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Tobacco – A Public Health Problem

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Abstract

The purpose of this paper is to review the epidemiologic evidence for the effects of tobacco use and tobacco use cessation on a variety of oral diseases and conditions. Exposures considered include cigarette and bidi smoking, pipe and cigar smoking and smokeless tobacco use. Oral diseases and disorders considered include oral cancer and precancer, periodontal disease, caries and tooth loss, gingival recession and other benign mucosal disorders as well as implant failure. Particular attention is given to the impact of tobacco use cessation on oral health outcomes. We conclude that robust epidemiologic evidence exists for adverse oral health effects of tobacco smoking and other types of tobacco use. In addition, there is compelling evidence to support significant benefits of tobacco use cessation with regard to various oral health outcomes. Substantial oral health benefits can be expected from abstention and successful smoking cessation in a variety of populations across all ages.

Keywords: Smoking, Smokeless Tobacco, Oral Cancer, Pre Cancer, Periodontal Disease, Tooth Loss, Implants, Dental Caries, Smoking Cessation

Introduction

Oral cancer is the sixth most common cancer globally; however, the incidence is much higher in the developing countries, including Pakistan, India, Sri Lanka.[1,2] In Central and Southeast Asia, oral cancer accounts for up to 40% of all cancers compared to less than 4% reported in most developed countries.[3,4] The aetiology of oral cancer is multifactorial with majority of the cases attributable to separate and combined use of tobacco (smoked and smokeless), excessive alcohol consumption, betel quid, and betel quid substitutes.[5-9]

Smokeless tobacco (ST) is referred to as tobacco products that are consumed by means other than smoking and include chewing, sniffing, placing the tobacco between the gums and teeth and application to the skin. Smokeless tobacco are broadly categorized into two main types: Chewing tobacco and snuff.[10] Chewing

tobacco is universally available in the form of loose, cut, and shredded leaf whereas snuff is available as fine ground tobacco that can be dry, moist, or in sachets. Different names are given to various ST products depending on where they are used such As Gutka, Betel Quid, Supari, Khaini, Mawa, Qiwam, Mainpuri, Zarda, Naswar, Nass, Gul, Mishri, Gudakhu, Shammah, Toombak, Plug and Snus.

Tobacco is used in a variety of ways, mostly as smoked, but many populations use smokeless tobacco, which comes in two main forms; snuff (finely ground or cut tobacco leaves that can be dry or moist, loose or portion packed in sachets) and chewing tobacco (loose leaf, in pouches of tobacco leaves, plug or twist form). This review examines the oral health risks of both smoked and smokeless tobacco.

According to the National Family Health Survey (NFHS)- 3 survey, conducted in 2005–06, tobacco use is more prevalent among men, rural population, illiterates, poor and vulnerable section of the society.[5] The estimates of the Global Adult Tobacco Survey (GATS) conducted among persons 15 years of age or older during 2009–10 indicate that 34.6% of the adults (47.9% males and 20.3% females) are current tobacco users. Fourteen percent of the adults smoke (24.3% males and 2.9% females) and 25.9% use smokeless tobacco (32.9% males and 18.4% females).[6] According to the Global Youth Tobacco Survey (GYTS) conducted among 24,000 students aged 13–15 years in 2009, 14.6% students were tobacco users.

India's tobacco problem is very complex, with a large use of a variety of smoking forms and an array of smokeless tobacco products. Many of these products are manufactured as cottage and small-scale industries using varying mixtures and widely differing processes of manufacturing.[8] Bidis are mostly manufactured in the unorganized sector while cigarettes are mainly manufactured in large-scale industries.

This literature review aims to present published evidence regarding our current understanding of the epidemiology, aetiology and pathogenesis of tobacco use-related disorders. In addition, we also review significant improvements in oral health following cessation. The focus of the review is on the adverse effects of tobacco on several oral disorders including oral cancer, other oral mucosal disorders, periodontal disease and tooth loss, and how tobacco affects clinical management such as implantology, and to discuss the oral health benefits of tobacco cessation.

Smoking

Oral cancer

Among sites that have been considered to be at highest relative risk for cancer due to smoking is the lung. Following lung cancer, the highest relative risks are observed for the larynx and oral cavity.[1] The risk of oral cancer has increased in recent decades in many countries in the world.[2]

In those countries in which epidemiological studies have been conducted, it is clear that oral cancer risk is high among smokers. A recent meta-analysis reported 12 studies that estimated oral cancer risk in the USA, Uruguay, Italy, Sweden, India, China, Taiwan and Korea.[3] The reported pooled cancer risk estimate was 3.43 times higher in smokers compared with non-smokers (95% CI 2.37, 4.94). The results for risks associated with tobacco smoking were generally consistent across countries entered into the meta-analysis except in the study conducted among females in Sweden.[4] In a study reported from Northern Italy, the single factor with the highest attributable risk was smoking, which accounted for 81-87% of oral cancers in males and for 42-47% in females.[5] It is evident that oral

cancer risk is related to both intensity and duration of tobacco smoking. The differential risk between non-smokers and heavy smokers, and the steady progression of risk with increasing amount smoked both provide sufficient evidence for tobacco as a major risk factor for oral cancer. Furthermore, most studies show an inverse relation with age when starting to smoke. Among young people in southern England, a significant risk among males (alcohol adjusted OR: 19.5) was associated with starting to smoke under the age of 16 years.[6] These risks are also increased synergistically with alcohol consumption. However, among never drinkers, cigarette smoking was associated with an increased risk of 2.13 confirming an independent association with tobacco use.[7] This had also been demonstrated in an earlier study among 19 cases and 213 controls who described themselves as non-drinkers; the ORs were 3.8 and 12.9 for smokers of < 15 or > 15 cigarettes per day, with a strong trend.[8]

In many European and US studies the risks for oral and oropharyngeal cancers are similar for cigarette and cigar smokers.[9] Smoking bidi (hand-rolled Indian cigarette consisting of flaked tobacco rolled in temburni leaf) is a common practice in India and this may be relevant for Indian ethnic migrants to Europe. A meta-analysis has shown that the risk of oral cancer associated with bidi smoking is about three times higher compared with cigarettes.[10]

Pathogenesis

Several lines of evidence indicate that oral cancers arise as a result of mutagenic events (arising mainly from tobacco and alcohol) causing multiple molecular genetic events in many chromosomes and genes. The consequence of this chromosomal (genetic) damage is the impairment of cell regulatory processes leading to acquired capabilities within cells such as self-sufficiency in growth signals, insensitivity to anti-growth signals, evading apoptosis, limitless replicative potential, sustained angiogenesis and tissue invasion and metastasis.[11] As shown by epidemiological data, exposure to tobacco is unquestionably the major risk factor for oral cancers, however, only a minority of those exposed develops a malignancy. The balance between how enzyme systems metabolise and deactivate tobacco carcinogens varies among individuals and is likely to contribute to cancer risk. An individual's susceptibility to cancer may therefore be explained by genetic polymorphisms in a number of enzymes involved in the metabolism of tobacco carcinogens.[12]

Two main carcinogens present in tobacco smoke are benzo(a) pyrine and tobacco smoke derived nitrosamines (TSNA). These are primarily metabolised to their activated molecules by cytochrome P450 and these intermediates are detoxified by glutathione Stransferase (GST) to hydrophilic and non-toxic GST conjugated substances.[12] Genetic polymorphisms in these metabolising enzyme systems (Cytochrome P450 and GST) and resulting variants are relatively common in populations and may partly explain susceptibility to cancer in various organs.[13-15] If detoxification does not take place, then the metabolically activated tobacco products would adduct to DNA. In studies of DNA obtained from clinically normal oral mucosa in patients with oral cancer, a significantly higher level of a variety of aromatic adducts was noted in smokers than from non or former smokers.[16,17] DNA adducts associated with tobacco smoking could provide a marker of the biologically effective dose of tobacco carcinogens and improve individual cancer risk prediction.[18]

Precancer

Several potentially malignant disorders (particularly oral leukoplakia and erythroplakia) are known[19] and a

proportion of these transforms to cancer over a period of time.[20] Presence of epithelial dysplasia in precancers is a hallmark for cancer development[21] and several studies from the US and the UK have demonstrated significant associations with smoking in relation to oral epithelial dysplasia.[22,23] Exclusive tobacco consumption appears to be more likely to contribute to epithelial dysplasia than exclusive alcohol use suggesting that tobacco has an independent role in the aetiology of oral epithelial dysplasia.[24]

Oral leukoplakia is the most common precancer associated with tobacco use. The clinical appearance of leukoplakias varies considerably. The lesion may appear smooth, fissured, nodular or corrugated and the colour is predominantly white. Leukoplakias also vary with regard to size and distribution in the oral cavity. They may be barely discernible clinically, or may cover entire mucosal surfaces.[25] Two different clinical types of oral leukoplakia exist: homogeneous and non-homogeneous. The distinction between these two entities is primarily based on their clinical appearance (surface colour and morphological characteristics), and has some bearing on the prognosis and risk for malignant transformation of the lesion.[20] Homogeneous lesions are uniformly flat, thin, and exhibit shallow cracks on their surface. Non-homogeneous lesions include speckled leukoplakias (white and red lesions with a pre-dominantly white appearance, also termed 'erythro-leukoplakia', nodular and verrucous lesions, as well as the widespread and multifocal rarer entity known as proliferative verrucous leukoplakia.[25]

Hazards of tobacco use

Tobacco is deadly in any form or disguise. Scientific evidence has unequivocally established that exposure to tobacco smoke causes death, disease and disability.[9] According to the International Agency for Research on Cancer (IARC) monograph, there is sufficient evidence in humans that tobacco smoking causes cancer of the lung, oral cavity and hypo-pharynx, nasal cavity and paranasal sinuses, larynx, oesophagus, stomach, pancreas, liver, kidney (body and pelvis), ureter, urinary bladder, uterine cervix and bone marrow (myeloid leukemia). Colorectal cancer is seen to be associated with cigarette smoking, although there is insufficient evidence for it to be causal.[10] Ninety percent of all lung cancer deaths in men and 80% in women are caused by smoking.[9] Causal associations have been clearly established between active smoking and adverse reproductive outcomes, chronic obstructive pulmonary disease and cardiovascular diseases.[10] Studies on bidi smoking, the most common form of tobacco smoking in India, provide evidence toward causality of it as carcinogenic substance. Case–control studies demonstrate a strong association of bidi smoking with cancers at various sites, such as oral cavity (including subsites), pharynx, larynx, oesophagus, lung and stomach. Almost all studies show significant trends with duration of bidi smoking and number of bidis smoked.[10] Forty percent of the tuberculosis burden in India may be attributed to smoking. Significant association is seen between passive or active exposure to tobacco smoke and tuberculous infection, disease and tuberculosis mortality.[11] Smoking was associated with excess deaths among smokers between 30 and 69 years, mainly from tuberculosis and also from respiratory, vascular or neoplastic disease.[2] The risk of tuberculosis deaths among bidi smokers was 2.60-times higher than never-smokers in Mumbai.[12] Workers engaged in tobacco cultivation suffer from an occupational illness known as green tobacco sickness (GTS), an acute form of nicotine toxicity resulting from absorption of nicotine through the skin.

Environment

Tobacco leads to clearing of forests for cultivation, stripping fuel wood for curing and forest resources for packaging thus damaging the environment. Tobacco depletes the soil nutrients at a very rapid rate and displaces the indigenous flora and fauna thus becoming a source of pests for other crops.[8]

Passive smoking

Second-hand tobacco smoke (SHS) kills 600,000 people each year. Globally, about one-third adults are regularly exposed to SHS. The GATS-India shows that 52% of the adults (rural-58%, urban-39%) were exposed to SHS at home.[6] SHS is three- to four-times more toxic per gram of particulate matter than mainstream tobacco smoke. More than 4000 chemicals have been identified in tobacco smoke, at least 250 of which are known to be harmful. Toxic chemicals from SHS cling to rugs, curtains, clothes, food, furniture and other materials. These toxins remain even in the presence of windows, fans or air filters, and can recycle back into the air through the filters. They coat the surfaces of rooms, materials and smoker's belongings, and are sometimes referred to as "third-hand smoke."[17] There is conclusive evidence linking passive smoking to an increased risk of cardiovascular diseases, lung cancer and other cancers, asthma and other respiratory diseases in adults and asthma and other respiratory diseases, ear infection and sudden infant death syndrome in children, to name but a few of passive smoking's harmful effects.

Conclusion

Measures that proved very effective in the developed world, like tax increases on all tobacco products, need to be enforced immediately and the taxes collected should be used to support health promotion and tobacco control programmes. Sustained efforts are needed from the Government to strengthen efforts on alternate cropping and alternate livelihoods to replace employment losses that may come up gradually.

Public health awareness, raising a mass movement against tobacco, sensitizing and educating all health care professionals for tobacco control and cessation by incorporating the topic in medical undergraduate curriculum, nursing curriculum, various CMEs, conferences, scientific meetings, workshops, etc. is vital. Eventually, if all healthcare professionals participate in tobacco control and cessation, it will have a huge impact. Expansion of TCCs to the periphery to reach the community, making them more accessible and widely acceptable, will facilitate millions of current tobacco users to quit the habit.

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