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Original research article

Evaluation of the left ventricular function in chronic asymptomatic alcoholics

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Abstract

Background and Objective: To evaluate preclinical left ventricular dysfunction with Doppler and two-dimensional echocardiography in long-term asymptomatic alcohol consumer. To establish a correlation between potential variations and the amount of alcohol consumed.

Methods: A observational study was carried out at Department of General Medicine, Aditya Hospital, Jangaon, Telangana India from January 2023 to December 2023 after informed consent signed from 60 subject to assess the ventricular function in chronic asymptomatic alcoholism**.**

Results: Alcoholics had a considerably greater mean end diastolic volume index (p<0.001) than controls. The mean ejection fraction did not differ statistically significantly between the two groups. Alcoholics had a statistically substantially higher estimated mean left ventricular mass index (p<0.001). Alcoholics had considerably longer isovolumic relaxation times and early transmitral flow velocity deceleration times than controls (p<0.001).

Conclusion: The consumers displayed characteristics of maintained ejection fraction along with reduced left ventricular relaxation. Increased left ventricular mass, posterior wall thickness, and increased left ventricular chamber volume in diastole were the abnormalities seen in alcoholics by 2-D and M-mode echocardiography. Prolonged isovolumic relaxation and deceleration times, higher peak late transmitral flow velocities, and decreased E/A ratios were the anomalies in the Doppler parameters.

Keywords: Left ventricular chamber volume, ejection fraction, alcohol abuse, echocardiography

Introduction

Alcohol abuse is pervasive across all countries. Variations exist in the patterns of alcohol intake according to age, education level, religion, and other sociodemographic variables. Since 1970, the percentage of people who drink alcohol has climbed by 47% in developing countries and by 35% in wealthy countries [1, 2, 3]. Alcohol itself causes 4% of Disability Adjusted Life Years (DALYs), but alcohol use disorders account for 1.4% of the total sickness burden. 10% of women and 20-38% of men are said to be alcohol consumers at the moment [4, 5]. According to studies done in northern India, between 25 and 40 percent of individuals drink alcohol annually. In southern India, alcohol use today ranges from 33 to 50%, with a higher prevalence among the impoverished and those with lower educational attainment [5, 6]. According to these research, men drink alcohol at much higher rates than women do. While a healthy individual may benefit from one to two drinks per day for cardiovascular health, bigger alcohol use is harmful to most organ systems [7, 8]. Increased ethanol dosages may cause arrhythmias, hypertension in the systemic circulation, malfunction of the ventricular systolic and/or diastolic function, and even sudden cardiac death. Extended excessive alcohol intake can cause dilated cardiomyopathy, which is a gradual heart failure [9, 10]. Before heart failure is evident, subtle symptoms may appear as the illness progresses. This is significant because an early cessation may be able to reverse the left ventricular dysfunction. The effects of chronic alcohol use on the left ventricle's (LV) diastolic and systolic functioning have been the subject of numerous studies [10, 11]. Studies have indicated that LV systolic function has been retained, despite occasional reports of decreased LV systolic performance. Additionally, reports of both normal and compromised LV filling have been made [11, 12].

Materials and Methods

An observational study was conducted at Department of General Medicine, Aditya Hospital, Jangaon, Telangana India from January 2023 to December 2023 with signed informed consent from sixty subjects in order to evaluate ventricular function in persistent asymptomatic alcoholism. Alcoholics who satisfied the criteria for alcohol abuse or dependence were classified as DSM IV patients. From a detailed history, the amount and duration of alcohol consumption were ascertained. The amount of alcohol was then converted to grams of ethanol using the following formulas [13, 14]. Next, the alcohol addicts were divided into three groups in an effort to evaluate any possible differences in left ventricular function based on the duration of alcohol consumption. The following tests were performed: complete blood count, renal function tests, fasting and postprandial blood sugar, fasting lipid profile, thyroid function tests, liver

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function tests, and echocardiography [14].

Inclusion criteria

- Positive DSM IV diagnostic criteria for alcohol abuse or dependence.
- 60 patients who attended the medical department and met the study's enrollment requirements.
- Age < 60 years.
- Daily ethanol intake ≥ 90 grammes.
- History of drinking ≥ 5 years.
- No history of systemic disorders.

Exclusion criteria

- Patient with Ischemic / rheumatic / congenital heart disease.
- Diabetes mellitus in subject.
- Hypertensive individuals.
- Patient with Hyperlipidemia.
- Thyroid disorders in patient.

Results

Table 1: Laboratory data

	Controls	Group 1	Group 2	Group 3
Hb (g/dL)	13.81±0.45	13.45±0.58	13.05±0.32	13.36±0.70
AST (U/L)	25.54±5.27	44.82±14.15	54.68±14.86	90.13±12.13
ALT (U/L)	29.05±5.51	44.45±11.30	55.62±18.35	50.35±29.15
GGT (U/L)	29.28±6.68	62.25±11.50	63±11.90	90.23±16.39
T. Bilirubin (mg/dL)	0.95±0.19	0.86±0.26	0.89±0.19	0.87±0.15
T. Cholesterol (mg/dL)	162.53±13.26	163±19.85	164.19±14.05	161.72±16.35
TGL (mg/dL)	133.89±9.15	121.79±8.87	116.59±18.35	125.70±9.89

Table 2: EDVI & ESVI

		Range	Mean	Std. Deviation	
EDVI (mL/m^2)	Controls	35.39-65.40	45.15	5.89	p = 0.000
	Group 1	40.12-56.77	48.43	4.65	
	Group 2	46.03-61.53	51.48	4.45	
	Group 3	48.56-65.43	53.65	4.89	
ESVI (mL/m^2)	Controls	11.15-15.86	13.17	1.58	p = 0.889
	Group 1	12.11-16.03	13.30	1.48	
	Group 2	12.73-18.86	15.28	1.67	
	Group 3	12.18-16.27	14.59	1.56	

Table 3: Ejection fraction & left ventricular mass index

		Range	Mean	Std. Deviation	
EF (%)	Controls	65-87	75.15	6.88	p = 0.685
	Group 1	66-82	76.32	5.73	
	Group 2	68-85	76.54	6.59	
	Group 3	68-89	76.64	6.32	
LVMI (g/m^2)	Controls	55.78-105.94	76.56	11.54	p = 0.000
	Group 1	72.54-96.50	76.31	8.95	
	Group 2	73.68-98.46	82.68	8.65	
	Group 3	85.65-103.55	95.32	7.85	

Table 4: Isovolumic relaxation time & deceleration time

		Range	Mean	Std. Deviation	
IVRT (ms)	Controls	64 - 128	98.54	14.56	p = 0.000
	Group 1	75 - 99	87.66	6.32	
	Group 2	73 - 105	98.86	7.54	
	Group 3	82 - 122	89.59	13.82	
DT (ms)	Controls	115 - 225	159.54	25.36	p = 0.000
	Group 1	133 - 186	161.68	16.40	
	Group 2	144 - 198	182.88	16.45	
	Group 3	178 - 232	199.35	16.78	

Discussion

Over time, dilated cardiomyopathy is brought on by prolonged alcohol usage. Nonetheless, certain heart abnormalities that occur before clinical symptoms appear can be detected by echocardiography. These could be reversible effects with an early cessation. Therefore, it is imperative that these developments be acknowledged. Extensive research in this field has produced a mixed bag of findings. Finding cardiac

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abnormalities in long-term, asymptomatic alcoholics was the aim of this investigation. Included in the study were sixty alcoholics who satisfied the screening criteria. Three groups were formed to examine whether the duration of their alcohol use had an impact on their left ventricular performance. The sixty healthy individuals who served as controls had similar baseline characteristics. The baseline characteristics for the case and control groups were similar, including age, body surface area, pulse, and blood pressure [15]. People's years of alcohol consumption ranged from six to twenty-three. A daily average of 137.4±41.84 grams of ethanol were consumed. On average, the group who drank for the longest also consumed the most ethanol (190±38.08 grams). Growing tolerance is the cause of this. The laboratory values for controls and alcoholics did not significantly differ, possibly with the exception of liver function tests, where alcoholics showed a slight increase in enzymes (gamma glutamyl transferase, aspartate & alanine aminotransferases), which may be related to long-term ethanol usage. The left ventricular function of 89 chronic, asymptomatic drinkers was assessed by Lazarevic *et al*. He had divided the alcoholics into three groups: short, moderate, and long, according to the length of time they had been drinking [14, 15]. Using 2-D and M-mode echocardiography, the left ventricular systolic function and chamber dimensions were evaluated. The results of Lazarevic *et al*. and Askanas *et al*. are in line with our research, which found no statistically significant difference in the ejection % between controls and alcoholics. There was a statistically significant difference in the end diastolic volume index between the drinkers and the controls in this study. The final systolic volume index showed no appreciable variation [15, 16]. In contrast, larger diastolic and systolic dimensions of the left ventricle were noted by Lazarevic *et al*. Frank Starling's law could account for the normal end systolic volume index in our investigation. By increasing the stroke volume and decreasing the end systolic volume, the heart contracts more as the end diastolic volume rises. Rats and chickens in animal experiments by Nancy Morris *et al*. and Shann D. Kim *et al*. have verified the larger LV chamber volumes. Among this study, the posterior wall thickness and left ventricular mass index were higher among drinkers. There was no appreciable shift in the thickness of the interventricular septum. The findings of AM Lazarevic *et al*., Kupari *et al*., and Mathews *et al*. were in line with this. The diastolic function was evaluated with Doppler echocardiography [16, 17]. Our results showed that alcoholism was linked to a longer deceleration time, a longer isovolumic relaxation duration, and a lower E/A ratio. A rise in the peak late transmitral flow velocity was also observed. The findings indicate a possible diagnosis of moderate diastolic dysfunction or reduced left ventricular relaxation. Analogous findings were reported by Lazarevic *et al*. 39. Additionally, research by Kupari *et al*. and De Castro *et al*. has addressed it. If there is a problem with the calcium ion transport from sarcoplasma into the sarcoplasmic reticulum, it could be the reason for the delayed relaxation. Actin myosin interaction may take longer to inactivate [17, 18]. Delayed relaxation may also result from concomitant ventricular dilatation or cardiac hypertrophy [18]. The purpose of this study's data analysis was to determine whether age, blood pressure, pulse rate, amount of alcohol taken, and duration of drinking were independent variables that might be correlated with changes in echocardiogram. The parameters of the echocardiography showed a significant relationship with the duration of alcohol use. The consumption of alcohol was found to considerably raise the end diastolic volume index, left ventricular mass index, posterior wall thickness, isovolumic relaxation time, deceleration time, and peak late transmitral flow velocity. This field has very little study done in it. According to Lazarevic *et al*., alcoholics with longer drinking sessions had shorter deceleration durations, higher a values, and lower E/A ratios. The posterior wall thickness and the length of binge drinking were found to be inversely correlated by Kupari *et al*. 35. Nevertheless, no correlation could be shown between the amount and duration of alcohol intake and left ventricular hypertrophy and dysfunction. Differences in the quantity and duration of alcohol drank may account for these variations [19, 20]. To find the earliest detectable change in echocardiography, the echocardiographic features of the three alcohol-related groups were compared with the controls. Group 1, which consumed alcohol for the least amount of time, did not exhibit any notable differences from the control group. In terms of deceleration time, isovolumic relaxation length, end diastolic volume index, and LV mass index, Group 2 was significantly different from the controls. A significant decrease in the E/A ratio and a notable rise in posterior wall thickness and peak late transmitral flow velocity (A) were also observed in the alcoholics with the longest drinking history (group 3), in addition to the changes shown in group 2. This suggests that a rise in left ventricular mass and end diastolic volume occurred first, and that impaired LV relaxation developed later. According to Lazarevic *et al*., after prior LV volume modifications, LV mass increased and hence had an impact on diastolic function [21, 22].

Conclusion

Long-term asymptomatic drinkers may have preclinical cardiac issues that can be detected by echocardiography. The drinkers showed signs of decreased left ventricular relaxation and maintained ejection fraction. By using 2-D and M-mode echocardiography, the anomalies seen in alcoholics were increased left ventricular mass, posterior wall thickness, and increased left ventricular chamber volume in diastole. The anomalies in the Doppler parameters were longer isovolumic relaxation and deceleration periods, larger peak late transmitral flow velocities, and lower E/A ratios. The first obvious change was

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an increase in the mass, volume, and thickness of the LV chamber's posterior wall. Following that, LV relaxation was compromised. Changes in LV mass, volume, posterior wall thickness, and diastolic measures were all highly linked with the duration of drinking. The previously described findings might indicate the onset of alcoholic cardiomyopathy, however more investigation is required to verify this before long-term investigations are carried out. Further research is required to demonstrate how these anomalies begin, progress, and ultimately reverse themselves with early cessation.

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Conflict of interest
None.

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