Treatment of Plaque-induced Gingivitis, Chronic Periodontitis, and Other Clinical Conditions

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Abstract

This paper has been prepared by the Research, Science and Therapy Committee of the American Academy of Periodontology and is intended for the information of the dental profession. It represents the position of the Academy regarding the current state of knowledge about treatment of plaque-induced gingivitis, chronic periodontitis, and some other clinical conditions. Two other papers entitled The Pathogenesis of Periodontal Diseases and Diagnosis of Periodontal Diseases also reflect the Academy's position on these subjects. J Periodontol 2001;72: 1790-1800.

Gingivitis and periodontitis are the two major forms of inflammatory diseases affecting the periodontium. Their primary etiology is bacterial plaque, which can initiate destruction of the gingival tissues and periodontal attachment apparatus.[1,2] Gingivitis is inflammation of the gingiva that does not result in clinical attachment loss. Periodontitis is inflammation of the gingiva and the adjacent attachment apparatus and is characterized by loss of connective tissue attachment and alveolar bone. Each of these diseases may be subclassified based upon etiology, clinical presentation, or associated complicating factors.[3]

Gingivitis is a reversible disease. Therapy is aimed primarily at reduction of etiologic factors to reduce or eliminate inflammation, thereby allowing gingival tissues to heal. Appropriate supportive periodontal maintenance that includes personal and professional care is important in preventing re-initiation of inflammation.

Therapeutic approaches for periodontitis fall into two major categories: 1) anti-infective treatment, which is designed to halt the progression of periodontal attachment loss by removing etiologic factors; and 2) regenerative therapy, which includes anti-infective treatment and is intended to restore structures destroyed by disease. Essential to both treatment approaches is the inclusion of periodontal maintenance procedures.[4]

Inflammation of the periodontium may result from many causes (e.g., bacteria, trauma). However, most forms of gingivitis and periodontitis result from the accumulation of tooth-adherent microorganisms.[5-7] Prominent risk factors for development of chronic periodontitis include the presence of specific subgingival bacteria,[8-10] tobacco use,[9-13] diabetes,[9,10,14] age,[9,10] and male gender.[9,10] Furthermore, there is evidence that other factors can contribute to periodontal disease pathogenesis: environmental, genetic, and systemic (e.g., diabetes).[14,15]

This paper primarily reviews the treatment of plaque-induced gingivitis and chronic periodontitis, but there might be some situations where the described therapies will not resolve disease or arrest disease progression. Furthermore, the treatments discussed should not be deemed inclusive of all possible therapies, or exclusive of methods of care reasonably directed at obtaining good results. The ultimate decision regarding the appropriateness of any specific procedure must be made by the practitioner in light of the circumstances presented by an individual patient.

Plaque-induced gingivitis

Therapy for individuals with chronic gingivitis is initially directed at reduction of oral bacteria and associated calcified and noncalcified deposits. Patients with chronic gingivitis, but without significant calculus, alterations in gingival morphology, or systemic diseases that affect oral health, may respond to a therapeutic regimen consisting of improved personal plaque control alone.[16] The periodontal literature documents the short- and long-term effects following self-treatment of gingivitis by personal plaque control.[16-20] However, while it may be possible under controlled conditions to remove most plaque with a variety of mechanical oral hygiene aids, many patients lack the motivation or skill to attain and maintain a plaque-free state for significant periods of time.[21-23] Clinical trials also indicate that self-administered plaque control programs alone, without periodic professional reinforcement, are inconsistent in providing long-term inhibition of gingivitis.[19,24,25]

Many patients with gingivitis have calculus or other associa-ted local factors (e.g., defective dental restorations) that interfere with personal oral hygiene and the ability to remove bacterial plaque. An acceptable therapeutic result for these individuals is usually obtained when personal plaque control measures are performed in conjunction with professional removal of plaque, calculus, and other local contributing factors.[26,27] Removal of dental calculus is accomplished by scaling and root planing procedures using hand, sonic, or ultrasonic instruments. The therapeutic objective of scaling and root planing is to remove plaque and calculus to reduce subgingival bacteria below a threshold level capable of initiating clinical inflammation. The success of instrumentation is determined by evaluating the periodontal tissues following treatment and during the maintenance phase of therapy.

The use of topical antibacterial agents to help reduce bacterial plaque may be beneficial for the prevention and treatment of gingivitis in some patients.[28-30]

A number of these agents in oral rinses and dentifrices have been tested in clinical trials.[28] However, to be accepted by the American Dental Association (**ADA**) Council on Dental Therapeutics as an effective agent for the treatment of gingivitis, a product must reduce plaque and demonstrate effective reduction of gingival inflammation over a period of at least 6 months. The agent must also be safe and not induce adverse side effects.

Three medicaments have been given the ADA Seal of Acceptance for the control of gingivitis. The active ingredients of one product are thymol, menthol, eucalyptol, and methyl salicylate.[29] Active ingredients in the other two are chlorhexidine digluconate and triclosan.[29] If properly used, the addition of a topical anti-plaque agent to a gingivitis treatment regimen for patients with deficient plaque control will likely result in reduction of gingivitis.[30] However, experimental evidence indicates that penetration of topically applied agents into the gingival crevice is minimal.[31] Therefore, these agents are useful for the control of supragingival, but not subgingival plaque. Among individuals who do not perform excellent oral hygiene, supragingival irrigation with and without medicaments is capable of reducing gingival inflammation beyond that normally achieved by toothbrushing alone. This effect is likely due to the flushing out of subgingival bacteria.[32]

If gingivitis remains following the removal of plaque and other contributing local factors, thorough evaluation should be undertaken of systemic factors (e.g., diabetes, pregnancy, etc.) If such conditions are present, gingival health may be attained once the systemic problem is resolved and plaque control is maintained.

Acute periodontal diseases

Necrotizing ulcerative gingivitis (**NUG**) is associated with specific bacterial accumulations occurring in individuals with lowered host resistance.[1] NUG usually responds rapidly to the reduction of oral bacteria by a combination of personal plaque control and professional debridement. If lymphadenopathy or fever accompanies oral symptoms, administration of systemic antibiotics may be indicated. The use of chemotherapeutic rinses by the patient may be beneficial during the initial treatment stages. After the acute inflammation of the NUG lesion is resolved, additional intervention may be indicated to prevent disease recurrence or to correct resultant soft tissue deformities.

Necrotizing ulcerative periodontitis (**NUP**) manifests as rapid necrosis and destruction of the gingiva and periodontal attachment apparatus. It may initiate gingival bleeding and pain, and it usually represents an extension of necrotizing ulcerative gingivitis in individuals with lowered host resistance. NUP has been reported among both HIV-positive and negative individuals, but its true prevalence is unknown.[33-38] Management of NUP involves debridement which may be combined with irrigation with antiseptics (e.g., povidone iodine), antimicrobial mouth rinses (e.g., chlorhexidine), and administration of systemic antibiotics.[39] There is also evidence that HIV-immune deficiency may be associated with severe loss of periodontal attachment that does not necessarily present clinically as an ulcerative lesion.[40] Although not an acute disease, linear gingival erythema (LGE) occurs in some HIV-infected individuals and does not appear to respond to conventional scaling, root planing, and plaque control.[39] Antibiotic therapy should be used in HIV-positive patients with caution due to the possibility of inducing opportunistic infections.[39,40]

The oral manifestations of a primary herpes simplex virus type I infection often include gingivitis. By the time gingivitis is present, patients are usually febrile, in pain, and have lymphadenopathy. Diagnosis is generally made from the clinical appearance of the oral soft tissues. Although not performed routinely, a viral culture may provide definitive identification of the infective agent. In otherwise healthy patients, treatment for herpetic gingivitis consists of palliative therapy. The infection is self-limiting and usually resolves in seven to 10 days. Systemic antiviral therapy with acyclovir is appropriate for immuno-compromised patients with herpetic gingivitis.[41]

Gingival enlargement

Chronic gingival inflammation may result in gingival enlargement. This overgrowth of gingiva may be exaggerated in patients with genetic or drug-related systemic factors (e.g., anticonvulsants, cyclosporine and calcium channel blocking drugs).[42-46] Among individuals taking phenytoin, gingival overgrowth may be minimized with appropriate personal oral hygiene and professional maintenance.[47,48] However, root debridement in patients with gingival overgrowth often does not return the periodontium to normal contour. The residual overgrowth may not only complicate the patient's ability to adequately clean the dentition, but it may also present esthetic and functional problems.[49]

For patients with gingival overgrowth, the modification of tissue topography by surgical recontouring may be undertaken to create a maintainable oral environment.[47,50] Postoperative management following tissue resection is important. The benefits of surgical reduction may be lost due to rapid proliferation of the tissues during the post-therapy phase.[51] Recurrence is common in many patients with drug-induced gingival overgrowth.[51] For these patients, consultation with the patient's physician is advisable to determine if it is possible to use an alternative drug therapy that does not induce gingival overgrowth. If not, then repeated surgical and/or non-surgical intervention may be required.

Chronic periodontitis

Appropriate therapy for patients with periodontitis varies considerably with the extent and pattern of attachment loss, local anatomical variations, type of periodontal disease, and therapeutic objectives. Periodontitis destroys the attachment apparatus of teeth resulting in periodontal pocket formation and alteration of normal osseous anatomy. The primary objectives of therapy for patients with chronic periodontitis are to halt disease progression and to resolve inflammation. Therapy at a diseased site is aimed at reducing etiologic factors below the threshold capable of producing breakdown, thereby allowing repair of the affected region. Regeneration of lost periodontal structures can be enhanced by specific procedures. However, many variables responsible for complete regeneration of the periodontium are unknown and research is ongoing in this area.

Scaling and root planing

The beneficial effects of scaling and root planing combined with personal plaque control in the treatment of chronic periodontitis have been validated.[52-65] These include reduction of clinical inflammation, microbial shifts to a less pathogenic subgingival flora, decreased probing depth, gain of clinical attachment, and less disease progression.[52-65]

Scaling and root planing procedures are technically demanding and time-consuming. Studies show that clinical conditions generally improve following root planing; nonetheless, some sites still do not respond to this therapy.[62,63,66,67] The addition of gingival curettage to root planing in the treatment of generalized chronic periodontitis with shallow suprabony pockets does not significantly reduce probing depth or gain clinical attachment beyond that attained by scaling and root planing alone.[68,69] The following factors may limit the success of treatment by root planing: root anatomy (e.g., concavities, furrows etc.), furcations,[66] and deep probing depths.[70-72]

Several weeks following the completion of root planing and efforts to improve personal plaque control, re-evaluation should be conducted to determine the treatment response. Several factors must be considered at sites that continue to exhibit signs of disease. If the patient's daily personal plaque control is not adequate to maintain gingival health, then additional instruction and motivation in personal plaque control and/or the use of topical chemotherapeutics (e.g., mouthrinses, local drug delivery devices) may be indicated. Anatomical factors that can limit the effectiveness of root instrumentation or limit the patient's ability to perform personal plaque control (e.g., deep probing depths, root concavities, furcations) may require additional therapy including surgery. Host response may also have an effect on treatment outcome and patients with systemic conditions (e.g., diabetes, pregnancy, stress, AIDS, immunodeficiencies, and blood dyscrasias) may not respond well to therapy that is directed solely at controlling local factors. In such patients, it is important that attempts be made to control the contributing systemic factors.

Pharmacological therapy

Pharmacotherapeutics may have an adjunctive role in the management of periodontitis in certain patients.[73] These adjunctive therapies are categorized by their route of administration to diseased sites: systemic or local drug delivery.

Systemic drug administration

Numerous investigations[73] have assessed the use of systemic antibiotics to halt or slow the progression of periodontitis or to improve periodontal status. The adjunctive use of systemically delivered antibiotics may be indicated in the following situations: patients with multiple sites unresponsive to mechanical debridement, acute infections, medically compromised patients, presence of tissue-invasive organisms and ongoing disease progression.[74-77] The administration of antibiotics for the treatment of chronic periodontitis should follow accepted pharmacological principles including, when appropriate, identification of pathogenic organisms and antibiotic sensitivity testing.

Considerable research efforts have focused on systemic application of host modulating agents such as non-steroidal anti-inflammatory drugs (**NSAIDS**)[78-80] and subantimicrobial dose doxycycline.[81-84] Investigators have reported some benefit when these medications are incorporated into treatment protocols.[78,81-84] Recently [year 2000], the United States Food and Drug Administration (**FDA**) approved the use of a systemically delivered collagenase inhibitor consisting of a 20-mg capsule of doxycycline hyclate as an adjunct to scaling and root planing for the treatment of periodontitis. Benefits included a statistically significant reduction in probing depths, a gain in clinical attachment levels and a reduction in the incidence of disease progression.[82-84] Overall, the data suggest that use of subantimicrobial dose doxycycline as an adjunct to scaling and root planing provides defined but limited improvement in periodontal status.

It is important to consider the potential benefits and side effects of systemic pharmacological therapy. Benefits may include the ability to treat patients unresponsive to conventional therapy or an individual with multiple sites experiencing

recurrent periodonitits. In contrast, potential risks associated with systemically administered antibiotics include development of resistant bacterial strains,[85] emergence of opportunistic infections, and possible allergic sensitization of patients.[73] With regard to the prolonged administration of NSAIDS, harmful effects may include gastrointestinal upset and hemorrhage, renal and hepatic impairment, central nervous system disturbances, inhibition of platelet aggregation, prolonged bleeding time, bone marrow damage, and hypersensitivity reactions.[73] At present, the incidence of negative side effects reported after root planing with or without administration of subantimicrobial dose doxy-cycline has been similar. In general, since patients with chronic periodontitis respond to conventional therapy, it is unnecessary to routinely administer systemic medications such as antibiotics, NSAIDS, or subantimicrobial dosing with doxycycline.

Local delivery

Controlled delivery of chemotherapeutic agents within periodontal pockets can alter the pathogenic flora and improve clinical signs of periodontitis.[86-94] Local drug delivery systems provide several benefits; the drug can be delivered to the site of disease activity at a bactericidal concentration and it can facilitate prolonged drug delivery. The FDA has approved the use of an ethylene vinyl acetate fiber that contains tetracycline,[86-91] a gelatin chip that contains chlorhexidine[93] and a minocycline polymer formulation[92] as adjuncts to scaling and root planing. The FDA has also approved doxycycline hyclate in a bioabsorbable polymer gel as a stand-alone therapy for the reduction of probing depths, bleeding upon probing, and gain of clinical attachment.[94]

Local delivery systems have potential limitations and benefits. If used as a monotherapy, problems associated with local delivery can include allergic reaction, possible inability to disrupt biofilms, and failure to remove calculus.[95] The benefits include the ease of application, selectively targeting a limited number of diseased sites that were unresponsive to conventional therapy, and possibly enhanced treatment results at specific locations. Local delivery modalities have shown beneficial clinical improvements with regard to probing depth reduction and gain in clinical attachment.[91-94] Furthermore, there are limited data to suggest that local delivery of antibiotics may also be beneficial in preventing recurrent attachment loss in the absence of maintenance therapy.[90]

Utilization of antibiotics at an individual site will depend on the discretion of the treating therapist after consultation with the patient. The greatest potential of local delivery devices may be to enhance therapy at sites that do not respond to conventional treatment. Ultimately, the results of local drug delivery must be evaluated with regard to the magnitude of improvement that can be attained relative to disease severity. A more complete review of local drug delivery can be found in the American Academy of Periodontology position paper "The Role of Controlled Drug Delivery for Periodontitis".[87]

Surgical therapy

Surgical access to facilitate mechanical instrumentation of the roots has been utilized to treat chronic periodontitis for decades. A surgical approach to the treatment of periodontitis is utilized in an attempt to: 1) provide better access for removal of etiologic factors; 2) reduce deep probing depths; and 3) regenerate or reconstruct lost periodontal tissues.[96-98]

Clinical trials indicate that both surgical and nonsurgical approaches can be effective in achieving stability of clinical attachment levels.[60-65,99-103] Flap reflection is capable, however, of increasing the efficacy of root debridement, especially at sites with deep probing depths or furcations.[60-65,70,72,99-104] Nevertheless, complete calculus removal, even with surgical access, may not always be achieved.[70,72,104] The addition of osseous resection during surgical procedures appears to produce greater reduction of probing depth due to gingival recession,[62,64,65] particularly in furcations.[66] Regardless of the type of therapy, furcated teeth are problematic since they are still more likely to lose clinical attachment than nonfurcated teeth.[66,67,105] While these overall findings are helpful, the practitioner should base specific decisions for therapy on findings for each individual patient.

Regenerative surgical therapy

The optimal goal of therapy for individuals who have lost a significant amount of periodontal attachment is regeneration of lost tissues. While root debridement in combination with plaque control has demonstrated efficacy in resolving inflammation and arresting periodontitis,[26,27,60-65] healing typically results in the formation of a long junctional epithelium[106-108] with remodeling of the alveolus.[109] Similarly, surgical debridement alone does not induce significant amounts of new connective tissue attachment.[110,111] However, some bone fill may occur in selected sites.[107,112]

Clinical trials suggest that obtaining new periodontal attachment or regenerating lost tissues is enhanced by the use of adjunctive surgical technique devices and materials. Chemical agents that modify the root surface, while promoting new attachment, have shown variable results when used in humans.[113-118] Bone grafting[119-125] and guided tissue regeneration (GTR) techniques, with or without bone replacement grafts,[126-133] may be successful when used at selected sites with advanced attachment loss. The use of biologically engineered tissue inductive proteins (e.g., growth factors, extracellular matrix proteins, and bone morphogenic proteins) to stimulate periodontal or osseous regeneration has also shown promise.[134-142] Literature reviews on periodontal regeneration[143,144] and mucogingival therapy[145] provide additional information regarding these therapies.

Regenerative therapy and other treatment modalities can be affected by several risk factors (e.g., diabetes and tobacco use) which can diminish periodontal treatment outcomes.[146] In this regard, cigarette smoking is associated with a high risk for progressive periodontitis[9-13,147] and treatment for periodontitis may be less effective in smokers than non-smokers.[148-150] These factors are reviewed in more depth in the Academy's position paper Tobacco Use and the Periodontal Patient.[151] To maximize effective prevention and treatment of periodontitis, patients should be encouraged to stop smoking and to stop using smokeless tobacco.

Occlusal management

Several studies indicated that excessive occlusal forces do not initiate plaque-induced periodontal disease or connective tissue attachment loss (periodontitis).[152-155] However, other investigations suggest that tooth mobility may be associated with adverse effects on the periodontium and affect the response to therapy with respect to gaining clinical attachment.[156,157] With regards to treatment, occlusal therapy may aid in reducing tooth mobility and gaining some bone lost due to traumatic occlusal forces.[158] Occlusal equilibration also may be used to ameliorate a variety of clinical problems related to occlusal instability and restorative needs.[159] Clinicians should use their judgment as to whether or not to perform an occlusal adjustment as a component of periodontal therapy based upon an evaluation of clinical factors related to patient comfort, health and function.[160]

Periodontal maintenance procedures

Periodic monitoring of periodontal status and appropriate maintenance procedures should be part of the long-term treatment plan for managing chronic periodontitis.[28] Although experimental studies have demonstrated very successful treatment outcomes when patients are professionally maintained at two-week intervals,[161] such a program is impractical for most chronic periodontitis patients. Therefore, to maximize successful therapeutic outcomes, patients must maintain effective daily plaque control. It also appears that in-office periodontal maintenance at three to four month intervals can be effective in maintaining most patients.[4] A more comprehensive review on this subject can be found in the American Academy of Periodontology's position paper entitled *Supportive Periodontal Therapy* (SPT).[162]

Summary

The inflammatory components of plaque induced gingivitis and chronic periodontitis can be managed effectively for the majority of patients with a plaque control program and non-surgical and/or surgical root debridement coupled with continued periodontal maintenance procedures. Some patients may need additional therapeutic procedures. All of the therapeutic modalities reviewed in this position paper may be utilized by the clinician at various times over the long-term management of the patient's periodontal condition.

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