The Egyptian Journal of Hospital Medicine (October 2025) Vol. 101, Page 5084-5093

Liver Function Abnormalities in Patients with Acute Heart Failure in Sohag University Hospitals

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ABSTRACT

Background: Liver dysfunction is a frequently under-recognized complication in patients with acute heart failure (AHF), contributing to adverse outcomes and reflecting underlying hemodynamic compromise.

Objective: This study aimed to evaluate liver function tests (LFTs) abnormalities in patients with AHF and to identify cardiac and non-cardiac factors associated with hepatic impairment. **Patients and methods:** This observational study included 100 adult patients presenting with AHF to the Internal Medicine and Cardiology Departments at Sohag University Hospitals. Clinical evaluation, laboratory investigations (including LFTs, CBC, renal function, lipid profile, and cardiac biomarkers), echocardiography, chest radiography, and abdominal ultrasound were performed. Patients were classified according to AHF type, ejection fraction phenotype, and presence of valvular disease. Associations between LFTs abnormalities and echocardiographic parameters, AHF subtypes, and comorbidities were statistically analyzed.

Results: Elevated liver enzymes were common, with ALT and AST abnormal in 85% and 92% of patients respectively. Hypoalbuminemia was observed in 60%, total bilirubin was elevated in 34%, and INR was abnormal in 27%. Tricuspid regurgitation (TR) and mitral regurgitation (MR) were present in 65% and 60% of cases respectively, and showed significant associations with worsening liver parameters. Patients with reduced ejection fraction (HFrEF) and those presenting with cardiogenic shock or acute coronary syndrome (ACS) had significantly higher levels of ALT, AST, and bilirubin. Hepatic ultrasound revealed congestive liver changes in 70% of patients. Significant correlations were found between LFT abnormalities and TR severity, MR severity, reduced EF, and presence of ACS ($p < 0.05$).

Conclusion: Hepatic dysfunction was common in AHF, associated with reduced function, regurgitation, shock, and ACS. Enzyme and bilirubin elevations reflected valvular severity, while hepatic congestion showed weaker associations.

Keywords: Acute heart failure**,** Liver function tests**,** Tricuspid regurgitation**,** Hepatic congestion**,** Ejection fraction.

INTRODUCTION

Acute heart failure (AHF) represents a common and life-threatening cardiovascular emergency associated with significant morbidity and mortality worldwide. It is characterized by the rapid onset or worsening of symptoms related to cardiac dysfunction, including pulmonary congestion, hypoperfusion, and multi-organ involvement. Among the extra-cardiac organs affected, the liver is particularly vulnerable due to its dual blood supply from the hepatic artery and portal vein, which makes it highly sensitive to hemodynamic alterations [1].

Hepatic dysfunction in the setting of AHF, often referred to as "cardiohepatic syndrome", arises from venous congestion, impaired cardiac output, and hypoxic injury. These pathophysiological mechanisms contribute to abnormalities in liver function tests (LFTs), including elevated transaminases, bilirubin, and cholestatic markers. Such abnormalities are not only common in AHF patients but also hold important prognostic implications [2]. Previous studies have demonstrated that both congestive hepatopathy and ischemic hepatitis can complicate the clinical course of AHF. Congestive hepatopathy results primarily from increased central venous pressure leading to sinusoidal congestion, while ischemic hepatitis is linked to reduced hepatic perfusion and hypoxia. The presence of either condition has been correlated with adverse outcomes, longer hospital stays, and higher in-hospital mortality rates [3]. Furthermore, liver function abnormalities may serve as biomarkers that reflect systemic congestion and disease severity in AHF [4]. Elevated bilirubin and alanine aminotransferase (ALT) levels have been reported as independent predictors of poor prognosis in hospitalized patients with decompensated heart failure. Hence, routine assessment of hepatic function is increasingly emphasized in clinical practice for early risk stratification and therapeutic decision-making [5].

Despite growing recognition, the exact prevalence and prognostic significance of liver dysfunction in AHF remain incompletely understood. Variability in definitions, diagnostic methods, and patient populations has led to conflicting results across studies [5]. Moreover, it is not fully established whether liver abnormalities directly contribute to worse outcomes or simply reflect the severity of the underlying cardiac dysfunction [6]. Therefore, our study aimed to address these gaps by systematically evaluating hepatic dysfunction among patients admitted with acute heart failure.

PATIENTS AND METHODS

Study design: This observational study was conducted in the Internal Medicine and Cardiology Departments of Sohag University Hospitals.

Patient selection: Eligible participants were adults (≥18 years) diagnosed with acute heart failure (AHF) per the 2021 European Society of Cardiology (ESC) guidelines, requiring urgent intervention and unplanned admission. Presentations included acute decompensated heart failure, pulmonary edema, hypertensive AHF, cardiogenic shock and ACS with heart failure. Patients with congenital heart disease or liver disorders affecting liver function were excluded.

Received: 14/07/2025
Accepted: 14/09/2025

Methods: Patients received structured clinical assessments, including medical history (symptoms, cardiovascular history, comorbidities and medications) and physical examination with focus on cardiovascular and respiratory systems, systemic congestion, volume overload, and hepatic signs. Laboratory investigations included liver function tests (ALT, AST, alkaline phosphatase, total bilirubin, serum albumin and prothrombin time). Additional tests included CBC, random blood sugar, lipid profile, serum creatinine and cardiac troponin. NT-proBNP was measured selectively in cases where heart failure diagnosis remained uncertain, serving as a biomarker of myocardial wall stress. All patients underwent 12-lead ECG for rhythm analysis, ischemic changes and conduction abnormalities. Transthoracic echocardiography was performed to evaluate left ventricular ejection fraction (LVEF), wall motion, and structural or valvular disease. Abdominal ultrasonography assessed liver size, contour, echotexture, and coexisting pathology potentially affecting LFTs. Chest radiographs were obtained to detect pulmonary congestion, cardiomegaly, and pleural effusion.

Ethical approval: This study was approved from the Faculty of Medicine Ethics' Committee, Sohag University. All participants provided written informed consents prior to enrollment. Throughout its implementation, the study complied with the Helsinki Declaration.

Statistical analysis

Data were analyzed using IBM SPSS version 27. Normality was assessed with the Kolmogorov–Smirnov test. Continuous variables were expressed as mean ± SD or median (IQR), and categorical variables as frequencies (%). Chi-square, independent t-test, Mann–Whitney U, and Friedman's test were applied as appropriate. Statistical significance was defined as $p \leq 0.05$ (highly significant at $p \leq 0.001$).

RESULTS

This study included 100 patients presenting with AHF. Among the study cohort, 60% were males and 40% were females, yielding a male-to-female ratio of 1.5:1. The age ranged from 18 to 96 years, with a mean of 62.18 ± 14.28 years and a median of 56 years. The mean BMI was 28.58 ± 6.87 kg/m², ranging from 16 to 40 kg/m² (Table 1).

Table (1): Demographic characteristics among the studied cases

Parameters		Studied cases (N= 100)	
		N	%
Gender	Male	60	60.0%
	Female	40	40.0%
Age (years)	Mean± SD	62.18± 14.28	
	Median (Range)	56 (18 – 96)	
BMI (Kg/m²)	Mean± SD	28.58± 6.87	
	Median (Range)	30 (16 – 40)	

All patients (100%) presented with dyspnea and orthopnea, while 34% experienced paroxysmal nocturnal dyspnea. Chest pain was reported in 22%, and jaundice was observed in 2%. Regarding ascites, 1% of patients had marked ascites, 2% had moderate ascites, 8% mild ascites, and 89% had no ascites. Lower limb edema was universally present: 41% of cases had mild, 38% moderate, and 21% marked edema. Comorbid conditions were common: 44% had diabetes mellitus (DM) and 41% had hypertension. A history of ischemic heart disease was found in 71% of patients, and 20% presented with ACS on admission. Regarding prior cardiac interventions, 18% had undergone PCI or CABG, 13% had previous myocardial infarction, and 12% reported a heart failure episode within the past six months. The most frequent type of AHF was pulmonary edema (35%), followed by de novo AHF (18%), and cardiogenic shock (16%). Other forms included ACS complicated by HF (14%), AHF on top of chronic HF (11%), and hypertensive pulmonary edema (6%) (Figure 1).

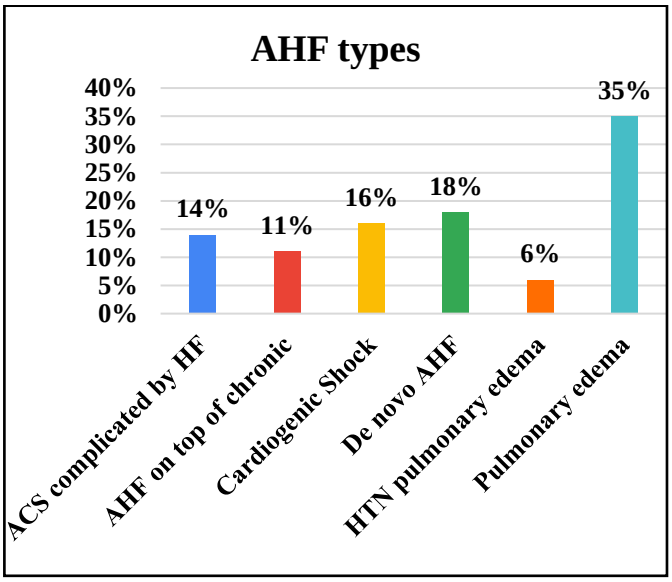

Figure (1): Distribution of the studied cases regarding AHF types.

Liver enzyme levels were significantly elevated in most patients. 82 out of 100 patients (82%) had an elevated ALT levels. The median ALT level was 116 IU/L (Range: 4.0- 1975 IU/l).92 % of the case had an elevated AST level, the median was 120 IU/L(Range: 14-2138 IU/L). 60% of patients exhibited aberrant albumin levels, with a median serum albumin concentration of 3.2 g/dL (Range: 1.8-205 g/dL). INR was elevated in 27% of instances, presenting a median of 1.1 (Range: 0.5-15). Abnormal serum total bilirubin levels were observed in 34% of cases, with a median of 0.8 mg/dL (Range: 0.2-23 mg/dL). Additionally, alkaline phosphatase (ALP) levels were elevated in 37% of cases, with the median level recorded at 103 IU/L (Range: 50-300 IU/L) (Table 2). Troponin levels were positive in 22% of patients, while 78% tested negative.

Table (2): Liver function test among the studied cases

	Studied cases (N= 100)				
	Median	IQR		Range	
ALT (IU/L)	116.0	79.0	200.0	4.0	1975.0
Abnormal ALT level	82 (85.0%)				
AST (IU/L)	120.0	67.5	157.5	14.0	2138.0
Abnormal AST level	92 (92.0%)				
Albumin (g/dL)	3.2	3.0	3.6	1.8	205.0
Abnormal albumin level	60 (60.0%)				
INR	1.1	1.0	1.3	0.5	15.0
Abnormal INR level	27 (27.0%)				
Total bilirubin (mg/ dL)	0.8	0.5	2.0	0.2	23.0
Abnormal bilirubin level	34 (34.0%)				
ALP (IU/L)	103.0	76.0	145.5	50.0	300.0
Abnormal ALP level	37 (37.0%)				

ALT: alanine aminotransferase, AST: aspartate aminotransferase, ALP: Alkaline phosphatase.

Tricuspid regurgitation (TR) was present in 65% of patients: 28% had mild, 22% moderate, and 15% severe TR. The mean ejection fraction (EF) was 42.07 ± 14.28%, with a range from 18% to 68%. Regarding mitral regurgitation (MR), 60% of patients had varying degrees: 22% mild, 27% moderate, and 11% severe MR. Based on EF, 46% had reduced EF (HFrEF), 20% mildly reduced (HFmrEF), and 34% preserved EF (HFpEF). Atrial fibrillation (AF) was noted in 20%, and global hypokinesia in 65%. Segmental wall motion abnormalities (SWMA) were observed in 75% of patients. Rheumatic heart disease was present in only 7%. Cardiomegaly was seen in 92%, pulmonary congestion in 96%, pericardial effusion in 6%, and pleural effusion in 75% (Table 3).

Table (3): Echocardiography findings among the studied cases

Parameters		Studied cases (N= 100)	
		N	**%**
TR	**Mild**	28	28.0%
	Moderate	22	22.0%
	Severe	15	15.0%
	No	35	35.0%
MR	**Mild**	22	22.0%
	Moderate	27	27.0%
	Severe	11	11.0%
	No	40	40.0%
EF (%)	**Mean± SD**	42.07± 14.28	
	Median (Range)	40 (18 – 68)	
EF HF	**HFrEF (Reduced EF)**	46	46.0%
	HFmrEF (Mildly Reduced EF)	20	20.0%
	HFpEF (Preserved EF)	34	34.0%
AF	**No**	80	80.0%
	Yes	20	20.0%
Global hypokinesia	**No**	35	35.0%
	Yes	65	65.0%
SWMA	**No**	25	25.0%
	Yes	75	75.0%
RHD	**No**	93	93.0%
	Yes	7	7.0%
Cardiomegaly	**No**	8	8.0%
	Yes	92	92.0%
Pulmonary congestion	**No**	4	4.0%
	Yes	96	96.0%
Pericardial effusion	**No**	94	94.0%
	Yes	6	6.0%
Pleural effusion	**No**	25	25.0%
	Yes	75	75.0%

EF: ejection fraction, TR: Tricuspid regurgitation, MR: Mitral regurgitation SWMA: segmental wall motion abnormalities, RHD: Rheumatic heart disease

Hepatic congestion with fatty changes was detected in 70% of patients, while 30% had no significant hepatic findings. Concerning, association of TR severity with LFTs, there was a significant correlation between TR severity and LFT abnormalities. As TR severity increased, ALT, AST, ALP, and total bilirubin levels rose significantly, while albumin levels decreased. For instance, median ALT increased from 95 IU/L in mild TR to 250 IU/L in severe TR (p = 0.005), and albumin declined from 3.5 to 2.8 g/dL (p = 0.027) (Table 4).

Table (4): Effect of TR severity on LFTs

Parameters	TR	Studied cases (N= 100)					Kruskal Wallis test	
		Median	IQR		Range		Kw	P-value
ALT (IU/L)	**Mild**	95	75	110.5	220	95	12.845	**0.005****
	Moderate	160	100	180	360	160		
	Severe	250	150	290	550	250		
	No	70	50	85	200	70		
AST (IU/L)	**Mild**	90	70	105	200	90	10.982	**0.012***
	Moderate	140	90	160	300	140		
	Severe	220	130	250	480	220		
	No	65	45	75	160	65		
Albumin (g/dL)	**Mild**	3.5	3.3	3.6	4.2	3.5	9.147	**0.027***
	Moderate	3.2	3	3.1	3.8	3.2		
	Severe	2.8	2.6	2.7	3.4	2.8		
	No	3.8	3.5	3.9	4.5	3.8		
INR	**Mild**	1.1	1	1.15	1.5	1.1	8.659	**0.034***
	Moderate	1.3	1.2	1.4	2	1.3		
	Severe	1.6	1.4	1.7	2.5	1.6		
	No	1	0.9	1.05	1.4	1		
Total bilirubin (mg/ dL)	**Mild**	0.8	0.6	0.9	2	0.8	11.241	**0.010***
	Moderate	1.3	0.9	1.5	3.5	1.3		
	Severe	2.4	1.8	2.6	5	2.4		
	No	0.6	0.4	0.7	1.5	0.6		
ALP (IU/L)	**Mild**	100	80	110	180	100	10.537	**0.015***
	Moderate	150	120	160	230	150		
	Severe	210	180	225	320	210		
	No	85	70	90	150	85		

P value >0.05: Not significant, *P value <0.05 is significant, **p<0.01 is highly significant.

Elevated ALT and AST levels were significantly associated with severe TR, HFrEF, and ACS. For example, 89.7% of patients with high ALT/AST had HFrEF (p = 0.007), and 51.7% had ACS (p = 0.002). Cardiogenic shock was more prevalent in patients with elevated enzymes (27.6% vs. 11.3%, p = 0.036) (Figure 2).

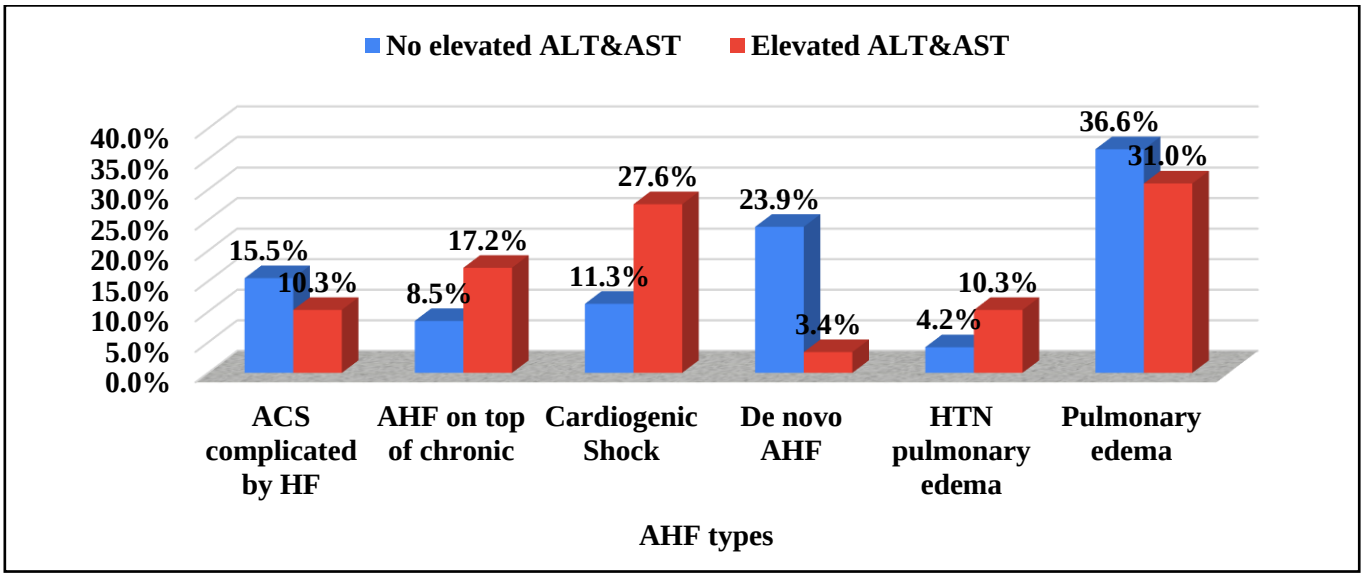

Figure (2): Relation between hepatocellular affection with types of AHF.

Mitral regurgitation severity was significantly associated with albumin and ALP levels. Severe MR was associated with lower albumin (median 3.1 g/dL, p = 0.045) and higher ALP (median 143 IU/L, p = 0.012). Other LFTs (ALT, AST, INR and bilirubin) showed no significant differences. No significant differences in TR or MR severity were found between patients with or without congested liver. However, global hypokinesia was significantly more common in patients with congested liver (p = 0.012). There was also no significant association between AHF type and liver congestion. Patients with ACS had significantly higher liver enzyme levels compared to those without ACS. Median ALT was 180 vs. 65 IU/L, AST 200 vs. 70 IU/L, bilirubin 2.1 vs. 0.7 mg/dL, and ALP 210 vs. 95 IU/L. Albumin and INR also significantly worsened in the ACS group (Table 5).

Table (5): Relation between LFTs tests and presence of ACS

Parameters	AHF patients without ACS (N= 80)			AHF patients with ACS (N= 20)			Kruskal Wallis test	
	Median	IQR		Median	IQR		Kw	P-value
ALT (IU/L)	65	50	100	180	130	250	10.845	**0.001****
AST (IU/L)	70	55	120	200	150	280	9.672	**0.002****
Albumin (g/dL)	3.7	3.5	4	3	2.8	3.2	7.931	**0.005****
INR	1.1	1	1.3	1.6	1.4	2	8.347	**0.004****
Total bilirubin (mg/dL)	0.7	0.5	1	2.1	1.7	3.5	11.154	**0.001****
ALP (IU/L)	95	75	130	210	170	280	8.765	**0.003****

P value >0.05: Not significant, *P value <0.05 is significant, **p<0.01 is highly significant.

Patients with elevated bilirubin were more likely to have moderate/severe TR, moderate MR, HFrEF, and ACS. For example, all patients with elevated bilirubin had reduced EF (p = 0.016), and 34.5% had ACS (p = 0.008) (Table 6). Pulmonary edema was significantly more common in this group (57.7% vs. 28.4%, p = 0.046).

Table (6): Relation between elevated bilirubin and echocardiography findings

		Elevated bilirubin				Chi- Square test	
		No (N=74)		Yes (N=26)			
		No.	%	No.	%	X^2	P-value
TR	**Mild**	23	31.10%	5	17.2%	9.85	**0.020***
	Moderate	10	13.50%	12	41.4%		
	Severe	6	8.10%	9	31.0%		
	No	35	47.30%	0	0.0%		
MR	**Mild**	19	25.7%	3	11.5%	8.638	**0.035***
	Moderate	15	20.3%	12	46.2%		
	Severe	7	9.5%	4	15.4%		
	No	33	44.6%	7	26.9%		
EF HF	**HFrEF (Reduced EF)**	20	27.00%	26	89.7%	8.267	**0.016***
	HFmrEF (Mildly Reduced EF)	20	27.00%	0	0.0%		
	HFpEF (Preserved EF)	34	45.90%	0	0.0%		
ACS	**No**	64	86.50%	16	55.2%	6.94	**0.008****
	Yes	10	13.50%	10	34.5%		
AF	**No**	61	82.4%	19	73.1%	1.052	0.305
	Yes	13	17.6%	7	26.9%		
AHF types	**ACS complicated by HF**	11	14.9%	3	11.5%	5.560	0.361^{MC}
	AHF on top of chronic	10	13.5%	1	3.8%		
	Cardiogenic Shock	10	13.5%	6	23.1%		
	De novo AHF	13	17.6%	5	19.2%		
	HTN pulmonary edema	6	8.1%	0	0.0%		
	Pulmonary edema	24	32.4%	11	42.3%		
Global hypokinesia	**No**	26	35.1%	9	34.6%	0.002	0.962
	Yes	48	64.9%	17	65.4%		
SWMA	**No**	17	23.0%	8	30.8%	0.624	0.430
	Yes	57	77.0%	18	69.2%		
RHD	**No**	71	95.9%	22	84.6%	3.794	0.185^{FET}
	Yes	3	4.1%	4	15.4%		
Cardiomegaly	**No**	6	8.1%	2	7.7%	0.005	$>0.999^{FET}$
	Yes	68	91.9%	24	92.3%		
Pulmonary congestion	**No**	4	5.4%	0	0.0%	1.464	0.346^{FET}
	Yes	70	94.6%	26	100.0%		
Pleural effusion	**No**	21	28.4%	4	15.4%	1.733	0.188
	Yes	53	71.6%	22	84.6%		
Pericardial effusion	**No**	70	94.6%	24	92.3%	0.178	0.680^{FET}
	Yes	4	5.4%	2	7.7%		

P value >0.05: Not significant, *P value <0.05 is significant, **p<0.01 is highly significant, X^2: Chi- Square test.

Significant differences in ALT, AST, and total bilirubin were observed across AHF types. The highest enzyme levels were recorded in cardiogenic shock, while the lowest were in de novo AHF. However, albumin, INR, and ALP did not significantly differ (Table 7)

Table (7): Effect of HF types on LFTs

Parameters	HF types	Studied cases					Kruskal Wallis test	
		Median	IQR		Range		Kw	P-value
ALT (IU/L)	ACS complicated by HF	180	85	307	4	1216	13.14	**0.022***
	AHF on top of chronic	114	89	122	10	820		
	Cardiogenic Shock	157	67	351	12	1628		
	De novo AHF	112	93	119	36	154		
	HTN pulmonary edema	116	113	180	100	223		
	Pulmonary edema	111	69	190	19	1975		
AST (IU/L)	ACS complicated by HF	164	89	251	14	403	12.97	**0.024***
	AHF on top of chronic	113	70	136	34	830		
	Cardiogenic Shock	122	81	357	26	2046		
	De novo AHF	124	60	137	38	155		
	HTN pulmonary edema	114	100	146	64	186		
	Pulmonary edema	114	64	153	27	2138		
Albumin (g/dL)	ACS complicated by HF	3.3	3.0	3.4	2.1	4.6	4.38	0.497
	AHF on top of chronic	3.1	2.6	3.5	1.8	3.7		
	Cardiogenic Shock	3.8	3.5	3.9	3.0	24.0		
	De novo AHF	3.2	3.0	3.5	2.4	205.0		
	HTN pulmonary edema	3.0	3.0	3.2	2.9	3.7		
	Pulmonary edema	3.2	2.8	3.5	2.2	4.1		
INR	ACS complicated by HF	1.1	1.0	1.2	0.7	15.0	10.06	0.074
	AHF on top of chronic	1.1	1.0	2.4	0.9	12.0		
	Cardiogenic Shock	1.2	1.1	1.3	0.5	3.2		
	De novo AHF	1.1	1.0	1.2	0.6	3.1		
	HTN pulmonary edema	1.0	0.7	1.1	0.7	1.1		
	Pulmonary edema	1.1	0.9	1.2	0.6	2.4		
Total bilirubin (mg/ dL)	ACS complicated by HF	1.4	0.6	1.9	0.4	6.2	14.7	**0.012***
	AHF on top of chronic	0.4	0.4	2.6	0.3	23.0		
	Cardiogenic Shock	0.9	0.7	3.9	0.2	16.0		
	De novo AHF	0.6	0.4	1.1	0.2	2.7		
	HTN pulmonary edema	0.8	0.5	1.0	0.4	1.2		
	Pulmonary edema	0.8	0.6	2.3	0.3	19.0		
ALP (IU/L)	ACS complicated by HF	130	78	143	51	300	6.17	0.290
	AHF on top of chronic	97	73	146	58	300		
	Cardiogenic Shock	105	94	140	65	280		
	De novo AHF	96	68	126	60	157		
	HTN pulmonary edema	70	52	106	50	110		
	Pulmonary edema	126	86	160	61	300		

P value >0.05: Not significant, *P value <0.05 is significant, **p<0.01 is highly significant.

DISCUSSION

Acute heart failure (AHF) is a medical emergency that can impair liver function due to hemodynamic disturbances. Hepatic congestion from elevated right heart pressures or reduced perfusion from low cardiac output can lead to liver enzyme elevation, hyperbilirubinemia, and hypoalbuminemia, collectively known as cardiac or congestive hepatopathy. In severe cases, such as cardiogenic shock, acute liver injury with marked enzyme elevation may occur [7].

The mean age was 62.18 ± 14.28 years, with a male predominance (60%), and a mean BMI of 28.58 ± 6.87 kg/m². These figures align closely with **Myhre *et al.*** [8] who reported a mean age of 63 ± 14 years with 61% male patients, and **Zymliński *et al.*** [9] who found a slightly older cohort with a mean age of 67 ± 12 years and 72% males. Clinically, all patients in our study presented with dyspnea and orthopnea, and 34% had paroxysmal nocturnal dyspnea (PND), indicating varying severity of pulmonary congestion. Chest pain was reported in 22% of cases, reflecting underlying ischemic pathology. Peripheral edema and ascites were also documented, consistent with volume overload, a common feature in decompensated heart failure. These findings are comparable to those of **Solela *et al.*** [10] who found dyspnea in 98.7% of AHF patients, orthopnea in 70%, and peripheral edema in 79%.

Comorbid conditions such as diabetes mellitus and hypertension were present in 44% and 41% of patients, respectively, both recognized as major risk factors for cardiovascular disease. Ischemic heart disease (IHD) was identified in 71% of patients, while 20% presented with acute coronary syndrome (ACS), and 18% had previously undergone coronary interventions such as PCI or CABG. These values are slightly higher than those reported by **Zymliński *et al.*** [9], where CAD and MI were present in 53% and 35%, respectively. The high prevalence of advanced NYHA class III and IV symptoms in our cohort reflects the severity of illness at presentation. Similarly, **Solela *et al.*** [10] noted that over 70% of their AHF population presented in NYHA class IV, further emphasizing the advanced functional limitation common in hospitalized AHF patients.

In terms of acute heart failure types, pulmonary edema was the most frequent presentation, affecting 35% of our patients, followed by de novo AHF (18%), cardiogenic shock (16%), and ACS-related AHF (14%). These distributions are similar to those reported by **Lasica *et al.*** [11] who documented pulmonary edema in 36.7% of cases and cardiogenic shock in 11.7%. **Degefu *et al.*** [12] and **Bazmpani *et al.*** [13] also confirmed pulmonary edema as the most prevalent AHF phenotype, followed by cardiogenic shock and ischemia-related presentations. The high rate of pulmonary edema in our study underscores the dominant role of fluid overload and venous hypertension in precipitating acute decompensation in these patients.

Hemodynamically, patients in our study had a wide range of blood pressure values. The mean systolic blood pressure was 119.7 ± 30.0 mmHg, and the diastolic pressure was 75.2 ± 16.48 mmHg, which is consistent with the findings of **Gheorghiade *et al.*** [14] who noted that AHF patients can present with a broad spectrum of blood pressure levels that correlate with disease severity and outcomes. **Kawase *et al.*** [15] also observed that patients with intermediate systolic blood pressure values (comparable to our cohort) had moderate prognostic risk, supporting the clinical relevance of blood pressure variability in AHF.

Hematological analysis in our cohort revealed mild anemia, with a mean hemoglobin of 11.48 ± 2.34 g/dL. This aligns with findings from **Li *et al.*** [16] and **Ye *et al.*** [17] who showed that anemia is common among AHF patients and is associated with worse clinical outcomes, including higher mortality and prolonged hospitalization. Our white blood cell and platelet counts also showed mild elevation and variation, indicating stress or inflammation, which is frequently observed in the acute setting.

The metabolic profile showed that mean cholesterol and LDL levels were within the lower to normal range, potentially reflecting chronic disease, statin therapy, or poor nutritional status. Triglycerides were elevated in some patients, and random blood glucose levels indicated a high prevalence of impaired glucose metabolism. Serum creatinine ranged widely, with a mean of 1.67 ± 2.17 mg/dL, consistent with moderate renal impairment. These values are in agreement with those reported by **Biegus *et al.*** [18] and **Horwich *et al.*** [19] who found similar biochemical profiles among patients hospitalized for AHF.

Liver function abnormalities were highly prevalent in our cohort. Elevated ALT and AST were observed in 85% and 92% of patients, respectively, suggesting significant hepatocellular injury. Hypoalbuminemia was found in 60%, and elevated total bilirubin and INR were seen in 34% and 27% of patients, respectively, indicating impaired hepatic synthesis and excretory function. These rates are higher than those reported by **Samsky *et al.*** [20] who found abnormal ALT in 22%, AST in 30%, and bilirubin in 42%. The greater frequency in our study likely reflects the inclusion of more severely decompensated patients. **Biegus *et al.*** [18] also observed AST and ALT abnormalities in 46% and 31%, respectively, and elevated bilirubin in 33%, all lower than the values observed in our cohort.

Echocardiographic assessment revealed reduced left ventricular ejection fraction (mean EF: 42.07 ± 14.28%) in most patients, with a high prevalence of tricuspid and mitral regurgitation. Tricuspid regurgitation (TR) was present in 65%, and mitral

regurgitation (MR) in 60% of cases. Increasing severity of TR was associated with significantly elevated levels of ALT, AST, ALP, and bilirubin, as well as reduced albumin and increased INR, reflecting the impact of right-sided volume overload on hepatic congestion and dysfunction. These associations are in agreement with findings from **Vyskocilova *et al.*** [21] who documented worsening LFTs with increasing TR severity. Similarly, MR severity correlated with hypoalbuminemia and elevated ALP, as supported by **Alvarez and Mukherjee** [22] who also reported a significant decline in albumin and rise in ALP with MR progression.

Ultrasonographic evaluation of the liver demonstrated hepatic congestion and fatty changes in 70% of cases. This structural hepatic alteration reflects chronic venous congestion and is pathophysiologically consistent with congestive hepatopathy, as described by **Konerman *et al.*** [23]**.** Global hypokinesia and reduced EF were significantly associated with these liver changes, suggesting a link between myocardial dysfunction and hepatic structural remodeling.

Patients presenting with ACS had significantly worse liver profiles, including elevated ALT, AST, ALP, bilirubin, and INR, along with lower albumin levels. This indicates that hepatic dysfunction in ACS-related AHF may stem from both ischemic injury and systemic inflammation. Similar findings were reported by **Vakilian *et al.*** [24] who showed that hepatic impairment in ACS was associated with adverse outcomes and correlated inversely with left ventricular ejection fraction.

Our results also demonstrated that LFTs varied significantly across different AHF types. Patients with cardiogenic shock had the highest levels of ALT, AST, and bilirubin, suggesting severe hypoperfusion-related liver injury. In contrast, de novo AHF had the lowest liver enzyme levels. These findings are consistent with **Vyskocilova *et al.*** [21] who also found significant liver dysfunction among patients with cardiogenic shock. Elevated bilirubin was more common in patients with moderate to severe TR and MR and all cases with elevated bilirubin had reduced EF, reinforcing the link between systolic dysfunction, valvular disease, and cholestatic hepatic injury. These observations were also supported by **Lau *et al.*** [25] and **Styczynski *et al.*** [26] who reported similar associations.

CONCLUSION

This study found liver dysfunction to be common in AHF, particularly in patients with reduced ejection fraction, valvular regurgitation, cardiogenic shock, and acute coronary syndrome. Elevated liver enzymes and bilirubin correlated significantly with tricuspid and mitral regurgitation severity and impaired cardiac function. Although hepatic congestion and fatty changes were frequently detected, they showed no strong association with valvular severity. These findings suggest that hepatic impairment in AHF reflects combined hemodynamic and structural cardiac abnormalities, underscoring the need for integrated cardiac–hepatic evaluation.

Conflict of interest: None.
Financial disclosures: None.

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