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Review Article

A closer look at the link between rheumatoid arthritis and periodontal disease

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ABSTRACT

Rheumatoid arthritis (RA) and periodontal diseases (PD) are chronic inflammatory conditions that share a complex and bidirectional relationship. Both conditions exhibit common pathogenic mechanisms, including chronic inflammation, immune system dysregulation, and tissue destruction, suggesting a potential close association. This abstract aims to highlight the emerging evidence on the correlation between RA and PD, focusing on their shared risk factors, underlying biological pathways, and the implications for patient management.

Studies have shown that individuals with RA are more likely to suffer from periodontal diseases, particularly periodontitis, which is characterized by the progressive destruction of the supporting structures of teeth. The presence of periodontal pathogens, notably Porphyromonas gingivalis, has been implicated in the citrullination of proteins—a key process in the development of RA autoantibodies, specifically anti-citrullinated protein antibodies (ACPAs). These antibodies play a central role in RA pathogenesis, indicating a potential triggering link between periodontal infection and RA onset.

Furthermore, systemic inflammation present in RA exacerbates periodontal inflammation and vice versa, creating a vicious cycle. The shared inflammatory mediators, including cytokines such as TNF-α, IL-1, and IL-6, further reinforce the connection between the two conditions. Management of PD has shown to improve clinical outcomes in RA patients, suggesting that periodontal health may influence RA disease activity.

The close association between rheumatoid arthritis and periodontal diseases highlights the need for an integrated approach to diagnosis and treatment. Recognizing and managing periodontal disease in RA patients may reduce systemic inflammation and improve overall disease outcomes. Future research should focus on exploring the molecular links between RA and PD, as well as the potential benefits of combined therapeutic interventions.

1. Introduction

Rheumatoid arthritis is an autoimmune disease which predominantly affects the joints.

It is characterized by the accumulation and persistence of an inflammatory infiltrate in the synovial membrane that leads to synovitis and the destruction of the joint architecture. The classic characteristic of this disease is bilateral and symmetric chronic inflammation of the synovium, a condition known as synovitis. This inflammatory response particularly affects small joints of the upper and lower extremities, and it often leads to the deterioration and eventual destruction of articular cartilage and juxta-articular bone, as well as to an inflammatory

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process surrounding tendons, all of which frequently results in deformities of the affected joints. However, numerous local factors other than bacteria and some systemic factors may also contribute to its aetiology.[1]

Periodontitis refers to an inflammatory condition which affects the tooth and its supporting structure which is initiated by the presence of microbial biofilm present on the surface of tooth and is often aggravated by dysregulated host response. Recent studies have shown that periodontal disease may be related to several systemic diseases including an increased incidence of atherosclerosis, coronary heart disease, myocardial infarction, and stroke. In addition to the above well documented examples, a few other chronic conditions of altered connective tissue metabolism, hormone imbalance and altered immune function have likewise been associated with increased risk of periodontal disease. Of these, rheumatoid arthritis is of particular interest since it is a chronic inflammatory disease which demonstrates remarkably similar patterns of soft and hard tissue destruction to those noted in chronic periodontitis.[2] Although the aetiologies of these diseases are distinctly separate, the underlying pathological processes are of sufficient similarity to warrant consideration of the hypothesis that individuals at risk of developing rheumatoid arthritis may also be at risk of developing periodontitis, or vice versa.

The similar pattern of natural history of RA and periodontitis provides useful insights into these diseases. For both diseases the host response, dictated by immunogenetics, largely determines the inflammatory responses Furthermore, the cells, enzymes and cytokines which determine the degree of tissue damage all share a common pathologic process in both RA and periodontitis. Finally, because of common shared pathologic processes, management strategies aimed at modulating these responses are similar. Finally, because of common shared pathologic processes, management strategies aimed at modulating these responses are similar.

2. Natural History

2.1. Rheumatoid arthritis

At least three types of disease manifestation can also be observed in RA populations:

1. Self-limited: in these cases, individuals originally presenting for RA have no evidence of disease 3 to 5 years later.
2. Easily controlled: the disease is relatively easily controlled with only nonsteroidal anti-inflammatory drugs (NSAIDs).
3. Progressive: these patients generally require second-line drugs, which often still do not fully control the disease.

Table 1: Pattern of disease progression in RA and periodontitis

Rheumatoid arthritis	**Periodontitis**
1. Self-limited RA The disease commences but it does not progress to cause significant damage. 2. Easily controlled RA The disease becomes established but it can be contained with first line medications. 3. Progressive RA The disease becomes established and it continues to progress. There is need of second line medications which themselves may be of little help in arresting the disease progression	1. Well maintained Periodontitis The disease commences but can be controlled with simple treatment such that little or no progression occurs 2. Downhill Periodontitis The disease becomes established and with the combination of simple and complex therapies it can largely be controlled. There may still be some ongoing destruction over the time 3. Extreme downhill periodontitis The disease becomes established and despite the simple and complex therapies it continues to progress and cause further tissue damage and tooth loss

Based on epidemiological studies of individuals originally presenting for RA, more than 75% of cases are self-limiting. Around 27% of the individuals originally diagnosed with RA may belong to the easily controlled category although this group has been identified in some populations as high as 85%.[3]

2.2. Periodontitis

As for RA, longitudinal studies on the natural history of untreated periodontal disease in humans have indicated the presence of three distinct subpopulations experiencing rapid progression (10–15%),

moderate progression (80%) and little or no progression of periodontal disease (5–10%).[4] These distributions seem to be independent of access to dental care or relative proportions of plaque deposits.

From etiological and pathogenesis viewpoints, the similar prevalence of severe forms of periodontitis in untreated and treated populations suggests that the amount of plaque does not determine the severity of the disease. Rather, the host response to the varying degrees of plaque challenge may determine the end point of periodontal disease. Increased risk of progressive periodontal disease has been associated with a multitude of factors including microbial parameters, smoking, familial or genetic background, diabetes mellitus, HLA-DR complex and IL-1b polymorphism.[5]

Table 2: Literature review

Years	Authors	Variables	Result
2023	Francessco Ichingolo, Angelo Michele Ichingolo et all	Non-surgical periodontal treatment, Disease modifying rheumatic drugs, periodontitis	The study discussed the effects of (NSPT) non-surgical periodontal treatment on rheumatoid arthritis and touched on the significance of (DMARDs)disease modifying anti- rheumatic drugs on periodontitis This study concluded that NSPT seems to have a positive effect on reduction of clinical disease activity indices more than of the reduction on ESR and CRP. Secondly it demonstrates that ex juvantibus that periodontal disease and RA influence each other via cytokine production and CMI. They concluded that DMARDs exert positive effects on clinical and immunological parameter of periodontium which led towards bidirectional pathogenic link between RA and Periodontitis.[6]
2022	Zhain Mustufvi Joshua Twigg	Periodontal treatment, ACPA level, DAS 28	Study evaluated the relation of people diagnosed with periodontal problems along with rheumatoid arthritis and further conducted a screening to analyse the effectiveness of periodontal therapy on RA. They included a total of 21 studies out of which 11 were non randomized experimental design trial and 10 were randomized control trials. In this DAS -28 (disease activity score) was the major outcome for most of these studies. In the given studies a total of 9/17 studies reported a significant intra group DAS score change. Out of the 21 studies 3 demonstrated a significant intra group improvement in ACPA level following NSPT. They concluded that there was evidence which suggested that periodontal treatment improved RA disease activity on short duration base.[7]
2022	Anna Krutyholowa et al	Periodontal pathogens, Molecular mechanism linking PD and RA, Antigens and Autoantibodies, Citrullination, HSP, Immune cells	They summarized that epidemiological studies establish corelation between PD and RA on multiple levels. Since corelation does not imply causation, the mechanism connecting these two however remain unclear. They share certain common features which may suggest similar underlying mechanism such as risk factors including HLA-DRB1-04 as the genetic factor, infection, smoking with cytomegalovirus and EBV. The study analysed the main virulence factors of the microorganism, their mechanism of action and effects on host immune response which led to providing cross talks between bacterial pathogens and to indicate the potential overlap between the pathogenic mechanism which may cause synergistic effects.[8]

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Table 2 continued

2022	Sivasankari Thilagar, Ramakrishnan Theyagarajan et al.	ACPA proteins; Chronic periodontitis; C Reactive proteins; Nonsurgical debridement; Rheumatoid arthritis; Rheumatoid facto	The aim of the study was to assess the extent of the effect of periodontal therapy on rheumatoid factor. They studied Anti citrullinated protein antibody reactive protein level, disease activity score 28 in patients with chronic periodontitis and rheumatoid arthritis. They analysed a sample which consisted of 28 patients with RA and CP. Study was designed to be randomized controlled double-blind study. According to the study the samples were randomly categorized based on either the control group (n -15) or the treatment group(n- 13). Chronic periodontitis status, biochemical status, clinical rheumatologist status was assessed at baseline along with the follow up dated at 8 -12. They further concluded that the treatment group exhibited significant reduction in probing pocket depth (P <.001), bleeding on probing (P < .005), C-reactive protein (P < .001), plaque index (P < b.001), improvement in the Disease Activity Score-28 and gain in the clinical attachment loss which was documented at reassessment after the Nonsurgical periodontal treatment in contrast to the control group. It summarized that the reduction of inflammation in the periodontium by the nonsurgical periodontal therapy led to no reduction of anti-citrullinated protein antibody along with the rheumatoid factor level. It however showed an improvement in the periodontal condition in addition to that remarkable changes were seen in the clinical Disease Activity Score and C reactive protein levels of those individuals with RA.[9]
2021	Hiroko Hashimoto; Shimpei Hashimoto; Yoshihiro Shimazaki	Periodontal inflammation ; Epidemiology; Rheumatoid Arthritis; Biological therapy	This study investigated the relationship between periodontal disease and functional impairment in patients with rheumatoid arthritis (RA) as a result of disease activity. Ninety-three patients with RA were included. As an indication of RA disease activity, serum levels of matrix metalloproteinase 3 were used. Probing depth (PD) and clinical attachment level (CAL) were used as indicators of periodontal status. They examined the association of RA severity and MMP-3 levels with periodontal status using a generalised linear model (GLM). They concluded that Functional impairment due to RA may affect PD, and high serum levels of MMP-3 may affect CAL.[10]
2021	Apoorva B. Badiger and Triveni M. Gowda	Rheumatoid Arthritis; Periodontal Pathogens; periodontitis	They observed that both Ra and periodontitis have a curiously parallel pathology. Those individuals affected with RA are more probable to experience amplified periodontal problems as compared to non-RA individuals. The emergent therapies have mainly focused on inhibition of destructive proteases in addition to the pro inflammatory cytokinin. They concluded that close attention to the oral health of these individuals would enhance their quality of life and provide them with important insight for treatment and prevention.[11]

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Table 2 continued

2021	Yoon Young Choi et al	RA [diagnostic code M05, M06, M08]; PD; Age; Gender; Household income; Smoking habits; Alcohol Consumption; BMI, systolic blood pressure; fasting glucose level; Total cholesterol	did a study to evaluate the association of Periodontitis with the development of Rheumatoid arthritis. Population- based cohort and longitudinal matched- cohort design was employed in the study. Participants were divided into 2 groups (Periodontitis and Non Periodontitis (control) between the years 2002-2006 and were monitored from 2007-2018. They found that periodontitis group were more prone to develop RA than the controls and multivariate analysis revealed higher risk of RA in periodontitis group. In conclusion it was noted that periodontitis group has higher risk of developing RA. [12]
2020	Stefen Renvert et al	RA; PD; Genetic predisposition; infection; gender; Other comorbidities; Age	This study focused on analysing the relation between rheumatoid arthritis and periodontitis. A total of 249 dentate individuals were analysed out of which one half suffered from RA and the other didn't. Both of the groups were subjected to panoramic radiograph and the patients affected by RA were evaluated and their medical histories were reviewed by rheumatologist. None of the participants in the control group had RA symptoms and their records were also negative. They concluded that RA was associated with the diagnosis of periodontitis. [13]
2020	Namrata S Jajoo	PPAD, ACPAs, Bacterial infection, Chronic inflammation, Vascular alteration, Role of genetics, Periodontal pathogens[7]	They summarized that in the last fifty years intensive studies have been performed based on potential function of inflammation as a causative factor of disease growth in RA. However there has been no proof conclusive of clear association between virus; bacteria and RA. They concluded that P. gingivalis PPAD enzyme may result in production of the citrullinated bacterial peptide and the citrullinate autoantibodies specific to human RA which has been boldly supported by human and animal test. [14]
2019	Rafael Scaf de Molon; Carlos Rossa Jr et al	RA; PD; immune pathological process; current treatment ; alveolar bone loss; bone resorption	Study focused on assessing people affected by both RA and PD. It mainly focused on translational research and on recent clinical studies that provided an overview of relationship between PD and RA and similarities in immune pathological aspect that could link the two diseases. They observed that recognization of association between RA and PD and the possible biological mechanism involved during the pathogenesis of the condition caused by them played an important role in the management of patients which required treatment for both the diseases Secondly, they concluded that the protocol care for RA affected patients should be altered to include periodontal examination and its resolution should be achieved by means of NSPT. Thirdly it was observed that in PD patients who are also affected by RA would show an improvement in their periodontal status with the help of medication such as DMARDS and NSAIDS given its immune modulatory effect for both diseases. [15]

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Table 2 continued

2019	Kaja Erikson et al	RA; PD; Anti citrullinated proteins; Autoantibodies; Smoking; Medications; porphyromon as gingivalis	They concluded that their findings demonstrated that patients with ACPA positive RA have found out to have more severe form of periodontitis irrespective of DMARD therapy or presence of the subgingival. gingivalis. In addition to that this study showed different subgingival microbial profile in RA patients with periodontitis versus no periodontitis. This study also reported that RA patients with moderate / severe periodontitis have increased serum and salivary levels of ligand APRIL. This cytokine was stated to be of a great importance for the survival and maturation of B cells and hence could potentially be involved in the link between RA and PD.[16]
2019	Railson de Oliveira et al	Periodontitis; predisposition; RA; Bacterial infection; pro Inflammatory profile; other comorbidities	They conducted a metaanalysis to find the corelation between periodontitis as a risk factor for Rheumatoid Arthritis. Several electronic databases were analysed and 9 relevant articles were screened for the possible association. They found that majority of the articles suggested possible association and similarity between the inflammatory profiles of the two diseases. They concluded that the pro-inflammatory nature of both diseases, bacterial infection, and genetics may make periodontitis a risk factor for RA.[17]
2016	Nicholas R. Fuggle et al	RA; PD; Age; Smokers and Nonsmokers; Gender; Years of RA; DAS28	This study focused on analysing the relation between rheumatoid arthritis and periodontitis. Twenty- one publications, comprising a total of 1378 participants, compared RA to osteoarthritis (OA), and 17 studies (involving 153,492 participants) compared RA to healthy controls. In comparison to healthy controls, individuals with RA had a significantly higher risk of developing periodontitis. They also had a significantly higher mean probing depth risk of bleeding on probing (BOP) and an absolute value of clinical attachment loss. The prevalence of periodontitis was not significantly changed between RA and OA; however, OA patients were at a higher risk of BOP. They concluded by stating a strong correlation exists between RA and OA.[18]
2015	Kobayashi T, Yoshie H	Periodontitis; RA; Cytokine genotypes; Coding proteins; Tumor necrosis factor alpha; interleukin 6; Targeted therapy	This study focused on analysing the relation between rheumatoid arthritis and periodontitis. They concluded that periodontitis and rheumatoid arthritis share a lot of similarities and pathologic features, which may be based on the similarity of the host response. Constitutive over production of pro- inflammatory cytokine, TNF-α, and IL-6 was implicated as one of the most common confounding factors for the two inflammatory diseases. Following treatment, results from animal and clinical intervention studies have shown an improvement in the inflammatory condition of periodontal. TNF-α and IL-6 receptor inhibitors.[19]

3. Discussion

3.1. Could periodontal pathogens be involved in the development of RA?

Many of the features that cause RA in a genetically susceptible host may also be present in periodontal pathogens. The periodontal pathogens co-exist with other groups of bacteria in a biofilm that may be able to cause chronic continuous infection. The suspect periodontal pathogens are consistently found in most people with various types of periodontal diseases. In periodontitis, the biofilm serves as an abundant source of LPS, meeting the fourth requirement for chronic LPS exposure easily. The local production of IgE and IgM has been shown to be present in periodontal disease.[20] Although the cross-reactivity of LPS from periodontal biofilms to the cartilage has not been demonstrated to cause an anticoagulant response, it is theoretically possible.

However, all these ideas are speculative unless the cause can be clearly identified. Despite extensive research and the best efforts of many researchers, no infectious agent has been identified as causing RA. Electron microscopy, molecular biology or molecular analysis of tissue infiltrating T cells do not support the idea that an antigen is causing synovial inflammations. It is also possible that there is not a single cause of RA, and that multiple mechanisms may cause the initial tissue damage and trigger synovial inflammatory responses

3.2. Are bacteria a common etiologic link between periodontitis and rheumatoid arthritis?

While there are many similarities between the microorganisms that can cause RA in a genetically sensitive host and the identified periodontal pathogens, RA is still not mostly recognized as a disease caused solely by bacterial challenge. Technological and conceptual advances have made it possible to identify bacteria, or groups of bacteria, that are associated with certain periodontal conditions. A careful examination of the factors that drive the virulence of the periodontal pathogens may indicate that such a response may be possible. Therefore, it is possible for ongoing periodontitis to cause RA in genetically sensitive individuals.

3.3. Immunogenetics of RA

While the exact cause of RA has yet to be fully elucidated, the realization that RA is autoimmune and not primarily a tissue degeneration disease has caught geneticists' and immunologists' attention.

Based on family and twin studies, RA has numerous characteristics that are typically associated with a complicated genetic disease, including genetic variation, non-inclusive penetrance, and multi-gene involvement. The application of DNA sequencing and molecular-based typing to detect HLA-DRB1 alleles exhibited that the definite disease-conferring portion of the D region is confined to a short

sequence encompassing amino-acid positions 67–74 of the third hypervariable region of the HLA- DRB1. The disease-associated alleles include the HLA-DRB1 0401, 0404 and 0408 in the white population, HLA-DRB1 0405 in the Asian population and HLA-DRB1 1402 in the Greek population.21

3.4. Immunogenetics in periodontitis

Concerning RA, immunogenetics has been the subject of extensive interest in periodontics. More prominent than half of the change found in grown-up periodontitis might be made sense of by hereditary variables.[20] In an irresistible sickness, for example, periodontal illness, the relationship between the HLA antigens and different types of the illness has been of interest with a few examinations detailing the occurrence of different class I and II HLA antigens in patients with beginning stage periodontal sickness. Specifically, the HLA antigens A9, A28, BW15 and DR4 have been viewed as related with beginning stage types of periodontitis.[21] Of interest is the perception that the HLA-A9 and HLABW15 antigens have been related with the summed up, however not restricted, types of adolescent periodontitis, inferring contrasting hereditary variables might be answerable for these two circumstances. Besides, extraordinary intronic quality varieties have been noted in the quality for HLA-DQb in patients (and some other typical relatives) appearing with beginning stage periodontitis. In any case, different examinations have demonstrated that there are no HLA relationship with signs of different kinds of periodontal illness.[22]

3.5. Immunogenetic features in common

In humans, most of the genes responsible for the regulation of monoclonal antibody responses are mapped to chromosome 5's HLA (human leukocyte-derived antigen) complex in the TNF (non-fungal factor-dependent) (TNF-b) gene family. Both Rheumatoid Arthritis (RA) and Progressive Periodontal Disease (PMS) are associated with the HLA complex. This indicates a genetic basis of the observed monocytes trait, which is associated with RA, PMS, and other systemic disorders. Part of the inter-individual variation in RA and PMS severity is due to the intrinsic differences in monocyte/ T cell response traits in both diseases. An antigen challenge (for example, LPS) on the monoclonal/ lymphocytes axis would lead to secretion of catabolic cytokines (including PGE2 and IL- 1), as well as inflammatory mediators (IL-1, IL-2, and IL-4).

Table 3: Current and emerging therapies

Mode of treatment	Effect
NSAIDs	1. Currently, NSAIDs (Non-Steroidal Anti-Inflammatory Drugs) are the most common "first-line" treatment for RA. 2. Aspirin, naproxen/diclofenac/ ibuprofen, and other NSAIDs work by inhibiting the COX synthesis, which produces both analgesics and antipyretics. 3. These medications are effective in relieving pain in RA, but do not significantly change its course.[23] NSAIDs have been used to treat periodontal disease for the past 20 years, and the results have been promising. 4. However, the wide clinical use of NSAIDs to treat periodontitis is not universal. 5. One issue with their use for periodontal disease is the "rebound" to baseline after stopping the medication.[24]
COX-2 inhibitors	1. With the disclosure of two COX catalysts liable for PGE2 creation, assigned COX-1 (constitutively communicated) and COX-2 (inducible), an assortment of COX-2 inhibitors has been read up for their capability to stop or dial back bone resorption. 2. Tenidap, one of the first COX-2 inhibitors developed, has been shown to prevent the production of IL-1, IL-6, and TNF-a as well as cyclooxygenase and PGE2. 3. Until now, COX-2 inhibitors have not been entirely read up for their capability to change bone resorption in periodontitis.
DMARDs	1. As opposed to the NSAIDS, which do not fundamentally change the direction of RA, a more up to date group of prescriptions assigned sickness altering hostile to rheumatic medications (DMARDs) has been created. 2. A medication must be able to alter the course of rheumatoid arthritis for at least one year to be considered a DMARD, as evidenced by sustained improvement in function, decreased synovitis, and prevention of further joint damage. 3. Instances of these meds incorporate parenteral gold salts, methotrexate, sulfasalazine, hydroxychloroquine (antimalarial drug), penicillamine, azathioprine, and leflunomide. 4. A significant disadvantage in the utilization of DMARDs is their impressive poisonousness. The utilization of DMARDs for the administration of periodontitis has been confined largely because of the harmfulness issues. 5. Gold salts, on the other hand, have been shown to reduce periodontal destruction in an animal model.[25] No human studies have yet been conducted.
MMPs inhibitor	1. Another emerging area of potential for host modulation in periodontitis and rheumatoid arthritis is control of the MMPs that are important mediators of connective tissue breakdown in both hard and soft tissues. 2. The role of MMP inhibitors in managing RA has been less well studied but promising results are emerging. 3. In this regard, tetracyclines and various chemical analogues have been found to inhibit MMP activity by a mechanism that is independent of their antimicrobial property.[26] 4. A number of clinical trials using low- dose tetracycline to modify periodontitis have been carried out, with the most recent data indicating that low-dose doxycycline is safe and significantly effective.[27] 5. The role of MMP inhibitors in managing RA has been less well studied but promising results are emerging. In particular, a recent study has demonstrated that low-dose and antimicrobial (higher) dose doxycycline, when used adjunctively with methotrexate, produces enhanced improvements in global scores of RA severity in humans than methotrexate combined with placebo.
IL-1 and TNF antagonists	1. Control of cytokines and their receptors is also emerging as a field of considerable promise. 2. For example, blocking the IL-1 receptor and using gene therapy to deliver IL-1 receptor antagonist are two strategies under investigation to modulate the effect of elevated IL-1 in inflamed tissues. 3. Similarly, other studies have shown that blocking the activity of another important inflammatory cytokine, TNF- a, has therapeutic efficacy in RA patients. 75-78 The roles of IL-1 and TNF antagonists in a primate model of periodontitis have demonstrated a reduction in the inflammatory infiltrate near bone as well as reduction in the formation of osteoclasts and reduced bone loss.[28]
Periodontal non-surgical treatment	1. It is an vital therapeutic device in periodontal therapy, and contains plaque control, supragingival scaling, root planning, subgingival scraping and improving and maintaining the health ofperiodontal tissues.

4. Conclusion

The relationship between rheumatoid arthritis (RA) and periodontitis is complex and bidirectional. Both conditions involve inflammation and immune dysregulation, and emerging evidence suggests they may influence each other's progression.

Rheumatoid arthritis is an autoimmune disease characterized by chronic inflammation of the joints, leading to pain, stiffness, and joint damage. Periodontitis is a chronic inflammatory condition affecting the gums and supporting structures of the teeth, resulting in gum recession, bone loss, and ultimately tooth loss.

Several studies have shown a higher prevalence of periodontitis in individuals with RA compared to the general population. The inflammatory processes involved in periodontitis may exacerbate systemic inflammation in RA, potentially worsening joint symptoms.

Moreover, the bacteria associated with periodontitis can trigger an immune response that may contribute to the development or progression of RA in susceptible individuals.

Conversely, RA itself may increase the risk of periodontitis. Chronic inflammation in RA can affect the immune response in the oral cavity, making individuals more susceptible to periodontal pathogens. Additionally, medications used to treat RA, such as corticosteroids and disease-modifying anti-rheumatic drugs (DMARDs), can have side effects that affect oral health, including increased susceptibility to infections and gingival hyperplasia.

The link between RA and periodontitis underscores the importance of comprehensive care for individuals with either condition. Managing inflammation through medications, lifestyle modifications, and regular dental care can help improve outcomes for both RA and periodontitis patients. Additionally, addressing one condition may have beneficial effects on the other, highlighting the potential for integrated treatment approaches.

5. Source of Funding

None.

6. Conflict of Interest

None.

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