International Journal of Community Medicine and Public Health
Joshi A et al. Int J Community Med Public Health. 2019 Aug;6(8):3625-3629
http://www.ijcmph.com
pISSN 2394-6032 | eISSN 2394-6040

DOI: http://dx.doi.org/10.18203/2394-6040.ijcmph20193123

Original Research Article

Association of oral pre-malignant lesions with the consumption of tobacco

Arpita Joshi[1], Chinmay Jani[2]*, Kushboo Jain[3], D. V. Bala[4], Divyesh Jetpariya[5], Harshal Jadav[6]

[1]Department of Medicine, Mount Sinai- Elmhurst, New York, USA
[2]Smt NHL Municipal Medical College, Ahmedabad, Gujarat, India
[3]Department of Radiation Oncology, GCRI Hospital, Ahmedabad, Gujarat, India
[4]Head of the Department, Department of Community Medicine, Smt NHL Municipal Medical College, Ahmedabad, Gujarat, India
[5]Department of Orthopedics, C U Shah Medical College, Surendranagar, Gujarat, India
[6]Department of Radiology, M P Shah Medical College, Jamnagar, Gujarat, India

Received: 17 June 2019
Revised: 02 July 2019
Accepted: 03 July 2019

***Correspondence:**
Dr. Chinmay Jani,
E-mail: ctjani1494@gmail.com

ABSTRACT

Background: Tobacco consumption in the form of chewing and smoking is one of the biggest addictions. It causes various oral pre-malignant lesions which mainly include oral sub-mucous fibrosis (OSMF) caused by betel nut consumption and tobacco quid lesion, leukoplakia, erythroplakia and smoker's palate caused by chewing and smoking tobacco.
Methods: The data about the past and present consumption of tobacco was collected from 909 individuals. Based on the tobacco habits, the oral cavity findings were assessed and the statistical analysis was carried out to find the significance of associations observed between tobacco consumption and lesions.
Results: Out of 909 subjects, 537 were consuming tobacco, out of which 284 were chewers, 65 were smokers and 76 were consuming in both forms. Out of the 284 tobacco chewers, 43% had oral lesions (19.4% had OSMF and 21.5% had tobacco quid) as compared to just 1% of workers having oral lesions (OSMF) amongst those who had no addiction. There was a high prevalence of lesion in tobacco chewers ($p<0.005$). Out of total smokers, 15.8% had smoker's palate whereas no lesion was seen in the non-addicted workers. The odds of developing oral pre-malignant lesion are 43.62 times higher in exposed individuals.
Conclusions: The tobacco consumption in any form is hazardous. Betel nut exposure causes more harmful effect which goes unnoticed. Awareness should be created about various lesions caused by tobacco as well as betel nut and special steps should be taken for early diagnosis and its treatment.

Keywords: Oral pre-malignant lesions, Tobacco smoking and chewing, Betel nut

INTRODUCTION

Tobacco use is a major public health problem globally. According to the World Health Organization (WHO), tobacco is the second most important cause of death in the world. It kills 5 million people every year.[1] Tobacco use is not only addictive but extremely harmful. All forms of tobacco cause fatal and disabling health problems throughout the life. Scientific evidence has linked tobacco use with the development of more than 25 diseases.

Tobacco is used in different forms-smoke and smokeless. Smoking is through cigarettes, bidis, hookkah and chilam

(ganja). Smokeless tobacco products include tobacco that is used in pan, gutkha, zarda, khaini, and dohra. Smoking tobacco is the major cause of lung cancer, chronic obstructive pulmonary disease (COPD), peripheral vascular disease, and various throat and mouth cancers.

Tobacco use is influenced by a variety of factors, including individual attitudes and beliefs, social norms and acceptability, availability, and advertising campaigns. There are many misperceptions with regard to tobacco use, for example that it aids concentration, suppresses appetite, reduces anxiety and tension, causes skeletal muscle relaxation, and induces feelings of pleasure. Partly as a result of these perceived benefits tobacco consumption is highest in the labor classes and among those from a low socioeconomic status. Several studies have shown that tobacco use is higher among the less educated or illiterate, and the poor and marginalized groups.[2,3]

Oral malignancies constitute 3% of all malignancies. Most of oral cancers occur as squamous cell carcinoma (SCC). Many oral squamous cell carcinomas develop from premalignant conditions of the oral cavity like oral sub mucous fibrosis, leukoplakia, smoker's palate, erythroplakia, tobacco quid lesion and non-healing ulcers. In order to prevent malignant transformation of these precursor lesions, multiple screening and detection techniques have been developed. The early detection of cancer is of critical importance because survival rates markedly improve when the oral lesion is identified at an early stage.

Common pre-malignant oral lesions

Leukoplakia: The World Health Organization (WHO) group has defined leukoplakia as "a white patch or plaque that cannot be characterized, clinically or pathologically, as any other disease". These lesions are potentially premalignant and vary in size, shape, consistency and macroscopically said to be homogenous and nodular. Histologic examination may reveal hyperkeratosis, dysplasia, carcinoma-in-situ (CIS) or invasive squamous cell carcinoma. Dysplasia occurs in as many as 30% of leukoplakic lesions, whereas a small percentage of lesions show invasive SCC on pathologic examination.[4]

Erythroplakia: Erythroplakia and/or erythroleukoplakia is a red or erythematous patch of the oral mucosa and is associated with significantly higher rates of dysplasia, CIS, and invasive carcinoma than leukoplakia.[5]

Oral sub mucous fibrosis: Oral sub-mucous fibrosis (OSMF) is a chronic, debilitating disease characterized by inflammation and progressive fibrosis of the sub-mucosal tissues (lamina propria and deeper connective tissues).[6] It results in marked rigidity and an eventual inability to open the mouth. The buccal mucosa is the most commonly involved site, but any part of the oral cavity can be involved, even the pharynx. The pathogenesis of OSMF is not well established and hence is believed to be multifactorial. The cascade begins with a juxtaepithelial inflammatory reaction in the oral mucosa, interspersed with healing and fibrosis, with trismus being the end result. Areca nut is considered to play an important causative role in OSMF.[7]

Smoker's palate: It is characterized by diffuse white patch on the hard palate, usually caused by tobacco smoking. It is painless and is caused by a response of the palatal oral mucosa to chronic heat. A more pronounced appearance can occur with reverse smoking, sometimes distinguished from stomatitis nicotina by the term reverse smoker's keratosis.[8]

Tobacco quid lesion: Tobacco-associated keratosis is an ill-defined area of white thickening at the sites where oral smokeless tobacco is habitually placed; most commonly, these areas involve the mandibular, labial and buccal mucosal folds. The continued use of smokeless tobacco causes the affected areas to become corrugated and gray in colour giving broken mud appearance.

In India, tobacco use is estimated to cause 800,000 deaths annually.[9] The WHO predicts that tobacco deaths in India may exceed 1.5 million annually by 2020.[10] The present study was done to determine prevalence of tobacco use in a vulnerable population, i.e., Fabrication industry workers, thermal power station workers.

The main aim of the study is to find out the prevalence of tobacco consumption in the study population and associate the prevalence of Oral Pre malignant lesions with the consumption of tobacco.

Other objectives of this study were:

- To differentiate the lesions seen in smokers and those consuming smokeless tobacco (SLT).
- To find out the difference in the risk of developing OMPLs in those currently consuming tobacco and those who quit the habit for more than 6 months.

METHODS

A cross-sectional observational study of 909 industrial workers working in four industries in Ahmedabad and Gandhinagar was undertaken from March 2014 to October 2014. The permission to carry out this study was taken from the Dean of NHLMMC and Management of the respective industries. Ethical clearance from the Institutional Review Board was taken. Informed consent was taken from each subject in the study.

Inclusion criteria

Inclusion criteria were population giving an informed consent to the study. Cases were those who had consumed tobacco in either form for over a year.

Exclusion criteria

Exclusion criteria were known cases of oral cancer taking treatment.

Sociodemographic history and the history of current and past tobacco consumption were recorded. Details of type of tobacco, amount and duration of consumption as well as any other history of habits were recorded. All the subjects underwent general health checkup, anthropometrical measurements and examination of the oral cavity.

The oral cavity was examined for pre-malignancy. Those detected with lesions were referred to dental hospitals for pathological diagnosis and further management. Entire study population was educated about the harmful effects of the habit.

Analysis was done using SPSS software version 20. Results of categorical responses were presented in terms of frequencies and percentages. Odds ratio was calculated to find out the association between tobacco consumption and oral pre-malignant lesions.

RESULTS

A total of 909 workers from four industries were examined out of which 97% were males.

Out of 909 subjects, 537 (59.1%) had a history of consuming tobacco; out of which, 360 (39.6%) were consumers of smokeless tobacco (SLT) and 141 (15.5%) were smokers.

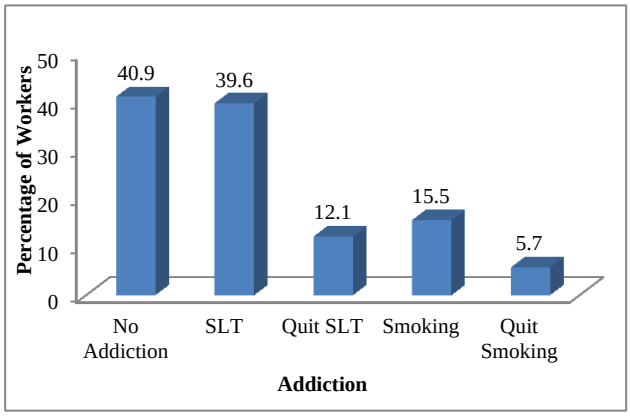

Figure 1: Tobacco consumption in industrial workers.

As suspected, workers having addiction of smokeless tobacco had highest proportion of tobacco quid lesion followed by OSMF. Tobacco quid lesions were seen even if the workers had quit the tobacco consumption. Whereas in smoke form of tobacco, smoker's palate was the most common lesion followed by leukoplakia and erythroplakia with no prevalence of quid lesions. Similar to smokeless form even in smoke form of tobacco, workers had smoker's palate even if they had quit tobacco consumption.

Amongst 909 workers, most common lesion seen was tobacco quid lesion (10.9%) followed by OSMF (9.8%). OSMF was mainly seen in the workers who were consuming areca nut along with other forms of tobacco.

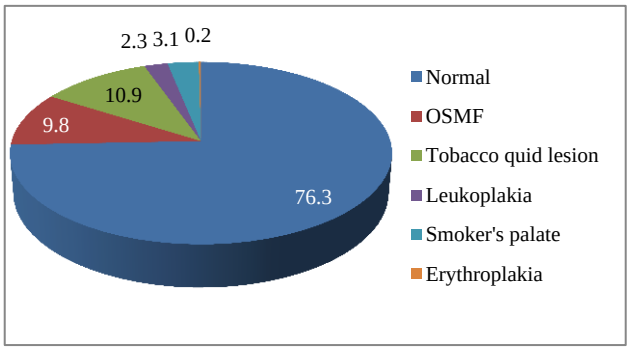

Figure 2: Prevalence of oral premalignant lesions.

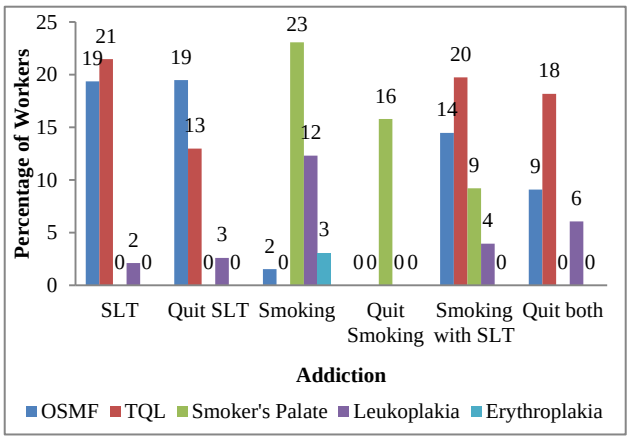

Figure 3: Prevalence of lesions associated with history of tobacco consumption.

Oral premalignant lesions were present in around 52% of the study population that was exposed to tobacco in the form of smoking or chewing; whereas a little over 2% of the subjects without exposure had a lesion. The odds of developing an oral premalignant lesion as a result of exposure to tobacco are 43.62 times more than the odds of acquiring a lesion without exposure. This result demonstrated the harmful effect of tobacco consumption in various forms on the prevalence of oral pre-malignant lesions.

The odds of developing an oral premalignant lesion after removing the exposure to tobacco for at least 6 months are still 18.4 times more than the odds of acquiring a lesion without exposure. From this result we can infer that even if workers stop consuming the tobacco, its harmful effect still remains very high as compared to those who have never had any tobacco consumption in the past.

Table 1: Association of tobacco consumption with oral pre-malignant lesions.

Exposure	Total	Lesions present	Lesions absent	Odds ratio
		N (%)	N (%)	
Tobacco consumers	416	217 (52.2)	199 (47.8)	43.62
No addiction	369	9 (2.4)	360 (97.6)	1

Table 2: Association of oral pre-malignant lesions in workers who had quit tobacco consumption.

Exposure	Total	Lesions present	Lesions absent	Odds ratio
		N (%)	N (%)	
Ex-tobacco consumers	124	39 (31.5)	85 (68.5)	18.39
No addiction	369	9 (2.4)	360 (97.6)	1

DISCUSSION

In our study we found association between the oral premalignant lesions and tobacco smoking and chewing. The oral mucosa is composed of different layers including stratified squamous epithelium, masticatory/ keratinized epithelium (hard palate, dorsum of the tongue, and keratinized gingival) and lining mucosa (floor of the mouth, ventrolateral surface of the tongue, soft palate complex, labial vestibule, and buccal mucosa). Tobacco use affects the surface epithelium, resulting in changes in the appearance of the tissues. The changes may range from an increase in pigmentation to thickening of the epithelium (white lesion). Tobacco use can also irritate the minor salivary glands on the hard palate and directly increase a person's risk for oral cancer as well as periodontal disease.

Out of total workers who had history of tobacco chewing 43% had oral lesions (out of which 19.4% had oral sub mucous fibrosis and 21.5% had tobacco quid lesion) as compared to just 1% of workers having oral lesion amongst those who had no addiction. This showed that there was a high prevalence of lesions in the tobacco chewers. Also out of total smokers 15.8% had smoker's palate whereas no similar lesion was seen in the workers having no addiction. This showed that smoker's palate had high prevalence in the smokers only.

Various studies have been carried out which shows correlation of tobacco with various oral lesions. In one such study by Pentenero et al, the tobacco association was linked to frictional lesions, leukoplakia, melanin pigmentation and smoker's palate.[11]

In different review articles, there were similar conclusions about higher prevalence of oral lesions in the tobacco smokers and chewers. These studies have stated that consumption of tobacco can result not only in a multitude of different general health problems like carcinoma of the lung, Ischemic cardiac diseases, peripheral vascular diseases, stroke, chronic-obstructive pulmonary diseases or peptic ulcers, but also in pathologic lesions of the oral mucosa. Tobacco-associated lesions like oral leukoplakia or oral squamous cell carcinoma are already potentially life-threatening diseases that in general require active treatment.[8,12-14]

In a study by Sujatha et al, 1028 patients with tobacco, alcohol and areca nut habits formed the study sample. The commonest habit in their study sample was smoking (39.2%) followed by smokeless tobacco use (28.1%). Also in their study they found leukoplakia as the most common lesion (14%) owing to higher prevalence of smoking addiction whereas in our study tobacco quid lesion was the commonest (10.9%) owing to higher smokeless tobacco consumption.[15]

In another study by Chandra et al a selection of 1525 patients visiting the Department of Oral Medicine and Radiology were interviewed regarding tobacco habits. 359 patients (23.5%) had the habit and 265 (73.8%) of them had oral mucosal lesions. About 75% of those who had habit of tobacco were having oral lesions which was higher as compared to our study where we found prevalence rate to be around 43%.[16]

In the study by Morger et al data on the oral health status of 615 Swiss Army recruits was collected using a standardized self-reported questionnaire, followed by an intraoral examination. They found that among young male adults a significant number of oral mucosal lesions could be identified, which strongly correlated with tobacco use.[17]

The condition of OSMF is well recognized for its malignant potential and is particularly associated with areca nut chewing, the main component of betel quid. The mixture of this quid or chew is a combination of the areca nut (fruit of the *Areca catechu* palm tree, erroneously termed betel nut) and betel leaf (from the *Piper betel*, a pepper shrub), tobacco, slaked lime (calcium hydroxide), and catechu (extract of the *Acacia catechu* tree) betel nut consumption is particularly seen in countries of Indian sub-continent which correlated with higher prevalence of OSMF in these countries.[18]

Various studies have tried to prove this association of areca nut with OSMF. In a study by Angadi et al carried

out at Belgaum, India a strong association with smokeless tobacco use especially areca-nut in the form of gutkha was established and was related to earlier development of OSMF, i.e., within a year of the habit.[19]

CONCLUSION

The tobacco consumption in any form is hazardous and causes various kinds of oral pre-malignant lesions. Betel nut exposure causes more harmful effect which goes unnoticed. Awareness should be created about various lesions caused by tobacco as well as betel nut and special steps should be taken for early diagnosis and its treatment.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. WHO report on the global tobacco epidemic 2013. Geneva: World Health Organisation; 2013.
2. Rani M, Bonu S, Jha P, Nguyen SN, Jamjoum L. Tobacco use in India: prevalence and predictors of smoking and chewing in a national cross sectional household survey. Tobacco Control. 2003;12(4):e4.
3. Thankappan KR, Thresia CU. Tobacco use & social status in Kerala. Indian J Med Res. 2007;126(4):300-8.
4. Ali AA, Al-Sharabi AK, Aguirre JM, Nahas R. A study of 342 oral keratotic white lesions induced by qat chewing among 2500 Yemeni. J Oral Pathol Med. 2004;33(6):368-72.
5. Reichart PA, Philipsen HP. Oral erythroplakia--a review. Oral Oncol. 2005;41(6):551-61.
6. Rajendran R, Vidyarani. Familial occurrence of oral submucous fibrosis: report of eight families from northern Kerala, south India. Indian J Dent Res. 2004;15(4):139-44.
7. Rooban T, Saraswathi TR, Al Zainab FH, Devi U, Eligabeth J, Ranganathan K. A light microscopic study of fibrosis involving muscle in oral submucous fibrosis. Indian J Dent Res. 2005;16(4):131-4.
8. Vellappally S, Fiala Z, Smejkalova J, Jacob V, Somanathan R. Smoking related systemic and oral diseases. Acta Medica. 2007;50(3):161-6.
9. Country profile: India. J Indian Med Assoc. 1999;97(9):377-8, 83.
10. Murray CJ, Lopez AD. Global mortality, disability, and the contribution of risk factors: Global Burden of Disease Study. Lancet. 1997;349(9063):1436-42.
11. Pentenero M, Broccoletti R, Carbone M, Conrotto D, Gandolfo S. The prevalence of oral mucosal lesions in adults from the Turin area. Oral Dis. 2008;14(4):356-66.
12. Taybos G. Oral changes associated with tobacco use. Am J Med Sci. 2003;326(4):179-82.
13. Bornstein MM, Klingler K, Saxer UP, Walter C, Ramseier CA. Tobacco-associated lesions of the oral mucosa. Schweiz Monatsschr Zahnmed. 2006;116(12):1261-74.
14. Schulz M, Reichart PA, Ramseier CA, Bornstein MM. Smokeless tobacco: a new risk factor for oral health? A review. Schweiz Monatsschr Zahnmed. 2009;119(11):1095-109.
15. Sujatha D, Hebbar PB, Pai A. Prevalence and correlation of oral lesions among tobacco smokers, tobacco chewers, areca nut and alcohol users. Asian Pacific J Cancer Prevent. 2012;13(4):1633-7.
16. Chandra P, Govindraju P. Prevalence of oral mucosal lesions among tobacco users. Oral Health Prevent Dent. 2012;10(2):149-53.
17. Morger R, Ramseier CA, Rees TD, Burgin WB, Bornstein MM. Oral mucosal findings related to tobacco use and alcohol consumption: a study on Swiss army recruits involving self-reported and clinical data. Oral Health Prevent Dent. 2010;8(2):143-51.
18. Ho PS, Yang YH, Shieh TY, Huang IY, Chen YK, Lin KN, et al. Consumption of areca quid, cigarettes, and alcohol related to the comorbidity of oral submucous fibrosis and oral cancer. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2007;104(5):647-52.
19. Angadi PV, Rekha KP. Oral submucous fibrosis: a clinicopathologic review of 205 cases in Indians. Oral Maxillofacial Surg. 2011;15(1):15-9.

Cite this article as: Joshi A, Jani C, Jain K, Bala DV, Jetpariya D, Jadav H. Association of oral pre-malignant lesions with the consumption of tobacco. Int J Community Med Public Health 2019;6:3625-9.