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Short Communication

The Deleterious Trilogy: Tobacco, Periodontal Disease and Cardiovascular Disease

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Abstract

Tobacco used in both smokeless and smoking form is deleterious to various body systems. Oral cavity bears both the imprints of the habit and also the brunt. This article attempts to highlight the impact of tobacco on periodontal tissues and cardiovascular tissues. Consequently how the periodontal disease can in turn pose as a risk factor for cardiovascular disease is brought forth in this article. The importance of intra oral examination by a clinician irrespective of specialty cannot be overemphasized.

INTRODUCTION

Tobacco kills around 6 million people each year. More than 5 million of those deaths are the result of direct tobacco use while more than 600 000 are the result of non-smokers being exposed to second-hand smoke. Nearly 80% of the world's 1 billion smokers live in low- and middle-income countries [1]. Tobacco use has a widespread deleterious systemic effects including oral cavity. In fact oral cavity is the first to face the onslaught of smoking and /or oral tobacco.

In view of current epidemic of tobacco related non communicable diseases such as hypertension, coronary artery disease (CAD), diabetes and cancers particularly oral cancer it is essential to examine the oral cavity of every subject who visits the hospital for any health related ailment.

DISCUSSION

Nicotine the alkaloid present in the leaves of tobacco plants, in bidi or cigarette smoke stimulates catecholamine hormones facilitating the secretion of adrenocortical hormones, leading to vascular contraction [2]. It also increases the concentration of epinephrine and nor-epinephrine in blood by stimulating the sympathetic nervous system and adrenal medulla, with a resultant increase in heart rate, blood pressure, and cardiac output. Consequently, it acts as a risk factor for cardiovascular diseases [3].

A study of the acute effect of smoking on vascular status reported, Blood Vessel Output Power (BVOP), was significantly lower in the smoking group than the non-smoking group. Both Remaining Blood Volume (RBV), and Blood Vessel Aging Level (BVAL) for the smoking group were higher than for the non-smoking group. These findings indicate that smoking weakens blood vessel function [4].

Carotid intima-media thickness (C-IMT), a marker of subclinical atherosclerosis, has been used as a surrogate end point to investigate the effects of cigarette smoking [5-9]. Some studies have failed to report a difference in C-IMT between former and current smokers [8,9] which is suggestive that the effect of smoking on vascular walls is irreversible.

Gingival vasculature also is affected by tobacco. A positive association between cigarette smoking and acute necrotizing ulcerative gingivitis (ANUG) was first reported over 4 decades ago. Smokers with excellent oral hygiene show significantly less periodontal bone height and attachment level than non-smokers. Recent studies have confirmed a greater prevalence of attachment loss [10,11] recession, severe destructive periodontal disease [12] and less favorable response to nonsurgical [10,11] or surgical [10] periodontal treatment in smokers, as compared to non-smokers. Regarding periodontal blood supply, several studies have reported that plaque-induced gingivitis, showed a reduction of clinical signs [13,14] with a smaller propensity for gingival bleeding [13,15] owing to vascular changes caused by smoking.

The oral effects of smokeless tobacco are localized to the site of placement. Localized gingival recession is commonly found at the site of tobacco quid placement.

Literature reports gingival recession occurs in 25-30 percent of these users, and white mucosal lesions occur in 50-60 percent of users [16-18].

The oral mucosa also shows manifestations of the effect of tobacco. The mealnocytes present in the oral mucosa act as a diffusible sink barrier to absorb the various aromatic hydrocarbons released in the tobacco smoke [19], this explains the increased melanin mucosal pigmentation seen on the lips, cheeks and palate of smokers.

The common site of placement of tobacco quid is the buccal vestibule. This explains the higher prevalence of mucosal lesions as compared to gingival lesions is probably due to the closer proximity of the tobacco to the mucosal tissues [20]. The severity of the white keratotic mucosal lesions or the ill effects of any habit depends on the triad of the quantity, frequency and duration of usage. Inflammatory mediators, such as prostaglandin E2 (PGE2) and interleukin–1 (IL- 1) are elevated in these developing sites [21], and IL-1 remains elevated in the established lesion [22]. In the developing lesion, the pro-inflammatory effects of these mediators likely contribute to the observed erythema.

Smokeless tobacco users develop keratotic white patch as a result of the constant friction the quid of tobacco causes against the mucosal tissues. The majority of the white mucosal lesions regress when the smokeless tobacco habit is discontinued. Literature reports 97 percent of smokeless tobacco-induced lesions resolved within six weeks of tobacco cessation [23]. In another study, 22 percent of lesions resolved one week after tobacco exposure ceased [21].

This is useful information for the clinician to time the need of biopsies in such individuals.

Premalignant lesions such as leukoplakia are also often seen in tobacco users.

The negative effects of smoking on bone metabolism are attributed to the effect of nicotine and bacterial lipopolysaccharide (LPS) which increase PGE2 secretion by peripheral monocytes. Smokers exposed to LPS had significantly higher plasma levels of TNF alpha and IL-6 than nonsmokers [24].

Systematic review and meta-analysis evaluating the relationship between periodontal disease, including gingivitis, bone loss, and missing teeth, concluded periodontal disease to be an independent, though relatively weak, risk factor for coronary heart disease (CHD) and that periodontal disease confer approximately a 24–35% increase in risk of CHD [25].

Periodontal disease represents a chronic infection resulting in a chronic inflammatory state. Data from recent literature suggest an important role for chronic inflammation in the development of coronary artery disease (CAD). This hypothesis is supported by many studies showing fibrinogen, CRP, serum amyloid A and Von Willebrand factor elevations in association with periodontal disease [26-31]. Notably, periodontal treatment studies have shown improvements in measures of systemic inflammation such as CRP and serum IL-6 with treatment [32].

Another biological consideration is the intermittent bacteremia associated with periodontal disease and its possible role either in the chronic inflammatory state or more directly on endothelial tissue surfaces. In one study, 80% of carotid endarterectomy specimens were positive for one or more polymerase chain reaction (PCR) assays of various oral pathogens [33].

Some studies have shown increased platelet activation *in vivo* in association with periodontal disease, which could contribute to plaque instability and thrombosis [34]. Relative risks for stroke in association with periodontal disease in the range of 1.2 to 3.0 [35-37]. In addition, a relationship has been shown among individuals with peripheral vascular disease and periodontal disease, as well as tooth loss [38,39]. These data support the role of periodontal disease in generalized atherosclerosis.

Data suggests that periodontal disease may be a marker of risk. This hypothesis implies that unexplained confounding by a factor associated with both periodontal disease and CAD explains the relationship. The most likely known confounders include: smoking, diet, diabetes and socio-economic factors [40-42].

Some investigators have hypothesized that genetic susceptibility to a strong inflammatory response mediates both CAD and periodontal disease.

CONCLUSION

Intra oral examination will yield immense clinical benefits in detecting premalignant lesion or condition such as leukoplakia, erythroplakia or oral submucous fibrosis due to smokeless tobacco use or smoking. Besides above lesions tobacco deposits in smokeless tobacco chewers and signs of periodontitis may also be seen which is now considered as risk factor for CAD.

Not uncommonly smoker or tobacco chewer may sometimes deny tobacco habit, a doubly alert attending physician may examine the mouth cavity and find evidence of smoking and / or tobacco particles in mouth providing a definite proof for tobacco usage. Tobacco stains on the teeth are a definite evidence for tobacco habit.

In all such circumstances patient needs to be told in no uncertain term that continued tobacco habit may compromise his prognosis after procedures like PTCA or CABG. It is therefore strongly advocated that oral cavity of every patients who visits any clinic whether medicine, chest, surgery, OBG-Gynae, emergency, ENT, general check up clinic etc. should be examined for evidence of smoking and /or oral tobacco .Such an examination pays rich dividends most time by providing clues about tobacco habits - a major risk factor for cardiovascular diseases and /or cancer.

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