ISSN: 2581-5989
PubMed - National Library of Medicine - ID: 101738774

International Journal of Dental Science and Innovative Research (IJDSIR)

IJDSIR : Dental Publication Service

Available Online at:www.ijdsir.com

Volume – 7, Issue – 4, August – 2024, Page No. : 179 - 185

Relation Between Smoking and Periodontal Disease: A Review

[1]Dr.Hemalatha D.M., M.D.S., Assistant Professor, Department of Periodontics, Mahe Institute of Dental Sciences, Chalakkara, Mahe- 673310

[2]Dr. Arjun M.R, M.D.S., Reader, Department of Periodontics, Mahe Institute of Dental Sciences, Chalakkara, Mahe- 673310

[3]Dr.Nanditha Chandran, M.D.S., Reader, Department of Periodontics, Mahe Institute of Dental Sciences, Chalakkara, Mahe- 673310

[4]Fitha Khan, Third year BDS Student, Mahe Institute of Dental Sciences, Chalakkara, Mahe- 673310

[5]Husana, Third year BDS Student, Mahe Institute of Dental Sciences, Chalakkara, Mahe- 673310

Corresponding Author: Dr. Hemalatha D.M., M.D.S., Assistant Professor, Department of Periodontics, Mahe Institute of Dental Sciences, Chalakkara, Mahe- 673310

Citation of this Article: Dr. Hemalatha D.M., Dr. Arjun M. R, Dr. Nanditha Chandran, Fitha Khan, Husana, "Relation Between Smoking and Periodontal Disease: A Review", IJDSIR- August – 2024, Volume –7, Issue - 4, P. No. 179 – 185.

Type of Publication: Review Article

Conflicts of Interest: Nil

Abstract

Periodontitis comprises a group of inflammatory conditions that impact the supportive tissues around the tooth, known as the periodontium. The four tissues that comprise periodontium are gingiva, cementum, alveolar bone and periodontal ligament. Tobacco has an immense effect on development, progression and treatment outcomes of periodontal disease. The link between smoking and periodontal health has been studied since the mid-20th century. Smoking independently increases the risk of both initiating and exacerbating periodontal disease. Eventually smoking reduces the chances for good prognosis. Tailored approaches are essential for identifying individual patient risks and achieving improved outcomes.

Keywords: Smoking, Periodontal Disease, Tobacco use, Periodontal pathogens, Smoking Cessation

Introduction

Periodontal diseases are highly prevalent and affects about 90 % of the world population. Gingivitis, being the mild form of periodontal disease, is due to the accumulation of bacterial plaque on teeth adjacent to the gums. Symptoms include red, swollen and bleeding gums. But gingivitis does not extend to supporting structures of teeth and is modifiable. But if left untreated it can lead to periodontitis, which can result in loss of connective tissue and bone support. However, genetic

and environmental factors have immense role in formation of periodontal disease. Tobacco use is a significant modifiable risk factor that greatly influences the development, progression, and treatment outcomes of periodontal disease. (1)

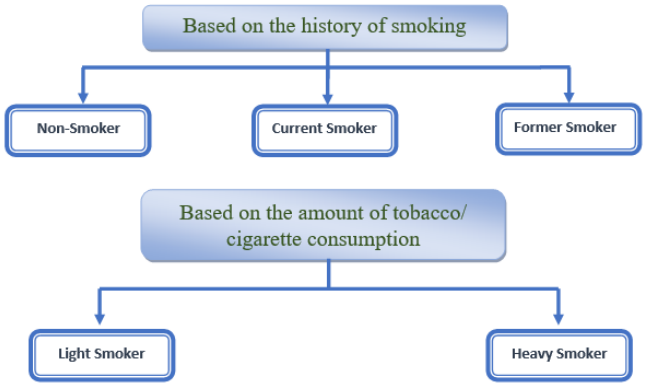
According to American Academy of Periodontology, periodontitis is classified into aggressive periodontitis (AgP), chronic periodontitis (CgP) and periodontitis due to systemic cause. Both AgP and CP have multifactorial causes, with dental plaque being the initial trigger. However, the onset and progression of periodontitis are also influenced by other factors, including tobacco use. (2)

Types of smoking

These are the categories for different types of smoking products

1. Smoked tobacco products: These are tobacco-containing products intended for smoking. Examples include cigarettes, cigars, and hookah tobacco (typically mu'assel), cigarillos, roll-your-own, dokha, pipe tobacco, kizami.
2. Smokeless tobacco products: These products contain tobacco but are used in a way that does not produce smoke, distinguishing them from heated tobacco products. Common examples include dipping tobacco (moist snuff or dip), snus, kuber, gutkha, mava plug, pellets and various forms of chewing tobacco.
3. Heated tobacco products: These products involve heating tobacco to generate an aerosol or particulate suspension that can be inhaled. Also known as heat-not-burn tobacco products or smokeless cigarettes, examples include IQOS tobacco cylinders and loose tobacco blends vaporized in Pax dry herb vaporizers.
4. Nicotine-only products: These products contain nicotine but do not include tobacco. Nicotine is typically extracted from tobacco or synthesized. Common examples include e-liquids (used in e-cigarettes or vapes), nicotine pouches, and various types of nicotine replacement therapy products. (3)

Classification of smokers



Non-Smoker: An adult who has never smoked or has smoked fewer than 100 cigarettes in their lifetime.

Current Smoker: A smoker who has consumed 100 cigarettes in their lifetime and currently continues to smoke cigarettes.

Former Smoker: An adult who has smoked at least 100 cigarettes during their lifetime but had stopped smoking by the time of the interview. (4)

Light Smoker: Using tobacco or cigarettes fewer than 10 times, with the first instance occurring more than 60 minutes after waking up.

Heavy Smoker: Using tobacco or cigarettes fewer than 40 times, with the first instance occurring more than 5 minutes after waking up. (5)

Pack Year

Pack years measure how much pack an individual has smoked over their lifetime. Contrary to what the name implies, it's not simply the number of years one has smoked. The calculation of pack years involves both the duration of smoking and the quantity smoked daily during that period

To calculate someone's pack-year history, you need two pieces of information:

1. The number of packs of cigarettes smoked per day (N).

2. The number of years they have smoked (T).

The formula for calculating pack years is:

Pack Year = N x T

This formula multiplies the average number of packs smoked per day (N) by the number of years of smoking (T) to determine the total pack years. (6)

Effects of smoking

Microbiological

A study examined subgingival biofilm from all teeth except third molars in 272 adult participants, including 50 current smokers, 98 former smokers, and 124 nonsmokers. Using checkerboard DNA-DNA hybridization technology, researchers screened for 29 subgingival species and found that members of the orange and red complex species—such as Eikenellanodatum, Fusobacteriumnucleatumssvincenti, Prevotella intermedia, Peptostreptococcus micros, Prevotellanigrescens, Tannerella forsythia, Porphyromonasgingivalis, and Treponemadenticola—were notably more prevalent in current smokers compared to nonsmokers and former smokers. This heightened prevalence among smokers was predominantly observed in shallow sites (pocket depth ≤4 mm), with no significant differences among the groups in pockets ≥4 mm. These pathogenic bacteria were more commonly found in the maxilla than in the mandible and these results indicate that smokers tend to harbor a greater quantity of periodontal pathogens than nonsmokers or former smokers, potentially increasing the risk of periodontal disease progression. (7)

Immunological

The immune response of host to plaque buildup is protective, maintaining balance in periodontal health and gingivitis without causing periodontal support loss. However, periodontitis results from an altered host-bacterial balance, majorly due to changes in subgingival plaque composition or immune response. Smoking significantly diminishes immune protection, exacerbating periodontal damage by suppressing the immune response against bacterial challenges. Neutrophils, critical in fighting infections, show impaired functions like chemotaxis, phagocytosis, and oxidative burst in smokers or when exposed to tobacco smoke. Smokers with periodontitis exhibit reduced levels of IgG2, crucial for bacterial clearance, and elevated inflammatory markers such as TNF-α, PGE2, neutrophil elastase, and MMP-8 in gingival fluid. Nicotine exposure also increases PGE2 secretion by monocytes in response to bacteria. (7)(16)

Physiological

Effects on microvascular perfusion

Diverse conclusions have been drawn from studies on how nicotine affects oral blood flow in the short term. Application or infusion of nicotine increased blood flow to the dental pulp and gums in animal studies. Nevertheless, nicotine reduced blood flow to the gums when it was injected directly into the rabbits' arteries. The type and duration of tobacco use appear to have an impact on oral microvascular perfusion. Chronic tobacco use is believed to disrupt oral microvascular perfusion, leading to a higher risk of periodontal disease. Repeated vasoconstrictive effects of nicotine may contribute to this disruption, reducing blood flow and oxygen delivery to tissues. Chronic tobacco users, show lower bleeding tendency in the gingiva and tongue, indicating lower perfusion in the oral mucosa. The vasoconstrictor effect of nicotine and decreased endothelial NO synthesis may contribute to this reduced perfusion. Tobacco smoke contains reactive oxygen species (ROS) that may

decrease NO bioavailability, leading to vasoconstriction and higher blood pressure. (8)

Effects on periodontal angiogenesis

Contrary to the increased angiogenesis observed in periodontal disease, chronic tobacco use has been linked to suppressed angiogenesis in patients with the disease. Patients with periodontal disease have higher levels of pro-angiogenic mediators such as vascular endothelial growth factor (VEGF) andbasic fibroblast growth factor (b-FGF), which encourage a greater capillary density and a propensity for bleeding. On the other hand, chronic tobacco use, especially smoking, inhibits angiogenesis, which lowers gingival perfusion and the propensity to bleed. The reason for this suppression is that smokers have lower levels of pro-angiogenic mediators than non-smokers do, such as VEGF and b-FGF. (8)

Etiological Factors	Changes on Periodontal Tissue
Microbiological	Increase in complexity of microbiome and colonization of periodontal pocket by periodontal pathogens
Immunological	Increase in tumour necrosis factor, neutrophil collagenase and elastase in gingival crevicular fluid. Increased production of prostaglandin E_2 by monocyte in response to lipopolysaccharide
Physiological	Decreased blood vessels with increased inflammation. Decreased gingival crevicular fluid flow and subgingival temperature. Bleeding on probing present

Effects of smoking on gingivitis and periodontitis:

GINGIVITIS

- Gingival inflammation and bleeding on probing is decreased (7)

PERIODONTITIS

- Prevalence and severity of periodontal destruction is increased.
- There is increased pocket depth, attachment loss and bone loss.
- Rate of periodontal destruction is increased.
- Tooth loss is increased
- Prevalence with increased number of cigarettes smoked per day is increased
- Prevalence and severity with smoking cessation is decreased (7)

Effects of smoking on the response to periodontal therapy

NONSURGICAL THERAPY

- Clinical response to root surface debridement is decreased
- Reduction in probing length is decreased
- Gain in clinical attachment levels is decreased
- Negative impact of smoking is decreased along with increased level of plaque control (7)

MAINTENANCE CARE

- Probing depth and attachment loss is increased during maintenance therapy
- Disease recurrence in smokers is increased
- Need for retreatment in smokers is increased
- Tooth loss in smokers after surgical therapy is increased (7)

SURGERY AND IMPLANT THERAPY

- Probing depth reduction is decreased and there is increased gain in clinical attachment levels after access flap surgery
- Deterioration of furcation is increased after surgery
- Gain in clinical attachment levels, bone fill is decreased and there is increase in recession and membrane exposure after guided tissue regeneration
- Root coverage after grafting procedures is decreased for localized gingival recession
- Probing depth reduction is decreased after bone graft procedures
- Implant failure and peri-implantitis risk is increased (7)

Effect of smoking in Implantology and osseointegration

Many of the existing studies on implant outcomes in smokers are retrospective or consist of case series,

incorporating a range of implant designs and surfaces like machined titanium, hydroxyapatite, and rough titanium, often within the same patient cohort. Smokers generally exhibit at least twice the implant failure rate compared to nonsmokers. Smoking is recognized as a predisposing factor for implant failure, especially in cases of repeated failures in the same patient. Nonetheless, survival rates range from 80% to 100% in smokers versus 93% to 98% in nonsmokers. (9)

A study initiated in 1991 involved 2887 implants, with 62% being hydroxyapatite-coated, placed across over 800 patients at 32 clinical sites. After three or more years, implant failure rates were 8.9% in smokers compared to 6% in never or former smokers. The administration of pre-operative antibiotics reduced failure rates in smokers by over 10%, while yielding a 3% reduction in the nonsmoker or former smoker group. Notably, maxillary implant failure rates among smokers (10.9%) were higher than those reported for nonsmokers or former smokers (6.4%). Other studies found minimal differences in implant loss in the anterior mandible between smokers and nonsmokers, although longitudinal bone loss around implants was greater for smokers in this location. (9)

While most studies focused on delayed implant placement, a retrospective analysis in private practice found smoking was not a significant factor in the failure of immediate implants. Some researchers argue that the introduction of moderately rough titanium surface implants has minimized disparities between smokers and nonsmokers, though the evidence from available studies is mixed. For instance, studies assessing early healing reported similar bone loss rates in smokers and nonsmokers with rough-surfaced titanium implants. Longer-term studies indicated that moderately rough-surfaced titanium implants experience greater bone loss in smokers, particularly in the maxillary arch. (9)

Shenava et al. reported a survival rate of 30.95% for implants in patients with >10 years of smoking versus 69.05% for those with <10 years of smoking. In addition, they found a higher incidence of implant failure with cigarette consumption of >20 packets/year compared to a consumption of <20 packets/year, a difference which was not found to be statistically significant. (9)(17)

A systematic review by Heitz-Mayfield and Huynh-Ba demonstrated an increased risk of peri-implantitis among smokers. In a follow-up study of 10 years, DeLuca et al. reported a significantly higher failure rate among smokers compared to nonsmokers. (10) There is particular evidence that smoking is a risk factor for peri-implantitis, with some studies showing that smoking has adverse effects on the treatment outcomes of peri-implantitis (11)(17)

In grafts of maxillary sinuses, smokers showed a 12.7% implant failure rate compared to 4.8% in nonsmokers, consistent with findings from Kan et al. Who reported a 17.1% implant loss rate in smokers versus 7% in nonsmokers following maxillary sinus augmentation over an average 41.6-month follow-up. (12)(13) A retrospective cohort study encompassing maxillary sinus and ridge augmentation procedures indicated smokers had a 4.4 times higher risk of implant failure, yet overall survival rates at 1 and 5 years remained 95.2% and 90.2%, respectively. Nevertheless, smokers experienced more postoperative complications after onlay bone grafting, such as graft exposure necessitating removal. (12)(17)

5-A for smoking cessation

- **ASK**- Identify and document tobacco use status for every patient at every visit.

- **ADVICE**- In a clear, strong, and personalized manner, urge every tobacco user to quit.
- **ASSESS**- Ask every tobacco user if he or she is willing to make a quit attempt at this time
- **ASSIST**- For the patient willing to make a quit attempt, use counselling, pharmacotherapy, and supplementary materials to help him or her quit.
- **ARRANGE**- Schedule follow-up contact, preferably within the first week after the quit date. (14)

5-R for smoking cessation

- **Relevance**- Encourage the patient to indicate why quitting is personally relevant.
- **Risks**- Ask the patient to identify potential negative consequences of tobacco use.
- **Rewards**- Ask the patient to identify potential benefits of stopping tobacco use.
- **Roadblocks**- Ask the patient to identify barriers or impediments to quitting.
- **Repetition**- The motivational intervention should be repeated every time an unmotivated patient has an interaction with a clinician. (15)

Conclusion

Smoking is a well-established risk factor for periodontal disease. It alters the human oral microflora and immune response, leading to the destruction of the tooth's supporting tissues. A challenging aspect is that symptoms of periodontal disease are often raised in smokers, which can delay patients seeking help until the condition has significantly progressed, making poor prognosis or even too late in some cases.

References

1. Tea Borojevic. Smoking and Periodontal Disease. 2012; 24(4): 274–276.
2. Pihlstrom BL, Michalowicz BS, Johnson NW. Periodontal disease. The Lancet. 2005;366(9499):1809–1820.
3. https://en.m.wikipedia.org/wiki/List_of_tobacco_products
4. https://www.cdc.gov/nchs/nhis/tobacco/tobacco_glossary.htm#:~:text=Every%20day%20smoker%3A%20An%20adult,at%20the%20time%20of%20interview.
5. Mark David Atkinson, Jonathan Ian Kennedy, Ann John, Keir E Lewis.Development of an algorithm for determining smoking status and behaviour over the life course from UK electronic primary care records. 2017 DOI:10.1186/s12911-016-0400-6
6. https://www.verywellhealth.com/definition-of-pack-years-of-smoking-2249140
7. Michael G. Newman, Henry H. Takei, Perry R. Klokkevold, Fermin A. Carranza. Smoking and Periodontal disease. Carranza's Clinical Periodontology, Tenth edition 2006
8. Henrique Silv. Tobacco Use and Periodontal Disease—The Role of Microvascular Dysfunction. 2021, 10(5), 441;DOI: 10.3390/biology10050441
9. Shenava S., Singh P., Sharath B.C., Kumar V., Jyoti B., Sharma S. Co-relation between smoking and bone healing around dental implants: A clinical study. J. Int. Oral Health. 2016;8:1–3
10. Heitz-Mayfield L.J., Huynh-Ba G. History of treated periodontitis and smoking as risks for implant therapy. Int. J. Oral Maxillofac. Implant. 2009;24:39–68
11. Haas R., Haimböck W., Mailath G., Watzek G. The relationship of smoking on peri-implant tissue: A retrospective study. J. Prosthet. Dent. 1996;76:592–596. Doi: 10.1016/S0022-3913(96)90435-7.
12. Mahmoud Abu-Ta'a. The Effects of Smoking on Periodontal therapy - An Evidence Based Comprehensive Literature Review. 2014 DOI:10.4236/ojst.2014.43022

13. Galindo-Moreno, P., Fernández-Jiménez, A., O'Valle, F., Silvestre, F.J., Sánchez-Fernández, E., Monje, A. And Catena, A. (2013) Marginal Bone Loss in Implants Placed in Grafted Maxillary Sinus. Clinical Implant Dentistry and Related Research. http://dx.doi.org/10.1111/cid.12092
14. Peter J. Lawson, Susan A. Flocke, and Brad Casucci. Development of an Instrument to Document the 5A's for Smoking Cessation doi: 10.1016/j.amepre.2009.04.027
15. https://www.nysmokefree.com/CME/PageView.aspx ?P=50&P1=5027
16. Heasman L, Stacey F, Preshaw PM, et al: The effect of smoking on periodontal treatment response: a review of clinical evidence, J Clin Periodontol 33:241-253, 2006.
17. Marwa Madi, Steph Smith, Sami Alshehri, Osama Zakaria, and Khalid Almas. Influence of Smoking on Periodontal and Implant Therapy: A Narrative Review. 2023; 20(7): 5368.