International Journal of Health Sciences and Research
www.ijhsr.org ISSN: 2249-9571

Review Article

Effects of Smoking on Periodontal Therapy

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Received: 20/03//2014 *Revised: 09/04/2014* *Accepted: 17/04/2014*

ABSTRACT

Smoking is considered to be one of the most significant risk factors associated with periodontal disease initiation and progression. Smoking may influence the clinical outcome of non-surgical and surgical periodontal therapy as well as the long-term success of implant placement. This article discusses the effects of smoking on the response to periodontal treatment and explores the beneficial effects of smoking cessation in periodontal therapy.

Key words: smoking, periodontal disease, periodontal therapy, smoking cessation

INTRODUCTION

Smoking exerts its harmful effects on virtually every tissue in the body, including the periodontium. [1] Haber has described a discrete, smoking-specific disease entity – smoking associated periodontitis – that is characterized by fibrotic gingiva, limited gingival redness and oedema relative to disease severity, proportionally greater pocketing in anterior and maxillary lingual sites, gingival recession at anterior sites and a lack of association between periodontal status and the level of oral hygiene. [2]

Cigarette smoking is a well-established risk factor for periodontitis and, second to bacterial plaque, is the strongest of the modifiable risk factors. Smoking increases the prevalence and severity of periodontal destruction. [1] Cross-sectional studies have consistently demonstrated that smokers present with less gingival inflammation than non-smokers suggesting that smokers have a decreased expression of clinical inflammation in the presence of plaque accumulation compared with non-smokers. [3-6] Multiple cross-sectional and longitudinal studies have demonstrated that pocket depth, attachment loss, and alveolar bone loss are more prevalent and severe in patients who smoke compared with non-smokers. [6-8] Smoking is associated with a two- to eight-fold increased risk for periodontal attachment and / or bone loss, depending on the definition of disease severity and smoking dose. [1,9]

Various factors contribute to the deleterious periodontal effects of smoking, including alterations in both microbial and host response factors. Systemic innate and immune responses are impacted by smoking, and tobacco components have toxic effects for local cell populations, and impact local host responses. Even though the precise mechanisms by which smoking exerts detrimental effects on the periodontium have

not yet been clearly understood, proposed mechanisms for the negative periodontal effects of smoking include vascular alterations, altered neutrophil function, decreased IgG production, decreased lymphocyte proliferation, increased prevalence of periodontal pathogens, altered fibroblast attachment and function, difficulty in eliminating pathogens by mechanical therapy, and negative local effects on cytokine and growth factor production. [1,6,7,10]

Non-Surgical Therapy

The majority of clinical research supports the observation that pocket depth reduction is more effective in non-smokers than in smokers after nonsurgical perio therapy (Phase I therapy), including oral hygiene instruction, scaling, and root planing. [11-16] In addition, gains in clinical attachment as a result of scaling and root planing are less pronounced in smokers than in non-smokers. [6]

Renvert et al, [17] in a study of patients with previously untreated advanced periodontal disease, demonstrated that scaling and root planing plus oral hygiene resulted in significantly greater average reductions in pocket depth and bleeding on probing in non-smokers than in smokers, when evaluated 6 months after completion of therapy. According to Georgia et al, [10] the numerical differences between smokers and non-smokers become more pronounced in probing depths ≥5 mm, where smokers demonstrated 0.4 mm to 0.6 mm less improvement in clinical attachment levels following scaling and root planing.

Jin et al, [18] however, reported significantly greater reductions of the order 1.0mm in non-smokers compared with smokers at 1 and 3 months following non-surgical therapy. Further, Papantonopoulos [19] noted that between 6 and 8 weeks following non-surgical therapy, significantly more smokers (42.8%) than non-smokers (11.5%) required further treatment and the smokers may have benefitted from a surgical approach in the first instance. Grossi et al, [12] showed that nonsurgical managements of pockets 5 mm or greater showed that smokers had less pocket depth reduction than non-smokers after 3 months as well as fewer gains in clinical attachment levels. When a higher level of plaque control can be achieved as part of nonsurgical care, the differences in the resolution of 4-mm to 6-mm pockets between non-smokers and smokers become clinically less significant. [16]

According to Carranza, the average pocket reductions of 2.5 mm for non-smokers and 1.9 mm for smokers were observed in periodontal pockets that averaged 7 mm before treatment. [6] However, the negative impact of smoking decreases with increased level of plaque control. When comparing current smokers with former smokers and non-smokers, the former smokers and non-smokers appear to respond equally well to non-surgical care, reinforcing the need for patients to be informed of the benefits of smoking cessation. [1, 6, 20]

In a group of patients with generalized aggressive periodontitis, Darby et al, [21] reported 0.7 mm less improvement in probing depth and 0.4 mm less attachment gain in smokers compared to non-smokers at the 6- to 8-week re-evaluation following scaling and root planing.

Preber et al, [22] studied the clinical and microbiological effects of non-surgical therapy and found that smokers had a less favourable outcome in terms of pocket depth reduction than did non-smokers. The study revealed no difference, however, between smokers and non-smokers in terms of the microbiological changes following therapy, i.e., the microflora was broadly similar in both categories of patients before and after treatment. In a microbiological study, of

particular interest was the observation that smokers do not respond to mechanical therapy as well as non-smokers; this is associated with increased levels of B. forsythus (now T. forsythia), A.actinomycetemcomitans, and P.gingivalis remaining in the pockets after therapy in the smoking group when compared with non-smokers. [13]

Machtei et al, [23] considered the changes in attachment level and alveolar bone levels approximately one year after the hygiene phase of therapy. Non-smokers had relatively stable bone height, whereas smokers exhibited an annualized rate of bone loss of 1. 17 mm. Furthermore, Bostrom et al, [24] in a five-year study, smokers were found to exhibit less improvement compared with non-smokers in terms of bone height.

According to McFarlane et al, [25] approximately 90% of patients who were categorized as having failed to respond to conventional therapy were smokers. Although smokers will also benefit from treatment, albeit to a lesser degree, treatment failures tend to predominate among smokers. Kinane and Radvar, [26] found that the response to non-surgical mechanical therapy is particularly poor in deep periodontal pockets in smokers. Although the attachment gain was also greater among the non-smokers than the smokers, this was not significant. This indicates that, after treatment, a greater degree of recession occurred among the non-smokers compared with the smokers. In the description of the appearance of smokers' periodontal condition, and in studies looking cross-sectionally at smokers, a commonly noted feature is the level of recession, which is often noted as worse in smokers than in non-smokers. [27, 28]

Not all studies, however, have shown unequivocally a more effective response in non-smokers compared with smokers. Pucher et al, [29] reported that smokers and non-smokers responded similarly to non-surgical therapy after 9 months with reference to reduction in probing depth, attachment level gain and reduction in bleeding on probing. Only non-smokers, however, showed a significant improvement in gingival index after 9 months compared with baseline. Further, in their post-non-surgical treatment evaluation of 12 smokers and 14 non-smokers, Zuabi et al, [30] reported no difference in post-treatment probing depth and clinical attachment level between smokers and non-smokers. There was, however, significantly more plaque in smokers compared with the non-smokers and the smokers had significantly greater probing depths at baseline compared with the non-smokers. Consequently, the greater probing depth reduction (0.81 mm) in smokers compared with non-smokers (0.5 mm) will itself have been a direct consequence of the greater depth of pocketing in smokers before treatment.

Thus, in general, there is decreased clinical response to non-surgical periodontal therapy in smokers as compared to non-smokers. However, the negative impact of smoking decreases with increased level of plaque control. When comparing current smokers with former smokers and non-smokers, the former smokers and non-smokers appear to respond equally well to non-surgical care, reinforcing the need for patients to be informed of the benefits of smoking cessation. [6]

Antimicrobial and Host Modulatory Therapy

Because of the diminished treatment response in smokers, clinicians may be more likely to utilize adjunctive antimicrobial therapy in these patients. This is a rational approach based on evidence suggesting that subgingival pathogens are more difficult to eliminate in smokers following scaling and root planing. [1]

Soder et al, [31] concluded that there was little adjunctive effect of systemic metronidazole on non-surgical therapy in smokers. On the other hand, in studies where scaling and root planing were combined with adjunctive doxycycline gel or minocycline microspheres, the response in smokers was similar to that seen for non-smokers receiving scaling and root planing alone. [32,33] In a study comparing adjunctive systemic antibiotic therapy to scaling and root planing alone, smokers receiving amoxicillin and metronidazole showed significantly more improvement in bleeding scores, probing depth and attachment levels than smokers receiving only scaling and root planing. [34] Smokers comprise a high percentage of refractory periodontitis patients. [25,35] The combination of intensive antimicrobial therapy including mechanical plaque removal, systemic antibiotics and locally delivered antibiotics has been shown to be of value in controlling disease progression in refractory patients. [36]

In addition to their antimicrobial activity, part of the benefit of locally delivered tetracycline derivatives may be derived from their anticollagenase activity. [1] Novak et al, [37] who reported a positive response to sub-antimicrobial doxycycline (anti-collagenase) therapy in combination with scaling and root planing in a group of patients with severe periodontitis that included smokers.

Unique regimens combining local antimicrobial delivery with host modulatory therapy or sequencing of host modulatory therapy following systemic antimicrobial therapy may offer clinicians and patients new therapeutic options that address both the microbial and host response alterations that are evident in smokers. [1]

Surgical Therapy

The less favourable response of periodontal tissues to non-surgical therapy that is observed in current smokers also appears to apply to surgical therapy. [6]

Open flap debridement surgery without regenerative or grafting procedures is the most common surgical procedure used for accessing the root and osseous surfaces. By 6 months after this procedure, smokers showed significantly less reduction of deep pockets (>7mm) than smokers and significantly less gain in clinical attachment, even though the patients received supportive periodontal therapy every month for 6 months. Of increased significance was the observation that only 16% of deep pockets in smokers returned to 3 mm or less at 6 months after surgery, whereas 47% of the deep pockets in non-smokers were 3 mm or less after completion of therapy. [38]

In a longitudinal comparative study of the effects of four different treatment modalities, including coronal scaling, root planing, modified Widman flap surgery, and osseous resection surgery, smokers consistently showed less pocket reduction and less gain in clinical attachment levels than non-smokers or former smokers. [15]

Following surgical treatment such as osseous surgery, modified Widman flap surgery, or flap debridement surgery, smokers had approximately 0.5 mm less improvement in probing depth and attachment levels, which was on average only 50–60% as much improvement as that in nonsmokers. [11, 39] In two studies there was a 1 mm difference between smokers and nonsmokers for both probing depth and clinical attachment level improvements at sites initially probing ≥ 7 mm. [11, 38] These differences remained over a 6-year maintenance period. At furcation sites both horizontal and vertical attachment gain were impaired by smoking. [11,40] Kaldahl et al, [15] noted a trend for heavy smokers (≥20 cigarettes per day) to respond less favourably to treatment than light smokers (<20 cigarettes per day).

Tonetti et al, [41] performed a retrospective study that examined the effect of cigarette smoking on the healing response following guided tissue regeneration (GTR) in deep infrabony defects. This study indicated that smoking was a significant factor in determining the clinical outcome. A risk-assessment analysis indicated that smokers had a significantly greater likelihood than non-smokers of having a reduced probing attachment level gain following GTR. [20]

Periodontal Soft Tissue Grafting

The majority of studies indicate that smokers exhibit three-quarters of the amount of root coverage shown by nonsmokers. [1] Also, sub-epithelial connective-tissue grafts were demonstrated to be less successful in smokers than nonsmokers. [42, 43]

In a case series including 100 patients, Harris consecutively treated recession sites using a connective tissue with a partial thickness pedicle graft and found no difference between the percentage of root coverage among light smokers (97%), heavy smokers (99%) or non-smokers (98%). [44]

Amarante et al, [45] found no difference in root coverage between smokers and non-smokers when recession defects were treated with a coronally repositioned flap alone or with a bio-absorbable membrane. On the other hand, when expanded polytetrafluoroethylene membranes were utilized in guided tissue regeneration procedures at recession sites, smokers had significantly less root coverage (57%) compared to non-smokers (78%). [46] The superior blood supply afforded by the sub-epithelial connective tissue graft might be more resistant to the effects of smoking as compared to the non-resorbable barrier membrane. [10]

In general, while smokers have more gingival recession than non-smokers, and can benefit from root coverage procedures to treat recession, studies suggest that smoking negatively impacts the clinical outcomes of root coverage surgery. [1]

Periodontal Hard Tissue Grafting

Smoking has also been reported to negatively impact regenerative procedures in interproximal and furcation defects, including osseous grafts alone, membranes alone or membranes in combination with osseous grafts. [1, 10]

Studies have demonstrated less than 50% as much improvement in clinical attachment levels in smokers compared to non-smokers, which amounted to differences ranging from 0.35 mm to 2.9 mm. [41] In studies that evaluated osseous changes by sound probing or re-entry, vertical bone gain in smokers ranged from 0.1 to 0.5 mm, whereas non-smokers demonstrated 0.9 to 3.7 mm improvement. [47, 48]

Bowers et al, [49] found significantly more residual class II defects among smokers than nonsmokers (62.5% vs. 14.3%) in furcations treated with a combination of demineralized freeze-dried bone allograft and a polytetrafluoroethylene membrane. Miller demonstrated that cigarette smoking has been associated with 80% failure rate in the treatment of furcation defects. [10] In contrast, Tsao et al, [50] found that smoking had no effect on regenerative outcomes of mandibular class II furcation defects. In terms of stability of treatment results, Cortellini et al, [51] found that stability was related to patient factors; patients who smoked, were non-compliant with recall, and had deteriorating oral hygiene lost attachment (2.2 to 2.4 mm) following both guided tissue regeneration and scaling and root planing treatment modalities.

Implant Therapy

Smoking is significantly associated with implant failure, based on a multivariate statistical model adjusted for age, gender, and jaw position. 0% to 17% of implants

placed in smokers were reported as failures as compared to 2% to 7% in non-smokers, with the majority of studies showing at least twice as many failed implants in smokers. [1, 10]

The largest data set on the influence of smoking on implant success comes from the Dental Implant Clinical Research Group (DICRG) of the Department of Veterans Affairs (DVA). This is an 8-year, randomized, prospective clinical study that includes more than 2,900 implants in more than 800 patients at 32 clinical centres. The 3-year data demonstrated that 8.9% of implants placed in smokers failed as compared to 6% in individuals who had never smoked or had quit smoking. The majority of implant failures in smokers occurred prior to prosthesis delivery; thereafter, the differences between smokers and non-smokers tended to disappear. [10, 52, 53] Smoking impacts implants placed in the maxillary arch more negatively than in the mandible. The DICRG reported that the percentage of maxillary implant failures among smokers (10.9%) was almost twice that reported for non-smokers or past smokers (6.4%). [53]

The impact of smoking on implant therapy is more dramatic in grafted maxillary sinuses compared to non-grafted sites, with percentage of implant failures in grafted sinuses in smokers being 12.7% compared to 4.8% in the non-smoker group. [10, 54, 55]

The effect of smoking on ridge augmentation procedures has been studied. Jones and Triplett, [56] reported that four of five smoking patients undergoing simultaneous onlay grafting and implant placement had impaired healing, as defined by loss of bone and/or implants. Another study reported that defect reduction in guided bone regeneration procedures around 36 implants placed in smokers was not significantly affected by smoking. In contrast to most other reports, smoking did not impact treatment success in these patients. [57]

Smoking cessation should be recommended prior to implants and patients should be advised and informed of the benefits of smoking cessation and the potential risks of smoking for implant failure. [6]

Maintenance Therapy

The detrimental effects of smoking on treatment outcomes appear to be long lasting and independent of the frequency of maintenance therapy. After 4 different modalities of therapy including scaling, SRP, modified Widman's flap surgery and osseous (reduction) surgery, maintenance therapy was performed every 3 months for 7 years. Smokers consistently had deeper pockets than non-smokers and less gain in attachment when evaluated each year for the 7-year period. [6]

Recurrent (Refractory) Disease

Because of the difficulty in controlling periodontal disease in smokers, many smokers become refractory to traditional periodontal treatment and tend to show more periodontal breakdown than non-smokers after therapy. It is now thought that patients formerly considered refractive to therapy actually undergo continuous or recurrent disease; for this reason the diagnosis of refractory periodontitis has been removed as a distinct classification. (Approximately, 90% of these so called 'refractory patients' were smokers). Smokers may present with periodontal disease at an early age, may be difficult to treat with conventional therapy and may continue to have progressive or recurrent periodontitis leading to tooth loss. [6, 20, 25]

Healing In Smokers

Healing following conventional scaling and root planing is seen clinically as a reduction in pocket depth and is the result of a reduction in inflammation which causes

tissue shrinkage or reduced inflammatory swelling and also an improved tissue tone. This improved tissue form is more resistant to pocket probing forces and is detected clinically as an increase in clinical attachment. The tissue shrinkage may result in recession which, together with the increased attachment, produces reduced probing depths. It has been hypothesized that, in smokers, much of the inflammatory tissue swelling before treatment may be absent, and thus this part of the post-therapy tissue change may not contribute as much to the post-treatment pocket depth reduction in smokers compared with non-smokers. [20, 26] All things being equal, smokers could therefore have deeper pockets after therapy than non-smokers, and these pockets will continue to harbour quantitatively and qualitatively more pathogenic bacteria than shallower pockets. Coupled with this are the reduced fibroblast, polymorphonuclear leukocytes (PMN), and epithelial cell function, reduced host defense response, less probing and reduced vascularity of the site. These tissue differences in smokers following the initial short-term healing after therapy (up to six weeks) may partly explain the differences in treatment response. [20]

Fibroblasts are an important cell in the periodontal healing response. Poorly functioning fibroblasts consequent to nicotine binding and internalization may not produce collagen fibres as efficiently, and thus gingival tissue support and adaptation will be impaired or at least slowed, and poor tissue form will often result in greater microbial plaque retention around teeth. [20, 58]

Another crucial cell of the dento-gingival barrier is the keratinocyte. Johnson et al, [59] have shown that human gingival keratinocytes are induced to produce significantly increased amounts of IL-1 and prostaglandin E-2 (PGE2) by tobacco extracts. Furthermore, a constant feature of the junctional epithelium is the presence of PMN migrating through the epithelial layers and into the gingival crevice. The PMN is considered an important cell in the local host defense at this site. [20] MacFarlane et al, [25] reported that phagocytosis of polymorphonuclear leukocytes in refractory periodontitis patients was impaired. They found that 90% of these patients were smokers compared with 21% of the controls. No direct assessment of smoking or tobacco products on PMN function was assessed, however. [20]

Smoking Cessation

Smoking cessation is beneficial to periodontal treatment outcomes and periodontal health. Observational studies comparing periodontal health between current, former, and non-smokers after periodontal treatment suggested that quitting smoking is beneficial to patients with periodontal diseases. [20,60] Smoking cessation cannot reverse the past effects of smoking; however, the rate of attachment and bone loss slows after patients quit smoking. Periodontal disease severity in former smokers falls between that of current and non-smokers. [1, 10, 20]

Smoking cessation should be considered an essential component of periodontal therapy. [1] The practice of periodontics offers multiple opportunities for interaction with patients: during active treatment and especially in the ongoing long-term maintenance phase of care. Because of the negative impact of tobacco use on periodontal treatment, an additional motivation for cessation can be demonstrated over time and used effectively to help patients ultimately achieve a tobacco-free life. [10,28] Smoking cessation intervention strategies, including behavioural therapy and pharmacotherapy may be integrated in existing procedures of dental treatment for achieving an improved outcome. [60]

CONCLUSION

Research has indicated that there is considerable evidence available for the role of smoking in the etiology of periodontal disease and its influence on the treatment of periodontal diseases. As compared to non-smokers, smokers respond less favourably to non-surgical therapy as well as surgical therapy, including soft tissue grafting and osseous regenerative therapy and implant therapy. Smokers more frequently experienced a recurrence of periodontal disease than non-smokers during supportive periodontal therapy. Tooth loss is a tangible outcome of periodontal treatment and also reflects the recurrence of periodontal disease. Therefore, advising patients of the negative effects of tobacco use and emphasizing on the benefits of smoking cessation are essential in the management of patients who smoke.

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How to cite this article: Lawande SA. Effects of smoking on periodontal therapy. Int J Health Sci Res. 2014;4(5):248-258.

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