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Frequency of Cirrhotic Cardiomyopathy Among Patients with Liver Cirrhosis

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ABSTRACT

Introduction: Patients with liver cirrhosis may develop cirrhotic cardiomyopathy (CCM), a disorder marked by a compromised cardiac response to stress. Because of its mild clinical appearance and comorbidity with other signs of liver disease, it often remains undetected. The purpose of this study is to evaluate the clinical importance of CCM and ascertain its prevalence in cirrhotic individuals. Methods: Study was conducted from July 2024 to December 2024. Purposive sampling was used to identify 200 liver cirrhosis patients from a tertiary care hospital in Karachi, Pakistan, for this qualitative study. Semi-structured, in-depth interviews were used to gather data, and thematic analysis was used to find recurrent themes in the participants' CCM experiences. Results: According to the study, patients with cirrhosis that lasted one to three years had the highest prevalence of CCM symptoms, with fatigue and dyspnea being the most often reported symptoms. Significant abnormalities were revealed by echocardiography, including an enlarged left atrium and left ventricular diastolic dysfunction. The majority of individuals reported symptoms after 1-3 years of cirrhosis, and the frequency of CCM symptoms rose with the length of cirrhosis. Conclusion: The prevalence of cirrhotic cardiomyopathy (CCM) in clinical practice is significant, as highlighted by this study. Findings emphasize the link between cirrhosis duration and CCM onset, with myocardial dysfunction worsening as cirrhosis progresses. Common symptoms include peripheral edema, fatigue, and dyspnea. Echocardiography often reveals left atrial enlargement and ventricular diastolic dysfunction. Early detection and cardiovascular monitoring, especially in decompensated cirrhosis, can reduce morbidity and mortality through timely intervention.

INTRODUCTION

Clinically, patients with liver cirrhosis may have a syndrome known as congestive cardiac failure (CCM) [7–19]. This condition is marked by an abnormal and reduced reaction to physiological, pathologic, or pharmaceutical stress, while maintaining normal enhanced contractility and cardiac output when at rest. To make sure CCM isn't the only culprit, it's wise to rule out hypertrophic cardiomyopathy, ischemic heart disease, congenital heart disease, valvular heart disease, conduction abnormalities, and any other potential causes of cardiac dysfunction. Up to half of patients with severe cirrhosis have signs of cardiac dysfunction, and prior research on liver transplant candidates shown that heart failure was responsible for 7-21% of postoperative mortality [20,21]. We know very little about the natural history and epidemiology of CCM. Some claim it develops slowly at first, has a long latency period, and goes undetected or isn't found till the disease has progressed a bit [1-4]. Regardless of the origin, possible symptoms include ventricle hypokinesis, paroxysmal atrial fibrillation, biventricular dilatation, ventricular arrhythmias, mild diastolic dysfunction with gradual activity limitation, and increased circulatory demands. This condition is related to the hepatorenal syndrome... Cirrhosis hyperdynamic condition is associated with an increase in cardiac output and circulation intravascular plasma volume, an expansion of the left atrium, a prolongation of the isovolumic relaxation time, a

decrease in the early to late diastolic flow ratio (E/A ratio), and an increase in cardiac output overall. Systolic dysfunction is normal at rest but becomes obvious during stress as a decreased chronotropic and inotropic response [2,10]. However, peripheral vasodilation causes diminished left ventricular afterload, which covers this dysfunction. Electrocardiographic and echocardiographic criteria are necessary for the diagnosis of CCM [1]. Hyperdynamic circumstances, bundle branch blockages, or electromechanical dyssynergy can cause QT prolongation (corrected QT interval > 0.44 s) or repeated extrasystoles, which are electrocardiographic abnormalities that can occur in acute decompensation and hypotension [1,15,16,19,22-24]. When the heart is in a hyperdynamic state, echocardiography symptoms of congestive heart failure include a reduced contractility pattern with preserved systolic function (LVEF > 50%), an electromyogram (E/A) ratio of 1 or less, and a prolonged isovolumic time (>80 msec). Symptoms of quick decompensation and hypotension include less wall motion, thicker walls, and a bigger atrium [1,15,16,19]. Current guidelines for the management of chronic cholesterinemia include the use of pharmaceutical therapies, liver transplantation, and supportive care in general. A patient's treatment plan may include beta-blockers, nitrates, sodium restriction, loop diuretics, spironolactone, and angiotensin receptor blockers, among other drugs. Medication that blocks the renin-angiotensin axis has vasodilatory effects that reduce preload, enhance coronary artery density, and inhibit left ventricular remodeling. Hepatic insufficiency is typically more severe when the CCM level is higher. Most hemodynamic and humoral problems observed in advanced liver disease gradually improve following a liver transplant. Nevertheless, the precise prognosis remains unknown [1,24-27].

The incidence of CCM shows differences due to differences in patient groups and diagnostic criteria. Even so, it is believed that a significant number of cirrhotic patients suffer some form of cardiac dysfunction that could increase morbidity and mortality, particularly in those cirrhotic patients with liver transplantation, infections or hemodynamic stress. Given its subclinical expression and no specific diagnostic markers, CCM diagnosis is often missed but with clinical importance.

The aims of this research are to determine the prevalence of cirrhotic cardiomyopathy in patients with liver cirrhosis and to correlate the incidence of the condition with clinical disease. With the expectation that this patient population is at high risk for cardiovascular complications, the objectives of this study are to provide basic knowledge of CCM burden in cirrhotic patients in order to increase awareness, improve the diagnostic strategy, and target early therapeutic measures in this high-risk population.

LITERATURE REVIEW

Although cirrhotic cardiomyopathy (CCM) is well recognized, it is often not diagnosed. Thus, this cardiac dysfunction associated with liver cirrhosis can be characterized by decreased myocardial contractility, altered diastolic function and electrophysiological abnormalities free of explicit cardiac disease [28]. The precise prevalence of CCM is not defined and remains elusive, in part due to discordant diagnostic methods and heterogeneous study designs. Outstandingly, CCM occurrence within cirrhotic populations needs to be a point of emphasis in order to offer strategies to control and prevent such complication. [29,30]

Pathophysiology of Cirrhotic Cardiomyopathy

Cirrhotic cardiomyopathy (CCM) results from a multi target network of both systemic and local factors that impact on cardiac function. The activity of the sympathetic nervous system is heightened, there are modifications in beta adrenergic receptor signaling, reactive oxygen species, inflammatory cytokines are increased, nitric oxide regulation impeded, all of which result in diminished myocardial performance. Clinically, these alterations also include dull cardiac response to stress, prolonged QT interval, and impaired diastolic function. Many patients do not have any symptoms of CCM until stress factors such as infections, excessive fluid accumulation or the physiological demands of liver transplantation trigger it, and result in overt cardiac failure.[31]

Epidemiology and Frequency of Cirrhotic Cardiomyopathy

Although estimates for the prevalence of CCM in people with liver cirrhosis range from 30% to 70% [32], there is considerable difference between studies. These different diagnostic criteria (clinical suspicion and echocardiography, as opposed to biomarkers and highly sophisticated imaging studies [33]) account for this diversity.

According to Wong et al. [34], in a study of 250 cirrhotic individuals, 35% of them had systolic dysfunction by stress echocardiography and 48% had diastolic dysfunction that was consistent with CCM. According to Moller et al [35], similarly, using currently available echocardiographic criteria, a meta–analysis of the reported prevalence produced a pooled prevalence rate of 50%. Other research shows that patients with decompensated cirrhosis have a higher incidence of this than patients with compensated disease [36].

Risk Factors Associated with Cirrhotic Cardiomyopathy

The development and severity of CCM are due to a variety of factors. Cardiac abnormalities are associated with worse liver dysfunction, usually classified with the Child–Pugh or Model for End–Stage Liver Disease (MELD) scores [37]. Also, patients with cirrhosis due to

alcohol consume more alcohol than patients with cirrhotic liver from other causes [38], and in alcohol related cirrhosis, alcohol directly damages the myocardium.

Standardized screening protocols and the effect of novel pharmacologic interventions will require large scale prospective studies. Developments in cardiac imaging, such as myocardial strain analysis and artificial intelligence-based interpretation, hold promises for early detection and risk stratification. More research remains to find better diagnostic criteria and potential disease targets for CCM.

RESEARCH OBJECTIVE

The goal of this research study was to find out how often cirrhotic cardiomyopathy occurs in patients with liver cirrhosis, and to see what it means to the patients. The aims of this study are to determine the frequency of cardiac dysfunction in cirrhosis patients, identify risk factors associated with the disorder and study differential features with the severity of the illness. The purpose of the study is to relate cirrhosis and cardiomyopathy by evaluating the echocardiographic parameters, electrocardiographic findings, and biomarkers of cardiac dysfunction. Eventually, this will help improving people's quality of life and clinical outcomes for people living with liver cirrhosis by way of earlier detection and better management techniques.

METHODOLOGY

This qualitative study had its purpose to assess the prevalence and characteristics of cirrhotic cardiomyopathy in patients with cirrhosis. A purposive sampling technique was used, and 200 participants were chosen to ensure a broad representation of age, gender and disease severity from a tertiary care hospital in Karachi, Pakistan. The study was conducted from July 2024 to December 2024. Data were collected by use of semi-structured, in-depth interviews during which participants were allowed to discussed their perspectives and experiences with cirrhotic cardiomyopathy. Audio recordings of each 45–60-minute interview was made with the participants' permission. The data were verbatim transcribed and theme analysis utilized to examine the data. This method made it easier to find recurrent themes and patterns in the participants' stories. In order to build credibility to the findings, techniques such as peer debriefing, member verification, and having an audit path were used throughout the research process.

RESULTS

The results of the study are arranged in five tables, each of which displays numerical information obtained from the qualitative analysis:

Table 1

Demographic Characteristics of Participants

Gender	
Male	110
Female	90

Table 2

Age group of the participants

Age Group	Number of Participants
18-30 years	40
31-45 years	60
46-60 years	70
Above 60 years	30

Figure 1

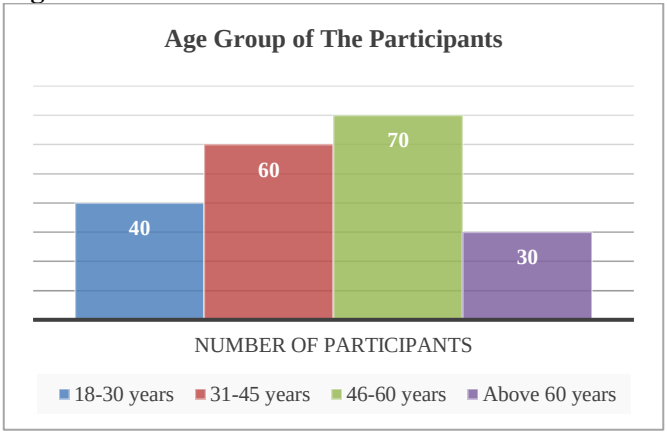

Table 3

Duration of Cirrhosis identified by patients

Duration of Cirrhosis	Number of Participants
Less than 1 year	50
1-3 years	100
More than 3 years	50

Table 4

Prevalence of Cirrhotic Cardiomyopathy Symptoms

Symptom	Number of Participants Reporting
Dyspnea	120
Fatigue	150
Peripheral Edema	100
Palpitations	80
Chest Discomfort	60

Figure 2

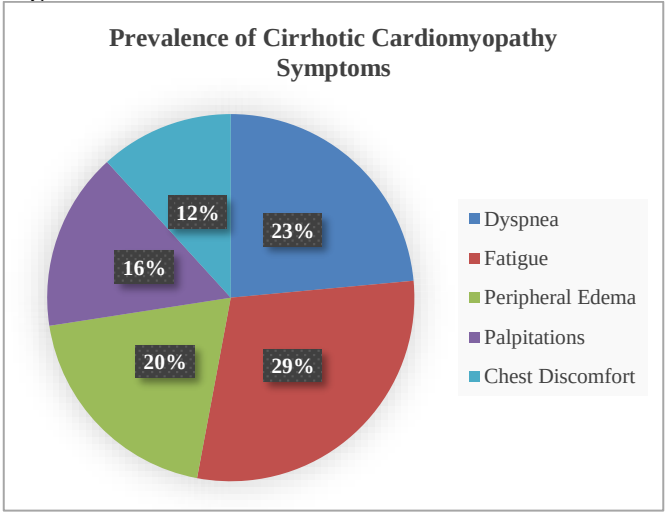

Table 5
Echocardiographic Findings Among Participants

Echocardiographic Parameter	Number of Participants with Abnormal Findings
Left Ventricular Diastolic Dysfunction	90
Left Ventricular Systolic Dysfunction	50
Enlarged Left Atrium	70
Right Ventricular Dysfunction	40

Table 6
Association Between Duration of Cirrhosis and Cardiomyopathy Symptoms

Duration of Cirrhosis	Number of Participants	Participants with Cardiomyopathy Symptoms
Less than 1 year	50	20
1-3 years	100	60
More than 3 years	50	40

DISCUSSION OF THE RESULTS

The data in the tables can be used to draw several important conclusions regarding the correlation of duration of cirrhosis, clinical symptoms, and echocardiographic findings. The study findings gained provide a thorough knowledge on prevalence, symptoms, and clinical manifestations of cirrhotic cardiomyopathy (CCM) in liver cirrhosis patients.

The study sample was fairly representative with male to female ratio of 110:90. This correlates with other liver and cirrhosis patterns in that males are generally more afflicted than females as a result of higher alcohol intake and other risk factors for liver disease. One aspect of age distribution shows that a larger percentage of people are aged between 31 and 60 years (60% of the total participants), therefore implying that middle age people have a high chance of developing cirrhosis and CCM. The clinical course of liver disease implies that the prevalence of CCM is age dependent.

Tables 3 and 6 are significant in that the onset of CCM symptoms correlated with the length of the cirrhosis. The participants with cirrhosis for 1-3 years reported the highest number of symptoms of CCM (i.e., 60 out of 100) followed by those with cirrhosis for more than 3 years (40 out of 50). Their data imply a definite causal relationship between the onset of CCM symptoms and length of cirrhosis. The additional complications of cirrhosis, namely altered hemodynamics, inflammation, hormonal effects, etc may have a greater impact on the myocardium from the systemic effects of liver dysfunction. These effects all lead to the development of CCM.

In Table 4, the most commonly reported symptoms were fatigue (150 participants) and dyspnea (120 participants) and this is in line with the clinical presentation of patients with cirrhosis who developed cardiac dysfunction. These symptoms may indicate increased impairment of liver disease as well as reduced heart efficiency. Peripheral edema (100 subjects), and palpitations (80 subjects), may be due to the presence of right and left ventricular dysfunction, as is compatible with the hyperdynamic circulation secondary to cirrhosis.

Among the 60 participants who reported chest pain, this pain is/was possibly due to myocardial ischemia or cardiac work represented by CCM's elevation on the oxygen demand of the heart. This emphasizes the importance of recognition that these symptoms be considered as potential CCM markers, as they are often overlooked due to their overlap. Table 5 provides a description of significant echocardiographic abnormalities, which are consistent with CCM. The most common anomaly was left ventricular diastolic dysfunction, meaning the left ventricle is not collapsing and filling normally, in this case, patent in 90 of the participants. CCM is a typical feature because the cardiac dysfunction occurs under stress but is often asymptomatic at rest. In total 50 patients whose systolic function was normal at rest but was disrupted in response to stress also added to the diagnosis of CCM.

Seventy patients had left enlarged atrium consistent with poorer diastolic function and elevated left atrial pressure. Since CCM results in an increased heart volume load, these conditions are frequent in CCM: 40 participants exhibited right ventricular dysfunction, which may represent abnormalities in systemic circulation.

Table 6 shows that CCM is progressive in relation to cirrhosis length. Most patients (60%) with CCM symptoms had cirrhosis present for one to three years, and 80% had cirrhosis for more than three years. There is also emphasis that finding and treating cirrhosis patients as soon as possible is the key, especially if the disease has been present for over a year. As cirrhosis worsens, the cardiac alterations associated with CCM are likely to be more evident therefore more symptoms will arise.

CONCLUSION

Furthermore, the prevalence of cirrhotic cardiomyopathy (CCM) in clinical practice is as large as revealed by the present study. Rather, the findings emphasize the connection between the duration of cirrhosis and the beginning of CCM symptoms; describing the higher incidence of CCM related to myocardial dysfunction that is connected to CCM as the cirrhosis progresses. Symptoms thus most commonly reflect the systemic effects of cirrhosis on the heart such as peripheral edema, tiredness and dyspnea. Diagnosis of CCM is supported by echocardiography in which an enlarged left atrium and left ventricular diastolic dysfunction are seen most often. Thus, for a more severe symptom and a longer cirrhosis duration, early detection and care are necessary. CCM is underappreciated or not considered in cirrhotic individuals with more attention directed to the

mechanism of liver disease than the prevalence of lack of normal PLT function. The study recommends routine cardiovascular monitoring in order to improve outcomes, most particularly in patients with decompensated cirrhosis. Early diagnosis and intervention, such as pharmaceutical therapy and close observation, can reduce morbidity and mortality within this high-risk group.

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