

“Getting it Right” in the Long-Term Management of Smoking-Related Chronic Periodontitis



Abstract

The US Surgeon General has charged oral health care professionals to develop interventions targeted at smoking-related diseases and disorders, including periodontal disease. As periodontists and periodontal therapists scramble to halt the disease progression of smoking-related chronic periodontitis among an aging population, new research applied to a common clinical pathway for nonsurgical treatment may signal “hope at

last.” This article provides hygienists who want to practice at the periodontal therapist level an opportunity to consider using a proposed clinical pathway to treat the kind of cases we see in everyday private practice involving smoking-related chronic periodontitis. Armed with host-modulatory therapy in addition to “one-stage” scaling and root planing and risk modification, we might just be able to “get it right.”

We all know the nightmare associated with “losing ground” when trying to stabilize the periodontium of our patients who smoke. Greater than 90% of patients with refractory periodontitis are smokers¹⁻³ whose case management continues to challenge even the most veteran of clinicians. In long-term management of smoking-related chronic periodontitis, we need to get it right.

In 2000, the US Surgeon General tasked the dental profession with creating and implementing actions to address the lack of periodontal health in tobacco use of the citizens of our country.⁴ The greatest promise for achieving therapeutic success appears to lie in the benefit derived from applying scientifically founded treatment models and modalities that address not only the bacterial component of periodontal disease, but the host response as well. Compelling evidence highlighting the importance of treating host response continues to increase in research findings. It is well documented that both nonsurgical and surgical periodontal therapies are less effective in smokers and that recurrence of disease is more likely in smokers after periodontal treatment.⁵⁻⁸ A more recent discovery may lend even greater support for modulation of the host response of smokers with chronic periodontitis. A study reported in 2001 examined the effect that a single session of scaling and root planing (SRP) had on gingival crevicular fluid (GCF) levels of biochemical markers associated with active bone resorption in the progression of periodontal disease.⁹ The findings of this study have great significance to the long-term manage-

ment of smokers because it was determined that a single episode of SRP did not significantly reduce GCF biochemical markers associated with bone resorption in smokers with chronic periodontitis. In fact, smokers continued to “lose ground” relative to alveolar bone height. Although smokers in this study exhibited improvements in clinical parameters, it may be reasoned that the improvement observed at the clinical level “may not represent the metabolic state of the tissues” within the periodontium.⁹ In other words, it appears that mechanisms of bone resorption may continue in smokers even after SRP procedures successfully disrupt subgingival biofilms. This helps explain why conventional periodontal therapies are just not cutting it with these patients.

Given this information, we should be looking more seriously at modulating the host response as a long-term strategy in stabilizing the periodontium of patients with smoking-related chronic periodontitis. The American Academy of Periodontology (AAP) supports the use of host-

modulatory therapy as follows: “in situations in which conventional therapy does not always achieve the desired clinical outcomes”; further, “certain patients possess non-microbial risk factors which are difficult to reduce or eliminate (e.g., smoking, diabetes)” or are beyond the clinician’s ability to control (e.g., genetic predisposition); and finally, “in these instances...the use of host-modulatory therapy in conjunction with antibiofilm treatment may prove to be advantageous (however, this concept needs to be proven in controlled clinical trials).”¹⁰ The only available chemotherapeutic agent approved by the Food and Drug Administration (FDA) and the American Dental Association for host-modulatory therapy, used to suppress tissue-destroying enzymes responsible for collagen degradation in the periodontium is subantimicrobial-dose doxycycline, (Periostat®, CollaGenex Pharmaceuticals, Inc, Newtown, Pa).¹¹ Despite the AAP’s validation of this therapeutic approach, its potential in everyday clinical practice is often overlooked. Periostat® has been approved by the FDA as an adjunct to SRP to promote attachment level gain and reduce pocket depths (PDs) in patients with adult periodontitis.^{12,13} Its therapeutic value is in inhibiting collagenase activity, thereby minimizing the degradation and preserving the content of collagen within the connective tissue of the periodontium. Of all the treatment modalities available for long-term

Learning Objectives

After reading this article, the reader should be able to:

- discuss the etiological challenges that smokers with chronic periodontitis face that are independent of microbial infection.
- identify treatment modalities that target the bacterial component of chronic periodontitis.
- discuss the effectiveness of host-modulatory therapy with subantimicrobial dose doxycycline (Periostat®) in cases of smoking-related, chronic periodontitis.
- describe a proposed clinical pathway for smoking-related chronic periodontitis.



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management of smoking-related chronic periodontitis, modulation of the host response may prove to be the most effective. While Periostat® is the only host modulatory agent approved by the FDA, other host modulators have been studied for use in the modulation of periodontal inflammation, ie, nonsteroidal anti-inflammatory drugs (NSAIDs) and bisphosphonates.¹⁴

The Epidemiology of Smoking and Chronic Periodontitis

The epidemiology of this addiction-related disease is daunting. Data derived from the National Health and Nutrition Examination Survey (NHANES III) survey period of 1988 through 1994 found that roughly 28% of dentate adults were current smokers and 23% were former smokers. By applying standard epidemiologic formulas, the same NHANES III survey calculated that about 42% of periodontitis cases (6.4 million cases) in the US adult population were attributable to current smoking status and about 11% (1.7 million cases) to former smoking status. Furthermore, among current smokers, 75% of their periodontitis was deemed attributable to smoking.¹⁵ The relationship between cigarette smoking and oral, pharyngeal, and lung cancers, and cardiovascular and respiratory diseases has been well publicized in the United States.¹⁶ However, the similarly strong association between cigarette smoking and periodontal disease remains relatively unknown among the general population.

There is substantial evidence that cigarette smoking is not only a risk factor for periodontitis, but also exacerbates its effects and compromises the effectiveness of available therapies.^{5,17} Emerging evidence also suggests that other forms of tobacco use (pipe and cigar smoking, and smokeless tobacco) also exhibit the same destructive characteristics.¹⁸ Not only is smoking a risk factor for periodontal disease,^{2,19-21} it also impacts the severity of the disease. Smokers have greater periodontal attachment loss, greater alveolar bone loss, a greater number of deep pockets, and increased tooth loss, yet variable levels of plaque and inflammation.^{9,18} Interestingly, the effect of smoking on treatment outcomes appears to be dose dependent, with heavy smokers (>20 cigarettes/day) exhibiting greater disease progression than light smokers.²

While the prevalence of smoking has decreased among US women over the past 30 years, close to 27% of women still smoke, and surprisingly, the prevalence of smoking has dropped more significantly among men than women.²² Combine this with the demographics and compromised health trends in women (osteoporosis/osteopenia, acquired immune diseases, xerostomia-inducing medications, etc) associated with an aging

cal mechanical therapy, exhibiting less posttreatment probing depth reduction and less gain in attachment and bone height than non-smokers.² Smokers have decreased wound healing ability as a result of impaired vascularization,²³ which jeopardizes the outcomes of surgical interventions. Smoking could lead to increased periodontal destruction by altering the host response by impairment of the normal host response in

odontal stability of refractory patients and/or make surgery a more viable option in Phase III treatment, which focuses on repairing periodontal defects that are the result of disease.

A Proposed Clinical Pathway

The clinical pathway presented in this article includes specific microbial and nonmicrobial components:

- Eliminating or reducing the risk factor of smoking or other tobacco use.
- Modulating the host response by enzyme suppression with Periostat®.
- “One-stage” SRP to prevent the recolonization of subgingival periodontal pathogens via translocation of bacteria from untreated quadrants to treated quadrants.
- Site-specific bacteriostatic alterations in PDs ≥ 5 mm by application of the locally delivered, subgingival, controlled-release antimicrobial Atridox® (CollaGenex Pharmaceuticals, Inc, Newtown, Pa) approved for gain in calculated attachment level^{26,27}. Other locally delivered antimicrobial agents include PerioChip™ (2.5 mg chlorhexadine gluconate; Astra Pharmaceuticals, London, UK); Actisite® (tetracycline hydrochloride fibers; (manufactured by Alza Pharmaceuticals, marketed by Procter & Gamble, Cincinnati, Ohio) and Arestin™ (1 mg minocycline).

continued on page 16

Smokers have greater periodontal attachment loss, greater alveolar bone loss, a greater number of deep pockets, and increased tooth loss, yet variable levels of plaque and inflammation.

population, and it seems inevitable that cases of progressive, chronic periodontitis among women smokers, in particular postmenopausal women, may become increasingly common in general practice.

As referenced above, recent research indicates that the increased risk for the development of periodontitis in patients who smoke appears to be independent of an altered microbial profile; rather, it may have more to do with individual change in host response.^{9,10} This may help explain why smokers are less responsive to surgical and nonsurgi-

neutralizing infection^{24,18} and alterations of normal functional mechanisms, ie, neutrophil functions, revascularization in soft and hard tissues, increased fibroblast collagenase activity, modified or intensified cytokines or proinflammatory mediators, etc, that result in destruction of the surrounding healthy periodontal tissues.^{18,25} Clearly, treatment plans that recommend surgery must consider patients' current use of tobacco when predicting outcome. Defining a prescribed clinical pathway that incorporates host-modulatory therapy may increase the peri-

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continued from page 15

Table 1—Therapeutic Outcomes Compared to Contributions to PD Reduction and Calculated Attachment Gain of Other Treatment Modalities/Models

	Mean Change In PD			Mean Change In CAL		
	4-6 mm	≥ 7 mm	All Teeth	4-6 mm	≥ 7 mm	All Teeth
Patient 4.5 months posttreatment	1.6	3.7	1.8	1.4	3.7	1.7
Generally accepted contributions of SRP alone ²⁹	1.3	2.2		0.6	1.3	
One-stage full-mouth SRP (sustained up to 8 mo after treatment) ⁴³	additional reduction of 0.9 mm–1.2 mm, depending on # of roots			additional corresponding gain of 0.8 mm–1.0 mm, depending on # of roots		
Host-modulatory therapy (Periostat®) ¹³ (at 3 mo)	0.22	0.6		0.2	0.5	
Locally delivered, subgingival, controlled-release antimicrobial (Atridox®)(tested as a monotherapy; reported at 2 and 4 mos) ²⁷	0.8	1.4	0.9	0.6	0.8	0.6

CAL = calculated attachment gain; PD = pocket depth

cline hydrochloride microspheres, OraPharma, Inc, Warminster, Pa).

- Irrigation of pockets <5 mm with an aqueous solution of 10% tetracycline immediately after SRP to render the shallow pockets bacteriostatic.
- Prescription of appropriate systemic antibiotics as determined by DNA-probe analysis and sensitivity testing.
- Comprehensive patient education in the etiological progression of periodontal disease, self-care training in plaque removal with an powered toothbrush and pulsated water irrigation; complete communication regarding the importance of compliance to medication regimens including both systemic antibiotics and Periostat®, and periodontal maintenance visits.

This proposed clinical pathway is the subject of the following case, which represents one of many cases in which disease progression was halted in a patient with smoking-related chronic periodontitis.

Clinical Case

This patient, a 59-year-old white woman, is now in Phase II treatment, which is designed to sustain disease inactivity. This case study sought to discover whether the disease activity in a type IV smoking-related, chronic periodontitis case could be halted and sustained by following a clinical pathway that utilized aggressive, nonsurgical treatment modalities as outlined in the clinical pathway presented above. Indeed, the clinical end points of Phase I (halting disease progression)

of this case mirrored the results of large, well-designed, clinical trials conducted to investigate the safety and efficacy of the various treatment modalities used in this case.^{13,27,28} In fact, the outcomes at 4.5 months posttreatment far surpassed the results of published collective data²⁹ related to average probing depth reduction and gains in calculated attachment level that can be expected from SRP (Table 1). The rather dramatic success in this patient's case may be a result of the combined effects of all therapies: Periostat® to suppress collagenase production within the periodontium¹, Atridox® to reduce the bacterial biomasses that inhabit the pocket environment; and SRP in “one stage” to prevent the translocation of periodontal pathogens from nontreated to treated sites. The combined effect of using these individual, evidence-based treatment modalities/models may prescribe a cumulative clinical pathway for achieving predictable and sustainable therapeutic end points in comprehensive management of chronic periodontitis in high-risk patients such as smokers.

Initial Presentation

Medical History

The patient described her present health as “good,” although she recently began taking Lipitor® (Parke-Davis, Morris Plains, NJ) for what she described as “borderline” high cholesterol. There is no history of major illnesses; however, she reported taking Fioricet® (Novartis Pharmaceutical Corp., East Hanover, NJ) for “occasional bouts” of

migraines and Premarin® (Wyeth-Ayerst, Philadelphia, Pa) for hormone replacement. She reported an allergy only to sulphur drugs. Other than smoking, nothing was significant to her periodontal status or presented a contraindication for treatment.

Dental History

The patient reported a long history (more than 15 years) of failed periodontal care that included surgical interventions and 3-month periodontal maintenance schedules that alternated between the general dentist's and periodontist's offices. This may explain the hopelessness she communicated during the comprehensive periodontal examination. A referral to a periodontist was recommended but the patient refused this attempt to refer, which was entered in her progress notes. She complained of food getting trapped in her full-mouth crown-and-bridge restoration. A review of her dental history indicated that she had experienced so much bone loss around teeth Nos. 7 and 10 that it was necessary to extract the roots of both teeth. Because the clinical crowns remain as part of the fixed prosthesis, it actually appears that the entire teeth are still present (Figure 1). Review of the full-mouth radiograph (Figure 2) taken 9 months before our comprehensive examination was performed, shows the absence of the root structure of teeth Nos. 7 and 10. At the time our comprehensive periodontal examination was performed, tooth No. 15 was being considered for extraction because of loss of bone support.



Figure 1—Baseline clinical condition.

Progress notes indicate that approximately 4 years before the comprehensive periodontal examination, the patient was referred to specialists for treatment of a combined periodontal-endodontic lesion on tooth No. 9 and simultaneous extraction of the root of tooth No. 7.

Initial Clinical and Radiographic Findings

Extra- and intraoral examination revealed face, skin, eyes, nodes, temporomandibular joint, lips, labial and buccal mucosa, tongue, floor of mouth, salivary flow, hard palate, soft palate, and tonsillar region all within normal limits. The patient is in Class I occlusion with a 4-mm overjet and a 6-mm overbite, with fremitus present on teeth Nos. 4 through 13. Teeth Nos. 4 through 6, 8, 9, 11 through 13, 28, 29, and 31 have Class I mobility; teeth Nos. 23 through 26 have Class I+ mobility; tooth No. 15 has Class III mobility and a Grade II furcation involvement. Tooth No. 31 has Grade I furcation involvement.

The patient presented with generalized, mild-to-moderate supra- and subgingival plaque and calculus, with generalized, moderate inflammation. Note the plaque and calculus that has

Quick Tips

The Material Safety Data Sheets (MSDSs) maintained on hazardous products used in your office are considered a part of the employee medical record that has to be maintained for 30 years, plus the length of employment. Instead of maintaining the MSDSs for products you no longer use, OSHA allows you to simply maintain the product name, manufacturer, time period for which the product was used in the office, and the location of use. This information should be included on your chemical inventory list; so keep the list instead of all of those outdated MSDSs.

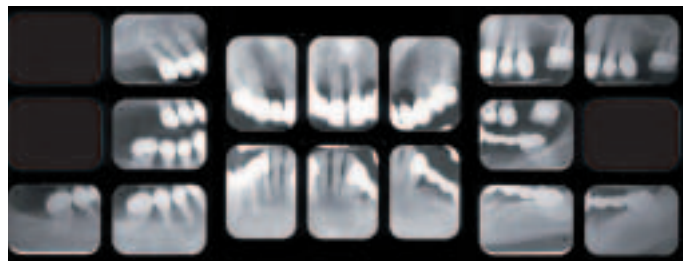


Figure 2—Full-mouth x-ray taken 9 months before treatment.

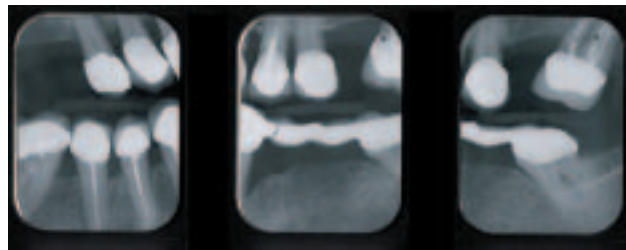


Figure 3—Bitewing x-rays 4.5 months post-treatment.



Figure 4—Clinical condition 4.5 months post-treatment.

Table 2—Comparison of Clinical End Points Pre- and Posttreatment

	PD < 4 mm	PD 4 mm-6 mm	PD ≥ 7 mm	BOPs	PD ≥ 5 mm w/BOP	Mean PD of Pockets 4-6 mm	Mean PD of Pockets ≥ 7 mm	Mean CAL of Pockets 4-6 mm	Mean CAL of Pockets ≥ 7 mm
Baseline	19%	62%	19%	74%	73%	4.9 mm	8.5 mm	6.6 mm	10.1 mm
4.5 Months Posttreatment	60%	38%	2%	5%	0%	4.5 mm	7.7 mm	6.5 mm	9 mm

CAL = calculated attachment gain; PD = pocket depth

accumulated around mandibular anterior teeth, and the accompanying fibrotic gingival inflammation (Figure 1). In addition, the presence of a McCall's festoon around the facial of tooth No. 27 is visible on the pre-treatment photograph. The amalgam restoration in this area may be iatrogenically contributing to the inflammatory change in the marginal tissue by increased plaque retention. Suppuration was present on probing at sites associated with teeth Nos. 4 through 6, 9, 12, 15, 25, 27 through 29, and 31. Eighty-four out of 114 sites had bleeding upon on probing (BOP).

Baseline measurements of 6 sites at each of the patient's 19 teeth were taken and recorded. Attachment levels were calculated from PD and recession measurements. Of all sites probed, 19% had PDs less than 4 mm and were considered shallow sites. Moderately deep pockets of 4 mm to 6 mm were found in 62% of the sites. Approximately 19% of the sites had PDs greater than 7 mm, which are classified as deep pockets. Average calculated attachment levels for shallow, moderate, and deep pockets as defined above were 4.4 mm, 6.6 mm, and 10.1 mm, respectively. These findings are summarized in Table 2.

Review of the full-mouth x-ray (FMX) taken 9 months before treatment (Figure 2) revealed generalized, severe horizontal bone loss with localized vertical defects either beginning or progressing at sites adjacent to teeth Nos. 4, 8, 9, 18, 22, 25, 28, and 29. Bone density around teeth Nos. 4, 26 through 29, and 31 appears to be diminished. Widened periodontal lig-

ament (PDL) space appears at teeth Nos. 4, 6, 8, 9, 11, 13, 15, 24, 25, and 29. Tooth No. 12 has a carious lesion on the mesial aspect.

Results of DNA-Probe Analysis and Sensitivity Testing

Intrapocket plaque sampling using paper points at six sites of six different teeth was performed and sent to a microbiological testing firm (Oral Microbiology Testing Service Laboratory; Department of Periodontology, Temple University, School of Dentistry; Philadelphia, Pa) 2 weeks before nonsurgical periodontal therapy. Results of the DNA-probe analysis indicated the following: high levels of *Porphyromonas gingivalis* that exceeded established threshold levels, levels of beta-hemolytic streptococci that indicate increased risk of disease, and high levels of *Bacteroides forsythus*.

Risk Assessment

As a smoker of 1 pack of cigarettes a day for more than 30 years, this patient presents with the risk factor with the strongest association to periodontal disease severity, calculated to be as high as 2.6 to 6 times greater risk in some studies.¹⁸ The patient also admitted to being under extreme levels of stress in her personal life.

Diagnosis/Prognosis

The patient's diagnosis included generalized advanced chronic periodontitis (case Type IV) complicated by smoking and exacerbated by occlusal trauma and possibly stress, and a defective restoration with caries on tooth No. 12. Her short-

term prognosis was questionable and her long-term prognosis was perhaps hopeless.

Prescribed Clinical Pathway Phase I Care: Halt Disease Progression

The patient was presented a treatment plan with therapeutic interventions aimed at halting disease progression. She was also presented with alternative treatment plans, including providing regular "cleanings" and "watching" her periodontal condition, or referral to another periodontist. It was explained that the treatment being recommended was intended to be only an "initial" therapy to halt disease progression, and referral for surgical reduction of PDs and/or repair of osseous defects might be necessary in the future. Because surgical interventions had failed in the past, the patient was encouraged to hear that there were nonsurgical options available. She consented to treatment, and was eager to begin.

Smoking Cessation and Stress Management

Although it was carefully explained to the patient that smoking was an established risk factor for periodontal disease, including its consequence relative to disease progression and how it would compromise therapeutic outcomes, the patient was not receptive to recommendations related to smoking cessation interventions at the time of active treatment.

Smoking cessation interventions that are reported to be successful include nicotine replacement therapy in the form of transdermal patches

and gum, the use of pharmaceuticals such as Zyban® (GlaxoSmithKline, Research Triangle Park, NC), reading self-help booklets and social support guides that teach habit-breaking techniques, signing stop-smoking contracts, listening to audio tapes with motivational or relaxation tips, obtaining tobacco cessation kits, calling toll-free hotlines for counseling, attending group support sessions, or getting individual counseling from allied health care providers such as nurses and dental hygienists, hypnotherapy, and professional help from psychologists and psychiatrists specialized in addiction counseling.³⁰⁻³³

The exact relationship of stress relative to periodontal disease progression is still under investigation.³⁴ Findings suggest that those who inadequately cope with financial strain may be at a higher risk of having more severe attachment loss.³⁴ Although further studies are needed to determine what might be the biological rationale for this relationship and to what degree modifying this risk through stress management counseling can be effective in halting periodontal disease progression,³⁴ counseling was recommended to the patient. She agreed at the time of treatment to consider this proposed interdisciplinary intervention.

Treating the Bacterial Challenge Systemic Antibiotics

Sensitivity testing is generally conducted to determine whether sampled organisms are susceptible or resistant to various antibiotic regimens that might be recommended for systemic antibacterial therapy of

continued on page 18

continued from page 17

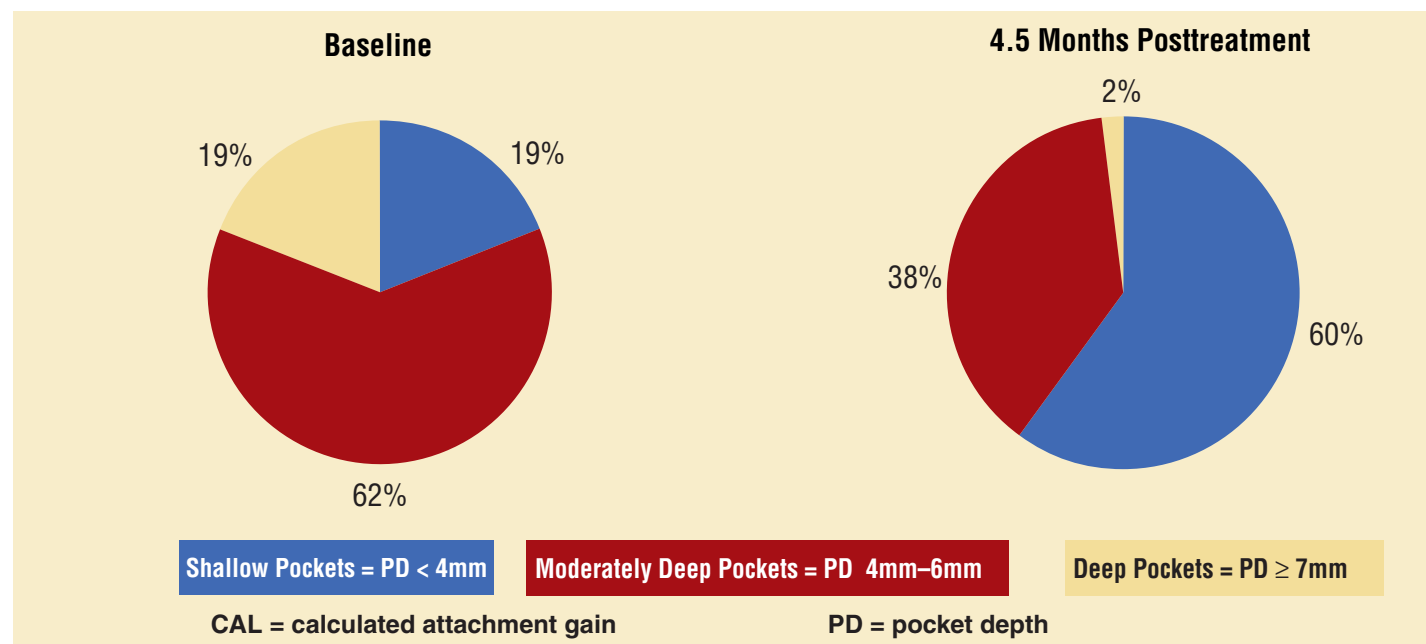


Figure 5—Illustration of patient's relative shift in disease severity.

chronic periodontitis.³⁵ The patient's microbiological report indicated the appropriate systemic regimen was azithromycin (500 mg/day for 4 days; taken as two 250 mg tablets 1 time/day). She was compliant with the antibiotic regimen and reported no side effects.

Thorough SRP

Instrumentation included the use of a piezo-ultrasonic device made by Satelec (Satelec, Inc, Mount Laurel, NJ). Research indicates that the most important factor related to optimal ultrasonic removal of the biofilm or plaque and calculus is the

configuration and size of the inserts.^{36,37} The thin size and configuration of Satelec's Gracey-like, micro-inserts allow excellent adaptation to curved root surfaces, flutes, furcations, and other areas that are relatively inaccessible, including the midproximal regions apical to contacts. Satelec recently added another line of extremely thin, curet-shaped diamond-coated micro-inserts. Their thinness allows highly sensitive tactile detection of subgingival deposits.³⁸ In addition, the diamond-coated surfaces of the blade on this new micro-insert allow removal of hard deposits with relative ease. The low power output setting recommended by the manufacturer makes it possible to remove residual, hard deposits without damaging soft tissues and cementum surfaces.

A list of other ultrasonic units currently sold in the United States is provided in Table 3. Gracey finishing curets, manufactured by Hu-Friedy (Hu-Friedy Manufacturing Company, Inc, Chicago, Ill), were used in final root planing.

Using a Local, Site-Specific Antibiotic for Deep Pockets

Atridox[®] was placed at all sites with PDs that were 5 mm or greater. No dressing was placed. This therapy was aimed at eliminating and/or reducing the number of periodontal pathogens within moderately deep and deep pockets. Atridox[®] delivers viable concentrations of doxycycline, greater than 100 times the minimum inhibitory concentration (MIC₉₀) for suspected periodontal pathogens, directly at the site and

maintains therapeutic levels for 7 to 10 days.³⁹ Additional clinical trials revealed a sustained reduction in periodontal pathogens 6 months postapplication.²⁸ Atridox[®] was approved by the FDA as a monotherapy (meaning that the average PD reduction of 0.8 mm to 1.4 mm and the average gain in calculated attachment levels of 0.6 mm to 0.8 mm achieved in the trials was accomplished as a stand-alone therapy without SRP).^{27,28} The clinical relevance of this is significant because it affords clinicians some level of assurance in that if they should fail to remove the critical mass of calculus at a deep site, PD reduction and calculated attachment gain are still likely to be achieved when this therapy is used. A recent study demonstrated a sort of "leveling" effect associated with the use of Atridox[®] in smokers. Smoking patients (and nonsmokers with a history of smoking) who received Atridox[®] therapy demonstrated gains in calculated attachment levels and reductions in PDs equivalent to nonsmokers. This finding helps "level the playing field" somewhat for smoking patients undergoing nonsurgical periodontal therapy.⁴⁰

Using a Local, Site-Specific Antibiotic for Shallow Pockets

An aqueous solution of 10% tetracycline (compounded by an independent apothecary), was irrigated subgingivally in this patient using a 23-gauge canula at all sites with PDs less than 5 mm. This antimicrobial irrigation, performed immediately after SRP, may have had a synergistic effect in promoting calculated attachment gain. The substantivity, anticol-

lagenase, and antimicrobial properties of tetracycline may explain the significant attachment gains (0.8 mm) reported in the literature when used as a subgingival irrigant in a 10% solution after a single session of SRP.^{41,42} Because of root surface affinity for tetracycline, its antimicrobial substantivity is great, even reported to be as long as 7 days after application as an irrigant in a 10% solution.⁴¹

One-Stage SRP

One-stage SRP, with local anesthetic (2% lidocaine with epinephrine) was performed within a 4-hour visit. This appointment time also included antimicrobial therapies and patient self-care training. The general findings offered by Quirymen et al⁴³ indicate that an average of 1 mm of PD reduction and calculated attachment gain is possible if instrumentation (with or without chlorhexidine disinfection) is completed within a 24-hour period. Although this is not the subject of this case presentation, use of this treatment model may have contributed to some extent to the dramatic improvement in posttreatment clinical parameters.

Self-Care Training

The patient's posttreatment self-care regimen incorporates the daily use of pulsating water irrigation (WaterPik[®], Water Pik Technologies, Fort Collins, Colo) directed at a 90-degree angle to the marginal gingiva. This home-care component of treatment may be yielding a trend in PD reduction and calculated attachment gain. Results of a recent study indicate that when daily oral irrigation was added to the routine self-care of moderate periodontitis patients, an average PD reduction of 0.4 mm and an average calculated attachment level gain of 0.5 mm was observed in as few as 7 days.⁴⁴ A decrease in markers of disease activity, such as interleukin 1 beta (IL-1 β) and prostaglandin E₂ (PGE₂), and an increase in IL-10, a beneficial cytokine, accompanied these clinical signs of improvement. Findings would seem to suggest that the therapeutic benefits related to use of irrigation in cases of chronic periodontitis can have more to do with the "specific host-microbiota alterations in the subgingival environment"⁴⁵ than the reduction of plaque.

Approximately 30 minutes of the 4-hour treatment appointment was dedicated to patient self-care training. Acquiring this included recom-

Table 3—Manufacturers of Ultrasonic Units Sold in the United States:

- Coltene/Whaledent Inc, Mahwah, NJ, (www.coltenewhaledent.com),
- DBI America/Amdent USA, Tampa, FL (www.amdentusa.com),
- Dentsply Professional, York, PA (www.professional.dentsply.com),
- Odonto-Wave, Fort Collins, CO (www.odontoson.com),
- Parkell Inc., Farmingdale, NY (www.parkell.com),
- Pro-Dentec, Batesville, AR (www.prodentec.com),
- Satelec, Mt. Laurel, NJ, (www.satelec.com)
- South East Instruments Corp, Gainesville, FL (www.autoscaler.com),
- Tony Riso Co. Inc, No. Miami Beach, FL (www.tonyriso.com).

mending using a powered toothbrush to prevent reinfection of the “freshly emptied” periodontal “niches” with pathogens that may be present on the patient’s old, used toothbrush⁴⁶ and frequent replacement of the brush heads.

Therapeutic Strategy for Modulation of the Host Response

The patient was placed on Periostat[®] (20 mg doxycycline hyclate) at the time SRP was performed. This coincided with a 1-week interval that followed her completion of the azithromycin regimen. The patient’s strict compliance to the directions related to Periostat[®] was excellent, probably owing to the time (about 15 minutes) spent communicating the value of its mechanism of action in slowing the rate of disease progression, and stressing the importance of absolute compliance. It was also explained that this chemotherapeutic therapy would likely be long term (risk for breakdown is 3 to 7 times greater for a smoker¹⁸), and would perhaps be indefinitely required to manage her periodontal condition.

Impressive data related to the improvement in clinical parameters achieved in phase III trials¹³ are recorded in Table 1. Yet, perhaps one of the most encouraging pieces of research regarding smoking-related chronic periodontitis is a 9-month phase IV study that was recently published.⁴⁷ Inclusion criteria in this study resulted in a subject population comprised of about 70% smokers. The study compared the addition of Periostat[®] to hygienist-delivered treatment that included full-mouth, supra- and subgingival debridement (within a 45-minute time frame) using ultrasonics and manual instruments, against the same treatment protocols with a placebo. At 8 months, it was found that patients who received Periostat[®] experienced an average of a 1.6 mm greater reduction in PD and an average of 0.8 mm greater gain in calculated attachment levels than those who received the placebo.⁴⁷

Reevaluation: 5 Weeks Posttreatment

The patient was seen again about 5 weeks after her nonsurgical periodontal therapy. Full-mouth probing was performed and recorded. Clinical improvements were seen in inflammation, BOP, and PD

indexes. The patient reported no root sensitivity, which usually accompanies root planing procedures, and was happy with her progress. Because Atridox[®] bio-adsorbs,²⁶ there was no residual material that needed to be removed. Light supragingival scaling and polishing with a low-abrasive pumice

was performed. Self-care instructions were reviewed and the patient reported strict compliance to the Periostat[®] regimen. She was reappointed for her first periodontal maintenance visit 11 weeks later, which would be about 4.5 months after the initial nonsurgical periodontal therapy.

First Periodontal Maintenance Visit: 4.5 Months Posttreatment

A bitewing survey was performed (Figure 3). Posttreatment clinical improvements were dramatic (Figure 4). PDs and recession were measured, gains or losses in calculated attachment

continued on page 20

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— Council on Scientific Affairs, ADA

† Values shown are the highest value for each product when compared to a negative control at study endpoint.

‡ Mean plaque buildup scores derived from Tinsley modification of Quigley-Hen Floss Index (study 1 and study 2, respectively): Listerine[®] (1.57, 2.22); floss (2.52, 2.77); control (2.52, 2.85). Mean gingivitis scores derived from Modified Gingival Index (study 1 and study 2, respectively): Listerine[®] (1.92, 1.79); floss (1.93, 1.92); control (2.07, 2.05).^{1,2}

§ p<0.001

|| p=0.134

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ment levels were determined, and BOPs were evaluated and recorded. Table 2 summarizes the differences in baseline and posttreatment outcomes.

PDs of 7 mm or greater at baseline with a reduction of at least 3 mm totaled 17/22 or 77%. The significance of this is that 77% of the pockets that were considered deep at baseline (≥ 7 mm) were reduced to moderate depths within 4.5 months posttreatment. The number of pockets ≥ 7 mm were reduced from 19% to only 2%, placing the patient in a more moderate disease category. The pockets with depths of 4 mm to 6 mm at baseline that showed a reduction of at least 3 mm totaled 14/71 or 20%. This shift in the severity of the disease is also represented by the reduction from 62% to 38% of the number of 4-mm to 6-mm pockets compared to baseline and 4.5 months posttreatment.

Another way to represent this data is in terms of relative shift in disease severity. Percentages of shallow, moderately deep, and deep PDs at baseline and posttreatment are illustrated in Figure 5. This may best represent the patient's trend toward a healthier periodontium.

Discussion

Table 1 demonstrates the contri-

butions to reduction in PD and gains in calculated attachment level associated with various treatment modalities and models. One-stage SRP, Atridox[®], and the use of a 10% tetracycline solution in shallow pockets, all have demonstrated an ability to enhance therapeutic outcomes when used adjunctively with SRP. However, it is critical to point out that these therapies are aimed at treating the microbial challenge, which, in the case of many patients with smoking-related chronic periodontitis, have failed to halt the progression of the disease. For this reason, it can be hypothesized that the dramatic results seen 4.5 months posttreatment in the case described in this article are primarily related to strategies that target modulation of the host response—namely the use of Periostat[®]. It is important to state that the therapies that address the bacterial challenge used in this patient's case must be implemented to ensure that the pocket ecosystem has the best opportunity for repopulation with beneficial bacteria. However, optimal therapeutic outcomes may only be possible by employing a treatment strategy that addresses the bacterial component, the host response component, and the risk component (namely smoking cessation), of this multifactorial disease entity.

Phase II therapy is designed to

sustain disease inactivity. This will likely be best accomplished by periodontal maintenance visits at 2-month intervals during the first year posttreatment. Assuming short-term therapeutic end points are sustained after this period, the frequency of periodontal visits may be adjusted to 3-month intervals. The patient has been advised to continue her present Periostat[®] regimen indefinitely and will be consistently urged to start smoking-cessation therapy and encouraged to seek help in learning coping skills for stress management.

In patients such as this with severe and extensive periodontal disease, therapy designed to be “initial” may become “definitive” if, as cotherapists, we can successfully manage their disease. Longer-term evidence of this would be resolution of osseous lesions, progress towards occlusal stability, and no detection of subgingival microbes from the red or orange complex of microbial species as determined by conducting new DNA-probe analyses. Should we start to “lose ground” again, this patient will be referred to a periodontist.

Conclusions

Research related to various adjunctive therapies and localized and systemic treatment is both relevant and applicable to the types of patients we see in everyday general practice. Disruption of the subgingival biofilm is still essential in long-term management of chronic periodontitis. However, we now know that there is an increased risk for the development of chronic periodontitis in patients who smoke because they have challenges with their host response independent of their microbial profile.^{9,16,18,23} Highly researched and scientifically validated treatment modalities and the use of the one-stage model for SRP promise predictable outcomes and should become components of a common clinical pathway. Given a certain level of technical competency in instrumentation, an adequate fund of knowledge related to risk assessment, risk modification, host-modulatory therapy, and the guidance of a defined clinical pathway, clinicians in general practice may predictably achieve optimal therapeutic outcomes in cases of smoking-related chronic periodontitis—outcomes that have previously eluded our best attempts at disease management.

Educating this patient on her risk susceptibility as illustrated in

Figure 6, her body's response by overproducing enzymes that destroy the collagen in her periodontium, and the threat of other systemic complications she faces as a result of her periodontal condition, has motivated her to “take charge” of her own condition. The other high note related to the success of this case is the professional encouragement it has given to clinicians who see this type of patient everyday. For hygienists (and dentists) who choose to practice at the periodontal therapist level, the proof of success in a case this compromised should be empowering. We might just be able to “get it right.” **COH**

Disclosure

The author has occasionally received honoraria from CollaGenix Pharmaceuticals, Inc, for lecturing.

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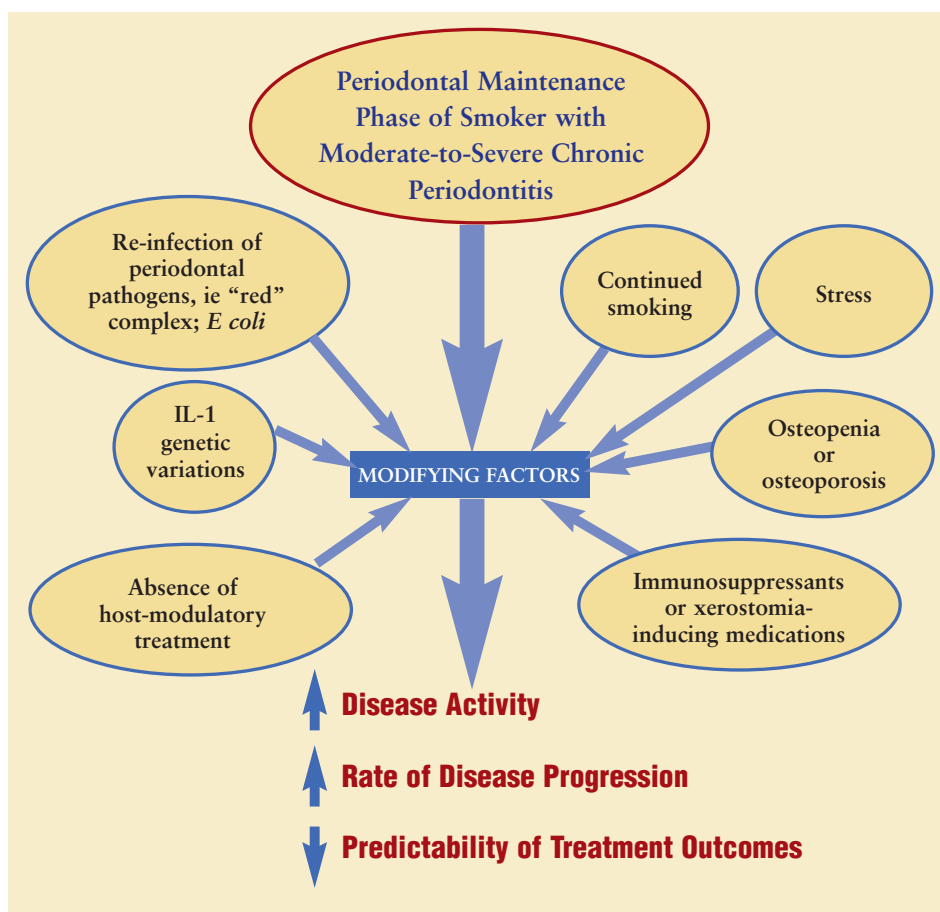


Figure 6—The risk susceptibility during periodontal maintenance phase for smoker.

QuickTips

Do not make your blood-borne pathogen exposure control plan too specific. Even if your employees are following what OSHA says is correct, if your policy manual says something else, you may be fined. For example, do not designate the brand name of surface disinfectant you are currently using, instead, state that you are using an EPA-registered tuberculocidal surface disinfectant. Then if you change brands in the middle of the year, you are still following your policy. (Hint: Make sure your disinfectant is EPA-registered to kill tuberculosis.)

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- The greatest promise for achieving therapeutic success appears to be in modalities that address:
 - salivary pH.
 - using fluoride toothpaste.
 - host response.
 - patient home care.
- Periostat® is the only FDA and ADA approved chemotherapeutic agent for host-modulatory therapy in suppression of the enzymes responsible for:
 - collagen degradation in the periodontium.
 - collagen production in the periodontium.
 - periodontal ligament formation.
 - cementum formation.
- What percentage of the US population is composed of smokers?
 - 10%
 - 25%
 - 50%
 - 60%
- What percentage of periodontal cases were attributable to smoking?
 - 18.7%
 - 29.3%
 - 41.9%
 - 53.4%
- Which of the following are considered compromised health trends associated with an aging population?
 - osteoporosis/osteopenia
 - acquired immune diseases
 - xerostomia-inducing medications
 - all of the above
- Smokers have a decreased wound-healing ability as a result of:
 - increased free radicals.
 - decreased free radicals.
 - impaired vascularization.
 - the synergistic effect of smoking on mitosis.
- One-stage SRP decreases the risk of recolonization of subgingival periopathogens via:
 - the meniscus phenomenon.
 - translocation of bacteria.
 - enzyme suppression.
 - promoting excessive hemorrhage.
- Prescribing the appropriate antibiotics is determined by:
 - DNA-probe analysis only.
 - sensitivity testing only.
 - DNA-probe analysis and sensitivity testing.
 - historic knowledge of appropriate antibiotics.
- The proposed clinical pathway in this case included:
 - comprehensive patient education in the etiological progression of periodontal disease and self-care training in plaque removal.
 - communication on the importance of compliance to medication regimens and periodontal maintenance visits.
 - all of the above
 - none of the above
- Atridox® has the ability to sustain a concentration greater than how much of the MIC for periodontal pathogens (6 mg/mL) for 7 days postapplication?
 - 10 times
 - 100 times
 - 1,000 times
 - 10,000 times