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Original Research Article

CORRELATION OF CARDIAC AND RENAL FUNCTIONS IN PATIENTS WITH HEPATIC CIRRHOSIS

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ABSTRACT

Background: Hepatic cirrhosis, a major contributor to illness and death worldwide, signifies the final phase of various chronic liver conditions marked by advancing fibrosis and the eventual substitution of healthy liver tissue with scar tissue. This progression hinders the liver's capabilities to perform vital functions such as metabolism, detoxifying the body, and managing blood circulation. Worldwide, cirrhosis continues to be a leading cause of illness and death, with more than 1.32 million fatalities documented in 2020, as reported by the Global Burden of Disease Study.

Materials and Methods: Patients who met the inclusion and exclusion criteria were enrolled in the study. Following the acquisition of informed valid consent, each patient underwent a comprehensive clinical evaluation, focusing on the symptoms and indicators of liver function. Biochemical tests, which are a part of standard of care, were conducted for complete blood picture, liver function, and renal function estimation. An ECG, abdominal and pelvic ultrasound, and 2D echocardiogram were carried out, and eGFR was calculated for all patients using the MDRD 6 formula.

Results: Most participants (41 out of 59) exhibited a decrease in eGFR, indicating impaired kidney function among individuals with cirrhosis. There was a significant correlation observed between reducing eGFR and diastolic dysfunction in Child Pugh classes. Among the decompensation markers studied, only INR showed a significant correlation with eGFR ($p<0.05$). There was significant correlation of reduced eGFR with reduced levels of serum albumin ($p<0.05$). This analysis demonstrates that QTc prolongation is significantly more common in patients with left ventricular hypertrophy compared to those without it ($p<0.05$).

Conclusion: Despite not having obvious symptoms, a considerable number of patients, particularly those in Child-Pugh classifications B and C, have diastolic dysfunction, QTc prolongation, and various levels of renal impairment. Reduced eGFR is further supported by the association between low blood albumin and abnormal INR and the interrelated pathophysiology of hepatic-cardio-renal syndrome.

Keywords: Hepatic cirrhosis, cardiac and renal functions, eGFR, serum albumin.

INTRODUCTION

Hepatic cirrhosis, a leading cause of morbidity and mortality worldwide, represents the terminal stage of various chronic liver diseases characterized by progressive fibrosis and the eventual replacement of healthy hepatic parenchyma with scar tissue.[1] This process impairs the liver's ability to carry out essential functions, including metabolism, detoxification, and regulation of blood flow. Globally, cirrhosis remains one of the leading causes of morbidity and mortality, with over 1.32 million deaths reported in 2020, according to the Global Burden of Disease Study.[2]

The prevalence of cirrhosis is rising due to several factors, including the increasing rates of chronic hepatitis B and C infections, alcohol-related liver disease, and the rapid surge in non-alcoholic fatty liver disease (NAFLD). NAFLD, closely tied to the global obesity epidemic, has become the most common cause of liver disease in many countries, especially in developed nations. As metabolic conditions such as obesity, type 2 diabetes, and metabolic syndrome become more widespread, cirrhosis is expected to increase in prevalence. Additionally, as global populations age, the incidence of cirrhosis is likely to rise further, contributing to a growing public health burden.[3]

In the United States alone, it is estimated that 4.9 million adults have been diagnosed with chronic liver disease, and cirrhosis is the 12th leading cause of death. Similarly, Europe and parts of Asia have seen increases in cirrhosis-related mortality, partly driven by alcohol consumption and viral hepatitis. In countries where viral hepatitis control measures have been successful, the rising prevalence of NAFLD has taken over as the primary driver of cirrhosis.[4]

One of the most clinically significant aspects of cirrhosis is the range of complications that arise as liver function deteriorates.[5] Among these are portal hypertension, ascites, hepatic encephalopathy, and an increased risk of hepatocellular carcinoma. In advanced stages, cirrhosis often leads to multi-organ dysfunction, particularly involving the cardiovascular and renal systems.[6] These complications are particularly critical as they significantly impact patient prognosis and management.

The interplay between cardiac and renal dysfunction in cirrhosis is of particular interest. Portal hypertension, a common consequence of advanced cirrhosis, alters systemic and splanchnic circulation, leading to a cascade of hemodynamic changes. This triggers compensatory mechanisms such as activation of the renin- angiotensin-aldosterone system (RAAS) and the sympathetic nervous system.[7] While these responses initially help to maintain circulation, they eventually contribute to renal sodium and water retention, increased cardiac preload, and subsequent cardiac dysfunction. Over time, these processes can lead to severe complications like hepatorenal syndrome (HRS), which is a form of renal failure specific to cirrhosis, and cirrhotic cardiomyopathy, characterized by impaired cardiac function.[8]

From a clinical perspective, the correlation between cardiac and renal dysfunctions in cirrhosis necessitates a comprehensive approach to patient management. The traditional management of cirrhotic patients has focused on controlling portal hypertension, managing ascites, and preventing variceal bleeding; however, the recognition of cardio-renal interactions demands a shift towards more integrated care models.[9] Monitoring cardiac function using echocardiography and assessing renal function through serum creatinine levels, glomerular filtration rate (GFR), and biomarkers such as cystatin C, are essential components of this approach. Moreover, the introduction of therapies targeting the RAAS and the sympathetic nervous system, alongside the use of vasodilators and beta-blockers, has shown promise in mitigating the progression of cardio-renal syndrome in cirrhotic patients.[10]

This study aims to explore the intricate correlation between cardiac and renal functions in patients with hepatic cirrhosis. Understanding the mechanisms driving this relationship is crucial for improving patient management and outcomes, particularly as the global burden of cirrhosis continues to evolve with changing etiological patterns.

MATERIALS AND METHODS

A cross-sectional, observational, comparative study conducted at OPD and IPD of the Dr. D.Y. Patil Medical College Hospital and Research Institute, Kolhapur. Study started after getting approval of the Institutional Research Committee and Institutional Ethical Committee. Consecutive Sampling method is used and sample size is 51. Considering a drop out rate of 15%, adjusted sample size is 60. One patient dropped out of the study.

Inclusion criteria:

Patients giving informed valid consent, Age > 18 years, all genders, Hepatic Cirrhosis of any etiology, compensated and decompensated cirrhosis patients.

Exclusion criteria:

Patients with pre-existing CKD, diagnosis of AKI or elevated creatinine, known cases of hypertension, CAD, IHD, previous MI, pre-existing complete bundle branch block, cardiomyopathy, valvular heart diseases, cases of severe anemia (Hb <6gm/dl), diagnosed cases of hyperthyroidism, malignancy, Pregnant females, Patients taking any drug known to have an effect on the QTc interval such as macrolides, chloroquine, antipsychotics, antidepressants, anti-arrhythmic, Patients with known or suspected parathyroid disorder, Cardiac cirrhosis.

Patients who met inclusion and exclusion criteria were recruited in the study. After informed valid consent was obtained, all patients underwent detailed clinical examination with emphasis on the symptoms and signs of hepatic function. Collecting a blood sample involved essential steps to maintain sample integrity and patient safety. After cleaning the site with alcohol, a sterile needle was inserted into the vein. Blood was collected into tubes. The tube was labelled with patient information, and samples were promptly transported to the lab. The entire procedure was meticulously documented in the patient's records, ensuring sample integrity and patient safety. Biochemical tests were sent (part of standard of care) for CBC, liver function test, renal function test estimation. ECG, ultrasonography of

abdomen and pelvis, and 2D ECHO were performed and eGFR is measured or calculated using MDRD 6 formula in all patients.

Investigation- Biochemistry: Complete blood count, Random blood sugar, Liver function test, Renal function test, PT INR, 12 lead Electrocardiogram, Radiological: Ultrasonography of abdomen and pelvis, 2D ECHO

Materials and Machines used: Weighing machine, stadiometer, measuring tape, ultrasonography machine (F8xpert), electrocardiography: BPL Cardiart 9108, 12 Channel ECG machine, 2D ECHO was done on Sonosite M-turbo- LVEF and E/A calculated, biochemical tests were performed on Horiba 5 part (CBC), agape Mispa Nanoplus and RAPIDLab® 348EX Blood Gas System: (RFT, LFT).

RESULTS

Majority of the participants in the study were male ($p \leq 0.001$)

Only 3.39% had type 2 diabetes mellitus in the study participants ($p \leq 0.001$)

Majority of the study participants (93.2%) did not report any major past medical illness.

Distribution of Alcohol consumption in cirrhotic population

Overwhelming majority (86.44%) of participants had history of alcohol addiction.

Clinical signs of hepatic failure

Commonest signs of liver cell failure noted within study participants were fetor hepaticus (13.56%) followed by spider nevi (11.86%) and scratch marks (8.47%)

The commonest clinical finding was ascites and splenomegaly and the least common finding was hepatomegaly.

Majority of patients with cirrhosis in this study did not have encephalopathy at time of inclusion.

Child Turcotte Pugh score

This categorizes patients by severity of liver disease using the Child Turcotte Pugh classification.

Most patients in the study fell under Child Turcotte Pugh Class B and Class C(decompensated) suggesting cohort predominantly has moderate to severe liver dysfunction and limited representation of compensated disease.

Correlation of Child-Turcotte-Pugh Score and BARD Score

The relationship between liver severity score and metabolic fibrosis index. Majority of the study population fell under a BARD score of 2 which did not correlate with the severity of the CTP score suggesting that fibrosis itself cannot determine the severity of the patient but plays a role as risk factor.

Table 1: Renal function (eGFR) in Cirrhotic Patients.

eGFR	No. of patients	Percentage (%)
< 60	22	37.29
60 - 90	19	32.20
> 90	18	30.51
Total	59	100.00

This table categorizes renal function of patients using eGFR.

The majority of participants (41 of 59) showed reduced eGFR, reflecting the presence of impaired renal function in patients with cirrhosis.

Correlation of eGFR and Child-Turcotte-Pugh Class.

The comparison of mean eGFR values across the Child Turcotte Pugh classes. The mean estimated glomerular filtration rate (eGFR) showed a declining trend with progression in Child Turcotte Pugh class, though the difference was not statistically significant ($p>0.05$).

Among the decompensation markers studied (splenomegaly, ascites, INR, platelet count), only INR showed a significant correlation with eGFR ($p<0.05$).

Table 2: Correlation of Albumin Levels with Mean eGFR.

Albumin	eGFR		p-value
	Mean	S.D.	
Hypoalbuminemia	66.75	30.61	0.0143
Normal	85.28	40.42	

This table presents mean eGFR values in relation to serum albumin levels.

There was significant correlation of reduced eGFR with reduced levels of serum albumin ($p<0.05$).

Table 3: Correlation of LVEF with Child-Turcotte-Pugh Score

CTP SCORE	Left Ventricular Ejection Fraction		p-value
	Mean	S.D.	
CLASS A	59.17	2.04	0.003
CLASS B	59.81	0.98	
CLASS C	57.22	4.00	

This table shows the variation in left ventricular ejection fraction across Child Turcotte Pugh classes. Although the absolute change in ejection fraction is small, the consistent trend of lower values in Class C (p value< 0.05) underscores the importance of cardiac monitoring in patients with advanced cirrhosis.

Correlation of Diastolic Dysfunction with Child-Turcotte-Pugh Class:

Although it is observed that higher grades of liver dysfunction (Class B and C) showed greater prevalence of diastolic dysfunction, the difference across the groups was not statistically significant in this cohort.

Table 4: (a) Correlation of Diastolic Dysfunction with mean eGFR.

Diastolic Dysfunction	**eGFR**		**p-value**
	Mean	**S.D.**	
Present	65.97	33.92	0.02087
Absent	93.43	20.21	

(b): Correlation of Diastolic Dysfunction with eGFR levels

Diastolic Dysfunction	**eGFR**			**p-value**
	< 60	**60 - 90**	**> 90**	
Present	21 (95.45)	18 (94.74)	13 (72.22)	0.0353
Absent	1 (4.55)	1 (5.26)	5 (27.78)	
Total	22 (100)	19 (100)	18 (100)	

This table correlates presence of diastolic dysfunction with mean eGFR values.

Patients with diastolic dysfunction showed significantly lower eGFR compared to those without diastolic dysfunction (p<0.05)

Participants with reduced eGFR (below 90) showed higher incidence of diastolic dysfunction (>94%) while those with normal eGFR had significantly lower incidence (p<0.05)

QTc prolongation in the study population

Majority participants showed presence of QTc prolongation in ECG though not statistically higher (p>0.05). Sub analysis done revealed that QTc prolongation did not show any significant results in relation to sodium and potassium levels in the serum.

Table 5: Association of LVH and QTc Prolongation

QTc PROLONGATION AND LVH CROSSTABULATION					
		Left Ventricular Hypertrophy		**Total**	**p-value**
		NO	**YES**		
QTc PROLONGATION	NO	3 (100)	22 (39.29)	25	
	YES	0 (0)	34 (60.71)	34	
Total		3 (100)	56 (100)	59	0.038

This table examines the relationship between left ventricular hypertrophy and QTc prolongation.

This analysis demonstrates that QTc prolongation is significantly more common in patients with left ventricular hypertrophy compared to those without it (p<0.05).

DISCUSSION

In the present study involving 59 patients with hepatic cirrhosis, a significant male predominance was noted ($p \leq 0.001$). This observation aligns with global and regional literature, which consistently reports higher cirrhosis prevalence in males. Several factors contribute to this pattern. Biologically, males may have a heightened susceptibility to hepatic injury from alcohol, as estrogen has shown some hepatoprotective effects in females. Socially and behaviorally, Indian men, particularly in lower- and middle-income groups, tend to have higher rates of alcohol consumption, which remains a principal risk factor for cirrhosis. The male dominance in this study is further reinforced by the overwhelmingly high prevalence of alcohol use (86.44%) among participants. This observation is consistent with findings from Asrani et al. (2019), who reported that alcohol-related liver disease has surpassed viral hepatitis as the leading cause of cirrhosis in many regions, including South Asia.[3] The concordance between our findings and those from the literature can be attributed to overlapping patient profiles—middle-aged males from semi-urban or rural backgrounds with high alcohol consumption patterns, limited health literacy, and often delayed access to tertiary care. This demographic profile often leads to late-stage presentations, frequently involving multi-organ dysfunction. In contrast to some international studies from Africa and East Asia, where hepatitis B and C continue to be dominant etiologies, our study showed a lower burden of viral hepatitis. This regional variation could reflect the successful implementation of national hepatitis vaccination programs and improved awareness regarding blood- borne infections in India. The second most frequent systemic finding was splenomegaly, noted in 79.66% of patients. Splenomegaly is also a classical consequence of portal hypertension, reflecting increased pressure in the splenic venous system and associated with hypersplenism, which can result in

cytopenias such as anemia, thrombocytopenia, and leukopenia. Its high prevalence in this cohort aligns with its well-established role as a surrogate marker of chronic liver disease severity and sustained portal pressure.

Hepatic Status: Child-Pugh and BARD Scores.

In addition to the Child-Pugh score, the BARD score was calculated for each patient to estimate fibrosis risk. The BARD score, a non-invasive index incorporating BMI, AST/ALT ratio, and presence of diabetes, has shown utility in predicting significant fibrosis in various liver disease etiologies, especially NAFLD.[11] In this study, most patients had a BARD score of 2, indicating an intermediate-to-high risk of advanced fibrosis. However, there was no statistically significant correlation between BARD score and Child-Pugh class ($p < 0.05$). This finding highlights a crucial clinical distinction: fibrosis severity does not necessarily parallel the functional capacity of the liver, particularly in alcohol-related cirrhosis. These results are in agreement with prior research conducted by Pan Z et al, (2020) and Praveen S et al, (2021) which has observed that fibrosis indices like BARD and FIB-4 often show limited concordance with dynamic liver function assessments such as the Child-Pugh or MELD scores.[12,13] Especially in alcohol-related cirrhosis, histological damage may include not only fibrosis but also fluctuating degrees of steatohepatitis, necrosis, and cholestasis, all of which may impact liver function disproportionately. Some studies, particularly those involving patients with viral hepatitis or NASH, have demonstrated moderate to strong correlations between fibrosis scores and hepatic staging.[14,15] This could be because disease progression in these populations is often slower, more linear, and less confounded by repeated inflammatory injury, allowing fibrosis burden to better reflect cumulative hepatic dysfunction.

Additionally, the components of the BARD score such as diabetes and BMI may have less relevance in our predominantly lean, alcohol-related cirrhotic population, thus diminishing its predictive value. In contrast, in NAFLD/NASH cohorts with high metabolic burden, these factors directly contribute to hepatic inflammation and fibrosis.

These findings reinforce the limited utility of fibrosis scores alone in gauging hepatic decompensation, particularly in alcohol-induced cirrhosis. They also underscore the need for comprehensive assessments that integrate structural, biochemical, and clinical parameters for staging liver disease, especially in heterogeneous populations.

Renal Dysfunction: eGFR Trends

Importantly, this study found statistically significant correlations between renal function and two key markers of hepatic synthetic function INR ($p < 0.05$) and serum albumin ($p < 0.05$) This finding suggests that renal impairment in cirrhosis may reflect not only hemodynamic alterations but also the degree of hepatic failure. Hypoalbuminemia, by reducing plasma oncotic pressure, contributes to intravascular volume depletion and renal hypo perfusion, while elevated INR is a marker of reduced hepatic synthesis of clotting factors and overall liver reserve. These associations support established concepts in the pathophysiology of hepatorenal syndrome and correlate well with earlier studies by Llacuna et al., which emphasized the role of low albumin in renal compromise due to poor vascular filling and systemic vasodilation.[16] Despite the statistical limitations, the trend of declining renal function with worsening hepatic status is clinically consistent with other studies, including those by Lora et al. and Wong et al., which describe progressive renal hypo perfusion, endothelial dysfunction, and subclinical renal damage in decompensated cirrhosis.[17,18] In contrast, studies such as that by G a Siregar and M Gurning, have shown statistically significant correlations between hepatic severity (using Child-Pugh and MELD scores) and declining eGFR.[19] The difference in findings may be due to larger sample sizes in those studies, inclusion of patients with more diverse etiologies (e.g., hepatitis C, NASH), or the use of cystatin C-based or measured GFR methods, which may offer greater accuracy in cirrhotic patients than creatinine-based eGFR calculations.

Decompensation Markers and Renal Function: Broader Correlations

When stratifying renal function by markers of hepatic compensation such as ascites, splenomegaly, INR, and platelet count the study found that only INR had a statistically significant correlation with reduced eGFR ($p<0.05$). This finding is consistent with the study by Nazar et al., who reported INR as an independent predictor of renal dysfunction and mortality in cirrhosis, supporting its use in assessing hepatorenal risk.[20] The lack of correlation between eGFR and ascites or splenomegaly in this cohort may reflect the subjective nature of clinical ascites grading or variability in volume status at the time of evaluation.

Although platelet count did not reach statistical significance ($p>0.05$), it has been shown in other studies (e.g., O'Leary et al.) to serve as an indirect marker of portal hypertension and hypersplenism, both of which may influence renal perfusion indirectly through hemodynamic shifts.[21]

Cardiac Systolic Function (LVEF) In cirrhotic cardiomyopathy, alterations in beta-adrenergic receptor signaling, membrane fluidity, myocardial fibrosis, and increased levels of circulating cytokines such as TNF-α contribute to impaired systolic function.

Our findings are consistent with the pathophysiological trajectory of cirrhotic cardiomyopathy, where the failing liver induces a cascade of systemic changes autonomic dysregulation, inflammation, and increased cardiac afterload due to hyper dynamic circulation that progressively impair myocardial performance. The normal range LVEF values may persist at rest, but

the inability to augment cardiac output under stress reflects early systolic compromise. Some studies have reported more pronounced reductions in LVEF, especially in populations with concurrent metabolic syndrome, hypertension, or longstanding alcohol use.[22] The less severe systolic impairment in our cohort could be due to several factors: relatively younger age, lower prevalence of diabetes and hypertension, and perhaps early-stage recognition of cardiac involvement. Moreover, compensatory sympathetic overactivity, common in cirrhosis, may help maintain resting LVEF despite underlying dysfunction. Also, echocardiographic limitations in cirrhotic patients such as altered acoustic windows due to ascites or hyper inflated lungs may introduce variability in LVEF estimation.

Diastolic Dysfunction Stratified by eGFR Categories A stratified analysis in the present study revealed a statistically significant association between diastolic dysfunction and worsening renal function among patients with hepatic cirrhosis. Specifically, the prevalence of diastolic dysfunction exceeded 94% among patients with eGFR < 90 mL/min/1.73 m², whereas only 72.2% of those with preserved renal function (eGFR > 90) demonstrated diastolic abnormalities ($p < 0.05$). This finding provides compelling evidence for the cardio-renal interplay in cirrhosis, wherein even subclinical cardiac dysfunction may predispose to, or aggravate, renal hypo perfusion. This observation aligns closely with the findings of Ma et al., who demonstrated that diastolic dysfunction independently predicted both renal impairment and mortality in cirrhotic patients, irrespective of the etiology of liver disease or baseline systolic function.[23] Their study proposed that impaired diastolic relaxation reduces left ventricular compliance, thereby limiting cardiac preload and effective forward flow, especially during physiological stress.

As renal perfusion is highly dependent on cardiac output in cirrhosis due to systemic vasodilation, even marginal reductions in diastolic efficiency can precipitate renal under-filling and glomerular hypo perfusion. Furthermore, Zardi et al. highlighted the critical hemodynamic consequences of diastolic dysfunction in cirrhosis, describing how elevated central venous pressures and impaired ventricular filling impede renal blood flow through both backward and forward failure mechanisms.[24]

QTc Prolongation

In this study, QTc prolongation was observed in 57.6% of cirrhotic patients, a finding that aligns with numerous prior reports establishing QTc interval changes as a hallmark of cirrhotic cardiomyopathy. While no significant association was found between QTc prolongation and Child-Pugh class, it was found to correlate positively with left ventricular hypertrophy (LVH) ($p<0.05$). This suggests that structural cardiac remodeling may contribute to electrophysiological instability in these patients.

Integrated Cardio-Renal Correlation

One of the key findings of this study is the significant association between diastolic dysfunction and renal impairment in cirrhotic patients.

The observed association between QTc prolongation and LVH further supports the hypothesis that structural and electrical remodeling in the myocardium occurs in parallel. Both may be markers of chronic subclinical stress on the heart due to hyper dynamic circulation, chronic inflammation, and hormonal dysregulation.

These observations support the model proposed by Luis M et al, who described a progressive, multisystem syndrome in cirrhosis marked by circulatory dysfunction, myocardial impairment, and renal hypo perfusion collectively termed "hepatic-cardio-renal syndrome".[25]

Compensated vs. Decompensated Cirrhosis: Comparative Trends

A comparative analysis between compensated cirrhosis (Child-Pugh Class A) and decompensated cirrhosis (Classes B and C) in this study, though limited by a small sample size in the compensated group (n = 6), revealed clear and clinically relevant trends reflective of the natural history and multisystem deterioration associated with cirrhosis progression.

QTc prolongation was more commonly observed in decompensated patients, suggesting a higher burden of electrophysiological instability, likely driven by chronic autonomic imbalance, systemic inflammation, and myocardial remodeling. These electrical changes are also reflective of subclinical myocardial dysfunction and portend a greater risk of arrhythmias.

These observations are in strong agreement with the findings of Siren Møller et al., who in a large-scale review emphasized that multi-organ involvement including cardiac and renal compromise, intensifies as cirrhosis progresses from compensated to decompensated stages.[26] Their work highlighted the importance of systemic circulatory dysfunction in the evolution of cirrhosis and emphasized that portal hypertension and impaired hepatic function are only part of a larger syndrome involving cardiac contractility, vascular tone, renal filtration, and neurohormonal status.

CONCLUSION

It underscores the importance of comprehensive systemic evaluation, revealing that a significant proportion of patients especially those in Child-Pugh classes B and C exhibit diastolic dysfunction, QTc prolongation, and varying degrees of renal impairment, even in the absence of overt symptoms. The correlation of low serum albumin and abnormal INR with reduced eGFR further supports the interconnected pathophysiology of hepatic-cardio-renal syndrome. A major strength of this study is its multi-system approach, assessing liver severity

alongside echocardiographic and renal parameters within the same cohort. Inclusion of QTc and BARD score analysis added a metabolic and electrophysiological dimension to the findings, broadening clinical relevance.

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