ORAL SQUAMOUS CELL CARCINOMA: EFFECT OF TOBACCO AND ALCOHOL ON CANCER LOCATION

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KEYWORDS

Oral Cancer, Squamous Cell Cancer, Carcinogen, Alcohol, Smoking, Tumour Site

ABSTRACT

Introduction- The root causes of oral squamous cell malignancies (OSCC) have been identified, however little research has concentrated on etiological distinctions between affected oral cavity regions. **Methodology:** A cross-sectional analysis of patients with primary OSCC was undertaken retrospectively. The patients' self-reported history of tobacco smoking and alcohol use was explained by tumour site, age, gender, tumour size, and lymph node status in a logistic regression model. The data on smoking and alcohol consumption was gathered from a patient background form. **Results-**In 519 patients, tumors occurred most often in the tongue (51%), gingiva (21%), or floor of the mouth (FOM; 15%). FOM had 26-fold greater odds for a history of smoking and alcohol use than other tumor sites (OR=25.78; 95% CI: 8.02–82.95; $p<0.001$). Gingival and buccal sites were associated significantly less with smoking and alcohol use (OR=0.43, 95% CI: 0.28–0.67; $p<0.001$ and OR=0.47; 95% CI: 0.25–0.92; $p<0.026$, respectively). **Conclusions-**OSCC of oral cavity sites has notable differences in etiology. FOM seems to be related almost exclusively to conventional smoking and heavy alcohol use.

Introduction

Cancer of the oral cavity is a major public health concern across globally, ranking sixth in terms of incidence and with a 5-year survival rate of 50%.[1] It constitutes around 3% of all malignancies reported each year in the United States. Approximately 90% of these cases are oral squamous cell carcinoma (OSCC). According to the American Cancer Society, the most recent predictions for oral cavity and oropharyngeal malignancies in the US are for 2024. (1) roughly 58,450 new instances of oral cavity or oropharyngeal cancer; (2) nearly 12,230 fatalities from oral cavity or oropharyngeal cancer.[2]

The kind of alcohol or tobacco contributes to the danger, with black tobacco and liquor particularly linked to OSCC formation. The amalgamation of both boosts the chance of OSCC by 15 times, particularly for floor-of-mouth (FOM) cancer.[1] Pipes and cigars appear to pose much greater danger for OSCC than normal cigarettes, most likely because to the alkaline smoke that stays in the oral cavity for extended. OSCC danger grows with everyday dosage of carcinogen items, period of intake, and lifelong cumulative intake of both.[3,4]

There are additional risks, as the occurrence of tongue squamous cell carcinoma appears increasing among younger non-smoking, non-drinking females. Betel quid chewing, diet, familial histories of cancer, oral and dental health difficulties, ill-fitting prosthetic teeth,

mechanical irritation, oral lichen planus, lichenoid responses, immunosuppression, and vitamin/nutrition deficiencies have all been identified as risk factors for OSCC. Human papillomavirus (HPV) is connected with malignancies of the base of the tongue and the oropharynx, but not OTSCC. These other risk factors appear to be more common in patients who never smoke or drink.[5,6]

The purpose of this study was to clarify the role of tobacco smoking and alcohol in different OSCC sites. Our hypothesis was that the role of preceding smoking and alcohol exposure varies according to the site of oral cancer.

Methodology

A cross-sectional analysis of individuals with acute OSCC was undertaken retrospectively at the Department of Oral and Maxillofacial Pathology, RKDF Dental College and Research Centre in Bhopal. Patient details were obtained from the OPD database. Any individuals who had a primary OSCC diagnosis across the research's duration were included. Patients with previous episodes of oral cavity malignancies were eliminated.

The information on tobacco smoking and alcohol consumption was obtained from a separate background data form that every person with cancer completed. The relationship amongst patients' self-reported smoking and alcohol usage and the site of OSCC was studied. The tumour locations were classified as tongue, buccal mucosa, gingiva, palate, and FOM. Additionally, an anamnestic admitted to background in alcohol and smoking was examined in more depth in individuals with OSCC of FOM.

The expressed smoking and alcohol use data were obtained using an individual's history form which all individuals with cancer complete in the medical facility. Subjects were divided in 2 categories based on their smoking habits: non-smokers (non-smokers and past smokers in cessation for at least 5 years) and smokers (present smokers and former smokers in cessation for less than 5 years). Occasionally smokers were classified as nonsmokers. In terms of alcohol, seven doses per week or more were considered heavy alcohol usage, as 70 g of alcohol per week or more is thought to raise the chance of developing cancer. One dose is equivalent to 10-12 grammes of pure alcohol.

Tumour locations were classified as tongue, buccal mucosa, gingiva, palate, and FOM using The International Classification of Diseases 10th coding. Other characteristics evaluated included age, gender, tumour size, and lymph node status. Tumour size was classified as T1-4 relying on the TNM staging of lip and oral cavity malignancies. Pathological lymph node grade was classified as N0, N1, or greater.

Statistical analysis

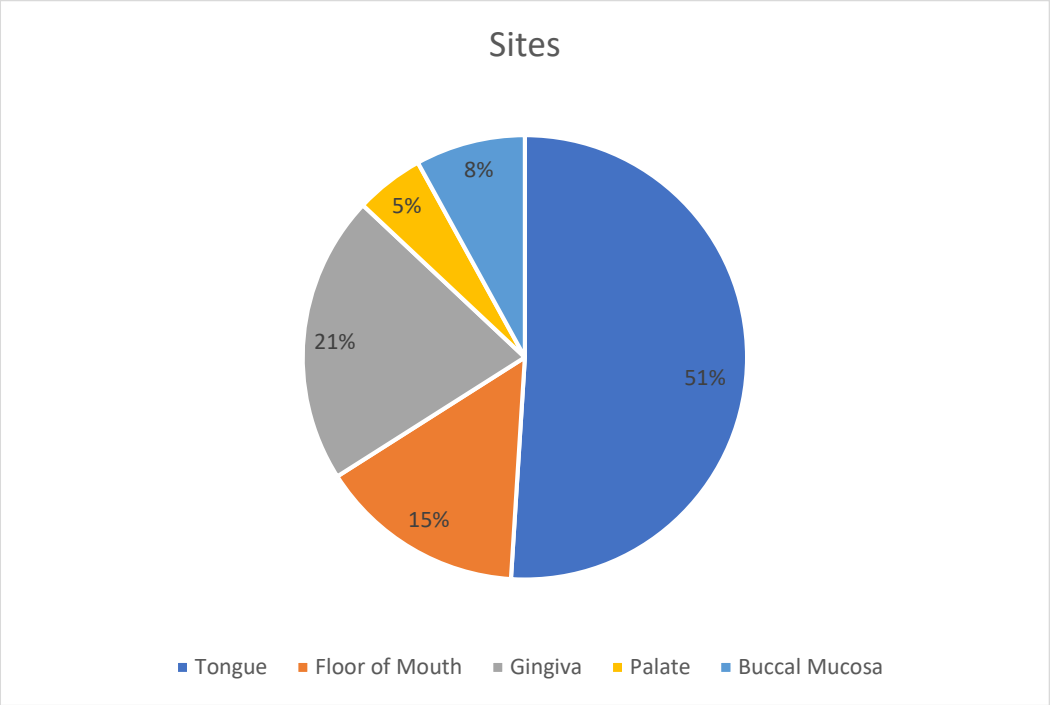
The self-reported history of tobacco smoking and heavy alcohol use (either or both) was explained with patients' clinical data in a logistic regression analysis. Logistic regression analyses were evaluated for goodness-of-fit using the Hosmer and Lemeshow test. A significance level of 0.05 was set for all analyses. Statistical analyses were performed using SPSS 23.

Results

In total, 519 OSCC patients met the inclusion criteria and were included in the final analysis. Respondent ages varied from 19 to 98 years (mean = 66.3 years). OSCC patients were more likely to be men (59%) than women (41%). More over half (57%) of the patients had a record of smoking, excessive alcohol consumption, or both. Half of the patients (50.5%) were smokers

(present and previous smokers in quit for less than 5 years). 38% of the OSCCs reported heavy alcohol intake. Overall, 202 (39%) of the 519 patients had tumours classified as T1.

Figure 1- Site distribution of Oral squamous cell Carcinoma



The preceding years of self-reported smoking and alcohol use vary among OSCC locations. Only half (52%) of those with OSCC stated having cigarettes or alcohol heavily. whereas nearly every one (96%) individuals with FOM squamous cell cancer (SCC) had previously been exposed to them.

Table 2-*Univariate logistic regression model explaining self-reported history of smoking and heavy alcohol use[*] among patients with oral squamous cell carcinoma sites,*

Variable	***OR***	***95% CI***	***p***
Tongue	0.68	0.48–0.97	**0.032**
Gingiva	0.43	0.28–0.67	**<0.001**
Floor of the mouth	25.78	8.02–82.95	**<0.001**
Palatinum	3.70	0.98–5.65	0.055
Buccal	0.47	0.25–0.92	**0.026**

Table 3.*Logistic regression model explaining the self-reported history of smoking and heavy alcohol use[*] with patient demographics, tumour size, and lymph node metastasis among patients with oral squamous cell carcinoma sites.*

Univariate logistic regression analysis				***Multivariate logistic regression analysis****			
Variable	***OR***	***95% CI***	***p***	***Variable***	***AOR***	***95% CI***	***p***
Age (years)	0.95	0.94–0.97	**<0.001**	Age (years)	0.95	0.94–0.97	**<0.001**
Sex (Ref. male)	0.33	0.23–0.48	**<0.001**	T-classification (Ref. T1-T2)	1.73	1.15–2.60	**0.009**
T-classification (Ref. T1-T2)	1.25	0.87–1.80	0.230	Floor of the mouth (Ref. no)	26.15	8.01–84.84	**<0.001**
Lymph node metastasis (Ref. no)	2.15	1.44–3.21	**<0.001**				
Floor of the mouth (Ref. no)	25.78	8.02–82.95	**<0.001**				

Further univariate logistic regression analyses showed statistical associations of sex and preceding history of smoking or alcohol use with FOM. Males were 2.5 times more likely to have SCC of FOM than females (OR=2.54; 95% CI: 1.47–4.40; p<0.001). Smoking and heavy alcohol use increased the odds for SCC of FOM significantly (OR=9.01; 95% CI: 4.52–17.93; p<0.001 and OR=5.63; 95% CI: 3.31–9.57; p<0.001, respectively).

Discussion

OSCC is the most common oral cavity cancer, and it can spread to any place within the mouth.[7] The tongue was the most common place in present investigation, which is consistent with previous research. In contrast to recent investigations in distinct categories, the gingiva is ranked second, the FOM third, and the buccal mucosa fourth. variances across the studies are most likely explained by variances in participants, particularly smoking and high alcohol consumption. Subjects in this research used alcohol and cigarettes frequently; 57% stated that they used either or both. [8]

This raises the percentage of FOM. As demonstrated in present research, smoking and alcohol consumption were 25.8 times greater to be associated with SCC of FOM than with other OSCC locations. Additional researchers have already noted the link, while the process causing the changes remains unknown.[9]

The architecture of FOM may explain why it is more susceptible to carcinogen. Tissue arrangement, molecular marker expression, and epithelial rates of turnover vary among the oral cavity locations.[7] FOM has a thin, non-keratinized mucosa, and carcinogens tend to gather while mixed with saliva, resulting in increased exposure to dangerous chemicals. The average turnover of non-keratinized epithelium is 25 days, while keratinised epithelium is around 50 days. The keratin covering works as a barrier, insulating the mucosa from external factors. Because FOM lacked this barrier, its structure makes it more prone to toxins and the high temperatures of tobacco smoke. OSCCs of the non-keratinized lining epithelium of the FOM and buccal mucosa are often associated to tobacco and alcohol use, but OSCCs of the keratinised masticatory epithelium of the gingiva and hard palate are more prevalent in women who do not engage in risky behaviour.[9,10]

The variations in structure may explain why FOM is more susceptible to carcinogens and thus more common among smokers and drinkers. Nevertheless, given there has been an upsurge in

non-smoking, non-drinking (NSND) individuals with carcinoma of the tongue, the burning issue is whether elements that do not alter FOM might be behind the boost in OTSCC instances.[10,11]

Conclusion

OSCC of different sites should be considered as different entities to find preventive and predisposing factors for malignancy. In the future, OSCC treatments may differ according to etiology.

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