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Impact of alcohol on coronary heart disease in Indian men

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ABSTRACT

Background: Moderate alcohol consumption is known to be protective against coronary heart disease (CHD). However, the INTERHEART study, a case–control study of acute myocardial infarction (MI) patients, revealed that alcohol consumption in South Asians was not protective against CHD. We therefore planned to study cardiovascular risk factor and CHD prevalence among male alcohol users as compared to age matched lifetime abstainers.

Methods: The subjects for this study were recruited from a cross-sectional survey carried out among employees and their family members aged 20–69 years in 10 medium-to-large industries from diverse sites in India, using a stratified random sampling technique. Information on education, behavioral, clinical and biochemical risk factors of CHD and alcohol use was obtained through standardized instruments. CHD diagnosis was based on Rose Questionnaire or a prior physician diagnosed CHD.

Results: A total of 4465 subjects were present or past alcohol users. The mean age of alcohol users and lifetime abstainers was 42.8 ± 11.0 years and 42.8 ± 11.1 years, respectively ($p = 0.90$). Systolic blood pressure and diastolic blood pressure were significantly higher in alcohol users (128.7 ± 17.6 mmHg/80.1 ± 11.3 mmHg) as compared to lifetime abstainers (126.9 ± 15.9 mmHg/79.5 ± 10.3 mmHg, $p < 0.01$). Fasting blood sugar in alcohol users (98.7 ± 30.5 mg%) was also significantly higher than lifetime abstainers (96.6 ± 26.0 mg%, $p < 0.01$). Total cholesterol was lower in alcohol users (179.1 ± 41.1 mg%) as compared to lifetime abstainers (182.7 ± 38.2 mg%, $p < 0.01$). HDL cholesterol was higher in alcohol users (42.9 ± 10.8 mg%) as compared to lifetime abstainers (41.3 ± 10.0 mg%, $p < 0.01$). Body mass index (BMI) was lower in alcohol users as compared to lifetime abstainers (22.7 ± 4.1 kg/m^2 vs. 24.0 ± 3.3 kg/m^2, $p < 0.001$). Tobacco use was significantly higher in alcohol users (63.1% vs. 20.7%). The odds ratio (OR) of having CHD after adjusting for tobacco use, BMI and education was 1.4 (95%CI 1.0–1.9) in alcohol users as compared to controls. The OR was 1.2 (95%CI 0.8–1.6) in occasional alcohol users, 1.6 (95%CI 1.0–2.2) in regular alcohol users and 2.1 (95% CI 1.1–3.0) in past alcohol users as compared to controls.

Conclusion: We did not observe an inverse (protective) association between alcohol intake and the prevalence of CHD. In contrast, our study indicated an association in the reverse direction, suggesting possible harm of alcohol for coronary risk in Indian men. This relationship needs to be further examined in large, prospective study.

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1. Introduction

Moderate alcohol consumption is believed to be protective against coronary heart disease (CHD). A 'J'- or 'U'-shaped relation between alcohol consumption and CHD is widely reported, with the lowest CHD and mortality rates being seen among light-to-moderate drinkers and higher rates among abstainers and heavy drinkers [1–4]. However, this hypothesis is challenged by a meta-analysis that demonstrated the lack of benefit of alcohol in preventing CHD [5]. In addition ethnicity [6], gender [1–4], type of alcoholic beverage [7,8] and pattern of alcohol intake [9] also influence the relation between alcohol and CHD in various studies. In a prospective study in African Americans no J-shaped curve was found in the relationship between average volume of alcohol consumption and mortality for male or female African Americans. Instead, there was no beneficial effect and mortality increased with increasing average consumption of more than one drink a day [6]. Limited data in Indians from the INTERHEART study, a case–control study of patients with incident acute myocardial infarction (MI), revealed that in South Asians alcohol consumption was not protective against CHD. Alcohol intake was associated with a higher odds ratio of MI particularly among Indians [10].

We therefore studied the effects of alcohol on cardiovascular risk profile and prevalence of CHD among present and past alcohol users and compared them with lifetime abstainers who participated in a surveillance program for CVD risk factors across 10 industrial sites in India.

2. Methods

2.1. Study setting

This study was part of cross-sectional data from a surveillance for CVD risk factors conducted in 10 industries across India. The detailed methodology of the study has been published elsewhere [11]. In brief 10 medium-to-large industries (defined as industries employing 1500–5000 people) in the organized sector were selected from different sites spread across India, from both public and private sectors, based on their willingness to participate in the study and proximity to an academic medical institution. All of the employees and their family members between the ages of 20 and 69 years were eligible to be included in the survey. At each participating center, detailed data were obtained from randomly selected employees and their eligible family members ($n = 2000$ at each center). Further, from this group, we chose 1000 individuals per center by stratified random subsampling for biochemical analysis.

A total of 19,973 (11,898 men) individuals consented to participate in this survey in the age group of 20–69 years at a response rate of 87.6%.

For the purpose of this particular study we investigated the impact of alcohol use on CHD risk factors and on presence of CHD among the population surveyed. Only males surveyed were analyzed for effect of alcohol intake on CHD as the number of alcohol users among women was very small.

2.2. Alcohol consumption data

Present or past consumption of alcohol was ascertained by a structured interview wherein all the participants were asked "Do you use alcoholic beverages?" The answers were reported as never used/currently using alcohol regularly/currently using alcohol occasionally/used alcohol in the past. Regular alcohol consumption was defined as once or more in a week; occasional was less than once a week; past alcohol consumption was defined as those who had quit at least 6 months ago. Current and past alcohol users were further enquired about the type, frequency and amount per occasion of alcohol consumed. Binge drinking was defined as ≥4 standard drinks at one time [12]. One standard drink for this study was defined as 30 ml of spirits, 285 ml of regular beer or 120 ml of wine [13]. Computer generated random number was used to identify an age matched (±2 years) lifetime abstainer for each case in the database.

2.3. Coronary heart disease definition

CHD diagnosis was based on Rose Angina Questionnaire or a prior physician diagnosed MI or angina.

2.4. Measurement of other covariates

Data related to the demographic profile, educational status (in terms of highest education level attained) and leisure time physical activity was recorded at the baseline. Lifetime exposure to tobacco was assessed on the basis of self-reported use of chewable or smoked tobacco. Body mass index, systolic and diastolic blood pressures were recorded by standardized methods. Fasting blood sugar, total cholesterol, HDL cholesterol, LDL cholesterol and triglycerides levels were measured at baseline.

2.5. Quality control measures

To ensure the accuracy, completeness, and comparability of blood pressure and anthropometric measurements and of interviewee responses across the 10 study sites, several quality control measures were included in the study protocol. Greater details are available in the methodology paper published earlier [11]. Briefly, the study followed a common study protocol and a manual of operation. In all participating centers standardized measurement techniques and structured pre-tested pro forma were used by trained study staff. Ten percent of the biochemical samples were reanalyzed at the central coordinating center laboratory. The analysis of the results of this 10% sample from all participating sites yielded <5% coefficient of variation between the central coordinating laboratory results and the individual laboratory results. The questionnaire for alcohol consumption was tested for face validity in a pilot study and in another large industrial survey [14]; however test–retest reliability was not performed due to logistic constraints.

2.6. Statistical methods

Baseline variables were compared in alcohol users and non-users and the data are summarized in mean and standard deviation. Independent '*t*' test was used to compare the mean difference in baseline variables between cases and control subjects. The frequency of outcome variables of interest, separately in cases and controls and its subgroups, are summarized in proportions. Multivariate logistic regression method was used to identify risk estimates (odds ratio and its 95% confidence interval). All analysis was performed in SAS 8.02 version (SAS Institute Inc., Cary, NC, USA) using Windows operating system.

3. Results

Of the 11,898 men surveyed 4465 (38%) reported consuming alcohol currently or in the past. Among them 2293 reported occasional alcohol consumption, 1852 reported regular alcohol consumption and 320 reported past consumption. Local (56.8%) or branded spirit (52.8%) was the most common type of alcohol used with very low rates of wine (0.3%) or beer consumption (2.8%). Addi-

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Table 1
Characteristics of alcohol users and non-users.

	Alcohol use ($N = 4465$)	Non-users ($N = 4465$)	p value
Age (years)	42.8 ± 11.0	42.8 ± 11.1	0.9
Systolic BP (mmHg)	128.7 ± 17.6	126.9 ± 15.9	<0.01
Diastolic BP (mmHg)	80.1 ± 11.3	79.5 ± 10.3	<0.01
Fasting blood sugar (mg%)	98.7 ± 30.5	96.6 ± 26.0	<0.01
Total cholesterol (mg%)	179.1 ± 41.1	182.7 ± 38.2	<0.01
HDL cholesterol (mg%)	42.9 ± 10.8	41.3 ± 10.0	<0.01
Triglycerides (mg%)	136.44 ± 83.82	125.93 ± 69.18	<0.001
Tobacco use (%)	63.1	20.7	<0.001
Leisure time physical activity (%)	34.0	32.9	0.28
Waist circumference (cm)	84.5 ± 11.5	87.7 ± 9.2	<0.001
BMI (kg/m^2)	22.7 ± 4.1	24.0 ± 3.3	<0.001

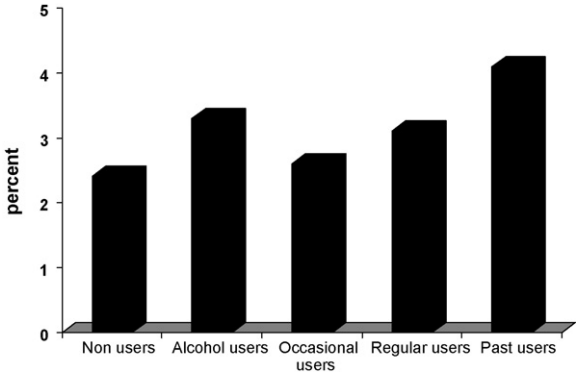

Fig. 1. CHD prevalence in different groups.

tionally, information on patterns of alcohol intake was available in 54% of alcohol users and of them 56.8% reported binge drinking.

3.1. Alcohol consumption and cardiovascular risk factors

The mean age of the alcohol users was 42.8 ± 11.0 years. The characteristics of alcohol users and non-users were as mentioned in Table 1. In unadjusted analyses, alcohol consumption was associated with higher systolic and diastolic blood pressure and HDL cholesterol and lower total cholesterol. Alcohol users had much higher tobacco consumption and lower BMI and waist circumference. Alcohol consumption was associated with higher fasting blood sugar. Leisure time physical activity was similar in alcohol users and non-users.

3.2. Alcohol consumption and CHD

Prevalence of CHD among alcohol users and non-users was 3.3% and 2.4%, respectively. Among the subgroups the prevalence of CHD was as in Fig. 1. The odds ratio of having CHD among alcohol users after adjusting for tobacco use, BMI and education was 1.4 (1.0–1.9, $p = 0.05$). Among the subgroups, occasional alcohol users had an odds ratio (OR) of 1.2 (95% CI 0.8–1.6, $p = 0.09$) of having CHD after adjusting for age, tobacco use, BMI and education. The OR was 1.6 (95% CI 1.0–2.2, $p = 0.06$) with regular alcohol consumption and 2.1 (95% CI 1.1–3.0, $p < 0.05$) in quitters as compared to age-matched controls after adjusting for age, tobacco use, BMI and education (Table 2). Among alcohol users the odd of CHD in light (<14 g/day), moderate (14–<28 g/day) and heavier (>28 g/day) alcohol users were 1.3 (95% CI 1.2–1.5, $p < 0.01$), 1.6 (95% CI 1.0–2.3, $p = 0.07$) and 2.0 (95% CI 1.2–2.6, $p < 0.05$), respectively after adjusting for age, tobacco use, BMI and education (Fig. 2). The odds of CHD among those consuming local spirits and those consuming branded spirits was 1.8 (95% CI 1.0–2.6, $p < 0.05$) and 1.5 (95% CI 0.9–2.2, $p = 0.07$), respectively after adjusting for age, tobacco use, BMI and education.

Table 2
Prevalence of CHD and odds ratio.

	Prevalence of CHD (%)	Odds ratio	p value
Non-users	2.4	1.0	
Alcohol users	3.3	1.4 (1.0–1.9)	0.05
Occasional users	2.6	1.2 (0.8–1.6)	0.09
Regular users	3.1	1.6 (1.0–2.2)	0.06
Past users	4.1	2.1 (1.1–3.0)	0.02
Type of alcohol			
Local spirits	3.6	1.8 (1.0–2.6)	0.06
Branded spirits	3.3	1.5 (0.9–2.2)	0.16

3.3. Alcohol consumption, tobacco use and CHD

We further analyzed odds of CHD in alcohol users by stratifying them as tobacco users and no tobacco users. The odds of CHD among alcohol and tobacco users was 2.9 (95% CI 1.8–4.0, $p = 0 < 0.05$), tobacco but no alcohol users was 2.4 (95% CI 1.9–2.9, $p < 0.01$), alcohol but no tobacco users was 1.7 (95% CI 1.1–2.3, $p < 0.05$) as compared to no tobacco and no alcohol users after adjusting for age, BMI and education (Fig. 3).

4. Discussion

Alcohol consumption in Indian men did not have an inverse association with CHD prevalence. On the contrary, the association was in the reverse direction suggesting possible harm. This trend was observed in alcohol users classified as occasional and regular as well as in light, moderate and heavier consumers of alcohol. Past users had a significantly higher prevalence of CHD, a well known phenomenon in the literature wherein individuals who develop illnesses like hypertension and CHD are known to reduce or give up their drinking habits [15]. On analysis of CHD prevalence among alcohol users by stratifying them as tobacco users and no tobacco users we observed that alcohol was associated with harm in both, with highest odds in those consuming both. Results of our study are

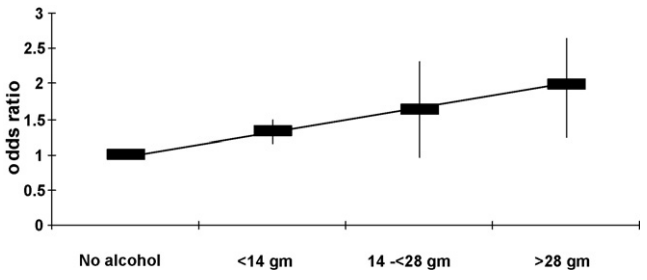

Fig. 2. Daily use of alcohol and risk of CHD.

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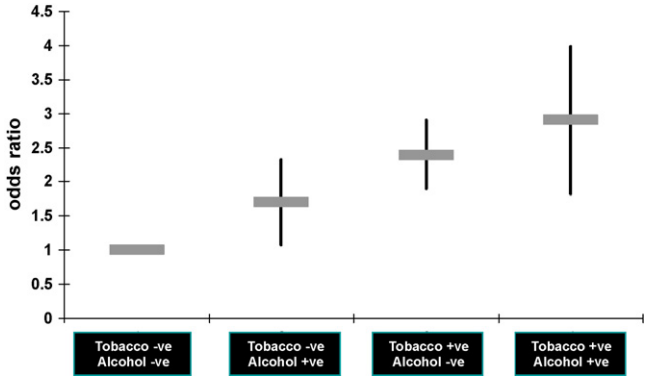

Fig. 3. Tobacco, alcohol and risk of CHD.

consistent with the INTERHEART study. This study reported that while alcohol use was protective against MI in the entire study population recruited from 52 countries around the globe, it was harmful in Indians. The OR of having MI was 1.6 (95% CI 1.2–2.3) among Indian alcohol users as compared to non-users [10]. Similar results have been reported in studies that included African Americans; like the CARDIA study where alcohol use was associated with higher prevalence of sub-clinical CHD [16]. Similarly a prospective cohort study among African Americans revealed no beneficial effect of alcohol and there was evidence of increased mortality with increasing average consumption of more than one drink a day [6].

A recent meta-analysis of 28 cohort studies by Corrao et al. revealed lower protective effect of alcohol than is generally assumed, especially in women and populations living out of the Mediterranean area [4]. Further, another meta-analysis by Fillmore et al. challenged the beneficial hypothesis of alcohol. Their contention being that controls in most prospective studies included past alcohol users and if such studies are eliminated, then overall survival in abstainers is similar to occasional/regular alcohol users [5]. These results are despite the fact that meta-analysis are inherently biased due to the phenomenon of fewer negative reports getting published as compared to positive associations.

The type of alcohol consumed in this study was different from previous reports with nearly half the men using locally brewed alcohol. The impact of using this form of spirit on CHD events is not known. However, the results in our study revealed similar impact of local and branded spirit on CHD.

The reasons for the lack of protective effect could be several. Firstly, there maybe heterogeneity in effect of alcohol on CHD in different ethnicities and the protective effect may be absent or more modest in populations other than that in Mediterranean or South Europeans. This could be hypothesized to be due to unfavorable variant of alcohol dehydrogenase which is known to impact the effect of alcohol on CHD [17]. Secondly drinking patterns may account for the difference in the results. In particular Mediterranean drinking patterns are characterized by the use of daily constant amounts of alcohol mainly in the form of wine which has been associated with protection against CHD as compared to irregular heavy or binge drinking that provides no favorable effect on CHD [9,18]. In this study a large proportion of individuals, in whom the pattern of drinking was available, reported binge drinking and thus the protective effect of alcohol was probably lost in them. Thirdly, alcohol intake in India is reported to be much higher among uneducated, poorer, socially disadvantaged and rural population [19]. Though we did adjust for some of these factors like education it was not possible to adjust for all. More recent data from India has shown the greater vulnerability of the poorer and less educated sections of the society to cardiovascular diseases [20,21] as is in the industrialized nations [22,23]. Finally, other residual confounders which co-vary with alcohol use and cannot be accounted for may also account for this difference.

The limitations of our study are one that it is cross-sectional in design and thus the results demonstrate associations but do not provide evidence for causality. Secondly the study population was mainly composed of industrial employees and may not be representative of the general population. Lastly, possibility of underreporting of alcohol use and also amount and frequency of alcohol consumption may be present due to its social unacceptability in certain segments of the society.

In conclusion, our study shows that alcohol use in Indian men, as practiced in this industrial population, is not protective against CHD rather it is associated with possible harm. This finding needs to be confirmed in a prospective study in the general population as it has important public health and policy implications in Indians who are at a high risk of developing CHD.

Acknowledgments

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