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CLINICAL AND BIOCHEMICAL SIGNIFICANCE OF LIVER FUNCTION TESTS IN CONGESTIVE CARDIAC FAILURE: AN OBSERVATIONAL STUDY FROM THE TRIBAL BELT OF GUJARAT, INDIA

Internal Medicine

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ABSTRACT

Background: Congestive cardiac failure (CCF) frequently causes secondary hepatic injury through elevated venous pressures and reduced perfusion, yet the clinical utility of liver function tests (LFTs) in this setting remains underexplored, particularly in resource-constrained tribal populations.**Objectives:** To characterize LFT abnormalities in CCF patients, examine their correlation with disease severity and etiology, assess their value during exacerbation and remission phases, and determine their prognostic utility for in-hospital outcomes and readmission.**Methods:** A prospective observational study enrolled 114 consecutive CCF patients at Zydus Medical College and Hospital, Dahod, from December 2023 to September 2025. Serum bilirubin (total and direct), AST, ALT, ALP, and albumin were measured. Clinical severity was graded by NYHA functional class. Statistical analysis employed one-way ANOVA, chi-square tests, and independent t-tests.**Results:** The mean total bilirubin was 1.72 ± 0.81 mg/dL, AST 66.4 ± 24.2 U/L, ALP 164.3 ± 49.6 U/L, and albumin 3.18 ± 0.54 g/dL across the cohort. A stepwise deterioration in all LFT parameters was observed with increasing NYHA class ($p < 0.001$ to $p = 0.015$). Elevated LFTs were present in 75.0% of patients during exacerbation versus 26.2% during remission ($p < 0.001$). Patients who deteriorated or expired had significantly higher mean total bilirubin (2.48 vs. 1.54 mg/dL; $p < 0.001$). Hypoalbuminaemia (< 3.0 g/dL) strongly predicted 30-day readmission (50.0% vs. 10.0%; $p = 0.011$). Inter-etiology LFT differences were statistically non-significant.**Conclusion:** LFT derangements in CCF are common, severity-linked, and largely reversible with decongestive therapy. Total bilirubin and serum albumin serve as accessible prognostic biomarkers. Routine LFT monitoring should be integrated into CCF management, especially in settings where advanced hemodynamic monitoring is unavailable.

KEYWORDS

Congestive Cardiac Failure; Liver Function Tests; Hepatic Congestion; Nyha Class; Tribal Population; Prognostic Biomarkers; Congestive Hepatopathy; Bilirubin; Hypoalbuminaemia

INTRODUCTION

The heart and liver maintain a close functional relationship that becomes pathologically apparent when cardiac output is compromised. In congestive cardiac failure (CCF), the simultaneous presence of elevated right-sided venous pressures and diminished forward flow creates a dual insult to the liver — passive congestion from the backpressure side and ischemic injury from the forward-failure component [1]. This combination disrupts hepatocyte function, impairs biliary excretion, and suppresses synthetic capacity, producing measurable biochemical changes that are readily detectable through routine liver function tests (LFTs) [2].

Hepatic involvement in CCF spans a spectrum from subtle transaminase elevations to frank congestive hepatopathy with perisinusoidal fibrosis. Bilirubin rises reflect cholestatic obstruction within congested sinusoids, while aminotransferase elevations signal hepatocellular injury from ischemia–reperfusion damage. Serum albumin, as a marker of hepatic synthetic function, declines with disease chronicity and serves as an indicator of both nutritional depletion and progressive parenchymal compromise [3]. These alterations are not mere epiphenomena — the Model for End-Stage Liver Disease excluding INR (MELD-XI), which integrates creatinine and bilirubin, independently predicts cardiac mortality and all-cause death across diverse heart failure cohorts, tying biochemical indices directly to outcome [4].

Despite this recognized association, the systematic use of LFTs as prognostic tools in CCF management remains inconsistent, particularly in peripheral and resource-limited settings. Most published evidence originates from urban tertiary referral centers, leaving tribal and rural populations undercharacterized. The tribal belt of North-East Gujarat represents a medically underserved region where late presentation, limited diagnostic resources, and high rates of comorbid hypertension and diabetes compound the disease burden [5]. In such contexts, low-cost biochemical markers like LFTs can potentially bridge the gap between symptomatic management and data-driven prognostication.

Emerging evidence also underlines the bidirectional nature of the cardiac-hepatic axis. Non-alcoholic fatty liver disease (NAFLD), prevalent in metabolically burdened tribal communities, sensitizes hepatocytes to congestion-mediated injury and independently elevates the risk of new-onset heart failure [6]. Serial LFT monitoring thus carries potential value not only for tracking disease progression but also for identifying individuals at heightened risk for decompensation before overt clinical deterioration occurs.

This study was therefore designed to prospectively characterize the pattern and severity of LFT abnormalities in CCF patients admitted at a tribal-belt tertiary hospital, assess their correlation with NYHA functional class and etiological subtype, track changes across exacerbation and remission phases, and evaluate their utility as accessible prognostic indicators for in-hospital outcomes and short-term readmission.

MATERIALS AND METHODS

Study Design and Setting

This prospective observational study was conducted in the Department of General Medicine, Zydus Medical College and Hospital (ZMCH), Dahod, Gujarat, from December 2023 to September 2025. ZMCH serves as the primary tertiary-care facility for the tribal belt of North-East Gujarat, drawing patients from a predominantly rural and tribal catchment area. The study was approved by the Institutional Ethics Committee of ZMCH (IEC Approval), and written informed consent was obtained from all participants prior to enrollment, in accordance with ICMR ethical guidelines and Good Clinical Practice standards.

Study Population and Sample Size

Consecutive patients admitted through the Outpatient Department, Inpatient Department, and Emergency Department with a clinical diagnosis of CCF were screened for eligibility. The sample size of 114 was determined using OpenEpi software (version 6), based on estimated prevalence of LFT abnormalities in heart failure populations, with a 95% confidence interval and alpha error of 0.05.

Inclusion criteria encompassed adult patients with CCF of any etiology — including ischemic heart disease (IHD), hypertensive heart disease (HHD), rheumatic valvular disease, dilated cardiomyopathy, and cor pulmonale — at first presentation or during exacerbation. Diagnosis was established using the Framingham criteria, requiring

two major or one major plus two minor criteria, supplemented by echocardiography where indicated.

Patients were excluded if they had a history of alcohol use, prior jaundice, active or resolved viral hepatitis (positive viral markers), or recent use of hepatotoxic medications such as rifampicin, isoniazid, phenytoin, valproate, steroids, or chlorpromazine, to minimize confounding hepatic pathology.

Clinical Assessment

A standardized pre-structured case-record form captured demographic details, symptom duration, comorbidities, and medication history. Clinical examination documented cardiovascular and hepatic findings including jugular venous distension, pedal edema, hepatomegaly, ascites, and signs of right or left ventricular failure. Heart failure severity was graded using the New York Heart Association (NYHA) functional classification. Abdominal ultrasonography was performed to assess hepatic size, echogenicity, and venous congestion graded from 0 (normal) to 3 (severe congestive hepatopathy).

Laboratory Investigations

Blood samples collected under aseptic conditions were analyzed at the Central Biochemistry Laboratory of ZMCH using standard automated analyzers with verified quality-control procedures. The LFT panel included total and direct serum bilirubin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), total protein, and serum albumin. Prothrombin time/INR, serum creatinine, urea, and electrolytes were measured as ancillary parameters. LFT measurements were repeated at remission wherever clinically feasible to assess dynamic changes.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 26. Continuous variables are expressed as mean ± standard deviation (SD); categorical variables as frequency and percentage. Between-group comparisons for continuous LFT parameters across NYHA classes and etiological subgroups were performed using one-way ANOVA. Chi-square tests were used for categorical comparisons. Independent samples t-test evaluated bilirubin differences by in-hospital outcome. A Jonckheere–Terpstra test assessed ordinal trends across ultrasound hepatopathy grades. Pearson correlation coefficient examined LFT-severity associations. A two-sided p-value < 0.05 was considered statistically significant throughout.

RESULTS

Demographic and Clinical Characteristics

A total of 114 patients were enrolled. The majority (58.8%) were aged ≥ 60 years, with a male preponderance (59.6%). Over four-fifths (80.7%) were from tribal communities within the ZMCH catchment area (Table 1). Ischemic heart disease was the leading etiology (40.4%), followed by hypertensive heart disease (28.1%) and valvular disease (17.5%). Most patients presented with advanced disease — 38.6% were in NYHA Class III and 24.6% in Class IV. Hypertension (54.4%) and diabetes mellitus (33.3%) were the most prevalent comorbidities; chronic kidney disease coexisted in 19.3% of patients.

Table 1. Baseline Demographic and Clinical Profile of the Study Population (n = 114)

Characteristic	Category	n	%
Age Group	< 40 years	8	7.0
	40–59 years	39	34.2
	≥ 60 years	67	58.8
Gender	Male	68	59.6
	Female	46	40.4
Residence	Tribal	92	80.7
	Non-tribal	22	19.3
Etiology of CCF	Ischemic Heart Disease	46	40.4
	Hypertensive Heart Disease	32	28.1
	Valvular Heart Disease	20	17.5
	Dilated Cardiomyopathy	10	8.8
	Others	6	5.3
NYHA Functional Class	Class I	10	8.8
	Class II	32	28.1
	Class III	44	38.6
	Class IV	28	24.6
Major Comorbidities	Hypertension	62	54.4
	Diabetes Mellitus	38	33.3
	Chronic Kidney Disease	22	19.3
	COPD/Asthma	12	10.5
	No major comorbidity	18	15.8

CCF = congestive cardiac failure; DCM = dilated cardiomyopathy; NYHA = New York Heart Association; HD = heart disease

Overall LFT Profile and Correlation with NYHA Class

Across the cohort, all LFT parameters were elevated above their respective reference upper limits. The mean total bilirubin was 1.72 ± 0.81 mg/dL, AST 66.4 ± 24.2 U/L, ALT 58.9 ± 21.7 U/L, and ALP 164.3 ± 49.6 U/L. Serum albumin was below the normal threshold at 3.18 ± 0.54 g/dL, indicating impaired hepatic synthetic function. A progressive, stepwise deterioration in all parameters was observed with increasing NYHA class. Total bilirubin rose from 1.12 mg/dL in NYHA I–II to 2.32 mg/dL in NYHA IV, while albumin declined from 3.42 to 2.86 g/dL over the same gradient (Table 2). ANOVA revealed highly significant differences for total bilirubin ($p < 0.001$), AST ($p < 0.001$), ALT ($p = 0.002$), ALP ($p = 0.015$), and albumin ($p = 0.004$), confirming a robust severity-dependent relationship.

Table 2. Overall LFT Parameters and Stepwise Correlation with NYHA Functional Class

LFT Parameter	Overall Mean ± SD	NYHA I–II	NYHA III	NYHA IV	p-value*
Total Bilirubin (mg/dL)	1.72 ± 0.81	1.12	1.68	2.32	< 0.001
Direct Bilirubin (mg/dL)	0.68 ± 0.35	—	—	—	—
AST (U/L)	66.4 ± 24.2	48.1	64.5	82.3	< 0.001
ALT (U/L)	58.9 ± 21.7	44.6	57.4	71.2	0.002
ALP (U/L)	164.3 ± 49.6	142.7	162.9	182.4	0.015
Serum Albumin (g/dL)	3.18 ± 0.54	3.42	3.12	2.86	0.004

*One-way ANOVA. AST = aspartate aminotransferase; ALT = alanine aminotransferase; ALP = alkaline phosphatase; NYHA = New York Heart Association; SD = standard deviation

LFT Patterns by Etiology and Imaging Correlation

Comparison of LFT parameters across etiological subgroups (IHD, HHD, valvular disease, DCM) revealed no statistically significant differences in total bilirubin, AST, ALP, or albumin (all $p > 0.05$), suggesting that hepatic dysfunction in CCF is driven primarily by shared hemodynamic mechanisms — namely venous congestion and reduced cardiac output — rather than the specific underlying etiology (Table 3). IHD patients exhibited marginally higher bilirubin (1.86 mg/dL) and AST (69.5 U/L), though within the overlap of other groups.

Ultrasound-based grading of congestive hepatopathy demonstrated a clear concordance with biochemical indices. Mean total bilirubin rose from 1.22 mg/dL at Grade 0 to 2.44 mg/dL at Grade 3, while ALP increased from 148 to 191 U/L across the same gradient (Jonckheere–Terpstra trend test; $p < 0.001$). This imaging–biochemistry concordance reinforces the pathophysiological basis for LFT derangements in CCF and validates their construct validity in this population.

Table 3. LFT Parameters by Etiology and Ultrasound Congestive Hepatopathy Grade

Group	Total Bilirubin (mg/dL)	AST (U/L)	ALP (U/L)	Serum Albumin (g/dL)	p-value*
Etiology					
IHD	1.86	69.5	—	3.10	> 0.05
Hypertensive HD	1.55	60.3	—	3.22	> 0.05
Valvular HD	1.74	63.8	—	3.16	> 0.05
DCM	1.63	66.1	—	3.09	> 0.05
Ultrasound Congestive Hepatopathy Grade					
Grade 0	1.22	—	148	—	< 0.001
Grade 1	1.61	—	159	—	
Grade 2	2.03	—	176	—	

Grade 3	2.44	—	191	—	

*One-way ANOVA for etiology comparisons; Jonckheere–Terpstra trend test for USG grades. IHD = ischemic heart disease; DCM = dilated cardiomyopathy; HD = heart disease; — = not assessed in this subset

Prognostic Significance of LFT Parameters

Elevated LFTs were present in 75.0% of patients during exacerbation phases, compared with only 26.2% during remission (chi-square, $p < 0.001$), confirming the state-dependent, largely reversible nature of hepatic injury in CCF (Table 4). Patients with adverse in-hospital outcomes (deterioration or death, n = 18) had a significantly higher mean total bilirubin of 2.48 mg/dL compared with 1.54 mg/dL in those who improved or remained stable ($n = 96$; $p < 0.001$).

Serum albumin emerged as a particularly powerful prognostic discriminator. Among patients with albumin < 3.0 g/dL, 35.7% experienced adverse in-hospital outcomes and 50.0% were readmitted within 30 days. In contrast, only 5.9% of patients with albumin > 3.5 g/dL experienced deterioration, and 10.0% were readmitted (chi-square, $p = 0.011$). A direct positive relationship between mean total bilirubin and length of hospital stay was also observed: bilirubin averaged 1.42 mg/dL in patients discharged within five days, rising to 2.21 mg/dL in those requiring more than ten days of hospitalization (one-way ANOVA, $p = 0.003$).

Table 4. Prognostic Significance of LFT Parameters: Clinical Phase, In-hospital Outcome, Length of Stay, and 30-day Readmission

Prognostic Parameter	Category	n	Mean Total Bilirubin (mg/dL) / Elevated LFTs (%)	p-value*
Clinical Phase				
Elevated LFTs (% patients)	Remission	42	26.2%	< 0.001
	Exacerbation	72	75.0%	
In-hospital Outcome				
Mean Total Bilirubin	Improved / Stable	96	1.54 mg/dL	< 0.001
	Deteriorated / Expired	18	2.48 mg/dL	
Albumin < 3.0 g/dL	Deteriorated / Expired	10/28	35.7% adverse outcome	< 0.001
Albumin 3.0–3.5 g/dL		6/52	11.5% adverse outcome	
Albumin > 3.5 g/dL		2/34	5.9% adverse outcome	
Length of Hospital Stay				
Mean Total Bilirubin	≤ 5 days	—	1.42 mg/dL	0.003
	6–10 days	—	1.88 mg/dL	
	> 10 days	—	2.21 mg/dL	
30-Day Readmission by Albumin Category				
Readmission rate	Albumin < 3.0 g/dL	28	50.0% readmitted	0.011
	Albumin 3.0–3.5 g/dL	46	21.7% readmitted	
	Albumin > 3.5 g/dL	40	10.0% readmitted	

**Chi-square test for categorical outcomes; one-way ANOVA for length-of-stay analysis; independent t-test for in-hospital outcome bilirubin comparison. LFT = liver function test*

DISCUSSION

This study provides a comprehensive characterization of hepatic biochemical alterations in CCF patients from a tribal-belt tertiary center, demonstrating that LFT derangements are highly prevalent, severity-linked, dynamically responsive to clinical phase, and prognostically informative. Taken together, our findings support integrating routine LFT monitoring into standard CCF management protocols, particularly in resource-limited settings where invasive hemodynamic assessment is impractical.

The demographic profile — predominantly elderly males with advanced NYHA class at presentation — reflects the epidemiological reality of CCF in peripheral India, where care-seeking is often delayed until symptoms are debilitating. The 80.7% tribal composition of the cohort mirrors the hospital catchment but also flags the geographic and socioeconomic determinants of late presentation, a pattern well recognized in rural cardiovascular cohorts [7]. Ischemic heart disease as the leading etiology (40.4%) is consistent with the national shift in CCF burden toward ischemic and hypertensive substrates [8]. The high prevalence of comorbid hypertension, diabetes, and CKD introduces compound hemodynamic and metabolic pressures on hepatic function, potentially amplifying LFT derangements beyond what the cardiac lesion alone would produce.

The overall LFT profile — moderately elevated bilirubin and transaminases with reduced albumin — mirrors patterns reported in multicenter studies of acute and chronic heart failure [1,2]. The mixed hepatocellular (raised AST/ALT) and cholestatic (raised bilirubin and ALP) biochemical picture is consistent with the pathophysiology of congestive hepatopathy, wherein sustained elevation of central venous pressure causes sinusoidal congestion, perisinusoidal oedema, and canalicular compression, while coexistent low cardiac output induces ischemia-reperfusion injury to zone 3 hepatocytes [3,9].

The stepwise deterioration of LFT parameters across NYHA classes ($p < 0.001$ to $p = 0.015$) provides strong statistical evidence for the severity-dependent nature of cardiac hepatopathy. This gradient corroborates findings from prior studies which demonstrated that the degree of LFT abnormality parallels hemodynamic burden [10]. The simultaneous decline in albumin across NYHA classes reflects not only reduced hepatic synthetic capacity but also the catabolic state and subclinical malnutrition that accompany advanced heart failure, both of which compound prognostic risk [11]. Notably, the absence of significant inter-etiology differences underscores that hepatic dysfunction in CCF represents a "final common pathway" of congestion, irrespective of whether the primary cardiac lesion is ischemic, hypertensive, valvular, or myopathic [7].

The sharp contrast in LFT abnormality rates between exacerbation (75.0%) and remission (26.2%) phases illustrates the dynamic and largely reversible nature of the hepatic insult in CCF. This reversibility is physiologically coherent: effective diuretic-mediated decongestion reduces sinusoidal hydrostatic pressure and sinusoidal oedema, allowing transaminase and bilirubin levels to normalize, typically within days of haemodynamic improvement [12]. However, the persistence of abnormal LFTs in a quarter of patients during remission suggests that a subset sustains irreversible architectural change — perisinusoidal fibrosis or parenchymal scarring — that does not resolve with short-term volume management alone [13].

The prognostic utility of LFTs in this cohort was clinically meaningful. A mean total bilirubin difference of nearly 1 mg/dL between adverse and favorable outcome groups ($p < 0.001$) positions bilirubin as a straightforward, widely available risk stratifier. Similar associations have been reported from studies incorporating bilirubin into composite scores such as MELD-XI, where higher values predicted mortality and rehospitalization independent of traditional cardiac variables [4,14]. The albumin-outcome relationship was equally compelling: hypoalbuminaemia below 3.0 g/dL concentrated adverse outcomes and doubled 30-day readmission risk compared with albumin above 3.5 g/dL ($p = 0.011$), consistent with albumin's role as an integrator of hepatic synthetic reserve, inflammatory catabolism, and nutritional depletion [15].

The concordance between ultrasound congestive hepatopathy grades and biochemical parameters ($p < 0.001$) validates a combined clinical-biochemical-imaging approach to severity assessment. In settings where elastography or advanced hepatic imaging is unavailable, conventional ultrasound combined with routine LFTs can provide comparable risk-stratification information at minimal cost.

From a practical standpoint, serial LFT monitoring in CCF can serve multiple clinical functions: flagging early decompensation before overt haemodynamic deterioration, guiding intensity of diuretic therapy, informing discharge readiness, and identifying patients requiring more intensive post-discharge follow-up. The present study adds region-specific evidence to support this practice within tribal-belt healthcare systems where point-of-care biochemistry remains more

accessible than echocardiography or invasive hemodynamic monitoring.

LIMITATIONS

This study has several limitations that warrant acknowledgment. The single-center design limits external generalizability. The sample size, while adequate for the primary analyses, may be insufficient for robust multivariate modeling. Longitudinal follow-up was limited, precluding assessment of chronic hepatic consequences. Advanced hepatic imaging (elastography, hepatic venous pressure gradient) was not uniformly performed. The observational design precludes causal inference. Future multicenter studies with extended follow-up are needed to characterize long-term LFT trajectories and validate composite prognostic models in this population.

CONCLUSION

Liver function test abnormalities are highly prevalent and clinically significant in congestive cardiac failure. Derangements in total bilirubin, AST, ALT, ALP, and serum albumin correlate strongly with NYHA functional class severity and normalize substantially during clinical remission, underscoring their dynamic, hemodynamically driven nature. Inter-etiology LFT differences are not statistically significant, supporting a common congestion-mediated mechanism. Total bilirubin and serum albumin are actionable prognostic biomarkers that predict in-hospital adverse outcomes, prolonged hospitalization, and 30-day readmission. Given their low cost, universal availability, and demonstrated clinical utility, LFTs should be incorporated as a routine component of CCF assessment and monitoring — particularly in tribal and rural settings where they can guide management decisions in the absence of advanced diagnostic infrastructure.

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DECLARATIONS

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