Research Article

Study of cardiovascular manifestations in individuals with alcohol abuse

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Received: June 19, 2019; **Accepted:** June 22, 2019

ABSTRACT

Background: For nearly 150 years, alcohol consumption has been associated with a variety of cardiovascular diseases. The relationship between alcohol consumption and cardiovascular diseases is complex. The effects of alcohol on the heart include modification of the risk of coronary artery disease, development of alcoholic cardiomyopathy, exacerbation of conduction disorders, atrial and ventricular dysrhythmias, increased risk of hypertension, and fetal heart abnormalities. The purpose of our research is to study the cardiovascular manifestations in individuals with alcohol abuse in our setup. **Objectives:** The objectives of this study were to assess the spectrum of cardiovascular manifestations using clinical examination, lipid profile, and echocardiography (ECHO) in individuals with alcohol abuse compared to normal population. **Materials and Methods:** This case–control study was done in the Department of Medicine, Civil Hospital, Aizawl. The total sample size of 154 individuals was selected. Of which there were 84 cases of individuals with alcohol abuse (as per Diagnostic and Statistical Manual -IV criteria) within the age group of 18–70 years. Seventy age-, sex-, and weight-matched lifetime abstainers, healthy individuals were taken as controls from medicine outpatient department. They underwent a detailed clinical examination, lipid profile, and two-dimensional ECHO to assess the cardiovascular manifestations. **Results:** About 14.3% of the individuals with alcohol abuse were hypertensive. There was positive correlation between alcohol abuse and hypertriglyceridemia (143.8 ± 43.8). Echocardiographic mean left ventricular mass index (LVMI) among the individuals with alcohol abuse was 96.9 ± 3.4 and among the controls was 89.7 ± 4.89. **Conclusion:** Our study showed that the prevalence of hypertension, hypertriglyceridemia, and echocardiographic findings of increased LVMI and diastolic dysfunction is higher in the individuals with alcohol abuse as compared to the normal population.

KEY WORDS: Cardiovascular Manifestations; Alcohol Abuse; Lipid Profile; Echocardiography

INTRODUCTION

Effect of alcohol on cardiovascular system is analogous to a double-edged sword. It can take opposite forms, depending on how, when and the quantity consumed by a particular person, as it has both, a debatable positive as well as negative impact on cardiovascular health. The effects of alcohol on the heart include modification of the risk of coronary artery disease, development of alcoholic cardiomyopathy, exacerbation of conduction disorders, increased risk of hypertension, hemorrhagic stroke, and infective endocarditis.[1]

Access this article online	
Website: http://www.ijmsph.com	Quick Response code
DOI: 10.5455/ijmsph.2019.0617922062019	

Numerous studies have shown that regular light-to-moderate drinking can have a beneficial impact on morbidity and mortality for ischemic heart disease and ischemic stroke, whereas excessive alcohol intake or binge drinking has detrimental effect on cardiovascular system. Pattern of alcohol intake also has an independent effect on cardiovascular morbidity and mortality. Roerecke and Rehm showed a meta analyzed the data on this issue and concluded

that cardioprotective effect of moderate alcohol consumption disappears when, on average, light-to-moderate drinking is mixed with irregular heavy drinking occasions.[2]

Various studies have proved chronic alcohol consumption as a cause of hypertension.[3,4] Single episodes of heavy drinking increase blood pressure and heart rate[5] and it may contribute to sudden cardiac death related to ventricular arrhythmias.[6] However, the studies from Southeast Asia have not shown any beneficial effect of alcohol consumption in this population. The study by Roy *et al*. showed that alcohol intake even in mild-to-moderate amount increases the risk of ischemic heart disease by 30–60% in Indian men when compared with the person who has never consumed alcohol.[7]

Proposed mechanism of the association between alcohol and hypertension: effects on the rennin-angiotensin-aldosterone axis, adrenergic nervous system discharge, catecholamine release, heart rate variability, ionic fluxes, cortisol secretion, insulin sensitivity, renopressor effect. Increased vascular responsiveness to pressor agents may also play an important role. Other possibilities include a direct pressor action of alcohol and a withdrawal effect from short periods of abstinence. Ogata *et al*.[8] in their study found that catecholamine excretion was elevated during acute and chronic alcohol administration. After abrupt withdrawal of alcohol, there was further increase in urinary epinephrine. Plasma norepinephrine levels are highest 13–24 h after alcohol cessation. They suggest stimulation of adrenal medullary secretion as well as changes in sympathetic nervous system activity as the cause of elevated blood pressure in alcoholics. An effect on the renin-angiotensin-aldosterone system was also postulated. Saunders *et al*.[9] reported elevated plasma renin activity in 28 of 48 chronic alcoholics and raised plasma aldosterone in eight individuals. They also noted an increased sympathetic nervous system activity (high dopamine beta-hydroxylase), suggesting an adrenergic mechanism.

MATERIALS AND METHODS

This case–control study was carried out in the Department of Medicine in Civil Hospital, Aizawl. Approval of the Institutional Ethical Committee was taken. The study was conducted between July 2015 and January 2017. Cases and controls were enrolled after meeting inclusion and exclusion criteria. Written, informed consent was obtained from all participants. The study included 84 individuals with alcohol abuse recruited from inpatient and outpatient departments of general medicine. A total of 70, age-, sex-, and weight-matched lifetime abstainers, healthy individuals were taken as controls. All the controls meeting the inclusion and exclusion criteria were taken from medicine outpatient department where they had reported for routine annual medical examination or other minor unrelated ailments.

Inclusion Criteria

Eighty-four fresh cases of individuals with alcohol abuse (as per Diagnostic and Statistical Manual [IV] criteria) within the age group of 18–70 years were included in the study.

Exclusion Criteria

Patients with a history of rheumatic/valvular heart disease, ischemic heart disease, congenital heart disease, smoking, and tobacco use were excluded from the study.

All the cases and controls underwent a detailed clinical examination, blood pressure, lipid profile, and two-dimensional (2D) echocardiography (ECHO) to assess the cardiovascular manifestations.

A detailed history of alcohol consumption, habits, both dose and duration, as well as type of alcohol consumed was asked from each subject. Under aseptic precautions, venous blood samples were taken for lipid profile. Using echocardiogram, each subject was screened for any chamber dilation, left ventricular ejection fraction, diastolic dysfunction, valvular lesion, left ventricular hypertrophy, regional wall motion abnormality, and pulmonary hypertension.

M-mode of 2D ECHO was used to measure interventricular septal thickness at end diastole (IVSD), left ventricular internal dimension at end diastole (LVIDD), and left ventricular posterior wall thickness at end diastole (LVPWD). Mitral valve inflow was assessed by pulsed wave Doppler at the level of the tips of the mitral leaflets in the apical four-chamber view. Early diastolic transmitral velocity (E) and late diastolic transmitral velocity (A) recorded at the tips of mitral valve. E/A ratio was derived for the assessment of the left ventricular compliance and stiffness and diastolic dysfunction. Abnormal mitral inflow pattern (abnormal E/A ratio) was further confirmed by pulmonary venous flow obtained by four-chamber view by color Doppler.

Blood pressure was measured in the arm after a 5 min rest in sitting position, using mercury sphygmomanometer with standard cuff size (to the nearest 2 mmHg), hypertension: >140/>90 mmHg.

Statistics

Statistical analysis of data was made using Chi-square/ Fisher's exact test for comparing categorical variables. Independent *t*-test and Mann–Whitney U-test for comparing mean values between two groups for quantitative variables. $P < 0.05$ was considered to be statistically significant.

RESULTS

The mean age of the individuals with alcohol abuse was 40.9 years and mean age of controls was 40.04 years. This

difference was not statistically significant with respect to the age of the cases and controls. About 41.7% of the individuals with alcohol abuse belonged to 31–40 years and 38.1% belonged to 41–50 years. About 44.3% of the controls belonged to 41–50 years and 41.4% belonged to 31–40 years [Table 1].

About 57.1% of the individuals with alcohol abuse had the habit of consuming alcohol for <15 years and 42.9% were consuming alcohol for ≥15 years [Table 2].

The mean pulse rate of the individuals with alcohol abuse was 73 ± 9.77 beats/min and controls was 64.4 ± 3.5 beats/min. This difference was statistically significant. The mean systolic blood pressure among alcohol abuse was 118.9 mmHg and 115.7 mmHg among the controls. The difference in between the cases and controls was statistically significant. The mean diastolic blood pressure among the individuals with alcohol abuse was 76.2 mmHg and 74.8 mmHg among the controls. The difference was not statistically significant [Table 3].

Table 4 shows that 14.3% of the individuals with alcohol abuse and 1.4% of the controls were hypertensive, 32.1% of the alcoholics, and 14.3% of the non-alcoholics were pre-hypertensive.

The mean high-density lipoprotein (HDL) among the cases was 41.1 mg/dl and among the controls was 36.8 mg/dl, the difference was statistically significant. The mean low-density lipoprotein was 123.8 mg/dl and serum cholesterol 178.4 mg/dl in alcoholics, among controls 114.5 and 177.4 mg/dl, respectively. The difference was statistically not significant. The mean triglycerides among the individuals with alcohol abuse were 143.8 mg/dl and among the controls were 117.1 mg/dl; this difference was statistically significant [Table 5].

The left ventricular ejection fraction among the alcoholics was 0.66 and 0.68 among non-alcoholics; the difference was not statistically significant. The mean LVIDD among the individuals with alcohol abuse was 49.04 and among the controls was 48.0; the difference was not significant. The mean LVPWD among the cases was 9.93 and 9.48 among the controls; this difference was not significant. The mean IVSD among the alcohol abuse was 9.79 and 9.37 among non-alcoholics; the difference was significant. The mean left ventricular mass index (LVMI) among the individuals with alcohol abuse was 96.9 ± 3.4 and among the controls was 89.7 ± 4.89; the difference was statistically significant [Table 6].

The E/A ratio was E < A among 9.5% of the individuals with alcohol abuse and 1.4% of the controls. The ratio was E > A among the 90.5% of the cases and 98.6% of the controls [Table 7].

Table 1: Distribution of the study groups according to age

Age group	Cases *n* (%)		Controls *n* (%)
<30 years	7 (8.3)		6 (8.6)
31–40 years	35 (41.7)		29 (41.4)
41–50 years	32 (38.1)		31 (44.3)
>50 years	10 (11.9)		4 (5.7)
Total	84 (100)		70 (100)
Mean±SD	40.9±7.94		40.04±7.27
t-value		0.677	
P-value		0.499	

SD: Standard deviation

Table 2: Distribution of the study groups according to the duration of drinking alcohol

Duration of drinking alcohol	Cases *n* (%)	Controls *n* (%)
Non-alcoholic	0	70 (100)
<15 years	48 (57.1)	0
≥15 years	36 (42.9)	0
Total	84 (100)	70 (100)

DISCUSSION

Alcohol abuse causes substantial morbidity and mortality due to its effect on cardiovascular system. It can lead to early onset of heart diseases. With that in mind, 84 cases of individuals with alcohol abuse from Civil Hospital, Aizawl, and 70 control non-alcoholic subjects were clinically evaluated. About 14.3% of the individuals with alcohol abuse were hypertensive. There was positive correlation between alcohol abuse and hypertriglyceridemia (143.8 ± 43.8). Echocardiographic mean LVMI among the individuals with alcohol abuse was 96.9 ± 3.4 and among the controls was 89.7 ± 4.89.

The mean age of the individuals with alcohol abuse was 40.9 ± 7.94 years ranging from 24 to 58 years and mean age of controls was 40.04 ± 7.27 years ranging from 26 to 59 years. In a similar study by Lazarević *et al*., the mean age of patients with alcohol abuse was 45 years and controls was 44 years.[10] In a study by Bell *et al*., the mean age of non-drinkers was 48.5 years, former drinker was 49.5 years, occasional

Table 3: Distribution of the study groups according to pulse rate, systolic blood pressure, and diastolic blood pressure

Parameters	Cases (mean±SD)	Controls (mean±SD)	*t*-value	*P*-value
Pulse rate	73.0±9.77	64.4±3.5	6.963	0.000, significant
Systolic blood pressure	118.9±6.5	115.7±12.8	2.013	0.046, significant
Diastolic blood pressure	76.2±8.5	74.8±7.3	1.11	0.269, non-significant

drinkers was 48.1 years, moderate drinkers was 45.8 years, and heavy drinker was 45.8 years [Table 1].[11] This study had shown that 42.9% of the individuals with alcohol abuse were consuming alcohol for ≥15 years. A study by Lazarević *et al*. had shown that the mean duration of drinking alcohol for non-alcohol abusers was 8 years and alcoholics was 13 years [Table 2].[10]

The mean pulse rate of the individuals with alcohol abuse was 73 ± 9.77 beats/min and controls was 64.4 ± 3.5 beats/min. Mean systolic and diastolic blood pressure among the cases was 118.9 and 76.2 mmHg [Table 3]. The mean systolic and diastolic blood pressure among the controls in a study by Lazarević *et al*. was 123 and 79 mmHg, among the cases was 123 and 80 mmHg, respectively.[10] It was noticed that 14.3% of the individuals with alcohol abuse and 1.4% of the controls were hypertensive. Hypertension was significantly associated with the cases compared with controls [Table 4]. A study by Ceccanti *et al*. reported the prevalence of hypertension in 55% of cases of chronic alcohol consumer group during the early stage of abstinence.[12]

Higher levels of HDL (41.1 ± 4.9) and serum triglycerides (143. 8 ± 43.8) were noticed in the individuals with alcohol abuse as compared to the controls [Table 5]. The overall lipid profile data in this study appear to be comparable with the conclusion in a meta-analysis done by Brien *et al*.[13] Meta-analysis by Rimm *et al*. also concluded similar findings regarding the lipid profile in person consuming moderate amount of alcohol.[14]

The mean left ventricular ejection fraction among the individuals with alcohol abuse was 0.66 and 0.68 in the controls [Table 6]. Lazrevic *et al.* compared the echocardiographic findings of 95 patients admitted in hospital, for the treatment for detoxification, with the control subjects. It reported no significant difference in mean ejection fraction between the alcoholics and the control group.[10] Urbano-Marquez *et al*. in their study reported that the total lifetime alcohol intake showed a significant negative correlation with ejection fraction and a significant positive correlation with the left ventricular mass.[15] The ratio was E < A among 9.5% of the individuals with alcohol abuse and 1.4% of the controls [Table 7]. These findings are indicating that impaired left ventricular relaxation or diastolic dysfunction is in concordance with studies by Kupari *et al*.[16] and Silberbauer *et al*.[17] Kupari *et al*. found a significant decrease in the peak early diastolic velocity in alcoholics. Silberbauer *et al*. also reported similar significant decrease in chronic alcoholics.

The mean LVPWD was 9.93 in the cases and 9.48 among the non-alcoholics. It was higher in individuals with alcohol abuse as compared to controls. These findings are in concordance with the study from Lazarević *et al*.[10] and Urbano-Marquez *et al*.[15] They found a significant thicker posterior wall and high LVMI in the alcohol consuming group. In our study, echocardiographic mean IVSD was 9.79 among the alcoholics as compared to 9.37 in controls. Conflicting results have been reported regarding interventricular septal thickness in various studies. Kino *et al*. in their study reported thicker interventricular wall in chronic alcoholics.[18] Mean LVMI among the alcohol abusers was 96.9 g/m^2 and 89.7 g/m^2

Table 4: Distribution of the study groups according to blood pressure

Blood pressure	Cases *n* (%)	Controls *n* (%)
Normal	45 (53.6)	59 (84.3)
Pre-hypertensive	27 (32.1)	10 (14.3)
Hypertensive	12 (14.3)	1 (1.4)
Total	84 (100)	70 (100)

χ^2 value=17.878, df=2, *P*=0.000, Significant

Table 5: Comparison of the study groups according to lipid profile

Mean±SD	Cases	Controls	*t*-value	*P*-value
HDL	41.1±4.9	36.8±5.1	5.31	0.000, significant
LDL	123.8±51.0	114.5±45.7	1.183	0.239, non-significant
Serum cholesterol	178.4±45.7	177.4±39.9	0.138	0.89, non-significant
Triglyceride	143. 8±43.8	117.1±20.6	4.672	0.000, significant

HDL: High-density lipoprotein, LDL: Low-density lipoprotein

Table 6: Comparison of cases and controls according to the echocardiographic left ventricular ejection fraction, LVIDD, LVPWD, IVSD, and LVMI

Parameters	Cases (Mean±SD)	Controls (Mean±SD)	*t*-value	*P*-value
LV ejection fraction	0.66±0.08	0.68±0.07	1.737	0.084, NS
LVIDD	49.04±5.6	48.0±7.1	1.02	0.309, NS
LVPWD	9.93±0.92	9.48±0.51	3.649	0.000, Sig
IVSD	9.79±0.78	9.37±0.39	4.159	0.000, Sig
LVMI	96.9±3.4	89.7±4.89	10.838	0.000, Sig

LVIDD: Left ventricular internal dimension at end diastole, LVPWD: Left ventricular posterior wall thickness at end diastole, IVSD: Interventricular septal thickness at end diastole, LVMI: Left ventricular mass index

Table 7: Distribution of the study groups according to E/A ratio

E/A ratio	Cases *n* (%)	Controls *n* (%)
E<A	8 (9.5)	1 (1.4)
E>A	76 (90.5)	69 (98.6)
Total	84 (100)	70 (100)

χ^2 value –4.547, df=1, *P*=0.033, Significant

among the controls. The study by Lazaveric *et al*., the mean LVMI was 79 g/m^2 among the controls and 91 g/m^2 among the alcoholics [Table 6].[10]

Strength

This study was mainly undertaken to compare the spectrum of cardiovascular manifestations in the individuals with alcohol abuse and normal population, thereby assessing the need for alcohol cessation, early recognition, and treatment of cases.

The limitation of the study is that it is a hospital-based, case–control study. Sample size was less. This type of study design may result in recall bias.

CONCLUSION

We conclude that the prevalence of hypertension, hypertriglyceridemia, and echocardiographic findings of increased LVMI, IVSD, and diastolic dysfunction is higher in the individuals with alcohol abuse as compared to the controls. Hence, cessation of alcohol is recommended to prevent its cardiovascular system-related morbidity and mortality.

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How to cite this article: Lalramenga PC, Savla HS, Naveen P. Study of cardiovascular manifestations in individuals with alcohol abuse. Int J Med Sci Public Health 2019;8(8):688-692.

Source of Support: Nil, **Conflict of Interest:** None declared.