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How Far Can We Really Go with Nonsurgical Periodontal Therapeutics?

As the trend toward conservative, nonsurgical treatment of chronic periodontal disease continues, many of us are haunted by an important question, "Can we really sustain or enhance positive therapeutic outcomes in more compromised cases by using nonsurgical therapies alone?" Many general dentists, periodontal therapists, and periodontists will admit that they are often overwhelmed by the amount of research coming through the pipeline, some of which may report conflicting findings. At the heart of this tension is the age-old debate on the difference between research findings that are statistically significant and those that are clinically significant. Another way of asking this question is, "Do research findings that appear to be very small in effect really make a difference in what we do in everyday dentistry?"

At an American Academy of Periodontology meeting several years ago, a highly respected periodontist researcher/academician made a profound statement regarding statistical vs clinical significance that I think is extremely noteworthy—for a treatment modality to claim true clinical significance, the treatment has to demonstrate that it can sustain positive therapeutic outcomes over a very long period of time. The real test, he said, was whether the treatment modality under research would help the patient keep his teeth for the next 20 years. If so, he claimed, then you could say it was a treatment modality that had true clinical significance. However, the researcher also remarked that if we wait for 20 years before we make an absolute statement that the treatment modality is clinically significant, we risk robbing the public of valuable therapeutic solutions.

I think there is great wisdom in that observation. The real benchmark for claiming successful case management of chronic periodontitis may not be in just achieving, but perhaps more importantly, sustaining optimal therapeutic outcomes, such as decreased probing depths. This enables us to enact a long-term trend towards wellness. A demonstration of long-term optimal outcomes is especially challenging in the case management of patients who present with known risk factors for chronic periodontitis, which include smoking, diabetes, and the IL-1â polymorphism (genetic susceptibility).[1-4] As challenging as these kinds of cases may be, it is my experience that site-specific antimicrobials, microbiological monitoring, chairside genetic testing, sophisticated ultrasonic technologies, and host modulatory therapy provide us with an arsenal of valuable chemotherapeutics, screening tools, and leading edge mechanics that are positively impacting the level of periodontal care we can render.

In the case of smokers, we know they are at increased risk for the development of chronic periodontitis primarily because they have challenges with their immune (host) response which are independent of the quantity or composition of the bacterial plaque that exists in the oral cavity.[1, 5-7] In addition, it is well documented that both nonsurgical and surgical periodontal therapies are less effective in smokers and recurrence of disease is more likely in smokers after periodontal treatment.[8-11] What this really means is that only treating the microbial side of the disease just isn't enough. This is reflected in the recent consensus opinion of the American Academy of Periodontology, which recognized that host response is a key component of the etiology of periodontal diseases and provides an important rationale for treating the host response, in addition to conventional treatments aimed at suppression of bacterial infections.[12] Overlooking the need to modulate the host response by using subdose doxycycline, (Periostat®, CollaGenex Pharmaceuticals, Inc, Newtown, Pa, www.collagenex.com) may ultimately compromise our ability to sustain optimal therapeutic outcomes with smokers and other high risk patients.

> "Do research findings that appear to be very small in effect really make a difference in what we do in everyday dentistry?"

Case Study Revisited

In the January 2003 issue of *Contemporary Oral Hygiene*, I presented "Getting it Right" in the Long-Term Management of Smoking-Related Chronic Periodontitis," which was the case of a 59-year-old Caucasian woman who was diagnosed with generalized, advanced chronic periodontitis (Case Type IV).[13] This case was complicated by smoking and possibly high stress, in addition to being exacerbated by overcontoured crown and bridge and occlusal trauma.[13]

When I first saw this patient, she reported a long history of failed periodontal care that included osseous surgeries 15 years before and 3-month periodontal maintenance schedules that alternated between the dental hygienist at a general dentist's office and a periodontist. At the time of my initial visit with this patient, this case had very a questionable short-term prognosis and the long-term prognosis seemed hopeless. The patient had all but given up on the hopes of retaining the teeth she had remaining. She seemed very resigned to this fate and refused a referral to a periodontist for comprehensive re-evaluation.

The initial presentation of this patient follows[13]:

Initial Presentation[13]

Medical History[13]

At the initial visit, the patient described her present health as "good" although she recently began taking Lipitor® (Pfizer, New York, NY, www.pfizer.com) for what she described as "borderline" high cholesterol. There is no history of major illnesses; however, she reported taking Fioricet® (Novartis Pharmaceuticals Corporation, East Hanover, NJ, www.pharma.us.novartis.com) for "occasional bouts" of "migraines" and Premarin® (Wyeth®, Madison, NJ, www.wyeth.com) for hormone replacement. She reported an allergy only to sulfur drugs. Other than smoking, nothing else was significant to her periodontal status or presented a contraindication for treatment.

Dental History[13]

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 taken 9 months before the comprehensive examination was performed, shows the absence of the root structure of teeth Nos. 7 and 10 (Figure 2). At the time the comprehensive periodontal examination was performed, tooth No. 15 was being considered for extraction because of bone support loss.

Progress notes indicate that approximately 4

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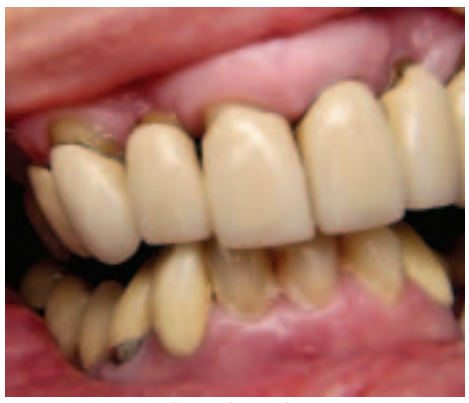

Figure 1—Baseline clinical presentation.

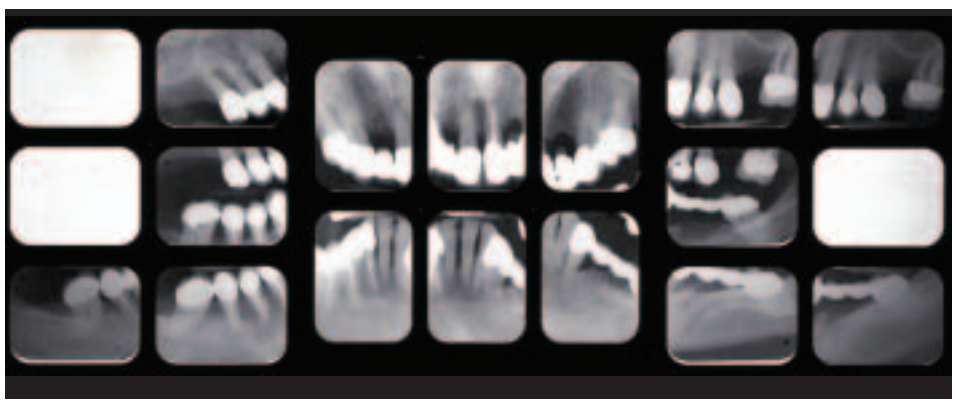

Figure 2—A review of the full-mouth x-ray. taken 9 months before treartment.

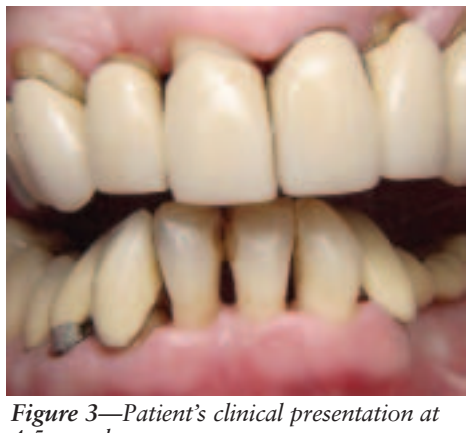

Figure 3—Patient's clinical presentation at 4.5 months posttreatment.

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 root extraction of tooth No. 7.

Initial Clinical and Radiographic Findings[13]

Extra- and intraoral examination revealed that the face, skin, eyes, nodes, temporomandibular joint, lips, labial and buccal mucosa, tongue, floor of mouth, salivary flow, hard palate, soft palate, and tonsillar region were all within normal limits. The patient is in Class I occlusion with 4 mm overjet and 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 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 pretreatment 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 bled on probing.

Baseline measurements of 6 sites at each of the patient's 19 teeth were taken and recorded. Attachment levels were calculated from pocket depth and recession measurements. Of all sites probed, 19% had pocket depths <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 probing depths >7 mm, which are classified as deep pockets.

A review of the full-mouth x-ray, taken 9 months before treatment, 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 (Figure 2). Bone density around teeth Nos. 4, 26 through 29, and 31 appears to be decreased. Widened periodontal ligament 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.

> The patient eagerly consented to treatment and definitive treatment was performed using a prescribed clinical pathway that targets the risk and microbial and nonmicrobial components of chronic periodontitis

Updated Risk Assessment and Prognosis at Initial Presentation

As a smoker of 1 pack of cigarettes a day for over 30 years, this patient presented 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 than nonsmokers in some studies.[1] This patient also presented with high levels of stress related to her husband's recent illnesses/surgery. Although it has not yet been conclusively determined that stress is a risk factor for chronic periodontitis, various models to evaluate its role in periodontal disease have been proposed.[14] More recent research findings seem to corroborate the theory that periodontitis patients with inadequate stress behavior strategies (defensive coping) are at greater risk for severe periodontal disease.[15]

Because this patient has a long history of failed periodontal care including osseous surgery, and she presents with advanced bone loss, infrabony defects, and multiple areas of pocket depths >7 mm, future bone loss seemed highly predictable.[16]

A subgingival intrapocket plaque sampling using paper points at 6 sites of 6 different teeth was performed and sent to an outside microbiological testing firm, Oral Microbiology Testing Service Laboratory at Temple University in Philadelphia, Pa, 2 weeks before nonsurgical periodontal therapy.[17] The results of the DNA-probe analysis indicated the following: high levels of *Porphyromonas gingivalis* that exceeded established threshold levels (*P. gingivalis* is present even in healthy oral tissue, but at a low level. Threshold levels refer to the value of microorganisms at a certain level where a psychological change will take place and there is a shift from health to periodontitis), levels of *beta-hemolytic streptococci* that indicate increased risk of disease and are often present in refractory cases, and high levels of *Tannerella forsythensis* (formerly *B. forsythus*). According to the research of Ximenez-Fyvie and colleagues, *P. gingivalis*, *T. forsythensis*, and *Treponema denticola* are organisms from the "red complex" species that are significantly more prevalent in plaque samples of subjects with periodontitis.[18]

Detectable levels of *P. gingivalis* have long been recognized to be elevated in periodontally active sites.[19] A more recent study examined the prevalence of *T. forsythensis* and *P. gingivalis* in subgingival plaque samples to assess the relationship of these bacteria with different categories of periodontal disease and health.[20] The study found that the distribution of bacteria differed in healthy and diseased sites. *T. forsythensis* was not detected in any sample from healthy sites, but was detected in 70% of sites with probing depth =5 mm and 100% of diseased sites with probing depth >5 mm. *P. gingivalis* was detected in only 1 sample from a healthy site, in 40% of sites with probing depth =5 mm, and in 90% of the sites with probing depth >5 mm. In addition, *T. forsythensis* and *P. gingivalis* were detected together in 30% of sites with probing depth =5 mm and 90% of sites with probing depth >5 mm. These findings seem to indicate that the specific combination of *T. forsythensis* and *P. gingivalis* may be associated with increased disease activity.

Other relevant research regarding *P. gingivalis* indicates that this particular organism is able to adhere to and invade the pocket epithelium, and that it is often transmitted between spouses.[21,22] Some important research findings may implicate a potential role for *P. gingivalis* in several steps involved in atherosclerotic lesion formation.[23] This may mean that the presence of this organism may have a serious systemic implication.

The PST® Genetic Test (Interleukin Genetics, Inc, Waltham, Mass, www.ilgenetics.com/Kimball Genet ics Inc, Denver, CO, www.kimballgenetics.com), which is a buccal

swab that can be performed chairside, was not performed on this patient. Had this patient tested PST-positive for the genotype (IL-1 polymorphism), the multifactoral risk posed by the combination of genetic susceptibility, high levels of certain periodontal pathogens from the red complex (as was found in this patient), and smoking would have significantly increased her risk of disease severity and therapeutic response to periodontal treatment.[24-26]

Definitive Care Using a Prescribed Clinical Pathway

The patient was presented with the optimal and 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, which followed a prescribed clinical pathway discussed below, was intended to be only an initial therapy to halt disease progression. The patient was also advised that because of the interference of smoking with treatment response, she was a poor surgical risk, but referral to a periodontist might still be necessary in the future.[7,27] The patient eagerly consented to treatment and definitive treatment was performed using a prescribed clinical pathway that targets the risk and microbial and nonmicrobial components of chronic periodontitis as described below.

- Risk elimination/modification. Recommendations for smoking cessation and stress management therapy.[28-30]
- Mechanical treatment. One-stage scaling and root planing with Satelec Ultrasonics (Satelec Inc U.S.A./Groupe ACTEON, Mount Laurel, NJ, www.satelecusa.com) manual instrumentation for finishing with Langer curettes.[31]
- Antimicrobial treatment. In office subgingival irrigation (tetracycline @ 10% solution), placement of site-specific antimicrobial, Atridox® (CollaGenex Pharmaceuticals, Inc, Newtown, Pa, www.collagenex.com) at all sites =5 mm with bleeding on probing.[32] Azithromycin 500 mg a day for 4 days as determined by DNA and sensitivity testing.[33-36]
- Patient self-care training. Training in the use of an automatic toothbrush interdental cleaning, home irrigation with the Waterpik® Dental Systems (Waterpik Technologies, Inc, Newport Beach, Calif, www.waterpik.com) and mild abrasive dentifrices.[37,38]
- Removal of iatrogenic factors. The patient was advised that she needed a new comprehensive restorative treatment plan, but it was recommended that she not proceed with restorative treatment until periodontal stability is achieved. She was also advised that smoking appears to be a negative risk factor for successful treatment of peri-implantitis, and that she was not a candidate for implant placement.[39]
- Host modulation. Continued, long-term regimen of Periostat.[40-44]

For further information on the clinical pathway that was followed on this patient and evidence based rationale for the treatment that was recommended, readers are referred to the original case study.[13]

The patient was re-evaluated 6 weeks after definitive care was performed and good healing of the soft-tissues was noted with no complications of therapy.

4.5 Months Post-Treatment Results

As was presented in the original case study, the clinical end points achieved at 4.5 months post-treatment compared to baseline clinical data showed rather dramatic improvements in all clinical parameters, including pocket depth reduction, reduction in bleeding on probing, the number of pockets = 5mm which also bled on probing, and clinical attachment gains. Figure 3 is the patient's clinical presentation at 4.5 months post-treatment.

A 2-month interval for periodontal maintenance was recommended for the first year to sustain disease inactivity in such a high-risk patient. It was explained to this patient that if short-term therapeutic outcomes were sustained after this period, the frequency of periodontal visits could be adjusted to 3-month intervals. Because of various complications related to her husband's illness, the patient was only able to commit to a 3-month interval. She complied with this periodontal maintenance schedule until her 5th visit (20 months post-treatment), at which time she was 3 months overdue. She was still smoking at that visit, and because she had forgotten to call for a prescription refill on her Periostat, 3 months had passed since she had taken the drug.

Conclusion

The purpose of updating this case study (which follows on page TK) is to compare the patient's clinical data 20 months after treatment with baseline clinical data and to determine whether the use of Periostat was as effective as the research claims in sustaining (and enhancing) positive therapeutic outcomes. And, finally, to address the haunting question, "Can positive therapeutic outcomes in risk-associated chronic periodontitis be sustained nonsurgically?" COH

Disclosure

The author has occasionally received honoraria from CollaGenex Pharmaceuticals, Inc to lecture and to write articles.

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