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Mini Review

Detrimental Effects of Smoking in Periodontal Disease

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Abstract

Periodontal Disease is a pathologic inflammatory disease affecting the supporting tissues of tooth. Smoking is a major risk factor in prevalence, extent and severity of periodontal disease. Smoking creates imbalance between bacterial challenge and host response, thus, it aggravates the severity of disease. It may influence the clinical outcome of non-surgical therapy and surgical therapy as well as implant and regeneration procedures. It causes some irreversible changes in periodontium such as pocket formation, attachment loss and more alveolar bone loss. Statistical analysis of smokers connected with periodontal disease constitutes major dental health problem. However, the deterioration of the periodontium doesn't continue after smoking cessation.

Keywords: Periodontal Disease; Smoking, Immune Response; Periodontal Therapy

Introduction

Periodontal Disease is a pathologic inflammatory disease affecting the supporting tissues of tooth. The earliest form is gingivitis which affects gingiva only whereas in periodontitis, all tissues surrounding the tooth may be affected. The microbial interaction with host determines the extent and severity of periodontal disease. Various environmental, genetic and lifestyle factors are responsible for progression of periodontal disease. Smoking is one of the major environmental risk factors in progression of periodontal disease [1]. Smokers tend to be more susceptible to advanced and aggressive form of periodontal disease and are at greater risk for exhibiting severe bone loss [2,3]. Results from first US National Health and Nutrition Examination Survey (NHANES I) has demonstrated that current smokers had more plaque and periodontal destruction than former or non-smokers [4]. Indeed, smoking has adverse effect on all periodontal therapy approaches ranging from mechanical debridement, systemic antimicrobial therapy to periodontal surgery including implant and regeneration procedures.

Smoking and Periodontal disease

The increased prevalence and severity of periodontal disease associated with smoking is due to imbalance between bacterial challenge and host response. Although smoking decreases gingival inflammation and bleeding on probing, but it increases pocket depth, attachment loss and bone loss. A meta-analysis of data from six such studies involving 2361 subjects indicated that current smokers were almost 3 times more likely to have severe periodontitis than former or non-smokers [5]. Grossi and colleagues demonstrated the relationship between smoking and attachment loss and showed a dose dependent response in which more severe attachment loss occurred in smokers compared with non-smokers, ranging from 2.05mm for light smokers to 4.75 mm in heavy smokers [6]. These findings support the fact that the adverse effects of smoking on periodontium is significantly correlated with pack years (i.e. no. of packs smoked per day multiplied by no. of years he subject has smoked) as well as no. of years since quitting. As far as the region is concerned, a study in Brazil shows that smokers exhibit higher alveolar bone resorption as compared to no smokers and confirmed that cigarette consumption affects maxillary region more than lower jaw and basically the anterior area [7]. Significant effects have been reported on the protective mechanism of host resulting in progression of disease. Neutrophil is an important component for chemotaxis, phagocytosis and oxidative bursts. Alteration in neutrophil activity may cause local infection. It has also shown that smoking increases PGE2 level, neutrophil elastase and MMP [8]. It might result in periodontal destruction. In vitro studies have also shown that there was no difference in bacterial count but alteration in subgingival temperature have been reported [9].

Zambon., *et al.* reported that smokers had significantly higher level of B. Forsythus and smokers were 2-3 times more likely to harbour this periodontal pathogen than former smokers or non-smokers [10]. This evidence indicates that rate of attachment loss and bone loss slows after patients quit smoking. In addition, the oxygen concentration in healthy tissues appears to be less in smokers than in non-smokers [11]. This data suggests that alteration in gingival vasculature leads to decreased blood flow and decreased clinical signs of inflammation.

Preber and Bergstorm reported that smokers didn't respond as much as non-smokers to non-surgical therapy [12]. Mc Guirre and Nunn found twice the risk of tooth loss in smokers undergoing periodontal treatment over a five-year period [13]. The impact of smoking on implant success is unclear. Several studies have shown that smoking reduces success rates [14], whereas other studies have shown no effect [15]. However, with existing evidence on detrimental effect of smoking on implant procedure, patient should be informed and advised to stop smoking due to its potential risks.

Conclusion

It can be concluded from the above written literature that smoking is major environmental risk factor in progression of periodontal disease. However, the exact mechanism by which smoking affects periodontal tissue is not clear, but with existing evidence indicates that it is a risk factor and it depends upon its frequency and duration. Although it aggravates the periodontal attachment loss, but smoking cessation respond more positively to periodontal therapy.

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