Adjunctive Implant Surface Decontamination for Treatment of Peri-Implant Disease

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Conflict of Interest Disclosure Statement

- Dr. Geisinger does not have any conflicts of interest in regard to the topic of this course. She has no relevant financial relationships to disclose.

Short Description – Implant Laser Therapy

Adjunctive Implant Surface Decontamination for Treatment of Peri-Implant Disease is a free dental continuing education course that covers a wide range of topics relevant to the oral healthcare professional community.

Course Contents

Overview

This course seeks to evaluate the effectiveness of implant surface decontamination therapies for treatment of peri-implant disease.

The placement of endosseous dental implants is a well-accepted treatment option for edentulism as implants have demonstrated high survival rates over long periods of time.[1] Dental implants are a common and growing treatment modality and it is estimated that up to 5 million dental implants are placed each year.[2] While dental implant survival rates remain high, reports indicate that a significant proportion of dental implants will develop peri-implant mucositis and peri-implantitis.[3-6] Prevalence data for peri-implant disease is heterogeneous, but estimates suggest that the prevalence of peri-implant mucositis and peri-implantitis range from 46-63% and 19-23%, respectively.[7] Inflammatory implant diseases are progressive and it has been shown that the inflammatory lesions associated with peri-implant diseases are larger than those around teeth with similar clinical presentation.[8,9] There are several risk factors that can lead to inflammation, peri-implant mucositis, and, later, peri-implantitis.[4,10-12] Systemic and environmental factors may play a secondary role in disease progression and susceptibility, but peri-implant diseases are initiated by accumulation of bacterial biofilm.[4,13,14] As bacterial plaque is the primary etiology for peri-implant disease, current therapies for peri-implantitis require dental plaque removal and implant surface detoxification as a critical step for successful surgical and/or nonsurgical treatment of peri-implantitis.[15,16] This surface detoxification has been accomplished with physical, chemical, laser therapy, and/or other means.

Treatment of peri-implantitis is unpredictable. It has been reported that even with strict supportive peri-implant therapy after active treatment for peri-implant disease, complete resolution of disease is not achieved in the majority of cases over the long term.[17,18] Given the paucity of evidence for efficacious treatments of peri-implant disease, it is imperative that clinicians monitor and prevent the development of inflammation and provide intervention at the earliest possible time in the disease process. Further, critical evaluation of available treatment modalities is imperative to understand best practices for treatment of implant diseases.

Learning Objectives

Upon completion of this course, the dental professional should be able to:
- Understand the current scientific literature about the prevalence, etiology, and stages of peri-implant diseases.
- Recognize, assess, and develop treatment and preventative strategies for peri-implant diseases.
- Evaluate the available evidence regarding the efficacy of implant surface disinfection

treatments to be performed as a part of surgical and nonsurgical treatment of peri-implant diseases.

• Discuss risks, benefits and therapeutic options with patients prior to implant therapy or with peri-implant diseases.

Introduction

Peri-implantitis has been defined as an inflammatory process that affects the soft and hard tissues surrounding an osseointegrated implant in function demonstrating loss of supporting marginal bone.[4,19] Peri-implantitis is, in many ways, an analogous disease to periodontitis, in that it affects the hard and soft tissues around implants and it is initiated by oral bacterial biofilms.[10,20,21] The definitive treatment of peri-implantitis may be more challenging than that of periodontitis. In a recent review of studies examining peri-implantitis treatment with at least a one-year follow up, peri-implantitis was shown to be difficult to fully resolve with up to 100% of cases recurring with some treatment modalities.[15] This and other reports indicate that frequent monitoring and retreatment of this chronic disease may be necessary.[15,17,18] Furthermore, a practitioner's ability to effectively treat peri-implantitis may differ based upon the severity of the presenting levels of attachment loss and the treatment modality used for treatment.[12,15,22,23] Based upon this, early intervention and preventive therapy of peri-implant diseases should be an integral part of therapy to maintaining implants in health and function.[12,24]

There are several risk factors that can lead to inflammation, peri-implant mucositis, and, later, peri-implantitis.[4,6,10] These include dental plaque accumulation, smoking, patients' systemic health conditions, implant design, surgical technique, prosthetic design/procedures, and occlusal forces.[10-12] While these systemic and environmental factors certainly play a role in disease progression and susceptibility, peri-implant diseases are initiated by accumulation of bacterial biofilm.[4,13,14] Therapies to treat peri-implant diseases have focused upon dental plaque removal and implant surface detoxification as a part of surgical and/or nonsurgical

treatments.[12,16] Many methods have been used to remove bacteria from dental implant surfaces and the surrounding inflamed tissues. These include: mechanical debridement, chemical detoxification, and laser therapy.[25-27]

Peri-implant Health and Disease

Missing teeth and supporting structures lost to dental diseases and trauma have been replaced in myriad ways through fixed and/or removable dental prostheses. In 1977, Dr. P.I. Brånemark demonstrated that bone will integrate into the surface of endosseous titanium dental implants,[28] and the modern era of root form endosseous dental implantology arose. It is estimated that up to 5 million dental implants are placed in the United States each year.[2] While longitudinal survival rates of osseointegrated dental implants range between 90-95%,[12,29] these numbers represent implants that are present and in function, but may not fully capture rates of peri-implant disease and or health. It is estimated that rates of peri-implantitis range from 10-47% and rates of peri-implant mucositis have been observed in up to 65% of subjects with dental implants.[30-34] Futhermore, it has also been demonstrated that these peri-implant diseases are increased in patients who smoke and have a history of periodontal disease, which may increase the difficulty in treating these implants.[35]

Given the high prevalence of peri-implant diseases, surveillance, early identification of disease, and intervention is critical. A key factor in long-term success of implants is proper maintenance of their surrounding soft and hard tissues. It has been shown that bacterial accumulation induces inflammatory changes in the tissues surrounding implants.[36] Furthermore, it has been estimated that a monitoring program including regular examination and supportive implant therapy to identify and intercept peri-implant mucositis is highly cost-effective and the economic advantage is increased in high risk patients.[37] In order to determine proper treatment steps to intervene for an implant with signs of peri-implant disease, it is essential to identify disease as early as possible to provide intervention. Clinicians must distinguish

between "ailing" and "failing"/"failed" implants to select an appropriate intervention.

The primary etiology for both peri-implant and periodontal diseases is virulent bacterial plaque.[36,38] While the inflammatory process that occurs around implants is similar to that around natural teeth, progression of disease is quicker in the peri-implant environment and the histologic peri-implant inflammatory lesions are larger and may prove more difficult to resolve at implant sites.[39] This may relate to the peri-implant attachment apparatus and lack of a periodontal ligament as well as the unique implant-soft tissue interface.[39] Multiple systematic reviews and randomized controlled trials have evaluated the efficacy of various treatment strategies for peri-implant diseases and identification of one ideal treatment strategy has proven elusive.[12,40,41] Ideal therapy of peri-implantitis would result in active disease resolution (no suppuration, bleeding on probing, no further bone loss) and the establishment and maintenance of healthy hard and soft peri-implant tissues in a patient and clinical environment where plaque removal was feasible over time.[12] Studies have shown that many therapies may be used to achieve these goals including non-surgical and surgical interventions, alone or combined, including mechanical debridement, pharmaceutical therapy, laser therapy, and open flap debridement with either resective or regenerative procedures.[12] This discussion will focus on laser interventions as an adjunct to improve overall implant health. . Table 1 describes the clinical and histologic characteristics of peri-implant health, peri-implant mucositis, and peri-implantitis.

Peri-implant Health
Healthy peri-implant mucosa is comprised by a core of connective tissue covered by either keratinized or non-keratinized mucosal epithelium. A healthy implant should demonstrate an endosseous implant surface is in contact with mineralized bone, while the remainder is associated with bone marrow, intraosseous vascular structures, and/or fibrous tissues. A healthy implant is asymptomatic and provides function in mastication, speech, and esthetics and the healthy peri-implant environment presents with an absence of clinical inflammation and progressive bone loss.[43]

Peri-implant Mucositis
Peri-implant mucositis is an inflammatory lesion confined to the soft tissues surrounding an endosseous dental implant without loss of supporting bone loss (Figure 1). While this stage of disease may still be reversible, it has been noted that the inflammatory lesion seen in experimental peri-implant mucositis is larger than that seen in experimental gingivitis of the same chronicity and that resolution of the clinical signs of peri-implant mucositis requires a longer time period than the 21-day time period required for resolution of experimental gingivitis.[8] This may indicate that more aggressive and/or invasive treatment may be indicated for such cases to allow for complete resolution of the defects; some authors have advocated earlier intervention, ideally as soon as disease is identified to prevent progression to bone loss and peri-implantitis.[8,9,44] Several factors have been identified as risk factors and/or potential risk factors for peri-implant mucositis, including plaque biofilm accumulation, smoking, head and neck radiation, diabetes mellitus/glycemic control, keratinized mucosa, and the presence of excess luting cement.[10,41,45-47] Successful treatment of peri-implant mucositis has been demonstrated through nonsurgical intervention consisting of supragingival and subgingival debridement with or without adjuncts such as laser and/or photodynamic therapy, locally delivered antibiotics, or chlorhexidine rinse.10 Regardless of treatment modality used, oral hygiene reinforcement, assurance of adequate plaque control, and regular maintenance is crucial to treat the inflammation and prevent future loss.

Peri-implantitis
Peri-implantitis is characterized by inflammation of peri-implant soft tissues and progressive loss of supporting bone that is often circumferential in nature (Figure 2).[48,49] Histologically, peri-implant lesions with similar clinical characteristics often have larger inflammatory lesions than periodontitis lesions around teeth.[50] At an implant presenting with clinical signs of inflammation and radiographic bone loss, systemic, oral and local risk factors should be assessed to determine all possible underlying etiologic factors. Initial therapy should include elimination of the etiologic factors to ensure success of reparative

Table 1: Case definitions for peri-implant health, peri-implant mucositis, and peri-implantitis[46]

	Peri-implant health	Peri-implant mucositis	Peri-implantitis
Definition	Implant without signs and symptoms of disease at either the soft or hard tissue attachment and without noted pathology	Reversible inflammation of the soft tissue mucosal attachment around dental implants without concomitant bone loss	An inflammatory lesion around dental implants that affects the soft and hard tissue attachment and characterized by bone loss.
Visual Inspection	No noted erythema No noted edema Firm mucosal consistency	Erythema may be present Edema may be present Soft mucosal consistency	Erythema may be present Edema may be present Soft mucosal consistency
Patient-Reported Signs/Symptoms	No pain No paresthesia No loss of function	Patients may report pain	Patients may report pain
Bleeding Upon Probing	Lack of profuse bleeding on probing.	Presence of profuse bleeding and/or suppuration on probing	Presence of profuse bleeding and/or suppuration on probing
Probing Pocket Depths	Probing pocket depths may vary based upon soft tissue height, but should not increase over time.	Probing depths are increased from baseline levels	Probing depths are increased from baseline levels
Radiographic Bone Loss	Absence of bone loss after initial remodeling.	Absence of bone loss after initial remodeling.	Progressive bone loss after initial remodeling.

Figure 1. Peri-implant mucositis clinical and radiographic presentation.

**Figure 2.** Peri-implantitis clinical and radiographic presentation.

treatment, if necessary. Establishing ideal overall health, plaque control, and compliance with professional maintenance is critical to the long-term success of therapy. Once it is established that it is possible for the patient to maintain good oral health, site-specific assessment should be performed.to determine if defects can be repaired. Additionally, assessment of the best treatment option for the individual patient and site should be undertaken. This assessment should include an evaluation of the implant and/or prosthetic component mobility, peri-implant defect dimensions, and condition of the implant should be undertaken to allow for optimal customization of the treatment protocol.

Risk Factors for Peri-implant Diseases

In clinical practice, achieving optimal oral health and esthetic results of implant procedures is dependent upon the patient, site, and treatment-related factors. Several risk factors are known to affect the development of peri-implant diseases.[4] Factors that can negatively impact implant health and treatment outcomes include: systemic inflammatory diseases, smoking status, poor plaque control, maintenance adherence, prosthetic design and occlusal overload, retained cement, soft tissue quality/quantity, and previous or active periodontal disease.[4] Due to these risk factors, proper patient selection, site development, and treatment planning are key to achieving high success rates and identification of early clinical signs of disease is critical to successful intervention for peri-implant diseases (Figure 3).[51]

Systemic Diseases

Dental implants are generally elective surgical procedures and should be undertaken on patients who are systemically healthy enough to undergo elective, outpatient procedures. Some common systemic diseases may also directly affect the rates of implant survival. It has been shown that implant failure rates were similar for patients with well-controlled diabetes (HbA1C < 8%) and patients without diabetes; with failure rates in patients with Type 2 diabetes overall demonstrating a marginally significant increase in failures.[52] Patients with uncontrolled diabetes may be poor implant and surgical candidates and demonstrate higher levels of early and late implant failures.[53] In humans, hyperglycemia is known to impair wound healing, impair host defense against pathogens, prolong the inflammatory response to injury, and impair new bone formation and bone repair.[53] The recommended osseointegration periods may be extended in diabetics due to this delay in wound healing caused by hyperglycemia.[54] Future studies are needed to identify distinct cut-off points and quantify the risks, if any, associated with diabetes and development of peri-implantitis. Osteoporosis may also potentially affect implant survival. Osteoporosis and osteopenia are diseases characterized by low bone mass and micro-architectural deterioration with a consequent increase in bone fragility and susceptibility to fracture.[55,56] Osteoporosis is diagnosed when bone mineral density (BMD) is 2.5 standard deviations or more below mean for age and gender-matched individuals and osteopenia is characterized as
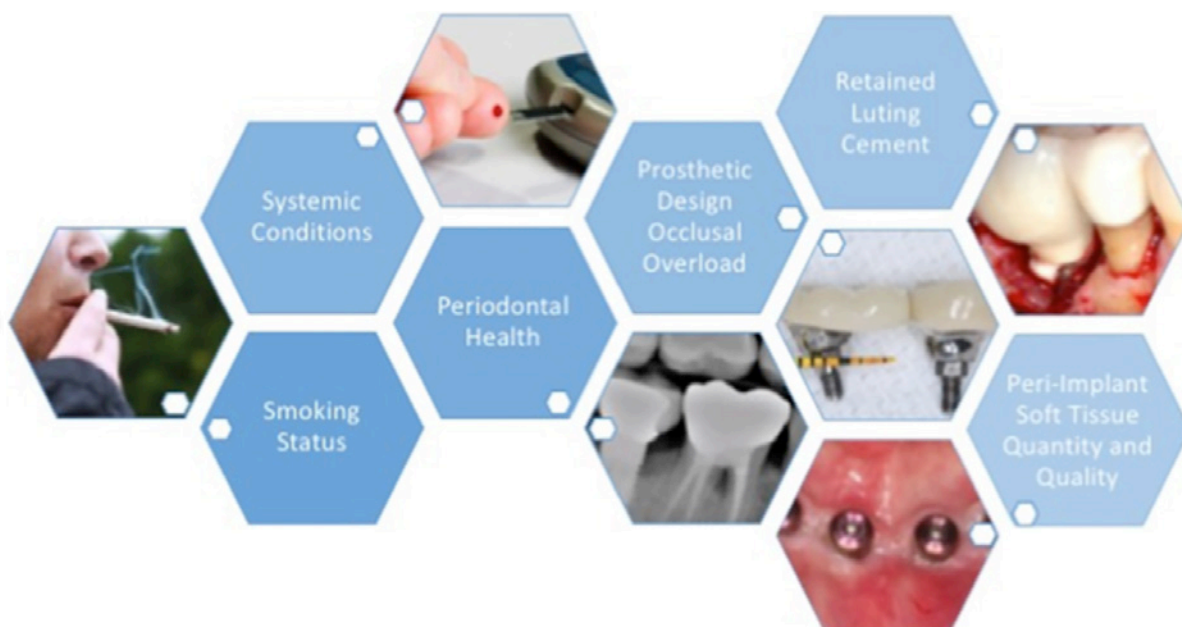
Figure 3. Risk factors for development of peri-implant diseases.

BMD between 1 and 2.5 standard deviations.[55,56] While osteoporosis/osteopenia have common risk factors with periodontal and peri-implant diseases, including cigarette smoking, dietary factors, and medications, periodontal disease has been independently associated with osteoporotic status.[57] Peri-implantitis, periodontal disease and osteoporosis are mediated by similar dysfunction in the bone remodeling process and the interaction between these diseases may be expected. Patients with osteoporosis demonstrated decreased alveolar and axial bone density and mass and thinner cortical bone than healthy counterparts.[58] To date, studies have not shown a definitive association of peri-implantitis with osteoporosis or osteopenia, although implant placement and use of bisphosphonate medications have been shown to potentially mitigate alveolar bone loss in osteoporotic patients.[4,59-61]

Smoking Status and/or Tobacco Cessation
Smoking has been shown to have many negative effects on the oral cavity and wound healing after procedures, such as reduction in neutrophil chemotactic response, vasoconstriction, alterations in innate and adaptive immune response, an increase in number or proportion of periopathogenic

bacteria, and a decrease in fibroblast number and collagen production.[62] These effects of smoking can lead to chronic inflammation at periodontal and peri-implant tissues. Patients who smoke have been shown to have up to two times the failure rate of implants compared to non-smokers.[63] Smoking itself, independent of periodontal health, is a predisposing factor in implant failure and development of peri-implant diseases.[63] Smoking cessation is an important contributing factor to implant success; even though cessation cannot reverse past effects it can increase implant success rates to that of nonsmokers.[64] Supportive implant therapy in patients who smoke has also shown a benefit in reducing rates of peri-implantitis that is of larger magnitude than that seen in non-smokers.[65]

Periodontal Health
Periodontal diseases affecting teeth can similarly affect implants. A history of periodontitis is a risk factor for peri-implantitis.[4,66,67] The primary cause of inflammation around peri-implant tissues is the presence of anaerobic bacteria and their byproducts.[68] Findings suggest that bacteria associated with periodontal disease and peri-implant diseases are similar and the principal pathogens in peri-implant disease

are *P gingivalis* and *A actinomycetemcomitans*.[69] Colonization of dental implants with these bacterial species has been shown to occur within the first 28 days after exposure to the oral environment and bacteria can be transferred from distant reservoirs at tooth sites within a patient's mouth.[70,71] Given the high prevalence of periodontal disease and the rate of tooth loss due to periodontal disease in adult patients, treatment of active periodontal disease and maintenance therapy of both natural dentition and/or dental implants is critical to overall implant success.[4,66]

Plaque Control and Adherence to Regular Supportive Implant Therapy

Plaque control for the prevention and management of peri-implant mucositis is essential in the long-term maintenance of implants in health.[72] Both patient-performed and professional plaque control can result in a reduction in clinical signs of peri-implant inflammation. Additionally, partially edentulous patients demonstrate higher rates of periopathogenic bacteria compared to fully edentulous patients likely resulting from transfer of bacteria and, in particular, pathogenic bacterial species from tooth sites to dental implant sites.[72] Therefore, regular maintenance protocols which reduce overall bacterial loads are critical to reduce the transmission of periodontal pathogenic bacteria from active periodontal sites to implant sites in same mouth.[73] Adherence to regular professional maintenance is key to detect and manage implants that are ailing or failing.[72] A lack of adherence to supportive peri-implant therapies results in significantly higher frequencies of sites with mucosal inflammation and peri-implant bone loss. Therefore, tailored supportive peri-implant therapies, such as reinforcement of personalized oral hygiene instructions combined with professional implant and/or tooth cleaning, should be an integral part of implant therapy.[72]

Prosthetic Design and Occlusal Load

It has been postulated that mechanical overloading is a contributing factor to many instances of peri-implant bone loss and late implant failures.[74] Occlusal load is influenced by prosthetic design but hard to study due to lack of quantification of overload. Some studies indicate that micromotion at the implant-abutment interface compromises the establishment of implant osseointegration during early healing[75] and that implants that have off-axis forces, such as a cantilever design demonstrate more peri-implant bone loss after loading.[74,75] Despite being difficult to quantify occlusal overload in the literature, a systematic review concluded that occlusal overloading was associated with peri-implant marginal bone loss caused by microtrauma concentrated at the marginal bone.[76] It follows that prosthetically-driven and biologically executed treatment planning as well as assessment of occlusal load, including inspection of implant prostheses for signs of potential grinding and other parafunctional habits and occlusal adjustment or prosthetic replacement when premature contacts or interferences are present should be undertaken during the maintenance phase to insure optimal dental implant health.[29]

Retained Cement

Retained cement has been indicated in a large number of peri-implant disease cases. Many dental implant cements are radiolucent and residual cement may not be detected radiographically, particularly if present on the buccal and/or lingual of the fixture. Residual cement may be rough and allow bacterial attachment and, subsequently, peri-implant inflammation.[77] Prosthesis design in combination with the additional irritant of subgingival cement may promote incomplete plaque removal due to the creation of non-cleansable sites.[78] Peri-implant disease prevalence is significantly higher at fixtures with cement-retained versus screw-retained restorations[77,79] and in a case-control study, within a group of implants with diagnosed peri-implantitis, 81% had excess cement present compared to no retained cement found at healthy, control implants.[80] Due to excess cement being a possible risk factor for peri-implant disease, it may be advisable to use screw-retained restorations when possible, practice techniques to avoid excess cement, allow for adequate soft tissue healing prior to seating of a permanent restoration, and allow for early follow up after initial restorative cementation to detect any early signs of cement retention.

Peri-implant Soft Tissue Quality/Quantity

While current evidence on the importance of keratinized and/or attached mucosa around teeth and implants for their health and survival is equivocal, it has been proposed that the establishment of a circumferential seal of tightly packed collagen around the implant-oral cavity interface may improve long-term implant success.[81] Implant survival rates have been shown to be equivalent for implants placed in keratinized and alveolar mucosa, but increased radiographic bone loss and higher levels of gingival inflammation are associated with a lack of keratinized mucosa.[82] Furthermore, increasing bands of keratinized peri-implant mucosa have been associated with improved clinical outcomes, particularly in individuals with erratic compliance to implant maintenance.[83] While there are no definitive studies to conclude that there is a benefit when implants have an adequate band of fixed and/or keratinized mucosa, in patients with other risk factors, including increased plaque accumulation and previous history of periodontitis, increased keratinized and/or fixed mucosa may be protective to allow for personal and professional plaque removal.[82,84]

Current Treatment Strategies for Peri-implant Diseases

Peri-implant mucositis is a reversible condition of the soft tissues around an implant and implants exhibiting inflammation limited to soft tissue and implants with peri-implant mucositis are sometimes described as "ailing" implants.[29] Clinical signs of peri-implant mucositis include presence of bleeding on probing, swelling of the peri-implant mucosa, increase of probing depth (pseudopockets), and/or erythema of surrounding tissues.[85] Peri-implantitis is a bacterially-initiated, inflammatory condition of the tissues around osseointegrated implants characterized by progressive loss of supporting bone that is verified by radiographs and clinical signs of inflammation (bleeding and/or suppuration on probing).[86] Implants with peri-implantitis are often categorized as "failing" implants and when these implants are refractory to treatment and/or present with clinical mobility they are classified as "failed."[87] Implants can fail at various stages in treatment and function. When implants fail due to lack of initial osseointegration, this is referred to as early implant failure. Early failures are influence by patient-specific impaired healing responses, acute infection, premature loading, and/or surgical trauma.[88] Late failure of implants occurs after the initial phase of osseointegration, remodeling and loading. Late failures are associated with occlusal overload, fixture or prosthetic fracture, and peri-implant disease.[89] Peri-implantitis has been seen in a mean of 22% of implants in place and has been noted in 10% of implants and 20% of implant patients within 10 years of surgical implant placement.[3-6,86] When either peri-implant mucositis or peri-implantitis is detected, it is imperative to initiate therapeutic intervention as soon as possible.[12]

Successful therapy to treat peri-implantitis can be assessed in a number of ways. Ideally, resolution of disease would mean absence of clinical inflammation (bleeding on probing) and a lack of progressive bone loss and/or regeneration of lost tissues.[40,41] At sites with peri-implant mucositis, biofilm control and adequate implant maintenance can reestablish implant health.[51,90] A systematic review examining various methods to treat peri-implant disease included studies that reported on implant loss, mean probing depth, percentage of sites or implants with bleeding and/or suppuration on probing, and radiographic bone levels at 12 months (or longer) following treatment. Successful treatment outcomes were defined as: implant survival with no mean probing depths ≥ 5 mm and no further bone loss 12 months after treatment.[40,41] Non-surgical treatment included debridement using manual or ultrasonic instruments, laser treatments in conjunction with local debridement, and adjunctive systemic or local application of antimicrobial agents.[40,41] Successful treatment outcomes for nonsurgical therapy ranged from 0%-84%.[91-93] Generally, these therapies were not successful at sites with initial peri-implantitis demonstrating extensive bone loss and/or deep probing depths.[91] At these sites, additional surgical intervention has been demonstrated to be necessary.[91] Surgical treatment of peri-implantitis included regenerative protocols, access surgery, and resective surgery.[94-102] The

success rates for regenerative surgery ranged from 0-100%[94-98,100,101] and included treatments of bone grafting without membrane using xenograft, autograft, or combination,[95,96] using non-resorbable membranes alone,[94] resorbable membrane in combination with bone graft materials,[95,96] or bone grafting in combination with subepithelial connective tissue graft.[97,98,100] Non-resorbable membranes included in this analysis had a high rate of exposure and did not demonstrate significant clinical improvements.[94] Access surgery using curettes for debridement of implant surface and saline soaked gauze for surface decontamination had success rate of 88% in achieving implant stability over time.[99] Resective surgery alone demonstrated success rate of 0% due to bone loss over 3 years, while implantoplasty with resective surgery had success rate of 100% and bone level remained unchanged over 3 years.[101,102] However, few implants were included in each group of treatment modalities and definitive identification of a superior therapy cannot be made from the current data.

Implant Surface Disinfection: Treatment Modalities and Effectiveness

The rationale for implant surface decontamination includes elimination of calculus, biofilm, and hard deposits such residual cement, to better prepare the implant surface for re-osseointegration prior to or during reconstructive therapy, and/or to establish an implant surface with decreased biofilm adhesion in advance of resective procedures.[103] Commonly used methods for implant surface detoxification include mechanical methods, chemical methods, and laser therapy.[104] It should be noted that many implant surface decontamination methods induce changes in implant surface chemical and physical properties, which may impact long-term clinical outcomes of surgical and nonsurgical therapies.[103,105] It is also notable that little evidence exists on the impact of implant surface geography and baseline surface characteristics on outcomes of decontamination procedures.[103] It is clinically evident that endosseous dental implants with undercuts and screw-form threads present real-life difficulties for thorough debridement of the implant surface while reducing deleterious

alterations to implant surface characteristics.[103] Further, there has been emerging evidence that some implant surface decontamination strategies targeting biofilm may lead to titanium particle dissolution.[103,105] Such titanium particles have also demonstrated cytotoxic effects on fibroblasts and osteoblasts and association with increased biofilm pathogenicity, which may lead to failed treatments.[103,105]

Mechanical Implant Surface Decontamination

Mechanical methods of surface decontamination include implantoplasty, use of hand or ultrasonic scalers, and the use of air powder abrasives.[104,106] Implantoplasty involves the use of rotary instruments to remove modified implant surfaces and to flatten or smooth threads on exposed implant surfaces.[106] Implantoplasty is often combined with resective approaches so that soft tissues are apically positioned and exposed implant surfaces demonstrate decreased plaque adherence.[107,108] Scaling with hand or ultrasonic scalers seeks to mechanically disrupt and remove biofilm deposits. The materials that the scalers and curettes consist of may influence both efficacy of biofilm removal and resultant damage and/or alterations to implant surfaces. Metal ultrasonic scaler tips and curettes are more efficient in removing biofilm and calculus, but alter implant surface topography.[109,110] It is often recommended that metals that are of equivalent or less hardness compared to the composition of dental implants (e.g. gold, titanium) should be use to minimize damage while maximizing biofilm removal.[111] Conversely, nonmetal curettes may be made of plastic, carbon, or resin. Such nonmetal curettes have demonstrated decreased effectiveness in biofilm removal and deposition of curette material on implant surfaces.[106,112] Air powder abrasives use an abrasive powder, including aluminum oxide, sodium bicarbonate, sodium hydrocarbonate, amino acid glycine, or erythritol, propelled by compressed air.[112] Effective biofilm and bacterial endotoxin removal has been demonstrated with minimal damage to implant surfaces.[109,113-115] However, some investigations have demonstrated that powder particles can stay attached to the implant surface after cleaning.[115] It should also be noted that subcutaneous emphysema have been reported after air abrasives around teeth and implants and care should be taken to avoid forcibly direct compressed air into tissues.[116-117]

Chemical Implant Surface Decontamination

Chemical surface decontamination involves the use of acidic and antimicrobial compounds to reduce biofilm on implant surfaces and they are often used in conjunction with nonsurgical and surgical regenerative approaches for treatment of peri-implant disease.[104] Commonly used chemical implant surface decontaminants include citric acid, chlorhexidine, ethylene diamine tetraacetic acid (EDTA), hydrogen peroxide, tetracycline, and saline.[104] Citric acid has a pH of 1 and has been used extensively for implant surface detoxification.[104] In vitro studies demonstrate endotoxin reduction and bacterial cell inactivation with citric acid applications ranging from 30 seconds to 2 minutes in smooth-surface and moderately rough implant surfaces.[118,119] Concentration of citric acid in studies were not consistent with concentrations ranging from 4-40%. Lower concentrations of citric acid demonstrated decreased osteoblastic cytotoxicity, but cell proliferation was impacted at all concentrations.[120,121] Chlorhexidine gluconate is a broad-spectrum antimicrobial and has demonstrated significant reduction in endotoxin and bacterial load on implant surfaces when applied to those surfaces.[118,122] Chlorhexidine has also been associated with induced apoptotic and autophagic/necrotic cell death of osteoblasts and fibroblasts.[123,124] EDTA is widely used in dentistry as a chelating agent and has the advantage of a neutral pH.[104] Little data is available on the clinical efficacy of EDTA for implant surface decontamination.[104] In vitro studies have demonstrated that hydrogen peroxide reduction of endotoxin and elimination of pathogenic bacteria from titanium surfaces.[125] Animal studies have also verified the capability of hydrogen peroxide to decontaminate implant surfaces in vivo and allow re-osseointegration.[126] Tetracycline is a bacteriostatic antibiotic that inhibits bacterial protein synthesis.[104] A slurry including tetracycline 50mg/mL applied to implant surfaces after resective implant surgery has been associated with arrest of peri-implant disease progression and radiographic bone fill.[127,128] Saline combined with mechanical implant surface debridement has been associated with clinical stability after treatment for up to 24 months.[129,130] It has also been reported that use of saline for decontamination provides similar results to other chemical disinfective processes and results in a more biocompatible surface with no deleterious effects on host tissues.[131]

Laser Implant Surface Decontamination

Commercially available lasers that have been used for implant surface disinfection include: carbon dioxide (CO2), diode, erbium yttrium aluminum garnet (Er:YAG), and neodymium yttrium aluminum garnet (Nd:YAG).[132-135] The specific functions and targets of these lasers are summarized in Table 2.

Adjunctive use of laser therapy has been shown to result in decreases in clinical signs of inflammation at 3 months[136-139] and bleeding on probing at six months after treatment, but the effect on other clinical parameters demonstrated minimal benefit.[136] While the preponderance of the current literature body presented does not present definitive findings demonstrating a clinical or microbiological improvement after adjunctive laser therapy, laser therapy with appropriate wavelength and settings can be used effectively to detoxify titanium surfaces without alteration of the surface morphology.[136,140] Additionally, some of the risks of laser therapy may be mitigated by the use of photodynamic therapy (PDT), which uses low-level laser therapy to perform surface decontamination. Recent in vitro studies have indicated that PDT may be more efficient than standard laser disinfection protocols without many of the associated risks.[141] Additionally, given the current evidence, only Er:YAG, diode, and CO2 lasers can be reliably assessed.[140,142, 143] Given the decreased risk of damage to tissues and implants with the use of appropriate time, wavelength, presence of cooling and laser power and the ability of the lasers to detoxify titanium surfaces, they may be a viable adjunctive therapy with nonsurgical and surgical implant treatment, although additional investigations are necessary to standardize protocols and classify expected outcomes.

Clinical Decision Making for Treatment of Patients with Peri-implant Diseases

Complications affecting implants are common and of concern for patients and practitioners alike. Proper maintenance of implants to insure health as well as identification and treatment of prosthetic and biologic complications is critical to the long-term function, esthetics, phonetics, and health of patients who have received endosseous dental implants for tooth replacement. It has been a great challenge of clinicians to properly manage and treat peri-implant disease. The decision tree presented here (Figure 4) was

Table 2. Laser types commonly used to treat peri-implant diseases and their properties.

Laser Type	Wavelength	Chromophore	Classification
Diode	450-1064 nm	Melanin, hemoglobin	Hot; soft tissue
Neodymium yttrium aluminum garnet (Nd:YAG)	1064 nm	Melanin, hemoglobin	Hot; soft tissue
Erbium yttrium aluminium garnet (Er:YAG)	2940 nm	Water, hydroxyapatite	Cold; hard or all tissue
Carbon dioxide (CO_2)	9300 to 10,600 nm/td>	Water, hydroxyapatite	Hot or cold; soft or all tissue

fabricated to guide treatment of these diseases and to possibly intervene at an earlier time point. This particular assessment emphasizes ongoing monitoring of implant health as a part of supportive peri-implant therapy and early detection of peri-implant disease. When treating peri-implant conditions, it is critically important to recognize peri-implant disease at the earliest stage of disease to start treatment immediately to reduce clinical signs of inflammation and progressive attachment loss. If peri-implant disease is identified, clinical decisions for nonsurgical and surgical intervention to treat that disease must be undertaken in a systematic manner. Because evidence suggests that traditional nonsurgical therapy alone is inadequate to treat peri-implantitis, use of adjunctive methods for implant surface decontamination should be considered by the clinician when appropriate. The decision tree in Figure 4 may help guide the management of clinical complications with implant therapy, such as peri-implant mucositis, peri-implantitis, and loss of osseointegration.

Importance of Supportive Peri-Implant Therapy

It is well-established that in the case of successful periodontal treatment, ongoing supportive periodontal therapy is essential to maintain health, prevent periodontal reinfection, reduce the risk of disease recurrence, and decrease the incidence of tooth loss.[144-147] Similarly, supportive peri-implant therapy both prior to any diagnosis of peri-implant disease and after treatment of peri-implant mucositis or peri-implantitis is critical to long-term implant success.[17,148] Patients who did not receive supportive peri-implant therapy, including ongoing surveillance for peri-implant disease, were over twice as likely to develop peri-implant mucositis and four times more likely to develop peri-implantitis than those who were compliant with at least annual supportive implant therapy over a 7 year period.[148] Given these findings, clinicians should discuss the need for supportive peri-implant therapy with patients during implant treatment planning and should utilize risk-based protocols[149] for maintenance interval selection after implant placement and restoration.

Conclusion

Peri-implant health, peri-implant mucositis, and peri-implantitis are distinct entities and present with distinct clinical and microbiologic findings. Ongoing appropriate implant maintenance and examination as well as early identification of peri-implant disease and treatment of reversible inflammation in peri-implant mucositis with nonsurgical therapy is critical for optimal outcomes of implant therapy. In cases where implants demonstrate peri-implantitis, adjunctive mechanical, chemical, and/or laser treatment may be considered for implant surface disinfection. To facilitate diagnosis and treatment of peri-implant diseases, a decision matrix was formulated to allow for identification and treatment for ailing/failing implants. More studies are needed to quantify best treatment options in the varying clinical scenarios seen in practice.
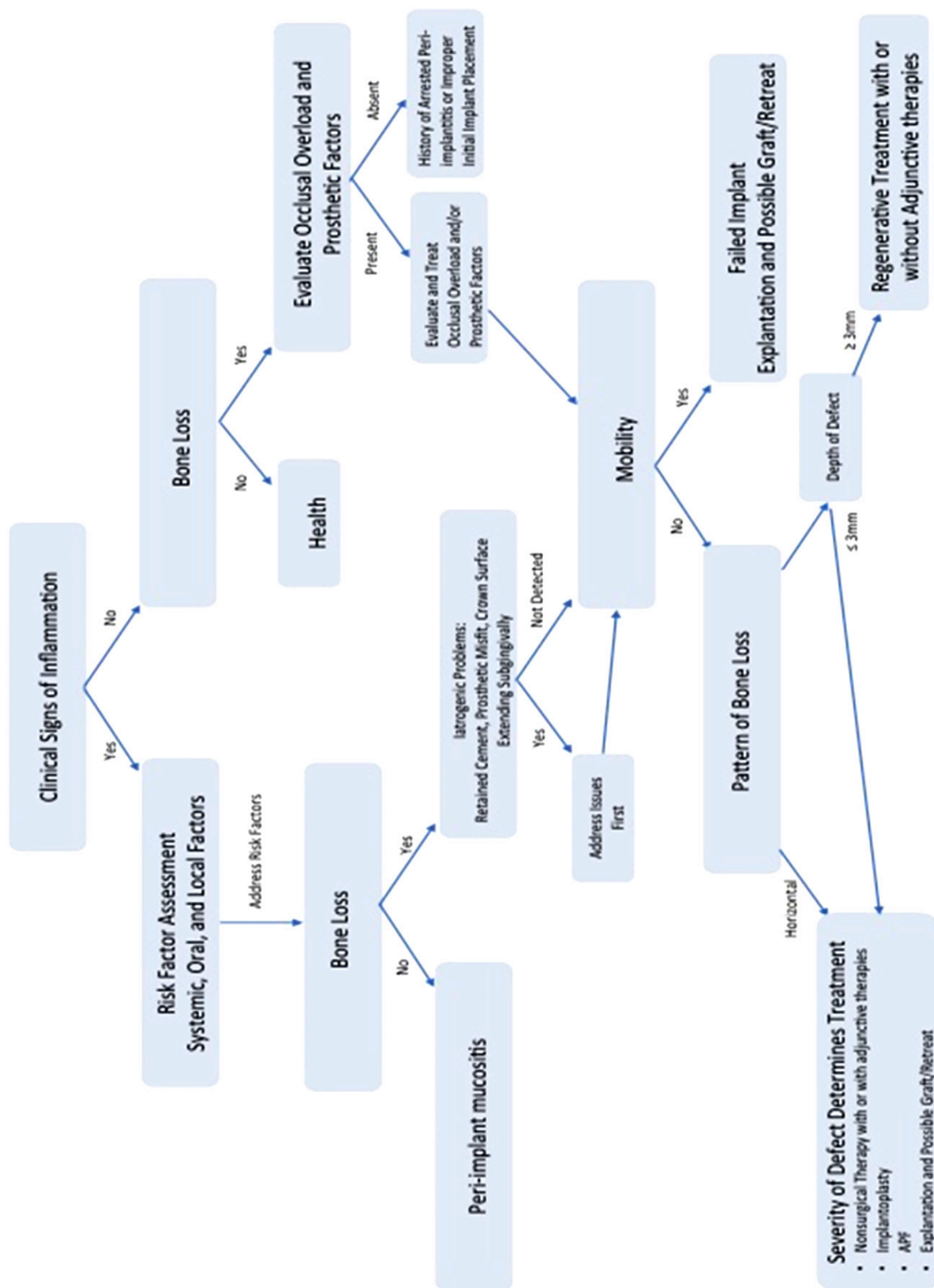
Figure 4. Decision matrix for peri-implant conditions.

Course Test Preview

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1. How many implants are placed annually in the US?
A. 500,000
B. 1 million
C. 5 million
D. 15 million

2. Definitive treatment of peri-implantitis has proven difficult and disease has been shown to recur in up to _____% of cases, depending upon the treatment modality used.
A. 50%
B. 60%
C. 80%
D. 100%

3. Peri-implant mucositis is defined as clinical inflammation without progressive bone loss and it has been observed in up to _____% of subjects with dental implants.
A. 10%
B. 27%
C. 47%
D. 65%

4. Healthy peri-implant mucosa is comprised of:
A. A core of connective tissue covered by either keratinized or non-keratinized epithelium
B. Connective tissue with significant inflammatory infiltrate
C. Keratinized mucosal epithelium only
D. A core of connective tissue with pseudostratified columnar epithelium

5. Peri-implant mucositis is reversible, the inflammatory lesion seen in experimental peri-implant mucositis is _____ that seen in experimental gingivitis of the same chronicity and with similar clinical signs.
A. Larger Than
B. Similar To
C. Smaller Than

6. All of the following have been associated with an increased risk of peri-implantitis, EXCEPT one. Which one is the exception?
A. Smoking cessation
B. Poor plaque control
C. Poor maintenance adherence
D. Periodontal disease

7. Failure rates for dental implants are increased in smokers up to _____ times.
A. 1.5
B. 2
C. 5
D. 10

8. How long does it take for periopathogenic bacteria to colonize dental implant components after their exposure to the oral environment?
A. 14 days
B. 28 days
C. 3-6 months
D. 12-24 months

9. In a case-control study, _____% of healthy implants and _____% of those diagnosed with peri-implantitis demonstrated retained cement upon surgical evaluation?
A. 7%; 76%
B. 25%; 88%
C. 0%; 81%
D. 14%; 94%

10. Which of the following is NOT a reason to perform implant surface decontamination?
A. Elimination of calculus and biofilm
B. To prepare the implant surface for re-osseointegration prior to or during reconstructive therapy
C. To remove the outer layer of surface roughness and alter implant surface topography
D. To establish an implant surface with decreased biofilm adhesion in advance of resective procedures.

11. Which of the following is a potentially harmful effect of implant surface decontamination?
A. Titanium particle dissolution
B. Endotoxin removal
C. Decreased colony forming units of P. gingivalis
D. Re-osseointegration

12. Which of the following has NOT been associated with air abrasion therapy?
A. Effective biofilm and bacterial endotoxin removal
B. minimal damage to implant surfaces
C. Retained powder particles at the implant surface
D. Change in pH after use

13. Which of the following chemical disinfection treatments has NOT been associated with deleterious impacts on host cells?
A. Chlorhexidine gluconate
B. Saline
C. Citric Acid

14. Which of the following commercially available lasers has the longest wavelength?
A. Diode
B. Nd:YAG
C. Er:YAG
D. Carbon dioxide

15. Adjunctive use of laser therapy has been shown to result in decreases in clinical signs of inflammation out to _____ months and bleeding on probing at _____months after treatment.
A. Six, Twelve
B. Six, Eighteen
C. Three, Six
D. Three, Twelve

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Additional Resources
• No Additional Resources Available

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