Creaky Joints and Bleeding Gums: The Interactions between Periodontal Disease and Rheumatoid Arthritis (RA)

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CE Credits: 1 hour
Intended Audience: Dentists, Dental Hygienists, Dental Assistants, Dental Students, Dental Hygiene Students, Dental Assistant Students
Date Course Online: 01/25/2017 Last Revision Date: N/a Course Expiration Date: 01/24/2020
Online Course: www.dentalcare.com/en-us/professional-education/ce-courses/ce519

Disclaimer: Participants must always be aware of the hazards of using limited knowledge in integrating new techniques or procedures into their practice. Only sound evidence-based dentistry should be used in patient therapy.

Introduction
This continuing education course will review the current scientific evidence about the association between rheumatoid arthritis and periodontal disease and to aid in the clinical decision making to care for patients with RA in a dental setting.

Conflict of Interest Disclosure Statement
• Dr. Garner reports no conflicts of interest associated with this course.
• Dr. Geisinger has been or is currently a co-investigator and/or principal investigator on research funded entirely or in part by The Procter & Gamble Company. All funds were used for research endeavors and not for personal gain. Dr. Geisinger has not accepted any payment from dentalcare.com for participation in this continuing education course.

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Provider ID# 211886
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Overview

Rheumatoid arthritis (RA) is a common inflammatory mediated arthritis that leads to joint damage and significant societal burden related to patient discomfort and treatment costs. Several studies have indicated patients with RA have an increased prevalence of periodontal disease than those without RA or with other forms of arthritis. Current understanding of the pathogenesis of RA lacks a clear picture of the autoantibody response and what stimulates it, particularly specific serum antibodies directed to citrullinated peptides, which are associated with smoking, disease severity, and periodontal disease (Figure 1). Previously, RA was thought to influence periodontal disease progress through a decrease in dexterity that led to an increase in etiologic factors secondary to poor oral hygiene, but despite similar levels of joint dysfunction, patients with RA demonstrate approximately three times higher rates of periodontitis than patients with osteoarthritis (OA). This suggests a potential underlying biologic mechanism of interaction between these two diseases.

Figure 1. Clinical and Pathological Features of RA.
This course seeks to improve the dental care provider's understanding of the interaction between periodontal disease and RA as well as aid in the clinical decision making to care for patients with RA in a dental setting.

Learning Objectives
Upon completion of this course, the dental professional should be able to:

- Understand the current scientific literature about the association between periodontal health and rheumatoid arthritis (RA) and discuss the interactions between these two conditions with patients.
- Be an active participant in an interdisciplinary team of health care providers treating patients with RA and periodontal disease.
- Evaluate patients' risk factors and oral home care practices based upon individualized patient needs and severity of RA disease markers.
- Understand the risks and benefits regarding routine, elective, and emergent dental care in patients with RA.
- Discuss with patients the common risk factors associated with periodontitis and RA and be familiar with strategies to treat those risk factors.
- Discuss with interdisciplinary colleagues the importance of and effective methods for treatment of periodontal disease in patients with RA.

Introduction
Due to similar features in the pathobiology and prevalence of periodontal disease and rheumatoid arthritis (RA), it has been proposed that these diseases have a biologic interrelationship. It has been reported that individuals with periodontitis are up to four times more likely to have a self-reported history of RA than those without periodontal disease. In the same population of patients with RA, 62.5% had advanced forms of periodontitis. For both of these diseases, the host response determines, in large part, the tissue destruction and inflammatory response. Additionally, because of the similarity of tissues destroyed by such a response, the cells, enzymes, and inflammatory mediators that cause the damage to bone and soft tissue share a common pathway. Finally, due to this common pathway, strategies to treat and/or modulate these diseases are similar and may have effects on both conditions. Therefore, it is imperative that physicians managing RA and oral health care providers are aware of this interaction and are able to identify and manage the common pathophysiology.

Epidemiology and Etiology of RA
RA is a chronic, destructive, inflammatory disease that is characterized by the accumulation and persistence of an inflammatory infiltrate within the synovial fluid of a patient's joints and the destruction of the bony architecture of the joint. This ultimately leads to irreversible joint damage, loss of function and a significant burden on patients and society related to discomfort and treatment costs. It is estimated to affect 1.3 million adults in the United States (U.S.), approximately 0.5-1% of the population over the age of 35 years. RA affects females more frequently than males, in a 3:1 ratio, and the onset is most commonly seen in the 4th and 5th decades of life. At least three types of RA have been described in clinical studies: 1) self-limiting, 2) easily controlled, and 3) progressive. Many patients who seek care in a rheumatology clinic have a progressive form of the disease and present with a number of markers of inflammation and autoimmune disease, including rheumatoid factor, rheumatoid nodules, high erythrocyte sedimentation rate, HLA-DR4 haplotype, autoantibodies against citrullinated peptides (ACPA), and high numbers and severity of joint involvement as measured by the disease activity (DAS) score.

The exact cause of RA is unknown, with many different stimuli having demonstrated an ability to activate the immune and inflammatory response seen in RA. Current understanding suggests RA may be initiated by exogenous infective agents as well as endogenous substances, such as connective-tissue proteins or immunoglobulins, in patients with a genetic predisposition. While infectious agents have been proposed as etiologic factors for RA, a single organism has not been identified, and it may be likely numerous agents may be able to initiate an RA response in susceptible individuals. Nonetheless, several agents...
including Gram negative anaerobic bacteria like those found in periodontitis have been implemented in the etiology of RA.\textsuperscript{27-29}

**Epidemiology and Etiology of Periodontal Disease**

Periodontitis is a chronic disease of the hard and soft tissue supporting the teeth caused by bacterial plaque resulting in progressive destruction of the periodontal ligament and alveolar bone.\textsuperscript{30,31} The disease typically has a slow to moderate rate of disease progression, but periods of accelerated attachment loss may be associated with local and/or systemic factors.\textsuperscript{32-34} The disease is classified as mild (1-2 mm of clinical attachment loss), moderate (3-4 mm of clinical attachment loss), or severe (≥5 mm clinical attachment loss).\textsuperscript{35,36} The prevalence of periodontitis has been estimated to be over 47% of U.S. adults, or 64.7 million individuals.\textsuperscript{37} Of those individuals, 8.7% showed mild disease, 30.0% demonstrated moderate disease, and 8.5% had severe chronic periodontitis.\textsuperscript{37} Risk indicators for periodontitis include male gender, Hispanic ethnicity, cigarette smoking, uncontrolled diabetes mellitus, and lower socioeconomic status.\textsuperscript{38} Prevalence of periodontitis varied two-fold between the lowest and the highest levels of socioeconomic status.\textsuperscript{39}

Disease progression of periodontitis has been categorized into subpopulations demonstrating rapid progression (10-15% of disease cases), moderate progression (80% of disease cases), and mild/no progression (5-10% of disease cases).\textsuperscript{32,40,41} The similar prevalence of disease and disease progression in treated and untreated populations,\textsuperscript{42} suggests that host factors may play the larger role in disease progression after bacterial initiation.\textsuperscript{43,44}

**Common Etiologic Factors and Epidemiology between RA and Periodontal Disease**

Rates of RA in patients with a diagnosis of periodontitis are significantly higher than in the general population, 3.95% versus less than 1%.\textsuperscript{5} Similarly, in patients with RA, periodontitis is at least two-fold more prevalent than in the general population. These findings are independent of smoking history, age, and gender.\textsuperscript{49} This increased disease prevalence may indicate common risk factors and/or common pathobiology. Both RA and periodontitis cause destruction of hard and soft tissue through similar pathways in that the pro-inflammatory cytokines, and inflammatory cells that result in gingival, collagen, and bone destruction are common between both diseases.\textsuperscript{49} The patterns and mechanisms of disease progression in periodontitis and rheumatoid arthritis indicate a high level of host susceptibility and may present analogous disease states (Table 1).

**Proposed Mechanisms of Interaction between Periodontal Disease and RA**

Several models of interaction have been proposed for the relationship between RA and periodontitis. Briefly: 1) Infection with periodontal pathogens, particularly *Porphyromonas gingivalis* (*P.g.*), initiates the alterations of host proteins through citrullination, which leads to the formation of autoantibodies and cross-reactivity causing autoimmunity and RA; 2) A common inflammatory burden activates both osteoclast function and vascular damage causing a predisposition to both RA and periodontitis; 3) periodontal disease and RA, when they exist together, can cause a cyclical exacerbation of systemic inflammation and a worsening of both diseases (“two hit model”).\textsuperscript{50} (Figure 2).

**Common Microbial Interactions**

The “red complex” of periodontal pathogens, composed of *Treponema denticola* (*T.d.*), *Tannerella forsythia* (*T.f.*), and *Porphyromonas gingivalis* (*P.g.*), are present in the majority of progressive gingival lesions of chronic periodontitis and have been identified as likely causative agents for periodontal tissue destruction.\textsuperscript{51} These organisms have a wide variety of virulence factors. *P.g.* expresses lipopolysaccharide (LPS), fimbrae, and hemagglutins, which allow the bacteria to invade periodontal pocket epithelium and initiate a host inflammatory response and also has a series of cysteine proteases, or gingipains, which render *P.g.* resistant to complement and create a feedback cycle in which the host’s attempts to clear the bacterial infection result in continued tissue damage, including bone resorption.\textsuperscript{52-56} This activation of bone resorption
Table 1. Common Pathologic Factors in RA and periodontitis.

<table>
<thead>
<tr>
<th>Pathogenesis of Rheumatoid Arthritis</th>
<th>Pathogenesis of Periodontitis</th>
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<tr>
<td>Chronic inflammatory disease</td>
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<td>Bacteria/peptide as adjuvant antigen in autoantibody production</td>
<td>Bacteria as etiological agent</td>
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<td>Role of macrophage and dendritic cells</td>
<td>Role of macrophage and dendritic cells</td>
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<td>IL-1, TNF-alpha, PG-E2</td>
<td>IL-1, TNF-alpha, PG-E2</td>
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<td>Immunoregulatory imbalance</td>
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<td>Th1=Th2</td>
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<td>Role of Nitric Oxide</td>
<td>Role of Nitric Oxide</td>
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<td>Genetic and environmental influences</td>
<td>Genetic and environmental influences</td>
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<td>Persistence of antigen/peptide</td>
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Figure 2. Concept of how periodontitis and rheumatoid arthritis might interact. A. Periodontitis Precedes Rheumatoid Arthritis. B. Common Inflammatory Pathways: Osteoclast Activation and Vascular Damage. C. Two Hit Model of Mutual Exacerbation.
through activation RANKL in osteoblasts may lead to increased bone resorption at distant sites as well.57

The majority of RA cases are triggered by an autoimmune response to citrullinated proteins, which occur when proteins are enzymatically modified to replace the amino acid arginine with citrulline. This may occur for many required cell functions, including terminal differentiation of the epidermis and regulation of gene expression via chromatin remodeling.58,59 In genetically susceptible individuals, however, the generation of autoantibodies against ACPAs in synovial fluid can lead to a subsequent development of RA.60-62

P.g. is the only bacterium identified that has the capability to develop ACPAs antibodies;5,66 it has also been linked to ACPAs and their antibody formation in patients with RA and their relatives.62-66 The presence of antibodies to P.g. is associated with the development of RA (OR=2.96; 95% CI: 2.00-4.37); the strength of this association is greater than known risk factors such as smoking (OR=1.37; 95% CI 1.07-1.74).67 Similarly, expression of ACPAs were higher in patients with subgingival P.g. and with anti-P.g. antibodies than in those without evidence of P.g. infection.68 Anti-P.g. titers have also been associated with development of RA symptoms and greater disease activity in early RA patients.69

Common Inflammatory Burden
Both RA and periodontal disease are associated with an increased inflammatory burden.70,71 RA subjects demonstrate higher levels of bleeding on probing (BOP) and higher pro-inflammatory cytokines, such as IL-1β and TNF-α levels in GCF than healthy controls.72 As the intrasulcular epithelial surface area differs between tooth type and location, periodontal inflamed surface area, PISA, aimed to measure the inflamed epithelial tissues.72 In an observational study, mean PISA scores were 291.9 mm² ± 328.7 in a cohort of patients with RA and 94% of RA patients in this study had moderate to severe periodontitis, as opposed to having mild or no periodontitis, which is higher than PISA findings in systemically healthy patients demonstrated in other studies.73 Similarly, in a group of patients with RA, PISA has been correlated with DAS on a linear regression model.74 While the latter finding was not statistically significant, it is consistent with other reports that correlate DAS with BOP and alveolar bone loss. These data in aggregate may indicate a positive, albeit weak, correlation was shown between periodontal disease and RA and the inflammation associated with both.

RA Therapies and the Effect on Oral Health and Periodontal Disease
Both disease modifying anti-rheumetic drugs (DMARDs) and anti-tumor necrosis factor alpha (TNF-α) have anti-inflammatory effects and are used to treat RA, although their effects on local periodontal inflammatory mediators have demonstrated inconsistent results.75 A recent study has demonstrated patients with chronic periodontitis demonstrated significantly less improvement in rheumatoid clinical parameters including DAS, erythrocyte sedimentation rates (ESR), and C-reactive proteint (CRP) levels than periodontally healthy patients with RA when treated with anti-TNF-α blockers.75 It is postulated the increased systemic inflammation due to periodontitis may dampen the effects of this therapy.76 Patients with a history of periodontal disease who were initially treated with a TNF-α blocker were also more likely to discontinue the drug than those without periodontal disease.77

RA symptoms may also be treated with steroid and non-steroidal anti-inflammatory drugs (NSAIDs). NSAID anti-inflammatory medications have shown an adjunctive benefit in reducing overall signs of periodontal inflammation in patients with periodontitis.78-81 Of particular interest, smokers and other subjects with increased inflammatory burden demonstrated improved treatment outcomes.82 Chronic corticosteroid stimulation, conversely, has been linked to an increased susceptibility to periodontitis.83,84 In patients with RA who are taking these medications for treatment of their arthritis symptoms, consultation with their rheumatologist or treating physician is critical to achieve optimal and safe results from therapy.

The use of many anti-rheumatic medications also poses a risk to patients undergoing treatment, as some patients may experience decreased immune response and higher
infection rates. Furthermore, the effectiveness of some DMARD and anti-rheumatic drugs may change based upon RA disease activity and patient age. As these may affect the ability of the patients to undergo invasive periodontal therapy, consultation with each patient's rheumatologist or treating physician and careful assessment of the risks and benefits to treatment should be performed.

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### Advanced Diagnostic and Screening Techniques for Patients with RA

Due to the high inflammatory burden associated with both RA and chronic periodontitis and the potential bidirectional influence this inflammation may have on the treatment of these conditions, it is critical to thoroughly assess and quantify the periodontal inflammatory burden to establish adequate baseline and therapy end points. This may include calculation of PISA scores, salivary or gingival crevicular fluid (GCF) biomarker sampling, and evaluation of subgingival plaque for the presence of *P. gingivalis*. This information can help with medication selection for RA treatment as well as improved monitoring for periodontal disease progression.

While the use of PISA scores and advanced biologic sampling may not be necessary in all cases of periodontal disease, incorporation of more in depth analysis of the periodontal inflammatory burden and underlying etiologic agents present may be critical to allow for more precise evaluation of overall periodontal inflammation and to assess the resultant changes after nonsurgical and/or surgical periodontal therapy. Additionally, coordination of care for patients with RA and chronic periodontitis and screening of RA patients for periodontitis should be performed in conjunction with the patients' rheumatologist or treating physician and periodontal practitioner to allow for assessment of the risks and benefits of therapy for each individual patient based upon their overall condition.

Accurate calculation of the overall inflammatory burden may also be critical in decision making by the patient's treating physician for drug selection as TNF-α blockers may be less effective in patients with RA and an inflammatory
Clinical Decision Making for Treatment of Patients with RA in a Dental Setting

Careful evaluation, quantification, and ongoing monitoring of periodontal disease and inflammation present in a patient’s mouth and consultation with the patient’s treating physician prior to initiation of periodontal therapy could allow for ideal management of RA and periodontitis in patients with both diseases (Figure 3).

Summary

Both periodontal disease and RA are unique diseases that have been suggested to require exposure to exogenous pathogens to initiate host inflammation, but the host factors propagate the destruction of hard and soft tissues at the site of local inflammation. In periodontal disease, this occurs at the junction of the gingival interface with the tooth, and in RA this occurs in the articular tissues. Many of the same proinflammatory mediators are present during tissue destruction in both diseases including IL-1, IL-6, CRP, TNF-α, INF-γ, and RANK ligand (RANKL). PISA and DAS values both characterize inflammation; PISA measures the inflamed surface area within a periodontal pocket, and DAS measures the articular and systemic disease activity of RA. Practitioners should consider utilizing more advanced clinical assessments of both RA and chronic periodontitis to allow for a more accurate evaluation of the disease conditions and to best select appropriate end points to therapy that relate to the overall inflammatory burden of each disease. Periodontal management of patients with RA should involve consultation with their rheumatologist and/or treating physician to ensure optimal patient care and safety.

Figure 3. Clinical decision making considerations for patients with RA and periodontitis.
Course Test Preview
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1. All of the following are risk indicators for periodontitis EXCEPT ___________.
   a. male gender
   b. hispanic ethnicity
   c. lower socioeconomic status
   d. uncontrolled diabetes mellitus
   e. insomnia

2. PISA measures the articular and systemic disease activity of RA; DAS measures the inflamed surface area within a periodontal pocket.
   a. Both statements are true.
   b. The first statement is true and the second statement is false.
   c. The first statement is false and the second statement is true.
   d. Both statements are false.

3. Studies show that patients with RA have an increased prevalence of periodontal disease versus those without RA or other forms of arthritis.
   a. True
   b. False

4. Do bacteria play a role in both the etiology of RA and periodontitis?
   a. Yes, with only one specific bacterium, *Streptococcus sanguinis*.
   b. Yes, with multiple types of bacteria, including *Porphyromonas gingivalis* and its role in citrullinating proteins in RA.
   c. No, there are bacteria present but they do not play a role in the etiology of either.
   d. No, there are no bacteria present in RA or periodontitis.

5. In patients with RA, periodontitis is at least ____-fold more prevalent than in the general population.
   a. one
   b. two
   c. three
   d. four

6. RA subjects do not demonstrate higher levels of bleeding on probing (BOP).
   a. True
   b. False

7. What medication has been shown as an adjunctive benefit the reduction of overall signs of periodontal inflammation in patients with periodontitis?
   a. Chronic corticosteroids
   b. Acetaminophen
   c. NSAIDS
   d. Amoxicillin
8. Non-surgical periodontal therapy decreased all of the following common inflammatory factors in patients with RA and periodontitis, EXCEPT __________.
   a. Erythrocyte Sedimentation Rate (ESR)
   b. TNF-α
   c. IL-1β
   d. CRP

9. In one cohort study of RA patients, what percentage of those RA patients had periodontitis?
   a. 26%
   b. 58%
   c. 73%
   d. 94%

10. Chronic corticosteroid stimulation has been an effective therapy for periodontitis and has been linked to a decreased susceptibility to periodontitis.
    a. True
    b. False

11. All of the following are considered virulence factors that bacteria may possess EXCEPT __________.
    a. mitochondria
    b. lipopolysaccharide (LPS)
    c. fimbrae
    d. hemagglutins

12. P.g. can elicit the host to respond and attempt to clear the bacterial infection, which can result in __________?
    a. increased calculus formation
    b. decreased calculus formation
    c. overall bone deposition
    d. overall bone resorption

13. The primary etiology of periodontitis is __________.
    a. smoking
    b. rheumatoid arthritis
    c. bacterial plaque
    d. diabetes

14. DMARDs and anti-TNF-α have consistent therapeutic results in both RA and periodontitis.
    a. True
    b. False

15. Management and treatment of RA and periodontitis is interdisciplinary and should include a dental professional, such as the patient’s dentist or periodontist, and a medical professional, such as the patient’s rheumatologist or treating physician.
    a. True
    b. False
References


About the Authors

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Dr. Garner is a postdoctoral resident in Periodontology at the University of Alabama at Birmingham (UAB). She received her Bachelor’s of Science in Biomedical Sciences from Troy University graduating summa cum laude and completed her dental training at the University of Alabama at Birmingham School of Dentistry. Dr. Garner was the recipient of multiple awards during her training, including the Dentsply Overall Winner for original research at UAB’s Scholar’s symposium for her work on the interaction of periodontal disease and rheumatoid arthritis. She is a member of ADA, AAP, ASDA, AAWD, IADR/AADR, and AGD.

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Dr. Geisinger is an Associate Professor at the University of Alabama at Birmingham (UAB) in the Department of Periodontology where she teaches a broad range of classes and serves as the Director of the Advanced Education in Periodontology Program. She received her Bachelor’s of Science in Biology from Duke University graduating cum laude and completed her dental training at Columbia University College of Dental Medicine. She completed her Certificate in Periodontology and Master’s of Clinical Science at the University of Texas Health Science Center in San Antonio. Dr. Geisinger is a Diplomate in the American Board of Periodontology. In her role at UAB, she is involved in clinical and translational research examining the interactions between periodontal diseases and systemic health. Her research focuses on periodontal-systemic interactions, periodontal regenerative therapies, implant dentistry, and educational technology. She is a member of the ADA, AAP, SAP, ADEA, AADR/IADR, AAWD, AAUW, and the President-elect of the AAPF.

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