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Study of Major Risk Factors Associated With Oral Cancer In Odisha, India

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ABSTRACT:
Oral squamous cell carcinoma (OSCC) is the sixth most common malignancy and is a major cause for morbidity and mortality in developing nations. Despite advances in surgery, radiotherapy and chemotherapy over the past three decades, no significant improvement in the prognosis for OSCC has been observed. Tobacco smoking and alcohol consumption are the principal etiological factors associated with oral cancer, which if controlled could help avoid many tumors. This study was done to find the burden of principal risk factors in the development of oral cancer in Odisha.

KEY WORDS: Oral squamous cell carcinoma, smoking, alcohol

I. INTRODUCTION

Cancer has a multifactorial aetiology and is a multistep process involving initiation, promotion and tumor progression. It arises by a complex process involving a series of genetic alterations which leads to cellular proliferation and differentiation[1].

Spectrums of epithelial alterations range from hyperplasia, atypical hyperplasia, dysplasia, carcinoma in situ to invasive carcinoma. As the degree of dysplasia increases from mild to severe, so does the risk of malignant transformation[2].

Among the various etiologic factors, the important role of smoking and alcohol in oral malignancy has drawn increasingly interest[3].

OSCC is most likely caused by a combination of extrinsic and intrinsic factors acting in concert over a (long) period of time.

Indications exist demonstrating that there is atleast a contributing component related to a genetic susceptibility to the individual exposed to carcinogens and a potential for malignant transformation of the oral/pharyngeal tissues.

Most published reports indicate that age, gender, race, tobacco use, alcohol use (especially tobacco and alcohol in combination),presence of a synchronous cancer of the upper aerodigestive tract, poor nutritional status, infection with certain viruses (HPV 16,18) , oral lichen planus,candida infections,poor oral hygiene and immune deficiencies all increase the relative risk for developing an oral cancer.

Typically 90% of men and 60% of women with oral carcinoma use tobacco (Baker SR. 1993). The incidence rate of oral carcinoma in smokers is six times greater when compared to non-smokers. Tobacco use over time may cause progressive morphologie changes to the oral mucosa with eventual malignant transformation. The risk of malignant transformation is related to the amount of tobacco used and the duration smoked.

Typically, 75% to 80% of patients with oral carcinoma use or have used alcohol. The incidence rate of oral carcinoma in this group is six times greater compared to non-drinker (Alvi A et al. 1996; Baker SR. 1993)[4,5]. It has been found that most heavy drinkers are also heavy smokers. The risk for a smoker to develop oral cancer is 5 times that of a non-smoker, and this risk increases to 15 times for a smoker who also uses alcohol (Alvi A et al. 1996)[4].

Considerable research has been focused in the recent past on the carcinogenic, mutagenic, and genotoxic potential of betel quid ingredients, especially tobacco and areca nut[6].

II. MATERIALS AND METHODS

Population of study- Patients undergoing surgery for oral intraepithelial lesions in department of surgery in Hi Tech Medical College and Hospital, Bhubaneswar, Odisha.
Study period- Nov 2015-Oct 2017
Study design – Prospective study
Study Area- Hi Tech Medical College and Hospital, Bhubaneswar, Department of pathology.

The study included 200 cases after getting approval by the Institutional Ethics Committee. Written consent was taken from the participants.

Selection of cases:
Inclusion criteria-
1. Cases cytological diagnosed and biopsy proven as intraepithelial lesions & invasive growths of oral mucosa.
2. Operated cases of growth without prior cytological evaluation in oral mucosa.

Exclusion criteria-
1. Non neoplastic inflammatory lesions.
2. Biopsy examined cases of oral mucosa with epithelial hyperplasia without dysplasia.

STATISTICAL ANALYSIS was made considering the clinical and histopathological data .Then transformed to a master chart by using Microsoft excel sheet, which was then subjected to statistical analysis using chi square test by using SPSS ,version 20.The findings were arranged in tables and graphs using Microsoft excel sheet. Analysis was done in the form of percentage and represented as tables and figures where necessary. P value of ≤ 0.05 is considered as statistically significant.

III. RESULTS

An observational clinical correlation study of 200 patients with oral dysplasia and SCC were undertaken for the study.

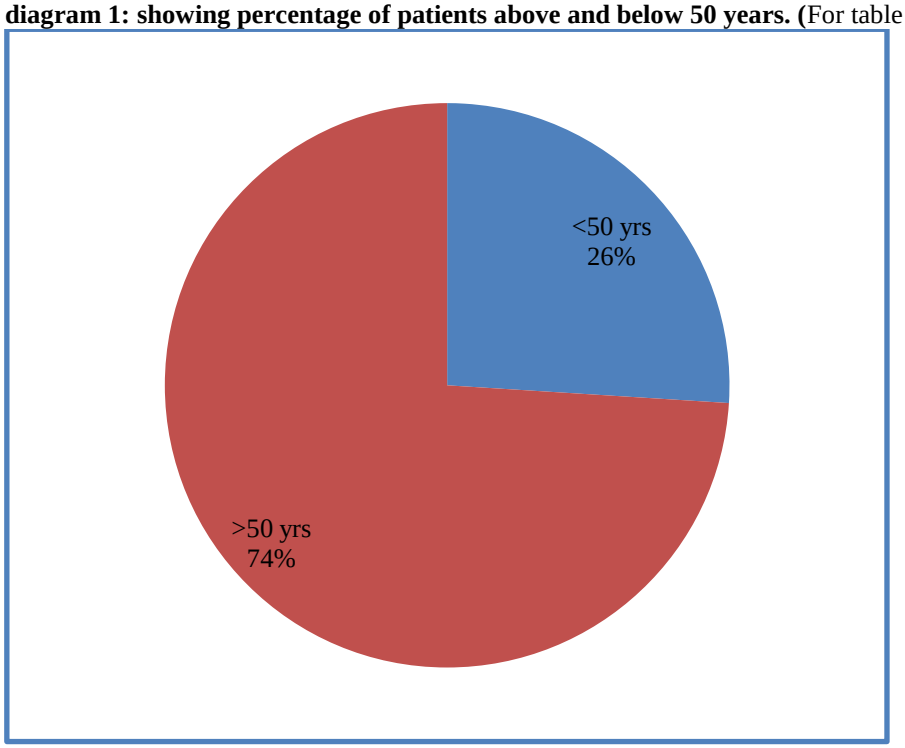
In the present study ,patients having dysplasia or carcinoma which are younger than 50 years were 26% (22% Male& 4% Female) and more than 50 years were74%(42% Male &32% Female). (table 1) **,(**pie diagram 1)

Table 1 : MEAN AGE DISTRIBUTION OF PATIENTS STUDIED

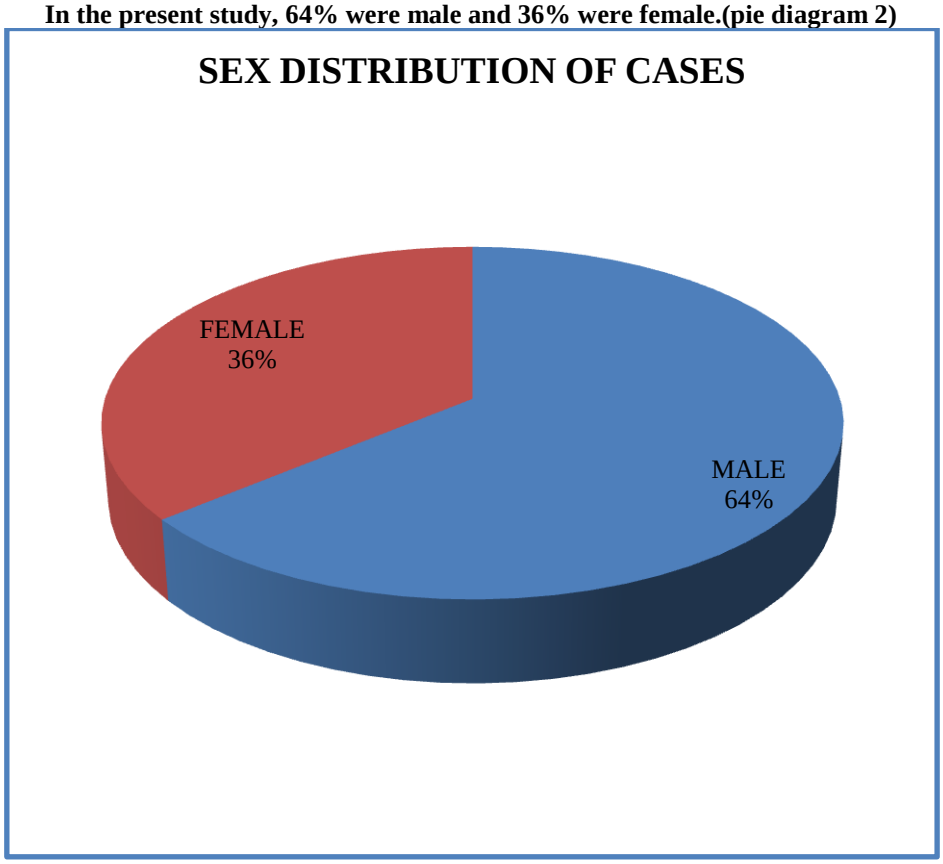
AGE IN YEARS	MALE(%)	FEMALE(%)	Total NO. OF PATIENTS (%)
<50 YEARS	**44(22%)**	**8(4%)**	**52 (26%)**
≥50 YEARS	**84(42%)**	**64(32%)**	**148 (74%)**
Total	**128(64%)**	**72(36%)**	**20 (100%)**

Chi Square=3.24 p value=0.07 Not Significant

Pie diagram 1: showing percentage of patients above and below 50 years. (For table 1)



In the present study, 64% were male and 36% were female.(pie diagram 2)



Pie diagram 2: showing Sex distribution of the cases.

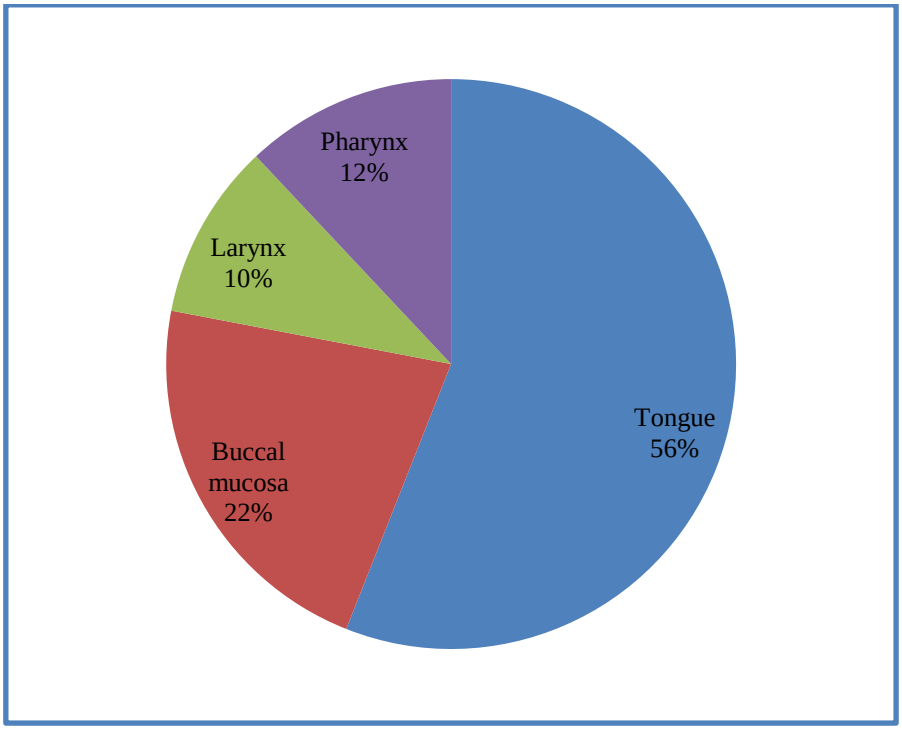
In our study, maximum number of cases (32%) had history of tobacco chewing alone, followed by 20% of cases with history of tobacco chewing & smoking; 14% cases with history of alcohol consumption; 14% cases with H/O tobacco chewing+smoking+alcohol;8% had the H/O smoking and 12% cases were Non-Addict. Total cases with H/O smoking were 25(50%); tobacco chewing were 33(66%); and alcohol consumption were 14(28%). (Table 2)

Table 2: History of smoking, alcohol and tobacco chewing

HISTORY	MALE (%)	FEMALE (%)	No. of Cases (%)
SMOKING	12(6%)	4(2%)	16(8%)
ALCOHOL	20(10%)	8(4%)	28(14%)
TOBACCO CHEWING	40(20%)	24(12%)	64(32%)
TOBACCO CHEWING+SMOKING	32(16%)	8(4%)	40(20%)
TOBACCO CHEWING+SMOKING+ALCOHOL	16(8%)	12(6%)	28(14%)
NON-ADDICT	8(4%)	16(8%)	24(12%)
TOTAL	128 (64%)	72 (36%)	200(100%)

(Chi Square=4.1 p value=0.53 Not Significant)

Majority of cases (56%) ,presented on tongue, followed by buccal mucosa (22%),pharynx (12%) and larynx(10%). (pie diagram 3)



Pie diagram 3: showing Percentage of sites involvement

Clinically, 60 (30%) cases were diagnosed as dysplasia and 140 (70%) cases were diagnosed as SCC.
Clinically, out of total 60 dysplasia cases,24 (40%) cases showed mild dysplasia,24 (40%) showed moderate dysplasia and 12 cases (20%) showed severe dysplasia. In our study, there were 80 (57.2%) cases of WD SCC,44 (31.4%) cases of MD SCC and 16 (11.4%) cases of PD SCC.

IV. DISCUSSION

Oral squamous cell carcinoma is one of the major cause of morbidity and morta.lity in India and in many developing nations Tobacco and alcohol are the most important contributing factors in oral cancer etiology.

This prospective study included 200 cases of oral squamous cell lesions which came to our institution Hi Tech Medical College and Hospital during the period of November 2015 to October 2017.
In the present study, mean age of study population was 50years ,which was similar to P. Baweja et al [7] ,but lower when compared to Deniz et al[8] and Ashraf et al[9].
In the present study, it was noted that the lesions were commoner in male than female. Similar observations were made by P Baweja et al[7] and Claudia et al[10] .But in the study done by Juan C et al[11] female involvement was more than male.

In the present study it was noted that majority of the patients had a history of tobacco chewing.Similar observations were noted in studies made by P Baweza et al[7] ,Deniz et al [8] and Fernandez et al [12] .

Correlation of smoking, alcohol and tobacco chewing history with other study

History	**Authors**			
	Fernandez et al[12]	Deniz et al [8]	P Baweja et al[7]	Present study
Smoking %	-	100	53	50
Alcohol %	75	24	37	28
Tobacco chewing %	85	-	58	66

In the present study, most of lesions were located in tongue and buccal mucosal regions. Similar observation was made by, P Baweja et al[7] Claudia et al[10], and Juan C et al [11].Incidence of tumors were highest in tongue in all these studies.

Almost 56% of our samples were tongue tumours, which is in accordance with the literature Neville & Dayet al[13]. and others.

The rich lymphatic network of the tongue and floor of the mouth seems to favour an early dissemination of the disease to cervical lymph nodes, decreasing the 5-year survival rate to values lower than 20% (Dias *et al.*)[14].

Moreover, experimental studies have shown that tongue tissue displays a higher amount of some enzymes responsible for the metabolic activation of chemical carcinogens than other regions in the oral tissues (Von Pressentin *et al.*)[120].

Nevertheless, the floor of the mouth and lateral and ventral tongue surfaces are characterized by a higher permeability due to their thinner, no keratinized mucosa, providing less protection against carcinogens (Neville & Day)[15].

These features may give a possible explanation for the high frequency and aggressiveness of tongue tumours.

The cessation of smoking leads to a progressively lower cancer risk. In support of this theory, Moore (1971) followed 203 smokers "cured" of their cancer of the upper aerodigestive tract over a 7 year period and found that 40% of patients who continued smoking developed second cancers compared to 6% of patients who stopped smoking.

Boffetta et al [16] have shown the carcinogenic effects of tobacco and alcohol to act through direct contact and tend to be site specific in the oral cavity. Tobacco smoking was more closely associated with carcinoma of the soft palate and alcohol was more closely associated with carcinoma of the floor of the mouth and tongue.

V. CONCLUSION

Our findings suggest that smoking and alcohol consumption have its greatest impact on oral carcinogenesis and is thus the major risk factors. Majority of the cases (66%) had history of tobacco chewing.

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