Smoking

Recently I have encountered many patients with deteriorating periodontal conditions who are smokers. Many of these patients have asked how smoking would affect their periodontal condition when questioned if they would ever think of quitting.

Because of the above I thought it would be a good idea to briefly review the effect of smoking on the periodontium at a cellular and vascular level. As well, what effect will smoking have on our periodontal treatments, both surgical and non-surgical therapies.

Many of these patients are already missing teeth and are concerned about their restorative options. Implants often times come up in discussion, I thought it would be a good idea to include some information on implant survival rates between the two groups.

1) Effects of smoking on the periodontium at a cellular and vascular level:
   a. Smoking and subgingival microflora:
      i. Largest studies support higher prevalence of Aggregatibacter actinomycetemcomitans, t. forsythia and P. gingivalis in smokers compared to former smokers (>1 year since quit) and never smokers.
      ii. To be exact:
         1. 3.1x for A.a
         2. 2.3x for T. forsythia
      iii. T. forsythia levels are also more likely to be dose-related to amount of smoking (packs per day for # for years)
      iv. The increased level of periodontal pathogenic bacteria explain why smokers are at a higher risk of periodontal disease
   b. Smoking and gingival blood flow:
      i. Smoking is thought to cause an initial hyperaemic response
         1. Increase blood flow to gingiva during smoking, but reduced flow to periphery
         2. HR and BP increased upon smoking due to sympathetic activity
   c. Smoking and bleeding on probing:
      i. Less bleeding on probing for smokers vs non-smokers, most likely due to decreased presence of gingival vessels
   d. Smoking and neutrophil function:
      i. E-selectin and Intercellular adhesion molecule (ICAM-1) are responsible for rolling of leukocytes for eventual migration through endothelial cells of vessels
      ii. Naturally there is increased vasculature in sites of inflammation
      iii. There are more vessels present in non-smokers vs smokers. As well, the number of vessels that are positive for E-selectin and ICAM-1 during inflammation is much lower in smokers. This results in an altered immune response (reduced neutrophil chemotaxis) to gingival inflammation caused by the increased level of periodontal pathogens explained above.
iv. In addition to chemotaxis, the oxidative burst of neutrophils (required to eliminate foreign bodies once phagocytosed) is reduced by half in smokers compared to non-smokers.

v. While there are direct effects of smoking on chemotaxis and function of neutrophils caused by smoking, we see there is an increase in the number of neutrophils in smokers compared to non-smokers. Our immune system is reacting to the inflammatory response in the gingival tissues, but the action is inefficient causing a prolonged and increased neutrophil response.

e. Smoking and bone healing:

i. This seems to be controversial, as this study had shown there is a negative effect on bony defect healing and fill following cigarette smoker inhalation. However, no clear answer on the process that is affected has been confirmed.

1. Nicotine levels affecting osteoblast function and revascularization.
2. Nicotine not playing a role, but other factors in cigarettes being responsible.

2) Effects of smoking on surgical and non-surgical therapy outcomes:

a. Smokers have a worse response to treatment compared to non-smokers

b. Non-surgical therapy (SRP):

i. Non-smokers with chronic periodontitis or aggressive periodontitis have a greater reduction in probing depth compared to smokers

ii. SRP was less successful in smokers at reducing probing depth

c. Surgical therapy

i. Most predictable surgical technique for root coverage: Connective tissue graft.

1. Superior reduction (~1mm) in gingival recession and in turn gain in clinical attachment in non-smokers treated with connective tissue grafts compared to smokers

2. Cigarette smoking affects root coverage

ii. Most commonly used surgical technique for lack of keratinized tissue: Free gingival graft

1. No significant difference in width of keratinized tissue following augmentation in smokers vs. non-smokers at 90 days (0.5mm difference recorded)

2. Donor site epithelialization is impaired in smokers. After 15 days only 20% of the palate was epithelialized in smokers, while after the same time period 92% of epithelization had occurred in non-smokers

3. Also, smoking reduces immediate post-operative bleeding incidence contributing to the decrease in post-operative healing

iii. Surgical therapies for periodontal regeneration: Guided tissue regeneration

1. Smokers treated with the above technique had reduced clinical
attachment gain and reduced probing bone level gain compared to non-smokers.

2. Smokers also had increased gingival recession following GTR.

3. When smokers were treated for intrabony defects with GTR and membrane there was an increase in membrane exposure.

3) Effects of smoking on implant placement:
   a. In smokers, there have been more reported cases of peri-implantitis and mucositis following implant placement compared to non-smokers.
   b. The overall failure rate of implants is higher in smokers (11.28%) compared to non-smokers (4.76%)
      i. There is no significant difference in the failure rate of implants placed in smokers (4.59%) vs non-smokers (3.79%) when placed in the posterior mandible
      1. All other regions had shown a significant difference in implant failure, with smokers showing greater rates of failure (maxilla 17-19% in smokers compared to 4-11% in non-smokers)
      ii. It was also found that longer implants had resulted in less failures irrespective of smoking status, especially in the posterior maxilla
      iii. HOWEVER, newer implant design and surface modification has reduced the failure rates of smokers and non-smokers to levels that are no longer significantly different.

Take Home Message

- Cigarette smoking is correlated with increased gingival levels of pathogenic bacteria.
- Cigarette smoking decreases gingival vasculature, increases immediate gingival blood flow and alters host response to gingival inflammation due to reduced neutrophil chemotaxis and oxidative burst.
- Cigarette smoking adversely affects the result of periodontal therapy, non-surgical and surgical alike. Although the improvements in smokers are less than those for non-smokers, it is important to recognize that smokers still benefit from both non-surgical and surgical therapy.
- Advancement in implant surface modifications and treatments have reduced implant failures in smokers vs. non-smokers to a level that is statistically insignificant.

References
