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Does Heavy Consumption of alcohol worsen the prognosis of Acute Myocardial Infarction? – An observational study undertaken in a rural South Indian Population

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Abstract
Although epidemiological evidence for the beneficial effects of low alcohol consumption on coronary artery diseases is strong, the impact of heavy drinking is less clear. There are plenty of evidences regarding the impact of alcohol consumption on acute myocardial infarction (AMI). The objective of the study is to determine the association of heavy alcohol consumption among patients with AMI with reference to Left Ventricular Ejection Fraction (LVEF), admitted at Rajah Muthiah Medical College Hospital, Annamalai University, Annamalai nagar, India. Adult inpatients to the hospital with clinical features suggestive of AMI were enrolled in this study. The total number of patients was one hundred. A detailed history was recorded and physical examination was carried out. Relevant investigations, i.e Electrocardiogram, Cardiac markers including Troponin-T, Creatine Phosphokinase (CPK-MB) and Echocardiogram were done. It was observed that the complexity of disease was significantly high in patients with history of heavy alcohol consumption. These patients were prone for high systolic and diastolic blood pressure, sinus tachycardia and high activity of CPK-MB and positive troponin-T. Captivatingly, most of the patients with a history of heavy alcohol consumption had reduced LVEF indicating the severity of Left ventricular (LV) dysfunction which was statistically significant. LV failure, with a poor prognosis, is strongly associated with LV dysfunction. These findings append to the already reputable fact that heavy alcohol consumption is a major risk factor for coronary artery disease and a predictor of poor prognosis of AMI.
Keywords: *Alcohol, Acute Myocardial Infarction, Creatine Phosphokinase, Troponin-T, Left Ventricular Ejection Fraction, Left Ventricular Dysfunction.*

Introduction

According to the World Health Organization's estimates, every year approximately six million people around the world develop myocardial infarction, and the lethal outcome occurs in over 25% of cases[1]. Myocardial infarction represents death of myocardial cells due to irreversible ischemia progressing to necrosis[1],[2]. Etiology of myocardial infarction is complex and still not completely elucidated[3], [4].

Alcohol use was implicated as a cause in >2.5 million deaths worldwide in 2010, primarily from injuries, cirrhosis, cardiovascular disease, and cancer, and was ranked the fifth most important risk factor contributing to the global burden of disease [5]. The relationship between alcohol consumption and acute myocardial infarction (AMI) is complex. There is now considerable evidence that episodic heavy drinking is associated with myocardial damage and is a risk factor for sudden cardiac death[6]. An episode of heavy drinking is associated with an increased risk of acute MI in the subsequent 24 hours, particularly in older individuals. Some studies have shown that heavy drinking, as well as binge drinking, can lead to AMI. The 'INTERHEART' study showed that episodes of binge drinking can lead to the onset of AMI and that especially in older people[7]. Age, smoking, hypertension, high cholesterol levels, diabetes mellitus and male sex are recognized as major risk factors for AMI [8]. Other risk factors include obesity, physical inactivity, dietary factors, positive family history and psychosocial factors. However, in some developing countries, the contribution of established myocardial infarction risk factors is not entirely known.

The potential biological mechanisms by which alcohol may predispose to AMI after ingestion have previously been reviewed [9]. When alcohol is consumed heavily in an episodic pattern, the withdrawal of the inhibitory effects of alcohol on platelet aggregation may create a rebound prothrombotic state[10]. This increases the risk of thrombotic occlusion that can lead to a heart attack or stroke. Episodic heavy drinking may also be associated with adverse changes in lipid profile such as an elevation in low density lipoprotein cholesterol (LDL) without protective increase in high-density lipoprotein cholesterol (HDL) observed with lower and more regular alcohol intake[11]. In addition, although alcohol ingestion lowers blood pressure within the first 4 hours after consumption, a significant elevation in blood pressure is observed at 20 to 24 hours, which may be mediated by vasoactive cytochrome P450,

ecosanoids, the concentration of which is affected immediately by alcohol consumption[12].

The present study is aimed to find out the relationship between the heavy alcoholics and the severity of AMI and its outcome compared with non-alcoholics AMI patients by assessment of left ventricular (LV) dysfunction.

Materials and Methods

A descriptive (non-experimental) study was undertaken at Rajah Muthiah Medical College, Annamalai University, Annamalai nagar, India after obtaining formal permission from the concerned authority. The diagnosis of MI was based on the history of acute and prolonged ischemic chest pain, characteristic electrocardiographic (ECG) changes, elevated creatine phosphokinase (CPK-MB) and positive Troponin-T within 12h after the onset of pain. All consecutive patients confirmed with the diagnosis of MI of all age groups admitted in the hospital were included in the study. The data regarding demographic, health and habitual status of patients were assessed by interview of the patients and supplemented from the patients' records. ECG and Color Doppler Echocardiography were done for all the patients. LV diameters and area were measured according to the criteria of American Society of Echocardiography[13]. Left Ventricular Ejection Fraction (LVEF) was estimated according to LV volumes evaluated by biplane method of disks (modified Simpson's rule). Routine biochemical investigations were done at the central diagnostic centre in the hospital. Serum CPK-MB was assayed by Immunoinhibition / Modified IFCC method. Blood pressure (BP) and heart rates (HR) were measured after a 5 to 10 minute rest using an automatic digital BP apparatus (Omron Digital HEM-907, Tokyo, Japan). Three measurements were made with 5 minutes interval and the average was recorded.

Patients with infection, inflammatory diseases, malignancy and congenital malformations of the heart or vessels or history of previous MI were excluded. Patients who drink more than 14 drinks in a week were considered as heavy alcoholics. The

patients were divided into two groups viz, alcoholics and non-alcoholics.

Statistical Analysis

Analysis was performed using the Statistical Package for Social Sciences (SPSS) Version 22.0 for descriptive analysis, frequency, mean, median and standard deviation and percentage. Student 't' test was performed between the two groups of alcoholics and non-alcoholics. Results were presented as means ± SEM and p value < 0.05 was regarded as statistically significant.

Results

In the present study, the total number of patients was hundred, in which forty nine of the study patients had the habit of consuming fourteen drinks per week for the last five or more years and fifty one had no habit of consuming alcohol. Majority of the patients i.e. 67% were hypertensive in alcoholic group whereas 53% were in non-alcoholic group. Diabetes was found to be as high as 69% of the alcoholic patients and 49% diabetic was found in non-alcoholic patients. The data regarding the symptoms, history, examinations, investigation and echocardiography are dissipated in Table 1.

Figure 1 shows the severe reduction in LVEF, suggesting severe LV dysfunction, as observed in the alcoholics (63%) than the non alcoholics (35%). Table 2 shows the results of the age, systolic BP, diastolic BP, pulse, CPK-MB activity and percentage of LVEF. Age was matched between the two groups. Systolic BP, diastolic BP and pulse were increased in the alcoholics than the non-alcoholics. The activity of CPK-MB of the group of alcoholics and non-alcoholics were significantly different. There was a significant reduction in the percentage of LVEF in alcoholics when compared to the non- alcoholics signifying that severe alcohol consumption has a negative impact on LV function.

Table 1: Distribution of study subject by different characteristics (n=100)

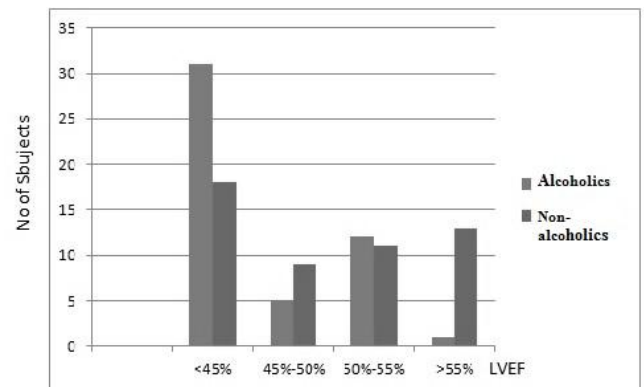|	Alcoholics (n= 49)		Non-alcoholics (n= 51)	
	Number of subjects	% of Subjects	Number of subjects	% of Subjects
Male	49	100	25	49
Female	-	-	26	51
History				
Chest pain	47	96	43	84
Syncope	34	69	25	49
Palpitation	25	51	17	33
Dyspnoea	19	39	13	25
Sweating	32	65	22	43
Vomiting	26	53	16	31
DM	34	69	25	49
HT	33	67	27	53
Family H/O IHD	26	53	16	31
Smoking	41	84	12	29
Investigations				
Troponin-T (Positive)	14	29	36	71

Figure 1: Distribution of Left Ventricular Ejection Fraction (LVEF) of study subjects

Table 2: Levels of physiological, biochemical and echocardiogram characteristics

S.No	Parameters	Alcoholics (n= 49)	Non alcoholics (n= 51)	t-value	p-value
1.	Age	56.67 ± 1.74	56.29 ± 1.69	0.197	0.876
2.	Systolic Blood Pressure (mm Hg)	143.10 ± 2.75	135.94 ± 3.58	1.577	0.118
3.	Diastolic Blood Pressure (mm Hg)	91.02 ± 1.63	88.58 ± 1.89	0.969	0.335
4.	Pulse	85.86 ± 2.18	83.04 ± 2.28	0.892	0.375
5.	Creatinine Phosphokinase-MB (IU/L)	89.00 ± 8.40	56.41 ± 4.43	3.467	0.001 *
6.	Ejection fraction (%)	42.14 ± 8.40	48.35 ± 1.13	-3.730	0.000 **

*represents significance at the level p < 0.05, **represents represents significance at the level p<0.001. Results were presented as means ± SEM

Discussion

The per capita alcohol consumption in India increased two folds between 2005 and 2016, according to the Global status report on alcohol and health 2018 released by the World Health Organization[14]. Indians consumed 2.4 litres of alcohol in 2005, which increased to 4.3 litres in 2010 and scaled up to 5.7 litres in 2016. The highest increase in alcohol consumption is expected in South-East Asia, with an increase of 2.2 litres in India alone, from 2005 to 2016. More than 3 million people died as a result of harmful use of alcohol in 2016. More than three quarters of those reported dead were men. Overall, the harmful use of alcohol causes more than 5% of the global disease burden. Of all deaths due to alcohol, 19% were due to cardiovascular diseases. According to the above report, in present study, out of one hundred subjects, 49 (100 %) men came under alcoholic groups whereas 25 (49%) men belong to non-alcoholic group, which indicate the prevalence of alcohol consumption has increased in Indian population.

Drinking more than the UK Chief Medical Officers' (CMO) low risk drinking guidelines, regularly and over a long period of time can increase the risk of developing heart disease. This is because; drinking at this level can increase the risk of high blood pressure. Drinking excessive amounts of alcohol causes raised blood pressure which is one of the most important risk factors for having a heart attack or a stroke. Increases in blood pressure can also be caused by weight gain from excessive drinking[15]. Heavy drinking weakens the heart muscle, which means the heart cannot pump blood as efficiently. It's known as cardiomyopathy and can cause premature death, usually through heart failure [16]. The heart may be enlarged [17]. According to the above report, in the present study, the systolic blood pressure and diastolic blood pressure are high in alcoholics when compared to non-alcoholic MI patients. In the present study, the pulse rate is high in the alcoholic patients than the non-alcoholic patients. After episodes of heavy drinking – usually at least 15 units (about seven and a half pints of 4% beer or one and a half bottles of 13% wine), the heart starts to beat irregularly making to feel breathless.

Creatine Phosphokinase (CPK) and more particularly its isoenzyme CPK-MB still have a formal place in defining myocardial infarction. CPK-MB is a cardiac enzyme marker for myocardial infarction [18]. These enzymes normally exist in cellular compartment and leak out into the plasma during myocardial injury due to disintegration of contractile elements and sarcoplasmic reticulum[19], [20]. It is documented that there is a significant incidence of increased CKMB activity in the alcoholic MI patients when compared to non-alcoholic MI patients. The present study confirms the previously reported increase in total CK activity in patients with acute ethanol ingestion in which patients confirms that acute alcohol intoxication frequently leads to increased MB-CK activity in plasma [21].

In the present study, severe reduction in LVEF, suggesting severe LV dysfunction, is observed in the alcoholics (63%) than the non alcoholics (35%). Heavy alcohol consumption should continue to be discouraged for patients with LV systolic dysfunction, because of the strong evidence that heavy alcohol consumption can lead to impairment of LV contractile function [22], [23], [24].

Conclusion

It is observed that raise in HR, SBP, DBP, increased activity of CPK-MB and reduced LVEF suggesting varying degrees of LV dysfunction were found in alcoholics with AMI. These findings suggest that the complexity and severity of coronary heart disease may be associated with heavy alcohol consumption and alcohol is regarded not only as a major risk factor but also behaves as a prognostic indicator, as LV dysfunction leads to LV de-compensation, thereby increasing the mortality and morbidity. Further studies are required to support our findings with regard to the association of alcohol and LV function in acute myocardial infarction.

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