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Original research article

To study the effect of alcohol on cardiac function in alcohol abuse patients

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Abstract

Aims and Objectives: To investigate how alcohol affects cardiac function in Indian hospital patients with alcoholism. Assess 2D ECHO results and ECG in patients with alcohol abuse.

Methods: Between September 2021 and September 2022, cardiology department of Kurnool Medical College, Kurnool, Andhra Pradesh, India, conducted the current cross-sectional study on cardiac function in alcohol abuse. A total of fifty alcohol abuse patients who met the inclusion and exclusion criteria were chosen.

Results: Ejection fraction or LVD is inversely correlated with the total lifetime alcohol intake. The majority of the patients under study had cardiomegaly visible on their chest X-rays, along with other radiological findings like pulmonary congestion and pleural effusion. ECG findings were normal in 18% of patients, atrial fibrillation was seen in 20% of patients, LVH was seen in 12% of patients, sinus tachycardia was seen in 12% of patients, LBBB was seen in 10% of patients, RBBB was seen in 12% of patients, SVT was seen in 10% of patients, and ectopic beats were seen in 6% of patients. The maximum number of patients with DT (150-200) was found in 42% of those who underwent echocardiography, and 64% of patients had prolonged EPSS (>12). Maximum patient numbers for LVSD (40%) and LVDD (52%), respectively, were found in the range of 6–6.9 cm and >7 cm, respectively. ESV between 50 and 74 ml is seen in 36% of patients, while EDV between 100 and 174 ml is seen in 64% of patients. Severe LVD was seen in only 16 percent of patients, moderate LVD in 24 percent, and mild LVD in 60 percent of patients.

Conclusion: Alcohol abuse leads to dilated cardiomyopathy, arrhythmias, heart failure, sudden deaths. Alcoholic dilated cardiomyopathy also risk factor for sudden death. Alcohol abstinence results in better prognosis.

Keywords: Cardiomyopathy, arrhythmias, heart failure, alcohol abuse

Introduction

The most commonly used toxic substance worldwide is alcohol [1]. The third-leading cause of death that can be prevented is alcoholism and alcohol abuse, both of which are serious health issues. An estimated 20–30% of admissions for cardiology will have a complicated multigenic etiology. Alcohol use contributed to 3.3 million deaths in 2012, or 5.9% of all deaths worldwide (7.6% of deaths among men and 4.0 percent of deaths among women) [1]. Alcohol use disorders, liver cirrhosis, cancers, and injuries are just a few of the illnesses and injury-related health conditions that alcohol is linked to [2]. Alcohol use accounted for 5.1% of disease and injury burden globally in 2012 (139 million disability-adjusted life years) [3]. In terms of risk factors for premature death and disability worldwide, alcohol abuse ranks first among those who are between the ages of 15 and 49 [4]. According to the WHO, alcohol is thought to be a contributing factor in about 25% of deaths among people aged 20 to 39 [5]. Despite the fact that drinking has long been a part of various societies' cultural traditions, alcohol use has recently come to be recognized as a serious public health issue. For both male and female patients, the prevalence of admissions related to alcohol was 13.3% and 2.3%, respectively. The majority of patients were between the ages of 31 and 50. In both sexes, chronic liver disease and its complications were the most typical presentation. Males were more likely to experience alcohol dependence syndrome than females, which has a big impact on the health and financial burden of society [6].

Prevalence of Alcoholic cardiomyopathy in idiopathic dilated cardiomyopathy according to various studies, varied from 23% to 47% like Halssaguerre *et al*., (47%), Prazak *et al*. (31%), Fauchier *et al*. (37%), McKenna *et al*. (40%), Gavazzi *et al*. (23%). Alcoholic cardiomyopathy is a form of dilated cardiomyopathy that causes heart failure as a result of chronic, long-term alcohol abuse (ethanol) [7]. Heart failure results from the heart's inability to pump blood effectively as a result of the direct toxic

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effects of alcohol on heart muscle. Males between the ages of 35 and 50 are the most susceptible. The most common underlying factor leading to dilated cardiomyopathy is idiopathic, followed by alcohol use [8]. Chronic alcohol use has been linked to the emergence of liver or pancreatic failure, oral and pharyngeal cancers, as well as an elevated risk of dementia. A J-shaped curve can be seen in a graph that compares all-cause mortality to alcohol consumption [9]. While binge drinking or heavy drinking on an irregular basis increases the risk of cardiovascular disease, there is a direct correlation between these behaviors and the risk of total mortality. Alcohol affects all bodily tissues and the majority of essential bodily functions. An important factor in the death of chronic alcoholics is heart disease, along with cirrhosis.

Consuming too much alcohol damages the myocardium, disrupting the myofibrillar architecture and reducing the contractility and ejection fractions of the heart. Cardiomegaly and congestive heart failure are the primary symptoms of alcoholic cardiomyopathy, which is characterized by cardiac hypertrophy and ventricular dilatation [1]. Although many alcohol-related deaths go unreported, many professionals think that alcoholism is likely the leading cause of death. One of the top ten leading causes of death, for instance, is liver cirrhosis, which is most frequently linked to alcoholism. Alcohol has the potential to seriously physically harm one or all of the body systems as an irritant. Medical professionals all too frequently delay making an alcoholism diagnosis until the onset of such physical symptoms. Alcoholism is a chronic, progressive illness, but despite this, the early signs are typically behavioral rather than physical. In the late, chronic stages of an illness, the majority of health issues typically manifest. The high death rate associated with this issue can be attributed to waiting for physical symptoms to show.

The secondary nature of alcoholism to the primary nature of medical complications. Many alcoholics can avoid more severe medical complications if the problem is evaluated and diagnosed properly. The exact prevalence of alcohol intake particularly in relation to cardiovascular system involvement are not known in Indians. There is no large-scale studies and no standard guidelines to show association between alcohol and heart disease. Alcohol abuse has been reported most common identifiable etiological factor, which causes up to one third of cases of dilated cardiomyopathy. Alcohol abuse also causes arrhythmias like atrial fibrillation, supra ventricular tachycardia, ventricular ectopic beats. Alcoholism is known to cause sudden cardiac death, which may be related to an arrhythmogenic effect of alcohol. Hence present study is to know the effects of alcohol on the heart.

Materials and Methods

A cross-sectional study was conducted at Kurnool Medical College, Kurnool, Andhra Pradesh, India, medical wards and cardiology department. From September 2021 to September 2022, fifty patients with alcohol abuse who met the inclusion and exclusion requirements were chosen.

Inclusion criteria

- Age > 18 years
- Patients with history of alcohol abuse admitted in medical wards.
- Patients willing to give written informed consent.

Exclusion criteria

- Patients with known cardiac disease like valvular heart diseases, congenital heart disease, ischemic heart disease, dyslipidemeas.
- Thyroid disorder
- Diabetes mellitus
- Hypertension
- Age <18 years
- Smokers

Study Methods

- Careful detailed history paying special attention towards
- Duration of alcohol consumption
- Type of alcohol and quantity of alcohol
- History was taken regarding the symptoms like breathlessness, palpitations, syncopal attacks, pedal edema, oliguria, jaundice, weight loss.
- Thorough clinical examination
- Relavent past history, family history, personal history were recorded, all the details were entered in the proforma.
- General physical examination including detailed cardiovascular system examination was done in all patients and appropriate investigations were done.

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Results

Table 1: Age Distribution

S. No.	Age	Frequency	Percentage
1.	<29 yr	6	12
2.	30-44 yr	30	60
3.	45-59 yr	14	28
4.	Total	50	100

Age distribution table showed that maximum number of patients(60%) were present between in the age group of 30-44 yr and least number of patients (12%) were in the group of <29 years.

Table 2: Symptoms at presentation

S. No.	Symptoms at Presentation	Frequency	Percentages
1.	Breathlessness	50	100
2.	Chest pain	11	22
3.	palpitations	43	86
4.	Pedal edema	33	66
5.	Oliguria	26	52

In present study the frequency of various symptoms at presentation are breathlessness in (100%) of patients, chest pain in (22%) of patients, palpitations in (86%) of patients, oliguria in (52%) of patients, pedal edema in (66%) of patients. Most common presenting symptom was breathlessness

Table 3: Pulse Regularity Distribution Table

S. No.	Pulse Rate	Frequency	Percentage
1	Irregular	10	20
2	Regular	40	80
3	Total	50	100

Present study showed that pulse among patients of alcohol abuse irregular in 20% of patients and regular in 80% of patients.

Table 4: Signs at Presentation

S. No.	Signs	Frequency	Percentages
1.	Raised JVP	33	66
2.	S3	21	42
3.	Cardiomegaly	34	68
4.	Organomegaly	11	22
5.	Ascites	21	42
6.	crepitations	22	44

Various signs at presentation in the present study were raised JVP in 66% of patients, S3 present in 42% of patients, cardiomegaly on chest X ray PA view in 68% of patients, organomegaly in 22% of patients, ascites in 42% of patients, crepitation in 44% of patients.

Table 5: Murmurs at the time of presentation

S. No.	Murmur	Frequency	Percentage
1.	No murmur	17	34
2.	PSM over TA	18	36
3.	PSM over MA	8	16
4	PSM over TA&MA	3	6
5	SM over PA	4	8
6	Total	50	100

Present study showed that no murmur in 34% of patients, PSM over TA in 36% of patients, PSM over MA 16% of patients, PSM over in TA & MA(both) in 6% of patients, SM over PA in 8% of patients.

Table 6: Relationship between Amount of Alcohol and Ejection Fraction

S. No.	Amount of Alcohol (gm)	Frequency	Mean EF	Standard deviation	ANOVA Test
1.	5 lakhs	17	45.18	5.790	f= 5.660 P=0.002
2.	5-10 lakhs	19	39.63	8.952	
3.	10-15 lakhs	9	43.22	6.418	

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4.	15-20 lakhs	5	31.00	3.674	

ANOVA TEST was applied f=5.660, P value =0.002.

In present study the amount of alcohol consumed in total life time by a patient and echocardiographic EF values were compared through ANOVA test, as amount of alcohol increases EF value decreases significantly, (P value=0.002).

Table 7: ECG Changes

S. No.	ECG Changes	Frequency	Percentages
1	Normal	9	18
2	Atrial Fibrillation	10	20
3	LVH	6	12
4	Sinus Tachycardia	6	12
5	SVT	5	10
6	LBBB	5	10
7	RBBB	6	12
8	Ectopics	3	6
9	Total	50	100

Present study showed various ECG changes, normal ECG noticed in 18% of patients, atrial fibrillation in 20% of patients, LVH in 12% of patients, Sinus tachycardia in 12% of patients, SVT in 10% of patients, LBBB in 10% of patients, RBBB in 12% of patients, ventricular ectopics in 6% of patients.

Table 8: ECHO Findings DT (Deceleration time)

S. No	DT	Frequency	Percentages
1.	<150	14	28
2.	150-200	21	42
3.	>200	15	30
4.	total	50	100

Present study showed that DT is <150 msec in 28% of patients, 150-200 msec in 42% of patients, >200 msec in 30% of patients.

Table 9: EPSS values

S. No.	EPSS	Frequency	Percentages
1	<5	1	2
2	6-11	17	34
3	>12	32	64
4	total	50	100

In present study the EPSS is <5 in 2% of patients, 6-12 in 34% of patients, >12 in 64% of patients. Maximum no.of patients present between EPSS; 6-12.

Table 10: LVSD & LVDD percentages

S. No.	Diameters Range(cm)	LVSD Percentages	Frequency	LVDD Percentages	Frequency
1	3-3.9	8	4	2	1
2	4-4.9	18	9	10	5
3	5-5.9	32	16	14	7
4	6-6.9	40	20	22	11
5	>7	2	1	52	26
	Total	100	50	100	50

LV systolic diameter normal 2.3-3.9 cm

In present study few of them had normal LV systolic diameter it is 3-3.9 cm in 8% of patients, 4-4.9 cm in 18% of patients, 5-5.9 cm in 32% of patients, 6-6.9 cm in 40% of patients,>7 cm in 2% of patients. Maximum no.of patients were found in 6-6.9cm.

LV Diastolic diameter normal 3.6-5.2 cm

In present study some of them had normal LV diastolic diameter 3-3.9 cm in 2% of patients, 4-4.9cm in 10% of patients, 5-5.9 cm in 14% of patients, 6-6.9 cm in 22% of patients, >7 cm in 52% of patients. Maximum no.of patients were found in >7 cm.

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Table 11: ESV ml Measurements

S. No.	ESV (ml)	Frequency	Percentage
1	<49	15	30
2	50-74	18	36
3	75-99	17	34
	Total	50	100

Normal ESV ml= 19-49 ml.

Present study showed that ESV <49ml in 30% of patients, 50-74 ml in 36% of patients, 75-99ml in 34% of patients. Maximum no.of patients were found in between ESV; 50-74 ml.

Table 12: EDV ml measurements

S. No.	EDV ml	Frequency	Percentage
1	50-74	1	2
2	75-99	15	30
3	100-124	10	20
4	125-149	10	20
4	150-174	12	24
5	175-199	2	4
	Total	50	100

Normal EDV ml =56-104 ml.

Present study revealed that EDV; 50-74ml in 2% of patients, 75-99ml in 30% of patients, 100-124ml in 20% of patients, 125-149 ml in 20% of patients, 150-174ml in 24% of patients, 175-199ml in 4% of patients.
Maximum No. of patients were found in 100-174 ml.

Table 13: LVD Percentages

S. No	LVD	Frequency	Percentage
1	Mild LVD	30	60
2	Moderate LVD	12	24
3	Severe LVD	8	16
	Total	50	100

Mild LV dysfunction means EF value 45-54%, moderate LV dysfunction means EF value 30-44%, severe LV dysfunction means EF value <30%.
LVD in present study showed, mild LVD in 60% of patients, moderate LVD in 24% of patients, severe LVD in 16% of patients. Maximum no.of patients were found in mild LVD.

Table 14: Diagnosis Chart

S. No.	Diagnosis	Frequency	Percentages
1.	AF	10	20
2.	DCM	21	42
3.	HF	14	28
4.	SVT	5	10
	Total	50	100

In present study, DCM is found in 42% of patients, AF in 20% of patients, HF in 28% of patients, SVT in 10% of patients.
Maximum no. of patients was diagnosed as DCM.

Discussion

The alcohol is most frequently abused substance throughout the world. There is compelling evidence of worldwide increase in alcohol consumption and also there is an increase in consumption of alcohol even by women and adolescents.
Alcohol permeates all tissues of the body and affects most vital functions. Significant effects of alcohol on the cardiovascular system have been seen over the past 20 years. While the hearts of many alcohol addicts exhibited subclinical abnormalities, only a minority experience symptom of cardiac issues. These include dilated cardiomyopathy, heart arrhythmias, and heart failure.
Alcohol abuse has been reported as the most common identifiable etiological factor accounting up to one third of cases of dilated cardiomyopathy. Although it has been shown that average alcohol consumption and cardiovascular disease have a J-shaped relationship for all-cause mortality, heavy drinking on the irregular basis increases the risk of cardiovascular disease.

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A total no of 50 patients were included in study. Patients with hypertension, diabetes mellitus, known valvular disease, congenital heart diseases, ischemic heart disease, smokers were excluded.
Patients of age >18 years were taken, study was done in symptomatic patients with history of alcohol abuse. The aim of present study is to find out the respective value of physical examination, chest X ray PA view, ECG and echocardiography in the diagnosis of cardiac dysfunction in alcohol abuse patients.

Age
In this study 50 patients were included. The highest incidence in present study was in age group of 30-44 years (60%), least number of patients in age group of <29 years (12%).

S. No.	Study	Most Common Age Group
1	Present Study	30-44 years
2	Regan TJ Study	30-55 years
3	Laurent D *et al*.	40-59 years
4	Piano MR *et al*.	38.5-44 years
5	Maisch B Study	30-50 years

Regan TJ, in his study on alcoholic cardiomyopathy observed maximum incidence in 30-55 years group people [10]. Burch GE *et al*., study showed most common patients were adults [11].
Laurent D, *et al*. study described more common in age group of 40-59 years [12]. Piano MR *et al*. study showed that the mean age of male subjects ranged from 38.5-44 years [6].
Maisch B, described that the highest prevalence is detected in the third to fifth decade of life [13].
Fuster V, *et al*., described that Alcoholic cardiomyopathy is observed most frequently in males age 30 to 55 years with a greater than 10 year history of heavy alcohol use [14].

Sex
In this study all patients were male, no female patients, similar to other studies like Timothy J Regan in his study male predominance in patients with alcoholic cardiomyopathy [10]. Fauchier L *et al*. study showed that 48 patients were male, 2 patients were females [25]. Similar results were observed in Laurent D *et al*., Piano MR *et al*., Richard A Lange *et al*., Fernandez Sola *et al*., Urbano Marquez *et al*.

S. No.	Study	Most Common Sex
1	Present study	All patients were males
2	Fauchier L, *et al*.	48 were males, 2 patients were females
3.	Laurent D, *et al*.	Most common were males
4.	Piano MR, *et al*.	Most common were males
5.	Richard A Lange	Most common were males
6.	Timothy J Regan, *et al*.	Most common were males

Laurent D *et al*. study showed most common patients were males in patients with alcoholic cardiomyopathy [12].
Piano MR *et al*. study showed that most commonly male were affected in case of alcoholic dilated cardiomyopathy [6].
Despite the fact that women consumed less alcohol overall throughout their lifetimes than men did, Fernandez-Sola and colleagues evaluated 10 women and 26 men who were alcohol abusers and found that both groups had a similar prevalence of cardiomyopathy [16].
Similar outcomes were found in a study of 50 women and 100 men who misused alcohol, according to Urbano-Marquez. According to the authors, a woman's lifetime alcohol consumption was 60% less than a man's [17].

Presenting complaints
Of the 50 cases studied, the most frequent symptom was breathlessness in 50 patients (100%), second most frequent symptom was palpitations 43 patients (86%) and chest pain in 11 patients (22%), pedal edema in 33% patients (66%), oliguria in 26 patients (52%).
Similar to Timothy J. Regan *et al*., study described that most common symptoms were breathlessness, heart failure symptoms [10].
Fauchier L *et al*., study showed similar symptoms like breathlessness, palpitations, pedal edema [15].
Richard A Lange *et al*. showed symptoms and signs of heart failure developed due to alcoholic cardiomyopathy [18].
Fuster V, O Rourke RA *et al*. described that in the symptomatic phase both systolic and diastolic dysfunction occur. Onset of symptoms ranges from progressive exercise limitation to acute fulminate heart failure. Not infrequently paroxysmal atrial fibrillation is the initial finding [14].
Patients with alcoholic cardiomyopathy typically exhibit heart failure symptoms, such as dyspnea, orthopnea, edema, nocturia, and/or tachycardia, according to Klatsky AL *et al*. [19].

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Similar symptoms were present in the current study at the time of presentation as in studies by Fauchier L. *et al*., Klatsky AL. *et al*., Richardson A. Lange. *et al*., and Timothy J. Regan.

Physical examination

During the current study's general physical examination, 33 patients (66%), 21 patients (42%), 22 patients (44%) and 11 patients (22%), respectively, had raised JVP, S3, crepitations, organomegaly, and ascites, all of which were indicators of heart failure.

Richard A Lange *et al*. study showed the signs of heart failure in alcoholic cardiomyopathy [18].

Piano MR *et al*., described that symptomatic ACM patients have signs and symptoms of heart failure, such as elevated jugular venous pressure, S3 heart sound, pulmonary rales, and peripheral edema [6].

According to George A. *et al*., clinical heart failure symptoms like a third heart sound, an elevated jugular venous pulse, and cardiomegaly with or without rales are more common in decompensate states [19].

As demonstrated by Johnson RA *et al*., heart failure symptoms can result from either early diastolic dysfunction or later systolic dysfunction. Due to atrial fibrillation in the later stages. In the dilated atria, thrombi are common. Up to two thirds of cases [20] have mitral regurgitation.

This study's physical examination findings were comparable to those of studies by George *et al*., Piano MR *et al*., and Johnson *et al*.

In this study irregular pulse was noticed in 10 patients (20%), regular pulse was noticed in 40 patients (80%). Irregular pulse observed is due to atrial fibrillation and extra systoles.

Richard A Lange *et al*., showed that the most common ethanol induced arrhythmia is atrial fibrillation. Ethanol is of casual importance in about a third of subjects with new onset atrial fibrillation, in those younger than 65 years. Most episodes occur after binge drinking, usually on weekends or holidays, hence a term holiday heart syndrome [18].

According to Engel TR *et al*., [11] heavy drinkers who did not have cardiomyopathy or overt heart failure were found to be susceptible to atrial fibrillation and flutter [21].

Holiday heart syndrome with a higher risk of supra ventricular tachyarrhythmia in alcoholics was described by Klatsky AL *et al*. [22].

Irregular pulse is due to atrial fibrillation and extra systoles. Atrial fibrillation is most common arrhythmia in alcohol abuse patients. The present study results were correlating with other studies like Richard A Lange, Engel TR *et al*., Klatsky AL *et al*., Tirumaran *et al*., Evan W study.

Alcohol consumption has been linked to reduced vagal activity, according to Tirumaran *et al*.'s report on the universal association between heart rate variability and alcohol consumption. Although the exact cause of the positive relationship between alcohol consumption and heart rate variability is unknown, potential explanations include an increase in sympathetic activity brought on by vasodilatation or calcium entering cardiac myocytes [23].

According to Even W's study [24], ectopic beats were common and frequently originated from multiple locations.

Chest X ray PA view

In this study cardiomegaly on chest X ray PA view was noticed in 34 patients (68%), similar to other studies like Phillippeguillo *et al*. [25], and Askanas *et al*. [26], Koide *et al*. [27] observed a similar mean cardiothoracic ratio.

Although a chest radiograph is a useful tool for identifying a large heart and pulmonary vascular changes in heart failure, it frequently offers little insight into the cause, the severity of cardiac dysfunction, and complicating factors like valve dysfunction. This is why cross sectional imaging tests, typically an echocardiogram, are added to radiography in most cases.

A worse prognosis is linked to the development of cardiomegaly, which can be easily monitored using chest radiography. Coronary artery calcification may be a sign of ischemic pathology, but it is rarely found.

Koide *et al*.'s 1975 study was the first to specifically examine the amount of alcohol required to cause ACM. The authors used chest x-rays to examine the prevalence of cardiomegaly and linked alcohol use to it in a group of Japanese men who were of working age. They discovered that cardiomegaly affected 2 out of the 6 people (or 33%) whose daily alcohol intake exceeded 125 mL. In contrast, only one out of every 25 subjects who drank moderately (4%), six out of every 105 people who drank very little (5.7%), and 4.5% of those who didn't drink were found to have an enlarged heart [27].

ECG changes

In this study, ECG findings of atrial fibrillation, LVH, and sinus tachycardia were observed in 10 (20%), 6 (12%), and 12% of the patients, respectively. 5 (10%) of the patients had supraventricular tachycardia, 5 (10%) had LBBB, 6 (12%) had RBBB, 3 (6%), had ventricular ectopics, and 9 (18%) had normal ECGs.

Eliaser M *et al*., described that electrocardiographic changes giving evidence of abnormal left ventricular

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repolarisation simulating a so called "digitalis effect" and evidence of left ventricular enlargement. These changes were present in 57.4 per cent of a group of 94 chronic alcoholic persons whose average age was 42 years [28].

The abnormal electrocardiographic patterns were considered to be the result of the cumulative effects of ethanol on cardiac muscle.

Engler R, *et al*., described that Atrial fibrillation and supraventricular tachyarrhythmias are common findings in 15 to 20% of alcohol abuse patients [29]. Where, as ventricular tachycardias were rare [30] in alcohol abuse patients as described by Follansbee WP, *et al*. [30].

The ECG can show non-specific abnormalities similar to those in idiopathic DCM, according to Marriott HJL [31]. These abnormalities include complete or incomplete left bundle branch block, atrioventricular conduction disturbances, changes in the ST segment, and P wave changes.

In the study by Brigden and Robinson, a wide range of abnormalities were discovered in the ECGs of alcohol abuse patients. These abnormalities progressed over the course of the disease, and in some patients, spontaneous improvement was noted [32].

Arrhythmias were common, and in half of the patients, atrial fibrillation with rapid ventricular beats occurred. Patients with sinus rhythm and those with atrial fibrillation both frequently experienced extra ventricular systoles. Two patients experienced nodal rhythm. Twelve cases had abnormal P waves. In chronic heart failure with myocardial disease, this was typically the pattern of right and left atrial hypertrophy. Eight patients had LBBB, four had RBBB, seven had a prolonged P-R interval, one patient had a complete heart block.

In two of these cases, necropsy confirmed the absence of coronary artery disease. Pathological Q waves were present in four cases. Six cases involved left ventricular hypertrophy as evidenced by a deep S wave in the right precordial leads.

In the majority of ECGs taken from these patients, T waves were abnormal. In a group of alcoholic patients, Brigden and Robinson also discovered T wave changes but thought they were non-specific [32]. The location and degree of muscle damage are to blame for the ECG results rather than the illness itself [32].

Among 20 patients with alcoholic cardiomyopathy, Evans W described changes in the ECG; of these, abnormalities were deemed to be distinctive in 17 patients and not specific in the other 3 patients [24].

The T wave, which is a depressed dimple in 8 patients, an upright wave in 7, and a spinous wave in 2, were the only notable ECG abnormalities. The presence of extra systoles in 3 patients supported the diagnosis because they were numerous, arose from various foci, or were connected to moderate tachycardia. Six other patients also had additional systoles in addition to the characteristic symptoms. In addition to paroxysmal atrial tachycardia, atrial fibrillation was discovered in 2 patients. Due to the fact that they occurred in patients who also had heart failure, bundle branch block in 2 patients and ST segment depression in 2 patients were regarded as sinister signs [24].

S. No	ECG description	George A study No of patients (1000 patients)	Present study No of patients (50 patients)
1	Normal	820	9
2	Sinus tachycardia	86	6
3	Non specific T wave changes	40	-
4	Extrasystoles	9	3
5	Atrial fibrillation	3	10
6	First degree AV block	2	-
7	CAD	24	-
8	RBBB	3	6
9	LBBB	6	5
10	LVH	7	6
11	SVT	-	5

George A described that ECG of 1000 chronic alcoholic patients were examined and analyzed. Evidence is presented that excessive consumption of alcohol, in the absence of organic heart disease, may produce changes in the electrocardiogram. The predominant abnormalities were sinus tachycardia and nonspecific T wave changes. These abnormalities were present in the majority of the patients, provided that electrocardiograms were taken while the patients were still intoxicated. It is suggested that the changes are caused by two known effects of alcohol, stimulation of catecholamine secretion from the adrenal medulla and alteration of cell membrane permeability [19].

T wave changes were found in 20 out of 37 chronic alcoholics, according to Priest *et al*. [33]. Richard A Lange *et al*., study showed that ethanol consumption is associated with variety of atrial, ventricular arrhythmias most commonly atrial fibrillation, supra ventricular tachycardia and atrial or ventricular premature beats, ventricular tachycardia, ventricular fibrillation. Most episodes occur after binge drinking, usually on weekends or holidays, hence the term holiday heart syndrome. Electrophysiological

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testing in humans without cardiac disease has shown that ethanol enhances vulnerability to induction of atrial flutter or fibrillation. Abstinence is the main form of treatment for these ethanol-induced arrhythmias [18].

Acute ethanol consumption causes diuresis, which results in concurrent urinary sodium, potassium, and magnesium loss. The presence of myocardial fibrosis, ventricular hypertrophy, cardiomyopathy or autonomic dysfunction may also enhance the likehood of dysarrhythmias. In patients who use alcohol, it has also been observed that the Q-T interval is prolonged, that heart rate variability is decreased, that vagal modulation is diminished, and that baroreflex sensitivity is decreased [18].

Excessive alcohol consumption has been linked to cardiac arrhythmia in both healthy people and patients with pre-existing heart disease, according to Kransniqi A, Bostaca *et al*. According to their study most common type of arrhythmia were atrial fibrillation 61%, VPCS, SVT, LBBB, RBBB. These were observed in patients who had a history of consumption of alcohol >150 gm/day >10 years [34].

When the Thirumaran M *et al*. results were analyzed, it was discovered that alcohol-dependent people had significantly lower heart rate variability than normal, healthy people.

These variations are due to cardiac autonomic neuropathy, which decrease the cardiovascular performances among the alcohol subjects. Alcohol affects the cardiac autonomic function [23].

In 88 patients with dilated cardiomyopathy and normal coronary angiograms, the prognostic significance of electrocardiographic variables was retrospectively investigated, according to Yoshinori Koga *et al*. The cumulative survival rate was 73% at 2 years and 60% at 5 years during a follow-up period of an average of 3.72.9 years. Univariate life table analysis revealed that abnormal Q-waves, a QRS duration 0.12s, a cardiothoracic ratio 60%, a systolic blood pressure 110 mmHg, and a left ventricular end-diastolic pressure 15 mmHg were all significantly linked to a higher 5-year mortality rate. The following list of the main independent risk factors is the result of multivariate analysis using Cox's proportional hazards model: All patients must have abnormal Q-waves, left bundle branch block or intraventricular conduction disturbances, left ventricular end-diastolic pressure, systolic blood pressure, and the cardiothoracic ratio. Patients without left bundle branch block or intraventricular conduction disturbances must also have abnormal Q-waves, left ventricular end-diastolic pressure, and systolic blood pressure. Thus, the study showed that the electrocardiogram could offer independent prognostic predictors in people with dilated cardiomyopathy, perhaps reflecting the severity of myocardial damage [35].

The phrase "Holiday heart syndrome" refers to a phenomenon that Ettinger and colleagues (1978) studied in a group of patients who were hospitalized for arrhythmia episodes after binge drinking over the weekend or during holidays. Palpitations and breathing difficulties are signs of an acute alcohol-induced arrhythmia. Beginning after heavy drinking is typical, and symptoms typically subside with abstinence, whether or not additional targeted therapy is used [36].

Numerous arrhythmias, including premature, erratic, and rapid contractions of the upper chambers of the heart, were discovered in a study involving about 4,000 participants. Six drinks a day users had a relative risk for these conditions that was at least twice as high (Cohen *et al*., 1988) [37].

In present study alcohol abuse patients are more prone for atrial fibrillation. ECG changes like non-specific T wave abnormalities correlates with other studies like Eliaser *et al*., Priest *et al*., George A study, Yoshinori Koga *et al*., Richard A Lange *et al*., Marriott HJL study, Engler R study, Brigden & Robinson study, Kransniqi A Bostaca *et al*., Cohen *et al*.

2D ECHO findings

In this study mild LVD is observed in60% (EF;45-54%), moderate LVD in 24% (EF;30-44%), severe LVD 16% (EF;<30%).

In this study DT (Deceleration time) values >200 msec was noticed in 30% of patients, DT value between 150-200 msec was noticed in 42% of patients, DT value <150 msec was noticed in 28% of patients. Maximum no. of patients were between 150-200 msec. DT change is earliest feature of diastolic dysfunction. Present study DT values were correlating with Lazarevic AM *et al*.

In present study EPSS value of >12 was noticed in 64% of patients. Normally in DCM, EPSS value is prolonged.

About 40% of the patients who could be found in the study have LVESD values between 6 and 6.9 cm. Maximum patient presence was 52%, and LVEDD values were >7 cm. In the current study, the majority of patients had elevated LVSD and LVDD. In DCM normally LVSD, LVDD are increased.

ESV in ml maximum percentage (36%) of patients were noticed between 50-74ml, EDV in ml maximum percentage (64%) of patients were noticed between 100-174 ml.

In the present study, the majority of patients had elevated ESV and EDV. These findings were consistent with those of studies by Kupari *et al*., Kasper *et al*., Gonclaves *et al*., and Mathew *et al*.

In present study alcohol was one of the risk factors for development of Atrial fibrillation, similar to others, Kosinen P *et al*. study, Lee WK *et al*.

The majority of studies have discovered that more LV dilation and increased cardiac mass are characteristics of symptomatic ACM. In contrast to asymptomatic alcoholics, symptomatic patients had twice the size of LVESD values and 40% and 60% higher values for LVEDD and LV mass, respectively,

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according to Mathews *et al*. [34].
According to Mathew *et al*., all 11 symptomatic patients had significantly reduced left ventricular percent fractional shortening (mean 14 percent, normal range 28 to 44), significant increases in left ventricular systolic and diastolic dimensions (mean increases of 105 and 48 percent above normal, respectively), left atrial dimension (mean increase of 21 percent), and estimated left ventricular mass (mean increase of 105 percent). 15 (68%) of the 22 asymptomatic patients had significant increases in at least one of the echocardiographic parameters measured, including left ventricular mass, left ventricular dimensions, septal and left ventricular wall thicknesses, and left atrial dimension [38].
Similar to other dilated cardiomyopathies, ACM is characterized by an elevated LV mass, dilated ventricles, ventricular dysfunction, and wall thinning. These alterations are present even in the absence of coronary artery disease and nutritional deficiencies. The stage and severity of ACM may influence the extent of LV dilation as well as changes in LV mass, wall circumference, and LV function.

Amount of alcohol consumed Vs Ejection fraction values
In present study the amount of alcohol consumed in total life time by a person is inversely proportion to EF values (P value was 0.002). The amount of alcohol consumption was inversely proportion to EF values irrespective of duration of drinking. The present study results were correlating with Urbano Marquez *et al*. study, Richard A Lange study, Regan TJ *et al*., Mckenna JC *et al*. study.
The International Society and Federation of Cardiology Task Force and the World Health Organization (1995) recommend that the dominant pathophysiology or, if possible, the etiologic/pathogenic factor be used to categorize cardiomyopathies. In light of this, ACM is regarded as a dilated and specific cardiomyopathy and is frequently mentioned in relation to substances that are toxic to the myocardium. A dilated left ventricle (LV), normal or reduced LV wall thickness, and other dilated cardiomyopathies (i.e., idiopathic, viral/immune) are features of ACM. It is highly individualized and poorly understood when these abnormalities start to manifest over the course of a person's lifetime of drinking.
ACM cannot be diagnosed using any particular immune histochemical, immunologic, or other criteria, in contrast to other cardiomyopathies like immunologic cardiomyopathies. As a result, the diagnosis of ACM is frequently regarded as speculative and is typically one of exclusion. A long history of heavy alcohol abuse is the main contributing factor to ACM. High levels of alcohol consumption are related to the occurrence of ACM.
According to Mckenna JC *et al*., 100 patients with DCM drank significantly more alcohol than the 211 participants in the control group (40% vs. 24%; p=0.01), and these patients also scored higher on the CAGE questionnaire for alcohol abuse (27% vs. 16%; p=0.05). In cases, the average total lifetime consumption of alcohol expressed in units was also significantly higher [39].
According to research by Urbano-Márquez and colleagues, the total lifetime dose of alcohol was associated with a rise in LV mass and a fall in ejection fraction (EF). Even though there isn't a clear dose response relationship and there are differences in the amount of alcohol consumed and the length of alcohol abuse across studies, some generalizations about alcohol consumption and ACM can be made. A history of alcohol consumption of > 90 gm/d for > 5 years was typically present in asymptomatic alcoholic patients with changes in cardiac structure and function [14].
This is the only study to demonstrate a direct linear correlation between lifetime alcohol consumption and left ventricular mass (r = 0.42), fractional shortening (r = 0.35), and ejection fraction (r = 0.46; all P 0.001). In a cohort of 52 alcoholics, they observed a pronounced decline in the ejection fraction that was inversely correlated with the total lifetime alcohol consumption of the patients [17].
The alcoholics had a significantly lower mean ejection fraction (59 vs. 67 percent), a lower mean fraction shortening (33 vs. 38 percent), a greater mean end-diastolic diameter (51 vs. 49 mm), and a higher mean left ventricular mass (123 vs. 106 g per square meter of body-surface area) in their cardiac studies when they were compared to 20 healthy controls. None of the controls had an ejection fraction of less than 55 percent, compared to one-third of the alcoholics. Muscular strength and the estimated lifetime total ethanol dose were inversely correlated (r = -0.65; P 0.001). They came to the conclusion that striated muscle is harmed by alcohol in a dose-dependent manner and that cardiomyopathy is common in people with chronic alcoholism [17].
Left ventricular dysfunction (LVD) is a common finding in chronic alcoholic subjects, according to Kupari M. *et al*. This work examined the relationship between LV function and alcohol consumption in the general population, where low and moderate consumption predominate. Multiple linear regression was used to examine the relationships between LV measurements and alcohol consumption. The range of the daily average ethanol intake was 0 to 1.2 gm/kg of body weight (median 0.2). For the LV end systolic diameter, fractional shortening, peak early trans mitral velocity, and peak atrial trans mitral velocity, statistically significant associations with square root of daily ethanol use were discovered [40]. The regression coefficient [b] +/- SE 4.0 +/- 1.5 mm/square root of g/kg, p 0.01, and the peak atrial trans mitral velocity, b = -4.9 +/- 1.9 cm/s, respectively,
According to Lazarevic AM *et al*., alcoholics had a longer IVRT (92 +/- 11 vs. 83 +/- 7 ms, p 0.001), a longer DT (180 +/- 20 vs. 170 +/- 10 ms, p 0.01), a smaller E/A (1.25 +/- 0.34 vs. 1.40 +/- 0.32, p The

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longest period of heavy drinking was found to have the greatest impact on DT and A by multiple regression analysis [41]. In comparison to the control group, left ventricular diameter and volume were significantly higher in subjects who had used alcohol for fewer years, between 5 and 10 years. Diastolic dysfunction (impaired deceleration time) was noted as an early side effect of alcohol abuse [41].

In both sexes, increasing alcohol consumption was linked to larger left ventricular diastolic and systolic diameters as well as larger left atrial diameter, according to Gonclaves *et al*. (P0.05). Alcohol consumption increased the left ventricular mass in men (8.23.8 g per consumption category; P=0.029) and the E/E' ratio (0.820.33 per consumption category; P=0.014). Increased alcohol consumption in women was linked to a decrease in left ventricular ejection fraction (-1.90.6% per consumption category; P=0.002) [42]. They showed that increasing alcohol consumption decreased the LV relative wall thickness and increased end diastolic diameter. In contrast to eccentric remodelling, this suggests that drinking alcohol may increase LV eccentricity [42].

According to a 1986 study by Komajda M *et al*., patients with DCM who were admitted due to heart failure consumed more alcohol than those who were admitted for surgical procedures (101 mL/d vs. 64 mL/d; RR(relative risk) = 7.6; P 0.001) [43]. They noted that patients consumed more alcohol than controls did before the onset of the first symptoms (101 vs 64 ml day-1, P less than 0.001); the excess consumption included all types of alcoholic beverages (wine, beer, etc.). Wine and other alcoholic beverages both independently contributed to the relative risk (RR), which was estimated from the odds ratio and increased only among heavy drinkers (greater than or equal to 110 ml day-1; RR: 7.6, P less than 0.001). In conclusion, alcohol is the risk factor with the highest likelihood of causing DCM, but the excess risk is only present in heavy drinkers and is unaffected by the type of beverage [43].

Additionally, Gillet published a related study in which a cohort of 23 patients with DCM reported higher average daily alcohol consumption (82 g/d vs 30 g/d; P 0.001) and a longer duration of that consumption (34 vs 22 years, P 0.001) than a second group of 46 people with other types of heart disease [44].

McKenna reported a 40% incidence of binge drinking in a group of 100 DCM patients in 1998, compared to 23% in a control group of 211 healthy subjects [44]. Alcohol use is a risk factor for dilated cardiomyopathy, the study found.

Fernández-Solá *et al*. [45] analysis of an alcoholic population also revealed a higher prevalence of DCM in alcoholics than in the general population. They demonstrated that alcohol abusers had an increased risk of developing alcoholic dilated cardiomyopathy.

According to the findings of the current study, alcoholic dilated cardiomyopathy was the most prevalent type; this finding was consistent with those of studies by Fernandez Sola *et al*. and Lee WK *et al*.

According to Scott D. *et al*., LVEF and increased mitral E point separation from the interventricular septum (EPSS) are two measures used to determine the severity of LV contractility impairment. LVEF decline and higher end diastolic and end systolic volumes, regardless of the underlying cause, are linked to a worse prognosis. The enlarged LV and RV cavity and systolic dysfunction are common features of dilated cardiomyopathies. A subnormal overall ejection fraction is present, along with increased LV end diastolic and end systolic volumes, end diastolic dimensions, and overall LV mass [46, 47].

Chronic heavy ethanol consumption, according to Richard A. Lange *et al*., may cause diastolic and systolic dysfunction in the left ventricle. The amount of ethanol consumed over the course of a lifetime is correlated with the likelihood that ethanol-induced dilated cardiomyopathy will develop. The majority of men who have consumed more than 80g of alcohol daily for at least five years develop it.

Regan TJ and co. It became abundantly clear that excessive alcohol consumption led to left ventricle dysfunction, including deterioration of kinetic properties and a significant drop in the ejection fraction [48].

According to Kosinen P *et al*., about one-third of patients experiencing their first episode of atrial fibrillation do not have any apparent heart conditions, but many of them (15–30%) are heavy drinkers who frequently consume alcohol. There is no question that one of the most prevalent aetiological causes of atrial fibrillation in people of working age is alcohol [49].

In chronic alcoholics, Levi *et al*. discovered that preclinical abnormalities of left ventricular function include a prolonged pre-ejection period (PEP) and a decreased left ventricular ejection time (LVET), both of which are signs of myocardial dysfunction. A smaller stroke volume is indicated by a shorter ejection time, and a higher PEP/LVET ratio also indicates subpar myocardial function [50].

According to Kasper *et al*., echocardiography can initially show hypertrophy as well as mild, moderate, or severe depressive symptoms of cardiac function and ejection fraction. Alcoholic cardiomyopathy is characterized by left or both ventricles that are dilated and exhibit impaired contraction [51].

When compared to controls of the same age and weight, Kupari *et al*. found that the left ventricular end diastolic diameters were larger, the left ventricular mass index was higher, and the left ventricular ejection fraction was significantly lower (45%) [52].

Alcohol can have unacceptable cardiac risks in chronic high doses, leading to a dilated cardiomyopathy and an associated increased risk for cardiovascular death, according to Lee WK *et al*. Alcoholism at high doses can lower the threshold for arrhythmia as well [53].

One can exhibit cardiac toxicity at a long-term high alcohol dose. High blood alcohol levels have been

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shown to have negligible inotropic effects on the myocardium.
When compared to healthy controls, Askanas *et al*. found that asymptomatic chronic alcoholics had significantly thicker left ventricular walls, a thicker interventricular septum, and larger left ventricles [26].
Without providing information regarding the length of consumption, Kino *et al*. discovered that ventricular thickness increased when ethanol consumption exceeded 75 mL/day (60 g), and the increase was greater among subjects who consumed over 125 mL/day (100 g) [54].
In the current study, which included 50 patients who misused alcohol, we found that DCM, AF, heart failure, and SVT affected 42%, 20%, 28%, and 10% of patients, respectively. Alcoholic dilated cardiomyopathy was the most typical presentation.

Conclusions
In this cross-sectional study of 50 alcohol abuse patients with satisfied inclusion criteria were examined and ECG, Echocardiography, chest X ray PA view and other investigation were carried out for those patients. Majority of the patients were in 30 -44 years age group and all patients were males. Among 50 alcohol abuse symptomatic patients, we observed alcohol dilated cardiomyopathy in 42% of patients, atrial fibrillation observed in 20% of patients, Heart failure observed in 28% of patients, SVT observed in 10% of patients. Alcohol abuse causes myocardial function abnormalities. It is a major cause of secondary non ischemic dilated cardiomyopathy. Alcohol abuse leads to dilated cardiomyopathy, arrhythmias, heart failure, sudden deaths. Alcoholic dilated cardiomyopathy also risk factor for sudden death. Alcohol abstinence results in better prognosis.

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