

SMOKING RELATED SYSTEMIC AND ORAL DISEASES

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Summary: This article reviewed smoking related systemic diseases and oral diseases. Smoking is related to lung cancer, cardiovascular diseases and many other systemic diseases. Cigarette smoke affects the oral cavity first, so it is evident that smoking has many negative influences on oral cavity, for example, staining of teeth and dental restorations, wound healing, reduction of the ability to smell and taste, and development of oral diseases such as oral cancer, periodontitis, smoker's palate, smoker's melanosis, hairy tongue, leukoplakia, oral candidiasis and implant survival rate. The article also discusses the relationship between smoking and dental caries in detail.

Key words: *Smoking; Systemic diseases; Oral diseases; Dental caries*

Introduction

Tobacco is one of the major toxic agents in our civilization. Tobacco use and smoking is one of the most common causes of mortality and morbidity in developed and developing countries in present times. Tobacco comes from a plant that is native to America, around Peru and Ecuador (51). Tobacco was introduced to Europe from America in the fifteenth century, first being used in medicinal purposes. Later, it came to be burnt in pipes for pleasure on a large scale for nearly 100 years in England and subsequently in Europe as well as the rest of the world. Pipe smoking gave way to the use of tobacco as snuff and in time to cigars and cigarettes varying from country to country, until cigarette smoking became the dominant form in most of the developed countries between the two world wars (20).

It has been reported that tobacco kills 5 million people annually, and has been estimated that by mid 2020, the figure will increase to about 10 million a year, which means it would be the cause of most death occurring in developing countries (75). Over 1.1 billion adults (29 % of the adult population) are current smokers of cigarettes worldwide (5). Cigarette smoke is highly dynamic and has a complex matrix consisting of a gas phase and a particulate phase with more than 3800 compounds. Among these compounds, 60 of them are well-established carcinogens in animals and 15 of them are carcinogens in humans. Some of the carcinogens found in cigarette smoke include polycyclic aromatic hydrocarbons, aldehydes, arsenic, nickel and cadmium (1, 26). Smoking not only harms smokers but also harms the people around them.

This review mainly discusses the systemic effect and oral effect of smoking and specifically analyzes the effect of smoking on dental caries.

Smoking and systemic diseases

Presently, cigarette smoking has been found to be positively associated with nearly 40 diseases and causes of death and to be negatively associated with eight or nine. In some instances the positive associations are largely or wholly due to confounding, but the great majority has been shown to be causal in character (20). Cigarette smoking reduces life span by an average of 7 years, and tobacco consumption accounts for a shortening of disease free life by 14 years (11).

The medical evidence of harm caused by smoking has been accumulating for 200 years, at first in relation to cancers of the lip and mouth, and then in relation to vascular diseases and cancer of the lung. In the beginning, the evidence was ignored until five case-control studies relating smoking to the development of lung cancer were published in 1950 (20, 51).

Lung cancer is the most common tobacco-related cause of cancer. Statistically, one case of pulmonary carcinoma has been reported for every 3 million cigarettes smoked (54). Cigarette smoke contains several carcinogens that alter biochemical defense systems in the body leading to lung cancer (17), the strongest determinant being the duration of smoking and risk increasing with the number of cigarettes smoked (53).

Smoking causes a wide range of diseases, including many types of cancers, coronary heart disease, stroke and

peripheral vascular diseases (22). Smoking also increases the chance of developing cancer of the pancreas, renal pelvis, and urinary bladder. It is also associated with the cancer of the liver, uterine cervix, nasal cavity, pharynx, larynx, esophagus, stomach and kidney (18, 29, 71).

Smoking is a known cause for chronic obstructive pulmonary disease (COPD) (8). Tars produced within tobacco combustion contain irritant agents (acrolein, formaldehyde, etc.) and oxidative substances that are responsible for chronic bronchitis and emphysema (40). It has been observed that 80 % of deaths from heart attacks in individuals under 45 are directly related to smoking. The chances of heart attack are twice as likely among smokers compared to non-smokers. When a smoker quits smoking, his/her chances of heart attack decline to those of a non-smoker in about 6 months (15).

The most harmful compounds in tobacco smoke include carbon monoxide, arsenic, cadmium, cobalt and polycyclic aromatic hydrocarbons. Carbon monoxide is bound with haemoglobin to form carboxyhaemoglobin, which is a major cause of hypoxia and vascular accidents (40). Peptic ulcer is also more common in smokers than in non-smokers (22). Other potentially fatal diseases caused by smoking include respiratory heart disease, pneumonia, aortic aneurysm and ischaemic heart disease, which are the most common causes of death in developed countries (71). There is also a strong association between smoking and a number of common eye diseases, which include Graves' ophthalmopathy, age-related macular degeneration, glaucoma, and cataract (14).

The health effects of smoking are more serious for women than for men. In addition to the general health problems common to both genders, women face additional hazards (47). An example of this situation is the exposure of the fetus to maternal smoking. The exposure of mother to environmental tobacco smoke (ETS) can have a serious, sometimes life-threatening impact on the fetus. Such exposure increases the risk of spontaneous abortion, ectopic pregnancy, intrauterine growth retardation, premature membrane rupture, pre-term birth, retroplacental haematoma, placenta praevia, and sudden infant death (74).

Besides women, children are a very vulnerable population, particularly during the first year of their life. Their exposure to environmental tobacco smoke, or passive smoking, can induce serious short-term as well as long-term health effects (8). Children exposed to passive smoking have increased chances of chronic middle ear effusion, asthma attack, and changes in pulmonary function (21). Children of parents who smoke are more likely to encounter respiratory infections, pneumonia and bronchiolitis (48). Children who are exposed to passive smoking are potentially in danger of chronic cough, wheezing, running nose and excessive sneezing (48). Passive exposure of smoking during childhood increases a child's risk of leukemia and lymphoma during adulthood (21). It has been reported that maternal smoking during childhood increased a child's risk to become a young adult smoker (53).

Smoking and oral diseases

Oral cancer

Cigarette smoking and use of other types of tobacco products cause oral cancer (72). Oral cancer affects mostly middle aged or elderly people and is more common in men than in women. It constitutes 2-3 % of all cancers worldwide (56). Cigarette smokers have two to five times higher risk of oral cancer than that of non-smokers (73), the risk increasing with the number of cigarettes and years smoked. On the other hand, cessation decreases the risk (26). Tobacco-specific N-nitrosamines, aromatic amines, and polycyclic aromatic hydrocarbons presented in mainstream tobacco smoke are considered the major carcinogens contributing to the risk of oral cancer from smoked tobacco products (73).

Although the underlying mechanisms are not known in detail, it is said that smoking could lead to cancer because carcinogens in tobacco smoke can induce changes in DNA. In recent years much attention has been given to smoking-related mutations in a tumor suppressor gene coding for p53 protein. This protein is important in regulating cell proliferation and has a role in the repair of DNA damage (52). Smoking-related mutations in the gene may lead to an accumulation of DNA damage in the cells, which may play an important role in the development of cancer. It has been estimated that between 75 % and 90 % of all cases of oral cancer can be explained by the combined effect of smoking and alcohol use. This could be because alcohol dissolves certain carcinogenic compounds in tobacco smoke and/or because alcohol increases the permeability of the oral epithelium (56). Smoking and excessive alcohol intake synergistically increases the risk of the development of oral cancer (26).

Oral leukoplakia

Leukoplakia is believed to be a premalignant lesion associated with development of oral cancer (44). Tobacco smoking is the most important known etiological factor in development of oral leukoplakia (7). Cross-sectional studies show a higher prevalence rate of leukoplakia among smokers, with a dose-response relationship between tobacco use and oral leukoplakia, and intervention studies show a regression of the lesion after the cessation of smoking (7). Leukoplakias of the floor of mouth appeared to be significantly more often present in smokers than in non-smokers (60). Smokers have a six-fold increase in the risk of developing leukoplakia of the oral mucosa regard to non-smokers (12). Six European studies found smoking to be a cause in 56-97 % of leukoplakia patients. One of these studies also showed that the majority of smokers with leukoplakia (74 %) smoked more than 20 cigarettes per day compared to 34.5 % of those without leukoplakia (12).

Palatal leukokeratosis and smoker's melanosis

Palatal leukokeratosis (smoker's palate) is an asymptomatic lesion associated with heavy pipe and cigar smoking

usually appearing as white changes in hard palate, often combined with multiple red dots located centrally in small elevated nodule. It may disappear after smoking cessation (34, 56). It does reveal premalignant potential. Premalignant lesion like palatal keratosis is primarily associated with reverse smoking found mostly in South Asia (49).

Melanin pigmentation of the oral mucosa is normally seen in coloured races. Among Caucasian heavy smokers, 30 % prevalence in pigmentation is seen, mostly on the attached gingiva (56). There are no symptoms, the change is not premalignant, and the pigmentation may be reversible upon cessation of smoking habit (34). A recent study in the Indian population showed that smokers were more likely to develop smoker's melanosis compared to other lesions (58).

Oral candidiasis and hairy tongue

Cigarette smoke is associated with a variety of changes in the oral cavity and it has an effect on oral commensal bacteria and fungi, mainly *Candida* species, which causes oral candidiasis. How cigarette smoke affects oral *Candida* is still controversial (65). Further studies and research need to find the exact etiology of smoking and oral candidiasis. It has been seen in the clinical experience that some *Candida* infections disappear following smoking cessation alone (34). Another oral lesion, "hairy tongue" or "black hairy tongue" is a benign condition characterized by hypertrophy of the filiform papillae that give the dorsum of the tongue a furry appearance associated sometimes with heavy smoking (76), but its etiology remains unclear.

Aesthetics, smell and taste

Discoloration of teeth, dental restorations and dentures are very frequent in smokers (34, 56). A recent cross sectional study conducted in British adults showed that 20 % of smokers reported to have moderate and severe levels of tooth discoloration compared to 15 % in non-smokers (4). Discoloration caused by smoking is more severe than that caused by tea and coffee consumption (56). Smoking is found to be associated with halitosis (30, 34). It has been seen that smoking influences the decreased function of smell (34, 56), and it is also associated with worsening of taste perception (59).

Periodontitis

Cigarette smoking is a significant risk factor for periodontal diseases (68), for example, increased loss of attachment (55), development and progression of periodontal inflammation (23, 32) and increased gingival recession (50). It has been found that smoking has a direct influence on the periodontal health status irrespective of the oral hygiene practice, age, race, gender, socioeconomic status or frequency of dental visits (35). Smoking affects various aspects of the host immune response. The mechanisms by which smoking enhances periodontal degradation are said to be the cumulative effect of elevation in levels of perio-

dontal pathogens and modulation of the host immune response (16, 70). There is sufficient evidence that shows that smoking affects the innate and specific immune host responses (33, 39).

Antibody production is another protective host mechanism that is altered by smoking. Generally, smoking decreases serum IgG concentrations and decreases IgG2 antibody production in patients with early onset periodontitis (25). Smoking does not alter the bacterial plaque composition but on the other hand, it has been observed that the host's response to bacterial plaque is disturbed (46). The severity of alveolar bone destruction was found to be more expressed in smokers than in non-smokers (10, 38).

Chemical products and toxins in tobacco smoke may delay wound healing by impairing the biologic progression of healing and by inhibiting the basic cellular functions responsible for its initiation (35). Smokers have a decreased response to periodontal therapy compared to non-smokers (32, 46). Smoking has a strong negative influence on regenerative therapy, which includes osseous grafting, guided tissue regeneration or a combination of these treatments (34). It has been seen that the hemorrhagic response of periodontium is decreased in smokers compared to non-smokers (9). On the whole, smoking related periodontal diseases consist of an increased and accelerated destruction of the supporting tissues of the teeth, with clinical symptoms of bone loss, pocket formation, and finally tooth loss.

Wound healing

Smoking appeared to have an adverse effect on the wound healing in the mouth after periodontal scaling, periodontal surgery or extraction wounds (34). It has been reported that increased frequency of smoking and smoking on the day of surgery significantly increased the incidence of alveolar osteitis known also as dry socket (3). The mechanism of impaired healing is likely associated with increased plasma levels of adrenaline and noradrenaline after smoking, leading to peripheral vasoconstriction and also impaired polymorphonuclear neutrophils function (34).

Dental implants

Smoking was found to be one of significant factors predisposing to implant failure. The use of tobacco involves a 15.8 % risk of implant failure and the consumption of more than 20 cigarettes per day increased this risk up to 30.8 % (57). Study has also suggested as an ongoing detrimental effect around the successfully integrated maxillary implants of smokers, with significantly greater bleeding index, mean peri-implant pocket depth, peri-implant inflammation and radiographically descendent mesial and distal bone loss (24). Studies on fixed implant prostheses proved that smoking correlated more strongly with marginal bone loss around implants (19). Because of all these proved effect of smoking on implant success, latest studies pertaining implants success exclude heavy smokers from their potential participants (64, 63).

Smoking and dental caries

Smoking and its relation to dental caries is a subject of controversy, however several studies indicate an association with smokeless tobacco and caries, particularly in terms of root surface caries. This may be due to high proportion of sugar in some type of smokeless tobacco (69). It is interesting to note that maternal smoking and environmental tobacco smoke is also considered as a risk factor for dental caries in children (62, 72).

Schmidt, in 1951, reported that the increase in tobacco smoking was followed by a decrease in caries rate (61). Smoking increases thiocyanate level in saliva. Thiocyanate, a normal constituent of saliva, was found to have a possible caries inhibiting effect (34, 56). On the other hand, studies showed that smoking is associated with lower salivary cystatin activity and output of cystatin C during gingival inflammation. Cystatins are thought to contribute to maintaining oral health by inhibiting certain proteolytic enzymes. In addition, studies have confirmed from earlier results that there were no significant differences in salivary flow rates between smokers and non-smokers (34, 41). The decreased buffering effect of smoker's saliva and the higher number of lactobacilli and *S. mutans* group may indicate an increased susceptibility to caries (34, 56). To date, several investigators have discovered a correlation between increased smoking level and occurrence of dental caries (6, 13, 31, 36). For example, in 1952, Ludwick and Massler reported that individuals who smoked more than 15 cigarettes daily had significantly higher number of decayed, missing, and filled teeth (43). Ainamo found that increased smoking results in significantly high number of decayed surfaces per dentition (2). In 1990, Zitterbart confirmed an association between smoking and the prevalence of dental caries in adult males. Smokers had a significantly higher DMFT (Decayed, Missing, and Filled Teeth) score, untreated decayed surfaces, and missing surfaces. More cigarettes consumed per day resulted in more missing tooth surfaces in a smoker's mouth (77). Statistical analyses from a study in Sweden in 1991 showed that smoking, as a habit and an increased number of cigarettes smoked per day, are positively correlated with the increased number of decayed, missing and filled teeth and number of initially decayed proximal surfaces (28). Although studies did not establish a causative relationship, a recent study done on American female population in 2006, showed a correlation between cigarette smoking and the presence of dental caries (27).

Most of the studies mentioned above have taken into consideration other contributing factors to dental caries such as age, tobacco habits other than smoking, oral hygiene habits, eating habits, and preventive visits to dentist (dental recalls) and overall health standards. Therefore elucidating the exact strength of dental caries in relation to smoking is difficult to identify.

Studies indicate that smokers not only had bad oral hygiene and less sophisticated outlook on health, but also had

different eating habits, presumably consuming higher amount of sugar containing products like soft drinks and snacks (6, 28). It is also seen that smokers have poorer brushing habits than non-smokers (2, 37, 45). Also current smokers were less likely to report regular preventive visits to dentists and were reluctant to use accessory dental aids such as dental floss (42).

In natural tobacco, sugar can be present in a level up to 20 %_{w_t}. In addition, various caries promoting factors such as sugars (4 %_{w_t} or can be up to 13 %_{w_t}) and sweeteners are added intentionally during tobacco manufacturing process (67). Sugars used as cigarette additive include glucose, fructose, invert sugar (glucose/fructose mixture) and sucrose. In addition, many tobacco additives contain high amount of sugars, for example fruit juices, honey, molasses extracts, cones, maple syrup and caramel. The added sugars are usually reported to serve as flavor/casing and humectants. However, sugars also promote tobacco smoking, because they generate acids that neutralize the harsh taste and throat impact of tobacco smoke. Moreover, the sweet taste and the agreeable smell of caramelized sugar flavors are appreciated in particular by starting adolescent smokers (67).

All the findings above can be argued for increased dental caries in smokers. Though a direct etiological relation is still lacking between smoking and dental caries, the above-mentioned studies and findings point to the conjecture that smoking has some influence in high caries incidence. Further studies, clinical trials and experiments are needed to confirm the independent effect of smoking as one of the causes of dental caries.

Conclusion

Smoking is evidently associated with systemic diseases like cardiovascular diseases, various lung disorders and different types of cancer. It has been proved that smoking is hazardous especially to women and children. Cigarette smoking negatively influences oral cavity. It has been established that it also causes diseases such as oral cancer, periodontitis, leukoplakia and several other oral lesions, but the direct influence of smoking on dental caries is still not verified. It has been proved that smoking along with bad oral hygiene, food habits, preventive dental visits and over all health standards, is associated with high caries incidence.

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