ALCOHOL AS A RISK FACTOR IN HNC, AN ENORMOUS TOLL ON THE LIVES AND COMMUNITIES

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ABSTRACT

INTRODUCTION

Head and neck cancer (HNC) is the seventh most common cancer in the world. After smoking, alcohol consumption is a strong associated risk factor for HNC. Alcohol consumption has negative impact on health and social consequences. The existing literature shows that alcohol consumption along with smoking is an important risk factor for HNC.

MATERIAL AND METHODS

In this retrospective analysis of 12-years from 2001 to 2012, total 9,950 patients of histopathologically proven head and neck cancers, were included in the study. The patients were analyzed for their association with alcohol as an associated risk factor along with smoking in different HNC subtypes.

RESULTS

Male to female ratio was 7:1 and median age of presentation was 54-years. 59% patients were alcoholics (alcoholics only+ smokers as well as alcoholics), out of which 6% were alcoholics only and the strength of association differed between different HNC subtypes; maximum for oropharyngeal group (35%), and 7.6%, 5% and 10% for oral cavity, hypopharynx and larynx respectively. Further evaluation has shown that total smokers (smokers only+ smokers as well as alcoholics) were 89%, out of which 36% were smokers only. At last follow-up, no evidence of disease (NED) was seen in 15% patients among total alcoholics and in 20% patients among non-alcoholics.

CONCLUSION

The present retrospective study of HNC patients at a tertiary care centre over a period of twelve years concluded the strong association of alcohol consumption with HNC and its different subtypes. Alcohol problems have an enormous toll on lives and communities of the developing nations particularly in the populous regions of the world (e.g. in China and India). There is a need to raise awareness among the population about the negative implications of alcohol consumption and to minimize them.

KEYWORDS

Alcohol, smoking, Risk factor, Oropharynx, Head and neck cancer, Association, Lifestyle, Carcinogen, Acetaldehyde, HNC subtypes, Drunkenness.

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INTRODUCTION: As per World Health Organization, the global data about the people who consume alcoholic beverages are around 2 billion and out of them 76.3 million are with diagnosable alcoholic disorders and the global burden related to alcohol consumption, both in terms of mortality and morbidity is substantially high throughout the world.[1] HNC is the seventh most common type of cancer in the world and constitute 5% of the entire cancers worldwide. The global burden of HNC accounts for 6,50,000 new cases and 3,50,000 deaths worldwide every year and a major proportion of regional malignancies in India.[2-4] Alcohol consumption has health and social consequences via intoxication (drunkenness), alcohol dependence and other biochemical effects of alcohol. Alcohol has consistently been related to the risk of cancer of the oral cavity, pharynx,

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larynx and hypopharynx. As per WHO Global Status Report on Alcohol 2004, the relative risk for mouth and oropharynx cancers is 1.45, 1.85 and 5.39 for category I to III alcohol users respectively.[1] More than 70% of squamous cell carcinoma of the head and neck are estimated to be avoidable by lifestyle changes, particularly by effective reduction of exposure to well-known risk factors such as tobacco smoking and alcohol drinking.[4-6] In this large retrospective analysis of 9,950 of head & neck cancer (HNC) patients spanning over 12-years (2001-12) were analyzed for different risk factors of HNC and its subtypes. It was observed that after smoking, alcohol consumption was the strong and independent risk factor responsible for increased risk of HNC overall. Among all the patients of HNCs, men were predominant. Most of the patients i.e. 92.3% presented as locally advanced head and neck cancer and most common presenting complaints observed was dysphagia (37%). Out of all the sub groups, oropharyngeal HNC group (35%) was most strongly associated with alcohol consumption. In this retrospective study of 9,950 patients, we have attributed the strong association of HNC with alcohol as 59% of these HNC patients were alcoholics.

AIMS AND OBJECTIVE: A retrospective analysis of 12-years from the year 2001 to 2012, of head and neck cancer patients attending Regional Cancer Centre, PGIMS Rohtak was done. Total numbers of cancer patients seen in a span of 12-years were26, 295, out of which 9,950 patients were of head & neck cancer (HNC). These patients were retrospectively analyzed for their association with alcohol as an associated risk factor in different HNC sub-types.

Sl. No.	Clinical Presentation	Total number of patients (n=9950)	Percentage of patients (%)
1	Bleeding from mouth	63	0.63
2	Hoarseness of voice	944	9.48
3	Dysphagia	3655	36.7
4	Odynophagia	1876	18.9
5	Ulcerative growth in mouth	585	5.87
6	Throat pain	343	3.44
7	Neck/cervical swelling	1843	18.5
8	Dyspnoea	160	1.60
9	Trismus	185	1.85
10	Headache/ earache	232	2.33
11	Nasal Obstruction/discharge	64	0.64

Table 1: Clinical presentation of HNC patients (n=9950)

Age groups (years)	Number of HNC patients	Percentage of patients (%)
<20	40	0.4
21-30	241	2.4
31-40	1127	11.3
41-50	2783	27.9
51-60	3091	31
61-70	2055	20.6
>70	613	6.2

Table 2: Age-wise distribution of HNC patients (n=9950)

OBSERVATIONS:

Stage-wise distribution	Number of HNC patients	Percentage of patients (%)
Stage I	180	1.8
Stage II	591	5.9
Stage III	2844	28.6
Stage IV	6335	63.7

Table 3: Stage-wise distribution of HNC patients (n=9950)

Sl. No.	HNC Sub-type		Number of alcoholics (n=9950) & Percentage (%)	Patient with NED status at last follow-up (alcoholic group)
1.	Oral Cavity (Lower lip, Tongue, Floor of mouth, Gingiva, Palate, Retromolar trigone, Upper lip & Buccal mucosa)		762(7.6%)	101
2.	Oropharynx	(Soft Palate & Lateral, Posterior Pharyngeal wall except base tongue & Tonsil)	92(0.9%)	8
		Base Tongue	2302(23%)	313
		Tonsil	1075(11%)	163
3.	Nasopharynx		33(0.3%)	5
4.	Hypopharynx	Post Cricoid	251(2.5%)	43
		Pyriform fossa	226(2.3%)	33
5.	Larynx	Supraglottic	907(9%)	197
		AE fold	23(0.2%)	9
		Epiglottis	58(0.6%)	17
		Vocal Cord	31(0.3%)	11
6.	Sinonasal (Maxilla, Nasal cavity)		51(0.5%)	4

7.	Salivary Gland	43(0.4%)	6
8.	EAC	7(0.1%)	0
	Total alcoholic patients (Out of total HNC patients, n=9950)	**5861(59%)**	**910**

Table 4: Alcoholic population with different HNC sub-types (n=9950)

Sl. No.	Total HNC patients	Total smokers (smokers only + smokers as well as alcoholics)	Smokers only	Total alcoholics (alcoholic only + smokers as well as alcoholics)	Alcoholic only	Alcoholic and smokers
1	9950	8859 (89%) (3605+5254)	3605 (36%)	5861 (59%) (607+5254)	607 (6%)	5254 (53%)

Table 5: Total smokers and alcoholics in HNC population (n=9950)

Sl. No.	Category of the Patient	Patient with NED status at last follow-up & percentage (%)
1	Non-alcoholic HNC patients	815(20.0%)
2	Alcoholic HNC patients	910(15.5%)
3	Total HNC patients of all sub-types	1725(17.3%)

Table 6: NED status of HNC Patients at last follow-up

RESULTS: This is a hospital-based retrospective study of 12-years participating all patients in Regional Cancer Centre, PGIMS Rohtak from the year 2001 to 2012. Total 26,295 cancer patients were analyzed, out of which, 9,950(37.8%) patients were of head & neck cancer (HNC), with a predominance of male population as 8,686(87%); and females were 1,264(13%). The male to female ratio was 7:1.

It was observed that most common age of presentation was between 51-60 years (3,091 patients) and next common age-group was 41-50 years (2,783 patients) and median age of presentation was 54-years. Most of the patients i.e. 92.3% presented as locally advanced head and neck cancer (LAHNC Stage III & IV). It was also observed that most common presenting complaints were dysphagia 3,655 (37%), odynophagia 1,876(19%) and cervical swelling 1,843(18.5%).

In this hospital-based retrospective analysis of 9,950 patients of HNC spanning a period of 12-years, 59% patients were total alcoholics (alcoholics only+ smokers as well as alcoholics), out of which 605 (6%) were alcoholics only. Further evaluation has shown that total smokers (smokers only+ smokers as well as alcoholics) were 89%, out of which 3605 (36%) were smokers only.

The presented data have shown a strong association of HNC with alcohol and hypothesized the effect of socioeconomic position on HNC in third world countries, which is in part explained by differential patterns of alcohol consumption across social strata. It was observed that the strength of the association, however, differed between different HNC-subtypes; i.e. oropharyngeal HNC group (35%) was most strongly associated with alcohol consumption, while oral cavity, hypopharynx and larynx HNC group contributed to 7.6%, 5% and 10% respectively. It was also observed that only 910 out of 5861 (15.5%) total alcoholic HNC patients (alcoholics only+ smokers as well as alcoholics) were having NED status at last follow-up, while 815 out of 4089 (20%) non-alcoholic HNC patients were having NED status at last follow-up.

In this analysis on different risk factors of HNC and its subtypes, we have attributed the association of HNC with smoking and alcohol in 89% smokers and 59% alcoholics respectively and observed that after smoking, alcohol consumption was the strong associated risk factor responsible for increased risk of HNC overall.

DISCUSSION: Head and neck cancer (HNC) includes several malignancies that originate in the paranasal sinuses, nasal cavity, salivary glands, oral cavity, nasopharynx, oropharynx, hypopharynx and larynx.[2] "Head and Neck Cancer" usually refers to neoplasms arising from below the skull base to the region of thoracic inlet. HNC is the seventh most common type of cancer in the world and constitute 5% of the entire cancers worldwide. The global burden of HNC accounts for 6,50,000 new cases and 3,50,000 deaths worldwide every year and a major proportion of regional malignancies in India.[2-4] According to hospital based cancer registries in India, head and neck cancers account for 29.8 to 50.4% of all cancers in males and 11.4 to 21.6% of all cancers in females. The incidence of HNC in our institute in past 20-years constitutes 30-35% of all the malignancies.[5,6] Epithelial carcinomas of head and neck arise from the mucosal surfaces and typically are squamous cell in nature and these squamous cell carcinomas (SCC) account for more than 90% of HNCs.[2]

How does Alcohol Increase the Risk of Cancer?: Alcohol consumption, tobacco and dietary factors, are established risk factors for HNC originating from the oral cavity, pharynx, and larynx, and are likely to be differentially associated with risk of different HNC-subtypes.[2]

The International Agency for Research on Cancer (IARC /Centre International de Recherché sur le Cancer) of the World Health Organization has classified alcohol as a Group 1 carcinogen (carcinogenic to humans). Its evaluation states, "There is sufficient evidence for the carcinogenicity

of alcoholic beverages in humans…. alcoholic beverages are carcinogenic to humans (Group 1)."[1] IARC classifies alcoholic beverage consumption as a cause of oral cavity, and pharynx cancers.[7]

Based on extensive reviews of research studies, there is a strong scientific consensus of an association between alcohol drinking and several types of cancer.[2,4,8] In its report on carcinogens, the National Toxicology Program (NTP) of the US Department of Health and Human Services lists consumption of alcoholic beverages as a known human carcinogen. The research evidence indicates that the more alcohol a person drinks; particularly the more alcohol a person drinks regularly over time, not the type of alcoholic beverage, higher is the risk of developing an alcohol-associated cancer. Based on data from 2009, an estimated 3.5 percent of all cancer deaths in the United States (about 19,500 deaths) were attributed to alcohol.[4,8,9]

Alcohol is the common term for ethanol or ethyl alcohol, a chemical substance found in beer, wine, and liquor (distilled spirits), as well as in some medicines, mouthwashes, household products, and essential oils (scented liquids taken from plants). Alcohol is produced by the fermentation of sugars and starches by yeast.

The main types of alcoholic drinks and their alcohol content are as follows:[10]

- Beers and hard ciders: 3-7 percent alcohol.
- Wines, including sake: 9-15 percent alcohol.
- Wines fortified with liquors, such as port: 16-20 percent alcohol.
- Liquor, or distilled spirits, such as gin, rum, vodka, and whiskey, which are produced by distilling the alcohol from fermented grains, fruits, or vegetables: usually 35-40 percent alcohol (70-80 proof), but can be higher.

The Federal Government's Dietary Guidelines for Americans 2010 defines moderate alcohol drinking as up to one drink per day for women and up to two drinks per day for men. Heavy alcohol drinking is defined as having more than three drinks on any day or more than seven drinks per week for women and more than four drinks on any day or more than 14 drinks per week for men.

As per WHO Global Status Report on Alcohol 2004, definition of drinking categories is: category I: for females not exceeding on average 0 to 19.99 g pure alcohol per day; for males not exceeding on average 0 to 39.99 g pure alcohol per day; category II: for females not exceeding on average 20 to 39.99 g pure alcohol per day; for males not exceeding on average 40 to 59.99 g pure alcohol per day; category III: for females on average 40 g pure alcohol and above per day; for males on average 60 g pure alcohol and above per day.[1] For comparison: a 75 cl. bottle of wine contains about 70 g of pure alcohol.

Researchers have identified multiple ways that alcohol may increase the risk of cancer, including: Alcohol probably acts as a local irritant especially in the anterior floor of mouth. It may also induce oral cancer via a coexisting malnutrition which in turn may depress immunological response.[2]

- Inside the body, alcohol (ethanol) is metabolized into a toxic chemical called acetaldehyde, which is a probable human carcinogen, which can cause cancer by damaging DNA and proteins and stopping our cells from repairing this damage. The International Agency for Research on Cancer (IARC) has classified acetaldehyde formed as a result of drinking alcohol as being a cause of cancer, along with alcohol itself. Acetaldehyde also causes liver cells to grow faster than normal. These regenerating cells are more likely to pick up changes in their genes that could lead to cancer. Ethanol is broken down mainly by the liver, but lots of other cell types can do this as well. Some of the bacteria that live in our mouths and the linings of our guts are also able to convert ethanol into acetaldehyde.[1-3,7]
- Generating reactive oxygen species (chemically reactive molecules that contain oxygen), which can damage DNA, proteins, and lipids through a process called oxidation.[2,3,7]
- Impairing the body's ability to break down and absorb a variety of nutrients that may be associated with cancer risk, including vitamin A; nutrients in the vitamin B complex, such as folate; vitamin C; vitamin D; vitamin E; and carotenoids. Alcohol use can lower the body's ability to absorb folate from foods, which is a vitamin that cells in the body need to stay healthy. This problem can be worse in heavy drinkers, who often do not get enough nutrients such as folate in their diet.[1-3]
- Category III/ Heavy alcohol user can add extra calories to the diet, which can contribute to weight gain in some people. Being overweight or obese is known to increase the risks of many types of cancer.[11]
- Increasing blood levels of oestrogen, a sex hormone linked to the risk of cancer.[3,7]
- Alcoholic beverages may also contain a variety of carcinogenic contaminants that are introduced during fermentation and production, such as nitrosamines, asbestos fibres, phenols, and hydrocarbons.[2,3]

In a review, Pöschl and Seitz list some possible mechanisms of alcohol as a carcinogen as 1) induction of CYP2E1, a member of the cytochrome P450 mixed-function oxidase system, which is an important enzyme for the conversion of ethanol to acetaldehyde;[12,13] 2) nutritional deficiencies; 3) interactions with retinoids; 4) alcohol and methylation; 5) alcohol and immune surveillance.[13] Purohita et al further evaluated some possible mechanisms of alcohol as a carcinogen as 1) production of acetaldehyde, which is a weak mutagen and carcinogen; 2) induction of cytochrome P450 2E1 and associated oxidative stress and conversion of procarcinogens to carcinogens; 3) depletion of S-adenosylmethionine and, consequently, induction of

global DNA hypomethylation; 4) induction of increased production of inhibitory guanine nucleotide regulatory proteins and components of extracellular signal-regulated kinase–mitogen-activated protein kinase signalling; 5) accumulation of iron and associated oxidative stress; 6) inactivation of the tumour suppressor gene BRCA1 and increased oestrogen responsiveness (primarily in breast); 7) impairment of retinoic acid metabolism.[14]

Other Risk Factors for HNCs: More than 70% of squamous cell carcinoma of the head and neck are estimated to be avoidable by lifestyle changes, particularly by effective reduction of exposure to well-known risk factors such as tobacco smoking and alcohol drinking.[6]

It was hypothesized in the literature[2] that 1) alcohol consumption and cigarette smoking are strongly, positively associated with HNC-risk, with multiplicative interaction; 2) alcohol and tobacco consumption are synergistic to each other; 3) these risks are different for oral cavity cancer (OCC), oro-/hypo-pharyngeal cancer (OHPC) and laryngeal cancer (LC).

The other factors are poor oral hygiene, nutritional deficiencies, chronic mechanical irritation in case of oral cavity cancers. Squamous cell carcinoma may also develop from premalignant conditions such as submucous fibrosis, which in return may be a result of tobacco or betel nut chewing.

There is increasing evidence that viruses may contribute to causation of head & neck cancers. Infection with the Epstein-Barr virus is clearly associated with nasopharyngeal carcinoma, and DNA from the human papilloma virus has also been detected in head and neck cancer.[15]

Epidemiologic research shows that people who use both alcohol and tobacco have much greater risks of developing cancers of the oral cavity, pharynx and larynx than people who use either alcohol or tobacco alone. In fact, for oral and pharyngeal cancers, the risks associated with using both alcohol and tobacco are multiplicative; that is, they are greater than would be expected from adding the individual risks associated with alcohol and tobacco together.[2,3]

In recent years, evidence has accumulated to support the hypothesis that vegetable and fruit consumption is inversely associated with risk of HNC overall. Meta-analysis by Dhull et al. compared the people who consumed ~1.5 portions of fruit and vegetables each day, with people consuming ~5.8 portions per day, had a relative risk of 0.71 of having HNC.[16]

Clinical Overview: The clinical manifestations of disease vary according to the stage and primary site of tumour. However, dysphagia, odynophagia, otalgia, hoarseness, mucosal ulceration, restricted tongue mobility, nasal obstruction, epistaxis, cranial nerve neuropathies and cervical lymphadenopathy are common presenting complaints. The advanced stages of all head and neck cancers have easily detectable signs and symptoms, including trismus, formation of fistulae etc.

Management of head and neck carcinoma has come a long way from the era of primitive surgery to advanced and state of the art treatment by Image Guided Radiation Therapy (IGRT). Similarly the diagnostic workup has evolved from the crude staging by only clinical methods to sophisticated and logically convincing biological imaging. History with emphasis on alcohol intake, smoking, tobacco chewing, general physical examination, examination of oral cavity, oropharynx, nasopharynx, laryngopharynx (direct & indirect laryngoscopy), examination of the lymph nodes and biopsy of any suspected areas is preliminary to confirm and stage HNC. Conventional radiographic tools such as plain radiographic studies have some role in the head and neck tumour evaluation and to rule out pulmonary metastatic disease. Computed tomography is useful in assessing the extent of bone invasion by the oral cavity tumours that are in the vicinity of mandible and maxilla. CT-MRI fusion is use to better define the target volume for Radiation treatment planning. Positron Emission Tomography (PET) and Single Photon Emission CT (SPECT) are considered new tools in the diagnostic work-up and evaluation of patients with HNC.[17] The uniformity and prognostication of HNSCC is agreed throughout the world with AJCC Staging.[18]

Over the past few years, oncology has rapidly advanced in various fields especially surgery, radiotherapy and chemotherapy. However for local cure, surgery and radiotherapy are the two standard curative modalities and have shown comparable cure rates in early stages (Stage-1 and II) of most squamous cell carcinomas of head and neck. In most cases of LAHNC, usually combined modality with surgery, radiotherapy alone or concomitant chemoradiation is preferred.

Alcohol consumption is a major risk factor for certain head and neck cancers, particularly cancers of the oral cavity (excluding the lips), pharynx (throat), and larynx (voice box). People who consume 50 or more grams of alcohol per day (approximately 3.5 or more drinks per day) have at least a two to three times greater risk of developing these cancers than non-drinkers.[1-4,13,14] Moreover, the risks of these cancers are substantially higher among persons who consume this amount of alcohol and also use tobacco.

Our results are in accordance with the existing literature, showing alcohol consumption to be an strong associated risk factor for the development of HNC (Table 1-6).[1-3] Alcoholic beverages and acetaldehyde, the main metabolite of ethanol, are classified as a class I carcinogen. There is a probability that alcohol after being metabolized acts both directly and indirectly in HNC carcinogenesis.[2,3]

Our present retrospective study confirms a multiplicative interaction between alcohol consumption in HNC overall.[1-3,13,14] The interaction effect between alcohol consumption and smoking is biologically plausible, since alcohol can act as a solvent for carcinogens in cigarette smoke and make the mucosa more permeable for these carcinogens; as a result, the carcinogenic properties of both factors are likely to be enhanced in the presence of one another.[1-3,13,14] It

has been reported that HNC is much more common in smokers than in non-smokers and most common in males over 50 years of age.[19]

For HNC overall, increased risks were found for every exposure combination of alcohol consumption and cigarette smoking, mostly statistically significantly, compared to never smokers and abstainers. In literature, a statistically significant, positive, multiplicative interaction was found between alcohol consumption group and cigarette smokers.[1-3,13,14]

The major risk factors for head and neck squamous cell cancer (HNSCC) are tobacco smoking and alcohol consumption and withdrawal of these environmental carcinogens remains the focus for primary and secondary prevention. Smoking and alcohol are the major independent risk factors for head and neck cancer. Patients who continue to drink heavily after treatment for head and neck cancer have a significantly worse quality of life and continued drinking has a negative impact on survival.[1-3,13,14] The beneficial effects of quitting alcohol, on the risk of developing HNC, are only observed after more than 20-years, when the level of risk reaches that of non-drinkers.

CONCLUSIONS: In conclusion, the present retrospective study of large population of HNC patients spanning over 12-years, confirms the principal role of alcohol consumption as associated risk factor in HNC-carcinogenesis, as well as the differential associations with HNC-subtypes. As the existing evidence is largely based on retrospective analysis, this contributes to establish the association between alcohol consumption with risk of HNC overall and, more specifically different HNC-subtypes.

Alcohol is not an ordinary commodity. While it carries connotations of pleasure and sociability in the minds of many, harmful consequences of its use are diverse and widespread. As documented in the literature, alcohol problems exert an enormous toll on the lives and communities of many nations, especially those in the developing world, particularly in the populous regions of the world (e.g. in China and India). Research has shown that when extrapolating from historical trends, the role of alcohol as a major factor in the burden of disease will be increasing in the future and risky patterns in drinking especially among young people.

A global perspective on alcohol policy needs to acknowledge and take into account the characteristics, effects and consequences of alcohol use in different societies, and yet to focus and act on the public health goal which is to minimize the harm caused by drinking. Alcohol related burden is linked to at least two different dimensions of consumption: average volume and patterns of drinking. Thus, in order to avoid or reduce burden, both dimensions should be taken into consideration.

It is evident from the WHO report that there is a need for countries to develop national monitoring systems to keep track of alcohol consumption and its health and social consequences. This would be particularly useful in raising awareness among the general public and policy-makers of the serious implications that alcohol use have within the public health domain. It is now the responsibility of Governments worldwide and concerned citizens to encourage healthy debate and formulate effective public health-oriented countermeasures in order to minimize the harmful social and health consequences from alcohol use.

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