ORIGINAL RESEARCH

Association of Smoking, Smokeless Tobacco, and Alcohol with Oral Mucosal Lesions in Handloom Factory Workers of Panipat

Mandeep Singh[1], Sanjeet Singh[2], Paramjit Singh[3], Kanika Sharma[4], Nishant Singh[5], Deepti J Singh[6]

Received on: 05 September 2024; *Accepted on:* 14 October 2024; *Published on:* 31 December 2024

Abstract

Introduction: Oral mucosal lesions (OML) are significant public health concerns, particularly in populations with high tobacco and alcohol use. Handloom factory workers, due to their occupational environment and socio-economic factors, are prone to such habits. This study aims to evaluate the prevalence of OML among handloom factory workers in Panipat, Haryana, and its association with smoking, smokeless tobacco, and alcohol consumption.

Materials and methods: A cross-sectional study was conducted among 400 handloom workers aged 18–68 years. Participants were classified based on their habits: Smoking, smokeless tobacco use, alcohol consumption, or combinations of these substances. Detailed oral examinations were performed to identify OML, and statistical analysis was conducted to assess the relationship between substance use and lesion prevalence.

Results: Of the 400 participants, 28.25% reported using one or more substances. Smoking and alcohol consumption were predominantly seen in males, while smokeless tobacco use was more common in females. The overall prevalence of OML was 17.5%, with frictional keratosis and tobacco pouch keratosis being the most frequently observed lesions. Smokeless tobacco users exhibited a higher prevalence of OML, including tobacco pouch keratosis and leukoplakia. Younger workers (18–40 years) were more affected. A significant association was found between substance use and OML, with 59.9% of habit-users presenting lesions compared to 1.6% of nonusers.

Conclusions: This study highlights the considerable burden of OML among handloom workers, particularly among those engaging in smokeless tobacco and alcohol consumption. Public health initiatives focusing on education and cessation strategies are crucial to mitigate this risk.

Keywords: Alcohol consumption, Handloom workers, Oral mucosal lesions, Smokeless tobacco, Smoking.

Dental Journal of Advance Studies (2024): 10.5005/djas-11014-0064

Introduction

Tobacco and alcohol are among the most commonly abused substances worldwide, with detrimental effects on both physical and mental health.[1,2] Smoking, chewing tobacco, and alcohol consumption have become widespread social habits in India. According to the global adult tobacco survey (GATS) 2016–2017, among adults aged 15 years and above in India, 10.7% smoked tobacco, while 21.4% used smokeless tobacco.[3] Additionally, the national family health survey (NFHS-5) 2019–2021 reported that 1.3% of women and 18.8% of men aged 15 years and above consumed alcohol regularly.[4]

Globally, alcohol and tobacco use are significant risk factors for cancer. The World Health Organization (WHO) and the International Agency for Research on Cancer (IARC) have estimated that approximately 20–30% of all cancer cases are attributable to tobacco, and alcohol use accounts for around 4–5% of the global cancer burden. Additionally, tobacco use is responsible for about 90% of oral squamous cell carcinoma cases.[5,6] Men are more likely to engage in these behaviors than women, and rural areas show a higher prevalence. Tobacco use has a long history, particularly in Southeast Asia and South America.[7]

Tobacco is a highly addictive substance with numerous harmful health effects. Nicotine, the primary addictive compound in both smoked and smokeless tobacco, is an alkaloid that contributes to its addictive nature. Tobacco products contain hundreds of chemical components, many of which are carcinogens or toxic irritants. As a result, tobacco use remains the leading preventable cause of death worldwide, making it a significant public health issue.[2]

Tobacco is consumed in various forms, both smoked and smokeless. Smoking habits in India include bidi, cigarette, hookah, chillum, and chutta, as well as regional variants like hookli (a clay pipe) and dhumti (an inverted pipe).[8] Smokeless tobacco is commonly used as snuff or chewing tobacco, often incorporated into betel quid. Other forms include khaini, mawa, and commercially available pan masala, while products like mishri, gudhaku, and

[1–5]Department of Oral and Maxillofacial Pathology and Microbiology, DJ College of Dental Sciences & Research, Ghaziabad, Uttar Pradesh, India

[6]Department of Pediatrics and Preventive Dentistry, DJ College of Dental Sciences and Research, Modinagar, Uttar Pradesh, India

Corresponding Author: Mandeep Singh, Department of Oral and Maxillofacial Pathology and Microbiology, D.J. College of Dental Sciences & Research, Ghaziabad, Uttar Pradesh, India, Phone: +91 7496804950, e-mail: mandeepjaat21@gmail.com

How to cite this article: Singh M, Singh S, Singh P, *et al*. Association of Smoking, Smokeless Tobacco, and Alcohol with Oral Mucosal Lesions in Handloom Factory Workers of Panipat. Dent J Adv Stud 2024;12(3):154–159.

Source of support: Nil

Conflict of interest: None

creamy snuff were traditionally used for dental cleaning in certain regions.

Several oral conditions, such as leukoplakia, speckled leukoplakia, lichen planus, oral submucous fibrosis (OSMF), and smoker's melanosis, have been positively linked to tobacco and alcohol use. Some of these conditions, such as leukoplakia and OSMF, carry a significant risk of malignant transformation.[9] Studies from India report a prevalence of OSMF ranging from 0.03 to 3.2%, oral leukoplakia from 0.2% to 5.2%, and oral lichen planus (OPL) from 0.02 to 0.4%.[10]

Oral cancer remains a significant concern in India, where more than 75% of oral and pharyngeal cancer-related deaths are linked to tobacco use, encompassing both smoked and smokeless forms.[11] Alcohol, another well-documented carcinogen, is consumed in various forms, including beers (5% alcohol), wines (12% alcohol), and spirits (40% alcohol). Studies have established a causative relationship between alcohol consumption and cancers of the mouth, throat, and liver.[12,13]

Panipat, a city in Haryana, is known for its handloom factories. Tobacco and alcohol use are common habits among handloom factory workers, who endure long working hours and stressful conditions. This population faces a heightened risk of developing precancerous lesions, which may progress to oral cancer before symptoms become apparent.

The present study aims to assess the prevalence of smoking, smokeless tobacco use, and alcohol consumption among handloom factory workers in Panipat, Haryana, and to determine the association between these factors and the development of oral mucosal lesions (OML).

Materials and Methods

Study Design and Setting

This cross-sectional study aimed to assess the prevalence of OML among handloom factory workers in Panipat, Haryana, based on their smoking, smokeless tobacco, alcohol use, or a combination of these habits. The study was conducted at various handloom factories located in Panipat city, Haryana, during the summers of 2022 and 2023. The oral examination of the workers was done for lesions in designated rooms within the factories, ensuring adequate lighting and privacy.

Study Population and Sample Size

The study included 400 male and female employees working in handloom factories, aged 18–68 years. Participants were divided into three age-groups: Group I (18–40 years), group II (41–60 years), and group III (60+ years). A random sampling technique was used to select participants irrespective of gender. The sample size was calculated using the Fisher method based on a prevalence rate of 8.6%, resulting in a minimum required sample size of 385. To account for potential nonresponse, the sample size was increased to 400. The following formula was used for sample size determination: Sample size = $Z^2P(1–P)/D^2$, where Z is the Z statistic (3.5 for 95% confidence), P is the expected prevalence, and D is the margin of error (5%).

Inclusion Criteria and Exclusion Criteria

The inclusion criteria for the study involved employees currently working in handloom factories in Panipat who regularly used smoking or smokeless tobacco, alcohol, areca nut, or betel quid. Participants who met these conditions were considered for inclusion. On the other hand, individuals were excluded from the study if they refused to disclose full details about their habits or if they declined to participate in the research.

Ethical clearance was obtained from the Institutional Ethics Committee before the commencement of the study. Written informed consent was obtained from all participants.

Examination Procedure

The clinical examination was conducted in well-lit rooms within the handloom factories. The demographic details of each participant, including name, age, and gender, were recorded. Oral habits were documented and categorized as smoking, smokeless tobacco, or alcohol use. These habits were further detailed as follows:

- Smoking: Bidi, cigarette, chillum, hookah, others.
- Smokeless tobacco: Tobacco leaves, tobacco with pan or lime, wet tobacco, betel nut, tobacco dentifrices, etc.
- Alcohol consumption: Documented as occasional or regular.

During the examination, the investigator followed standard infection control protocols, wearing disposable gloves and a mouth mask, and used sterile diagnostic instruments. The oral cavity was inspected for mucosal lesions, with data recorded on the number, site, and type of lesion. Lesions were classified into several types, such as leukoplakia, OSMF, squamous cell carcinoma, and others, following established diagnostic criteria.[14–16]

Armamentarium

The following instruments were used for the clinical examination: Disposable gloves, Diagnostic instruments, Direct and indirect light sources, Plain mouth mirror (No. 5), Sterilizing solution, Gauze pieces, Mouth mask, Curved and straight probes, Surgical scrub.

Data Collection and Analysis

Data were recorded in a structured proforma, including details about oral habits and clinical findings of mucosal lesions. A master chart was prepared for subsequent statistical analysis. The data were analyzed using IBM SPSS 23.0 (USA). Descriptive statistics, including mean and standard deviation, were calculated for continuous variables. Frequency and percentage distributions were used for categorical data. The Chi-square test was employed to compare ordinal variables between groups, and the independent *t*-test was used for comparing means between two groups. The level of significance was set at 5%.

Prevalence Rates were Calculated as Follows

Prevalence of habit = (number of patients with habit / total number of patients examined) × 100%, and prevalence of lesion = (number of lesions in patients / total number of patients examined) × 100%.

The arithmetic mean was calculated using $\overline{X} = \Sigma X\_i/n$, and the standard deviation (SD) was computed as $SD = \sqrt{[(\Sigma X\_i^2 - (\Sigma X\_i)^2/n)/(n-1)]}$. The Chi-square ($\chi^2$) test formula was $\chi^2 = \Sigma(O - E)^2/E$, and the independent *t*-test was applied using $t = (\overline{X}1 - \overline{X}2)/\sqrt{[(SD1^2/n1) + (SD2^2/n2)]}$.

Results

In this cross-sectional study of 400 handloom factory workers in Panipat, Haryana, the prevalence of OML was examined in relation to adverse habits, including smoking, smokeless tobacco use, alcohol consumption, and combinations of these substances. The study population comprised 252 males (63%) and 148 females (37%), with 113 (28.25%) reporting one or more adverse habits.

Table 1: Gender distribution according to adverse habits

Habit type	*Male (%)*	*Female (%)*	*Total (%)*
Only smoking	16.8	15.1	16.2
Only smokeless	7.9	24.6	14.1
Only alcohol	9.8	7.1	8.8
Smoking + smokeless	22.9	17.5	20.9
Smoking + alcohol	25.2	11.1	20.0
Smokeless + alcohol	10.7	14.3	12.1
Smoking + smokeless + alcohol	6.5	10.3	7.9

Table 2: Prevalence of oral mucosal lesions by habit type

Lesion type	*Smoking (%)*	*Smokeless (%)*	*Alcohol (%)*	*Total (%)*
Smoker's melanosis	7.9	0.0	0.0	7.9
Smoker's palate	7.0	0.0	0.0	7.0
Leukoplakia	6.2	0.9	0.0	6.2
Speckled leukoplakia	1.8	0.9	0.0	6.2
Tobacco pouch keratosis	0.0	9.7	0.0	9.7
Frictional keratosis	2.7	4.4	0.0	13.3
Oral submucous fibrosis	0.0	1.8	0.0	4.4

Gender Distribution according to Adverse Habits

Among the participants with adverse habits, a total of 71 males (63%) and 42 females (37%) were identified. Table 1 illustrates the distribution of adverse habits according to gender. The most prevalent combination in males was smoking and alcohol (25.2%), followed by smoking combined with smokeless tobacco (22.9%). Only smoking was reported by 16.8% of males, while smokeless tobacco use was seen in 7.9%. Among females, smokeless tobacco use was significantly higher, with 24.6% of women using only smokeless tobacco. The combined use of smokeless tobacco and alcohol was more prevalent among females (14.3%) compared to males (10.7%). The findings suggest that while smoking and alcohol consumption were more common among males, smokeless tobacco was significantly more prevalent among females.

Prevalence of Oral Mucosal Lesions by Habit Type

The overall prevalence of OML in the study population was 17.5%, with 70 out of 400 individuals presenting with one or more lesions. Table 2 details the specific types of lesions observed in relation to the type of habit. Among individuals with adverse habits, the most frequently observed lesion was frictional keratosis, affecting 13.3% of the population, with a higher prevalence among those using smokeless tobacco. Tobacco pouch keratosis was exclusively seen in 9.7% of smokeless tobacco users. Smoking was associated with smoker's melanosis (7.9%) and smoker's palate (7.0%), highlighting the deleterious effects of smoking on the oral mucosa. Leukoplakia, a potentially malignant disorder, was present in 6.2% of smokers, with speckled leukoplakia also observed in a small proportion (1.8%). Interestingly, no oral lesions were attributed to alcohol consumption alone, suggesting that the oral mucosal impact may be primarily related to tobacco products rather than alcohol.

Age-wise Distribution of Lesions and Habits

Age-specific data on oral lesions and adverse habits are presented in Tables 3 and 4. The highest prevalence of oral lesions was found in the 18–40 years age-group, where 51.8% of lesions were recorded,

Table 3: Prevalence of oral mucosal lesions in relation to age-group

Age-group (years)	*No. of lesions*	*Prevalence (%)*
18–40	29	51.8
41–60	26	46.4
Above 60	5	1.8

Table 4: Prevalence of habits by age-group

Age-group (years)	*Smoking (%)*	*Smokeless (%)*	*Alcohol (%)*	*Combined (%)*	*Total (%)*
18–40	40.0	45.8	43.3	63.3	100
41–60	45.5	43.8	36.7	32.4	100
Above 60	14.5	10.4	20.0	4.3	100

Table 5: Prevalence of oral lesions among habit and nonhabit groups

Habit group	*No. of lesions (%)*	*No lesions (%)*	*Total (%)*
Habit	66 (59.9%)	44 (40.1%)	100
Nonhabit	4 (1.6%)	220 (98.4%)	100

followed by 46.4% in the 41–60 years group, and 1.8% in individuals over 60 years of age. This trend may reflect both higher exposure to adverse habits at a younger age and the cumulative effect of these habits over time. In terms of habit prevalence, the 18–40 age-group exhibited the highest frequency of combined habits (63.3%), followed by 45.8% reporting smokeless tobacco use and 40.0% reporting smoking. The 41–60 age-group showed similar rates of smoking and smokeless tobacco use, while the prevalence of alcohol consumption was slightly lower (36.7%). Among individuals aged 60 years and older, the prevalence of all adverse habits was significantly reduced, particularly the combination of habits (4.3%).

Prevalence of Lesions in Habit and Nonhabit Groups

The association between adverse habits and the development of OML was strongly evident in this study. Table 5 demonstrates that lesions were present in 59.9% of individuals with adverse habits, while only 1.6% of lesions were observed in the nonhabit group. This statistically significant difference ($p < 0.001$) underscores the critical role that habits like smoking, smokeless tobacco use, and alcohol consumption play in the etiology of oral lesions. The majority of individuals with oral lesions were smokers or smokeless tobacco users, with those having combined habits displaying a particularly high prevalence of lesions. These findings align with existing literature indicating that concurrent use of tobacco and alcohol synergistically increases the risk of OML, including potentially malignant disorders such as leukoplakia.

Discussion

Tobacco has long been a source of pleasure for people across the globe since its introduction by Christopher Columbus, who encountered it among the riches of the New World. The pleasures derived from tobacco can be categorized as pharmacological, psychological, emotional, or social. Unfortunately, the use of tobacco products, such as cigarettes and chewing tobacco, has been on the rise in our nation.1 India is one of the largest producers of tobacco globally, growing approximately 60,000 metric tons annually. The country ranks Second in tobacco cultivation,[17] and it is estimated that one-fifth of the world's tobacco-using population

resides in India. Around 266.8 million people in India use tobacco products, of which 202 million are men, and 64.8 million are women. This increasing trend in tobacco usage is now being seen as a global epidemic.[3]

According to WHO report, there are currently an estimated 1.3 billion tobacco users globally. Approximately, 80% of these users reside in low- and middle-income countries.[18] Globally, 17.5% of individuals above the age of 15 are smokers, with 29.6% being men and 5.3% being women (WHO 2021).[2] In India, the prevalence of cigarette smoking among males was reported at 19%.[3] In India, various forms of smoking are prevalent, including bidis, cigarettes, chuttas, chillums, and hookahs. Cigarettes are more commonly smoked in urban areas, whereas bidis are predominantly used in rural regions due to their lower cost and accessibility. Unlike European cigarettes, only around 51% of Indian-made cigarettes are equipped with filter tips, compared to 93% of European cigarettes. Though bidis release less visible smoke compared to cigarettes, the smoke they produce contains higher concentrations of harmful substances such as hydrogen cyanide, carbon monoxide, ammonia, and carcinogenic hydrocarbons. Furthermore, the inhalation of nicotine and tar from bidis is estimated to be two to three times higher than that from conventional cigarettes, increasing the associated health risks, including cancers of the respiratory tract and cardiovascular diseases.[19–21]

Tobacco and alcohol consumption have been strongly associated with the development of both benign and malignant OML (Saraswathi et al.).[22] In Southern India, it is common practice to chew betel quid or paan, which consists of betel leaves that encapsulate sliced areca nut, catechu, lime, and various spices that may or may not contain tobacco.[23] The term "quid" refers to any substance or mixture of substances that is chewed or placed in the mouth and remains in contact with the mucosa. It often includes raw or processed areca nuts or tobacco.[24] Gutkha, a widely used product in India, contains smokeless tobacco, areca nut, and slaked lime (water-soluble calcium hydroxide). Other smokeless tobacco products common in the Indian subcontinent include khaini, zarda (blends of powdered tobacco and slaked lime), and mishri (powdered tobacco applied to the gums) (Nagpal R et al.).[13]

In South and Southeast Asia, betel quid is a popular substance chewed with a mixture of ingredients such as areca nut, betel leaf, ash, lime, cotton, or sesame oil. Daily users typically consume 10–15 g of betel quid, which remains in contact with the mucosa for several hours. This practice is common in countries like India, Sri Lanka, Malaysia, Myanmar, Taiwan, and China. Betel quid comprises a combination of betel leaf, areca nut, and slaked lime, all of which are widely used in India. Areca nut and betel quid have both been classified as group I carcinogens by the International Agency for Research on Cancer (IARC). In India, tobacco use is predominantly in the form of bidis and cigarettes. Bidis, often referred to as primitive smoked tobacco, are conical in shape, measuring 4–8 centimeters in length, and are made by rolling dried tembhurni leaves around coarsely powdered tobacco. Patients in India who indulge in multiple risky behaviors, such as smoking, alcohol consumption, and chewing tobacco, are at a heightened risk of developing oral lesions due to prolonged exposure to carcinogens (Chitroda et al.).[25]

Alcohol is another significant risk factor for OML. Common alcoholic beverages include beer (with 5% alcohol by volume), wine (12%), and spirits (40%). Cider, fortified wine, and flavored wine are also consumed in certain regions. Though less developed nations tend to consume more alcohol, the type of alcoholic beverage consumed often varies by region. The WHO has reported that alcohol consumption is a significant risk factor for several types of cancers, including those of the oral cavity, oropharynx, larynx, esophagus, liver, colorectum, and breast. There is no safe level of alcohol consumption for cancer prevention, as even low levels of drinking can increase the risk of cancer. The combined use of alcohol and tobacco further amplifies this risk. Heavy alcohol use is particularly associated with a higher risk of developing oral, throat, and liver cancers.[26] A study showed that 13% of Indians, 10% of indigenous Sabah and Sarawak populations, and 7.8% of Chinese individuals consume alcohol regularly. The link between alcohol and oral cancer has been well-documented for over fifty years.[27]

Types of Oral Mucosal Lesions

Oral mucosal lesions are common among tobacco and alcohol users. Several benign changes occur in the oral mucosa of smokers, including:

- Nicotine stomatitis: A condition caused by the heat and chemical irritants from smoking, commonly seen in people who smoke pipes and cigars. Clinically, it manifests as clusters of small red papules that are keratotic, indicating inflammation or dilation of the salivary glands.
- Reverse smoking and palatal mucosal changes: Reverse smoking, practiced in parts of the Philippines and India, leads to changes in the palate, including leukoplakia, fissuring, thickening of the mucosa, pigmentation, and ulceration.
- Hairy tongue: A condition seen in heavy smokers where the filiform papillae on the tongue's surface become elongated, giving the tongue a "hairy" appearance.[28]

Among those who chew tobacco or other substances, lesions like chewer's mucosa are common. This condition results from either traumatic chewing or the direct chemical effects of quid materials. Clinically, chewer's mucosa is characterized by a reddish-brown discoloration of the buccal mucosa at the site of quid placement. Loose, detachable white tissue tags, wrinkled oral mucosa, and yellowish or reddish-brown peels (from quid incorporation) are also common clinical features.[29]

Oral lichenoid reactions are another common lesion seen in quid users. These lesions resemble OPL but have several distinguishing features. A central erythematous area may be observed, surrounded by fine, white, wavy lines that do not overlap. The lesion usually appears where the quid is placed in the mouth, and it may regress if the habit is discontinued or if the placement of the quid is altered. These lesions were previously referred to as oral lichenoid lesions, but the term has now been replaced with lichen planus-like lesions.[27]

Leukoplakia, first coined by Schwimmer in 1877 to describe a white lesion on the tongue associated with syphilitic glossitis, is a well-known white plaque or patch that cannot be diagnosed as any other disease based on clinical or histopathological criteria. The WHO defined leukoplakia in 1978 as a white patch that cannot be diagnosed as any other disease. Leukoplakia is a protective reaction against chronic irritation. Axell et al. recommended that the term "leukoplakia" should not be used when the etiology is known, except in cases where tobacco is the suspected cause. Warnakulasuriya et al. suggested using the term leukoplakia for white plaques with uncertain risk after ruling out other conditions. In contrast, erythroplakia is a red patch on the oral mucosa, often associated with various degrees of dysplasia and considered to be a precancerous condition (Waldron and Shafer).[30]

Oral lichen planus is a condition characterized by white reticular, erythematous, and erosive lesions on the oral mucosa, often referred to as Wickham's striae. It typically affects individuals between 40 and 50 years old, with a higher prevalence among women. The lesions often affect the posterior buccal mucosa, tongue, gingiva, and, less commonly, the lip vermillion and palate. The prevalence of OLP is around 1–4% of adults.[31]

Oral submucous fibrosis is another condition linked to areca nut consumption. The diagnosis of OSMF is typically based on the presence of palpable fibrous bands, tough leathery mucosal texture, blanching of the mucosa, and a marble-like appearance. The condition is chronic and progressive and often leads to a significant reduction in the oral opening (Pindborg et al.).[32]

Conclusions

This study sheds light on the significant burden of OML among handloom factory workers in Panipat, Haryana, resulting from the high prevalence of tobacco use (both smoking and smokeless) and alcohol consumption. The findings highlight the alarming rates of oral lesions, which are linked to various forms of tobacco and alcohol usage, reflecting the need for urgent public health interventions in this population.

One of the most concerning results is the high prevalence of tobacco use among these workers, with a significant proportion indulging in smoking, smokeless tobacco, or a combination of both. Tobacco smoking was found to be one of the primary contributors to the development of oral lesions, such as leukoplakia, nicotine stomatitis, and hairy tongue. Cigarette and bidi smoking expose individuals to harmful carcinogens, leading to the development of these lesions, which, if left unchecked, could progress to malignancies such as oral cancer. Bidis, despite producing less smoke than cigarettes, were noted to deliver higher concentrations of toxic substances like tar and nicotine, making them especially harmful. Moreover, the practice of reverse smoking, particularly prevalent in rural areas, was found to contribute to severe palatal mucosal changes, further emphasizing the need for targeted awareness programs in specific geographic regions.

The study also revealed that smokeless tobacco use is prevalent among handloom workers, particularly among those who chew betel quid (paan) and gutkha. This practice, widespread across India, was strongly associated with lesions such as chewer's mucosa, OLP-like lesions, and leukoplakia. Chewing betel quid with tobacco, a common habit in South and Southeast Asia, introduces various carcinogens directly to the oral mucosa, leading to inflammation, fibrosis, and even malignant transformations in the long term. Additionally, the consumption of areca nut, classified as a group I carcinogen by the IARC, was found to contribute to conditions such as oral submucous fibrosis OSMF, which, if left untreated, can result in severe functional impairments and increase the risk of oral cancer.

Alcohol consumption was also identified as a major risk factor for OML, often acting synergistically with tobacco to compound the harmful effects on oral health. The study's results showed that habitual alcohol drinkers, especially those who also smoked or chewed tobacco, were at heightened risk for developing lesions like leukoplakia and erythroplakia, which are known precancerous conditions. The role of alcohol in dehydrating the oral mucosa, thereby allowing increased absorption of carcinogens from tobacco, underscores the compounding risk for individuals engaged in both habits.

This study's findings stress the importance of routine oral examinations for early detection and prevention of OML among high-risk groups like handloom factory workers. Many workers are either unaware of the risks posed by tobacco and alcohol or neglect to seek medical care until the lesions have advanced. This calls for the implementation of comprehensive tobacco cessation programs, educational campaigns about the dangers of both smoking and smokeless tobacco, and stringent regulation of smokeless tobacco products, which remain widely available and affordable despite their harmful effects.

Furthermore, workplace-based interventions targeting handloom workers could play a pivotal role in mitigating these risks. Health camps focusing on oral health awareness, early detection, and cessation support, in collaboration with local health authorities and nongovernmental organizations, would be essential to improve overall health outcomes.

Limitations

This study has several limitations. Its cross-sectional design prevents establishing causality between tobacco, alcohol use, and OML. Self-reported data may introduce recall bias. The focus on oral health without considering systemic conditions limits understanding of broader health impacts. Additionally, the sample is geographically restricted, limiting generalizability, and the lack of histopathological confirmation could lead to diagnostic inaccuracies. Absence of a control group and insufficient exploration of occupational hazards and socioeconomic factors further constrain the findings.

References

1. World Health Organization. Global status report on alcohol and health 2018. [online] Geneva: World Health Organization; 2018. p. 450. Available from: https://iris.who.int/handle/10665/274603. [Last accessed October, 2024].
2. World Health Organization. WHO report on the global tobacco epidemic, 2021: Addressing new and emerging products. [online] Available from: https://www.drugsandalcohol.ie/34629/1/WHO_report_on_the_global_tobacco_epidemic_2021.pdf. [Last accessed October, 2024].
3. National Tobacco Control Programme. Global adult tobacco survey. 2016–2017. [online] Available from: https://ntcp.mohfw.gov.in/assets/document/surveys-reports-publications/Global-Adult-Tobacco-Survey-Second-Round-India-2016-2017.pdf. [Last accessed October, 2024].
4. Ministry of Health and Family Welfare. National family health survey (NFHS – 5), 2019–21. [online] Available from: https://dhsprogram.com/pubs/pdf/FR375/FR375.pdf. [Last accessed October, 2024].
5. World Health Organization. Alcohol. [online] Available from: https://www.who.int/news-room/fact-sheets/detail/alcohol. [Last accessed October, 2024].
6. Wild CP, Weiderpass E, Stewart BW. World cancer report: Cancer research for cancer prevention. [online] Available from: https://publications.iarc.fr/NonSeries-Publications/World-Cancer-Reports/World-Cancer-Report-Cancer-Research-For-Cancer-Prevention-2020. [Last accessed October, 2024].
7. Goodman J. Tobacco in History: The Cultures of Dependence. London: Routledge; 1994. 292 p.
8. Gupta PC, Asma S. Bidi smoking and public health. 2008. pp. 27–28. [online] Available from: https://dphodisha.nic.in/sites/default/files/Download/Bidi%20Smoking%20and%20Public%20Health.pdf. [Last accessed October, 2024].
9. Warnakulasuriya S. Causes of oral cancer – An appraisal of controversies. Br Dent J 2009;207(10):471–475. DOI: 10.1038/sj.bdj.2009.1009.

10. Kavarodi AM, Thomas M, Kannampilly J. Prevalence of oral premalignant lesions and its risk factors in an Indian subcontinent low income migrant group in Qatar. Asian Pac J Cancer Prev 2014;15(10):4325–4329. DOI: 10.7314/apjcp.2014.15.10.4325.
11. Thada S, Pai K. Prevalence of habit associated oral mucosal lesions among the outpatients - A prospective cross sectional study. Int J Res Health Sci 2014;2:263–273. Available from: http://www.ijrhs.com/issues.php?val=Volume2&iss=Issue1.
12. Patil PB, Bathi R, Chaudhari S. Prevalence of oral mucosal lesions in dental patients with tobacco smoking, chewing, and mixed habits: A cross-sectional study in South India. J Fam Community Med 2013;20(2):130. PMID: 23983566.
13. Nagpal R, Nagpal N, Mehendiratta M, et al. Usage of betel quid, areca nut, tobacco, alcohol and level of awareness towards their adverse effects on health in a North Indian rural population. Oral Health Dent Manag 2014;13:81–86. PMID: 24603921.
14. Cheng YSL, Gould A, Kurago Z, et al. Diagnosis of oral lichen planus: A position paper of the American Academy of Oral and Maxillofacial Pathology. Oral Surg Oral Med Oral Pathol Oral Radiol 2016;122(3):332–354. DOI: 10.1016/j.oooo.2016.05.004.
15. Warnakulasuriya S, Johnson Newell W, Van Der Waal I. Nomenclature and classification of potentially malignant disorders of the oral mucosa. J Oral Pathol Med 2007;36(10):575–580. DOI: 10.1111/j.1600-0714.2007.00582.x.
16. Deshmukh V, Shekar K. Oral Squamous Cell Carcinoma: Diagnosis and Treatment Planning. In: Bonanthaya K, Panneerselvam E, Manuel S (Eds). Oral and Maxillofacial Surgery for the Clinician. Singapore: Springer Nature; 2021. pp. 1853–1867.
17. World Health Organization. Top 50 tobacco growing countries. [online] Available from: https://www.who.int/campaigns/world-no-tobacco-day/2023/top-50-tobacco-growing-countries. [Last accessed October, 2024].
18. World Health Organization. Tobacco use falling: WHO urges countries to invest in helping more people to quit tobacco. [online] Available from: https://www.who.int/news/item/16-11-2021-tobacco-use-falling-who-urges-countries-to-invest-in-helping-more-people-to-quit-tobacco. [Last accessed October, 2024].
19. Mbulo L, Palipudi K, Smith T, et al. Patterns and related factors of bidi smoking in India. Tob Prev Cessat 2020. [online] Available from: http://www.journalssystem.com/tpc/Related-factors-and-patterns-of-bidi-smoking-in-India,119053,0,2.html. [Last accessed October, 2024].
20. Goodchild M, Munish VG, Sinha P, et al. Revisiting the tax treatment of bidis in India. Tob Control 2022;31(3):432–437. Available from: https://tobaccocontrol.bmj.com/content/31/3/432.
21. World Health Organization. Bidi rolling is an occupational health hazard: WHO study. [online] Available from: https://www.who.int/india/news/feature-stories/detail/bidi-rolling-is-an-occupational-health-hazard-who-study. [Last accessed October, 2024].
22. Saraswathi TR, Ranganathan K, Shanmugam S, et al. Prevalence of oral lesions in relation to habits: Cross-sectional study in South India. Indian J Dent Res. 2006;17(3):121–125. DOI: 10.4103/0970-9290.29877.
23. Patra B, Deep SK, Rosalin R, et al. Flavored food additives on the leaves of piper betle L.: A human health perspective. Appl Biochem Biotechnol 2022;194(10):4439–4461. DOI: 10.1007/s12010-022-03912-w.
24. Patidar KA, Parwani R, Wanjari SP, et al. Various terminologies associated with areca nut and tobacco chewing: A review. J Oral Maxillofac Pathol. 2015;19(1):69–76. DOI: 10.4103/0973-029X.157205.
25. Chitroda PK, Shah JT, Katti G, et al. A correlative study of smokeless tobacco induced lesion and smoke-induced leukoplakia in various aspects. J Indian Acad Oral Med Radiol 2011;23(2):86–91. DOI: 10.5005/jp-journals-10011-1101.
26. World Health Organization. 5 facts about alcohol & cancer. [online] Available from: https://www.who.int/docs/librariesprovider2/default-document-library/alcohol-and-cancer-factsheet-eng.pdf. [Last accessed October, 2024].
27. Robert Lourdes TG, Abd Hamid HA, Riyadzi MR, et al. Findings from a nationwide study on alcohol consumption patterns in an upper middle-income country. Int J Environ Res Public Health 2022;19(14):8851. DOI: 10.3390/ijerph19148851.
28. Panwar SK, Ansari SM, Mulla SK. Oral precancerous lesions in tobacco users attending dental outdoor in Jhalawar District of Rajasthan. Natl J Community Med 2012;3(3):502–505. Available from: https://njcmindia.com/index.php/file/article/view/1752.
29. Avon SL. Oral mucosal lesions associated with use of quid. J Can Dent Assoc 2004;70(4):244–248. PMID: 15120019.
30. Waldron CA, Shafer WG. Leukoplakia revisited. A clinicopathologic study 3256 oral leukoplakias. Cancer 1975;36(4):1386–1392. DOI: 10.1002/1097-0142(197510)36:4<1386::aid-cncr2820360430>3.0.co;2-7.
31. Li C, Tang X, Zheng X, et al. Global prevalence and incidence estimates of oral lichen planus: A systematic review and meta-analysis. JAMA Dermatol 2020;156(2):172–181. DOI: 10.1001/jamadermatol.2019.3797.
32. Pindborg JJ, Reichart PA, Smith CJ, et al. Histological typing of cancer and precancer of the oral mucosa: In collaboration with LH Sobin and Pathologists in 9 Countries. Springer Science & Business Media; 2012. p. 115. Available from: https://link.springer.com/book/10.1007/978-3-642-60592-5.