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Effects of Alcohol Consumption on Cardiovascular Features among Iraqi Individuals with Alcohol Abuse

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Abstract: ***Effects of alcohol consumption 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.Alcohol consumption has been associated with a variety of cardiovascular diseases, and 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. This study aimed to assess the spectrum of cardiovascular manifestations using clinical examination, lipid profile, and echocardiography (ECHO) in individuals with alcohol abuse. This case–control study was done in Ibn Al-Nafees hospital in Baghdad/ Iraq during the period from July 2015 to January 2017 on 80 individuals with alcohol abuse (as per diagnostic and statistical manual-IV criteria) within the age group of 18–70 years. From the hospital's outpatient clinic, 70 age-, sex-, and weight-matched healthy individuals were taken as controls. They underwent a detailed clinical examination, lipid profile, and two-dimensional ECHO to assess the cardiovascular manifestations. Results showed that13(16.3%) of the individuals with alcohol abuse were hypertensive. There was positive correlation between alcohol abuse and hypertriglyceridemia (145.3 ± 44.5). Echocardiographic mean left ventricular mass index (LVMI) among the individuals with alcohol abuse was 97.1 ± 4.41 and among the control group was 90.3 ± 4.72. Our study concluded that the prevalence of hypertension, hypertriglyceridemia and echocardiographic with increased LVMI and diastolic dysfunction were higher in the alcoholic groupwhen as compared with the normal people.***

Keywords: Alcohol abuse,Cardiovascular features, Lipid profile, Echocardiography

1. Introduction

Theeffects 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]

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 concludedthat 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.

2. Materials and Methods

This case–control study was conducted in Ibn Al-Nafees hospital in Baghdad / Iraq during the period from July 2015 to January 2017 on 80 individuals with alcohol abuse, and 70 healthy persons as a control group after meeting inclusion

and exclusion criteria. Approval of the institutional ethical committee was taken, and a written and informed consent was obtained from all participants.

The 80 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.

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 features.
A detailed history of alcohol consumption was taken from each subject. 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 trans mitral velocity (E) and late diastolic trans mitral 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, hypertension: >140/>90 mmHg. Statistical analysis of data was done usingSPSS version 21 program; Chi-square/ Fisher's exact test for comparing variables. Independent t-test and Mann–Whitney U-test for comparing mean values between two groups for quantitative variables. $P < 0.05$ was considered statistically significant.

3. Results

The mean age of the individuals with alcohol abuse was 45.2 ±12.5 years and mean age of controls was 42.4±10.9 years, with no significant difference. In regard to age groups, 32(40.0%) of the individuals with alcohol abuse were withinthe age group 31–40 years and 35(43.7%)were within the age group41–50 years, while30(42.8%) of the controls were within the age group 41–50 years and 30(42.8%)within the age group31–40 years as shown in table (1).

Table 1: Distribution of the study groups according to age

Age group	Cases n (%)	Controls n (%)
<30 years	3 (3.7)	6 (8.6)
31–40 years	32 (40.0)	30 (42.8)
41–50 years	35 (43.7)	30 (42.8)
>50 years	10 (12.6)	4 (5.8)
Total	80 (100)	70 (100)
Mean±SD	45.2±12.5	42.4±10.9
t-value	1.45	
P-value	0.148	

SD: Standard deviation

The mean pulse rate of the individuals with alcohol abuse was 79± 9.5 beats/min and in the controls was 69.5 ± 4.5 beats/min, with a significantdifference. The mean systolic blood pressure among alcohol abuse was 120.5±7.5 mmHg and 115.5±9.8 mmHg among the controls, with a significant difference, whereas the mean diastolic blood pressure among the individuals with alcohol abuse was 78.2±8.5 mmHg and 77.8±7.3 mmHg among the controls with no statistically significant difference as seen in table (2).

Table 2: Distribution of the study groups according to pulse rates, systolic and diastolic blood pressures

Parameter	Cases (mean ±SD)	Controls (mean ±SD)	t-value	P-value
Pulse rate	79±9.5	69.5±4.5	7.65	0.005, significant
Systolic blood pressure	120.5±7.5	115.5±9.8	3.53	0.005, significant
Diastolic blood pressure	78.2±8.5	77.8±7.3	0.31	0.759, non-significant

Table (3) shows that 13(16.3%) of the individuals with alcohol abuse and 5(7.2%) of the controls were hypertensive, 27(33.7%) of the alcoholics, and 10(14.3%) of the non-alcoholics were pre-hypertensive respectively.

Table 3: Distribution of the study groups according to blood pressure

Blood pressure	Cases n (%)	Controls n (%)
Normal	40 (50.0)	55 (78.5)
Pre-hypertensive	27 (33.7)	10 (14.3)
Hypertensive	13 (16.3)	5 (7.2)
Total	80 (100)	70 (100)
$\chi 2$ value=13.13, df=2, P=0.001, Significant		

The mean high-density lipoprotein (HDL) among the cases was 41.5±4.5 mg/dl and among the controls was 43.8±4.3 mg/d with a significantdifference, while the mean low-density lipoprotein was 125.2±43.9 mg/dl and serum cholesterol 185.4±45.1 mg/dl in alcoholics, and among controls they were114.9±45.5 and 179.2±40.5 mg/dl, respectively, with no statistically significant difference. The mean triglycerides among the individuals with alcohol abuse were 145.3±44.5 mg/dl and among the controls were 112.9±26.1 mg/dl with a significant difference as shown in table (4).

Table 4: Comparison of the study groups according to lipid profile

Mean ±SD	Cases	Controls	t-value	P-value
HDL	41.5±4.5	43.8±4.3	3.19	0.001, significant
LDL	125.2±43.9	114.9±45.5	1.41	0.160, non-significant
Serum cholesterol	185.4±45.1	179.2±40.5	0.88	0.379, non-significant
Triglyceride	145.3±44.5	112.9±26.1	5.34	0.005, significant

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

The left ventricular ejection fraction among the alcoholics was 0.67±0.07 and 0.69±0.08 among non-alcoholics; the difference was not statistically significant, while the mean LVIDD among the individuals with alcohol abuse was 49.15±5.1 and among the controls was 48.8±7.5 withno significant difference. The mean LVPWD among the cases was 9.81±0.90 and 9.52±0.53 among the controls, and this difference was statistically not significant. The mean IVSD among the alcohol abuse was 9.83±0.81 and 9.31±0.41 among non-alcoholics, with a significantdifference. The mean left ventricular mass index (LVMI) among the individuals with alcohol abuse was 97.1±4.41 and among the controls was90.3±4.72, with a statistically significantdifference as illustrated in table (5).

Table 5: Comparison between 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	0.67±0.07	0.69±0.08	1.63	0.104, NS
LVIDD	49.15±5.1	48.8±7.5	0.34	0.736, NS
LVPWD	9.81±0.90	9.52±0.53	2.36	0.019, Sig
IVSD	9.83±0.81	9.31±0.41	4.85	0.005, Sig
LVMI	97.1±4.41	90.3±4.72	9.12	0.005, 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

The E/A ratio was E < A among 6(7.5%) of the individuals with alcohol abuse and 1(1.4%) of the controls. The ratio was E > A among the 74(92.5%) of the cases and 69(98.6%) of the controls as seen in table (6).

Table 6: Distribution of the study groups according to E/A ratios

E/A ratio	Cases n (%)	Controls n (%)
E<A	6 (7.5)	1 (1.4)
E>A	74 (92.5)	69 (98.6)
Total	80 (100)	70 (100)

$\chi 2$ value =2.87, df=1, P=0.090, non-significant

4. Discussion

Alcohol abuse causes substantial morbidity and mortality due to its effect on cardiovascular system. It can lead to early onset of heart diseases. In our study, 16.3% of the individuals with alcohol abuse were hypertensive. There was positive correlation between alcohol abuse and hypertriglyceridemia (145.3±44.5). Echocardiographic mean LVMI among the individuals with alcohol abuse was 97.1±4.41 and among the controls was 90.3±4.72. The mean age of the individuals with alcohol abuse was 45.2±12.5 years and mean age of controls was 42.4±10.9 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,, and heavy drinker was 45.8 years[11].

The mean pulse rate of the individuals with alcohol abuse was 79±9.5 beats/min and controls were69.5±4.5beats/min. Mean systolic and diastolic blood pressure among the cases was 120.5±7.5and 78.2±8.5mmHg, 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 16.3% of the individuals with alcohol abuse and 7.2% of the controls were hypertensive. Hypertension was significantly associated with the cases compared with controls, table (3). 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 (41.5 ± 4.5) and serum triglycerides (145.3±44.5) were noticed in the individuals with alcohol abuse as compared to the controls [Table 4]. The overall lipid profile data in this study appear to be comparable with theconclusion in a meta-analysis done by Brien et al [13]. Metaanalysis 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 0.67 and 0.69 in the controls [Table 5]. 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 7.5% of the individuals with alcohol abuse and 1.4% of the controls [Table 6]. 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 9.81±0.90 in the cases and 9.52±0.53 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 Lazarevićet 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 9.83±0.81 among the alcoholics as compared to 9.31±0.41 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 97.1±4.41 g/m^2 and 90.3±4.72 g/m^2among 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].

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