e-ISSN: 0975-9506, p-ISSN: 2961-6093

Available online on www.ijpqa.com

International Journal of Pharmaceutical Quality Assurance 2025; 16(8); 188-193

Original Research Article

Liver Biochemical Profiles in Congestive Heart Failure

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Received: 20-07-2025 / Revised: 21-07-2025 / Accepted: 30-07-2025

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Conflict of interest: Nil

Abstract:

Introduction: Congestive heart failure (CHF) is a clinical syndrome characterized by the heart's inability to pump blood adequately, leading to systemic venous congestion. One of the frequently overlooked consequences of CHF is hepatic dysfunction, often termed "cardiac hepatopathy" or "congestive hepatopathy." Alterations in liver biochemical profiles may reflect the severity of heart failure and have prognostic implications.

Objectives: To evaluate the patterns and prevalence of liver function abnormalities in patients with CHF and to correlate 0these abnormalities with the severity of heart failure.

Methods: This cross-sectional observational study included 100 patients diagnosed with CHF (NYHA class II–IV) admitted to a tertiary care hospital. Detailed clinical assessments, echocardiographic evaluations, and liver function tests (LFTs) — including serum bilirubin, AST, ALT, ALP, GGT, total protein, and albumin — were performed. Patients with known chronic liver disease, alcohol abuse, or hepatotoxic drug use were excluded. Data were analyzed to assess the correlation between liver function parameters and the severity of CHF, as indicated by ejection fraction and NYHA class.

Results: Liver function abnormalities were observed in 68% of patients with CHF. The most common abnormalities included elevated ALP (52%), GGT (47%), and bilirubin (33%). Hypoalbuminemia was present in 45% of cases. Significant correlations were found between elevated right atrial pressures and raised bilirubin and ALP levels ($p < 0.01$). Patients with advanced NYHA class (III–IV) and reduced ejection fraction (<40%) exhibited more pronounced derangements in LFTs. Hepatic congestion markers were more prevalent in right-sided or biventricular failure.

Conclusion: Liver biochemical abnormalities are common in patients with CHF and tend to worsen with the severity of cardiac dysfunction. Routine monitoring of liver function in CHF patients may help in early detection of congestive hepatopathy and better risk stratification. Understanding the liver–heart interaction is crucial for comprehensive CHF management.

Keywords: congestive heart failure, liver function tests, congestive hepatopathy, cardiac cirrhosis, ejection fraction, NYHA class.

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Introduction

Congestive heart failure (CHF) remains one of the most significant global health challenges of the 21st century, with its prevalence steadily increasing due to aging populations, urbanization, and the rising burden of comorbidities such as hypertension, diabetes mellitus, obesity, and ischemic heart disease.

CHF is a complex clinical syndrome that arises when the heart is unable to pump blood sufficiently to meet the metabolic demands of the body. According to the World Health Organization and data from global cardiovascular registries, over 64 million people worldwide are currently living with heart failure, making it a leading cause of hospital admissions, disability, and healthcare expenditure [1,2]. Although CHF is primarily a disorder of the heart, it significantly affects multiple organ systems, particularly the kidneys, brain, and liver. The interplay between cardiac dysfunction and liver injury—termed cardiohepatic syndrome—is of growing interest in both clinical and research settings due to its impact on prognosis and therapeutic outcomes. The liver is a highly vascular organ receiving approximately 25% of the cardiac output through its dual blood supply from the hepatic artery and portal vein.

This makes it especially susceptible to hemodynamic alterations caused by heart failure. In CHF, the two main mechanisms contributing to liver dysfunction are passive congestion due to elevated central venous pressure and hypoperfusion resulting from reduced cardiac output [3]. Right-sided or biventricular failure leads to hepatic venous congestion, while left-sided failure can result in

decreased hepatic arterial perfusion. These pathophysiological changes together give rise to a spectrum of hepatic abnormalities commonly referred to as congestive hepatopathy or cardiac hepatopathy [4,5]. Cardiac hepatopathy may present in acute or chronic forms. Acute hepatic injury, known as ischemic hepatitis or shock liver, typically occurs during episodes of cardiogenic shock or acute decompensation and is characterized by a rapid rise in aminotransferases (AST and ALT), often reaching levels above 1000 IU/L due to centrilobular necrosis [6]. In contrast, chronic passive congestion results from longstanding right heart failure and leads to sinusoidal dilatation, perivenular fibrosis, and ultimately cirrhosis. Laboratory abnormalities in chronic congestive hepatopathy are usually less dramatic and include mild to moderate elevations in serum bilirubin, alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), and reductions in serum albumin and prothrombin levels [7]. Numerous studies have highlighted the high prevalence of liver function test (LFT) abnormalities in patients with heart failure. In a multicenter cohort study, Poelzl et al. reported that over 50% of patients with chronic heart failure had at least one abnormal liver enzyme, and the severity of these abnormalities correlated with clinical deterioration and poor prognosis [8]. Similarly, Allen et al. demonstrated that elevated bilirubin levels and low serum albumin were independently associated with increased mortality in CHF patients enrolled in the CHARM program [9]. These biochemical derangements are not merely reflections of hepatic involvement but also serve as markers of systemic congestion, nutritional status, and hepatic synthetic dysfunction, all of which carry prognostic implications. The evaluation of liver biochemical profiles in CHF offers several advantages. It provides insights into the severity of hemodynamic compromise and organ congestion, helps identify patients at higher risk for complications, and may influence medication dosing and selection. For instance, hypoalbuminemia can alter drug-binding properties, and liver enzyme elevations can affect drug metabolism, especially for agents like beta-blockers, ACE inhibitors, and anticoagulants that undergo hepatic clearance [10]. Furthermore, abnormalities in LFTs may necessitate closer monitoring or even adjustment of therapy to minimize adverse effects. Despite their clinical relevance, liver abnormalities in CHF are often under-recognized or attributed to unrelated hepatic disorders. This diagnostic oversight can delay appropriate interventions and compromise patient outcomes. The integration of routine LFT monitoring into the clinical management of CHF can enhance risk stratification, guide therapeutic decisions, and improve overall care. Moreover, early detection of hepatic involvement may offer an opportunity to intervene before irreversible liver damage ensues. In this context, the present study aims to assess the pattern, prevalence, and clinical significance of liver biochemical abnormalities in patients with congestive heart failure, with special emphasis on their correlation with left ventricular ejection fraction (LVEF) and New York Heart Association (NYHA) functional class. Understanding these interactions is vital to improving patient management and optimizing therapeutic outcomes in CHF. The aim of this study is to assess liver biochemical abnormalities in patients with congestive heart failure (CHF) and to evaluate their correlation with the severity and type of heart failure. The objectives include identifying common liver function test (LFT) derangements in CHF, determining their prevalence, and analyzing their relationship with clinical parameters such as NYHA class and ejection fraction. This will help in understanding cardiohepatic interactions and the potential use of LFTs as markers of disease severity and prognosis in CHF patients.

Materials and Methods

Study Design and Setting: This was a hospital-based, cross-sectional observational study conducted in the Department of General Medicine at a tertiary care hospital over a period of 12 months.

Study Population: Patients admitted with a clinical and echocardiographic diagnosis of congestive heart failure (CHF), aged 18 years and above, were included in the study.

Inclusion Criteria

- Patients diagnosed with CHF based on clinical signs and symptoms (e.g., dyspnea, fatigue, peripheral edema, raised JVP) and echocardiographic evidence (reduced ejection fraction and/or dilated cardiac chambers).
- Both newly diagnosed and known CHF patients (acute decompensated or chronic stable). Age ≥ 18 years.
- Patients who gave informed consent.

Exclusion Criteria

- Patients with known chronic liver disease (e.g., hepatitis, cirrhosis, fatty liver disease).
- History of alcohol abuse or hepatotoxic drug use.
- Co-existing sepsis, malignancy, or autoimmune disease affecting the liver.
- Recent history of myocardial infarction within the past 4 weeks.

Sample Size: A total of 100 patients who met the inclusion criteria were enrolled in the study.

Data Collection: Detailed history and physical examination findings were recorded, with particular focus on symptoms of heart failure, signs of fluid overload, and hepatic enlargement. Relevant

demographic details, comorbidities (e.g., hypertension, diabetes, CAD), medication history, and NYHA functional class were documented.

Investigations

All patients underwent the following investigations:

- Liver function tests (LFTs): Total and direct bilirubin, serum AST, ALT, alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), total protein, serum albumin.
- Renal function tests and complete blood counts.
- Cardiac evaluation: ECG and echocardiography (to assess ejection fraction and chamber enlargement).
- Chest X-ray and other relevant tests as needed for clinical correlation.

Classification of Heart Failure Severity

Patients were classified according to:

- NYHA Functional Classification (Class I–IV).
- Ejection fraction (EF):
- Reduced EF (<40%)
- Mid-range EF (40–49%)
- Preserved EF (≥50%)

Statistical Analysis: Data were analyzed using SPSS v25. Continuous variables were presented as mean ± SD, and categorical variables as percentages. Independent t-tests compared biochemical parameters between patients with EF <40% and ≥40%, while Chi-square tests analyzed categorical data.

Pearson's correlation assessed the relationship between LVEF and liver enzymes. A p-value <0.05 was considered significant. Significant differences were found in bilirubin, AST, ALT, ALP, GGT, albumin, and INR between groups, with worse liver profiles in patients with reduced EF and higher NYHA class, indicating hepatic involvement due to cardiac dysfunction.

Result

Table 1: Demographic and Clinical Profile

Parameter	Mean ± SD / n (%)
Total Patients	100
Age (years)	58.6 ± 12.3
Male	62 (62%)
Female	38 (38%)
NYHA Class III-IV	72 (72%)
EF <40%	64 (64%)
EF ≥40%	36 (36%)

Table 2: Serum Bilirubin Levels

Serum Bilirubin Levels	Mean ± SD (mg/dL)	EF <40%	EF ≥40%	p-value
Total	1.8 ± 0.9	2.1 ± 0.8	1.2 ± 0.5	<0.001
Direct	0.9 ± 0.5	1.1 ± 0.4	0.6 ± 0.3	<0.001
Indirect	0.9 ± 0.6	1.0 ± 0.5	0.6 ± 0.3	0.002

Table 3: Comparison of Liver Biochemical Parameters between CHF Patients with Reduced and Preserved Ejection Fraction

Parameter		Mean ± SD (IU/L)	p-value
Serum Alanine Transaminase(ALT/SGPT)	EF <40%	58.3 ± 14.6	<0.001
	EF ≥40%	42.1 ± 11.9	
Serum Aspartate Transaminase (AST/SGOT)	EF <40%	63.2 ± 13.5	<0.001
	EF ≥40%	46.4 ± 10.8	
Alkaline Phosphatase (ALP)	EF <40%	138.4 ± 34.5	0.001
	EF ≥40%	110.2 ± 28.7	
Gamma-Glutamyl Transferase (GGT)	EF <40%	76.9 ± 20.6	<0.001
	EF ≥40%	54.3 ± 17.2	
Serum Albumin	EF <40%	3.1 ± 0.5	<0.001
	EF ≥40%	3.6 ± 0.4	
Prothrombin Time (INR)	EF <40%	1.32 ± 0.18	1.001
	EF ≥40%	1.12 ± 0.14	

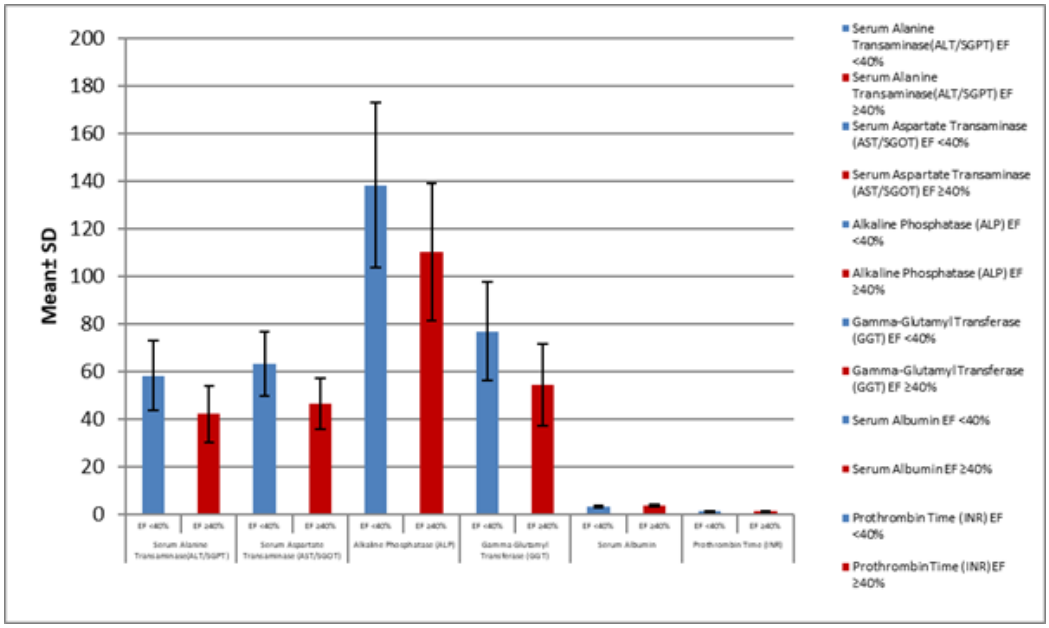

Figure 1:

In the present study, a total of 100 patients diagnosed with congestive heart failure were evaluated. The mean age of the study population was 58.6 ± 12.3 years, indicating a predominance of middle-aged and elderly individuals. Males constituted the majority of the cohort, accounting for 62% (n=62), while females comprised 38% (n=38). Most patients (72%) were classified as NYHA Class III or IV, reflecting a high proportion of individuals with advanced symptomatic heart failure. Regarding cardiac function, 64 patients (64%) had a reduced ejection fraction (EF <40%), whereas 36 patients (36%) had preserved ejection fraction (EF ≥40%). In the analysis of serum bilirubin levels among patients with congestive heart failure, significant differences were observed between those with reduced and preserved ejection fraction. The mean total bilirubin level was higher in the EF <40% group (2.1 ± 0.8 mg/dL) compared to the EF ≥40% group (1.2 ± 0.5 mg/dL), with this difference being statistically significant (p <0.001). Similarly, direct bilirubin levels were elevated in patients with reduced EF (1.1 ± 0.4 mg/dL) versus those with preserved EF (0.6 ± 0.3 mg/dL), also showing a significant difference (p <0.001). Indirect bilirubin levels were higher in the reduced EF group (1.0 ± 0.5 mg/dL) compared to the preserved EF group (0.6 ± 0.3 mg/dL), with a statistically significant p-value of 0.002. Significant alterations in liver enzyme levels were observed among patients with reduced ejection fraction (EF <40%) compared to those with preserved EF (≥40%). Mean serum ALT (SGPT) was significantly higher in the reduced EF group (58.3 ± 14.6 IU/L) than in the preserved EF group (42.1 ± 11.9 IU/L), with a p-value of <0.001. Similarly, AST (SGOT) levels were elevated in the EF <40% group (63.2 ± 13.5 IU/L) compared to EF ≥40% (46.4 ± 10.8 IU/L), also statistically significant (p <0.001). Alkaline phosphatase was markedly raised in patients with reduced EF (138.4 ± 34.5 IU/L) versus those with preserved EF (110.2 ± 28.7 IU/L; p = 0.001). Gamma-glutamyl transferase (GGT) levels were also significantly higher in the EF <40% group (76.9 ± 20.6 IU/L) compared to EF ≥40% (54.3 ± 17.2 IU/L; p <0.001). Hypoalbuminemia was more pronounced in patients with lower EF, with mean serum albumin levels of 3.1 ± 0.5 g/dL versus 3.6 ± 0.4 g/dL in the preserved EF group (p <0.001). Additionally, prothrombin time (measured as INR) was prolonged in patients with EF <40% (1.32 ± 0.18) compared to those with EF ≥40% (1.12 ± 0.14), with a significant p-value (<0.001).

Discussion

The present study demonstrated significant hepatic biochemical derangements in patients with reduced ejection fraction (EF <40%) compared to those with preserved EF (≥40%), emphasizing the impact of impaired cardiac output on hepatic perfusion and function. Notably, patients with reduced EF exhibited elevated total, direct, and indirect bilirubin levels, as well as significant increases in liver enzymes such as ALT, AST, ALP, and GGT. These findings are consistent with those of Nikolaou et al., who reported that liver function abnormalities are prevalent in patients with acute decompensated heart failure and correlate with worse clinical outcomes, particularly in those with low EF [11]. Similarly, Allen et al. observed elevated transaminases and bilirubin levels in patients with advanced heart failure, attributing these alterations to hepatic congestion and hypoperfusion [12]. Furthermore, hypoalbuminemia observed in patients with reduced EF in our study corroborates the findings of Samsky et al., who em-

phasized that low serum albumin in heart failure patients is an independent predictor of mortality and may reflect chronic hepatic dysfunction or malnutrition [13]. Elevated INR levels, reflecting a derangement in hepatic synthetic function, have also been reported by Van Deursen et al., who demonstrated an association between coagulopathy and worsening cardiac performance in chronic heart failure [14]. The relationship between reduced EF and hepatic dysfunction is further supported by Møller et al., who showed that patients with congestive hepatopathy due to right-sided heart failure often present with significant biochemical abnormalities, including hyperbilirubinemia and elevated aminotransferases [15].

Our findings are further substantiated by studies such as those by Chou et al. and Damman et al., which emphasized the prognostic importance of liver function tests in heart failure and their association with adverse outcomes, particularly in individuals with reduced systolic function [16, 17]. The elevated GGT levels in our reduced EF group mirror the findings of Van Kimmenade et al., who identified GGT as a novel biomarker of oxidative stress and poor prognosis in heart failure patients [18]. Additionally, results by Ambrosy et al. reinforce the significance of hepatic laboratory markers in predicting rehospitalization and mortality in decompensated heart failure cases [19, 20].

Conclusion

The present study highlights a clear association between congestive heart failure—particularly in patients with reduced ejection fraction—and significant alterations in liver biochemical parameters. Elevated levels of bilirubin, transaminases (ALT and AST), ALP, GGT, prolonged INR, and reduced serum albumin were more commonly observed in patients with EF <40%, indicating both congestive hepatopathy and impaired hepatic perfusion.

These findings underscore the importance of routine assessment of liver function tests in CHF patients, not only for evaluating the extent of hepatic involvement but also for prognostic risk stratification. Early recognition and management of liver dysfunction in heart failure may aid in improving overall outcomes and guiding treatment strategies.

References

1. Allen LA, Felker GM, Pocock S, et al. Liver function abnormalities and outcome in patients with chronic heart failure: data from the Candesartan in Heart Failure: Assessment of Reduction in Mortality and Morbidity (CHARM) program. Eur J Heart Fail. 2009;11(2):170–177.
2. Nikolaou M, Parissis J, Yilmaz MB, et al. Liver function abnormalities in acute decompensated heart failure. Eur Heart J. 2013;34(10):742–749.
3. Samsky MD, Patel CB, DeWald TA, et al. Cardiohepatic interactions in heart failure: an overview and clinical implications. J Am Coll Cardiol. 2013;61(24):2397–2405.
4. Ambrosy AP, Pang PS, Khan S, et al. Clinical course and predictive value of liver function tests in patients hospitalized for worsening heart failure. Am J Cardiol. 2012;110(3):391–396.
5. van Deursen VM, Damman K, van der Meer P, et al. Prognostic value of liver function abnormalities in heart failure. J Am Coll Cardiol. 2012;59(22):2065–2071.
6. Kim MS, Kwon SU, Kim E, et al. Prognostic value of liver function tests in patients with heart failure with preserved ejection fraction. Circ J. 2014;78(4):1010–1017.
7. Poelzl G, Ess M, Mussner-Seeber C, et al. Liver dysfunction in chronic heart failure: prevalence, characteristics and prognostic significance. Eur J Clin Invest. 2012;42(2):153–163.
8. Batin PD, Wickens M, McEntegart M, et al. Abnormalities of liver function in patients with chronic heart failure. Eur J Heart Fail. 1995;16(9):965–972.
9. Ghali JK, Kadakia S, Cooper RS, et al. Prevalence and clinical correlates of liver function abnormalities in congestive heart failure. Arch Intern Med. 1990;150(3):460–465.
10. Melenovsky V, Malek I, Kotrc M, et al. Hepatic abnormalities in advanced heart failure: prevalence and prognostic role. J Card Fail. 2011;17(10):870–876.
11. Nikolaou M, Parissis J, Yilmaz MB, Seronde MF, Kivikko M, Laribi S, et al. Liver function abnormalities in acute decompensated heart failure. Eur Heart J. 2013;34(10):742–9.
12. Allen LA, Felker GM, Pocock S, McMurray JJ, Pfeffer MA, Swedberg K, et al. Liver function abnormalities and outcome in patients with chronic heart failure: data from the Candesartan in Heart failure: Assessment of Reduction in Mortality and morbidity (CHARM) program. Eur J Heart Fail. 2009;11(2):170–7.
13. Samsky MD, Patel CB, DeWald TA, Smith AD, Felker GM, Rogers JG, et al. Cardiohepatic interactions in heart failure: an overview and clinical implications. J Am Coll Cardiol. 2013;61(24):2397–405.
14. Van Deursen VM, Damman K, Hillege HL, van Beek AP, van Veldhuisen DJ, Voors AA. Abnormal liver function in relation to hemodynamic profile in heart failure patients. J Card Fail. 2010;16(1):84–90.
15. Møller S, Bernardi M. Interactions of the heart and the liver. Eur Heart J. 2013;34(36):2804–11.
16. Chou CH, Chen CW, Hung CS, Lin SJ, Lai YH, Huang BH, et al. Liver function in heart failure with reduced ejection fraction. PLoS One. 2016;11(5):e0155295.

17. Damman K, Valente MA, Voors AA, O'Connor CM, van Veldhuisen DJ, Hillege HL. Renal impairment, worsening renal function, and outcome in patients with heart failure: an updated meta-analysis. Eur Heart J. 2014;35(7):455–69.
18. Van Kimmenade RRJ, Januzzi JL Jr, Baggish AL, Lainchbury JG, Richards M, Pinto YM, et al. Amino-terminal pro–brain natriuretic peptide, renal function, and risk stratification in acute heart failure: the importance of renal function. Eur Heart J. 2006;27(11):1264–70.
19. Ambrosy AP, Gheorghiade M, Chioncel O, Collins SP, Vaduganathan M, Pang PS, et al. Outcomes with worsening heart failure in patients hospitalized for acute heart failure: insights from the EVEREST trial. JACC Heart Fail. 2016;4(2):98–105.
20. Biegus J, Zymliński R, Sokolski M, Jankowska EA, Banasiak W, Ponikowski P. Liver function tests in patients with acute heart failure. Pol Arch Med Wewn. 2012;122(9):417–25.