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SMOLDERING THREAT: EXPLORING THE IMPACT OF SMOKING ON PERIODONTAL HEALTH

Periodontology

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ABSTRACT

Etiology of inflammatory periodontal disease is multifactorial with both infectious and inflammatory components. The etiological significance of biological and behavioral risk factors includes smoking. Smokers have been classified as current smokers, former smokers and passive smokers. Byproducts of smoking inhibit the mechanisms that restrict the growth of pathogenic bacteria in the oral cavity and gives a favourable environment for bacteria in the mouth like P.gingivalis, P.intermedia and A.actinomycetemcomitans. This paper reviews the effects of smoking on various aspects of periodontal disease process and focuses on the change brought about by smoking and its effect on the initiation and progression of periodontal diseases.

KEYWORDS

: Smoking, Effects On Plaque, Risk, Periodontal Diseases

INTRODUCTION:

Smoking, an addictive habit, is the cause of preventable death and disease in the Western world. It is the major risk factor for periodontitis. First introduced in Europe in the 16th century, tobacco smoke can produce local as well as systemic effect. It adversely impacts the clinical outcome of non-surgical and surgical periodontal therapy and the long term success of implant placement. It is an identified and proven risk factor that alters or compromises the physiological, immunological, and biochemical functions of the periodontium. With that said, smoking can encourage the early stages of periodontal lesions and is considered a risk factor in periodontal diseases. Prolonged irritation from tobacco smoke can lead to nicotina stomatitis, benign tumours, smoker's keratosis and leukoplakia.

Components of cigarette smoke:

Tobacco contains benzene, formaldehyde and ammonia.[1] Benzene is a solvent used in fuel manufacture. Formaldehyde is a highly poisonous colourless liquid. Ammonia is a chemical found in cleaning fluid.

Tobacco smoke - grouped under gas or vapour phase. Tobacco smoke contains a group of chemicals including polycyclic aromatic hydrocarbons, toluene, aromatic amines or aldehydes. The chemical composition varies according to the frequency, intensity, volume and duration at different stages of cigarette consumption.

Hydrogen cyanide, poisonous gas used in the manufacture of plastics, dyes and pesticides. Cadmium is extremely poisonous metal found in batteries. Acetone is a solvent found in nail polish remover. Potent carcinogens are nitrosamines, polycyclic aromatic hydrocarbons and radiation emitting polonium.

Toxicity:

Different compounds have been identified in tobacco smoke that occur in concentrations harmful to health. Some of these substances are carcinogenic; the important carcinogens are the polycyclic aromatic hydrocarbons and N-nitroso compounds found in tar residue.[2]

Noxious substances like benzanthracene and hydrogen cyanide are present in tobacco smoke. Alkaloid nicotine is the major substance present in tobacco smoke. Even though nicotine has actions on almost all the organs of the body, it has a predilection for brain and other nervous tissues.

Pharmacology of tobacco products:

Components of inhaled smoke contains nicotine, carbon monoxide. Nicotine is the most pharmacologically active compound in tobacco smoke. Nicotine is absorbed through lung alveoli and it can also be absorbed through oral mucosa in sufficient quantities to have pharmacological effect. During smoking nicotine increases heart rate, cardiac output and blood pressure by autonomic stimulation that affects peripheral vasoconstriction.[3] It directly acts on the blood vessels and capillaries to produce vasoconstriction.

Carbon monoxide reduces the amount of oxygen carried in the blood and this changes the consistency of the blood. Thicker consistency of the blood puts the heart under increased strain as it pumps blood around the body. Irritants found in tar damages the lungs causing narrowing of the bronchioles and damage the cilia which protects the lungs from infection.

Effect on dental tissues:

Tobacco smoking produces brown or black stains on the tooth surface, due to tar products of tobacco consumption. The degree of staining in smokers does not correlate with the amount of tobacco consumed, but depends on a considerable degree on pre-existing acquired coatings that attaches to tobacco products on the tooth surface.[4] Experimental studies show poor oral hygiene in smokers than nonsmokers.[5]

Effect on calculus formation:

Studies stated that there was a positive correlation between smoking and calculus formation. The more tobacco the subjects smoked, the greater were the quantities of calculus.[6,7] NHANES in the United States reported that the smokers have more calculus than nonsmokers and it might be due to an effect of tobacco smoke upon properties of saliva.[8]

Smoking markedly increased the flow rate of saliva which is a reflex phenomenon produced by irritant particulate matter of the smoke. An increase of parotid salivary flow results in raise of pH and calcium concentration of parotid saliva which produces changes that favour the precipitation of calcium phosphate. This favours the statement that smoking may increase the mineralisation of saliva.

Effects on exocrine glands:

The alterations of the salivary flow or microflora may be responsible for the increased calculus prevalent in smokers. Parotid flow rate was increased following cigarette consumption. In novice smoker, increase flow of saliva is noted and in regular smoker there was no significant alteration in salivary secretion, whereas calculus formation is more

abundant. The increase in salivation in smokers is attributed to the smoke irritants and the acidity of cigarette smoke.[9] Nicotine present in tobacco smoke increases the salivary and bronchial secretions followed by inhibition of the secretions. Vasoconstriction of the peripheral blood vessels caused by smoking can also affect the periodontal tissue in smokers.

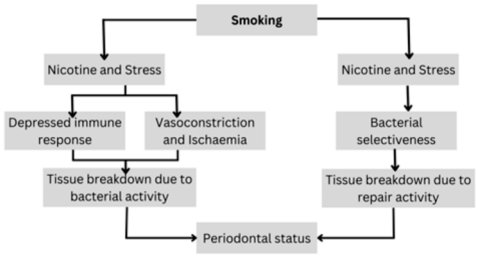
Clinical features of smoking associated periodontitis:
Smokers seem to have a decreased severity of gingival inflammation and it was coincident with decreased oral hygiene and due to vasoconstriction of gingival blood vessels, but may also be attributed to the heavier keratinization of gingiva in smokers.

In clinical appearance of smoking associated periodontitis, the gingiva tends to be fibrotic with thickened rolled margins, minimal gingival redness or oedema relative to disease activity, relatively severe and widespread disease compared with nonsmokers of same age, greater pocketing in anterior and maxillary palatal sites, gingival recession in anterior segment, not associated with periodontal status.[10] Increased plaque and calculus scores are also seen in smoking associated periodontitis. Clinical characteristics of smoking associated periodontitis includes relatively early onset of disease at age 20 or 30 years, rapid disease progression, minimal reduction in gingival pocket depth following scaling, repocketing within 1 year of surgical treatment and resistance to conventional therapy.

Periodontal disease, Attachment loss and Alveolar bone loss in smokers:
Cigarette smokers have significantly greater probing depth and bone loss, increased tooth mobility than nonsmokers. It affects the periodontal ligament by interfering with host response mechanism. Nicotine present in the root surface of periodontally diseased teeth in smokers acts to affect the fibroblasts by increasing the production of collagen and also impairs its secretion. In smokers cell structure of the fibroblasts changed and they were not firmly attached to the tooth surface and thus it makes more susceptible to periodontal damage.

Smokers suffer more tooth loss than nonsmokers, and smoking may be the possible causative factor. Clinical and radiological survey done by Feldman et al concluded that the mean periodontal pocket depth and alveolar bone loss are significantly higher in cigarette smokers compared to nonsmokers.[11]

Pathophysiology of periodontitis:



Courtesy: Dr.Anto et al, 1996[12]

Pre-existing gingivitis, stress and smoking form a triad of interrelated predisposing factors in the etiology of periodontal diseases. Host immuno-inflammatory response is activated in the presence of specific pathogenic bacteria. The antibody and PMNs are produced. Initiation and progression of periodontal disease is mainly due to the release of cytokines, PGE2 and MMP into the connective tissue and bone metabolism.

Microbiology:
Cigarette smoking could cause a lowering of the oxidation - reduction potential (Eh) that could cause an increase in anaerobic plaque bacteria.

Tobacco smoke contains phenols and cyanides, which can account for antibacterial and toxic properties. Smokers harbour significantly higher levels of T.forsythia than non-smokers with greater risk of infection.[13]

Branhemella catarrhalis, Neisseria flava and Neisseria sicca are the species of gram negative bacteria that are more susceptible to cigarette smoke than the species of gram positive bacteria such as Streptococcus mitis, S.salivarius and S.sanguis.

Acute Necrotizing Ulcerative Gingivitis (ANUG):
Possible correlation between tobacco smoking and ANUG is noted in a series of studies and it was found that with an increase in the use of tobacco, there was an increase in the frequency of ANUG.[14] According to Pindborg the tar in the smoke exerts a direct irritating effect on the gingiva giving rise to gingivitis.[15] Nicotine could cause vasoconstriction, interfering with the nutrition of the gingiva, which becomes less resistant to infection. The possible mechanisms for the increased susceptibility of ANUG in tobacco smokers are

1. Vasoconstriction of gingival blood vessels
2. Decreased activity of oral leucocytes
3. Proliferation of anaerobic microorganisms
4. Stress

Smoking activates the release of epinephrine. It promotes contraction of peripheral vessels, reducing blood flow to the gingiva. Imbalance of defensive reparative mechanism allows the initiation of the disease due to reduction in blood flow to the gingiva. Secondary to stress, if changes in gingival crevicular fluid chemical composition, could induce a shift in the composition of the bacteria towards more periodontopathic flora, could conceivably facilitate the initiation of ANUG.

Effects on immune system:
Polymorphonuclear leukocytes (PMN) are the first line of defence in an inflammatory response and decreased effectiveness of PMN may result in the degranulation and release of more lysosomal enzymes that result in severity of periodontal disease. Smokers have higher blood PMN count than do nonsmokers.

Vasoconstrictive effect of nicotine causes a reduction in the gingival blood flow, decreases the number of cells, the amount of oxygen and blood constituents that reach the gingiva and reduces the capacity to remove tissue waste products which weakens the defence reparative posture of the gingiva.

Smoking directly weakens the host defense response by depressing the primary and secondary immune responses by reducing the levels of circulating antibody and by depressing the chemotactic and phagocytic activities of oral PMN. Fewer crevicular PMN and less crevicular phagocytosis could decrease the release of lysosomal enzymes and thus decreases the level of inflammation in the superficial layers of periodontal tissues. Suppression of gingival inflammation may be due to high cAMP level together with lowered GCF flow in heavy smokers.[16]

Effect on periodontal wound healing:
Controversial statements are there on effects on periodontal wound healing in smokers. Smokers suffer from painful socket after removal of impacted third molars. There are studies that show probing depth reductions after modified Widman flap were significantly less in smokers than nonsmokers. There is good evidence that smoking does have a detrimental effect on the outcome of periodontal treatment. Study done by Miller found a strong correlation between smoking and failure to obtain optimum root coverage after the soft tissue autograft in 100 graft sites.[17]

Healing following scaling and root planing, an improved tissue tone which is more resistant to pocket probing force and on examination increase in clinical attachment level is noticed. Poorly functioning fibroblasts may not produce collagen fibers as efficiently and the gingival tissue support and adaptation will be impaired or at least slowed.[18]

DISCUSSION:
Periodontal disease contributes significantly to the global burden of oral diseases. The role of smoking as a contributing factor in the progression of periodontal disease process has been long suspected and large number of studies have been published. Smoking is now considered as an important risk factor for periodontal disease.

Smoking increases the risk of periodontal disease by two to seven fold. Smoking increases the damage that periodontal disease does and decreases gingival response to treatment, possibly causing refractory

disease. Smokers are four times as likely to have periodontitis as nonsmokers.

Smoking alters the body's immune response and causes bad breath. Salivary immunoglobulin A has also been found to be significantly decreased in smokers. Continuous exposure to smoke has been shown to affect both humoral and cellular immune response, initially there is an acute depression followed by stimulation and finally depression as the immune sets in. Nicotine is also believed to affect T lymphocytes. It increases the number, decreases the proliferation, decreases their response to antigen. It also decreases T helper cells and increases T suppressor cells.

Smoking decreases salivary IgA and serum IgG2 levels against A.actinomycetemcomitans. Smoking decreases the proliferation of T and B lymphocytes resulting in diminished production of protective antibodies. IgM, IgD and IgE are significantly elevated in smokers. Decreased ability of T cell proliferation and reduced natural killer cell activity against cultured cancer cells indicated deficient cell mediated immune response in smokers. Non surgical therapy in smokers showed poor clinical response. Surgical therapy in smokers also showed poor prognosis and increased risk of implant failure and peri-implantitis. It is clear that smokers presenting with periodontitis is difficult to treat with conventional therapy without quitting the habit. Asking about the use of tobacco, advising them to quit smoking, assessing their willingness to quit the habit, assisting them in quitting attempt and arranging for follow up will help in managing the patient with smoking habit.

SUMMARY AND CONCLUSION:

Smokers tend to accumulate more amount of calculus but there is no evidence that smoking increases the rate at which plaque develops. Smoking can predispose an individual to initiation and progression of periodontal disease. Smoking appears to suppress visible gingival inflammation at a clinical level in response to plaque accumulation. This might be the reason for failure to recognise the prevalence of gingivitis in early stage itself. By diminishing the oral cellular immunity tobacco may have a more destructive effect in predisposition of periodontal diseases. Smoking impairs the periodontal wound healing after non-surgical and surgical therapy by decreasing the cellular and humoral immune components in the region of gingival sulcus in response to decreased blood flow and crevicular fluid level. Smoking does not ordinarily lead to tooth loss but has harmful effect on teeth. This statement and evidence on effects of smoking might stimulate the dentist to advise the patient to stop smoking by pointing out the risks involved and positive benefits gained from stopping the habit.

REFERENCES:

1. How Tobacco Smoke Causes Disease: The Biology and Behavioral Basis for Smoking-Attributable Disease: A Report of the Surgeon General. Atlanta (GA): Centers for Disease Control and Prevention (US); 2010. 3, Chemistry and Toxicology of Cigarette Smoke and Biomarkers of Exposure and Harm. Available from: https://www.ncbi.nlm.nih.gov/books/NBK53014/
2. Li Y, Hecht SS. Carcinogenic components of tobacco and tobacco smoke: A 2022 update. Food Chem Toxicol. 2022 Jul;165:113179. doi: 10.1016/j.fct.2022.113179. Epub 2022 May 25. PMID: 35643228; PMCID: PMC9616535.
3. Benowitz NL. Pharmacology of nicotine: addiction, smoking-induced disease, and therapeutics. Annu Rev Pharmacol Toxicol. 2009;49:57-71. doi: 10.1146/annurev.pharmtox.48.113006.094742. PMID: 18834313; PMCID: PMC 2946180.
4. Karanjkar RR, Preshaw PM, Ellis JS, Holliday R. Effect of tobacco and nicotine in causing staining of dental hard tissues and dental materials: A systematic review and meta-analysis. Clin Exp Dent Res. 2023 Feb;9(1):150-164. doi: 10.1002/cre2.683. Epub 2022 Nov 13. PMID: 36372903; PMCID: PMC9932248.
5. Arowojolu MO, Fawole OI, Dosumu EB, Opeodu OI. A comparative study of the oral hygiene status of smokers and non-smokers in Ibadan, Oyo state. Niger Med J. 2013 Jul;54(4):240-3. doi: 10.4103/0300-1652.119627. PMID: 24249949; PMCID: PMC 3821224.
6. Bergström J. Tobacco smoking and supragingival dental calculus. J Clin Periodontol. 1999;26(8):541-547. doi:10.1034/j.1600-051x.1999.260808.x
7. Bergström J. Tobacco smoking and subgingival dental calculus. J Clin Periodontol. 2005;32:81-8. Doi:10.1111/j.1600-051X.2004.00638.x.
8. Tomar SL, Asma S. Smoking-attributable periodontitis in the United States: Findings from NHANES III. National health and nutrition examination survey. J Periodontol. 2000;71:743–51.
9. Petrušić N, Posavac M, Sabol I, Mravak-Stipetić M. The Effect of Tobacco Smoking on Salivation. Acta Stomatol Croat. 2015 Dec;49(4):309-15. doi: 10.15644/asc49/4/6. PMID: 27688415; PMCID: PMC4945334.
10. Muniandy S. Knowledge on smoking and periodontal disease: A cross-sectional survey among targeted respondents. J Indian Soc Periodontol. 2019 May-Jun;23(3):275-280. doi: 10.4103/jisp.jisp_479_18. PMID: 31143010; PMCID: PMC6519089.
11. Feldman RS, Bravacos JS, Rose CL. Association between smoking different tobacco products and periodontal disease indexes. J Periodontol. 1983;54:481–7.
12. Anto et al. Effects of smoking on periodontium. 1996;20(3):62.
13. Kenney EB, Saxe SR, Bowles RD. The effect of cigarette smoking on anaerobiosis in the oral cavity. J Periodontol. 1975;46(2):82-85. doi:10.1902/jop.1975.46.2.82
14. Luzzi LI, Greghi SL, Passanezi E, Sant'ana AC, Lauris JR, Cestari TM. Evaluation of clinical periodontal conditions in smokers and non-smokers. J Appl Oral Sci. 2007 Dec;15(6):512-7. doi: 10.1590/s1678-77572007000600011. PMID: 19089190; PMCID: PMC4327501.
15. Pindborg JJ. Tobacco and gingivitis. II. Correlation between consumption of tobacco, ulceromembranous gingivitis and calculus. J Dent Res. 1949;28(5):460–463.
16. Qiu F, Liang CL, Liu H, Zeng YQ, Hou S, Huang S, Lai X, Dai Z. Impacts of cigarette smoking on immune responsiveness: Up and down or upside down? Oncotarget. 2017 Jan 3;8(1):268-284. doi: 10.18632/oncotarget.13613. PMID: 27902485; PMCID: PMC 5352117.
17. Miller PD. Root coverage with free gingival graft. Factors associated with incomplete root coverage. J Periodontol 1987;58:674-681.
18. Ah MK, Johnson GK, Kaldahl WB, Patil KD, Kalkwarf KL. The effect of smoking on the response to periodontal therapy. J Clin Periodontol. 1994;21(2):91-97. doi:10.1111/j.1600-051x.1994.tb00285.x