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ORIGINAL RESEARCH

Biochemical Indicators and Cardiac Function Tests in Chronic Alcohol Abuse

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ABSTRACT

Background: Chronic alcohol abuse is a significant risk factor for the development of alcoholic cardiomyopathy, characterized by impaired cardiac function and increased risk of heart failure. This study aimed to investigate the utility of biochemical indicators and cardiac function tests in assessing the extent and severity of cardiac dysfunction in patients with chronic alcohol abuse. **Methods**: A prospective observational study was conducted on 100 patients with a history of chronic alcohol abuse. Patients underwent biochemical testing, including liver enzymes (AST, ALT, and GGT) and markers of cardiac injury (troponin I and BNP), and cardiac function tests, including echocardiography and cardiac magnetic resonance imaging (CMR). Patients were divided into two groups based on the presence or absence of alcoholic cardiomyopathy. **Results**: Thirty patients (30%) were diagnosed with alcoholic cardiomyopathy. Patients with alcoholic cardiomyopathy had significantly higher levels of AST (85 [60-120] vs. 45 [30-65] U/L, $p<0.001$), ALT (70 [50-95] vs. 40 [25-60] U/L, $p<0.001$), GGT (150 [110-220] vs. 90 [60-130] U/L, $p<0.001$), troponin I (0.08 [0.05-0.12] vs. 0.02 [0.01-0.04] ng/mL, $p<0.001$), and BNP (180 [120-250] vs. 60 [40-90] pg/mL, $p<0.001$) compared to those without alcoholic cardiomyopathy. Echocardiographic and CMR parameters showed significant differences in LVEF, left ventricular dimensions, and diastolic function between the two groups ($p<0.001$ for all comparisons). Logistic regression analysis identified several independent predictors of alcoholic cardiomyopathy, including age, male gender, duration and quantity of alcohol consumption, liver enzymes, markers of cardiac injury, and echocardiographic parameters. **Conclusion**: Biochemical indicators and cardiac function tests are valuable tools in assessing the extent and severity of cardiac dysfunction in patients with chronic alcohol abuse. The identified predictors of alcoholic cardiomyopathy can aid in the early detection and risk stratification of these patients, guiding targeted prevention and treatment strategies.

Keywords: Alcoholic Cardiomyopathy, Biochemical Indicators, Cardiac Function Tests, Echocardiography, Cardiac Magnetic Resonance Imaging

INTRODUCTION

Chronic alcohol abuse is a significant global health problem, with an estimated 283 million people aged 15 years and older suffering from alcohol use disorders worldwide in 2016.[1] Alcohol abuse has been linked to a wide range of adverse health effects, including liver disease, cancer, and cardiovascular disease.[2] In particular, the impact of chronic alcohol abuse on cardiac function has been extensively studied, with evidence suggesting that alcohol abuse can lead to the development of alcoholic cardiomyopathy, a form of dilated cardiomyopathy characterized by impaired cardiac function and increased risk of heart failure.[3]The pathophysiology of alcoholic cardiomyopathy is complex and multifactorial, involving direct toxic effects of alcohol on cardiomyocytes, oxidative stress, mitochondrial dysfunction, and neurohumoral activation.[4] These pathophysiological changes can lead to structural and functional alterations in the heart, including left

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ventricular dilatation, impaired contractility, and diastolic dysfunction.[5] Early detection and diagnosis of alcoholic cardiomyopathy is critical for initiating appropriate interventions and improving patient outcomes.Biochemical indicators and cardiac function tests play a crucial role in the assessment and monitoring of patients with chronic alcohol abuse. These tests can provide valuable insights into the extent and severity of alcohol-induced cardiac damage, as well as guide treatment decisions and monitor response to therapy. Commonly used biochemical indicators in the context of alcohol abuse include liver enzymes, such as aspartate aminotransferase (AST), alanine aminotransferase (ALT), and gamma-glutamyl transferase (GGT), which can reflect the degree of alcohol-induced liver damage.[6] Additionally, markers of cardiac injury and dysfunction, such as troponins and B-type natriuretic peptide (BNP), have been shown to be elevated in patients with alcoholic cardiomyopathy.[7,8]Cardiac function tests, including electrocardiography (ECG), echocardiography, and cardiac magnetic resonance imaging (CMR), are essential tools for evaluating the structural and functional changes associated with alcoholic cardiomyopathy. ECG can detect rhythm disturbances, conduction abnormalities, and signs of left ventricular hypertrophy, which are common findings in patients with chronic alcohol abuse.[9] Echocardiography is a widely used imaging modality that allows for the assessment of left ventricular size, wall thickness, and systolic and diastolic function.[10] CMR provides detailed information on cardiac morphology, function, and tissue characterization, and has emerged as a valuable tool for the diagnosis and monitoring of alcoholic cardiomyopathy.[11]Several studies have investigated the utility of biochemical indicators and cardiac function tests in the assessment of patients with chronic alcohol abuse. A prospective observational study by Fernandez-Sola et al.[12] evaluated the prevalence and characteristics of alcoholic cardiomyopathy in a cohort of 52 patients with chronic alcohol abuse. The study found that 29% of patients had evidence of alcoholic cardiomyopathy based on echocardiographic criteria, and that these patients had significantly higher levels of AST, ALT, and GGT compared to those without cardiomyopathy. The authors concluded that liver enzymes could serve as potential markers for identifying patients at risk of developing alcoholic cardiomyopathy.Another study by Urbano-Marquez et al.[13]investigated the relationship between lifetime alcohol consumption and cardiac function in a sample of 129 chronic alcoholics. The study utilized echocardiography to assess left ventricular function and found a significant correlation between total lifetime dose of ethanol and left ventricular mass, wall thickness, and end-diastolic diameter. The authors also observed a dose-dependent relationship between alcohol consumption and the prevalence of left ventricular hypertrophy and diastolic dysfunction, highlighting the cumulative effects of alcohol abuse on cardiac structure and function.A systematic review and meta-analysis by Piano et al.[14] examined the association between alcohol consumption and risk of heart failure. The analysis included 22 prospective studies with over 600,000 participants and found that heavy alcohol consumption (defined as >14 drinks per week for men and >7 drinks per week for women) was associated with a significantly increased risk of heart failure compared to no alcohol consumption (relative risk: 1.75, 95% confidence interval: 1.39-2.21). The authors also noted a J-shaped relationship between alcohol consumption and heart failure risk, with light to moderate drinking conferring a lower risk compared to abstinence or heavy drinking.

Recent advancements in cardiac imaging techniques have further enhanced the ability to detect and characterize alcohol-induced cardiac damage. A study by Lazăret al.[15]evaluated the role of speckle tracking echocardiography in detecting subclinical left ventricular dysfunction in patients with chronic alcohol abuse. The study included 50 chronic alcoholics and 50 healthy controls and found that alcoholic patients had significantly lower global longitudinal strain (GLS) values compared to controls, despite having preserved left ventricular ejection fraction. The authors suggested that GLS could serve as a sensitive marker for detecting early cardiac dysfunction in patients with chronic alcohol abuse.In conclusion, chronic alcohol abuse is a significant risk factor for the development of alcoholic cardiomyopathy and associated cardiac dysfunction. Biochemical indicators, such as liver enzymes and markers of cardiac injury, and cardiac function tests, including ECG, echocardiography, and CMR, play a crucial role in the assessment and monitoring of patients with chronic alcohol abuse. Prospective studies have demonstrated the utility of these tools in identifying patients at risk of developing alcohol-induced cardiac damage and guiding treatment decisions. As the burden of alcohol abuse continues to grow worldwide, further research is needed to refine the use of biochemical indicators and cardiac function tests in the early detection, risk stratification, and management of patients with alcoholic cardiomyopathy.

AIMS AND OBJECTIVES

The primary aim of this prospective observational study was to investigate the utility of biochemical indicators and cardiac function tests in assessing the extent and severity of cardiac dysfunction in patients with chronic alcohol abuse. The specific objectives were to evaluate the prevalence of alcoholic cardiomyopathy in a cohort of chronic alcohol abusers, examine the relationship between liver enzymes and markers of cardiac injury with echocardiographic parameters of cardiac function, and identify potential biochemical and imaging predictors of alcohol-induced cardiac damage.

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MATERIALS AND METHODS

Study Design and Patient Population

This prospective observational study was conducted over a period of one year at a tertiary care center. A total of 100 patients with a history of chronic alcohol abuse were enrolled in the study. Chronic alcohol abuse was defined as the consumption of more than 80 grams of ethanol per day for men and 40 grams per day for women for at least 5 years. Patients were excluded if they had a history of congenital heart disease, valvular heart disease, coronary artery disease, or any other significant comorbidities that could affect cardiac function.

Data Collection and Clinical Assessment

Detailed medical history, including duration and quantity of alcohol consumption, was obtained from all participants. Physical examination, including vital signs and cardiovascular assessment, was performed by experienced clinicians. Blood samples were collected for the measurement of liver enzymes (AST, ALT, and GGT), markers of cardiac injury (troponin I and BNP), and other routine laboratory tests.

Cardiac Function Tests

All participants underwent a comprehensive cardiac evaluation, including 12-lead electrocardiography (ECG), transthoracic echocardiography, and cardiac magnetic resonance imaging (CMR). ECG was performed to assess cardiac rhythm, conduction abnormalities, and signs of left ventricular hypertrophy. Echocardiography was used to evaluate left ventricular size, wall thickness, and systolic and diastolic function. Left ventricular ejection fraction (LVEF) was calculated using the biplane Simpson's method. Diastolic function was assessed using pulsed-wave Doppler and tissue Doppler imaging. CMR was performed to provide detailed information on cardiac morphology, function, and tissue characterization. Left ventricular mass, volumes, and ejection fraction were quantified using standard protocols.

Diagnosis of Alcoholic Cardiomyopathy

The diagnosis of alcoholic cardiomyopathy was based on a combination of clinical, biochemical, and imaging findings. Patients were considered to have alcoholic cardiomyopathy if they had a history of chronic alcohol abuse, evidence of cardiac dysfunction on echocardiography (LVEF <50% or diastolic dysfunction), and no other identifiable causes of cardiomyopathy.

Statistical Analysis

Continuous variables were expressed as mean ± standard deviation or median (interquartile range), depending on their distribution. Categorical variables were presented as frequencies and percentages. Comparisons between groups were performed using Student's t-test or Mann-Whitney U test for continuous variables and chi-square test or Fisher's exact test for categorical variables. Correlations between biochemical indicators and echocardiographic parameters were assessed using Pearson's or Spearman's correlation coefficients. Logistic regression analysis was used to identify independent predictors of alcoholic cardiomyopathy. A p-value of <0.05 was considered statistically significant. All statistical analyses were performed using SPSS software version 25.0 (IBM Corp., Armonk, NY, USA).

Ethical Considerations

The study protocol was approved by the Institutional Review Board of the hospital, and written informed consent was obtained from all participants. The study was conducted in accordance with the principles of the Declaration of Helsinki and adhered to Good Clinical Practice guidelines.

RESULTS

The study included 100 patients with a history of chronic alcohol abuse. The mean age of the participants was 48.5 ± 10.2 years, and the majority were male (85%). The median duration of alcohol abuse was 12 years (IQR: 8-18 years), and the median quantity of alcohol consumption was 120 grams per day (IQR: 90-160 grams per day). Comorbidities among the participants included hypertension (30%), diabetes mellitus (15%), and dyslipidemia (25%). The mean heart rate was 88 ± 12 bpm, while the mean systolic and diastolic blood pressures were 135 ± 18 mmHg and 85 ± 12 mmHg, respectively (Table 1).Thirty patients (30%) were diagnosed with alcoholic cardiomyopathy based on the study criteria. Patients with alcoholic cardiomyopathy had significantly higher levels of liver enzymes compared to those without alcoholic cardiomyopathy. The median AST, ALT, and GGT levels were 85 U/L (IQR: 60-120 U/L), 70 U/L (IQR: 50-95 U/L), and 150 U/L (IQR: 110-220 U/L) in the alcoholic cardiomyopathy group, compared to 45 U/L (IQR: 30-65 U/L), 40 U/L (IQR: 25-60 U/L), and 90 U/L (IQR: 60-130 U/L) in the group without alcoholic cardiomyopathy, respectively ($p<0.001$ for all comparisons). Similarly, markers of cardiac injury were significantly elevated in patients with alcoholic cardiomyopathy. The median troponin I and BNP levels were 0.08 ng/mL (IQR: 0.05-0.12 ng/mL) and 180 pg/mL (IQR: 120-250 pg/mL) in the alcoholic cardiomyopathy group, compared to 0.02 ng/mL (IQR: 0.01-0.04 ng/mL) and 60 pg/mL (IQR: 40-90 pg/mL) in the group without alcoholic cardiomyopathy, respectively ($p<0.001$ for both comparisons) (Table 2).Echocardiographic parameters were significantly different between patients with and without alcoholic cardiomyopathy. The mean LVEF was significantly lower in the alcoholic cardiomyopathy group (35 ± 8%) compared to the group without alcoholic cardiomyopathy (58 ± 6%, $p<0.001$). Left ventricular end-diastolic and end-systolic diameters were significantly larger in patients

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with alcoholic cardiomyopathy (62 ± 6 mm and 48 ± 5 mm, respectively) compared to those without alcoholic cardiomyopathy (50 ± 4 mm and 32 ± 3 mm, respectively; p<0.001 for both comparisons). Interventricular septum and left ventricular posterior wall thickness were also significantly greater in the alcoholic cardiomyopathy group (12 ± 2 mm and 11 ± 2 mm, respectively) compared to the group without alcoholic cardiomyopathy (10 ± 1 mm and 9 ± 1 mm, respectively; p=0.001 for both comparisons). Diastolic function parameters, including E/A ratio and E/e' ratio, were significantly different between the two groups. The mean E/A ratio was lower (1.2 ± 0.4 vs. 1.5 ± 0.3, p=0.002), and the mean E/e' ratio was higher (15 ± 4 vs. 8 ± 2, p<0.001) in patients with alcoholic cardiomyopathy compared to those without alcoholic cardiomyopathy (Table 3).CMR parameters also demonstrated significant differences between the two groups. Left ventricular mass, end-diastolic volume, and end-systolic volume were significantly higher in patients with alcoholic cardiomyopathy (220 ± 40 g, 180 ± 30 mL, and 120 ± 25 mL, respectively) compared to those without alcoholic cardiomyopathy (150 ± 30 g, 120 ± 20 mL, and 50 ± 10 mL, respectively; p<0.001 for all comparisons). The mean LVEF was significantly lower in the alcoholic cardiomyopathy group (34 ± 7%) compared to the group without alcoholic cardiomyopathy (59 ± 5%, p<0.001) (Table 4).Correlation analysis revealed significant associations between biochemical indicators and echocardiographic parameters. AST, ALT, GGT, troponin I, and BNP levels were negatively correlated with LVEF (r=-0.65, -0.60, -0.55, -0.70, and -0.75, respectively; p<0.001 for all correlations) and E/A ratio (r=-0.45, -0.40, -0.35, -0.50, and -0.55, respectively; p<0.001 for all correlations except GGT, where p=0.002). These biochemical indicators were positively correlated with E/e' ratio (r=0.55, 0.50, 0.45, 0.60, and 0.65, respectively; p<0.001 for all correlations) (Table 5).Logistic regression analysis identified several independent predictors of alcoholic cardiomyopathy. Age (OR: 1.05, 95% CI: 1.01-1.10, p=0.02), male gender (OR: 2.50, 95% CI: 1.10-5.80, p=0.03), duration of alcohol abuse (OR: 1.15 per year increase, 95% CI: 1.05-1.25, p=0.002), quantity of alcohol consumption (OR: 1.20 per 10 g/day increase, 95% CI: 1.10-1.30, p<0.001), AST (OR: 1.25 per 10 U/L increase, 95% CI: 1.10-1.40, p<0.001), ALT (OR: 1.20 per 10 U/L increase, 95% CI: 1.05-1.35, p=0.005), GGT (OR: 1.15 per 10 U/L increase, 95% CI: 1.05-1.25, p=0.002), troponin I (OR: 1.40 per 0.01 ng/mL increase, 95% CI: 1.20-1.60, p<0.001), BNP (OR: 1.30 per 10 pg/mL increase, 95% CI: 1.15-1.45, p<0.001), LVEF (OR: 2.00 per 5% decrease, 95% CI: 1.50-2.70, p<0.001), E/A ratio (OR: 1.25 per 0.1 decrease, 95% CI: 1.10-1.45, p=0.001), and E/e' ratio (OR: 1.35 per 1 unit increase, 95% CI: 1.20-1.55, p<0.001) were all significant predictors of alcoholic cardiomyopathy (Table 6).

Table 1: Baseline characteristics of the study population

Characteristic	Value
Age (years), mean ± SD	48.5 ± 10.2
Gender, n (%)	
Male	85 (85%)
Female	15 (15%)
Duration of alcohol abuse (years),	
median [IQR]	12 [8-18]
Quantity of alcohol consumption	
(grams/day), median [IQR]	120 [90-160]
Comorbidities, n (%)	
Hypertension	30 (30%)
Diabetes mellitus	15 (15%)
Dyslipidemia	25 (25%)
Vital signs, mean ± SD	
Heart rate (bpm)	88 ± 12
Systolic blood pressure (mmHg)	135 ± 18
Diastolic blood pressure (mmHg)	85 ± 12

Table 2: Biochemical indicators in patients with and without alcoholic cardiomyopathy

Biochemical Indicator	Alcoholic Cardiomyopathy (n=30)	No Alcoholic Cardiomyopathy (n=70)	p-value
AST (U/L)	85 [60-120]	45 [30-65]	<0.001
ALT (U/L)	70 [50-95]	40 [25-60]	<0.001
GGT (U/L)	150 [110-220]	90 [60-130]	<0.001
Troponin I (ng/mL)	0.08 [0.05-0.12]	0.02 [0.01-0.04]	<0.001
BNP (pg/mL)	180 [120-250]	60 [40-90]	<0.001

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Table 3: Echocardiographic parameters in patients with and without alcoholic cardiomyopathy

Echocardiographic Parameter	Alcoholic Cardiomyopathy (n=30)	No Alcoholic Cardiomyopathy (n=70)	p-value
LVEF (%)	35 ± 8	58 ± 6	<0.001
Left ventricular end-diastolic diameter (mm)	62 ± 6	50 ± 4	<0.001
Left ventricular end-systolic diameter (mm)	48 ± 5	32 ± 3	<0.001
Interventricular septum thickness (mm)	12 ± 2	10 ± 1	0.001
Left ventricular posterior wall thickness(mm)	11 ± 2	9 ± 1	0.001
E/A ratio	1.2 ± 0.4	1.5 ± 0.3	0.002
E/e' ratio	15 ± 4	8 ± 2	<0.001

Table 4: CMR parameters in patients with and without alcoholic cardiomyopathy

CMR Parameter	Alcoholic Cardiomyopathy (n=30)	No Alcoholic Cardiomyopathy (n=70)	p-value
Left ventricular mass (g)	220 ± 40	150 ± 30	<0.001
Left ventricular end-diastolic volume(mL)	180 ± 30	120 ± 20	<0.001
Left ventricular end-systolic volume (mL)	120 ± 25	50 ± 10	<0.001
LVEF (%)	34 ± 7	59 ± 5	<0.001

Table 5: Correlation between biochemical indicators and echocardiographic parameters

Biochemical Indicator	LVEF	E/A ratio	E/e' ratio
AST	r=-0.65	r=-0.45	r=0.55
	p<0.001	p<0.001	p<0.001
ALT	r=-0.60	r=-0.40	r=0.50
	p<0.001	p<0.001	p<0.001
GGT	r=-0.55	r=-0.35	r=0.45
	p<0.001	p=0.002	p<0.001
Troponin I	r=-0.70	r=-0.50	r=0.60
	p<0.001	p<0.001	p<0.001
BNP	r=-0.75	r=-0.55	r=0.65
	p<0.001	p<0.001	p<0.001

Table 6: Predictors of alcoholic cardiomyopathy (logistic regression analysis)

Variable	Odds Ratio (95% CI)	p-value
Age (per year increase)	1.05 (1.01-1.10)	0.02
Male gender	2.50 (1.10-5.80)	0.03
Duration of alcohol abuse (per year increase)	1.15 (1.05-1.25)	0.002
Quantity of alcohol consumption (per 10 g/day increase)	1.20 (1.10-1.30)	<0.001
AST (per 10 U/L increase)	1.25 (1.10-1.40)	<0.001
ALT (per 10 U/L increase)	1.20 (1.05-1.35)	0.005
GGT (per 10 U/L increase)	1.15 (1.05-1.25)	0.002
Troponin I (per 0.01 ng/mL increase)	1.40 (1.20-1.60)	<0.001
BNP (per 10 pg/mL increase)	1.30 (1.15-1.45)	<0.001
LVEF (per 5% decrease)	2.00 (1.50-2.70)	<0.001
E/A ratio (per 0.1 decrease)	1.25 (1.10-1.45)	0.001
E/e' ratio (per 1 unit increase)	1.35 (1.20-1.55)	<0.001

DISCUSSION

The present study investigated the utility of biochemical indicators and cardiac function tests in assessing the extent and severity of cardiac dysfunction in patients with chronic alcohol abuse. The findings demonstrated that patients with alcoholic cardiomyopathy had significantly higher levels of liver enzymes (AST, ALT, and GGT) and markers of cardiac injury (troponin I and BNP) compared to those without alcoholic cardiomyopathy. Additionally,

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echocardiographic and CMR parameters revealed significant differences in cardiac structure and function between the two groups. Several independent predictors of alcoholic cardiomyopathy were identified, including age, male gender, duration and quantity of alcohol consumption, liver enzymes, markers of cardiac injury, and echocardiographic parameters.The prevalence of alcoholic cardiomyopathy in this study was 30%, which is consistent with the findings of a meta-analysis by Rehmet al.[16], who reported a prevalence of alcoholic cardiomyopathy ranging from 23% to 40% in patients with chronic alcohol abuse. However, a study by Piano et al.[17] found a lower prevalence of 14% in a cohort of 1,204 patients with a history of alcohol abuse. This discrepancy may be attributed to differences in the study populations and diagnostic criteria used.The significantly higher levels of liver enzymes in patients with alcoholic cardiomyopathy observed in this study are in line with the findings of Torruellas et al.,[18] who reported that AST, ALT, and GGT were significantly elevated in patients with alcoholic liver disease compared to healthy controls ($p<0.001$). The authors also found that the severity of liver disease was positively correlated with the degree of alcohol consumption ($r=0.62$, $p<0.001$). Similarly, a study by Alataloet al.[19] demonstrated that GGT was a strong predictor of all-cause mortality (hazard ratio: 2.03, 95% CI: 1.44-2.85, $p<0.001$) and cardiovascular mortality (hazard ratio: 2.06, 95% CI: 1.28-3.32, $p=0.003$) in patients with alcohol dependence.The elevated levels of troponin I and BNP in patients with alcoholic cardiomyopathy found in this study are consistent with the results of Arutiunov et al.[20], who reported significantly higher troponin T levels in patients with chronic alcohol abuse compared to healthy controls (0.028 ± 0.014 ng/mL vs. 0.010 ± 0.004 ng/mL, $p<0.001$). The authors also found that troponin T levels were positively correlated with the duration of alcohol abuse ($r=0.47$, $p<0.05$). A study by Şafaket al.[21] demonstrated that BNP levels were significantly higher in patients with alcoholic cardiomyopathy compared to those with idiopathic dilated cardiomyopathy (1,102 ± 1,048 pg/mL vs. 335 ± 371 pg/mL, $p<0.001$) and healthy controls (1,102 ± 1,048 pg/mL vs. 28 ± 17 pg/mL, $p<0.001$).The echocardiographic findings of this study, which showed significant differences in LVEF, left ventricular dimensions, and diastolic function parameters between patients with and without alcoholic cardiomyopathy, are in agreement with the results of previous studies. A study by Lazăret al.[22] found that patients with alcoholic cardiomyopathy had significantly lower LVEF (32.4 ± 8.2% vs. 60.3 ± 4.7%, $p<0.001$) and larger left ventricular end-diastolic diameter (67.5 ± 7.2 mm vs. 48.3 ± 4.1 mm, $p<0.001$) compared to healthy controls. The authors also reported that E/A ratio was significantly lower (0.9 ± 0.3 vs. 1.3 ± 0.2, $p<0.001$) and E/e' ratio was significantly higher (15.2 ± 4.1 vs. 7.4 ± 1.8, $p<0.001$) in patients with alcoholic cardiomyopathy compared to controls.The CMR findings of this study are consistent with the results of Maceira et al.[23], who demonstrated that patients with alcoholic cardiomyopathy had significantly higher left ventricular mass (191 ± 54 g vs. 119 ± 24 g, $p<0.001$), end-diastolic volume (257 ± 72 mL vs. 146 ± 28 mL, $p<0.001$), and end-systolic volume (179 ± 66 mL vs. 55 ± 15 mL, $p<0.001$) compared to healthy controls. The authors also found that LVEF was significantly lower in patients with alcoholic cardiomyopathy (32 ± 11% vs. 62 ± 6%, $p<0.001$).The correlation analysis in this study revealed significant associations between biochemical indicators and echocardiographic parameters, which is in line with the findings of Ren et al.[24]. The authors reported that GGT was negatively correlated with LVEF ($r=-0.41$, $p<0.001$) and positively correlated with left ventricular end-diastolic diameter ($r=0.36$, $p<0.001$) in patients with chronic alcohol abuse. Similarly, a study by Erbaşet al.[25] found that troponin I levels were negatively correlated with LVEF ($r=-0.58$, $p<0.001$) and positively correlated with left ventricular end-diastolic diameter ($r=0.52$, $p<0.001$) in patients with alcoholic cardiomyopathy.The logistic regression analysis in this study identified several independent predictors of alcoholic cardiomyopathy, which is consistent with the findings of previous studies. A study by Guzzo-Merelloet al.[26] found that age (OR: 1.04, 95% CI: 1.01-1.07, $p=0.02$), male gender (OR: 2.68, 95% CI: 1.16-6.19, $p=0.02$), and total lifetime dose of ethanol (OR: 1.02 per 100 kg increase, 95% CI: 1.01-1.03, $p<0.001$) were significant predictors of alcoholic cardiomyopathy. Similarly, a study by Fernández-Solàet al.[27] demonstrated that the duration of alcohol abuse (OR: 1.10 per year increase, 95% CI: 1.04-1.17, $p=0.001$) and the quantity of alcohol consumption (OR: 1.22 per 10 g/day increase, 95% CI: 1.10-1.35, $p<0.001$) were independent predictors of alcoholic cardiomyopathy.This study highlights the importance of biochemical indicators and cardiac function tests in the assessment and risk stratification of patients with chronic alcohol abuse. The findings suggest that elevated liver enzymes, markers of cardiac injury, and echocardiographic parameters of cardiac dysfunction are strongly associated with the presence of alcoholic cardiomyopathy. Furthermore, the study identifies several independent predictors of alcoholic cardiomyopathy, which may aid in the early detection and management of this condition. Future research should focus on developing risk prediction models incorporating these biochemical and imaging markers to improve the diagnosis and prognosis of patients with alcoholic cardiomyopathy.

CONCLUSION

The present study demonstrates the significant role of biochemical indicators and cardiac function tests in assessing the extent and severity of cardiac dysfunction in patients with chronic alcohol abuse.

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The findings reveal that patients with alcoholic cardiomyopathy have significantly higher levels of liver enzymes (AST, ALT, and GGT) and markers of cardiac injury (troponin I and BNP) compared to those without alcoholic cardiomyopathy. Furthermore, echocardiographic and CMR parameters show significant differences in cardiac structure and function between the two groups, with patients with alcoholic cardiomyopathy exhibiting lower LVEF, larger left ventricular dimensions, and impaired diastolic function.The correlation analysis highlights the strong associations between biochemical indicators and echocardiographic parameters, suggesting that these markers can serve as valuable tools in the assessment of alcohol-induced cardiac damage. The logistic regression analysis identifies several independent predictors of alcoholic cardiomyopathy, including age, male gender, duration and quantity of alcohol consumption, liver enzymes, markers of cardiac injury, and echocardiographic parameters.These findings have important clinical implications for the early detection, risk stratification, and management of patients with alcoholic cardiomyopathy. Incorporating biochemical indicators and cardiac function tests into the routine evaluation of patients with chronic alcohol abuse can aid in the timely diagnosis and initiation of appropriate interventions. Furthermore, the identified predictors of alcoholic cardiomyopathy can be used to develop risk prediction models and guide targeted prevention and treatment strategies.In conclusion, this study underscores the importance of a comprehensive approach to the assessment of patients with chronic alcohol abuse, integrating biochemical indicators, cardiac function tests, and clinical risk factors. Future research should focus on validating these findings in larger, prospective cohorts and exploring the potential of novel biomarkers and imaging techniques in improving the diagnosis and prognosis of alcoholic cardiomyopathy. By advancing our understanding of the mechanisms and markers of alcohol-induced cardiac damage, we can work towards reducing the burden of this preventable and potentially reversible condition.

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