Periodontal Disease
Risk Factors and Treatment

by Richard Champagne DMD, FAGD
Periodontal Disease, Risk Factors and Treatment

**ABSTRACT**

Periodontal disease with clinical attachment loss affects approximately 47% of American adults over the age of 30. The development and progression of chronic periodontitis depends on a number of factors including risk and the host response. Treatment options include nonsurgical therapy which may also use adjunctive therapies, and surgical therapy. Regular re-evaluation and periodontal maintenance is essential for patients who have been treated for periodontal disease.

**LEARNING OBJECTIVES**

The overall objective of this course is to provide an overview on periodontal disease, risk actors and treatment. After completing this article, the reader will be able to:

- Describe the etiology, onset and progression of periodontal disease
- List and describe risk factors for periodontal disease
- Review periodontal treatment options

**Introduction**

The Centers for Disease Control and Prevention (CDC) conducted a survey spanning 2009-2010, and reports that approximately 47%, or 65 million American adults over the age of 30, have periodontitis. Among older adults in the United States, the prevalence of periodontitis is higher. Based on the National Health and Nutrition Examination Survey (NHANES) III data from 2009-2012, the overall prevalence of periodontitis is approximately 66% in adults ≥ 65 years-of-age, with approximately 12% having severe periodontitis. In those over 75 years-of-age, periodontitis is more prevalent than in all other age groups and its prevalence increases with age. Periodontal disease is associated with periodontal pathogens in dental plaque, with initiation and progression influenced by risk factors and the host response.
The majority of the population experiences gingivitis, an inflammatory process that does not include bone loss, the early stage of periodontal disease. Gingivitis may be acute or chronic, and require professional care. Initially, supragingival dental plaque formation begins with the adhesion of oral bacteria to the acquired pellicle, and if undisturbed dental plaque can accumulate. Supragingival and subgingival plaque matures over a period of several weeks. Subgingival dental plaque is well-developed by between three and twelve weeks of plaque maturation, and largely contains gram-negative bacteria. Current recommendations for preventive home care include twice-daily oral hygiene involving brushing, interdental cleaning and use of a toothpaste, to help prevent plaque build-up and oral disease.

Periodontitis and the Disease Process

Periodontitis is an inflammatory process, but in contrast to gingivitis it involves clinical attachment loss. There is also a lack of evidence to support the concept that unresolved gingivitis always results in progression to periodontitis. Microbial complexes were identified in 1998, based on subgingival plaque samples from 185 adults, and categorized into six specific microbial groups of bacterial species. The red complex compromised \textit{P. gingivalis}, \textit{B. forsythus} and \textit{T. denticola} was considered to be the most virulent based on the research, followed by the orange complex with 12 microorganisms including \textit{P. intermedia} and \textit{P. nigrescens}, while \textit{A. actinomycetemcomitans} was determined to belong in the blue complex.

At the current time, more than 600 species of bacteria have been identified and may be found in subgingival plaque from different patients, and up to 100 may be identifiable in one sampled site. Diseased periodontal pockets typically contain an abundance of \textit{Porphyromonas gingivalis}, \textit{Tannerella forsythia}, and \textit{Treponema denticola}, and a significant number of other anaerobes including but not limited to \textit{A. actinomycetemcomitans}, \textit{Fusobacterium nucleatum} and \textit{Campylobacter} spp. are also found in diseases sites. It is clear that some microorganisms involved in periodontal disease are more virulent and more frequently found in diseased sites than others. It is the host response that influences the inflammatory process and progression.

Host response mechanisms in periodontal disease involve many interactions, based on the inflammatory and immune responses. Initially, bacterial antigens and irritants cause the release of antibodies, neutrophils and lymphocytes. Bacterial lipopolysaccharides stimulate the immune response, resulting in increased levels neutrophils and then of cytokines produced by macrophages and neutrophils. The cytokines and chemokines then result in bone loss. Cytokines involved in the destruction of periodontal tissues include interleukin-1 and tumor necrosis factor alpha. This is followed by increased activation of matrix metalloproteinases (MMPs), in particular collagenase, which causes collagen breakdown within the periodontal matrix. Tumor necrosis factor alpha increases osteoclast activity which causes bone resorption. Other chemicals are also produced that increase the destructive inflammatory response.

Risk factors for Periodontitis

Periodontitis, associated with gram-negative microorganisms contained in subgingival dental plaque and the host response, has both modifiable and non-modifiable risk factors.

**Modifiable risk factors**

Modifiable risk factors include poor oral hygiene, tobacco smoking, stress, obesity, and the use of certain types of medications are also modifiable risk factors (Table 1). Poor oral hygiene includes those individuals who are unable or unwilling to practice proper oral hygiene and regular professional care visits. Local factors can increase plaque accumulation and make it difficult to perform proper oral hygiene, such as the presence of cervical caries, overhangs and other defective restorations, and interdental areas where food impaction occurs.

Tobacco smoking is a significant modifiable risk for periodontal disease. Based on a number of studies, the risk for
Periodontal disease is greater, periodontitis is more severe in smokers, and progression is more rapid. One study assessing NHANES III data found a four-fold risk of periodontitis in tobacco smokers compared to nonsmokers. Tobacco smoking also results in vasoconstriction within gingival tissues, resulting in less propensity for bleeding even in the presence of gingival inflammation compared to nonsmokers. Substance abuse also increases risk for periodontal disease.

Increased stress levels have been found to be associated with periodontal disease, and can promote poor oral hygiene habits. An inability or poor ability to cope with stressors leads to clinically evident responses. Increased production of cortisol, a glucocorticoid, can occur. This diminishes immunoglobulin production, negatively affecting the immune system. Poor coping of stress has also been found to be an indicator for more severe periodontal disease. It is hypothesized that this may be associated with pro-inflammatory cytokines.

Diabetes mellitus, if uncontrolled or poorly controlled, places patients at increased risk for periodontitis. Patients with diabetes and untreated periodontal disease experience difficulty controlling their blood sugar levels. An association between obesity and increased risk for periodontitis in females has been demonstrated, and high body mass indexes was associated with periodontal disease in NHANES III data.

Specific medications also affect the periodontium both from a disease and treatment perspective. Therefore, this must also be considered in assessing a patient’s risk level. If the medication is essential this is a nonmodifiable risk.

**Non-modifiable risk factors**

Periodontal disease risk factors that are nonmodifiable include gender, race and genetics (Table 2). Males are at greater risk for periodontal disease than females. Data also supports a strong genetic component for risk and progression of periodontal disease.

In addition to diabetes mellitus, autoimmune diseases and acquired immunodeficiency place patients at risk for periodontitis. It is evident from studies that patients with rheumatoid arthritis are at increased risk for periodontitis. Patients with established rheumatoid arthritis are at risk for more severe periodontal disease than those in the early stages of rheumatoid arthritis. Another factor is that methotrexate and glucocorticosteroids that are used to manage rheumatoid arthritis promote periodontal disease.

Other risk factors for periodontal disease include socioeconomic status, hematological disorders, osteoporosis and fluctuations in hormone levels. The effect of osteoporosis and osteopenia on periodontal health, induced by post-menopausal hormonal changes, may be related to the effect that both of these conditions have on bone mass density. One theory is that the resultant systemic reduction in bone mass density caused by osteoporosis may make alveolar bone more susceptible to the periodontal disease process than bone that has a greater bone mass density. Pregnancy gingivitis occurs in up to 75% of women, related to increased levels of estrogen and progesterone which amplifies the body’s response to oral bacteria. This usually reverses after delivery of the baby, not progressing to clinical attachment loss. Some studies support an association between periodontal disease and premature birth and low birth weight babies, while a number do not.

Once periodontal disease is present, periodontal treatment is aimed at eliminating bacteria, preventing its progression and gaining clinical attachment.

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<th>TABLE 1. Modifiable risk factors</th>
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<td>Poor oral hygiene</td>
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<td>Tobacco smoking</td>
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<th>TABLE 2. Nonmodifiable risk factors</th>
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<td>Socioeconomic status</td>
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<td>Rheumatoid arthritis</td>
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<td>Hematological disorders</td>
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Periodontal Therapies

Periodontal therapies can be separated into two categories, nonsurgical and surgical. Typically, behavioral changes that will improve modifiable risk factors are required in conjunction with active treatment. The goal of treatment is to halt periodontal disease progression and reduce pocket depths.

Nonsurgical periodontal therapy includes scaling and root planing followed by regular periodontal maintenance, typically at 3-month intervals. The interval between periodontal maintenance appointments is determined for patients based on severity of disease, risk factors, complexity of medical conditions with individual considerations. Scaling and root planning may be performed by hand instruments, ultrasonic scalers, lasers or combinations of these. It involves debridement for the removal of supra- and sub-gingival plaque and calculus, debris, subgingival bacteria and toxins. Calculus harbors bacteria and toxin, and provides a rough area where new plaque accumulation is fostered. Periodontal bacteria can recolonize areas and accumulate to the pre-treatment level within days or weeks. Improved oral hygiene is needed following therapy to control plaque and control bacteria, and regular periodontal maintenance is critical.

Depending on the host, response to treatment and associated factors such as smoking status or systemic disease, a decision may be made to use locally-applied or systemic agents to aid in the treatment of chronic periodontitis. Medications, both localized and systemic, may be used as an adjunct to scaling and root planing. Locally-applied therapeutic antimicrobials can be used in situations where patients respond poorly and/or may have conditions suggesting their use. Since they are applied locally, unlike systemic treatments they are used where they are needed and are designed to maintain a higher concentration at the site over a prolonged period of time that is sufficient to inhibit periodontal bacteria. Options used include minocycline applied as microspheres (Arestin, Orapharma Inc.) and doxycycline gel (Atridox, Tolmar Inc.). Determining whether to use a locally-applied antimicrobial in addition to scaling and root planning should consider the patient, host response, risk factors such as smoking, severity, and what additional benefit might be achieved.

Systemically, and as an adjunct to scaling and root planning, twice-daily 20 mg capsules of doxycycline (Periostat) are subantimicrobial and have been taken orally to inhibit collagenase activity and help protect against degradation of collagen in periodontal tissues. The changes in pocket depth vary by patient, site and initial pocket depth, and it is used for long-term control of periodontal disease in high risk patients, such as smokers. It is not a substitute for thorough home care or nonsurgical periodontal therapy.

Surgical periodontal therapy may be required under some circumstances, including when access is needed to treat areas such as furcations and deep periodontal pockets. This type of pocket reduction surgery involves making a full thickness flap to facilitate the complete removal of all deposits, and remodeling bone if indicated. Grafting procedures using an autograft, allograft, xenograft, or alloplast may also be performed to augment hard tissue, soft tissue, or both. Guided tissue regeneration can improve bony defects using bone graft material and a resorbable or non-resorbable barrier membrane to facilitate hard tissue growth. Some non-resorbable membranes have titanium reinforcement which can prove beneficial in situations that require that the graft be tented.

The case below discusses a patient where non-surgical scaling and root planing was performed along with adjunctive locally-applied antimicrobial therapy.

Case study

The patient in this study is a 48-year-old female. She is a nonsmoker with rheumatoid arthritis for which she takes methotrexate. Other than this, she has no relevant medical history and is healthy. The patient has attended our office regularly since 2013 for check-ups and has regularly received a periodontal evaluation and routine dental care, including thorough scaling and prophylaxis. She has good oral hygiene, and also uses a prescription-strength fluoride toothpaste as well as an antimicrobial essential oils mouthrinse on a daily basis that helps reduce plaque and gingivitis.

The patient presented for her check-up and periodontal evaluation on 3/22/2016. Periodontal charting revealed multiple periodontal pockets with a depth of 4 mm to 5 mm
in all four quadrants of the mouth. Prior to this visit, no pocket depth had exceeded 3 mm. No local factors, dietary changes, or home care changes were reported by the patient or identified as potential explanations for the increase in pocket depths, and her level of oral hygiene was unchanged. However, the association between rheumatoid arthritis and periodontal disease was flagged as a possible contributing factor to the change in her oral health. After reviewing the periodontal
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Charting findings, treatment options and recommendations were discussed with the patient. These included scaling and root planing, locally applied antimicrobial therapy where required, a re-evaluation at 7-10 days, and three-monthly periodontal maintenance. These recommendations were all declined by the patient.

On 10/6/16, periodontal charting was again performed. The number of 4 mm to 5 mm pockets had increased, as well as bleeding on probing (Figure 1, 2). The findings of the periodontal evaluation were again discussed with the patient, including the deterioration which had occurred since her previous evaluation.

The treatment now recommended included scaling and root planing in all four quadrants, including the upper right (teeth #2-#4), upper left (#11-#14), and the entire lower left quadrant. The use of a locally applied antimicrobial agent (Arestin, Orapharma) was also recommended at specific sites: teeth #2 distal lingual (5 mm; Figure 3), #13 distal facial (5 mm), #15 distal facial (5 mm) and #20 distal facial (5 mm). These treatment recommendations were accepted by the patient, and the treatment was completed over two visits, on 10/11/16 & 10/27/16.

At the patient’s eight-week follow up hygiene appointment on 12/6/16, significant improvement was noted and all periodontal tissues had returned to excellent health. It is important to note that all 5 mm pockets had reduced to 2-3 mm (Figure 4).

The patient indicated that she would keep three-month periodontal evaluation and maintenance visits. At each of these visits full periodontal charting will be performed so that any pocket depth increase will be caught at a very early stage of disease progression and treated conservatively.

Conclusions

Periodontal disease with clinical attachment loss continues to be a significant oral health issue. Risk factors exist that are modifiable and nonmodifiable. Among modifiable risk factors, tobacco use and poor oral hygiene are significant factors for the onset and progression of periodontal disease. Nonmodifiable risk factors include diseases such as diabetes mellitus, autoimmune disorders and hormonal changes. Once periodontal disease exists, treatment is required to halt progression and preferably obtain clinical attachment gains. Treatment options include nonsurgical periodontal therapy with or without adjunctive treatment, and in more advanced cases surgical periodontal therapy is indicated. Following periodontal therapy, regular periodontal maintenance therapy and good home care are essential to help prevent disease recurrence. The treatment recommended and provided for the individual patient is based on a full periodontal evaluation, the patient’s medical and dental history and risk factors. In determining treatment, patient needs and preferences and clinical judgement are required.

References


Webliography


Periodontal Disease, Risk Factors and Treatment

1. The Centers for Disease Control and Prevention (CDC) reports that approximately _________ of American adults over the age of 30 have periodontitis.
   a. 27%
   b. 35%
   c. 47%
   d. 55%

2. There is evidence to support the concept that unresolved gingivitis always results in progression to periodontitis.
   a. True
   b. False

3. Bacterial lipopolysaccharides stimulate the immune response, resulting in increased levels of _________ and then of _________.
   a. red blood cells; cytokines
   b. cytokines; macrophages
   c. neutrophils; cytokines
   d. bacterial byproducts; statins

4. At the current time, more than 600 species of bacteria have been identified and may be found in subgingival plaque from different patients, and up to _________ may be identifiable in one sampled site.
   a. 30
   b. 50
   c. 75
   d. 100

5. Tumor necrosis factor alpha increases _________.
   a. the bacterial load
   b. osteoclast activity
   c. destruction of collagen
   d. elastin activity

6. _________ is a modifiable risk factor.
   a. poor oral hygiene
   b. tobacco smoking
   c. stress
   d. all of the above

7. Tobacco smoking has been shown to result in a _________ risk of periodontitis.
   a. three-fold
   b. four-fold
   c. six-fold
   d. eight-fold

8. A _________ was associated with periodontal disease in NHANES III data.
   a. high body mass index
   b. high triglyceride level
   c. high cholesterol level
   d. low cortisol level

9. Data supports a strong genetic component for risk and progression of periodontal disease.
   a. True
   b. False

10. Methotrexate and glucocorticosteroids are used to manage rheumatoid arthritis, and _________.
    a. promote memory loss
    b. promote periodontal disease
    c. reduce anti-inflammatory chemicals
    d. reduce the level of periodontal bacteria

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11. Post-menopausal hormonal changes induce ____________.
   a. osteoporosis and osteopenia
   b. osteoblast activity
   c. lower bone mass indices
   d. higher levels of anti-inflammatory agents

12. Pregnancy gingivitis occurs in up to ____________ of women.
   a. 55%
   b. 65%
   c. 75%
   d. 85%

13. Periodontal treatment is aimed at ____________.
   a. eliminating bacteria
   b. preventing periodontal disease progression
   c. gaining clinical attachment
   d. all of the above

14. Typically, behavioral changes that will improve ____________ risk factors are required.
   a. genetic
   b. indeterminate
   c. modifiable
   d. most significant

15. Scaling and root planing involves debridement for the removal of ____________.
   a. supra-and sub-gingival plaque and calculus
   b. debris
   c. subgingival bacteria and toxins
   d. all of the above

16. A decision may be made to use locally-applied or systemic agents to aid in the treatment of chronic periodontitis and help with periodontal improvements, depending on ____________.
   a. the patient’s ability to perform thorough oral hygiene post-treatment
   b. mitigating circumstances
   c. the host response and associated factors such as smoking status
   d. the patient’s age

17. Locally-applied therapeutic antimicrobials are applied locally and are designed to ____________.
   a. maintain a low concentration at the site over a prolonged period of time
   b. maintain a high concentration at the site over a prolonged period of time
   c. prevent overdosing of antimicrobials
   d. provide for a tapering off effect

18. Determining whether to use a locally-applied antimicrobial in addition to scaling and root planing need only consider what additional benefit might be achieved.
   a. True
   b. False

19. Some non-resorbable membranes have ____________ reinforcement which can prove beneficial in situations that require that the graft be tented.
   a. zirconium
   b. titanium
   c. lithium
   d. gut

20. Following periodontal therapy, ____________ are essential to help prevent disease recurrence.
   a. regular periodontal maintenance therapy and good home care
   b. antimicrobials
   c. mouthrinses
   d. all of the above
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- Describe the etiology, onset and progression of periodontal disease;
- List and describe risk factors for periodontal disease;
- Review periodontal treatment options.

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