Original Article

Risk Factors Associated with Oral Cancer: A Hospital-based Case–control Study in Telangana State, India

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Abstract

Introduction: The use of tobacco products and alcohol consumption are two of the main risk factors for cancer of the oral cavity. The contribution of other factors such as diet, genetic predisposition, oral hygiene, and demographic variables to the occurrence of oral cancer has been debatable. This study explored the association between different risk factors and the risk of oral cancer using propensity score methods. **Methodology:** A case–control study was conducted in Hyderabad, Telangana state, India in cancer hospitals between October 2022 and July 2023. Direct interviews with 238 cases and 450 controls were used to gather information about the different risk factors using structured data collection forms. Chi-square tests and multivariable logistic regression along with computation of propensity scores and inverse probability of treatment weighting for odds ratios (ORs) with weighted logistic regression were conducted using R version 4.3.2. **Results:** Bivariate analysis found a significant difference in tobacco use, duration of use, frequency of alcohol consumption, and vegetable consumption between the cases and controls. Comparing cases of oral cancer to controls, the propensity weighed ORs were higher for those who had a habit of using smokeless tobacco (OR = 9.09, 95% confidence interval [CI]: 5.624–15.143) and smoking (OR = 8.96, 95% CI: 4.988–16.113) for more than 10 years. Oral cancer risk factors also included a history of chronic trauma in the mouth (OR = 3.00, 95% CI: 1.287–6.996) and daily alcohol consumption (OR = 5.69, 95% CI: 3.518–9.220). **Conclusion:** This study establishes the role of tobacco use as a risk factor for oral cancer, with an emphasis on duration of use, and provides supporting evidence for the role of chronic oral trauma in oral cavity cancer.

Keywords: *Mouth neoplasms, propensity score, risk factors, tobacco use*

Introduction

In the head-and-neck region, one of the most prevalent forms of cancer is oral squamous cell carcinoma (OSCC), which originates from the lips and oral cavity. According to data from the Global Cancer Observatory, there were 377,713 cases of OSCC on an annual basis in 2020 throughout the world with the Asian region having the highest number of cases.[1] Oral cancer is a worldwide issue, with a mortality rate of around 50%.[1] Even though oral cancers can be easily examined, these cancers present late and have a catastrophic impact on survival and quality of life.[2] Delays in diagnosis are the main cause of this unacceptable high death rate from oral cancer, and they may be linked to a complex interaction between factors related to the patient as well as those related to healthcare providers and services.[3] Asian countries would largely benefit from screening programs because the region accounts for two-thirds of oral cancer cases and three-quarters of cancer-related deaths.[2]

The risk of oral cancer is determined by a multitude of genetic, environmental, and behavioral factors. Heavy alcohol consumption and tobacco use, both smokeless and smoked, are the main established risk factors for oral cavity cancer.[4] There is uncertainty surrounding the contribution of additional risk factors, including diet, oral health and hygiene, persistent trauma, hormones, stress, and genetic, occupational, and socioeconomic status, to the risk of oral cancer.[5]

Around 50% of oral cancer patients go undiagnosed until the disease has significantly progressed mainly due to a diagnostic delay caused by a severe lack of public awareness and a lack of readily available screening tools.[6] Oral cancer and its precancerous lesions have been linked to

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smoking, drinking alcohol, and chewing tobacco, poor diet, chronic trauma, and poor oral health based on numerous case–control study findings.[6,7] However, the presence of confounders in case–control studies severely restricts how these results are interpreted.[8] Although using different study designs, such as cohort studies or nested case–control studies are advised,[9] practical difficulties may prevent this from being possible.

In medical research, multivariable methods have grown in popularity as a means of data analysis. Regression analysis is widely used to provide a quantitative estimate of risk for the individual effect and to determine the impact of individual variables in a multivariable context adjusting for known confounders.[10] Nevertheless, when these techniques are used, some essential precautions and assumptions are frequently overlooked or not documented.[11] A skewed odds ratio (OR), for instance, is often the consequence of breaking the recommended threshold of 10–20 events per variable.[11]

In recent times, propensity scores are gaining popularity to be used to account for measured confounding. Propensity scores have certain advantages over multivariable outcome models, one of which is their capacity to display the balance of covariates, which is required to eliminate confounding, the trimming of patients outside of a typical range of covariates for which there is no information to calculate the impact of treatment, and the capacity to calculate causal contrasts when treatment effect heterogeneity is present.[12] For example, employing a large-scale propensity score-based study to reduce potential selection bias and confounding effects, Deng *et al*. demonstrated that poor oral hygiene was associated with an increased risk of oral cancer.[13]

One of the techniques for incorporating propensity scores into study designs and data analysis is the inverse probability of treatment weighting (IPTW).[14] Despite various studies emphasizing the link between tobacco usage and oral cavity cancer, the accuracy in establishing a causal relationship between them can be achieved using advanced statistical techniques to adjust for confounders.[15] This study used IPTW to minimize potential confounding effects to further explore the relationship between various risk factors and oral cancer risk, given the relatively high incidence of oral cancer in this region. The specific research hypothesis was that the combination of tobacco use, alcohol consumption, and chronic oral trauma significantly increases the risk of oral cancer compared to individuals with none or only one of these risk factors.

Methodology

Study design and setting

A case–control study was carried out in Telangana, India, between October 2022 and July 2023. From three cancer hospitals, where 238 newly diagnosed cases of cancer of the oral cavity were gathered. Telangana, India's 29th state, was formed on June 2, 2014. According to the 2011 census, the state's population is 35,003,674. With Hyderabad as its capital, 60% of the state's population lives in rural areas. Based on information from India's population-based cancer registries, the most common cancers among men in the Hyderabad district were tongue, lung, and mouth cancers.[16]

Selection of cases and controls

Cases were defined as those with newly diagnosed oral cancer, a histopathologically confirmed report, and visiting the selected cancer hospital during the study period. The anatomical sites for oral cancer included were C00–C06 based on the International Classification of Diseases, Oncology, 3rd edition which refers to cancer of the oral cavity.[17] Patients over the age of 18 years and individuals who had a histologically verified diagnosis of oral cancer were included in the case group. Patients with a diagnosis of cancer recurrence or metastasized cancer in the advance stage, and individuals incapable of cooperating during the data collection process were excluded. The recruitment of cases started in November 2022 with a weekly visit to each of the hospitals and an average of 5–10 incident cases of oral cavity cancer being recruited at each visit. The median time of recent case inclusion was reached by March 2023. Recruitment of cases continued up to July 2023 after which the recruitment was stopped. Controls were recruited during the same period simultaneously with cases. Healthy controls were recruited from the same hospitals as cases and were relatives, friends, or accompanying persons of the patients or visitors to the hospital. Subjects without any history of oral cancer and above the age of 18 years were selected as controls. Those suffering from any other malignancy associated with tobacco or alcohol usage such as lung, liver, or esophageal cancer were not included. Group matching was done among cases and controls based on age (±2 years) and gender.

Sample size estimation

The anticipated OR for a tobacco user was taken as 2.0 from a previous study.[18] With the proportion of cases exposed as 20% and proportion of controls exposed as 10% and at 90% power of the study, the minimum sample size was estimated as 175 in the case group and 350 in the control group. Sample size was estimated in R studio (developed by Posit, PBC, a public-benefit corporation founded by J. J. Allaire creator of the programming language ColdFusion) using the package epiR for sample size estimation.[19]

Exposure ascertainment

Direct interviews were conducted by a single researcher who is a dentist with previous experience in conducting epidemiological studies, using a predesigned schedule to gather data.

This schedule was designed after a review of literature related to existing risk factors for oral cavity cancer and

content validation by a group of experts which included specialists in oral medicine, oral surgeons, and oncologists. The content validation was carried out as per the methods suggested by Yusoff.[20] The CVI for the schedule used to ascertain exposure was 0.86 which was satisfactory. Demographic details such as age, gender, socioeconomic status based on occupation of the head of the household, area of residence, and family history of cancer were obtained from the patient in the interview. Occupation of the head of the household was considered as a surrogate for socioeconomic status. People with steady jobs, such as teachers, clerks, professionals, or business owners, were categorized as belonging to the upper economic group and those without jobs, farmers, small-time traders, or manual laborers were classified as members of the lower economic group.[21] Participants were interviewed about their, lifestyle choices (such as tobacco usage and alcohol consumption), dental hygiene, family history of cancer, and food preferences (such as how often they ate fruits and vegetables). Assistance was sought from the caregivers in case the participants had difficulty in speaking.

A person who has used any form of tobacco at least once daily for a period of 1 year or more was considered as tobacco user and those who have never used or reported occasional use of tobacco were categorized as never users. Smoking forms include cigarette, bidi, chutta, cigar, or pipe and smokeless forms include chewable forms such as betel quid with tobacco, gutkha, khaini, zarda, or any other form specified by the participant. The history of usage of tobacco was recorded based on the duration, which was categorized into "<10 years" or "more than 10 years" for smoking or smokeless form of tobacco use. Alcohol consumption was recorded as never for those who "never" consumed any form of alcohol, "occasional" who consume alcohol less than once in a month, and "daily" as those who consume alcohol at least once a day for at least 6 months. History related to dietary habits included information about the consumption of vegetables and fruits, which was taken on a 4-point scale ranging from weekly, daily, occasionally, and never. This was later combined only into two categories of consumption <3 times a week and consumption more than 3 times a week. Data regarding oral hygiene practices included information on regular brushing habits and the habit of mouth rinsing with water were also collected. History of any recurrent trauma or chronic injury to the oral mucosa exceeding a duration of 3 weeks was also collected. The study methods, analysis, and presentation of results are conducted in accordance with the STROBE checklist[22] for case–control studies [Annexure 1]. Figure 1 presents a directed acyclic graph that illustrates the hypothesized associations between variables.[23]

Statistical analysis

Data collected using paper-based forms were entered in MS Excel, and data cleaning was later conducted. Statistical analysis was performed with R version 4.3.2. Using the ipw package in R, IPTW was performed to evaluate the impact of different risk factors on oral cancer.[24] Inverse probability of treatment weights was computed using estimated propensity scores to achieve a balance between measured baseline covariates among cases and controls.[25] Two key steps in IPTW were followed.[15] First, given an individual's characteristics, the probability – or propensity – of being exposed was computed. Second, each person's weight was determined by taking the inverse of the probability that they will experience their actual level of exposure. A pseudo population with equally distributed measured confounders across groups was produced by applying these weights to the study population.[15] When the absolute standardized mean difference was obtained at <0.1, the covariates were considered balanced.

The risk factors, such as tobacco use, food habits, oral hygiene, and chronic trauma, were compared between cases and controls using the Chi-square test in the weighted population next, using a weighted regression analysis, ORs with 95% confidence intervals (CIs) were determined. Multivariable binary logistic regression was also done on the original dataset, controlling for age, gender, socioeconomic status, and place of residence, to evaluate the relationship between different risk factors and oral cancer. A significance threshold of $P < 0.05$ was applied.

Results

The study involved 688 participants in total, 238 of whom were patients with oral cancer and 450 were healthy controls. Table 1 displays the baseline characteristics of the participants in the case and control groups. There was a notable imbalance in the demographic characteristics.

IPTW was done after generating propensity scores. After trimming extreme weights in the sample of 214 cases of oral cancer and 423 controls, the imbalance in the demographic characteristics was reduced and attributes were evenly balanced between the two groups (standardized mean difference < 0.15). Figure 2 depicts the distribution of baseline covariates among the study population before and after IPTW. There was significant overlap among the cases and controls before weighting. A balance in the confounding variables was achieved after trimming extreme weights. Table 2 shows a significant difference between the cases and controls when comparing different risk factors associated with oral cancer, such as diet, tobacco habits, and alcohol consumption, but there was no significant difference in the dental factors such as chronic trauma and mouthwashing after eating food and family history of cancer.

Binary logistic regression was carried out on the original dataset after adjusting for age, gender, occupation, and area of residence and on the weighted dataset. Although the dental factors were not significant in the bivariate

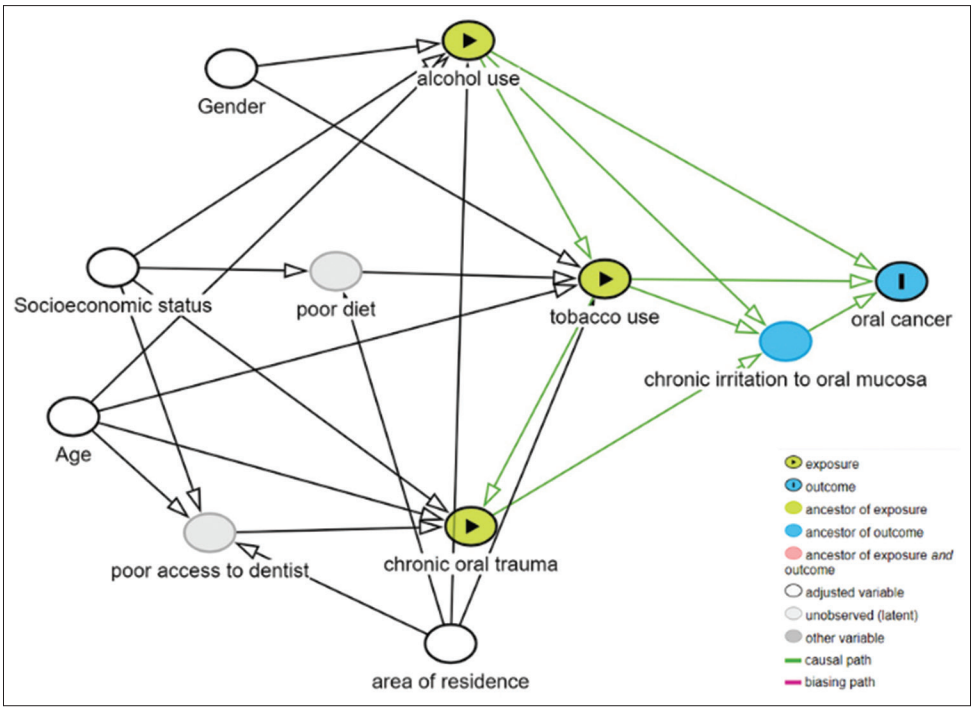

Figure 1: Directed acyclic graph for role of various risk factors on risk of oral cancer

Table 1: Distribution of study subjects based on demographic variables at baseline and after inverse probability of treatment weighting analysis

Characteristics	At baseline			After IPTW		
	Case (*n*=238), *n* (%)	Controls (*n*=450), *n* (%)	SMD	Cases (*n*=214), *n* (%)	Controls (*n*=420), *n* (%)	SMD
Age (years)						
35–44	19 (7.23)	31 (5.04)	0.203	19 (9.1)	27 (6.3)	0.120
45–54	52 (21.46)	132 (25.50)		56 (26.0)	124 (29.5)	
55–64	102 (42.90)	193 (47.87)		99 (46.3)	193 (46.0)	
65–74	65 (28.41)	94 (21.58)		40 (18.7)	76 (18.0)	
Gender						
Female	33 (13.9)	62 (13.8)	0.003	31 (14.5)	60 (14.3)	0.007
Male	205 (86.1)	388 (86.2)		183 (85.5)	360 (85.7)	
Occupation of head of household						
Employed	164 (68.9)	243 (54)	0.146	94 (44.1)	238 (56.7)	0.017
Unemployed	74 (31.1)	207 (46)		120 (55.9)	182 (43.3)	
Residence						
Rural	181 (76.1)	313 (69.6)	0.310	148 (69.3)	294 (70.1)	0.018
Urban	57 (23.9)	137 (30.4)		66 (30.9)	126 (29.9)	

IPTW: Inverse probability of treatment weighting analysis; SMD: Standardized mean difference

analysis due to the relative importance of these factors in previous research, they were included in the multivariable analysis. Table 3 depicts the multivariable binary logistic regression of risk factors and oral cancer in the adjusted and weighted datasets. The highest risks of oral cancer were seen among smokers who have the habit for more than 10 years (OR = 7.57, 95% CI: 4.192–13.901) and users of smokeless tobacco (OR = 9.09, 95% CI: 5.367–15.693) with a history of use more than 10 years. The likelihood of developing oral cancer was higher in alcoholics (OR = 5.18, 95% CI: 3.214–8.468). Occasional consumption of vegetables increased the risk. Patients with a history of persistent trauma from sharp teeth or ill-fitting dentures were statistically significantly more likely to acquire oral cancer (OR = 3.24, 95% CI: 1.408–7.352).

Discussion

Using propensity score analysis, the current case–control study investigated the risk factors for cancer of the oral cavity. To improve the covariate balance between cases

Table 2: Comparison of behavioral factors, diet, family history and dental factors among cases and controls

Characteristics	Cases (*n*=214), *n* (%)	Controls (*n*=420), *n* (%)	*P*
Tobacco usage			
Never	53 (24.5)	283 (67.4)	<0.001
Ever	161 (75.5)	137 (32.6)	
Smoking type			
Cigarette	22 (9.5)	25 (5.5)	0.512
Bidi/chutta	33 (13.8)	29 (5.7)	
Not applicable	159 (70.6)	366 (84.4)	
Years of smoking			
Never	159 (74.3)	366 (87.2)	<0.001
<10	11 (5.2)	25 (6.0)	
>10	44 (20.5)	29 (6.8)	
Smokeless type			
Gutkha/Khaini	97 (48.3)	68 (20.4)	0.625
Quid without tobacco	10 (4.2)	14 (3.11)	
Not applicable	107 (47.5)	338 (76.4)	
Years of smokeless tobacco			
Never	107 (50.2)	338 (80.5)	<0.001
<10	47 (21.9)	40 (9.5)	
>10	60 (27.9)	42 (10.0)	
Alcohol frequency			
Never	92 (42.8)	276 (65.8)	<0.001
Occasional	42 (19.7)	91 (21.6)	
Daily	80 (37.5)	53 (12.6)	
Vegetables (times/week)			
>3	161 (75.7)	376 (89.4)	<0.001
<3	53 (24.6)	44 (10.6)	
Fruits (times/week)			
>3	33 (15.4)	72 (17.1)	0.611
<3	181 (84.6)	348 (82.9)	
Mouth rinsing			
Daily	54 (25.2)	82 (19.5)	0.065
Occasional	160 (74.8)	338 (80.5)	
History of chronic trauma			
Absent	199 (93.1)	400 (95.2)	0.267
Present	15 (6.9)	20 (4.8)	
Family history of cancer			
No	204 (95.6)	405 (96.4)	0.599
Yes	9 (4.4)	15 (3.6)	

and controls and to lessen the confounding effects of demographic variables, we employed IPTW. In line with earlier research,[7] our study has also demonstrated that tobacco use and alcohol consumption in any form is associated with a higher risk of oral cavity cancer.

Tobacco and excessive alcohol consumption are two well-known risk factors for the occurrence of oral cancer.[26] Acetaldehyde, a byproduct of alcohol metabolism, primarily affects DNA by binding to it and forming genotoxic DNA adducts. Tobacco uses other carcinogens and tobacco-specific nitrosamines to cause cancer. Highly reactive molecules are produced when N'-nitroso nor nicotine is metabolized. These molecules may form DNA adducts, leading to miscoding during DNA replication. Double-strand breaks, point mutations in DNA, and other structural alterations in the genome can be brought on by these.[27]

In clinical practice, research, and healthcare policy, duration (years smoked) is a cumulative exposure indicator of tobacco usage burden that is frequently used to estimate patients' risk of developing tobacco-related diseases.[28] In the current study for smokers who have smoked for <10 years, the risk of oral cancer did not seem to increase significantly (OR = 1.84; CI: 0.77–4.18). This is consistent with a similar study by Gupta *et al*. in Maharashtra, India, where smokers with a habit of smoking for up to 25 years showed no significant increase in the risk of oral cancer.[7] This has significant implications and should

Table 3: Multivariable binary logistic regression of risk factors and oral cancer in adjusted and weighted dataset

Characteristic	Cases (*n*=238), *n* (%)	Controls (*n*=450), *n* (%)	Adjusted OR	Cases (*n*=215), *n* (%)	Controls (*n*=423), *n* (%)	OR after IPTW
Years of smoking						
Never	172 (72.3)	391 (86.9)	Reference	160 (74.3)	367 (86.7)	Reference
<0	10 (4.2)	25 (5.6)	1.710 (0.701–3.932)	11 (5.1)	25 (6.0)	1.84 (0.775–4.184)
>10	56 (23.5)	34 (7.6)	8.965 (5.030–16.283)	44 (20.6)	31.0 (7.3)	7.57 (4.192–13.901)
Years of smokeless tobacco						
Never	123 (51.7)	364 (80.9)	Reference	108 (50.3)	341 (80.6)	Reference
<10	46 (19.3)	40 (8.9)	7.638 (4.368–13.535)	47 (21.9)	40 (9.5)	7.36 (4.254–12.945)
>10	69 (29.0)	46 (10.2)	9.528 (5.694–16.299)	60 (27.8)	42 (10.0)	9.09 (5.367–15.693)
Alcohol frequency						
Never	98 (41.2)	294 (65.3)	Reference	91 (42.3)	278 (65.8)	Reference
Occasional	48 (20.2)	98 (21.8)	1.41 (0.875–2.303)	42 (19.5)	92 (21.7)	1.35 (0.824–2.215)
Daily	92 (38.7)	58 (12.9)	5.69 (3.518–9.220)	82 (38.2)	53 (12.5)	5.18 (3.214–8.468)
Vegetables (times/week)						
>3	184 (77.3)	401 (89.1)	Reference	163 (75.8)	377 (89.2)	Reference
<3	54 (22.7)	49 (10.9)	2.96 (1.766–4.985)	52 (24.2)	46 (10.8)	2.77 (1.665–4.648)
Mouth rinsing						
Daily	69 (29)	88 (19.15)	Reference	55 (25.8)	82 (19.4)	Reference
Occasional	169 (71)	362 (80.85)	0.80 (0.511–1.256)	160 (74.2)	341 (80.6)	0.85 (0.533–1.366)
History of chronic trauma						
Absent	223 (93.7)	430 (95.6)	Reference	200 (93.1)	403 (95.2)	Reference
Present	15 (6.3)	20 (4.4)	3.00 (1.287–6.996)	15.0 (6.9)	20 (4.8)	3.24 (1.408–7.352)

IPTW: Inverse probability of treatment weighting analysis; OR: Odds ratio

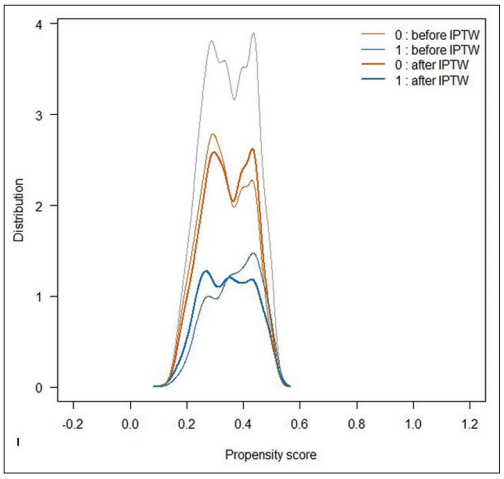

Figure 2: Balance in the case and control groups before and after inverse probability of treatment weighting

serve as justification for smokers who have not smoked for a long time to strongly consider giving up, if not completely abstaining from smoking to prevent oral cavity cancer.[29]

Among the smokeless tobacco users, the risk was significantly higher even among those who chewed tobacco for <10 years (OR = 7.36; CI: 4.25–12.94). These findings align with a nested case–control study conducted in Trivandrum, India in 2008 where the risk of oral cancer was higher in smokeless tobacco users of 20–40-year duration (OR = 7.5; CI: 5.0–11.4).[9] The mechanical trauma caused by chewing or holding smokeless tobacco products can lead to the formation of precancerous lesions, providing a fertile ground for malignant transformation over time.[30] In addition, synergistic interactions between smokeless tobacco use and other risk factors, such as alcohol consumption further escalate the risk of oral cancer development.

Gathering information about alcohol consumption can be challenging because it tends to vary more than smoking history, which is generally consistent over time.[31] Daily consumption of alcohol also significantly (OR = 5.18; CI: 3.21–8.46) contributed to the risk of oral cancer in the present study. There is no clear explanation for this finding, but one possibility is that the mucosa of the upper aerodigestive tract may become more susceptible to malignant transformations because of repeated exposure to salivary AA, a carcinogen.[30,31] Drinking 0.5 g of pure alcohol per kilogram of body weight can easily reach concentrations of higher than 40–50 μM, which is typically the threshold at which the direct carcinogenic or mutagenic effects of AA in saliva are observed.[32] Consequently, compared to drinking larger amounts over fewer instances, frequent social amount drinking may lead to prolonged exposure to the direct carcinogenic activity of AA.

Usage of ill-fitting dentures has frequently been implicated in the development of cancer as a result of chronic mucosal insult.[33] Continuous mechanical irritation has been suggested to damage DNA and may eventually lead to the development of cancer. The elevated activity of

poly-ADP-ribose polymerase in chronic trauma cases has demonstrated this.[34] Using multivariable regression, it was possible to find a significant association between the history of chronic mucosal trauma and oral cancer risk (OR = 3.2; CI: 1.40–7.35) even though the risk did not reach a significant level in bivariate analysis. Denture sores were found to be significantly correlated with the risk of oral cancer in a study by Jain *et al*.[33] A significant risk factor that requires more research involves continual trauma from a sharp cusp or an ill-fitting denture combined with poor oral hygiene.

One of the limitations of a case–control research is the utilization of closed-ended questionnaires for data collection, which precludes the collection of supplementary information on variables linked to cancer of the oral cavity. However, limiting the information sought for a small number of specific factors and avoiding overwhelming participants with extensive questionnaires, allowed us to minimize recall bias.[13] The propensity scores calculated by IPTW used in the present study allow dimension reduction given multiple confounders even in the presence of interactions and nonlinearities,[15] which is a major strength of the study compared to previous similar studies. The present study showed a lack of any significant interaction between smokers, smokeless tobacco users, and alcohol consumers or those with chronic trauma. The complexity of detecting any kind of interaction between these variables may require larger sample sizes to understand the combined effects of these variables. Nevertheless, these factors remain significant independently and highlight the need for public health initiatives aimed at preventing smoking and chewing tobacco, as well as reducing alcohol consumption. The public needs to be informed about the increased risk of oral cancer linked to chewing, smoking, and a combination of tobacco and alcohol consumption. The implementation of tobacco control legislation, as well as mass media communications, has resulted in a decrease in tobacco use.[13] However, the growing popularity of highly carcinogenic tobacco products comprising areca nut, combined with evasion of their bans, is increasing oral cancer incidence. Future research should be directed toward the carcinogenic potential of these products. Tobacco habits have shown a declining trend in India[35] mainly due to the increasing awareness of its ill effects and local legislations. However, the results of this study are unlikely to vary, given the role of these established risk factors. The increasing prevalence of oral cancer among nontobacco users' needs further exploration.[36] Chronic trauma due to ill-fitting dentures is a significant but often overlooked risk factor for oral cancer. Clinicians play a critical role in preventing this risk by ensuring proper denture fit, providing patient education, and monitoring for precancerous changes. Public health policies should focus on improving access to dental care, raising awareness, and establishing preventive measures for high-risk populations, particularly the elderly and socioeconomically disadvantaged.[37] Further research among these populations will help channelize the resources in an efficient manner for public health interventions.

Conclusion

To effectively prevent and control oral cancer, cessation of tobacco and alcohol use remains crucial, given the relatively low survival rates of patients diagnosed with the disease. In addition to quitting tobacco use, another important risk factor for oral cancer is a history of chronic trauma in the mouth. It is essential to see a dentist regularly and to be aware of the importance of self-examining your oral cavity to prevent disease in its early stages. Frequent dental checkups to detect oral cavity risk factors, such as an ill-fitting denture, ongoing trauma, or any early lesions, are easy steps that can significantly reduce the threat of oral cancer.

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Conflicts of interest

There are no conflicts of interest.

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Annexure 1

Annexure Table 1: STROBE Statement—Checklist of items that should be included in reports of case-control studies

	Item No	Recommendation	Page No
Title and abstract	1	(a) Indicate the study's design with a commonly used term in the title or the abstract	1
		(b) Provide in the abstract an informative and balanced summary of what was done and what was found	1
		Introduction	
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	1,2
Objectives	3	State specific objectives, including any prespecified hypotheses	2
		Methods	
Study design	4	Present key elements of study design early in the paper	2
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	2
Participants	6	(a) Give the eligibility criteria, and the sources and methods of case ascertainment and control selection. Give the rationale for the choice of cases and controls	2
		(b) For matched studies, give matching criteria and the number of controls per case	NA
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	2,3
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	2,3
Bias	9	Describe any efforts to address potential sources of bias	3
Study size	10	Explain how the study size was arrived at	2
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	2,3
Statistical methods	12	(a) Describe all statistical methods, including those used to control for confounding	3
		(b) Describe any methods used to examine subgroups and interactions	3
		(c) Explain how missing data were addressed	NR
		(d) If applicable, explain how matching of cases and controls was addressed	NR
		(e) Describe any sensitivity analyses	NR
		Results	
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	2
		(b) Give reasons for non-participation at each stage	NA
		(c) Consider use of a flow diagram	NA
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	8
		(b) Indicate number of participants with missing data for each variable of interest	NA
Outcome data	15*	Report numbers in each exposure category, or summary measures of exposure	5
Main results	16	(a) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	6
		(b) Report category boundaries when continuous variables were categorized	NA
		(c) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	NA
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	NA
		Discussion	
Key results	18	Summarise key results with reference to study objectives	11
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	4,5
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	5,6
Generalisability	21	Discuss the generalisability (external validity) of the study results	7
		Other information	
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	7

*Give information separately for cases and controls.