

ACTA SCIENTIFIC DENTAL SCIENCES (ISSN: 2581-4893)

Volume 3 Issue 11 November 2019

Review Article

Smoking and Periodontal Disease - Review

Rajni Mishra*

General Dentist, Glasgow Medical Center, Dubai

*Corresponding Author: Rajni Mishra, General Dentist, Glasgow Medical Center, Dubai.

Received: September 11, 2019; Published: October 31, 2019

DOI: 10.31080/ASDS.2019.03.0686

Abstract

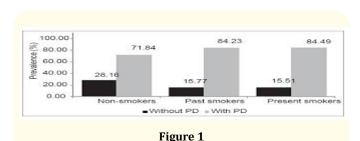
Cigarette smoking represents a major preventable cause of human disease. Smoking is an environmental factor which affects the oral cavity to a severe extent. A precise causal relationship between smoking exposure and the prevalence and the severity of periodontal disease has been resolutely recognized. Smoking is an independent risk factor for the initiation, extent and severity of the periodontal disease. A direct causal relationship between smoking exposure and the prevalence and the severity of periodontal disease has been firmly established. Smoking causes periodontitis which affects the periodontium of the tooth which comprises of alveolar bone, gingiva, periodontal ligament and cementum. The relationship between smoking and periodontal health was investigated as early as the middle of last century. This article presents the effect of smoking on the periodontium, etiology and pathogenesis of periodontal disease and response to periodontal therapy.

Keywords: Smoking; Periodontal Disease

Introduction

Periodontal disease is one of the most common chronic diseases in adults. It is the second most cause of tooth loss after dental caries among adults in developed countries. Tobacco smoking is an environmental factor and is now acknowledged as a major risk factor in the development and progression of periodontal disease. Smokers are almost thrice as likely to have severe periodontitis compared to nonsmokers and associated with 90% of refractory periodontitis. There is an abundance of scientific evidence that smoking has an additive effect on the progression of periodontal disease and is detrimental to healing after periodontal therapy. In smokers there is decreased gingival inflammation and bleeding on probing, but increased prevalence and severity of periodontal disease. Overall, smoking is probably the single most significant, modifiable risk factor for periodontal diseases.

Effect of Smoking on the prevalence of periodontal disease



Epidemiological evidence

- (Hill's criteria) Cross-sectional studies and case-control studies reveal a strong correlation between smoking and periodontal disease.
- Up to 90% of patients established with refractory periodontitis are smokers.

- Smoking may be accountable for more than half of the periodontal disease among adult population.
- Smokers are four times as likely to develop periodontitis as non-smokers.
- Carranza asserted women from ages 20 to 39 and men from ages 30 to 59 who smoke cigarette have twice the likelihood of having periodontal disease or becoming edentulous as do non-smokers.
- The impacts of smoking on periodontal status to be more evident in younger women.

Effect of smoking and severity of periodontal disease Gingivitis

There is decreased gingival inflammation and bleeding on probing.

Periodontitis

Increased prevalence and severity of periodontal disease; Increased pocket depth, attachment loss, bone loss and tooth loss; Increased rate of periodontal destruction.

According to a study, Before the age of 50 years about 80% of myocardial infarctions and 70% of chronic lung diseases are caused due tobacco smoking.

The most important environmental risk factor in periodontal disease is the cigarette form of tobacco smoking. In case of a periodontal disease there is a dynamic phenomenon with cyclical patterns of progression and resolution at any given site. Smoking impairs the immune response and compromises the ability of periodontal tissues to heal, following a period of disease activity.

Etiology

Tobacco products such as Nicotine have been found to have adverse effects on cells of the periodontium, including gingival fibroblasts and cells of the immune system. Nicotine inhibits attachment and growth of human PDL fibroblasts. This may partly explain the role of these substances in the progression of periodontitis. Tobacco smoking, mostly in the form of cigarette smoking, is recognized as the most important environmental risk factor in periodontitis. Tobacco smoking probably plays a significant role in the development of refractory periodontitis. There is increased production of inflammatory mediators. Fibroblast production is diminished, and it also impairs the normal reparative and regenerative potential of periodontium.

Microbiology

Smokers had higher level of Tinnerello forsythia. They do not respond to mechanical therapy due to increased level of T. forsythia, Aggregatibacteractinomycetemcomitans and Prohormones gingival is which remains in the pocket even after therapy when compared to non-smokers.

Immunology

Literature suggests that tobacco product such as nicotine affects gingival blood flow, cytokine production, neutrophil chemotaxis, phagocytosis, connective tissue turnover and other immune cell functions which can be the possible mechanisms responsible for overall effects of tobacco on periodontal tissues. There is increased TNF alpha (Tumour Necrosis factor alpha) and PGE two (prostaglandin two) in gingival crevicular fluid. ImmunoglobulinG2 level is reduced suggestive of reduced protection against the periodontal infection.

Nicotine being a major component of tobacco, adversely affects fibroblast function and suppresses the osteoblast proliferation while stimulating the alkaline phosphatase activity.

Normal reparative and regenerative process of the periodontium is altered by the tobacco products which leads to the diseased condition of the periodontium.

Physical condition of the diseased periodontium

Smokers may present with lower levels of gingival inflammation to a specific level of plaque than nonsmokers. Smokers have a higher proportion of sites with deeper probing depths and clinical attachment loss compared with nonsmokers. Smoking induces chronic hypoxia of periodontal tissues causes greater severity of periodontal disease seen in smokers.

An in vitro study done by Tanur., *et al.* showed that the nature of cell attachment to root surfaces is altered by nicotine.

Results of the study by Silva., *et al.* showed that cigarette smoke, but not nicotine, may significantly alter cell viability, cell migration, and myofibroblast differentiation in gingival mesenchymal cells.

Effect of smoking on the response to periodontal therapy

Smoking has a strong negative impact on regenerative therapy, including osseous grafting, guided tissue regeneration, or a combination of this treatment. Tobacco is a peripheral vasoconstrictor in-

fluencing the rate at which the oral wound heals. Carbon monoxide and other chemicals produced during the combustion of tobacco can reduce the capillary blood flow. A clinical study has shown that a single cigarette can reduce the peripheral blood velocity by 40% in one hour. The mechanism by which smoking may affect the wound healing is unknown. One possible explanation is there which states that the substances in tobacco and its smoke, particularly nicotine, cotinine, carbon monoxide, and hydrogen cyanide are cytotoxic to those cells that are involved in wound healing. Nicotine increases platelet adhesiveness, raising the risk of microvascular occlusion, and tissue ischemia. Smoking is associated with catecholamines release, resulting in vasoconstriction and decreased tissue perfusion.

Non-surgical therapy

- Decreased clinical response to scaling and root planning.
- Decreased reduction in pocket depth.
- Decreased gain in clinical attachment level.
- Decreased negative impact of smoking with increased level of plaque control.

Surgery and implant

- Decreased pocket depth reduction post surgery.
- Increased deterioration of furcation post surgery.
- Decreased bone formation.
- Decreased gain in CAL (clinical attachment level)
- Increased recession and membrane exposure following GTR (guided tissue regeneration)
- Decreased pocket depth reduction after DFDBA (demineralized freeze-dried bone allograft)
- Increased risk of periimplantitis.

Effect of smoking on maintenance therapy

- · Increased pocket depth
- Decreased gain in clinical attachment level
- Refractory disease.
- Increased need for retreatment in smokers.
- Increased need for antibiotics in smokers due to increased risk of periodontal infection
- Increased tooth loss in smokers after surgery.

Conclusion

Though we all know that smoking is strongly associated with pre-cancerous and cancerous lesions of the oral cavity. But we cannot refuse that tobacco smoking also causes periodontal problems and it is the most common cause for periodontal disease in adults. Tobacco smoking negatively affects the outcome of the routine periodontal therapeutic procedures performed in the oral cavity. Dentist should assist smoking patients to become tobacco free and edify them about the undesirable effects of smoking.

Volume 3 Issue 11 November 2019
© All rights are reserved by Rajni Mishra.