Pockets, pus and periodontitis: Non-surgical treatment strategies

By Michael Danesh-Meyer BDS, MDS (Perio)

Periodontitis is a chronic inflammatory disease of the periodontium that involves the gingival tissues and bone around the tooth roots. The progression of periodontitis is associated with the presence of a subgingival plaque biofilm and subsequent clinical attachment loss and pocket formation (Figure 1) due to destruction of the periodontal ligament attachment and associated alveolar bone.

Traditionally, the treatment of periodontitis has involved a number of different therapeutic strategies aimed principally at eradication of periodontal pathogens. These strategies have involved both surgical and non-surgical approaches to management depending on the severity of disease. Both surgical and non-surgical debridement results in clinical attachment gains and reduction in pocket depths. In shallow to moderate pockets, a non-surgical approach can be as effective as a surgical approach, with deeper pockets (>6mm) responding better following surgical therapy with greater probing depth reduction and clinical attachment gains.[1]

Our current understanding of periodontal disease recognizes that while periodontal pathogens are necessary to initiate disease, the extent and severity of tissue destruction is also related to the host response-microbial interactions. This has lead to new research looking specifically into how we may modulate the host response to ultimately reduce tissue destruction and disease progression.

Systemic antibiotics

Systemic antibiotics enter the gingival sulcus by transudation through the crevicular and junctional epithelium from the serum. However, the concentration of the antibiotic can often be so low as to be ineffective unless the bacterial biofilm is also disrupted through mechanical debridement. This highlights the need to undertake effective mechanical treatment (scaling, root planning) in conjunction with the use of systemic antibiotic therapy for the management of periodontitis. Thus, systemic antibiotics should not really be provided in isolation for the management of chronic periodontitis.

A number of different systemic antibiotic classes have been recommend as adjuncts to mechanical debridement of periodontal pockets (Table 1).

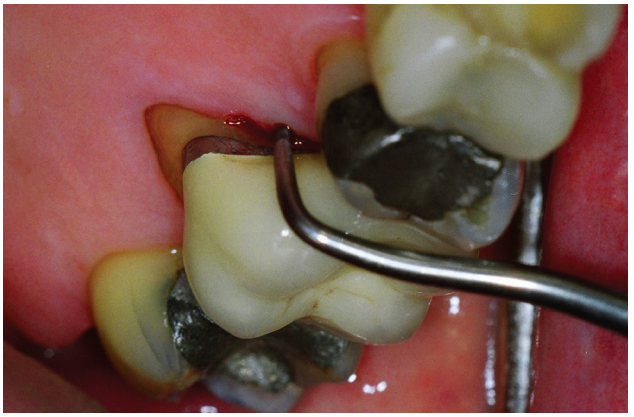

Figure 1. A deep periodontal pocket related to mesiopalatal furcation involvement.

One of the more effective antibiotic regimes is the use of a combination therapy of Amoxycilin and Metronidizole as proposed by Winkel etal.[2] The recommended dose being 250 mg of each three times daily for 8 days. If the patient has sensitivity to penicillin, then a combination of Clindamycin and Metronidizole can also be very effective.[3]

Recent data also supports the use of Azithromycin in the management of periodontitis in combination with mechanical debridement. This particular antibiotic has the advantage of being therapeutic at a low dose. Azithromycin has also been shown to reduce bleeding on probing and increase the rate of wound healing.[4,5]

Local antibiotic therapy

Problems associated with the use of systemic antibiotics in the management of periodontitis include: risk of antibiotic sensitivity; increasing bacterial resistance to antibiotic medications; gastrointestinal side effects; and low therapeutic dose reaching the target "organ" i.e. the periodontal pocket. This has lead to interest in delivering local antibiotic treatment, directly into the periodontal pocket. One of the first attempts in this regard was the use of Actisite™, which consisted of fine fiber willed with tetracycline. These fibers were inserted into the pockets for around

10 days. While Actisite™ was found to be effective, it was also labour intensive, which ultimately led to the search for a resorbable delivery system.

Atridox™ (Atrix Laboratories) was a flowable gel containing Doxycycline that could be "injected" into the periodontal pocket were it would flow and adapt around the root, setting and then allowing the release of the Doxycycline to the surrounding tissues. The other big advantage was that Atridox was resorbable. One of the disadvantages of this material is that it sets quite hard and can result in irritation and inflammation in the periodontal pocket.

Aristin™ (OraPharma) is minocycline incorporated into tiny bioabsorable polymer microspheres. When delivered into the periodontal pocket, it maintains a therapeutic concentration within the pocket for up to 14 days.

A recent meta-analysis of over 50 published papers on the subject of local delivery of antibiotics suggested that while overall treatment results were favorable, the differences were minimal when compared to scaling and root planning alone.[6] Such findings as well as difficulty in access to these products due to limited distribution and availability have meant that their use has not become widespread among clinicians.

Table 1. Systemic antibiotic classes

Penicillin	-	Augmentin, Amoxycilin
Tetracycline	-	Tetracycline, Minocycline, Doxycycline
Quinolone	-	Ciprofloxacin
Lincomycin	-	Clindomycin
Nitroimidazle	-	Metronidazole
Macrolide	-	Azithromycin

Antiseptics

The use of chemical agents with anti-plaque action as adjuncts to periodontal therapy appears to be of limited value. This is due principally to the inability of such mouthwashes to penetrate into the periodontal pocket. While oral antiseptics such as Phenolic compounds (Listerine™), Chorhexidine gluconate, Triclosan and cetylpyridium chloride can be useful in reducing supragingival plaque and gingivitis, they are ineffective in the management of periodontal pockets.

Anti-inflammatory medications

In recent years, research has shed more light on the important role that the host immune response and inflammatory response to microbiological challenge mediates tissue damage. This has resulted in considerable interest in the potential use of modulating agents as adjunctive therapy in managing periodontitis. In general terms, there are three main areas of investigation: anti-inflammatory agents; anti-proteinases; and anti-resorptive agents.

Non-steroidal anti-inflammatory drugs (NSAIDs) are known to inhibit prostanoid formation and have thus been the source of much interest as inhibitors of the host immune response in periodontal disease. Prostanoids produced during activation of the cyclooxygenase pathway during periodontal disease have been associated with tissue destruction and bone loss. Research suggests that selective NSAIDs (COX-2 inhibitors) may reduce bone loss associated with periodontal disease.[7] A recent review concluded that the use of NSAIDs in conjunction with mechanical therapy helped with bone maintenance in patients treated for periodontitis.[8]

The use of low dose doxycycline has also been advocated in modulating host response due to its ability to inhibit collagenolytic activity and thus mitigate tissue destruction leading to clinical attachment loss and pocket formation. This sub antimicrobial dose of doxycycline gets around the concerns regarding the development of antibiotic resistance and in one study was shown to have a statistically significant benefit on clinical attachment levels and probing depth when used in combination with scaling and root planning.[9]

Future directions

At present, scaling and root planing remain central to the non-surgical management of periodontitis. Through the removal of pathogenic bacteria and mineralized deposit; disruption of the biofilm; and provision of a clean root surface, scaling and root planning can initiate a healing response which can result in new clinical attachment and pocket depth reduction. Future work is directed at how we may enhance this process though adjunctive therapies such as those outlined here. It is clear that modulation of the host response and in particular the negative effects of inflammation shows considerable promise in enhancing the results of non-surgical therapy.

About the author

Dr Michael Danesh-Meyer is a specialist periodontist in private practice in Auckland, New Zealand. He was a Clinical Assistant Professor in Periodontology and Associate Scientist in the Laboratory for Applied Periodontal and Craniofacial Regeneration at Temple University, School of Dentistry in Philadelphia, USA. He has been involved in pre-clinical and clinical research involving Guided Tissue Regeneration/Guided Bone Regeneration and dental implants since 1991, has authored numerous scientific articles and lectures both nationally and internationally on topics related to implant dentistry and tissue regeneration therapy. He established the Institute of Dental Implants & Periodontics and Auckland Clinical Training Centre in 2000 and is Director of Dental Education Continuum and DentalMentor.org.

References

1. Heitz-Mayfield LJ etal. A systematic review of the effect of surgical debridement vs. non-surgical debridement for the treatment of chronic periodontitis. J Clin Periodontol 2002; 29(3); 92-102.
2. Winkel EG, et al. Amoxicillin plus metronidazole in the treatment of adjult periodontitis patients. A douple blind placebo-controlled study. J Clin Periodontol. 2001; 28(4);296-305.
3. Sigusch B et al. A 2-step non-surgical procedure and systemic antibiotics in the treatment of rapidly progressive periodontitis. J Periodontol 2001; 72(3); 275-83.
4. Gomi K, etal. Effects of full-mouth scaling and and root planning in conjunction with systemically administered azithromycin. J Periodont 2007; 78(3); 422-9.
5. Dasstoor SF, et al. Effect of adjunctive systemic azithromycin with periodontal surgery in the treatment of chronic periodontitis in smokers. A pilot study. J Periodontol 2007; 78(10); 1887-96.
6. Bonito AJ , Lux L, Lohr KN. Impact of local adjuncts to scaling and root planning in periodontal disease therapy. A systematic review. J Perioodontol 2005; 76(8); 1227-36.
7. Holzhausen M etal. Protective effects of etoricoxib, a selective inhibitor of cyclooxygenase-2, in experimental periodontitis in rats. J Periodontal Res 2005; 40(3); 208-11.
8. Reddy MS etal. Periodontal host modulation with antiproteinase, anti-inflammatory and bone sparing agents. A systematic review. Ann Periodontol. 2003: 8(1); 12-37.
9. Golub LM etal. Low-dose doxycycline therapy: effect on gingival crevicular fluid collagenase activity in humans. J Periodontal Res 1990; 25(6) 321-30.