

Research Article

Patterns of Tobacco Usage and Oral Mucosal Lesions of Industrial Workers: A Cross Sectional Study

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Abstract

Tobacco use is associated with a variety of oral precancerous lesions and cancer. The lesions caused by smokeless tobacco can be reversed by quitting the habit at an earlier stage and providing appropriate treatment. Thus, proving the importance of early diagnosis in prevention of debilitating diseases caused by tobacco use. This study was conducted to assess the prevalence of oral mucosal lesions and tobacco consumption patterns among industrial workers of an Indian city. A descriptive cross-sectional study was conducted with 1500 industrial workers and ethical approval was obtained and informed consent was obtained from the subjects. Tobacco related habits and oral mucosal lesions were recorded using WHO pro forma for recording Oral Mucosal diseases. Tobacco usage was prevalent among 70.8% and prevalence of oral mucosal lesions was 30.8%. The odds ratio (OR) for smoking, gutkha chewing, ST (Smokeless Tobacco) and alcohol consumption was 1.08, 1.7, 5.2 and 1.6 respectively. After adjusting for confounding factors, the adjusted OR for smokeless tobacco usage (i.e. ST and gutkha) was 12.1. Smokeless tobacco usage was found to be the strongest risk factor for precancerous and cancerous lesions of oral cavity. It is recommended to conduct regular screening and health education program for the industrial workers.

Keywords: Smokeless tobacco; Industrial workers; Precancerous lesions; Gutkha

Introduction

Tobacco is one of the most important plant products, which gathers attention from all the health professionals, just because of the variety of diseases caused by its use. Tobacco use causes a wide range of major diseases which impact nearly every organ of the body. These include several types of cancers, heart diseases and lung diseases. It is estimated that 250 million children and adolescents who are alive today, would die prematurely because of tobacco, most of them in developing countries [1].

India currently being the second largest populated country, India's share of the global burden of tobacco-induced disease and death is substantial. India has another distinction; it is the third largest tobacco growing country approximately producing six lakhs tones of tobacco annually. According to the World Health Organization (WHO) estimates, in India, 194 million men and 45 million women use tobacco in smoked or smokeless forms [2]. The WHO also predicts that India will have the fastest rate of rise in deaths attributable to tobacco in the future years. India also has one of the highest rates of oral cancers in the world, partly attributed to high prevalence of tobacco chewing [3].

Tobacco use is basically of two forms – smoking and smokeless tobacco. In general, cigarettes account for only 20% of all tobacco consumed, while gutkha and chewing tobacco each account for about 40% of tobacco consumption [4]. In India, the prevalence of cigarette smoking is very less. On the other hand, over half of all tobacco consumed in India is smoked as bidis and about one-fourth of tobacco consumption is in smokeless form. Bidi smoking and

smokeless tobacco use continues to be practiced by a large percentage of the population in India. Smokeless tobacco use is twice as high as bidi smoking among adolescents. Initiation of use of these products among youth leads to lifelong adult use [5]. In many cultures, particularly in India, smokeless tobacco use is more socially acceptable than smoking [6], and it is usually easy to practice without detection. Tobacco manufacturers encourage the use of smokeless tobacco products by smokers on occasions when they are not permitted to smoke and thereby promote individuals to adopt smokeless tobacco use in conjunction with continued smoking.

Use of tobacco in the form of cigarettes, bidis and smokeless tobacco has been associated with oral mucosal lesions and some of these lesions may eventually become malignant. There is already evidence in India of an increased occurrence of oral sub mucous fibrosis [7] due to the habit of chewing betel quid, areca nut, pan masala and Gutkha, and it is likely to reach an alarming proportion in the near future. Occurrence of oral mucosal lesions at the site of smokeless tobacco placement [8] is reported and the preliminary evidence suggests that these lesions are associated with the duration and amount of smokeless tobacco use.

Studies have showed that use of chewing tobacco has been found to be associated with other less severe oral lesions [9]. Histologically, these smokeless tobacco lesions are characterized by hyperkeratinisation of epithelium, acanthosis and proliferation of inflammatory cells.

Tobacco use is more common among males when compared with females [10,11]. Youth are especially vulnerable to initiating tobacco

use. In many cultures, particularly in India, smokeless tobacco use is more socially acceptable than smoking [12]. Anecdotal evidence suggests that the age of initiation of tobacco use is declining, with reports of children beginning to use tobacco as early as the age of 10 [13]. Evidence suggests that tobacco use is more common among the people from lower socio-economic class. Thus, the high-risk group of tobacco usage is mainly the young adults of low socio-economic status [14].

There is a clear benefit to quitting tobacco use because the risks of oral cancer decline with increasing time after tobacco cessation and some oral mucosal lesions may resolve with cessation of smokeless tobacco use [15]. The lesions caused by smokeless tobacco can be reversed by quitting the habit at an earlier stage and by availing appropriate treatment [16]. Thus, it proves the importance of identifying the high risk groups and educating them about ill-effects of tobacco, along with early diagnosis and prevention of debilitating diseases caused by tobacco use.

Most of the people working in the industries belong to lower socio-economic and have low literacy rate. The industrial workers thus form the high-risk group in whom, it was observed to have increased prevalence of tobacco related habits.

However, there are no studies exploring the prevalence of tobacco related habits and oral mucosal lesions among this group. Hence, this study was done to assess the prevalence of oral mucosal lesions and tobacco consumption patterns among the industrial workers.

Materials and Methods

The present study conducted using a cross-sectional design to study the prevalence of oral mucosal lesions among industrial workers of Belgaum city, Karnataka. Approval from the ethical committee and the institutional review board. Informed consent was obtained from all subjects who participated in the study.

Single examiner was involved in the collection of data; hence intra-examiner calibration was done. Twenty-five patients coming to outpatient department were examined and WHO proforma were recorded. The subjects with oral mucosal lesions were examined by the specialist (Oral Diagnosis) who confirmed the diagnosis given by the examiner.

A pilot study was conducted to determine the final sample size which was 1500. Industries were stratified as small, medium and large scale based on production parameters. Equal study subjects (500) were taken from each of stratified industries. This was a quota based sampling. Thus, in small scale 18 industries were covered to include 500 subjects and 7 industries were covered in medium scale strata and 2 industries from large scale strata using random number (Tables 1-5).

WHO assessment form for oral mucosal diseases was used to record the findings in this study. The study was conducted over a period of 6 months.

Permissions were obtained from the authorities like Director of Belgaum Industries before the start of the study. After the selection of industries, managers of each industry were explained, about the objectives of the study and date at which examination was done were fixed. Thirty subjects were examined per day. WHO pro forma for

Table 1: Distribution of subjects according to SES and precancerous lesion.

S.no	SES	Precancerous lesions		Chi square(df)	p-value
		Yes n(%)	No n(%)		
1.	Upper middle	4(1.5)	4(0.3)	5.65(2)	0.06
2.	Lower middle	235(88)	1094(88.9)		
3.	Upper lower	28(10.5)	135(10.8)		
	Total	267(100)	1233(100)		

Table 2: Distribution of subjects according to type of tobacco related habits and precancerous lesions.

S.no	Type of tobacco habit	Precancerous lesions		Chi-square(df)	P-value
		Yes n(%)	No n(%)		
1.	Smoking only	0	111(9)	196.9(7)	<0.001*
2.	Gutka only	65(24.3)	320(26)		
3.	ST only	81(30.3)	134(10.9)		
4.	Smoking and gutkha	9(3.4)	41(3.3)		
5.	Smoking and ST	14(5.2)	13(1.1)		
6.	ST and Gutka	59(22.1)	157(12.7)		
7.	Smoking, gutka and ST	23(8.6)	34(2.8)		
8.	None	16(6)	423(34.2)		
	Total	267(100)	1233(100)		

*-Statistically significant

recording oral mucosal diseases was used to record the findings. The demographic details and tobacco usage status was recorded by interviewing the subjects. The patient was positioned by facing the window for good illumination and Type III clinical examination was done. 2x2 inch gauze pieces were used to dry the mucosa and two plane mouth mirrors were used to observe the oral mucosa.

Data thus collected was entered in computer using Ms Office Excel windows 2007 and analyzed using SPSS version 17 (Chicago, IL) statistical software package.

The results for continuous variable were given as numbers, standard deviations, mean values (Quantitative data) and proportions (Qualitative data) as percentages. Differences between the groups were assessed by the Chi-square test. Student t-test was used to compare the means of lifetime exposure of tobacco habits between regular and occasional smokers. Multiple logistic regressions were used to remove the effects of confounding variable in assessing the risk of tobacco and alcohol habits in precancerous lesions.

Results

The present study was conducted using a cross sectional design using a WHO pro forma for recording oral mucosal lesions and tobacco related habits among the industrial workers of Belgaum city.

A total of 1500 workers who met the inclusion and exclusion criteria were selected for the study. The mean age of the study population was 32.14+9.86 years, ranged from 18-59 years. Out of the 267 subjects with precancerous lesions, 4(1.5%) belonged to the upper middle class, 235(88%) belonged to the lower middle class and 28(10.5%) belonged to the upper lower class. Of the 1233 subjects without precancerous lesions, 4 (0.3%) belonged to the upper middle class, 1094(88.9%) belonged to the lower middle class and 135(10.8%)

Table 3: Comparison of mean lifetime exposure of different forms of tobacco between subjects with and without precancerous lesions.

S.no	Tobacco related habits	Precancerous lesion	Mean	S.D	t-value	p-value
1.	ST lifetime exposure(packets)	No	4465.41	6134.10	-2.885	0.001*
		Yes	7170.91	11847.68		
2.	Gutkha lifetime exposure(packets)	No	6094.48	9636.85	169.356	<0.001*
		Yes	14660.83	23993.17		
3.	Cigarette/bidi lifetime exposure(numbers)	No	2902.59	6341.72	-.806	0.421*
		Yes	3715.06	5310.91		

*-Statistically significant

Table 4: Distribution of subjects according to the tobacco related habits and lesions.

Groups	Cancer n(%)	Leukoplakia n(%)	Erythroplakia n(%)	OSMF n(%)	Nicotina palatine n(%)
Smoking only	0	0	0	0	24(34.2)
Gutka only	0	18(13.3)	0	54(51.4)	0
ST only	0	69(51.1)	7(46.6)	12(11.4)	4(5.7)
Smoking and gutkha	0	0	0	9(8.5)	6(8.5)
Smoking and ST	0	10(7.4)	0	0	13(18.5)
ST and Gutka	0	28(20.7)	8(53.4)	21(20)	10(14.2)
Smoking,gutka and ST	4(100)	10(7.4)	0	9(8.5)	13(18.5)
None	0	0	0	0	0
Total	4(100)	135(100)	15	105(100)	70(100)
Chi-square (df)	101.5(7)	243.2 (7)	37.7(7)	91.80(7)	278.8 (7)
p-value	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*

*-Statistically significant

Table 5: Association of tobacco related habits and precancerous lesions using logistic regression model.

S.no	Habits	Precancerous lesions		p-value	Odds Ratio (CI)	Adjusted odds ratio (CI)	
		Yes	No				
1.	Smoking (n=245)	Yes	46	199	0.36	1.08(0.7-1.5)	1.5(0.7-1.5)
		No	221	1034			
2.	ST usage(n=515)	Yes	177	338	<0.001*	5.2(3.2-6.9)	5.02(3.7-6.6)
		No	90	895			
3.	Gutkha usage(n=707)	Yes	156	551	<0.001*	1.7(1.3-2.2)	1.5(1.1-2.07)
		No	111	681			
4.	Smokeless tobacco Both gutkha and ST(n=950)	Yes	251	699	<0.001	11.9(7.1-20.1)	12.1(7.2-20.5)
		No	16	534			
5.	Alcohol usage(n=133)	Yes	34	99	0.01*	1.67(1.1-2.5)	1.1(0.7-1.7)
		No	233	1134			

belonged to the upper lower class. The association between SES and precancerous lesions was not statistically significant $p=0.06$.

In the study population, 245(16.3%) were smokers, 515(34.3%) were ST users, 707(47.1%) were gutkha users and 133(8.8%) were alcohol consumers. Out of the 245 smokers, 38(15.6%) were occasional smokers i.e.; did not smoke in the last 30 days before the examination and 207(84.4%) were regular smokers. Out of the 515 ST users, 119(23.1%) were occasional users and 396(76.8%) were regular users. Out of the 707 gutkha chewers, 255(36.1%) were occasional users and 452(63.9%) were regular users. Out of the 133 alcohol consumers, 106(79.6%) were occasional users and 27(20.4%) were regular users.

The mean lifetime exposure was calculated by frequency (in days)* duration (in years) *365. Mean lifetime exposure of ST in subjects without precancerous lesions was 4465.1(+6134.1) and with precancerous lesions were 7170.91(+11847.6). The mean lifetime exposure of ST was found statistically significant $p=0.001$ Mean lifetime exposure of Gutkha in subjects without precancerous lesions was 6094.48(+9636.8) and with precancerous lesions were 14660.83(+23993.1). The mean lifetime exposure of gutkha was found statistically significant $p<0.001$. Mean lifetime exposure of cigarette/bidi in subjects without precancerous lesions was 2902.59(+6341.7) and with precancerous lesions were 3715.06(+5310.9). The mean lifetime exposure of cigarette/bidis was not statistically significant $p=0.421$.

Among the total study subjects, 457(30.39%) had oral mucosal lesions. Cancer was seen in 4(0.26%) subjects, aphthous ulcers were seen in 24(1.6%) subjects, leukoplakia was seen in 135(9%). Fifteen (1%) subjects had erythroplakia, 48(3.2%) had lichen planus, 105(7%) had OSMF and 42(2.8%) had candidiasis. Herpetic gingivitis was seen in 6 (0.4%) subjects, nicotina palatine was seen in 70(4.6%) and cancrum oris was seen in 8(0.53%) subjects.

Out of the four cancer cases all were seen in subjects consuming all three habits cigarettes/bidis, gutkha and ST. There was a statistically significant association between type of tobacco habits and cancer $p < 0.001$. Out of the 135 subjects who had leukoplakia, 18(13.3%) belonged to gutkha only group, 69(51.1%) belonged to the ST only group and 10(7.4%) belonged to the group of subjects consuming both cigarettes/bidis and ST and 28(20.7%) belonged to the subjects consuming ST and gutkha and 10(7.4%) belonged to the subjects consuming cigarettes/bidis, gutkha and ST. There was a statistically significant association between type of tobacco habits and leukoplakia $p < 0.001$. Out of the 15 subjects who had erythroplakia, 7(46.6%) consumed only ST and 8(53.4%) consumed ST and gutkha. There was a statistically significant association between type of tobacco habits and erythroplakia $p < 0.001$. Out of the 105 subjects who had OSMF, 54(51.4%) consumed only gutkha, 12(11.4%) consumed only ST, 9(8.5%) consumed gutkha and cigarettes/bidis, 21(20%) consumed ST and gutkha and 9 (8.5%) consumed cigarettes/bidis, gutkha and ST. There was a statistically significant association between type of tobacco habits and OSMF $p < 0.001$. Out of the 70 subjects who had nicotina palatini, 24(34.2%) consumed only cigarettes/bidis, 4(5.7%) consumed only ST, 6(8.5%) consumed gutkha and cigarettes/bidis, 13(18.5%) consumed ST and cigarettes/bidis, 10(14.2%) consumed ST and gutkha and 13 (18.5%) consumed cigarettes/bidis, gutkha and ST. There was a statistically significant association between type of tobacco habits and nicotina palatini $p < 0.001$.

Among the smokers, 18.2% had precancerous lesions, among the ST/Quid users 34/3% had precancerous lesions and among the gutkha users 22% had precancerous lesions. Among the subjects who only smoked, none of them had precancerous lesions. Among the subjects who only consumed gutkha, 65(24.3%) subjects had precancerous lesions, in the subjects consuming only ST/Quid 81(30.3%) subjects had precancerous lesions. Among the subjects consuming both smoking and gutkha 9(3.4%) had precancerous lesions and among the subjects consuming both smoking and ST 14(5.2%) had precancerous lesions. In subjects, consuming ST and gutkha 59(22.1%) had precancerous lesions and in subjects consuming smoking, gutkha and ST 23(8.6%) had precancerous lesions. There was a statistical significant association between different type of tobacco habits and precancerous lesions $p < 0.001$.

Among the smokers, 46 subjects had precancerous lesions, 199 did not have precancerous lesions, and among the non-smokers, 221 had precancerous lesions and 1034 did not have precancerous lesions. The association between smoking and precancerous lesions was not statistically significant and the odds ratio was 1.08 (0.7-1.5). When the other factors like ST usage, gutkha usage and alcohol usage were adjusted using logistic regression model, the adjusted odds ratio was found to be 1.5(0.7-1.5), but this was statistically not significant.

Among the 515 ST users, 177 subjects had precancerous lesions,

338 did not have precancerous lesions, and among the non-ST users, 90 had precancerous lesions and 895 did not have precancerous lesions. The association between ST usage and precancerous lesions was statistically significant and the odds ratio was 5.2(3.2-6.9). When the other confounding factors like smoking, gutkha usage and alcohol usage were adjusted using logistic regression model, the adjusted odds ratio was found to be 5.02(3.7-6.6), which was statistically significant.

Among the 707-gutkha users, 156 subjects had precancerous lesions, 552 did not have precancerous lesions, and among the non-gutkha users, 111 had precancerous lesions and 681 did not have precancerous lesions. The association between gutkha usage and precancerous lesions was statistically significant and the odds ratio was 1.7(1.3-2.2). When the other confounding factors like smoking, ST usage and alcohol usage were adjusted using logistic regression model, the adjusted odds ratio was found to be 1.5(1.1-2.07), which was statistically significant.

Among the 950 smokeless tobacco users i.e. ST and gutkha users, 251 subjects had precancerous lesions, 699 did not have precancerous lesions, and among the non smokeless tobacco users, 16 had precancerous lesions and 534 did not have precancerous lesions. The association between smokeless tobacco usage and precancerous lesions was statistically significant and the odds ratio was 11.9(7.1-20.1). When the other confounding factors like smoking and alcohol usage were adjusted using logistic regression model, the adjusted odds ratio was found to be 12.1(7.2-20.5), which was statistically significant.

Among the 133 alcohol consumers, 34 subjects had precancerous lesions, 99 did not have precancerous lesions, and among the non-alcohol consumers, 233 had precancerous lesions and 1134 did not have precancerous lesions. The association between alcohol usage and precancerous lesions was statistically significant and the odds ratio was 1.67(1.1-2.5). When the other confounding factors like smoking, gutkha usage and ST usage were adjusted using logistic regression model, the adjusted odds ratio was found to be 1.1(0.7-1.7), which was not statistically significant.

Discussion

The present study included subjects from the various stratum of industries i.e., small-scale, medium scale and large-scale industries. Only males were considered for this study. As this study population was derived from different type of industries of the city, it can be claimed to be representative of all the industrial workers of the city. The mean age of the study group was 32.14+9.86 years, ranging from 18-59 years, entailing the majority was of middle-aged subjects.

The study population included all types of occupational categories. The majority were skilled workers representing the nature of industries and its requirements of labor. As this study population was obtained from foundries where it requires various skills like melting of metals, molding, drilling, bracing of metals etc.

The common habit among the study group was gutkha chewing (47.1%) especially among the younger age group 18- 25 years. This shows the penchant of younger population towards gutkha chewing. The ST/quid prevalence (34.3%) was preferred next to gutkha chewing and this was mostly practiced by middle-aged population

(25-34 years). This could be due to the fact that young people start up with gutkha as experiment and continue with ST/quid. ST/quid is comparatively cheaper to gutkha usage and it can be kept in the mouth for a longer time than gutkha, this could explain the preference of ST in the later ages. This was found similar to the study conducted by Gupta PC et al. [17] and also concurred with the finding from the national family health survey 2005-2006 [18], where gutkha usage was more in younger group and increased prevalence. Smoking prevalence (16.3%) was least among the tobacco habits practiced by the subjects and mainly because of the cost and restriction to use of smoking habits in the industrial premises. Thus, ST or gutkha is preferred as this can be practiced without detection.

The prevalence of precancerous lesions was high in the age group of 25-34 years (31.1%) followed by 18-24 years (23.1%). This was in accordance with higher prevalence of tobacco usage among these age groups. Similar finding was reported by Mehrotra et al. [19] clearly demonstrating the increased risk of tobacco habits in the causation of precancerous lesions.

Majority of the gutkha chewers (26.6%) had received intermediate/PUC education and it was similar to the subjects, who received high or middle school education. Similar trend was seen in ST users and smokers, majority of subjects had received intermediate/PUC education and next majority belonged to the subjects who received high or middle school education. The association between educational status and usage of tobacco was found statistically significant in this study. This was in line with studies conducted by Hashibe et al., [20] in Kerala, India and Amarasinghe et al., [21] in Sri Lanka. There is general opinion that, people with higher educational qualification tend to avoid tobacco as they have better knowledge about ill-effects about tobacco, as found in the studies [20,21].

Majority of gutkha chewers, ST users and smokers belonged to lower middle class. Similarly higher prevalence of precancerous lesions (88%) was also present in lower middle class as the prevalence of habits was high. This was found in accordance with studies conducted by Ahmad et al., [22] and Sinor PN et al., [23] in India, Shiau et al., [24] in Taiwan and Ramanathan et al., [25] in Malaysia. There is general perception among the subjects, that tobacco chewing decreases the appetite and makes them alert. This could be attributed to the nicotine content of the tobacco. The reason for high prevalence of precancerous lesions among lower socio-economic class is due to poor quality of food i.e. food deficient in vitamins and other nutrients, coupled with poor health consciousness.

In this present study 1062 (70.8%) subjects consumed tobacco in any form and 438(29.2%) were free from tobacco habits. The subjects were classified as never users i.e. has not used tobacco in the past one year, occasional users i.e. has not used tobacco in the past one month from the date of examination and regular users i.e. are using tobacco currently. The prevalence of tobacco in this study group is similar to the trend seen in other states [26-29] of India, where the prevalence ranges as low 15% in Goa to as high as 67% in Andhra Pradesh. The prevalence of smoking in this study group was 16.3%, which was lesser compared to the general population [30] (22.3%), as more subjects preferred smokeless tobacco than cigarettes/bidis because of the cost.

The prevalence of alcohol usage (8.8%) was low compared to the general population (18.8%) as shown by the study conducted in Mumbai [31] this could be explained by the fact that alcohol consumption is more among the elderly aged group like above 45 years and in this study majority fell in the younger age group of 18-34 years. Other reason could be self-reported data that may not be reliable, as observed among the subjects their reluctance to reveal about drinking habits compared to tobacco habits.

The prevalence of precancerous lesions was higher in subjects with higher lifetime exposure of ST and Gutkha, implying higher risk for precancerous lesion for a longer time exposure to these habits. This result is in agreement with study conducted by Renganathan et al., [32] in India.

The overall prevalence of oral mucosal lesions in the present study was 30.39%, this is way higher compared to the previous study conducted by Prasad et al. [33,34] in Karnataka in 2004 where it was 7.53%. This major difference is mainly because, in the study conducted by Prasad et al included general population comprising of children and women. Leukoplakia (9%) was the most prevalent lesion followed by OSMF (7%) and nicotina palatine (4.6%). This was predictable as more than seventy percent were using tobacco in any form.

Cancer was seen in subjects using all forms of tobacco (i.e. smoking, ST and Gutkha). Highest percentage of leukoplakia (51.1%) was seen in subjects using only ST as it is retained in the mucosa for a longer time than other forms of tobacco and its absorption is more by the mucous membrane, thus illustrate the dose-response relationship in the causation of lesions [35].

Erythroplakia is a rare oral precancerous lesion in the oral cavity and in this present study also it was seen in just 1% of the subjects. Erythroplakia was seen more in subjects consuming both ST and gutkha, showing that both in combination increase the risk of disease. This finding was in accordance with the studies conducted by Hashibe et al., [36] in Kerala and Chung et al., [37] in Taiwan. OSMF (7%) was the common lesion next to leukoplakia among the study group. OSMF was prevalent in over 50% of only Gutkha chewers followed by subjects consuming both ST and gutkha, as shown in previous studies [38-40]. The gutkha chewing releases an alkaloid arecaidine from the areca nut, which is proved the major contributor in etiology of OSMF. The other factors increases the risk is nutritional deficiency and genetic predisposition.

The prevalence of precancerous lesions was higher in the subjects consuming ST only followed by gutkha only and its combination. This demonstrates the increased risk of precancerous lesions by smokeless form tobacco. This was also found statistically significant $p < 0.001$, as shown in various other studies [39-41].

When the risk of the tobacco related habits with presence or absence of precancerous lesions were assessed using odds ratio and exact risk for individual risk factor by removing the effects of other components was assessed using Logistic regression to yield adjusted odds ratio. Smoking showed an increased risk of 1.08 times for the occurrence of precancerous lesions. Smoking was not found to be an independent risk in this study and this finding concurred with study conducted by Sankaranarayanan et al., in Kerala [29]. ST usage alone has shown to increase the risk of precancerous lesions by 5.2 times,

when adjusted of effects of smoking and alcohol the risk decreased to 5.02 times, but still its contribution is higher. This finding was in accordance with study by Ariyawardane et al., [42], where quid chewing increased the risk by 3 times. This could be attributed to the fact that ST/quid is placed in the mouth rather than chewing, which increases the time tobacco is in contact with the oral mucosa. Gutkha usage alone increases the risk of precancerous lesions by only 1.7 times and when adjusted for smoking, alcohol and ST usage it decreased to 1.5 times. Alcohol usage increases risk by 1.6 times, when adjusted for effects of other factors the risk decreased to 1.1 times and was not significant. This finding was similar to various other studies. This study also shows that alcohol increases the risk of precancerous lesions, but its role is only synergistic with presence of tobacco factors. The other reason could be under-reporting of alcohol habit by subjects as alcohol is considered to be a social evil in Indian society [29]. Thus, the effect of alcohol may be stronger than that observed.

Our study confirmed the fact that tobacco chewing is the strongest risk for cancer and precancerous lesions. This was shown as smokeless tobacco usage (i.e. gutkha and ST/quid usage) increased the risk by 11.9 times and when adjusted for smoking and alcohol the risk increased to 12.1 times. This was consistent to the previous findings [43-49].

The limitation of any cross-sectional study emphasizes the need to undertake a cautious interpretation of results, because the study measures the cause and effect at the same point in time, its design introduces the problem of temporal ambiguity and the inability to establish causal relationships.

Detection bias is a possibility, knowing the exposure status of subjects looking harder for lesions in those subjects. As the self-reported tobacco and alcohol usage status was not verified using other means like urinary cotinine test and EtG (Ethyl Glucuronide) test [50], there is a possibility of reporting bias, although self-reports are reliable and commonly used in epidemiological research [51].

Conclusion

This present study showed that chewing tobacco is the strongest risk factor for precancerous and cancerous lesions of the oral cavity. Our study also revealed that high proportion of young adults are using tobacco and have oral precancerous lesions. These people forms the high-risk group, where obvious implication of preventive measures, like curbing tobacco and alcohol use through public education is required. This study forms the baseline information for planning preventive programs for these high-risk groups.

References

- World Health Organization. Tobacco Free Initiative: Why is tobacco a public health priority? Geneva: World Health Organization; 2006.
- Reddy KS, Gupta PC. Report on tobacco control in India. New Delhi, India: Ministry of Health and Family Welfare, Government of India. 2004.
- Consolidated Report of Population based cancer registries Consolidated Report - (1990 -19 96). National cancer registry programme, Indian council of medical research, New Delhi. 2001.
- World Health Organization Tobacco or health: a global status report, country profiles by region. Geneva: World Health Organization. 1997.
- Ray CS, Gupta PC. Bidis and smokeless tobacco. *Current Science*. 2009; 96: 1324-1334.
- Gupta PC, Ray SR. Tobacco and youth in the South East Asia Region. *Indian Journal of Cancer*. 2002; 39: 3-34.
- Gupta PC, Ray CS. Smokeless tobacco and health in India and South Asia. *Respirology*. 2003; 8: 419-431.
- Greer RO Jr. Oral manifestations of smokeless tobacco use. *Otolaryngol Clin North Am*. 2011; 44: 31-56, v.
- Holmstrup P, Pindborg JJ. Oral mucosal lesions in smokeless tobacco users. *CA Cancer J Clin*. 1988; 38: 230-235.
- Gupta PC, Sinor PN, Bhonsle RB, Pawar VS, Mehta HC. Oral submucous fibrosis in India: a new epidemic? *Natl Med J India*. 1998; 11: 113-116.
- Gupta PC, Hebert JR, Bhonsle RB, Sinor PN, Mehta H, Mehta FS. Dietary factors in oral leukoplakia and submucous fibrosis in a population-based case control study in Gujarat, India. *Oral Dis*. 1998; 4: 200-206.
- Mackay J, Eriksen M. *The Tobacco Atlas*. Geneva, World Health Organization. 2002.
- Patel D R. Smoking and children. *Indian J Pediatr*. 1999; 66: 817-824.
- Gilman SE, Abrams DB, Buka SL. Socioeconomic status over the life course and stages of cigarette use: initiation, regular use, and cessation. *J Epidemiol Community Health*. 2003; 57: 802-808.
- Winn DM. Tobacco use and oral disease. *J Dent Educ*. 2001; 65: 306-312.
- Ebbert JO, Rowland LC, Montori V, Vickers KS, Erwin PC, Dale LC, et al. Interventions for smokeless tobacco use cessation. *Cochrane Database Syst Rev*. 2004; 3: CD004306.
- Gupta PC, Ray CS. Epidemiology of betel quid usage. *Ann Acad Med Singapore*. 2004; 33: 31-36.
- Rooban T, Elizabeth J, Umadevi KR, Ranganathan K. Sociodemographic correlates of male chewable smokeless tobacco users in India: a preliminary report of analysis of National Family Health Survey, 2005-2006. *Indian J Cancer*. 2010; 47: 91-100.
- Mehrotra R, Thomas S, Nair P, Pandya S, Singh M, Nigam NS, et al. Prevalence of oral soft tissue lesions in Vidisha. *BMC Res Notes*. 2010; 3: 23.
- Hashibe M, Sankaranarayanan R, Thomas G, Kuruvilla B, Mathew B, Somanathan T, et al. Body mass index, tobacco chewing, alcohol drinking and the risk of oral submucous fibrosis in Kerala, India. *Cancer Causes Control*. 2002; 13: 55-64.
- Amarasinghe HK, Usgodaarachchi US, Johnson NW, Lalloo R, Warnakulasuriya S. Betel-quid chewing with or without tobacco is a major risk factor for oral potentially malignant disorders in Sri Lanka: a case-control study. *Oral Oncol*. 2010; 46: 297-301.
- Ahmad MS, Ali SA, Ali AS, Chaubey KK. Epidemiological and etiological study of oral submucous fibrosis among gutkha chewers of Patna, Bihar, India. *J Indian Soc Pedod Prev Dent*. 2006; 24: 84-89.
- Sinor PN, Gupta PC, Murti PR, Bhonsle RB, Daftary DK, Mehta FS, et al. A case-control study of oral submucous fibrosis with special reference to the etiologic role of areca nut. *J Oral Pathol Med*. 1990; 19: 94-98.
- Shiau YY, Kwan HW. Submucous fibrosis in Taiwan. *Oral Surg Oral Med Oral Pathol*. 1979; 47: 453-457.
- Ramanathan K. Oral submucous fibrosis--an alternative hypothesis as to its causes. *Med J Malaysia*. 1981; 36: 243-245.
- Mehta FS, Pindborg JJ, Gupta PC, Daftary DK. Epidemiologic and histologic study of oral cancer and leukoplakia among 50,915 villagers in India. *Cancer*. 1969; 24: 832-849.
- Mehta FS, Gupta PC, Daftary DK, Pindborg JJ, Choksi SK. An epidemiologic study of oral cancer and precancerous conditions among 101,761 villagers in Maharashtra, India. *Int J Cancer*. 1972; 10: 134-141.
- Bhonsle RB, Murti PR, Gupta PC, Mehta FS. Reverse dhumti smoking in Goa: an epidemiologic study of 5449 villagers for oral precancerous lesions. *Indian J Cancer*. 1976; 13: 301-305.

29. Sankaranarayanan R, Mathew B, Jacob BJ, Thomas G, Somanathan T, Pisani P, et al. Early findings from a community-based, cluster-randomized, controlled oral cancer screening trial in Kerala, India. The Trivandrum Oral Cancer Screening Study Group. *Cancer*. 2000; 88: 664-673.
30. National Oral Health Survey and Fluoride Mapping. An Epidemiological Study of Oral Health Problems and Estimation of Fluoride Levels in Drinking Water. Dental Council of India, New Delhi. 2004.
31. Gupta PC, Saxena S, Pednekar MS, Maulik PK. Alcohol consumption among middle-aged and elderly men: a community study from western India. *Alcohol*. 2003; 38: 327-331.
32. Ranganathan K, Devi MU, Joshua E, Kirankumar K, Saraswathi TR. Oral submucous fibrosis: a case-control study in Chennai, South India. *J Oral Pathol Med*. 2004; 33: 274-277.
33. Prasad KVV, Javali SR, Rajesh G, Jithendra A. An epidemiological study of oral mucosal lesions in Karnataka state, India. *JIAOMR*. 2004; 16: 9-18.
34. Hashibe M, Mathew B, Kuruvilla B, Thomas G, Sankaranarayanan R, Parkin DM, et al. Chewing tobacco, alcohol, and the risk of erythroplakia. *Cancer Epidemiol Biomarkers Prev*. 2000; 9: 639-645.
35. Chung CH, Yang YH, Wang TY, Shieh TY, Warnakulasuriya S. Oral precancerous disorders associated with areca quid chewing, smoking, and alcohol drinking in southern Taiwan. *J Oral Pathol Med*. 2005; 34: 460-466.
36. Shah N, Sharma PP. Role of chewing and smoking habits in the etiology of oral submucous fibrosis (OSF): a case-control study. *J Oral Pathol Med*. 1998; 27: 475-479.
37. Hashibe M, Sankaranarayanan R, Thomas G, Kuruvilla B, Mathew B, Somanathan T, et al. Body mass index, tobacco chewing, alcohol drinking and the risk of oral submucous fibrosis in Kerala, India. *Cancer Causes Control*. 2002; 13: 55-64.
38. Lee CH, Ko YC, Huang HL, Chao YY, Tsai CC, Shieh TY, et al. The precancer risk of betel quid chewing, tobacco use and alcohol consumption in oral leukoplakia and oral submucous fibrosis in southern Taiwan. *Br J Cancer*. 2003; 88: 366-372.
39. Yang YH, Lien YC, Ho PS, Chen CH, Chang JS, Cheng TC, et al. The effects of chewing areca/betel quid with and without cigarette smoking on oral submucous fibrosis and oral mucosal lesions. *Oral Dis*. 2005; 11: 88-94.
40. Scheifele C, Nassar A, Reichart PA. Prevalence of oral cancer and potentially malignant lesions among shammah users in Yemen. *Oral Oncol*. 2007; 43: 42-50.
41. Amarasinghe HK, Usgodaarachchi US, Johnson NW, Lalloo R, Warnakulasuriya S. Betel-quid chewing with or without tobacco is a major risk factor for oral potentially malignant disorders in Sri Lanka: a case-control study. *Oral Oncol*. 2010; 46: 297-301.
42. Ariyawardana A, Athukorala AD, Arulanandam A. Effect of betel chewing, tobacco smoking and alcohol consumption on oral submucous fibrosis: a case-control study in Sri Lanka. *J Oral Pathol Med*. 2006; 35: 197-201.
43. Balaram P, Sridhar H, Rajkumar T, Vaccarella S, Herrero R, Nandakumar A, et al. Oral cancer in southern India: the influence of smoking, drinking, paan-chewing and oral hygiene. *Int J Cancer*. 2002; 98: 440-445.
44. Znaor A, Brennan P, Gajalakshmi V, Mathew A, Shanta V, Varghese C, et al. Independent and combined effects of tobacco smoking, chewing and alcohol drinking on the risk of oral, pharyngeal and esophageal cancers in Indian men. *Int J Cancer*. 2003; 105: 681-686.
45. Sankaranarayanan R, Duffy SW, Padmakumary G, Day NE, Padmanabhan TK. Tobacco chewing, alcohol and nasal snuff in cancer of the gingiva in Kerala, India. *Br J Cancer*. 1989; 60: 638-643.
46. Sankaranarayanan R, Duffy SW, Padmakumary G, Day NE, Krishan Nair M. Risk factors for cancer of the buccal and labial mucosa in Kerala, southern India. *J Epidemiol Community Health*. 1990; 44: 286-292.
47. Sankaranarayanan R, Duffy SW, Day NE, Nair MK, Padmakumary G. A case-control investigation of cancer of the oral tongue and the floor of the mouth in southern India. *Int J Cancer*. 1989; 44: 617-621.
48. Nandakumar A, Thimmasetty KT, Sreeramareddy NM, Venugopal TC, Rajanna, Vinutha AT, et al. A population-based case-control investigation on cancers of the oral cavity in Bangalore, India. *Br J Cancer*. 1990; 62: 847-851.
49. Jayant K, Balakrishnan V, Sanghvi LD, Jussawalla DJ. Quantification of the role of smoking and chewing tobacco in oral, pharyngeal, and oesophageal cancers. *Br J Cancer*. 1977; 35: 232-235.
50. Kharbouche H, Faouzi M, Sanchez N, Daeppen JB, Augsburger M, Mangin P, et al. Diagnostic performance of ethyl glucuronide in hair for the investigation of alcohol drinking behavior: a comparison with traditional biomarkers. *Int J Legal Med*. 2012; 126: 243-250.
51. Pradeepkumar AS, Mohan S, Gopalakrishnan P, Sarma PS, Thankappan KR, Nichter M. Tobacco use in Kerala: findings from three recent studies. *Natl Med J India*. 2005; 18: 148-153.