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A descriptive study regarding prevalence and correlates of cardiomyopathy among patients of alcohol dependence syndrome attending a tertiary care hospital in North India

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ABSTRACT

Background

Alcoholic cardiomyopathy (ACM) is a significant cause of dilated cardiomyopathy (DCM). ACM progresses from subclinical diastolic dysfunction, detectable through advanced imaging techniques like echocardiography and MRI, to systolic dysfunction. There is a scarcity of Indian studies investigating the prevalence and characteristics of ACM. Hence, this study was conducted to assess the prevalence of cardiomyopathy among patients diagnosed with Alcohol Dependence Syndrome (ADS) and to assess reversibility of the cardiac changes observed longitudinally with 6 months abstinence from alcohol.

Methodology

The study was conducted in the Department of Psychiatry at a tertiary care hospital in North India. 80 male in-patients with newly diagnosed ADS were included in study. Transthoracic 2D Echocardiography was performed to detect any electrophysiological or structural changes in the heart. Statistical analysis was done using the Mann-Whitney Test, Chi-Square test, or unpaired T test.

Results

Cardiomyopathy was present in 20%(n=16) ADS patients. Out of 16 patients, 12(75%) had grade 1 and 4(25%) had Grade 3 CM. The patients were consuming alcohol over a mean period of 11.75 years with mean consumption of 230.29 kg of alcohol and there was a significant association seen in years of drinking with cardiomyopathy (P=0.044). The abstinence from alcohol led to an improvement of structural changes in the heart in 37.50 % of patients.

Conclusion

It becomes important to monitor heart health in chronic alcoholics. A multidisciplinary approach involving cardiologists, psychiatrists, and addiction specialists should be adopted in the management of patients with alcohol dependence syndrome.

Keywords: Acute cardiomyopathy, alcohol dependence syndrome, diastolic dysfunction, reversibility.

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INTRODUCTION

Alcoholic cardiomyopathy (ACM) is a significant cause of dilated cardiomyopathy (DCM), accounting for approximately 40% of all DCM cases.[1] The European Society of Cardiology (ESC) and the American Heart Association (AHA) classify ACM as a specific form of DCM, typically diagnosed through exclusion, as it lacks distinctive clinical or histological features.[2,3] Heavy, chronic alcohol consumption—usually more than 80 g per day over five years—has been associated with the development of ACM.[4–6] While acute binge drinking can result in transient left ventricular (LV) dysfunction, long-term alcohol abuse leads to more permanent cardiac damage, manifesting as reduced left ventricular ejection fraction (LVEF) and increased left ventricular end-diastolic diameter (LVEDD).[7,8] ACM progresses from subclinical diastolic dysfunction, detectable through advanced imaging techniques like echocardiography and MRI, to systolic dysfunction and further increases in LV size and mass.[9,10] Though alcohol dependence is a well-established cause of DCM, genetic predisposition plays a critical role in determining who will develop ACM, with studies showing that individuals with a genetic vulnerability to heart disease are more likely to be affected.[11] Globally, ACM is a major contributor to non-ischemic cardiomyopathies, with its prevalence estimated at 21-36% of such cases in the United States.[12] In India, alcohol-related cardiomyopathy accounts for approximately 16% of DCM cases.[11] However, the true prevalence of ACM may be underestimated due to the lack of comprehensive autopsies in cases of idiopathic heart failure. As alcohol consumption continues to rise, particularly in developing countries like India, ACM is becoming a growing public health concern. It is a significant risk factor for cardiovascular diseases, including hypertension, coronary artery disease, and stroke.[13,14] Despite this, there is a scarcity of Indian studies investigating the prevalence and characteristics of ACM, highlighting the need for more research in this area. This study aims to fill this gap by assessing the prevalence of cardiomyopathy among patients diagnosed with Alcohol Dependence Syndrome (ADS) and exploring the associated cardiac changes due to chronic alcohol consumption. The study also aims to assess reversibility of the cardiac changes observed longitudinally with 6 months abstinence from alcohol.

Methodology

The study was conducted in the Department of Psychiatry at a tertiary care hospital in a metro city in North India. It was a prospective observational study with a six-month follow-up period, carried out from January 2019 to March 2020. The study population included all in-patients diagnosed with Alcohol Dependence Syndrome (ADS) during the study period, based on specified inclusion and exclusion criteria. The study included freshly diagnosed cases of Alcohol Dependence Syndrome (ADS), with participants providing informed consent and being adult male patients aged over 18 years. Patients were excluded if they had pre-existing cardiac illnesses, determined through their medical history, symptoms of chest pain, coronary artery stenosis (>50%), exercise stress tests, or electrocardiogram (ECG) findings such as atrioventricular conduction abnormalities or bundle branch blocks. Additionally, patients with cardiomyopathy (CMP) diagnosed prior to alcohol consumption were excluded, provided they could recall and present previous medical reports as evidence.

The sample size was calculated as 80 by using formula as

$N \geq (i\,(1-i))/(ME/z\alpha)2$

Where Zα is value of Z at two-sided alpha error of 5%, ME is margin of error as 0.10 and i is incidence rate. Based on Fang W et al. (2018)[15] who reported that ACM accounts for 40% of DCM cases, and Rudrajit Paul et al.[16], who observed an incidence of DCM at 6.95/100,000/year, the incidence of ACM was estimated at 2.78/100,000/year whereas non response rate was taken as 5%. After obtaining informed written consent, 80 male in-patients with newly diagnosed ADS, according to ICD-10 criteria, were evaluated for cardiomyopathy. These patients were admitted for alcohol detoxification, during which demographic data (age, alcohol use history, withdrawal symptoms) were recorded.

Following detoxification and stabilization, patients underwent a four- to six-week treatment regimen including:

- Detoxification with benzodiazepines (Lorazepam or Chlordiazepoxide) based on liver function tests.
- Naltrexone, Acamprosate, Baclofen, or Fluoxetine (in case of comorbid depression) during de-addiction.

Patients were evaluated for cardiac changes through various diagnostic tools:

- MCV, SGOT, SGPT, and GGT levels were tested to assess target organ damage.
- Ultrasound (USG) of the abdomen was performed to check for fatty liver changes.
- Cardiac assessments were conducted using ECG, Chest X-ray PA view, and Transthoracic 2D Echocardiography, performed by a cardiologist to detect any electrophysiological or structural changes in the heart. Cardiomyopathy was classified based on echocardiographic findings.

Upon completion of treatment and data collection, patients were discharged with advice on alcohol abstinence and maintenance medications. They were re-evaluated after six months for a detailed review, including history, examination, and repeat investigations of cardiac parameters (systolic and diastolic function). Data were entered into Microsoft Excel and analyzed using SPSS version 21.0. Categorical variables were presented as numbers and percentages, while continuous variables were presented as mean ± SD or median. Statistical analysis was done using the Mann-Whitney Test, Chi-Square test, or Fisher's Exact test, with a p-value of <0.05 considered statistically significant. No interventions were introduced specifically for the study, and patients received standard care. Those with significant cardiac abnormalities requiring treatment were excluded. Institutional ethics committee approval was obtained, and confidentiality of patient data was maintained. Written informed consent was secured before enrollment.

Results

In the present study, 80 patients diagnosed with ADS were included in the study. The sociodemographic charaterstics like age, BMI, family history ete. are described in Table 1.

Table 1Socio demographic and alcohol use characteristics of patients

Age(years)	**Frequency**	**Percentage**
<=30	20	25.00%
31-40	49	61.25%
>40	11	13.75%
Body mass index(kg/m²)	**Frequency**	**Percentage**
Normal{18.5-24.9}	47	58.75%
Overweight{25-29.9}	32	40.00%
Obese{>=30}	1	1.25%
Family history		
Nil	72 (100%)	90 %
Present	8 (100%)	10 %
Pattern of use		
Binge	5	6.25%
Regular	75	93.75%
Years of drinking	Mean ± SD	11.75 ± 4.92
Age at dependence(years)	Mean ± SD	30.74 ± 4.45
Years since dependence	Mean ± SD	4.75 ± 2.51
Life time alcohol use(kg)	Mean ± SD	230.29 ± 141.75

None of the patients had EF<50% and hence systolic dysfunction. Mean value of IVSd(mm), PWd(mm), IVSs(mm), LVIDs(mm), LVIDd(mm), stroke volume(ml) and EF(%) of study subjects was 9.58 ± 1.85, 9.26 ± 2.37, 11.93 ± 3.8, 29.31 ± 5.33, 45.02 ± 9.21, 69.22 ± 1.74 and 65.41 ± 7.8 with median(25th-75th percentile) of 9.1(8.2-11.325), 9(8-10.4), 11.9(9-13.42), 29.05(25.7-33.25), 45.3(42.6-51), 68.5(68-69.125) and 63.95(58.375-71.2) respectively as shown in Table 2.

Table 2Distribution of systolic function of study subjects

Systolic function	Mean ± SD	Median(25th-75th percentile)	Range
IVSd(mm)	9.58 ± 1.85	9.1(8.2-11.325)	5-13.7
PWd(mm)	9.26 ± 2.37	9(8-10.4)	5-18.2
IVSs(mm)	11.93 ± 3.8	11.9(9-13.42)	5-23.8
LVIDs(mm)	29.31 ± 5.33	29.05(25.7-33.25)	11.9-39.4
LVIDd(mm)	45.02 ± 9.21	45.3(42.6-51)	4.17-59.3
Stroke volume(ml)	69.22 ± 1.74	68.5(68-69.125)	68-75
EF(%)	65.41 ± 7.8	63.95(58.375-71.2)	55-84.5
EF<50%	Nil		
Systolic dysfunction	Nil		

Diastolic dysfunction was present in only 16 out of 80 patients. Mean value of E/A ratio and e' of study subjects was 1.35 ± 0.38 and 12.33 ± 3.53 with median (25th-75th percentile) of 1.35(1.198-1.558) and 12.3(9.95-14.85) respectively. In the majority (80.00%) of patients, diastolic dysfunction was absent. In the present study, out of 16 patients of alcohol CMP, in majority 12(75.00%) of patients, grade was 1, with Grade 2 in 0 patient and Grade 3 in only 4(25%) out of 16 patients as shown in Table 3.

Table 3: Distribution of diastolic function of study subjects

Diastolic function	Mean ± SD	Median(25th-75th percentile)	Range
E/A ratio	1.35 ± 0.38	1.35(1.198-1.558)	0.59-2.5
e'	12.33 ± 3.53	12.3(9.95-14.85)	6.3-19.5

The table 4 shows the association between alcohol CMP with various demographic factors and blood investigations. Most participants are between 31-40 years (49 total), with 24.49% reporting CMP. The association between age and alcohol CMP is not statistically significant (P = 0.472). Most participants have a normal BMI (47 total), and 19.15% of this group report CMP. Among those who are overweight, 18.75% report CMP. The only obese participant reports CMP (100%). The BMI does not show a significant association with alcohol CMP (P = 0.268).The association between family history and alcohol CMP is not statistically significant (P = 0.576).Participants with deranged MCV (66 total)

show a lower proportion of CMP (13.64%).A significantly higher proportion (50%) of participants with normal MCV report CMP. The association between MCV and alcohol CMP is statistically significant (P = 0.002). Other factors such as GGT, ALT, AST did not show statistically significant associations with CMP in the study.

Table 4: Association of alcohol CMP with demographic and blood investigations

Variable	Distribution	No (n=64)	Yes (n=16)	Total	P value
Age	18-30	18 (90%)	2 (10%)	20 (100%)	0.472
	31-40	37 (75.51%)	12 (24.49%)	49 (100%)	
	>40	9 (81.82%)	2 (18.18%)	11 (100%)	
Body mass index(kg/m²)	Normal{18.5-24.9}	38 (80.85%)	9 (19.15%)	47 (100%)	0.268
	Overweight{25-29.9}	26 (81.25%)	6 (18.75%)	32 (100%)	
	Obese{>=30}	0 (0%)	1 (100%)	1 (100%)	
Family history	Nil	57 (79.17%)	15 (20.83%)	72 (100%)	0.576
	Present	7 (87.50%)	1 (12.50%)	8 (100%)	
MCV	Deranged	57 (86.36%)	9 (13.64%)	66 (100%)	0.002
	Normal	7 (50%)	7 (50%)	14 (100%)	
Gamma Glutamyl transferase (15-85 IU/L)	Deranged	54 (79.41%)	14 (20.59%)	68 (100%)	0.754
	Normal	10 (83.33%)	2 (16.67%)	12 (100%)	
Aspartate Aminotransferase (10-40 IU/L)	Deranged	59 (80.82%)	14 (19.18%)	73 (100%)	0.622
	Normal	10 (83.33%)	2 (16.67%)	12 (100%)	
Alanine Aminotransferase (10-40 IU/L)	Deranged	58 (79.45%)	15 (20.55%)	73 (100%)	0.692
	Normal	6 (85.71%)	1 (14.29%)	7 (100%)	

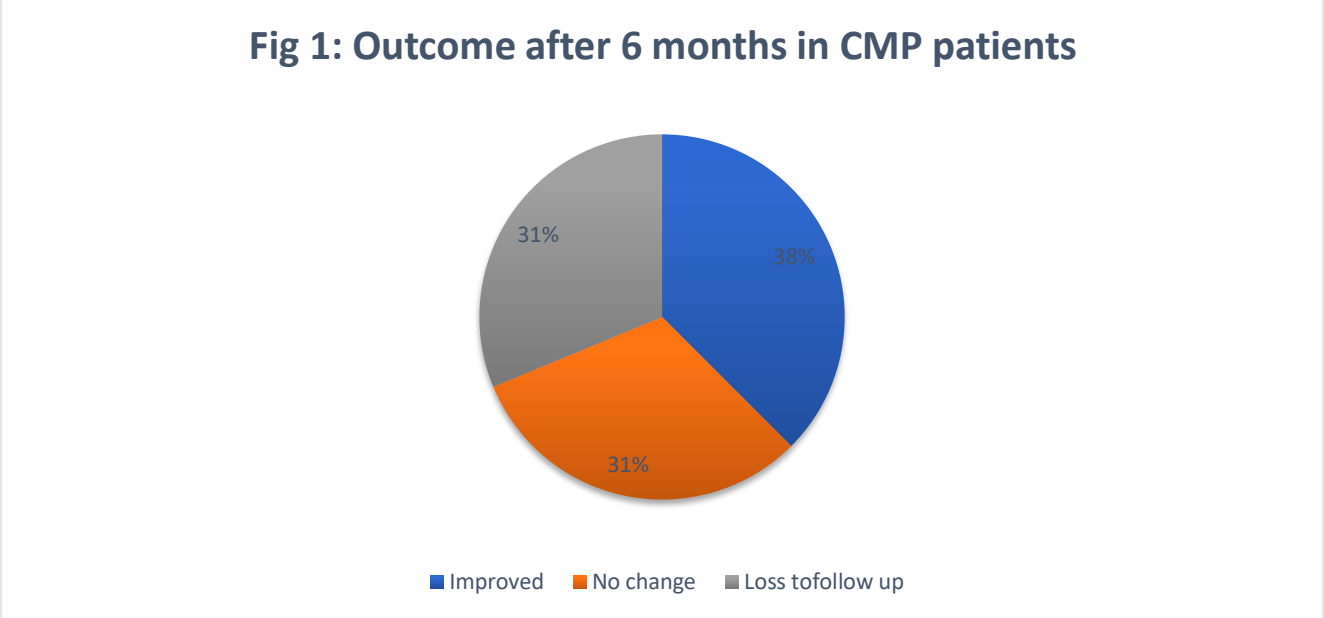
The Table 5 explores the association between CMP and various clinical profiles and alcohol consumption patterns. The years of drinking is the only variable that shows a statistically significant association with CMP (P = 0.044), indicating that individuals with CMP have been drinking for a longer period. Other factors, such as liver span, lifetime alcohol use, age at dependence, years since dependence and CIWA-Ar score, do not show significant associations with CMP. There is a near-significant association with USG abdomen echotexture (P = 0.094), which might indicate some relevance but requires more data for confirmation.

Table 5Table 5: Association of CMP with clinical profile and alcohol use

Variable	Characteristics	No (n=64)	Yes (n=16)	P value
USG abdomen Echotexture	Normal	35 (87.50%)	5 (12.50%)	0.094[1]
	Abnormal	29 (72.50%)	11 (27.50%)	
Liver span (cm)	Mean ± SD	13.94 ± 0.31	13.82 ± 0.42	0.202 [2]
Lifetime alcohol use (kg)	Mean ± SD	219.25 ± 142.16	274.42 ± 135.42	0.165 [2]
Pattern of use	Binge	5 (83.3%)	1 (16.7%)	0.83 [1]
	Regular	59 (79.7%)	15 (20.3%)	
Years of drinking	Mean ± SD*	11.22 ± 4.62	13.98 ± 5.64	0.044 [2]
Age at dependence(years)	Mean ± SD	30.8 ± 4.72	30.5 ± 3.2	0.81[2]
Years since dependence	Mean ± SD	5.81 ± 2.59	4.75 ± 2.51	0.145[3]
Initial CIWA-Ar score	Mean ± SD	10.58 ± 6.67	12.06 ± 6.86	0.432[3]

*1= Chi Square test, 2=unpaired T test, 3=Mann Whitney U test,*indicates p value<0.05*

In present study, in 6 (38 %) of patients, outcome after 6 months in cardiomyopathy patients improved. No change was seen in only 5(31%) out of 16 patients and 5 (31%) patients were lost to follow up as shown in Fig 1.



DISCUSSION

The current study examined the prevalence of CMP in alcoholic patients and it also studies. The association of CMP with various factors such as duration of alcohol consumption, amount of alcohol use in detail among 80 patients of ADS, it was found that CM was present in 20%(n=16) patients. Out of 16 patients of ACM, 12(75%) had grade 1 and 4(25%) had Grade 3 CM with none having Grade 2 CM. The prevalence of ACM as seen in this study was slightly less that reported in study by Fernández-Solà J et al.[17] Their study was conducted on 112 alcoholics and the reported incidence was 31.25%. Shaaban A et al (2020) estimates the prevalence of alcoholic cardiomyopathy in addiction units to be around 21-32%.[18] Their reported prevalence was comparable to our study. The prevalence of ACM among alcoholics thus varies from 20-32% depending upon the different strata of population (Age), family history and the lifestyles.[17,18] It was seen that 20% among the freshly diagnosed ADS patients had diastolic dysfunction as per the ECHO parameters. Compared to the present study, Fernández-Solà J et al,[17] reported that out of 35 patients with alcoholic cardiomyopathy, 23.33% had diastolic function impairment. Park et al[19] reported a further association that compared to occasional and light drinker groups, heavy and very heavy drinker groups had the higher *E*/*e*' ratio, the lower *E*/*A* ratio and septal *e*' velocity. The current study and other previous study results show that cardiac abnormalities can be subtle and only diastolic dysfunction can occur in patients with ACM where they may have normal systolic function. This also forms the basis of defining the natural history of ACM where ACM is considered to be a chronic disease that initiates with a subclinical phase in which the only change observed is left ventricular diastolic dysfunction;[17] which is followed by the systolic dysfunction phase, with a gradual decrease in left ventricular ejection fraction (LVEF).[20] However since all cases in the present study had LVEF more than 50%, systolic dysfunction phase might have not appeared among our cases. In the present study, the mean age (SD) of ADS patients was 35.46 (5.8) years with all males in the population . Similar age of patients with alcohol dependence syndrome was reported by K. Chatterjee et al.,[21] where mean age was 36.4 years. No significant association was seen in age(years) with cardiomyopathy (p >.05), which is in line with the study by Wang et al[22] as no such association of ACM was found with age. Number of patients with cardiomyopathy was significantly higher in patients with normal MCV (70-90 fl) (50%) as compared to patients with deranged MCV (70-90 fl) (13.64%). (p value = 0.002). Thus, there was significant association of MCV with ACM. Similarly, Wang et al[22] found that the ACM group had significantly higher levels of mean corpuscular volume than the non-ACM group (96.9 ±6.7 vs. 88.9 ±4.2, P=0.001). These findings indicate the usefulness of laboratory tests for providing indirect evidence of heavy drinking among patients who have heart failure. There was no significant association of LFT (AST, ALT, GGT and liver span) with ACM (P>0.05). Among the previous studies, Wang et al[22] reported that the ACM group had significantly higher levels of GGT. Additionally, the differences in lactate dehydrogenase approached statistical significance between ACM and non-ACM groups. GGT test is commonly used as a laboratory marker of heavy alcohol consumption because this test is less expensive as well as extensively accessible. There is a correlation between its level and the amount of alcohol consumed. However, the efficacy is challenged because it is also assessed in hepatobiliary disease, microsomal system-inducing drugs, and heart failure.[22] Among the other parameters, even their study found no significant association with ACM. The occurrence of ACM has been associated with various alcohol related factors. Among the alcohol consumption related parameters, we found a significant association between years of drinking and cardiomyopathy (p value <.05). However, we found no association of occurrence of CMP with pattern of alcohol use, age of onset, years of dependence and Initial CIWA-Ar score.(p>0.05).The findings were in line with the studies by Mathews et al.[23] and Urbano-Marquez et al.61[20] who found that among alcoholics consuming the same amount of alcohol, those with a longer duration of drinking had more ACM. Another study by Lazarevic AM et al,[10] examining the duration of alcohol use reported that LV dilation occurs within 5–9 years in those consuming >90 g alcohol/day for ≥ 4 days/week and precedes the development of diastolic dysfunction and LV enlargement, which occurred with drinking durations between 10 and 15 years.[10] A prospective, cross-sectional study by Urbano-Marquez et al.[20] reported that among

alcoholics (n = 52) there was a significant negative correlation (r = −0.46, p < 0.001) between ejection fraction and lifetime alcohol intake and a positive correlation (r = 0.42, p < 0.001) between LV mass and lifetime alcohol consumption signifying that increased duration of alcohol consumption significantly affects the heart.-For many years, the potential reversibility of ACM has been a subject of medical interest. In the present study, the patients were treated with drugs such as BZDs, T Naltrexone, T Acamprosate and group therapy. The outcome after 6 months in cardiomyopathy improved in 37.5% patients with no change seen in 5 out of 16 patients. All those 5 patients who did not show improvement had not abstained from alcohol whereas the 6 patients that improved showed complete abstinence. The reasons for continued alcohol use in those 5 patients were discontinuation of medication, no follow up at psy OPD, craving & withdrawal. Nicolás et al.[24] also found that complete abstinence among ACM patients helped them to normalize mean LVEF in the follow-up period. Fauchier et al.[5] also observed a similar long-term LVEF increase among ACM patients after detoxification and management. The mechanism behind the reversibility rests on the pathogenesis of ACM, which is currently considered to be a direct toxic effect of alcohol through multifactorial and synergistic mechanisms which include Ca2+-dependent signal transduction changes affecting myocardial contractility, the antioxidant and proinflammatory effects of alcohol and acetaldehyde, changes in the synthesis of structural and contractile proteins, and apoptosis induction, with consequent cardiomyocyte loss and replacement by subendocardial and interstitial fibrosis.7,100 This also explains the cumulative effects of alcohol with increasing duration of consumption and decreasing effects with abstinence or reduction of alcohol. The reversibility of ACM shows a positive hope of treatment in such patients and may help decrease morbidity and mortality in them. The thorough monitoring of both systolic and diastolic parameters by ECHO holds the key for an early diagnosis and treatment. This also warrants a combined modality management approach of psychiatry and cardiology department for cases of ACM for a better outcome.

Limitations

The study only included male subjects, which limits the generalizability of the findings to female patients. The effects of chronic alcoholism on cardiac functions in women could not be evaluated. The absence of a control group consisting of healthy individuals or patients with other causes of cardiomyopathy made it difficult to compare echocardiographic parameters and assess the specific impact of alcohol on cardiac health. The study's sample size of 80 patients may not be large enough to draw definitive conclusions, and larger studies would be needed to validate the findings.

Conclusion

The conclusion of the study indicates that cardiomyopathy was present in 20% of the study subjects, with 75% having grade 1 and 25% having grade 3 cardiomyopathy. The duration of alcohol consumption showed a significant association with cardiomyopathy, highlighting the importance of monitoring heart health in chronic alcoholics. Notably, abstinence from alcohol led to an improvement of structural changes in the heart.

Recommendation

It is recommended that future studies should include a more diverse population by incorporating female participants to assess the effects of chronic alcohol consumption on cardiac health in women. The studies with longer follow-up periods beyond 6 months should be conducted to better understand the long-term effects of alcohol abstinence on cardiac structure and function. Future research should include control groups of healthy individuals and patients with other causes of cardiomyopathy. This will allow for more robust comparisons and a clearer understanding of the specific impact of alcohol on cardiac health. A multidisciplinary approach involving cardiologists, psychiatrists, and addiction specialists should be adopted in the management of patients with alcohol dependence syndrome. This can ensure comprehensive care that addresses both cardiac health and addiction treatment.

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