IMPACT OF SMOKING AND SMOKELESS TOBACCO ON ORAL CAVITY

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Abstract

Aim: To investigate the effect of Smokeless Tobacco and Smoking on hard and soft tissues of oral cavity.

Material and method: We conducted a cross-sectional Study on 150 subjects in regular OPD of Karnavati School of dentistry. A visual oral soft tissue examination and a questionnaire-based Performa were part of the study methodology. The study included individuals who had a history of tobacco use and smoking. Patients were told verbally about the study, and those who agreed to share their personal habits and agreed for an oral examination were chosen as study subjects. A complete history of tobacco consumption and smoking was documented from the patient, including the kind, manner of usage, time span, frequency, and location of the tobacco (if chewable), as well as other harmful habits.

Patients with tobacco habits were categorized into three groups based on the inclusion and exclusion criteria, namely as Group A: only Smoking, Group B: only tobacco, Group C: only areca nut Group D: mixed tobacco and areca nut.

Result: Dental caries was found in 97(60.2%) and was found more in Group D patients. Gingival recession was found in 86(53.4%) and was found more in group d patients. Smokers' palate and Smoker's melanosis was found be associated with group A patients which is 19(61.3%) and 9 (29.9%) respectively. Leukoplakia was found to be more in group A patients which is 99(29.0%). Erythroplakia was found to be more prevalent in group B patients which is 4 (7.4%).Tobacco quid lesion was found to be more prevalent in group D patients which is 20(21%).OSMF was found to be more prevalent in group B patients which is 12 (52.2%). Lichenoid reaction was found to be more prevalent in Group B patients which is 3 (13%).Carcinoma of oral cavity was to be more prevalent in Group D patients which is 10 (10.9%).

Conclusion: Smoking and using smokeless tobacco both raise a person's risk of developing oral premalignant lesions and oral cancer. It also has a lot of negative consequences on the teeth and other structures. All medical professionals should evaluate their patients' tobacco use patterns and actively implement programs for tobacco prevention, cessation, and treatment.

Keywords: Nitrosamines, Nicotinic stomatitis, Leukoplakia, Hyperkeratosis, Human papilloma virus, Carcinoma.

INTRODUCTION

Oral mucosa is made up of stratified squamous epithelium. The two types of epithelial mucosa are masticatory (keratinized) and lining epithelial mucosa (nonkeratinized). The masticatory mucosa is thick, with a denser and less vascular connective tissue component. Keratin functions as a barrier towards stimuli such as unpleasant stimuli from ill-fitting dentures or cigarette smoking, as well as traumatic pressures from daily activities such as drinking, eating, swallowing and speaking, Tobacco and smoking are linked to a variety of carcinogenic, precancerous, and noncancerous changes in the oral and perioral environment.

The changes might range from enhanced pigmentation to significant thickness of the skin. Hyperkeratosis, results in a noticeable white lesion. Tobacco smoking can also irritate the small salivary glands on the hard palate, enhancing the risk of periodontal disease. (1) Keratinized and non-keratinized mucosa both border the oral cavity, operating as a physical barrier and serving a range of functions including protective, sensory, and secretory.(2) People who chew tobacco or smoke will have an effect on their oral cavity, and when it is affected it produces thicker epithelial layers and more keratinization.(3) A variety of functions, including defensive, sensory, and secretory activities.(2) People with habit of tobacco or smoke will have an influence on their oral cavity, which will result in thicker epithelial layers and increased keratinization.(3) Oral cancers account for 4% of all cancers in men and 2% in women. (4)

Lifestyle changes, poverty, a lack of awareness, and dangerous habits, as well as restricted access to health care, all contribute to an increased risk of cancer development and morbidity. (5) Chewing tobacco is the most popular smokeless tobacco in India, and it can cause potentially malignant disorders such as oral submucous fibrosis (OSMF), leukoplakia, erythroplakia tobacco pouch keratosis, and malignancies such as oropharyngeal tumors.

Smokeless tobacco products include pan masala, paan (betel quid), gutkha, khaini, mawa, snuff, and Swedish snuff.(6)(7) Tobacco smoke and unburned tobacco used in chewing or snuff dipping contain a number of potent nitrosamines. These substances may have a role in the onset and development of malignancies such as oral squamous cell carcinoma, verrucous carcinoma, and others.(8) The chance of developing oral squamous cell carcinoma increases with age, cigarette usage, and alcohol use.

HPV is also linked to cancer development. (2) The poor prognosis of this ailment is mostly due to the disease's advanced stage at the time of diagnosis. This is due to either the patient's failure to seek medical advice from a doctor for rare, usually asymptomatic oral lesions or the health-care personnel's failure to appropriately analyze possibly malignant mucosal lesions.(2)(3)(9) According to a study performed by the Indian government's National Sample Survey Organization, about 20 million children aged 10 to 14 are tobacco addicts.

Tobacco smoking's harmful effects on oral health are well known. Common and rare conditions ranging from benign to life-threatening diseases include tooth restoration discoloration, 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. (10)(7) (11)

Taking consideration into adverse impact of tobacco, this study was conducted to evaluate the oral mucosal changes and hard tissue changes such as stains, calculus, dental caries, and wasting diseases that may occur in patients due to the use of tobacco and smoking in various forms, as well as to collect a detailed history from the patients about the type of tobacco used, duration, frequency, and site of placement. Tobacco use, both smoked and smokeless, has an effect on the soft and hard tissues in and around the mouth. Because many of these changes are clinically visible, dentists, dental hygienists, and other health professionals are in a unique position to provide patients with detailed information on the harmful effects of tobacco use on the mouth.

Material and methods:

One hundred and fifty patients were examined over a period of 9 months (November 2020–August 2021) who came to regular OPD of Karnavati School of Dentistry. A visual oral soft tissue examination and a questionnaire-based Performa were part of the study methodology. The study included individuals who had a history of tobacco use and smoking. Patients were told verbally about the study, and those who agreed to share their personal habits and agreed for an oral examination were chosen as study subjects. A complete history of tobacco consumption and smoking was documented from the patient, including the kind, manner of usage, time span, frequency, and location of the tobacco (if chewable), as well as other harmful habits. Ethical committee clearance was obtained from the college for the present study.

Patients with tobacco habits were categorized into three groups based on the inclusion and exclusion criteria, namely as Group A: only Smoking, Group B: only tobacco, Group C: only areca nut Group D: mixed tobacco and areca nut.

Inclusion criteria

- Above 10 years of age including male and female.
- Patients who admit to tobacco and smoking use and to agree to an oral examination.
- Patients who continued the habit for 1 year minimum period and still consuming.

Exclusion criteria

- Patients who were being treated for any tobacco associated lesions
- Patients who discontinued the habit completely for the past 1 year

Result: Result shows a strong correlation between extrinsic stains and habit which is found in 150 (95.7%) and p value was calculated using chi square test and was 0.05 which is significant. Extrinsic stains were found to be more significant in group D patients. Attrition was found in 78 (48.4%) and was present more in group d patients and p value is 0.01 which is highly significant. Abrasion was found to be less associated with habit which was found in 23(14.3%).Dental caries was found in 97 (60.2%) and was found more in Group D patients. Gingival recession was found in 86 (53.4%) and was found more in group d patients. Smokers' palate and smoker's melanosis was found be associated with group A patients which is 19(61.3%) and 9 (29.9%) respectively. Leukoplakia was found to be more in group A patients which is 9 9(29.0%). Erythroplakia was found to be more prevalent in group B patients which is 4 (7.4%).Tobacco quid lesion was found to be more prevalent in group D patients which is 20(21%).OSMF was found to be more prevalent in group B patients which is 12 (52.2%). Lichenoid reaction was found to be more prevalent in Group B patients which is 3 (13%).Carcinoma of oral cavity was to be more prevalent in Group D patients which is 10 (10.9%).

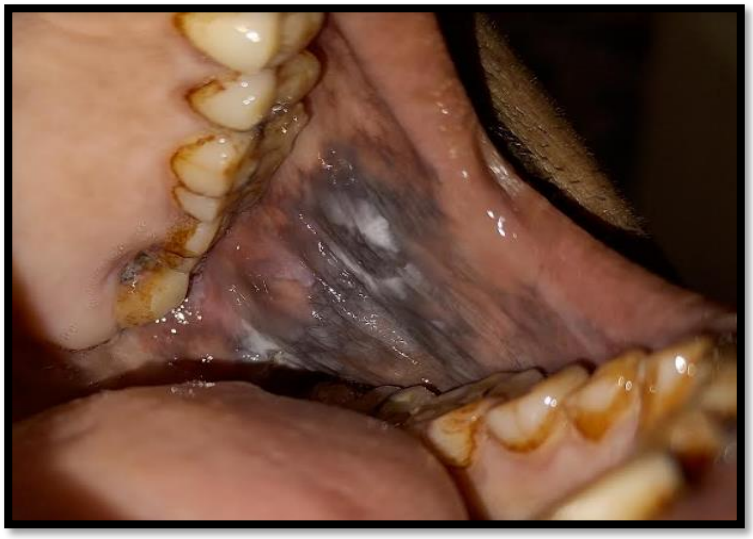

Figure 1: Leukoplakia on Left Buccal Mucosa

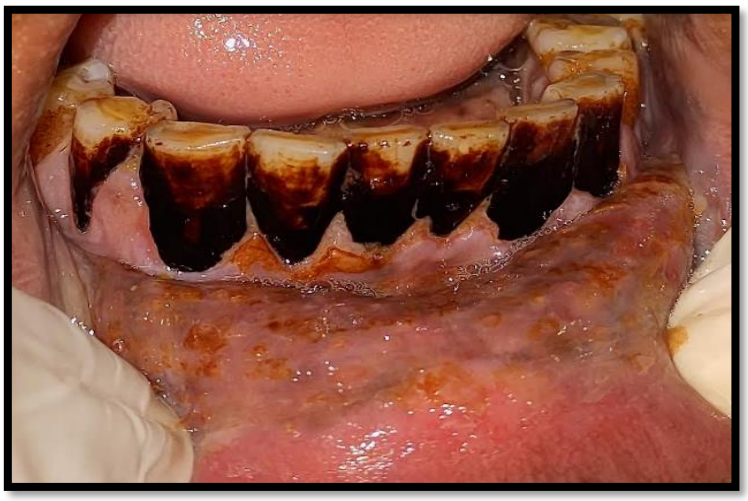

Figure 2: Tobacco Quid Lesion on Lower Labial Vestibule

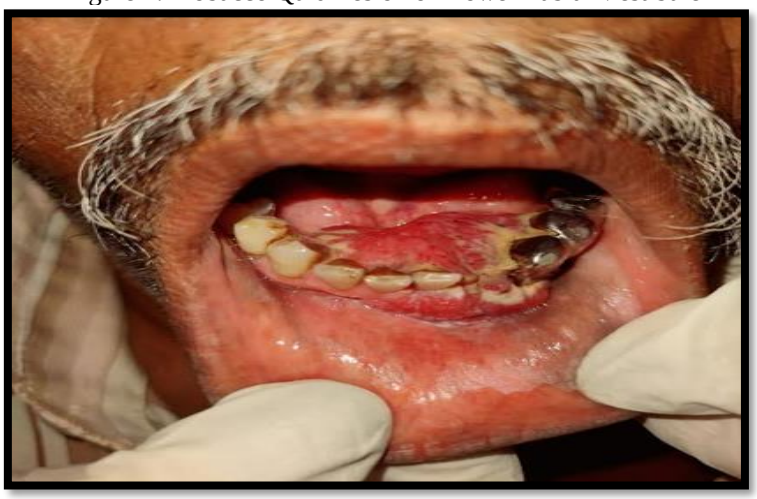

Figure 3: Carcinoma

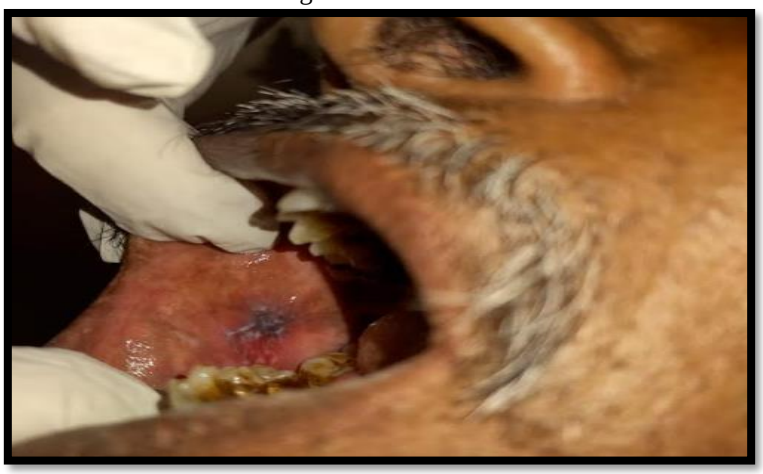

Figure 4: Tobacco Induced Lichenoid Reaction

Table 1: Shows descriptive analysis between habit and smokers palate					
			SMOKERSPALATE		Total
			Yes	No	
Habit	Smoking	Count	19	12	31
		% within Habit	61.3%	38.7%	100.0%
	Tobacco Chewing	Count	0	23	23
		% within Habit	0.0%	100.0%	100.0%
	Arecanut Chewing	Count	0	15	15
		% within Habit	0.0%	100.0%	100.0%
	Mixed	Count	0	92	92
		% within Habit	0.0%	100.0%	100.0%
	Total	Count	19	142	161
		% within Habit	11.8%	88.2%	100.0%

Table 2: Shows descriptive statistics between habit and carcinoma					
			CARCINOMA		Total
			Yes	No	
Habit	Smoking	Count	0	31	31
		% within Habit	0.0%	100.0%	100.0%
	Tobacco Chewing	Count	3	20	23
		% within Habit	13.0%	87.0%	100.0%
	Arecanut Chewing	Count	0	15	15
		% within Habit	0.0%	100.0%	100.0%
	Mixed	Count	10	82	92
		% within Habit	10.9%	89.1%	100.0%
	Total	Count	13	148	161
		% within Habit	8.1%	91.9%	100.0%

Table 3: Shows p value using chi square test for corelation between habit and carcinoma						
	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)	Point Probability
Pearson Chi-Square	5.774[a]	3	.123	.105		
Likelihood Ratio	9.281	3	.026	.035		
Fisher's Exact Test	5.416			.118		
Linear-by-Linear Association	2.323[b]	1	.128	.156	.076	.032
N of Valid Cases	161					

Discussion

Smokers melanosis: Smoker's melanosis is linked with both pipe smoking and cigarette. Tobacco smoke contains irritating components that either encourage melanin formation or induce melanin to bind to the noxious molecules in the smoke, resulting in pigmentation. Heavy smokers have increased pigmentation. Cigarette smoking causes pigmentation of the maxillary and mandibular alveolar mucosa, whereas pipe smoking causes pigmentation of the buccal mucosa and commissures. Women are more likely to develop smoker's melanosis, reflecting a possible synergistic impact of hormones and smoking. When you stop smoking, the pigmentation will fade away in 6 to 36 months. (12) (13) (14)

Nicotinic stomatitis (Smoker's Palate): Nicotinic stomatitis affects pipe smokers' hard palate, it can also affect cigar or cigarette users. In Men over the age of shows more prevalence of smoker's palate. The palate is keratotic, and the minor salivary glands of palate have been inflamed for a long period. It is possible to have several papules with punctuate reddish centres. Here are depicted irritable tiny salivary glands with inflamed duct orifices. (13) (14). It is a heat-induced response rather than a chemical reaction to tobacco, hence there is no danger of cancer. Tobacco usage, on the other hand, increases the incidence of squamous cell carcinoma of the tonsillar area, retromolar region, and respiratory tract. Nicotinic stomatitis is entirely reversible once tobacco and smoking is stopped. In 1 to 2 weeks, the palate tissue will recover to its natural look.

Periodontal disease: Smokers accounts for > 90% of individuals with refractory periodontitis, and smokers are more prone to loss of teeth due to periodontitis than non smokers (15). The higher frequency of periodontal disease in smokers may be due to a combination of the host response and the activities of periodontal bacteria. When it comes to periodontal infections, smokers have decreased oxygen tension in the periodontal pocket, which favors anaerobic organisms. (16) A positive BANA response is ten times more common among smokers.(17) Smoking reduces the capacity of bone and soft tissue revascularization.(18) as well as neutrophil activity. Neutrophils in smokers had decreased chemotaxis, phagocytosis, and adhesion, as well as a lesser capacity to eliminate bacteria.(19) (20) (21) The data is unambiguous that smoking contributes significantly to the development of many cases of periodontal disease, and that smoking during periodontal treatment results in a less-than-ideal outcome.

Tooth abrasion, dental stains and dental caries: In addition to the epithelial surface changes noted above, snuff/spit tobacco induces gingival keratin loss and tooth abrasion which leads to periodontal issues at the tobacco-placement area. The amount of root surface exposed will represent the degree of gingival recession and bone loss. Caries is more prone to develop on exposed root surfaces caused by cementum loss and dentin loss. As long as people smoke, the problem will not be fixed. (1)Tobacco stains (tar) are particularly visible in natural teeth's cervical (root) area. It's most visible on the lingual surfaces of the anterior teeth as brownish black discoloration and has ability to perforate exposed dentinal tubules. The surfaces more prone to discoloration are palatal surface of denture base and denture teeth.(21)

Oral cancer: Cancer has created a sense of morbidity and death in both the medical community and the general populace. The most common epithelial tumor of the oral cavity is oral squamous cell carcinoma, which is caused by genetic damage which results in uncontrolled cell growth of damaged cells. Tobacco-related oral cancers include epidermoid carcinoma (the most common malignant tumor of the oral cavity), erythroplakia, verrucous carcinoma, and (22) (23) (24) .Five-year survival rates are affected by the kind and size of cancer, the continuous use or nonuse of tobacco products, and the location of involvement.

Nitrosamines, one of the most powerful carcinogens known, are particularly prevalent in ST products among the many carcinogens found in both smoking and smokeless tobacco. (25) In terms of cancer of oral cavity, N′-nitrosonornicotine (NNN) and polycyclic aromatic hydrocarbons (PAHs) are the most significant carcinogens in cigarette (PAHs). NNN is a carcinogen produced by tobacco alkaloids called "tobacco-specific nitrosamines."

NNN and other tobacco-specific nitrosamines are present in all tobacco products. They are produced during the processing and curing of tobacco and are generally found in parts per million concentrations in tobacco, much above the levels of carcinogenic nitrosamines found in any other consumer product designed for human consumption. (26)

Leukoplakia and erythroplakia: Oral leukoplakia, white patch on the oral mucosa that cannot be scraped away and is not classified as any other lesion.(27) Its appearance might range from a smooth, somewhat transparent white spot to a thickened, cracked. Keratin layers accumulating on the oral epithelium generate the white patch. It has been linked to tobacco users' use of both smoking and smokeless tobacco. The type of tobacco product consumed influences the location and frequency of lesion development. Leukoplakias are commonly found in the labial and buccal mucosa (the mucobuccal fold region), the floor of the mouth, commissural area, the lateral border of the tongue, and the alveolar ridge of the mandible (28).

Both lesions are six times more likely among Tobacco users of smokeless type than in non-tobacco users and are more prevalent in men. Buccal and labial mucosa and lower buccal groves are typically involved sites among Smokeless tobacco users because of tobacco quid insertion at these regions. Three clinical forms of non-homogeneous varieties: I Speckled: a mixture of white and red (also known as erythroleukoplakia) with a prevalent white appearance; (ii) Nodular: small polypoid outgrowths with rounded red or white excrescences; and (iii) Verrucous: corrugated surface appearance.

Oral submucous fibrosis: Oral submucous fibrosis is characterized by scarring, tissue fibrosis, and premalignant lesions. It is quite frequent in the buccal mucosa (29) (30) .Pathological features include chronic inflammation, deposition of collagen in connective tissues underneath the mucosal epithelium, local inflammation in the lamina propria, and degeneration of muscles. OSMF individuals get a extreme burning sensation in their mouth after consuming spicy foods. Additional symptoms include dry mouth, pain, trismus, tongue movement restricted, difficulty in chewing, and altered tone. Because of its high malignant transformation rate (1.5-15%), this illness contributes considerably to death (31). OSMF is associated with lifestyle, nutrition, and culture, and its prevalence differ according to ethnicity and location (32) (33) (34) .India counts the highest number of OSMF patients in the world.

In Present study Dental caries was found in 97(60.2%) and was found more in Group D patients. Gingival recession was found in 86(53.4%) and was found more in group d patients. Smokers' palate and smoker's melanosis was found be associated with group A patients which is 19(61.3%) and 9 (29.9%) respectively. Leukoplakia was found to be more in group A patients which is 99 (29.0%). Erythroplakia was found to be more prevalent in group B patients which is 4 (7.4%).Tobacco quid lesion was found to be more prevalent in group D patients which is 20(21%).OSMF was found to be more prevalent in group B patients which is 12 (52.2%). Lichenoid reaction was found to be more prevalent in Group B patients which is 3 (13%). Carcinoma of oral cavity was to be more prevalent in Group D patients which is 10 (10.9%).

According to study conducted by Smitha et al, Keerthilatha et al Prevalence of oral habits was 17.54%. Indulgence in habit was seen more among males between age group 26-55yrs and most of them had education above high school level. Smoking (51.9%) was most common oral habit, followed by chewing tobacco (47.52%), consuming alcohol (29.7%) and chewing pan without tobacco (16.7%).Tobacco chewing was more common single habit. Prevalence of oral mucosal lesions was 46.1%. Reactive lesions (55.41%) were the most common followed by premalignant lesions/conditions(27.67%),malignant lesions (12.40%) and other lesions (4.49%). Reactive lesions were more common among smokers (68.98%) and alcoholics (40.03%). Premalignant (31.46%), malignant (12.08%) and other lesions (3.99%) were more common among tobacco chewers. Increase in number of habits was associated with increase in number of lesions. (35)

According to study conducted by Sanjay Kumar Singh, Priyanka Singh et al Almost all 98.6% inmates had adverse habits, exclusively use of tobacco (chewable + smoking). In male's gingival condition and oral sub mucous fibrosis was present in 79.2% inmates. In female inmates leukoplakia was present in 1.7%. Most common location for oral mucosal lesion was alveolar ridge/gingiva- 96.6% followed by buccal mucosa 40.9 %.(36)

Conclusion:

Both smoking and using smokeless tobacco increase the chance of acquiring oral premalignant lesions and mouth cancer. It also has a number of harmful effects on teeth and other structures. Oral cancer may be prevented by studying precancerous lesions and conditions in their early stages, the research of oral precancerous lesions and condition is important (37). Oral cancer must be diagnosed as soon as possible in order to have the highest chance of a successful treatment (38). Tobacco usage patterns should be evaluated by all medical providers, and tobacco prevention, cessation, and treatment programmes should be aggressively implemented.

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