link, as well as an overview of previous studies conducted on the relationship between these two diseases.

Keywords: Inflammation; Periodontitis; Bone resorption; Citrullinated protein; Rheumatoid arthritis

Introduction

Rheumatoid arthritis is the chronic multisystem disease of unknown aetiology with a characteristic feature is presence of persistent inflammatory synovitis which may involve the peripheral joint in a symmetric distribution. It is a progressive disease of synovial lining of peripheral joints characterized by symmetrical inflammation leading to potentially deforming polyarthritis. It is the most common systemic inflammatory disease characterized by symmetrical joint involvement. Extra-articular involvement, including rheumatoid nodules, vasculitis, eye inflammation, neurologic dysfunction, lymphadenopathy and splenomegaly, can be the manifestations of the disease [1].

It affects up to 1% of population worldwide and is more commonly seen in females with significant evidence of co-morbidity including Periarticular bone loss, juxta-articular bone erosion, joint ankyloses and fractures. It increases with age and mostly affecting the third and fifth decade of life [2]. This auto immune disease has a complex association between environmental and infectious risk factors results in the release of autoantibodies and the rise of rheumatoid arthritis. On the other hand, periodontitis is a dysbiotic inflammatory condition of hard and soft tissue surrounded by teeth caused by local anaerobic bacterial colonization especially by "red complex" microorganisms Porphyromonas gingivalis which activates the neutrophilic and immune complex, resulting in soft tissue destruction and alveolar bone loss. Recent evidence indicates that there is a bidirectional link between periodontal disease and number of systemic diseases such as DM, COPD, Pre-term low birth weight, cardiovascular disease and osteoporosis [3].

Snyderman and McCarty were the 1st ones to identify the Periodontitis and RA's common features in 1982. Periodontitis has a remarkably similar pathobiology to Rheumatoid Arthritis. Progress of both the diseases consists of high levels of Proinflammatory cytokines, Low levels of tissue inhibitors of metalloproteinases, High levels of MMP and PGE_2. In addition to this, a well-established risk factor for both diseases is cigarette smoking. There is a higher rate of bone loss, attachment loss and edentulism in case of smokers as compared to non-smokers [4]. In RA, cigarette smoking has been reported to increases the risk of seropositive disease (both ACPA and RF) in genetically predisposed individuals, and has also demonstrated a dose-dependent effect regarding the risk

of ACPA-positive disease [5, 6]. Furthermore, increased levels of citrullinated proteins have been reported in inflamed periodontal tissue, and expression of ACPAs has been demonstrated in gingival crevicular fluid (GCF) and saliva of patients with RA [7]. Similarly, RF has been reported in gingiva, subgingival plaques and in serum of patients with periodontitis.

Similar Features of Rheumatoid Arthritis and Periodontitis

1. The "red complex" microorganisms *P. gingivalis* is the biological link between periodontal and rheumatic pathology via citrullination [8]. It may result in local activation of defense system, change in the microbial composition and chronic inflammation due to release of gingipains and protease enzymes.

2. *P. gingivalis* generates gingipains which degrade complement components such as the C3, C4 and C5a, resulting in inhibition of the complement activation and immune complex deposition.

3. Genetically, human leukocyte antigen DR4 (HLA-DR4) and IL-1, IL-10 gene polymorphism are the markers which triggers the inflammatory process in both the diseases [9].

4. Immunomodulatory cells such as T-cells, plasma cells, macrophages, polymorphonuclear leukocytes release various factors such as pro-inflammatory cytokines, chemokines and MMPs, which are actively involved in the pathogenesis of periodontal disease and RA.

5. Production of cytokine profiles characterized by high levels of inflammatory mediators (such as PGE2, TNF-α, IL-6 and IL-1α, IL-1β, IL-8) and degradation enzymes (e.g. MMP1, MMP9 and MMP13), and decreased levels of anti-inflammatory mediators (such as IL-10 and TGF-β)

6. Because of release of large number of signalling molecules by inflammatory cells in the synovia/gingiva may result in degradation of tissue and bone.

7. Bone resorption is one of the important parameters in both the diseases. There is a osteoclast activation (RANKL overregulation) which actively promote the bone destruction through increasing the RANKL expression and reducing the OPG synthesis in osteoblast and stromal cells.

8. Citrullinated protein results in the generation of neoepitopes, another important link between periodontitis and RA which produces anti-cyclic citrullinated peptide autoantibodies affects the periodontal/synovial tissue.

9. Because of release of ample amount of pro inflammatory mediators ROS may directly cause bone resorption, degradation of ground substance and an imbalance between pro and anti-inflammatory systems in the periodontal/synovial micro-environment.

Hypothetical two-hit model

This model investigates the results from animal models and supported by the evidence of human clinical studies and hypothesize the idea of destruction of bone and connective tissue at one point for example synovial joints in patients with RA which through the generation of endotoxins from the microorganisms in subgingival biofilm may communicate with the tissues of the periodontium to co-induce periodontitis. Additionally, there is a systemic destruction of multi joints because of the elevation of host derived MMPs such as collagenase and gelatinase in the circulation but also cause local destruction of soft and hard tissue resulting in bone loss and tooth mobility.

Both the diseases share common genetic and environmental factor including smoking and even the infection caused by periodontal pathogen *P. gingivalis*. These risk factors are the linking evidence to establish RA and periodontitis. There are two schools of thought for both the disease to establish this relationship [10, 11].

1. In the "first hit" because of chronic inflammation the microbial products such as endotoxins becomes active which initiates the autoantibody system, protein antibodies (ACPAs) and citrullination process.

2. In the "second hit" there is an involvement of systemic diseases which may increase the biomarkers such as C reactive proteins, cytokines, MMPs, and prostaglandins in the circulation.

In another words there is a mutual exacerbation of inflammatory response resulting in tissue destruction including bone loss in both periodontitis and RA.

Biological link of RA and periodontitis- cross roads

Periodontal pathogens such as *P. gingivalis* present in subgingival biofilm played a central role and link the disease as a bidirectional relationship. There is a production of autoantibodies against the self-antigens which are expressed in inflamed joints. Literature studies stated that the synovial fluid contain the DNA of *P. gingivalis* and there is a migration of DNA of *P. gingivalis* from oral cavity to the joints in a free form. *P. gingivalis* is the only periodontal pathogen which produced Peptidyl Arginine Deiminase (PAD) enzyme and takes an important step of citrullination process. During physiological citrullination the enzyme Peptidyl Arginine Deiminase generate citrullinated peptides and arginine group is replaced by citrulline. The enzymatic deimination of arginine residuals to citrulline through the enzyme PAD is a form of post translational protein modification. Valesini et al in 2015 identified 5 types of PAD enzymes (PAD-1, PAD-2, PAD-3, PAD-4 and PAD-6) with different role and functions performed by them. PAD2 and PAD4 are the most strongly implicated PAD enzymes in rheumatoid arthritis (RA) pathogenesis [12]. *P. gingivalis* is a pathogen of red complex produces the virulence factor gingipain cysteine proteinases, specific for arginine or lysine. *P. gingivalis* has the potential to change the protective immune response to pathogenic one because of changing the cytokine level through lysine gingipains.

There are some other factors which activate the proteins such as cytokines, chemokines, immunoglobulins, complement proteins and host cell receptors that involve in the destruction of host proteins. These factors also degrade the collagen and proteins present in the basement membrane of periodontal tissues. Immunologically *P. gingivalis* may also change the adaptive immune response through the interaction with dendritic cells resulting in

cytokine release and stimulates the development of T helper 17 cells (Th17) with simultaneous downregulation of Th1 cells. *P. gingivalis* also influences the imbalance between Th1 and Th17 lymphocytes, supporting the line T h17 responsible for inflammation [13].

PAD is an important pathogenic factor and pivotal process in the pathogenesis of RA. The breakdown of mucosal tolerance results in aberrant interactions between the mucosal immune system and local microbiota, which eventually lead to inflammation in synovial joints. Alterations in mucosal microbiota (dysbiosis) have been observed in the oral cavity, and in patients with early and established RA. In patients with RA, *P. gingivalis* is known to induce the citrullination process. There is decreased immunotolerance to citrullinated proteins which is the key problem especially for RAs with high severity results in an increased formation of autoantibodies. *P. gingivalis* alter the integrity of epithelial tissue, infiltrate human endothelial cells, and affect transcription and protein synthesis and facilitate the direct systemic entry into bloodstream. The virulence factors gingipains facilitate the activation of proteolytic enzymes such as MMP-1,3 and 9 resulting in degradation of extracellular matrix host proteins including collagen, fibronectin, and laminin. Also, gingipains induce the degradation of complement factors and enhance the permeability of blood vessels [14, 15].

There is a genetic predisposition of *P. gingivalis* which supports both inflammatory conditions RA and periodontal disease. The most prominent genetic associations are the Human Leukocyte Antigen (HLA)-DR gene, in particular HLA-DRB1 located on chromosome no. 6 This association has suggested a role for antigen presenting cells (APCs) governing T cell range selection as well as their hyper-activation in RA pathogenesis [16]. Both RA and periodontal disease shared the same epitope genotype along with DRB1 which play an important role in the inflammation process.

Literature Evidence Summarizies the Relationship Between RA and Periodontal Status

Periodontitis and RA shared similar characteristics and risk factors including pathogenic link through periodontal pathogen such as *P. gingivalis,* pathways of tissue destruction and environmental risk factors such as smoking. Epidemiological studies suggests that an association exists based on increased incidence of periodontitis in patient with RA and a dose-response pattern between periodontitis severity and RA disease activity. The association between periodontitis and RA hypothesizing the bidirectional relationship and both the diseases crosses the roads based on pathogenic mechanism at some point. Literature studies showed the association of both the diseases with increased incidence/prevalence and disease activity of RA in patients with periodontitis or vice versa.

Table 1, Table 2

Table 1: Incidence/Prevalene and Disease Activity of RA in Patients with Perioontitis.

STUDY / AUTHOR	COUNTRY	SAMPLE SIZE	RA DIAGNOSTIC CRITERIA + PERIODONTAL PARAMETERS	FINDINGS/CONCLUSION
Scher JU et al 2012 [17]	New York	83	RA patients meeting 2010 ACR/EULAR criteria for RA, including presence of rheumatoid factor (RF) and/or anti-citrullinated peptide antibodies. Periodontal status was assessed according to AAP. **Periodontal parameters** included probing depth, clinical attachment level and bleeding on probing	new-onset rheumatoid arthritis (NORA) patients exhibit a high prevalence of PD at disease onset, despite their young age and paucity of smoking history. The subgingival microbiota of NORA patients is similar to CRA and healthy subjects of comparable PD severity.
Chen et al 2013 [18]	China	13779	RA patients (International Classification of Diseases, 9th revision, clinical modification (ICD9-CM) code 714.0) with a certificate of catastrophic illness between 2001 and 2006. American College of Rheumatology classification criteria for RA (1987) were used for RA diagnosis. periodontitis severity included the cumulative number of periodontitis-related visits (i.e., 1–2, 3–4, 5–7 and ≥8), the cumulative cost of periodontitis-related visits according to the 25th, 50th and 75th centiles, and the receipt of periodontal surgery before the index date.	Association was found between a history of periodontitis and newly diagnosed RA. The strength of this association remained statistically significant after adjustment for potential confounders. He association was dose- and time-dependent and was strongest when the interval between the last periodontitis-related visit and the index date was <3 months.
De Smit M et al 2012 [19]	Netherland	95	RA patients classified according to American College of Rheumatology classification 1987 criteria including Disease Activity Score 28 joint count (DAS28). Periodontitis was diagnosed using Dutch periodontal screening index (DPSI) score was assessed during one year (2010).	A higher prevalence of severe periodontitis was observed in RA patients in comparison to matched non-RA controls. RA patients with severe periodontitis have a more robust antibody response against P. gingivalis than non-RA controls, but not all RA patients have cultivable P. gingivalis.
Potikuri et al 2012 [20]	India	91	RA patients classified according to American College of Rheumatology classification 1987 criteria including Disease Activity Score 28 joint count (DAS28). Periodontal disease diagnosed with mean pocket depth (MPD) is ≥3 mm.	Patients with RA had significantly higher mean pocket depth, IgM-RF titres and ACPA titres as compared to those without PD. PD is more frequent and severe in non-smoking DMARD-naive RA patients compared with healthy controls. PD in RA is associated with high titres of ACPAs.

Mikuls TR et al 2014[21]	USA	287	RA patients classified according to American College of Rheumatology classification 1987 criteria Periodontitis was diagnosed using Probing depth, gingival recession and Bleeding on probing.	PD was more common in RA and aCCP positive RA. PD was associated with increased swollen joint counts, total Sharp scores, aCCP and RF. Associations of PD with established seropositive RA were independent of all covariates examined including evidence of Pg infection.
Choi IA et al 2016 [22]	China	264	RA patients classified according to American College of Rheumatology classification 1987 criteria clinical parameters including 68 tender joint count (TJC), 66 swollen joint count (SJC), disease du ration and medication were evaluated and erythrocyte sedimentation rate (ESR) and serum C-reactive protein (CRP)	The prevalence of moderate or severe periodontitis was higher in RA patients compared to controls. Periodontal structural damage represented by probing pocket depth and clinical attachment level were less in RA patients with human leukocyte antigen (HLA) DRB1 shared epitope compared than those without shared epitope.
Gonzalez SM et al 2015 [23]	United States	617	Cases satisfied the 1987 American College of Rheumatology classification criteria for RA (age of disease onset >18 years). Panoramic radiographs were taken; patients were categorized into low, moderate, or high tertiles based on mean percentage ABL.	ACPA-positive patients with RA had a significantly higher mean percentage of sites with ABL >20% compared with patients with osteoarthritis. ACPAs targeting citrullinated vimentin and histone were significantly higher with greater ABL regardless of smoking status. These results suggest that ACPA targeting, potentially of both vimentin and histones, could provide novel insights between RA and ABL.

Table 2: Prevalene/Severity of Periodontits in Patients with RA.

STUDY / AUTHOR	COUNTRY	SAMPLE SIZE	RA DIAGNOSTIC CRITERIA + PERIODONTAL PARAMETERS	FINDINGS/CONCLUSION
Mercado FB et al 2001 [24]	Australia	65	RA patients classified according to American College of Rheumatology classification 1987 criteria Measures of rheumatoid arthritis included tender joint analysis, swollen joint analysis, pain index, physician's global assessment on a visual analogue scale, health assessment questionnaire, levels of C-reactive protein, and erythrocyte sedimentation rate Periodontitis measures included probing depth, clinical attachment loss and bleeding score, plaque score and radiographic bone loss score.	There is evidence of a significant association between periodontitis and rheumatoid arthritis. This association may be a reflection of a common underlying dis-regulation of the inflammatory response in these individuals.
Ayravainen L et al 2017 [25]	Finland	124	RA patients classified according to American College of Rheumatology classification 1987 criteria. The number of swollen (66 joint count and 28 joint count) and tender (68 joint count and 28 joint count) joints were recorded. Disease Activity Score (DAS28) was calculated from the number of tender and swollen joints (28-joint count), PGA and erythrocyte sedimentation rate (ESR). Periodontitis was diagnosed based on probing depth, clinical attachment loss, Periodontal Inflammatory Burden Index (PIBI)	Moderate periodontitis was more frequent in patients with RA than in controls. Patients with ERA and CRA exhibited poorer periodontal health parameters when compared with controls.
Dissick A et al 2010 [26]	Finland	69	RA patients classified according to American College of Rheumatology classification 1987 criteria Measures of RA disease activity included a multi-dimensional health assessment questionnaire (MD HAQ), C-reactive protein (CRP) concentration, and the four-variable disease activity score-28 [DAS28(4v)] Periodontal disease diagnosed with Bleeding on probing, clinical attachment level, tooth mobility and digital panoramic radiographs to evaluate the presence and severity of periodontitis.	Periodontitis was more common and severe in patients with RA compared to patients with OA. Although unrelated to disease activity, the presence of periodontitis in patients with RA was associated with seropositivity for RF and the anti-CCP antibody, which was highly relevant given the associations of these autoantibodies with poor outcomes and disease pathogenesis in RA.
Coburn BW et al 2014 [27]	USA	617	RA patients classified according to American College of Rheumatology classification 1987 criteria Periodontitis was diagnosed using Probing depth, attachment loss and radiographically alveolar bone loss	Patient self-report, when combined with other risk factors, performs well in identifying periodontitis among patients with RA and OA. Self-report questions related to alveolar bone loss exhibit excellent convergent validity in these patient subsets.
Hiroko H, et al 2021 [28]	Japan	98	RA patients classified according to American College of Rheumatology classification 1987 criteria Periodontitis was diagnosed by probing pocket depth, bleeding on probing and periodontal inflamed surface area (PISA) was used as an indicator of periodontal status.	The strength of the association between the periodontal in flamed surface area (PISA) and the severity of RA increased after adjustment for RA medications, suggesting that severe inflammation of periodontal tissue may increase the severity of RA.

Concluding Remarks

There is growing evidence which supported the direct or indirect link of periodontal disease with RA. Various cross sectional, cohort and case control studies have suggested an association between RA and periodontitis. RA and periodontitis shared similarities in their pathogenesis process, microbiologically the role of p gingivalis in both the diseases and an environmental factor such as smoking may aggravate the inflammatory condition. Additionally genetic risk factor HLA-DRB1 SE alleles have also been implicated as a risk factor for both periodontitis and RA.

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Conflicts of Interest

No conflicts of interest.

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