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Literature Review

THE EFFECTS OF NICOTINE ON THE PERIODONTAL TISSUE

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ABSTRACT

Tobacco contains thousands of chemical substances which known to be harmful to periodontal tissues. Nicotine was considered as the most toxic substances to periodontal tissues. The datas in this review indicate that smoking may have a significant role in the initiation and progression of periodontal destruction. The conclusion of this and the other studies indicate that smokers have a less favorable response to periodontal therapy than non smoker. Nicotine is potentially toxic substances that have a detrimental effect on periodontal tissue, by altering the host response or directly damage the cells of normal periodontium.

Key words: *nicotine, risk factor, periodontal disease, vasocontriction, host response*

INTRODUCTION

Smoking had been a human's habit for almost four centuries. At the first time, Columbus brought tobacco to Europe which he gets from the Indians in America. From Europe, smoking was spread throughout the world by Portuguese and Spanish's world exploration and became an epidemic throughout the world. But recent studies found that smoking had a detrimental effect to our health.[1]

Smoking may be considered as a serious health issue as its epidemiological studies had shown that it may have a strong relationship to cancer and may cause physiological disturbances in pulmonary, cardiovascular and gastrointestinal. Smoking was also associated to various changes in the oral cavity which was related to oral cancer. In the last two decades, the awareness of smoking effect was increased, especially on its detrimental effect towards periodontal tissue which may be followed by loose teeth.[2]

Recent researches revealed that smoking might be the most potential risk factor for periodontal disease. Tobacco contains thousands of chemical substances e.c. nicotine, tar, and carbonmonoxydes. Therefore, smoking and periodontal disease may be considered as public health problems.

Nicotine

Nicotine is an alkaloid, colorless, and highly volatile liquid. Nicotine change its color to brown after contact with air and smell like tobacco. Tobacco containes 1–2% of nicotine.[5]

Nicotine is an alkaloid derived from dried leaves of *Nicotiana tabacum* and *Nicotiana rustica*. Nicotine is a tertiary amine of the pyridine and pyrrolidine rings. Nicotine is a weak alkali (pKa=8). At physiologic pH, 31% nicotine will not be ionized, therefore may penetrate the cell membrane. Nicotine is water-and alcohol-soluble. Cigarette smoke is an acidic substances (pH 5.5). In this acidic pH, nicotine is in ionic form and cannot easily penetrate the membrane so nicotine absorption in cheek mucosa occurs only from the smoke.[6]

Nicotine is easily absorbed from the airway, mucous membranes, and skin. Percutaneous nicotine absorption may be poisonous. While smoking, the nicotine is removed from the alveoli into the blood. Under acidic condition in alveolus, small particles undergo diffusion through the alveolar membrane or in the form of nicotine salt will removed as uncharged particles. These particles form nicotine which will be attached to the hair on mucosal lining of airway duct.[6]

Compared to the lipid solubility, pH differences may have stronger influence towards the distribution of nicotine throughout body's organs and tissues. At physiological pH, small changes in pH may be happened due to non ionized substantial changes in nicotine's fraction. Lipid soluble amine bases such as nicotine was known to

have higher levels in tissue than in the blood. Nicotine administered intravenously degrades slower in the arterial blood compared to when the nicotine ia absorbed during smoking. In addition, the nicotine in blood lipid fractions will be degraded completely by the liver.[7]

Inhaled nicotine fraction is metabolized by the lungs. Nicotine's metabolytes are known as konitin and nicotine-N-oxide which is formed from α-carbon oxide and N-oxidation of the pirolidin ring. Half-life nicotine elimination obtained from smoking or parenteral injection is 30–60 menit.[8]

Nicotine and metabolites can be eliminated by the kidneys. The excretion rate of nicotine via urine is dependent upon pH. Under alkaline condition, the excretion rate will be decreased. Nicotine may also be excreted in breast milk in woman.[1]

Once it in the body, it might bind nicotinicacethylcholine receptors located in peripheral (neuromuscular junction, adrenal medulla, autonomic ganglia) and central nervous system. Peripheral effects, included changes in the endocrine, and alteration in metabolic function of systemic blood vasoconstriction. These effects developed into body's tolerance.[6]

Nicotine facilitated platelet adhesion associated with cardiovascular disease and hypertension. Nicotine easily penetrated the blood brain barrier (blood-brain barrier) and stimulated the brain and released several neurochemical acetylcholine, beta-endorphin, dopamine, norepinephrine and vasopressin. In the medulla oblongata nicotine directly effect to the respiratory center. Here, the nicotine in small doses stimulates breathing activity. But in large doses, nicotine will suppress respiration, and an overdose may cause death.[9]

Smoking can affect the behavior due to carbon monoxide and nicotine poisoning to the body. Nicotine stimulates the production of adrenaline and cortisone. This hormone decreases the serotonin activity which is soothing the brain.[10]

When people smoke, the nicotine come into the brain within seconds, which cause someone to feel comfortable. Psychological effects of nicotine can cause addiction. An important indicator of physical dependence is the presence of withdrawal. The signs and symptoms of the withdrawal symptoms are dizziness, constipation, diarrhea, drowsiness, fatigue, unable to sleep, unable to concentrate, and depression. The degree of withdrawal symptoms for each person may be different.[7]

It might be said that smoking was dangerous, so smoking habit should be stopped. Smoking cessation is often as a result of the development of nicotine withdrawal symptoms. Nicotine dependence can be treated by behavior and psychological counseling. The other nicotine replacement are nicotine chewing gum and nicotine transdermal. Transdermal therapy was proven to be more secure and effective to stop smoking. Terapi used for patients who are determined to stop smoking.[1]

Oral cavity is the first organ exposed to harmful effects of smoking. The alteration in oral cavity may be present as it become the first place to absorp any poisonous substances from smoking. The temperature cigarette on lips is approximately 30° C. While the temperature at the ends of burning cigarette is approximately 900° C.[11]

Hot smoke continually blowing into the oral cavity acts as a heat stimulus that causes alteration in blood flow and reduces saliva production. As a result the oral cavity becomes dry and anaerobic, thus providing a suitable environment for the anaerobic bacteria in plaque. Smokers may have a higher risk of periodontal disease than those who do not smoke. Effect of cigarette smoke irritates the gingiva directly and may affect the body through the bloodstream and saliva indirectly.[12]

Periodontal tissues such as gingiva, periodontal ligament, alveolar bone may be damaged by disruption of the normal function of host response to infection and can stimulate the body to destroy the surrrounding healthy tissues.[13]

Nicotine may play a role in initiating the periodontal disease because nicotine may be absorbed by soft tissues in oral cavity including the gingiva through the bloodstream and gingival attachment to the tooth surface and roots. Nicotine can be found on the surface of the tooth's root and the continin as it metabolism result may be found in gingival crevicular fluid.[14]

Periodontitis

Periodontitis is defined as an inflammatory disease of the supporting tissues of the teeth caused by specific microorganism or groups of specific microorganisms, resulting in progressive destruction of periodontal ligament and alveolar bone with pocket formation, recession, or both. Periodontal disease occurs when bacterial toxins and enzymes destroy the supporting tissues of teeth and bone. Plaque, attached to the teeth, may form hard deposits called calculus or tar within 48 hours. It is easily happened in smokers.[15] Once the calculus has been attached to the teeth, it will not be easily cleaned with regular toothbrushing. Scaling is the only available method to remove calculus. Calculus which is located under the gums may cause inflammation and infection in periodontal tissue. Because the symptoms are absent, very few people may recognize this disease at the early phase. People will usually recognize it in later phase. Periodontal disease has some characteristics that can be described clinically.

a. Gingival inflammation

Gingival inflammation and the presence of bleeding after a light touches are the early signs of periodontitis. Healthy gingiva is charaterized with hard-consistency, live pink coloured, and normal contours. There is no sign of bleeding after probing and the patient has no bleeding complaint when brushing the tooth. The severity of inflammation depends on the status of oral hygiene, lack

of oral hiegene may exhibit gingival infection and bleeding when brushing teeth or sometimes spontaneous bleeding.

b. Periodontal pocket

Pocket is a gap between the teeth and gums are interpreted as an increase in the sulcus ginginva be pathological. Normal gingival sulcus has a depth of between 2–3 mm. The pocket depth measurement is an important part in periodontitis' diagnosis. Some factors may cause the deepening of normal gingival sulcus: 1) coronal movement of the gingival due to gingival inflammation; 2) apical displacement of the gingival attachment; and 3) a combination of both. Pocket with a depth of 4 mm is considered as a initial sign of periodontal disease.

c. Gingival Recession

Gingival recession is the exposure of tooth roots, which is happened together with chronic peridontitis but was not considered as a sign of periodontal disease. If there is a recession, pocket depth measurement is only a partial reflection of the amount of the whole periodontal damage.[16]

DISCUSSION

WHO developed a Community Periodontal Index of Treatment Needs (CPITN). This index is received and used by several countries. Some researchers conducted a study to compare between smokers and nonsmokers using CPITN reference. It was found that smoking was harmful to health. In oral region it may cause periodontal disease. Several studies using CPITN revealed that smoking may harm periodontal health.[17]

There was a strong relationship between smoking and periodontal disease, it is expected that the smokers have more routine periodontal treatment. The researchers reported that smokers have a higher CPITN scores than non-smokers. Smoking can also change the body's response to various periodontal therapies. Besides, smoking may also play a role in the development of refractory periodontitis.[17]

The number of cigarretes consumption determined the severity of periodontal destruction, especially the risk of oral cancer. There were some differences in the severity of periodontal destruction indicated that smoking might worsen host's response to periodontal therapy. This means that smoking had a negative impact on periodontal treatment.[18] Healing following periodontal therapy in smokers might need a longer time because the attachment of connective tissue and collagen fibers was inhibited, so it would disturb healing and tissue regeneration after treatment.[19]

Nicotine was toxic ingredients contained in cigarettes which was known to have a harmful effect on periodontal tissue, it may alter host's tissue response or directly damage the cells of the normal periodontium. It could be proven that small amount of nicotine was stored-in and released from fibroblasts in the periodontal tissues.[20] Nicotine was known to be able to inhibit cells splitting processes in osteoblast cultures by stimulating osteoblast alkaline phosphate activity in vitro. Nicotine might also alter the periodontal cells in vivo. This meaned that nicotine had a tendency to disturb reparative and generative potential of the periodontium.[21]

Nicotine in cigarettes stimulates the sympathetic ganglia to produce neurotransmitters including katekolamin.[22] It stimulates alpha (α) receptor in blood vessels which then may cause vasoconstriction. Vasoconstriction of peripheral blood vessels caused by smoking may also affect the periodontal tissues.[23]

Nicotine metabolism might cause vasoconstriction and suppress functional activity of polimorphonuclear (PMN) cells and macrophages. The quantity of neutrophils in the peripheral blood increased and penetrated cappilary wall.[24] Polimorphonuclear (PMN) are phagocytes which are present in most common location of acute inflammation and have an important role in the defense of the periodontal tissue from bacterial invasion on gingival margin.[15]

Several studies showed that the effects of nicotine in cigarettes can reduce levels of salivary antibodies (IgA) and serum IgG antibody, which serve as a defence against Porphyromonas intermedia and Fusobacterium nucleatum.[25]

It becomes clear that periodontium in smokers are prone to be infected with pathogens compared to non-smokers. Nicotine in cigarettes could reduce levels of serum IgG2. Besides, it could also reduce the local oxygen tension and this can cause the growth of anaerobic bacteria. Nicotine can also facilitate pathogen adhesion to epithelial cells. Deep periodontal pockets may facilitate the growth of pathogenic anaerobic bacteria by providing a low-oxgygen environment.[15]

In microbiology, it was hypothesized that the smokers had more plaque bacteria in periodontal disease compared to the non-smokers,. The smokers have more calculus accumulation. Calculus may irritate the local tissue and provide a local environment filled with bacteria phatogens.[26]

Severity of periodontal disease was determined by the amount of cigarettes consumption and the duration of smoking. Until now the data measuring the effectiveness of smoking cessation programs in reducing or inhibiting diseases periodontal has not been available yet.[4]

In addition, nicotine may suppress the production of pro-inflammatory cytokines interleukin 1 (IL-1) and tumor necrosis factor-alpha (TNF-α) which was considered as a key in regulating the host response against microbial infection.[15,27]

Periodontitis is destructive inflammation which causes loss of periodontal attachment and the supporting alveolar bone. However, the use of nicotine in tobacco cause damage to the collagen tissues, by increasing the production of colagenase.[4]

Nicotine suppress the growth of gingival fibroblast, and the production of collagen and fibronectin. In addition it also had an impact to leukocytes, by decreasing the migration ability of neutrophils and phagositosis.[20]

As a conclusion from this literature that nicotine may be a cause of periodontal disease. Smoking may suppress the immune response and destroy vascular endothelial glands. From this literature study was also showed that smokers had less response to periodontal therapy.

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