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Smoking and Periodontal Disease

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Abstract:
Cigarette smoking is a well-established risk factor for periodontal and oral related disease. It causes harm to the oral cavity majorly affecting the soft tissues – periodontium and oral mucosa and the hard tissues – teeth[1]. Periodontitis is a group of inflammatory diseases which affects the supporting tissues of the tooth - periodontium. The components of periodontium are: gingiva, alveolar bone and periodontal ligaments. Tobacco smoking is one of the modifiable risk factors and has enormous influence on the development, progress and treatment results of periodontal disease[2]. The relationship between smoking and periodontal health was found as early as the middle of last century. Smoking is an independent risk factor for the initiation, extent and severity of periodontal disease. Additionally, smoking can lower the chances for successful oral and other treatment.[3]

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I. Introduction:

Smoking can affect the function and proliferation of periodontal cells such as gingival fibroblasts, periodontal membrane cells, periodontal ligament cells and other cells, thus inducing cell apoptosis.

Research evidence suggests that smokers have a higher tendency to problems such as teeth and bone loss and gingival recession, invasion of periodontal disease, inhibit the autoimmune defence, and aggravate the inflammation reaction to damage and destroy the alveolar bone compared to non-smokers, and to the formation of periodontal pockets, which increase the probability to suffer from more severe periodontal disease. [4]

Few studies show that periodontal disease is an independent risk factor for oral cancer, hence smoking promotes this correlation and smokers are 7 to 10 times more likely to develop oral cancer and 3 times more likely to develop a second primary cancer than non-smokers.[5][6]

In the 2004 United States Surgeon General's report, *The Health Consequences of Smoking*, four major conclusions were listed:

1.Smoking harms nearly every organ of the body, causing many diseases and reducing the health of smokers in general.

2.Quitting smoking has immediate as well as long-term benefits, reducing risks for diseases caused by smoking and improving health in general.

3.Smoking cigarettes with lower machine-measured yields of tar and nicotine provides no clear benefit to health

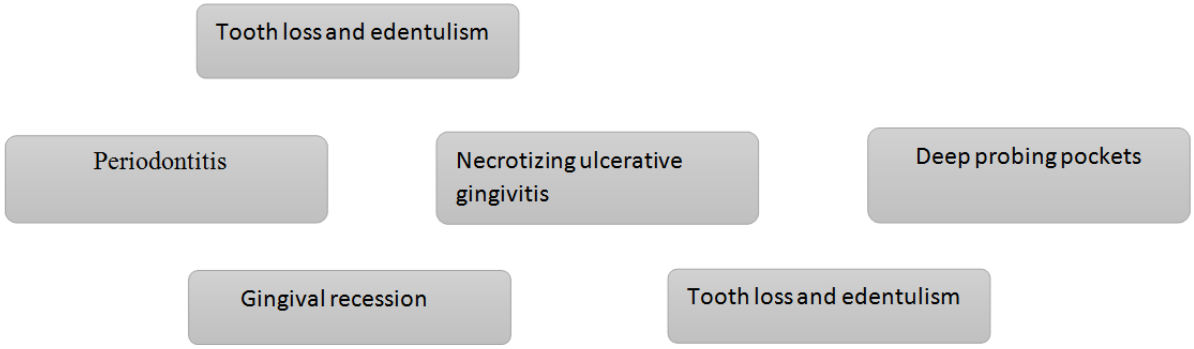
4. The list of diseases caused by smoking has been expanded to include abdominal aortic aneurysm, acute myeloid leukemia, pancreatic cancer, pneumonia, and periodontitis. These are in addition to diseases previously known to be caused by smoking, including bladder, esophageal, laryngeal, lung, oral, and throat cancers, as well as reproductive effects and sudden infant death syndrome[7]

SMOKING: ITS EFFECT ON ORAL HEALTH

The adverse effects of tobacco smoking on oral health are well established. It includes common and rare conditions, from benign to life-threatening diseases such as discoloration of teeth and dental restorations, bad breath, taste and smell disorders, impaired wound healing, periodontal disease, short-term and long-term implant success, oral mucosal lesions such as smoker's melanosis and smoker's palate, potentially malignant lesions and oral cancer.[8]

HOW SMOKING AFFECTS PERIODONTIUM

Smoking is a significant risk factor for the development of periodontal diseases. Risk calculations suggested that 40 per cent of chronic periodontitis cases may be attributed to smoking. The following pictorial representation shows how the periodontium if affected by tobacco smoking



The effect of smoking on the periodontal tissues is dose-dependent. Both daily consumption quantity and duration of smoking are related. [9]

PERIODONTIUM: STRUCTURE, COMPONENTS, HISTOLOGY AND ITS FUNCTION

The periodontium is the specialized tissues that both surround and support the teeth, maintaining them in the maxillary and mandibular bones.

Each of these components is distinct in location, architecture, and biochemical properties, which adapt during the life of the structure. It consists of four principal components,

Periodontal ligament	Allows the tooth to be suspended in the alveolar bone and respond to the forces.
Gingiva	Surrounds the tooth structure and protects it
Cementum	Adapts to wear on the occlusal surfaces of the teeth by apical deposition
Alveolar bone	Hold the soft tissues

A constant state of balance always exists between the periodontal structures and the external forces.[10]

EFFECT OF TOBACCO SMOKING ON VARIOUS CELLS AND TISSUES

- PERIODONTAL CELLS

Periodontal ligament (hPDLCs) cells are the main cell components in the periodontal membrane. They contain chemotactic adhesion, proliferation, biosynthesis and differentiation into cementite and osteoblasts. Many of the underlying effects of tobacco products on periodontal tissues may be due to a direct inhibition of normal fibroblast function. These cells are sensitive to nicotine. Nicotine activates the autophagy of hPDLCs by increasing the number of autophagosomes.

-Periodontal attachment loss: smokers had more attachment loss than never smokers particularly at maxillary lingual sites and at lower anterior teeth.[11][12]

- GINGIVAL CREVICULAR FLUID

Gingival crevicular fluid (GCF) is an inflammatory exudate. It is in present gingival sulci. GCF volume is a well-known marker of gingival health. The amount of gingival fluid is greater when inflammation is present [13][14]

-GCF volume change: The amount of gingival fluid is greater when inflammation is present and is sometimes proportional to the severity of the inflammation. Smoking was reported to produce an immediate transient but marked increase in gingival fluid flow but the cumulative effect of smoking in GCF have not been examined. smoking causes vasoconstriction as a consequence, gingival blood flow increased during smoking. On the other hand lower GCF volumes have been reported.[25] The lower GCF volumes were linked with diminished gingival blood flow in smokers.[15]

- ALVEOLAR BONE

Tobacco smoking can adversely affect the physiology of alveolar bone. Various studies were done to investigate the effect of smoking on alveolar bone[16]
Study 1 – Alveolar bone resorption.
Radiographic examination of all present teeth was conducted: (Age group:20-60)

30 smokers (12 men and 18 women)	30 non-smokers (13 men and 17 women, control group)
high values of alveolar bone resorption (3.16 ± 2.07 mm)	Less when compare to smokers (1.72 ± 1.02 mm).

Conclusion: The present results have shown that smoking increases alveolar bone resorption and that the period of smoking affects the level of resorption

Study 2 - Smokers and non-smokers were compared with respect to alveolar bone height.

The study covered 235 subjects aged 21- 60 years, 72 of whom were smokers. Oral hygiene status and dental care habits were above average and of equal standard in both groups. Alveolar bone height was assessed on radiographs
Conclusion: Alveolar bone height was significantly reduced in smokers as compared to non-smokers
It is concluded that smoking is a risk factor for periodontal health.[17]

- TOOTH:

Cigarette smoking was associated with higher prevalence of tooth loss at baseline as well as higher incidence of tooth loss during follow-up.[18]

- SALIVA:

Cigarette smoke contains free radicals that can cause cellular damage. It reduced the value of total protein, Ca and Pb of saliva. smoking slightly changes the value of zinc .It did not have an impact on Na, K and Mg of saliva. The mean total protein, calcium and Pb2+of whole saliva in smokers was lower than non-smokers but the difference was not significant. It is noteworthy that zinc concentration of smoker was lower than that non-smoker.[19][20]

COMPARISON BETWEEN A SMOKER AND A NON SMOKER

- Tooth brushing behaviour

Analysis of tooth brushing behaviour in different people shows how the oral cavity and its hygiene maintenance differs accordingly. Smokers have more plaque than non-smokers, and whether higher plaque scores subsequently found in smokers could be explained by differences in toothbrushing time, efficiency and frequency. Studies were done in males and females, male smokers brushed for a shorter time, and had more plaque alter toothbrushing, than male non-smokers. A similar, though non-significant trend was found in females.[21]

- Oral health

The analysis of oral health conditions revealed that 64% of smokers had tooth sensitivity compared with 43% of non-smokers. It has been observed that smoking is a predictor of dentine hypersensitivity. [22]

- Immune response [23][24][25][26][27]

T lymphocytes	Helper t cells	Dendritic cells	NK cells	Macrophages	B cells
-Major subset of immune cells -Mediates adaptive immunity. -Responds to specific antigens through their helper, effector, cytotoxic or regulatory capacities.	Epidemiological studies have suggested that either first hand or second-hand tobacco smoking is an important contributor in the development of many diseases. It's been known that cigarette smoking is a major cause of COPD characterized by chronic airflow obstruction - Crohn's disease (CD) is a chronic inflammatory bowel disease that leads to obvious morbidity and is epidemiologically correlated with cigarette smoking	-DCs are derived from a hematopoietic lineage of bone narrow -Cigarette smoke alters the number, distribution and development of DCs and Langerhans cells (LCs).	It suppress the expression of IFN-γ and TNF-α in human NK cells -Cigarette smoke apparently attenuated the activation and cytolytic capacity of human NK cells with decreased expression of activation marker CD69	Human macrophages to release IL-8, facilitating inflammation rather than directly enhancing their function . -cigarette smoking suppresses the phagocytosis of murine macrophages	Cigarette smoking resulted in higher prevalence of memory B cells in peripheral blood and memory IgG+ B cells in the lung - Smokers with Helicobacter pylori (H. pylori) infection had a lower number and impaired function of regulatory B cells than non-smokers with also H. pylori infection

CALCULUS FORM ATION DUE TO SMOKING

The influence of tobacco smoking on the occurrence and severity of supragingival calculus has received surprisingly little attention. Calculus forming was increased in smokers, due to the increased flow of saliva and concentration of calcium present in fresh saliva of smokers, immediately after smoking. we can observe increased accumulation of supra gingival and subgingival dental calculus.[28]

PERIODONTAL DISEASE ASSOCIATED WITH SMOKING

1. CHRONIC PERIODONTITIS;

-Plaque induced gingival disease
-non plaque induced gingival lesion

Periodontitis, also known as gum disease is a serious gum infection and a chronic inflammatory response leading to irreversible destruction of tissues and bone that support the tooth structure

2. GINGIVAL DISEASE

Its is the bleeding and swelling of gingiva which is early stage of periodontal disease
-Localizes
-generalised

3. AGGRESSIVE PERIODONTITIS

-Localised
-Generalised

4. NECROTIZING PERIODONTITIS

-Necrotizing ulcerative gingivitis (NUG)

It's is also known as Vincent's infection; it is a specific type of gingivitis with characteristic signs and symptoms. Inflammatory condition involves primarily free gingival margin, crest of the gingiva and interdental papillae. When its spreads to the soft palate and tonsillar areas its Vincent's Angina.

-Necrotising ulcerative periodontitis (NUP)

Necrotizing periodontitis (NP), previously referred to as necrotizing ulcerative periodontitis, is a specific subset of necrotizing periodontal diseases (NPD). In general, NP is a reactive, destructive inflammatory process in response to bacterial inflammation and represents the most severe form of biofilm-related diseases[29]

5. ABSCESSES OF PERIODONTIUM

Common and painful dental emergency resulting from bacterial accumulation or foreign body impaction in periodontal pockets. They affect both patients with or without active periodontal disease and require prompt management acute and long-term management.

-Gingival abscess
- Periodontal disease
-Peri coronal abscess

6. PERIODONTITIS ASSOCIATED WITH ENDODONTIC LESION

-Endodontic- Periodontal lesion
-Periodontal- endodontic lesion

RELATIONSHIP BETWEEN SMOKING, PERIODONTAL DISEASE AND ORAL CANCER

Oral cancer is known to have a multi-factorial aetiology tobacco, alcohol and betel quid being the major risk factors. Tooth loss and periodontal disease have been implicated to increase the risk of developing various cancers.

The link between tobacco products and human cancers results from a powerful combination of two factors — nicotine and carcinogens. Nicotine causes addiction and toxicity, but there is no scientific evidence that nicotine is a carcinogen, and nicotine is not classified as a carcinogen by the IARC. However, this addiction causes people to use tobacco products continually, and these products contain many carcinogens. More than 60 carcinogens were found in cigarette smoke and at least 16 in unburned tobacco.[30]

SMOKING: ITS EFFECT ON PERIODONTAL TREATEMENT:

Smokers have a higher risk for recurrence of periodontal disease and the response to non-surgical as well as surgical periodontal treatment is not as good as that of non-smokers. Moreover, there is a dose-response effect in the adverse effects of smoking on periodontal health. Compared to non-smokers, smoker patients with periodontitis tend to respond less favourably to non-surgical and surgical periodontal treatment, and exhibit recurrence more frequently during supportive periodontal treatment.[31]

- NON SURGICAL THERAPY

Smokers do not respond well to periodontal treatment and non-smoker or former smokers do. Most clinical research supports the observation that probing the depth reductions are generally greater in non-smokers than on smokers after non-surgical periodontal therapy. In addition, gains in clinical attachment as a result of non-smokers. When a higher level of oral hygiene was achieved as part of non-surgical between non-smokers and smokers became clinically less significant.[32]

- SURGICAL THERAPHY

The less favourable response of the periodontal tissues to non-surgical therapy that is observed after surgical therapy. In a longitudinal comparative study of the effects of four different treatment modalities smokers consistently showed less pocket reduction and less gain in clinical attachment as compared with non-smokers or former smokers. These difference were evident immediately after the completion of therapy and continued throughout 7 years of supportive periodontal therapy. During the 7 years deterioration at furcation areas was greater in heavy and light smokers and non smokers. Smoking has also been shown to have negative impact on the outcomes of guided tissues regeneration and the treatment of infrabony defects by bone grafts. By 12 months after guided tissue regeneration therapy at deep infrabony defects smokers demonstrated less than half the attachment gain that was observed in non smokers.

Open flap access surgery without regenerative or grafting procedures is a common surgical procedure used for accessing the root and bone surfaces. By 6 months after this procedure smokers showed significantly less reduction of deep pockets even though all of the patient's received supportive periodontal therapy every month for 6 months

Tobacco smoking also affects the outcomes of periodontal plastic surgery. For instance, a systematic review assessed the influence of smoking on the outcomes achieved by root coverage procedure. Significantly greater root coverage and greater gains in clinical attachment level were recorder for non-smokers as compared to smokers after the treatment of gingival recession defects by sub epithelial connective tissue grafting. Furthermore smokers showed significantly fewer sites with complete root coverage that were seen in the non-smokers [33]

- MAINTENANCE THERAPY

The detrimental effect of smoking on treatment outcomes appears to be long lasting and independent of the frequency of maintenance therapy. After 4 modalities of therapy [scaling, scling and root planning, modified Widman flap surgery and osseous surgery], maintenance therapy was performed y a hygienist every 3 months for 7 years. Smokers consistently had deeper pockets than non-smokers and less gain in attachment when evaluated each year for 7-year period. Even with more intensive maintenance therapy given every month for 6 months after flap surgery smokers had deeper and more residual pockets than smokers, smokers had deeper and more residual pockets than non-smokers. Tobacco, smoking was positively associated with tooth loss even when regular recall maintenance care was performed. Similarly smoking had detrimental effect on peri implant tissue status, even when patients are under strict peri implant preventive maintenance care [34]

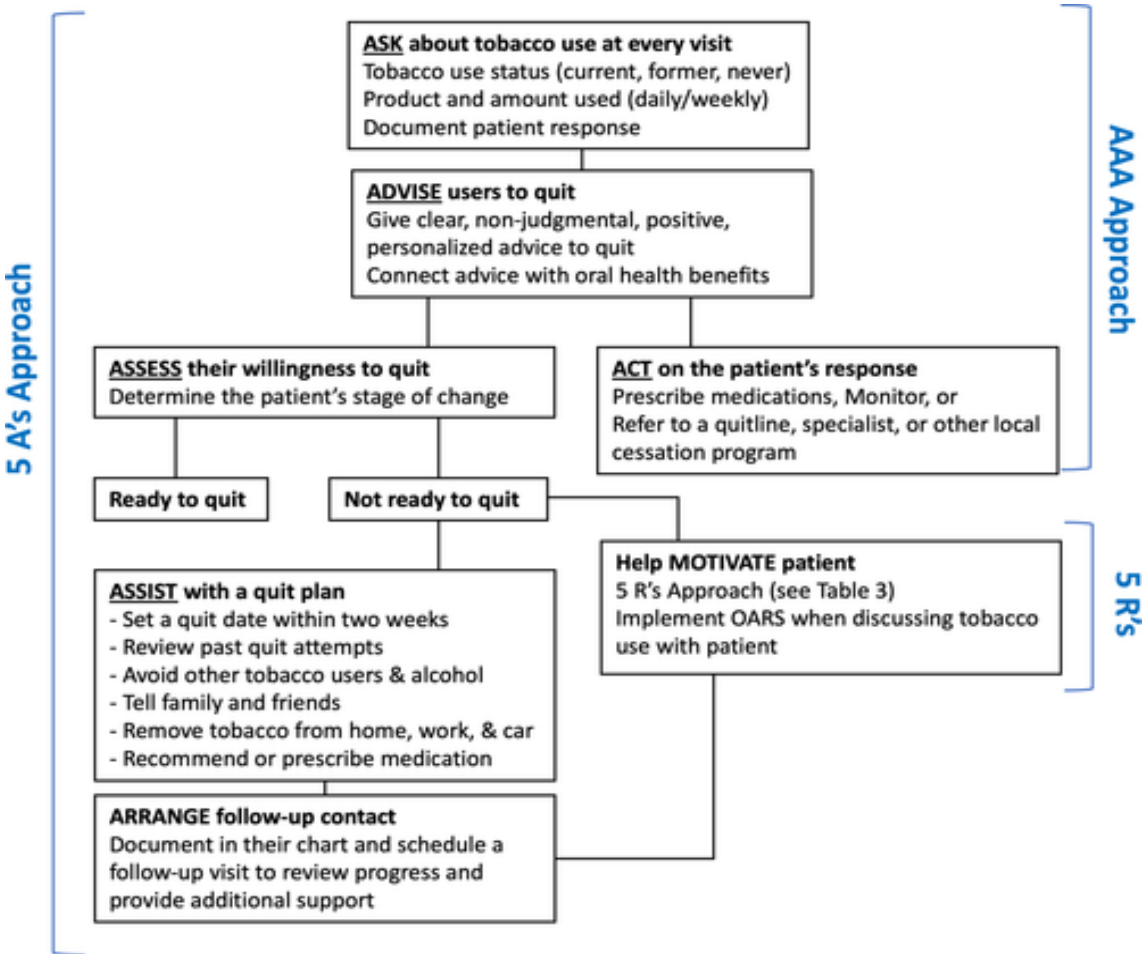
IMPACT OF SMOKING CESSATION ON PERIODONTAL STATUS AND TREATMENT

- TOBACCO SMOKING COUNSELLING: A COMPONENT OF PERIODONTAL THERAPY

Smoking cessation positively influenced periodontal treatment outcomes. When patients received non-surgical therapy as treatment for their periodontitis in addition to smoking cessation counselling for a period of 12 month, those individuals who study had the best response to the periodontal treatment

The benefit of smoking cessation on the periodontium is likely to be mediated through various pathways such as a shift toward a less pathogenic microbiome, the recovery of the gingival microcirculation and improvements in certain aspects of immune inflammatory response [35]

a. THE 5A FOR SMOKING CESSATION



II. Conclusion

In conclusion smoking is a major risk factor for periodontitis and smoking cessation should be integral part of the periodontal therapy among patients who smoke, smoking cessation should be considered a priority for the management of periodontitis in smokers [36]

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