Deliberations on Non-Surgical Periodontal Therapy

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Abstract

Non-surgical periodontal therapy remains the gold standard for resolution of dental plaque biofilm induced oral disease. This therapy involves patient oral home care on a daily basis for success. In incidents of NSPT failure, more than just patient compliance should be considered. Periodontal disease is a multi-factorial disease and needs to be considered in multi-modal therapeutic regimens. Practitioner's deliberations around failures after NSPT ought to evaluate the very nature of disease, the biology inherent in tooth to gum interactions, and the rather limited means of addressing such factors.

Keywords: Nonsurgical periodontal therapy; Failed NSPT; Diabetes in NSPT; Smoking with NSPT; NSPT outcomes; Antimicrobial therapy and NSPT

Introduction

This paper is written to stimulate practitioners' thinking upon evaluating failed outcomes in non-surgical periodontal therapy (NSPT). As practitioners we aim to reduce tooth loss and overall function with measures including prevention, maintenance through repair, replacement of missing teeth and many other heroic efforts that could be patient or practitioner driven. A large percentage of patient requests are for cosmetic reasons, function or for reduction of pain. As is expected practitioners evaluate each patient independently and configure treatment plans that consider cost, benefit, satisfaction, longevity, and biological science behind such measures. Practitioners usually consider patient compliance to treatment provided and the oral hygiene efforts necessary to ensure success. However, even the best intentions in a properly laid out treatment plan may result in failure. Periodontists and dental hygienists have to face this fact quite often. As periodontal disease is multifactorial, we as knowledgeable practitioners should be cognizant of multimodal therapies available for use. The ultimate challenge comes from prediction of the future outcome with the correct mode or modes of treatment/therapy. Regardless of treatment modes and patient intention, the biologic outcomes inherent in the integrity of the oral cavity will prevail. Humankind's greatest wish to bend nature to his will fails miserably when it involves a multifactorial disease with the limited selection of multimodal treatments. Prevention of disease and disease factors would serve humankind imminently better than all the therapy we can dispense at this point.

Literature Review

Classifications of dental root furcation

A furcation defect is alveolar bone loss because of periodontal disease that affects the trunk of the root in a tooth having two or more roots [1]. When carrying out a diagnosis and drawing up a plan for treatment, the shape and the magnitude of the furcation are major factors. The furcation itself is designated according to the passage of a furcation probe through and through, (Class III), part-way through, (Class II), or just presenting as a pronounced curvature or fluting between the roots (Class I). As imagined, for such access between the roots of a multirooted tooth, a fair amount of alveolar bone has become unavailable for firm attachment of the tooth.

Non-invasive treatment

Patient homecare: While periodontal disease is initiated by dental plaque biofilm and patients are not highly skilled in mechanical plaque removal, invoking perfection in home care is marked for failure. Reliance on professional dental hygiene care that includes supra and sub-gingival debridement via non-surgical periodontal therapy (NSPT) is almost universally indicated to sustain long-term stability of the periodontium [2]. The effectiveness of NSPT has been proved over many years and despite the advancements in periodontal surgical therapy, it is still considered the gold standard for dental plaque biofilm associated periodontal infections.

Patient compliance: Successful NSPT is based on patient compliance [3-5] and personal plaque control [6-9]. Incorporation of these aspects of maintenance improves the odds of long term success of the periodontal therapy. However, even university-based studies frequently have a high percentage of noncompliance problems with regards to dental hygiene [3,10] and maintenance visits [11]. Similarly, in private practice, it is often difficult to keep patients motivated. Wilson and co-workers reported that during an 8-year period in a

periodontal practice, only 16% of the treated patients were good compliers; 49% were erratic; and 34% were poor compliers [12]. Smoking habits are also related to patient compliance and will hamper the effect of non-surgical therapy making the patient more susceptible to disease progression.

Smoking cessation: One minimal invasion treatment is that of patient tobacco cessation. Smoking is one of the strongest modifiable risk factors for developing periodontal disease [13]. According to Johnson and Guthmiller it increases the odds of developing periodontal disease 2 to 8 -fold, depending on the definition of disease severity and smoking dose. The exact mechanism of smoking action is still unknown however several mechanisms for negative periodontal effects of smoking are proposed which include decreased Ig G2 production [14], chronic reduction in blood flow and vascularity [15], increased prevalence of potential periodontal pathogens [16], shift in neutrophil function towards destructive activities [17], negative effects on cytokine and growth factor production [18], and inhibition of fibroblast growth, attachment and collagen production [19]. These mechanisms in one way or another, makes individuals more susceptible to periodontal disease and compromise the healing response. The response to nonsurgical therapy in smokers is inferior to non-smokers and much less pocket reduction and/or gain in attachment can be noted in smokers. It is encouraging though, that a good response to nonsurgical therapy can be achieved in the smoker if they quit smoking [13,20]. From a clinical aspect it is important to include smoking cessation in the treatment plan of smokers.

Many studies show the prevalence of periodontitis in smokers. These studies allow one to assume that upon cessation of tobacco products, healthy outcomes are not far behind. It is believed that within 5 years, a smoker begins to attain the benefits of not smoking [21]. It is rather presumptuous to believe that just stopping smoking a while before and quitting completely after periodontal therapy, oral tissue can magically restore itself to the level of a non-smoker. If body tissues such as the lungs, take over 5 years to heal to the point of showing less damage on a chest X-ray, why do we expect instant healing with improved periodontal attachment when the patient quits? Consideration must also be made for the possibility that human neutrophils have altered themselves according to the onslaught of tobacco use and will forever remain in that state, or take a few years to adjust to the new healthier lifestyle. Newer studies are needed in this arena.

Glycemic control: The presence of systemic conditions such as diabetes mellitus has been linked as a factor in periodontal disease. The presence of diabetes has also been seen to affect the outcome of nonsurgical therapy in a negative manner [22]. In the diabetic patient, neutrophil function and collagen metabolism is altered which increases the susceptibility of periodontal infection. The short and long term response to periodontal therapy in well controlled diabetics with regular maintenance therapy is as good as non-diabetic patients [23]. However, long-term response to nonsurgical periodontal therapy in a poorly controlled diabetic patient would result in

rapid recurrence of the original deep pockets [24]. Hence, by maintaining good oral hygiene and keeping blood sugar level in check, failure of nonsurgical periodontal therapy can be prevented. More recent evidence suggests that diabetes and periodontitis have a two-way effect which means that untreated periodontal therapy will also affect the diabetic's control on sugars [25].

Body art: Although there are no references to piercings of the lip or tongue contiguous with periodontal disease, gingival recession in anterior teeth is mentioned by Chambrone [26] in that context. As we are talking about furca in this paper, such a reference may not be as pertinent. However, with the more creative kinds of body art in society, piercings in the posterior of the tongue or even through and through buccal mucosa piercings may not be that far-fetched. As with the anterior lip piercing, recession could be caused on the buccal or lingual surfaces of posterior teeth. Recessions plus inadequate plaque biofilm control could support the recommendation to the patient of removal of the jewellery.

Non-surgical periodontal therapy

In dentistry, non-surgical periodontal therapy (NSPT) is recommended in patients with periodontal disease. Such therapy is carried out by periodontists, general dentists and dental hygienists. Although considered the gold standard for dental biofilm associated periodontal disease, if the conditions mentioned above remain, NSPT may be doomed to fail. The failures of NSPT in the patient who does not have or carries out recommended activities as above may indicate factors pertaining to the actual tooth or teeth concerned. For instance, location of the tooth, number of roots, space available inter-proximally, manual dexterity of the patient, and even the genetics of the patient of concern.

Single rooted versus multi-rooted teeth: As in the incidence of a lip piercing, only a single-rooted tooth may be involved. These teeth respond better to nonsurgical therapy and show more pocket depth reduction and gain in clinical attachment as compared to multi-rooted teeth. In multi-rooted teeth, factors such as furcation involvement, presence of plaque at tooth level [27], visibility, and access can affect the result of nonsurgical therapy. Root surface debridement under higher magnification with mini-curettes plus the use of headlight might help to overcome the access and visibility in posterior multi-rooted teeth [28].

Initial probing pocket depth: Non-surgical therapy in teeth with deeper initial probing pocket depth may fail. Evidence suggests that the rate of failure increases when the initial probing pocket depth is more than 6 mm. Several reasons are proposed for this failure and they are 1) the amount of residual calculus being dependent on the size of the surface to be scaled; 2) with deeper pockets more irregularities are observed on the tooth surface; and 3) in deeper pockets the apical portion of the pocket is narrower making accessibility to the bottom of the pocket difficult with complete removal of calculus unlikely [29]. However, in an early paper, Badersten et al. [30], NSPT showed some improvement in clinical attachment even in sites with probing pocket depth more than

7 mm. On the other hand, over the long-term there is a possibility of further attachment loss in the sites with deep residual probing depths particularly if combined with bleeding on probing [31].

Experienced versus non-experienced operators: Operator experience can also influence the treatment outcome of nonsurgical therapy. Continuous training and practice provides knowledge and proficiency [29]. Therefore, in cases of nonsurgical therapy, experienced operators would have more knowledge of root surface anatomy, manual dexterity and proper instrument selection and application than the inexperienced one. Evidence suggests that nonsurgical therapy performed by an experienced operator shows better results in moderate or deep probing depth pocketed teeth than the inexperienced operator [32]. The differences in effectiveness of nonsurgical therapy in moderate or deep pockets were explained by the presence of residual calculus. Studies showed that an experienced operator produced fewer root surfaces bearing residual calculus than a less experienced one [27], and that the clinical success or failure of scaling and root planning may be dependent on the critical mass of residual calculus rather than total elimination of calculus [31].

Teeth with and teeth without furcation involvement: Attachment loss in the furcation area of multi-rooted teeth provides an ideal environment for highly virulent anaerobic bacteria to grow. Even after sub-gingival debridement the furcation sites in multi-rooted teeth showed higher microbial counts and greater proportions of suspected microbes [33]. Loos et al. evaluated changes in the subgingival microbial flora of molars with and without furcation lesions following scaling and root planning [33]. At 12 months post therapy, reductions in spirochetes and *P. gingivalis* were significantly less for molars with furcation lesions compared to those without such defects. The furcal anatomy and lack of access of the furcation area not only makes patient-performed home care and professionally performed sub-gingival debridement difficult but also provides sheltered anatomic sites for virulent peridontopathogens [34]. These observations explained the persistence of a pathogenic microbial flora after scaling and root debridement and the less or un-responsive nature of nonsurgical therapy in the furcation.

The initial probing depth of furcation site might also affect the response of nonsurgical therapy in that furcation site. Norland et al. [35], after monitoring the healing response for 24 months in non-molar sites, molar flat surface sites, and molar furcation sites after nonsurgical therapy, found that the frequency of attachment loss in the furcation area with initial probing depth of 4 mm was higher than non-molar sites or molar flat surface sites. The frequency of attachment loss was 3 times higher in those than in sites with initial probing pocket depth of more than 7 mm.

Refractory periodontitis: Refractory periodontitis could be another name for failed initial NSPT because it does not respond to nonsurgical periodontal therapy. In the 1989 classification, refractory periodontitis was categorized as a separate entity but later in the 1999 Armitage [36] classification it was excluded because of the diversity of clinical conditions and treatment under which periodontal therapy fails to arrest the progression of periodontitis. However the Armitage group proposed that "refractory" could be applied to all forms of periodontitis which despite excellent patient compliance and well-executed therapy still failed to arrest the progression of disease.

In refractory cases, it is possible that the host and parasite interaction is altered by genetic factors and/or prolonged by exogenous factors such as smoking [37]. About 90% of refractory cases in the Macfarlane study [38] were smokers which suggest a possibility of smoking having had a role in the pathogenesis of the refractory cases. Some authors also suggested that the differences in sub-gingival microbiota as a possible reason for refractory cases [38-42]. The exact pathogenesis of refractory cases is still unknown however more recent research on refractory cases tells us that it is not related with systemic disease, inadequate debridement and/or patient compliance [37]. Clinically it is important to know that refractory cases may then not be related to the factors mentioned earlier as it will help the clinician in diagnosing and planning the future course of treatment of refractory cases. Clinical refractory cases may present with the following features [38].

- Multiple sites show clinically detectable disease progression.
- Disease progression is unrelated to previous severity which means progression occurs even in sites of minimal or no previous disease.
- Disease progression is not stopped by conventional periodontal therapy and regular supportive care.

Host modulation therapy: Refractory cases are generalized non-responsive cases and remain a challenge to any periodontist. The treatment modalities proposed for refractory cases are broadly based on either host modulation therapy or on antimicrobial therapy. Host modulation therapy is a means of treating the host in the host-bacterial interaction [43]. In periodontitis, the body responds to bacterial challenge with an elevated inflammatory response wherein tissue break down occurs, leading to the clinical sign of periodontitis. Host modulation therapy offers the potential for down-regulating the destruction aspects and up-regulating protective aspects of the host response to the bacterial challenge to enhance the opportunities for wound healing and periodontal stability [44]. Sub-antimicrobial-dose doxycycline (SDD) has an anti-collagenolytic action for which the adjunct use of it with nonsurgical therapy was proposed in the treatment of aggressive, severe chronic and refractory periodontitis cases. Ryan et al. [43] suggested that 20 mg of SDD used as an adjunct to non-surgical therapy could be helpful in long-term maintenance of refractory, aggressive and severe chronic periodontitis cases.

Host modulation could also be effective in smokers and in this regard the sub-antimicrobial dose of doxycycline is of importance. A sub-antimicrobial dose of doxycycline has anti-collagenolytic activity [44-49] and therefore provides the rationale for its use in smokers. Evidence suggests that adjunctive use of SDD along with nonsurgical therapy could

provide results equivalent to non-smoker at least for 9 months. However long-term benefit can also be achieved if the patient stops smoking. Another unique treatment regime such as sequencing host modulation therapy following systemic antimicrobial therapy may offer clinicians and patients new therapeutic options that address both the microbial and the host response alterations that are evident in smokers [49]. However, care must be taken in prescribing systemic antimicrobials as resistance to them is rising [50]. Host modulation therapy has shown some promise but the evidence is not conclusive as yet and requires more studies with longer duration follow up. The most therapeutic approaches to the management of refractory cases of periodontitis center round the use of antimicrobials to aggressively reduce the bacterial challenge below the threshold of these apparently compromised patients [22].

Antimicrobial therapy: Adjunctive systemic antimicrobial therapy along with nonsurgical periodontal therapy is probably the most effective way to manage refractory cases [44]. Mechanical periodontal therapy disrupts the bacterial biofilm and enhances antimicrobial effect of antibiotics so it may be productive to use antibiotics as an adjunct to mechanical therapy in generalized non-responsive cases [45]. The systemic antibiotics including clindamycin [46], Augmentin [41], tetracycline [39], metronidazole [47] are found to be effective in treating refractory cases. Periodontitis being an infectious disease caused by groups of bacteria acting alongside the immune system cannot kill all the bacteria; therefore, a combination of amoxicillin and metronidazole has been proposed to treat refractory cases by Herrera et al. [44].

Winkel [48] and Flemmig [49] suggested that the use of amoxicillin and metronidazole with mechanical debridement shows improvement in generalized refractory cases. However, even with adjunctive antimicrobial therapy along with mechanical therapy, failure might occur due to inadequate personal oral hygiene [37]. Considering the role of antimicrobial therapy, mechanical debridement and the role of oral hygiene the following guidelines were suggested by Kornman [37] in the therapy of refractory periodontitis.

- Determine the microbial profile of the severely involved sites.
- Treat with appropriate systemic antibiotics and establish good plaque control.
- Confirm elimination of the target bacteria.
- If the target bacteria have been successfully eliminated, place the patient on supportive periodontal therapy and re-monitor the bacterial profiles annually.
- If the target bacteria have not been eliminated retreat with other antibiotics and reinforce plaque control.
- Control systemic risk factors where possible.
- If the patient has a history of frequent antibiotic exposure for any other reasons, the therapy should consider the possibility of enteric microorganisms and/or antibiotic resistance in the normal periodontal microbiota.

Points to ponder: As dental practitioners, we have learned that it is difficult to prove cause and effect in scientific studies.

Statistical analysis and control of extraneous factors can whittle away at our collective scientific natures in experimental studies. As we are practitioners of therapy based on scientific evidence, why are we casting about knowing there is no 1:1 cause and effect and are still delivering only 1 therapy at a time. If there are multiple factors involved, why not hit them all in one swoop rather than trying one or two and when they fail try another one or two therapies? If we have scientific studies on all the different ways to come at a problem why only select one and doom the tooth to failure?

In the wild, mammals that have tooth problems lose teeth [51,52]. If they lose too many they die. In man, there may be some specific truth to the loss of one tooth being a saving grace to the rest of the dentition. By losing a tooth, one might be removing the nidus of infection or allowing for better cleansing of the general area with toothbrushes or motions of the tongue and chewing with the rest of the firm teeth.

Teeth and their attachments are very fragile interfaces. Having a tooth in the mouth predisposes us to getting caries or periodontal disease. It is a miraculous feat to see so many teeth in so many mouths existing for such long periods of time. The innate immune system co-exists and responds to many assaults in the digestive tube from source to exit with relatively few exacerbations of disease. The real miracle would be to be able to harness the effects and use it in instances of periodontal disease, irritable bowel syndrome and the myriad of other immune diseases of mankind.

Discussion

Suggestions for management post NSPT

The gold standard for periodontitis remains as meticulous NSPT. The gold standard for a substantive therapy post-operative to NSPT is Chlorhexidine rinses or swabs. Meticulous homecare performed by the patient is mandatory and should be entered into prior to NSPT with re-evaluations at each subsequent maintenance visit. The literature is rife with suggestions for maintaining the results of successful NSPT. Additionally, there are thousands of articles and textbooks written on surgical processes to enhance homecare efforts, or regain esthetics in patients presenting with periodontitis. There are many success stories in print and very few failures actually reported in the research. The reality is that not all NSPT's are successful. We need to recognize the multifactorial nature of the disease and apply multimodal therapy for good outcomes.

Conclusion

Dental practitioners, be they dentists, specialists, or dental hygienists, all want the best outcomes on every single patient, every single time. The patients' themselves want the same. It is difficult to accept the fact that not every therapy will be a success story. However, it means that to be credible practitioners practicing evidence based dentistry, we need to

support lifelong learning and keep practicing. Maybe there will be a magic bullet for periodontal disease eventually.

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