Peri-implant Disease: Pathogenesis, Risk Factors, Diagnosis, Prevention and Treatment

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Continuing Education Units: 1 hour


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Participants in this course will be introduced to evidence-based information related to (1) the pathogenesis, (2) risk factors, (3) diagnosis, and (4) prevention and treatment of peri-implant mucositis and peri-implantitis.

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• Dr. Palomo reports no conflicts of interest associated with this work.
• Dr. Terézhalmy has done consulting work for Procter & Gamble and is a member of the dentalcare.com Advisory Board.

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Overview

Participants in this course will be introduced to evidence-based information related to (1) the pathogenesis, (2) risk factors, (3) diagnosis, and (4) prevention and treatment of peri-implant mucositis and peri-implantitis.

Learning Objectives

Upon completion of this course, the dental professional should be able to:
• Discuss the pathogenesis and risk factors associated with peri-implant disease.
• Diagnose peri-implant mucositis and peri-implantitis.
• Develop and initiate strategies for the prevention of peri-implant disease.
• Implement appropriate non-surgical and surgical intervention for peri-implantitis.

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Introduction

Dental implants have a very high survival rate. However, the use of survival rate as a metric for success does not address infection-induced inflammation and its consequences in surviving implants. As the link between oral health and systemic health is elucidated, and infection and inflammation are emerging as primary links, the issue is paramount. This is especially poignant in light of reports that up to 48% of dental implants show soft tissue inflammation.

Peri-implant disease is an infection-induced inflammatory process associated with dental implants. Since the condition can affect both soft and hard tissues, it can be classified into two categories: peri-implant mucositis and peri-implantitis. Peri-implant mucositis is soft tissue inflammation surrounding dental implants without evidence of bone loss. Peri-implantitis reflects progression of peri-implant mucositis and has both soft- and hard-tissue components.

Pathogenesis

Although the term mucositis is used to describe soft tissue inflammation, there is disagreement in the literature whether histologically the soft tissue around a dental implant more closely resembles mucosa or gingiva. Nevertheless, the obvious pathogenic comparison of peri-implant mucositis is to gingivitis, where only the surrounding soft tissue shows inflammation and the alveolar crestal bone is intact. Predictably, like gingivitis, peri-implant mucositis is reversible.

Peri-implantitis mirrors the pathogenesis of periodontitis. Exposed titanium surfaces accumulate glycoproteins forming a salivary pellicle and as bacteria move onto a surface, the dynamic process of biofilm formation begins. Implant-associated biofilm resembles that of chronic periodontitis, i.e., mixed, non-specific microbes, dominated by gram-negative anaerobes. A notable difference between the two conditions is the association of S. aureus with peri-implantitis.

In peri-implantitis, as in periodontitis, the biofilm triggers an inflammatory response. Blood vessels adjacent to the gingivae/mucosal tissue enlarge and become permeable, allowing the migration of neutrophils (PMNs) into the pocket space around the implant. As inflammation progresses, collagen around the blood vessels is lost and lymphocytes, which subsequently transform into plasma cells and macrophages accumulate in the area.
Fibroblast- and PMN-derived collagenases catalyze collagen loss apical to the pocket epithelium. The underlying connective tissue exhibits increasing lymphocytic infiltrates. Pocket formation is enhanced in the peri-implant space characterized by “pot-hole”-like defects (Figure 1), creating an environment that favors microbial proliferation. The products of these pathogens further challenge host immune defenses and degenerative changes progress apically into the underlying connective tissue.

At this point, the analogy between periodontitis and peri-implantitis briefly diverges. Inflammatory activity around implants is more pronounced than that observed around natural teeth and the tissues are more susceptible to the spread of plaque-associated infection into alveolar bone.14,15 When cases of peri-implantitis were systematically compared to cases of periodontitis, the results revealed that tissue destruction is more severe in association with peri-implantitis.16,17

One explanation for the apparent greater severity and increased rate of progression of tissue destruction is the structural differences between periodontal and peri-implant tissues. Unlike natural teeth, dental implants do not have cementum or Sharpey’s fibers, they are not bounded by periodontal ligament, and, consequently, there is direct contact between bone and implant surface. It is axiomatic that infection can progress without impediments from soft to hard tissue.

Ultimately, as the inflammatory process reaches the crest of alveolar bone, both in peri-implantitis and periodontitis, osteoclastic bone resorption begins (Figures 2 & 3). The inflammatory cells release cytokines such as interleukin-1 (IL-1), tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6). Bone destruction occurs though osteoclastic action which is triggered by cytokines and other inflammatory mediators, including IL-1β and PGE2.
Risk Factors
While dental implants have a very high survival rate, a number of factors (e.g., plaque control, previous periodontal disease, restorative issues, concomitant systemic diseases, and smoking) have been identified as prerequisites to the initiation and progression of peri-implant disease.

Plaque
The most notable risk factor for peri-implant disease is poor plaque control. This may reflect a patient’s inability or unwillingness to maintain optimal oral hygiene. Other impediments may include prosthesis design, adjacent restoration contour and margins, and/or loose or broken restorative components, which interfere with oral hygiene. Some of these problems may be avoided by designing removable superstructures, such as screw retained crowns.

It has also been shown that maxillary soft tissues adjacent to implants are at increased risk for plaque-induced inflammation when compared to the gingivae of natural dentition. Biofilm associated with peri-implant disease is also more complex than that with periodontitis. Common periopathogenic bacteria show low prevalence, and several bacteria, such as S. aureus, Fusobacterium, and Streptococcus species, have been identified as candidate pathogens in peri-implantitis.

Previous Periodontal Disease
Prospective studies have shown that patients with history of generalized aggressive periodontitis are more susceptible to peri-implantitis. Subgingival tissues around implants show higher levels of periopathogenic and superinfecting bacteria compared to sites from non-periodontal disease patients. Other studies have found that while implants are a viable treatment option in sockets affected by chronic periapical pathoses, the risk of implant failure is increased when placed adjacent to teeth with periapical radiolucencies.

Occlusal Overload
Finite element studies have shown that occlusal load is concentrated at the implant-marginal bone interface. Consequently, occlusal loading is an important aspect of prosthetic design, since dental implants do not have a periodontal ligament to adjust to varying loads and they are less resistant to load variations than natural teeth. A systematic review of the literature concluded that in the presence of poor oral hygiene, occlusal overload leads to peri-implant bone loss.

Prosthesis-related Factors
Although there are no published randomized clinical trials which directly prove crown design is linked to peri-implantitis, it has long been established that inadequate subgingival margins of crowns change the microflora and lead to inflammation around natural teeth. It is intuitive that the same principles should apply when considering crown design on dental implants to minimize the likelihood of peri-implant disease (Figure 4A, 4B, and 4C).

Cone Beam CT prior to implant placement facilitates optimal placement of the soft tissue component of the fixture, i.e., the coronal most portion of the dental implant with a 1.8 mm polished collar, so that the implant shoulder and crown margin are located close to the mucosal surface. Non-submerged implant fixtures with passive fits to the other components, should minimize irritation to adjacent soft tissue.

Residual cement associated with temporary or permanent crown placement on a dental implant may irritate the surrounding soft tissues, contribute to poor plaque control directly or by creating a rough surface, and promote bacterial plaque formation. Accumulation of biofilm, in turn, triggers soft tissue inflammation, which if unchecked progresses to peri-implantitis.

Figure 4A. Poorly contoured temporary restoration with plaque retentive surfaces covered with biofilm.
Diabetes Mellitus
Just as periodontitis is more common in persons with diabetes, poor glycemic control is also associated with peri-implant disease. Although the role of distinct phlogistic (inflammatory) mediators in its pathogenesis is not fully elucidated, evidence suggests proinflammatory gene expression at peri-implantitis sites is affected by glycemic control. Indeed, the prognosis with dental implants is improved in patients with glycosylated hemoglobin levels below 7 (normal range: 4 to 5.7%).

Smoking
The incidence of peri-implantitis is increased in smokers with an odds ratio of 3.6 to 4.6. A meta-analysis across 13 studies found that smoking increased the annual rate of bone loss around dental implants by 0.164mm/year. In smokers, even apparently healthy peri-implant sites have increased levels of IL-1β, TNF-α, and prostaglandin E2 levels in peri-implant crevicular fluid when compared to non-smokers.

Genetic Factors
A genetic disorder characterized by IL-1 gene polymorphism has been suggested as a risk factor for peri-implantitis. However, based on a systematic review of 27 relevant articles, no definitive conclusion can be drawn. In contradistinction, chronic inflammatory diseases, such as rheumatoid arthritis, do appear to increase the risk of peri-implantitis. A determination of the odds ratios through meta-analytic studies and systematic reviews is currently under way.

Diagnosis
A combination of diagnostic tools is needed to evaluate the health status of dental implants. Presence or absence of a single sign or symptom is generally not sufficient to establish a diagnosis of peri-implantitis or to distinguish between peri-implant mucositis and frank peri-implantitis.

Clinical Appearance, Probing, Bleeding, and/or Suppuration
Signs of peri-implant mucositis are similar to those of gingival disease. In mucositis, soft tissue color changes from pale pink to more red, or may even appear bluish or cyanotic (Figure 5A). Edema of the gingival margin may present as rolled or thickened instead of knife-edged (Figure 5B). Interdental papilla may look blunted (Figure 5C). Spontaneous bleeding or bleeding upon probing and/or suppuration may be noted.

Probing with traditional light force (0.25N) protects adjacent soft tissues. Using a plastic probe protects the implant surface from scratches. Increasing probing depths, especially with bleeding and/or suppuration, when compared to baseline probing (when the final restoration was placed) can be helpful in early diagnosis. However, increasing probing depths and the presence of bleeding and/or suppuration do not distinguish between peri-implant mucositis and peri-implantitis.

Radiographs
Either conventional or digital periapical radiographs are useful in evaluating interproximal bone levels (Figure 6). These radiographs
Figure 5A. Note cyanotic gingiva.

Figure 5B. Note thickened rolled margin around implant.

Figure 5C. Note bulbous, red, papilla with spontaneous bleeding, distal to #8 implant.

Figure 6. Note bone loss around the coronal-most implant threads.

Radiographs taken perpendicular to the implant can show clear thread demarcation, useful for comparison.

**Mobility**

Once implant mobility is present, the implant is considered to have a hopeless prognosis and removal is the default treatment plan. Often, it is the restoration or a component of the abutment that is mobile. Such a situation may lead to future bone loss, but does not significantly affect immediate implant prognosis. However, loose restorative components create space for pellicle adhesion and trigger the start of the inflammatory cascade leading to peri-implant disease.

**Other Diagnostics**

Just as with other oral infections, microbial diagnosis of peri-implantitis may be performed using direct microscopy, Gram’s Method of staining, culture, immunoserologic identification and nucleic acid methods. In certain situations inflammatory markers and genetic diagnostics may also be used.

**Preventive Strategies**

Since one of the marked differences between peri-implantitis and periodontitis is the more rapid progression of peri-implantitis and the severity of associated tissue destruction, treatment success should be compared to baseline radiographs obtained when the final restoration was placed. Radiographic comparison with baseline values is particularly useful. Subtraction programs are also available to further define radiographic changes. In the absence of such a program, however,
(outcome, prognosis) relies heavily on prevention, and early diagnosis and treatment.

**Toothbrushes**

In general, electro-mechanical toothbrushes have been shown to be more effective in plaque removal than manual toothbrushes, especially in mandibular lingual areas. While well controlled prospective studies that demonstrate the superiority of powered brushes specifically around dental implants have not been done, it is intuitive that maintaining good plaque control around dental implants is beneficial.

**Dentifrices**

There are no controlled, prospective studies comparing the efficacy of various toothpaste formulations around dental implants. However, there is robust evidence that, dentifrices with stannous fluoride and those containing triclosan with a copolymer have statistically significant antiplaque and antigingivitis activity.

More recently, in controlled 6-month clinical trials, a stannous fluoride-sodium hexametaphosphate containing dentifrice has been shown to have superior antiplaque and antigingivitis efficacy. In addition, the stannous fluoride-sodium hexametaphosphate formulation has been shown to have antigingivitis activity in subjects previously found to be non-responsive to a triclosan-copolymer containing dentifrice.

**Mouthwashes**

There are no controlled, prospective studies comparing the efficacy of various mouthwash formulations around dental implants. However, there is robust evidence that mouthwash formulations containing chlorhexidine and essential oils have statistically significant antiplaque and antigingivitis activity. The same meta-analysis also concluded that the anti-plaque and anti-gingivitis effects of cetylpyridinium chloride (CPC) mouthwashes are formulation-dependent.

In a 6-month placebo controlled clinical trial, a 0.07% cetylpyridinium chloride mouthrinse was found to be statistically superior to placebo in reducing plaque and gingivitis. Another 6-month study showed no statistically significant difference in the antiplaque and antigingivitis effects of a 0.07% cetylpyridinium chloride mouthrinse when compared to an essential oil-containing mouthrinse.

A recent study evaluated the performance of four commercially available CPC-containing mouthrinses versus a negative control (CTR) using the Disk Retention Assay (DRA) and the Plaque Glycolysis and Regrowth Method (PGRM). The DRA assessed the percentage of CPC adsorption onto anionic cellulose discs and provided a measure of the substantivity and bioavailability of CPC mouthrinses. The PGRM test examined the effects of CPC on the metabolism and growth properties of sampled in vivo plaque following treatment.

Products tested were Crest Pro Health (CPH700 ppm); Colgate Total US (CT750 ppm); Scope Mouthwash (SCP450 ppm); and Colgate Total Puerto Rico (CT450). Comparison of DRA to PGRM showed a linear relation between CPC bioavailability and its clinical antimicrobial performance with rank ordered efficacy, i.e., CPH700>CT750>SCP450>CT450>CTR. The study concluded that the antiplaque and antigingivitis activity of CPC-containing mouthrinses is predicated on optimal CPC substantivity and bioavailability.

**Treatment Strategies**

Just as mechanically disrupting the causative biofilm from the surface of a tooth can reverse the effects of gingivitis and prevent progression to periodontitis, so too is the case for peri-implant mucositis. Therefore, the treatment of peri-implant mucositis and initial therapy for frank peri-implantitis aims to eliminate the biofilm from the surface of the dental implant.

**Non-surgical Treatment**

Conventional non-surgical therapy appears to successfully reverse peri-implant mucositis. Laser therapy alone or as an adjunct to conventional therapy has been evaluated, however, the superiority of laser treatment has not be established. Adjunctive antibiotic therapy (both locally applied and systemic) in association with mechanical removal of plaque had only limited success. Failure may be related to the frequent presence of bacteria resistant to clindamycin, amoxicillin, doxycycline, or metronidazole.
Implantoplasty, removal of the micro- and macro-roughened implant surface has also been evaluated as a means to attain absolute decontamination of the implant surface.\textsuperscript{51,62} However, procedure-related complications, e.g., heat production, deposits of implant material into the surgical field, damage to the implant surface, and weakening of the implant structure appear to negatively affect prognosis.

**Conclusion**

The challenges of diagnosing and treating peri-implant disease will become much more widespread as the popularity of dental implants continues to rise. The paucity of well controlled scientific evidence against the backdrop of increasing prevalence of peri-implant mucositis and peri-implantitis, and a lack of robust evidence-based treatment options make the prevention of peri-implant disease a priority.
Course Test Preview
To receive Continuing Education credit for this course, you must complete the online test. Please go to:

1. ______________ is defined as soft tissue inflammation surrounding dental implants without
evidence of bone loss.
   a. Peri-implantitis
   b. Peri-implant mucositis
   c. Peri-implant disease
   d. None of the above.

2. All of the following statements are correct with respect to the pathogenesis of peri-implantitis
   except which one?
   a. The obvious pathogenic comparison of peri-implantitis is to gingivitis.
   b. Peri-implantitis mirrors the pathogenesis of periodontitis.
   c. Implant-associated biofilm resembles that of chronic periodontitis.
   d. In peri-implantitis, as in periodontitis, the biofilm triggers an inflammatory response.

3. Which of the following statements is correct with respect to peri-implantitis?
   a. Inflammatory activity around implants is more pronounced than that observed around natural teeth.
   b. When cases of peri-implantitis were systematically compared to cases of periodontitis, the results
      revealed that tissue destruction is more severe in association with peri-implantitis.
   c. As the inflammatory process reaches the crest of alveolar bone, both in peri-implantitis and
      periodontitis, osteoclastic bone resorption begins.
   d. All of the above.

4. All of the following statements are correct with respect to peri-implantitis except which one?
   a. The most notable risk factor for peri-implant disease is poor plaque control.
   b. Maxillary soft tissues adjacent to implants are at reduced risk for plaque-induced inflammation when
      compared to the gingivae of natural teeth.
   d. Biofilm associated with peri-implant disease is more complex than that with periodontitis.
   e. S. aureus, Fusobacterium, and Streptococcal species have been identified as candidate pathogens
      in peri-implantitis.

5. Which of the following statements is correct with respect to peri-implantitis?
   a. Prospective studies have shown that patients with a history of chronic and/or aggressive periodontitis
      are more susceptible to peri-implantitis.
   b. Studies have shown that while implants are a viable treatment option in sockets affected by chronic
      periapical pathoses, the risk of implant failure is increased when placed adjacent to teeth with
      periapical radiolucencies.
   c. A systematic review of the literature concluded that in the presence of poor oral hygiene, occlusal
      overload leads to peri-implant bone loss.
   d. All of the above.

6. Which of the following statements is correct with respect to prosthesis-related factors and
   the likelihood of peri-implant disease?
   a. It is intuitive that crown design, i.e., gingival margins of crowns, on dental implants can affect the
      likelihood of peri-implant disease.
   b. Non-submerged implant fixtures should have passive fits to the other prosthetic components, so as
      not to impinge on adjacent soft tissue.
   c. Residual cement associated temporary or permanent crown placement on a dental implant promotes
      bacterial plaque formation and triggers inflammation.
   d. All of the above.
7. **All of the following statements are correct with respect to peri-implantitis except which one?**
   a. The prognosis with dental implants is improved in patients with diabetes mellitus when their glycosylated hemoglobin levels are below 7%.
   b. The incidence of peri-implantitis increases in smokers with an odds ratio of 3.6 to 4.6.
   c. It has been concluded that a genetic disorder characterized by IL-1 gene polymorphism is a definitive risk factor for peri-implantitis.
   d. Chronic inflammatory diseases, such as rheumatoid arthritis, appear to increase the risk of peri-implantitis.

8. **Signs of peri-implant mucositis may include __________.**
   a. soft tissue color changes from pale pink to more red
   b. bluish or cyanotic soft tissue
   c. edema of the gingival margin rolled margin around the implant
   d. All of the above.

9. **Which of the following diagnostic criteria must be met to distinguish between peri-implant mucositis and peri-implantitis?**
   a. Spontaneous bleeding or bleeding upon probing and/or suppuration.
   b. Increased probing depth.
   c. Evidence of radiographic bone loss over time in comparison to baseline values.
   d. Mobility

10. **In general, all of the following statements are correct with respect to the plaque removal efficacy of toothbrushes except which one?**
    a. Electro-mechanical toothbrushes have been shown to be more effective in plaque removal than manual toothbrushes.
    b. Manual toothbrushes have been shown to be more effective in plaque removal than electromechanical toothbrushes in mandibular lingual areas.
    c. Well controlled prospective studies that demonstrate the superiority of powered brushes specifically around dental implants have not been done.
    d. It is intuitive that maintaining good plaque control around dental implants is beneficial.

11. **In general, which of the following statements is correct with respect to the antiplaque and antigingivitis efficacy of dentifrices?**
    a. There are no controlled, prospective studies comparing the efficacy of various toothpaste formulations around dental implants.
    b. There is robust evidence that in general, dentifrices with stannous fluoride or triclosan with a copolymer have significant antiplaque and antigingivitis activity.
    c. Stannous fluoride-sodium hexametaphosphate has been shown to have activity in subjects non-responsive to a triclosan-copolymer containing dentifrice.
    d. All of the above.

12. **In general, which of the following statements is correct with respect to the antiplaque and antigingivitis efficacy of mouthwashes?**
    a. There are no controlled, prospective studies comparing the efficacy of various mouthwash formulations around dental implants.
    b. There is robust evidence that mouthwash formulations containing chlorhexidine and essential oils have significant antiplaque and antigingivitis activity.
    c. Recent studies have concluded that a 0.07% cetylpyridinium chloride mouthrinse formulation is as effective as essential oil-containing mouthrinses and has superior substantivity and bioavailability compared to other CPC formulations.
    d. All of the above.
13. Which of the following statements is correct with respect to non-surgical treatment of peri-implant mucositis?
   a. Conventional non-surgical therapy appears to successfully reverse peri-implant mucositis.
   b. Laser therapy alone or as an adjunct to conventional therapy has been evaluated, however, the superioriety of laser treatment has not be established.
   c. Adjunctive antibiotic therapy in association with mechanical removal of plaque had only limited success.
   d. All of the above.

14. Which of the following statements is correct with respect to surgical treatment of peri-implantitis?
   a. Reversing frank peri-implantitis successfully hinges on bone regeneration.
   b. Surgical treatment of peri-implantitis is complicated by the fact that there is no cementum on the surface of dental implants and the periodontal ligament, which communicates with marrow spaces in bone, is also absent.
   c. The first step in successful surgical treatment hinges on effective decontamination of the affected site, i.e., establishment of surgical access followed by removal of granulation tissue, calculus, and biofilm.
   d. All of the above.

15. All of the following statements are correct with respect to surgical treatment of peri-implantitis except which one?
   a. Air powder abrasive treatment of the implant surface in association with surgical treatment offers no advantage over traditional decontamination.
   b. Er:Yag laser decontamination in association with surgical treatment was found to be less effective than traditional decontamination.
   c. Implantoplasty, removal of the micro- and macro-roughened implant surface, has been shown to attain absolute decontamination of the implant surface.
   d. All of the above.
References
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Dr. Palomo is Associate Professor of Periodontics and director of the undergraduate (DMD) program in periodontics at School of Dental Medicine, Case Western Reserve University, Cleveland, Ohio. Dr. Palomo earned her undergraduate as well as her DDS (1996) and MSD (2004) degrees from Case Western Reserve University. Dr. Palomo is certified of the American Board of Periodontology. Dr. Palomo has published several articles in refereed medical and dental journals and has been invited as a featured speaker by many local, state, national, and international professional societies.

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