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High Burden of Biochemical Liver Function Test Abnormalities and Clinical Implications in Chronic Heart Failure Patients: A Cross-Sectional Study at a Tertiary Hospital in Rwanda

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ABSTRACT

Background: Chronic heart failure (CHF), a significant global health burden, precipitates multi-organ dysfunction, including liver impairment. However, data on biochemical liver function test (LFT) profiles in Africa settings remain limited. This study aimed to determine the prevalence and severity of biochemical LFT abnormalities among Rwandan CHF patients and examine their association with the cardiac function category (New York Heart Association [NYHA] class).

Methods: A hospital-based cross-sectional study was conducted among 65 adults with confirmed CHF at the University Teaching Hospital of Butare (CHUB) from March to May 2025. Consecutive adult patients (≥18 years) with CHF were recruited from both outpatient and inpatient wards. Participants provided written informed consent before commencing study procedures. Sociodemographic and clinical data were collected using a structured questionnaire. Serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), gammaglutamyl transferase (GGT), total bilirubin (TBil) and albumin were analysed using the Architect ci4100 chemistry autoanalyser. Biochemical LFT abnormalities were defined according to manufacturer-specified reference intervals. Associations between LFTs and NYHA class were analysed using the chi-square test.

Results: Liver function test abnormalities were observed in most study participants. The most frequent derangements were elevated ALP (n=54, 83.08%), TBil (n=51, 78.46%), hypoalbuminaemia (n=48, 73.85%), and elevated GGT (n=46, 70.77%). Aminotransferase elevations were less common with ALT abnormal (n=32, 49.23%) and AST (n=24, 36.92%). Significant associations were identified between NYHA class and hypoalbuminaemia (P=.012) as well as elevated GGT (P=.034), with abnormalities becoming more frequent in patients with more severe functional classes.

Conclusion: Biochemical liver dysfunction, predominantly cholestatic abnormalities and hypoalbuminaemia, is common among Rwandan CHF patients and increases with advancing symptom severity. Routine monitoring of liver function tests can aid early detection of decompensation, guide timely nutritional and decongestive interventions, and support individualized management strategies to improve outcomes.

BACKGROUND

Chronic heart failure (CHF) has emerged as a critical global public health challenge, affecting an estimated 64 million individuals worldwide, with its impact particularly severe in low-resource settings.[1] Chronic heart failure (CHF) not only impairs cardiac output but also disrupts liver function through passive hepatic congestion caused by elevated rightsided heart pressures, leading to backward transmission of venous pressure.[2] This backward pressure results in sinusoidal hypertension and hepatocellular injury, collectively known as congestive hepatopathy.[3] Reduced perfusion in advanced CHF may further exacerbate liver dysfunction through ischemic injury. These insults manifest biochemically as distinct patterns of liver function test (LFT) abnormalities, including hypoalbuminaemia (reflecting impaired synthetic function), elevated cholestatic enzymes (indicating biliary stasis), and aminotransferases elevations (marking hepatocellular injury).[4] The severity of these abnormalities often parallels the degree of cardiac dysfunction, suggesting potential utility as accessible biomarkers.

In sub-Saharan Africa (SSA), the growing burden of CHF reflects epidemiological transitions, where non-communicable diseases (NCD) now account for nearly half of adult mortality.[1] Rwanda exemplifies this trend, with cardiovascular diseases (CVD) constituting a leading cause of death driven by a rising prevalence of hypertension (24.5% in adults) and

and frequent late-stage presentations of cardiac disease.[5] Recent studies across diverse populations demonstrate significant variation in the prevalence of LFT abnormalities in CHF patients.[6,7] Research conducted in India reported a 63% prevalence of LFT derangements among patients with heart failure and reduced ejection fraction (HFrEF), compared with 33% among those with preserved ejection fraction.[8] Chinese cohorts reported even higher rates of LFT abnormalities (71.9%), with hypoalbuminaemia (33.4%), hyperbilirubinaemia (32.5%), and alanine aminotransferase (ALT) elevation (30.1%) being the frequent findings.[9] European data similarly confirm the prognostic significance of these LFT abnormalities, with elevated serum gamma-glutamyl transferases (GGT) and bilirubin predicting increased risk of mortality independent of traditional cardiac markers.[9] A study done in Morrocco reported liver dysfunction in 13.7% of the study participants with elevated transaminases associated with increased tricuspid regurgitation, pulmonary hypertension, right ventricular dysfunction, kidney dysfunction, and higher diuretic doses while beta-blocker use was inversely associated. However, the generalisability of these findings to African populations remains uncertain due to differences in CHF aetiologies, comorbidities, and healthcare access patterns.[2]

The Rwandan context presents unique considerations for cardiac hepatopathy. First, the CHF population includes substantial proportions of post-infectious (rheumatic) and hypertensive cardiomyopathies, which may differentially affect hepatic congestion patterns.[10] Secondly, the high background prevalence of potential hepatotoxic exposures, including chronic viral hepatitis (13% HBsAg positivity) and aflatoxin contamination of staple foods - could amplify liver injury in CHF patients.[2] Thirdly, limited access to advanced cardiac imaging (echocardiography available in only 35% of Rwandan hospitals) creates pressing need for alternative, low-cost markers of disease severity.[11] These factors underscore the importance of population-specific data to guide clinical management.

Previous studies on heart failure outcomes in SSA have primarily focused on mortality, length of stay, hospital readmissions rates, electrolyte imbalances, and anaemia,[12] as well as long-term prognosis and comorbidities.[13] However, these studies have not thoroughly explored liver pathology outcomes in CHF patients. Notably, there is a significant gap in data from SSA regarding liver-related outcomes in CHF patients. To our knowledge, this is the first study in Rwanda to systematically evaluate biochemical LFT abnormalities among patients with CHF.

This study investigated the prevalence, severity, and clinical utility of LFT abnormalities among patients with CHF at a tertiary referral centre in Rwanda, focussing on their association with disease severity and potential role in risk stratification. The findings provide systematic characterisation of cardiac hepatopathy in Rwanda, filling an important evidence gap for similar low-resource settings across the region.

METHODS

Study Design and Setting

This hospital-based cross-sectional study was conducted at the Cardiology Department of the University Teaching Hospital of Butare (CHUB), the largest tertiary referral hospital in southern Rwanda, serving a catchment population of approximately 3 million people. The study was conducted between 1 March 2025 and 15 May 2025, and enrolled consecutive adult patients with confirmed CHF attending outpatient clinics or admitted to inpatient wards during the study period. The hospital was chosen as the study site because of its specialised cardiology services and high patient volume, making it an ideal setting to examine the hepatic manifestations of CHF in this population.

Study Population and Sampling

The study population comprised adult patients aged ≥18 years ensuring clinical and physiological homogeneity, as heart failure in children is typically driven by congenital or structural cardiac anomalies that follow different patterns of progression and management compared with adults. The CHF was diagnosed according to the European Society of Cardiology guidelines,[14] incorporating both clinical criteria (e.g., typical symptoms and signs) and objective evidence of cardiac dysfunction from echocardiography or other imaging modalities. Consecutive sampling was employed with all eligible patients meeting the inclusion criteria enrolled during the study period.

Eligibility Criteria

To be eligible for enrolment, participants had to be at least 18 years old, with a confirmed diagnosis of CHF regardless of the underlying cause, willing and able to provide written informed consent. Participants who had a known history of liver disease, such as cirrhosis or chronic viral hepatitis, acute hepatitis or other acute liver conditions were excluded. Patients with incomplete clinical data were excluded, including those lacking echocardiography findings (such as left ventricular ejection fraction or structural abnormalities), essential clinical assessment information (symptoms, physical examination findings, or medical history) or imaging results required to confirm or rule out heart failure as these omissions made verification of the CHF diagnosis impossible.

Sample Size

In this study, a total of 65 patients with chronic heart failure were consecutively recruited during the study period. The sample size represents all eligible individuals who met the inclusion criteria and presented for care within this timeframe, thereby forming a representative cohort for evaluating biochemical liver function abnormalities in this population.

Data collection procedures

Sociodemographic and clinical data

A standardised and structured questionnaire was used to collect data on sociodemographic variables (age, gender, comorbidities, smoking, and alcohol consumption), symptomatology (dyspnoea, chest pain, fatigue, palpitations), and response to physical activity, based on the New York Heart Association (NYHA) functional classification.[15] The questionnaire was translated into Kinyarwanda for ease of administration and participant comprehension.

Blood Sample Collection and Laboratory Analysis

Following consent, 5 ml of venous blood was collected from each participant under aseptic conditions into red-top blood collection tubes. Samples were transported promptly to the hospital laboratory within one hour of collection. After clotting at ambient temperature, the blood samples were centrifuged at 3000 revolution per minute for 3 minutes. Serum aliquots were preserved at –20 °C until analysis.

Liver function tests conducted on serum included alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), total bilirubin, and albumin. These tests were performed using the Abbot Architect ci4100 chemistry autoanalyser (Abbott Laboratories, Abbott Park, Illinois, USA), following standard operating procedures and quality assurance protocols consistent with good clinical laboratory practice. The results were interpreted against the established reference ranges as follows: ALT (10–40 IU/l), AST (10–45 IU/l), ALP (32–80 IU/l), GGT (11–49 IU/l), Total bilirubin (0–17 µmol/l), Albumin (35–50 g/l).[16]

Data Management and Statistical Analysis

Collected data were entered into a Microsoft Excel spreadsheet and subjected to cleaning and verification for logical consistency and completeness. The clean dataset was exported to Stata Version 13 (StataCorp, College Station, Texas, USA) for statistical analysis.[11] Continuous variables were summarised using medians and interquartile ranges (IQRs) due to non-parametric distribution. Categorical data were summarised using frequencies and percentages. The association between liver function test abnormalities and heart failure severity, as classified by NYHA functional class, was examined using the chi-square test or Fisher's exact test where appropriate. Statistical significance was set at $P<.05$.

Ethical Considerations

Ethical clearance was obtained from the Institutional Review Board of the University of Rwanda, College of Medicine and Health Sciences (CMHS/IRB/043/2025), and additional approval was granted by the CHUB Research Ethics Committee (REC/CHUB/009/2025). Written and signed informed consent was obtained from all participants prior to enrolment. The consent form was initially prepared in English and subsequently translated into the local language for participants who could not read or understand English. Participation in the study was entirely voluntary, and individuals were free to withdraw at any time without providing a reason. Confidentiality was ensured through participant anonymisation using unique study identification codes, encrypted data storage, and restricted access to study files. All study procedures were conducted in accordance with the principles of the Declaration of Helsinki.[17]

RESULTS

In this study 65 participants with CHF were enrolled from CHUB. Of these, the majority, 47 (72.3%) were female and 18 (27.7% were male. The overall median (IQR) age of the study participants was 54 (43-64) years with males having a median age of 59.5 (52-74) and females 52 (40-59) years. There was a statistically significant difference in median age according to participant sex ($P=.031$). Most of the participants 55 (84.62%) did not drink alcohol, however, among those who did, significantly more were males 7 (38.9%) compared with females, 3 (6.4%) ($P=.004$). The participants were stratified into three CHF classes II, III, and IV according to NYHA Classification. The socioclinicodemographic characteristics of the study participants are summarised in Table 1.

The LFT profile results are presented in Table 2. Overall, all study participants had at least an abnormality in one of the measured LFT profile parameters. Among the study participants, the highest prevalence of liver function test abnormalities was observed in ALP (n=54, 83.1%), followed by total bilirubin in (n=51, 78.5%), albumin (n=48,73.9%), and GGT (n=46,70.8%). Lower prevalence rates were noted for AST in 24 (36.9%) and ALT in 32 (49.2%) participants. The frequency of biochemical LFT parameter abnormalities was also stratified according to sex and no statistically significant differences were observed by gender ($P>.05$), Table 2.

The prevalence of LFT parameter abnormalities was also stratified according to the NYHA heart failure classes and the results are presented in Table 3. The prevalence of hypoalbuminaemia was statistically significantly different across the NYHA heart failure classes ($P=.012$) being higher in classes III and IV patients compared with class II individuals. Similarly, the frequency of GGT abnormalities was statistically significantly different ($P=.034$) according to the NYHA heart failure classes increasing with severity of HF, from class II (42.9%) to over 70% in classes III and IV. Serum ALT abnormalities also showed a rising trend 3 (21.4% in class II compared with 12 (54.5%) in classes III and IV, although this difference did not reach statistical significance ($P=.061$), Table 3.

Moreover, the findings showed no statistically significant association between lifestyle/clinical factors, duration of heart failure and LFT abnormalities. However, smoking status shows borderline non-statistically significant associations with total bilirubin ($P=.054$) and albumin (.090), which might warrant further investigation in future studies with a larger sample size, Table 4.

TABLE 1: Sociodemographic Characteristics and Clinical Data of Study Participants (N=65)

Variable	Total n (%)	Males n (%)	Females n (%)	*P Value*
Age (years)				
Age, (median, IQR)	54 (43-64)	59.5(52-74)	52 (40-59)	.031
Drinking status				
No	55 (84.62)	11 (61.1)	44 (93.6)	
Yes	10 (15.38)	7 (38.9)	3 (6.4)	.001
Smoking status				
No	64 (98.5)	17 (94.4)	47 (100)	
Yes	1 (1.5)	1 (5.6)	0	.103
Educational status				
No formal education	26 (40.0)	7 (38.9)	19 (40.4)	.731
Primary	23 (35.38)	7 (38.9)	16 (34)	
Secondary	11 (16.92)	4 (22.2)	7 (15.0S)	
University	4 (6.15)	0 (0.00)	4 (8.5)	
Vocational education	1 (1.54)	0 (0.00)	1 (2.1)	
NYHA class of HF				
Class 2	14 (21.54)	2 (11.11)	12 (25.5)	.353
Class 3	29 (44.61)	8 (44.44)	21(44.7)	
Class 4	22 (33.85)	8 (44.44)	14 (29.8)	
Chronic co-morbidities				
No	55 (84.62)	16 (88.9)	39 (83)	.555
Yes	10 (15.38)	2 (11.1)	8 (17)	
Total participants	65	18	47	

Key: IQR, Interquartile range; NYHA, New York Heart Association; HF, Heart failure.
Proportions were compared using a chi-square test, and the medians were compared using a Wilcoxon rank-sum test.
The level of statistical significance was set at $P<.05$.

TABLE 2: Prevalence of Biochemical LFT Abnormalities Stratified by Sex, N=65

LFT Markers	Total n (%)	Sex		*P Value*
		Males n (%)	Females n (%)	
AST (>45U/l)	24 (36.92)	7 (38.9)	17 (36.2)	.839
ALT (>40 U/l)	32 (49.23)	9 (50.0)	23 (48.9)	.939
GGT (>49 U/l)	46 (70.77)	15 (83.3)	31 (66.0)	.168
ALP (>80 U/l)	54 (83.08)	15 (83.3)	39 (83.0)	.973
ALB (<35 g/l)	48 (73.85)	15 (83.3)	33 (70.2)	.281
T. Bili (>17 µmol/l)	51 (78.46)	13 (72.2)	38 (80.9)	.449
Total participants	65	18	47	

Key: LFT, Liver function tests; AST, Aspartate Aminotransferase; ALT, Alanine aminotransferase; GGT, Gamma glutamyl transferase; ALP, Alkaline phosphatase; ALB, Albumin; T. Bili, Total bilirubin.
Proportions were compared using a chi-square test.
The level of significance was set at $P<.05$

TABLE 3: Association Between NYHA Class and Biochemical LFT Abnormalities (N=65)

LFT Markers	Class II n (%)	Class III n (%)	Class IV n (%)	$\chi 2$ (P *Value*)
AST (>45 U/l)	5 (35.7%)	12 (41.4%)	7 (31.8%)	0.5 (.778)
ALT (>40 U/l)	3 (21.4%)	17 (58.6%)	12 (54.5%)	5.6 (.061)
ALP (>80 U/l)	12 (85.7%)	23 (79.3%)	19 (86.4%)	0.53 (.767)
T. Bili (>17 µmol/l)	13 (92.9%)	22 (75.9%)	16 (72.7%)	2.26 (.323)
Alb (<35 g/l)	6 (42.9%)	24 (82.8%)	18 (81.8%)	8.88 (.012)
GGT (>49 U/l)	6 (42.9%)	23 (79.3%)	17 (77.3%)	6.75 (.034)
Number of participants	14	29	22	-

Key: AST, Aspartate Aminotransferase; ALT, Alanine aminotransferase; GGT, Gamma glutamyl transferase; ALP, Alkaline phosphatase; ALB, Albumin; T. Bili, Total bilirubin; LFT, Liver function tests; $\chi 2$, chi-square.
Proportions were compared using the chi-square test. The level of statistical significance was set at $P<.05$. Percentages are based on rows.

TABLE 4: Association Between Lifestyle and Clinical Factors and Liver Function Test Abnormalities (N=65)

LFT Markers	Lifestyle & Cinical Factors								
	Alcohol Use n (%)			Smoking n (%)			Comorbidities n (%)		
	Yes n=10	No n=55	*P Value*	Yes n=1	No n=64	*P Value*	Yes n=10	No n=55	*P Value*
ALT (>40IU/l)	6 (60.0)	26 (47.3)	.459	0 (0.0)	32 (50.0)	.321	4 (40.0)	28 (50.9)	.526
AST (>45IU/L)	4 (40.0)	20 (36.4)	.827	0 (0.0)	24 (37.5)	.441	3 (30.0)	21 (38.2)	.622
ALP (>80IU/l)	8 (80.0)	46 (83.6)	.778	1 (100)	53 (82.8)	.649	7 (70.0)	47 (85.5)	.231
GGT (>49IU/l)	8 (80.0)	38 (69.1)	.485	0 (0.0)	46 (71.9)	.117	8 (80.0)	38 (69.1)	.485
Alb (<35g/l)	8 (80.0)	40 (72.7)	.630	0 (0.0)	48 (75.0)	.090	8 (80.0)	40 (72.7)	.630
T. Bil (>17 µmol/l)	7 (70.0)	44 (80.0)	.479	0 (0.0)	51 (79.7)	.054	8 (80.0)	43 (78.2)	.898

Key: P, *P* Value; AST, Aspartate Aminotransferase; ALT, Alanine aminotransferase; GGT, Gamma glutamyl transferase; ALP, Alkaline phosphatase; ALB, Albumin; T. Bili, Total bilirubin; LFT: Liver function tests.
Proportions were compared using the chi-square test at a level of significance set at $P<.05$

DISCUSSION

This study aimed to investigate the prevalence, severity, and clinical utility of LFT abnormalities among patients with CHF at a tertiary referral hospital in Rwanda. In line with this objective, our findings demonstrate a high burden of biochemical LFT abnormalities in this population, with the highest prevalence observed for serum ALP, total bilirubin, GGT, and albumin levels. Notably, hypoalbuminaemia and GGT elevations were statistically significantly associated with worsening NYHA class, suggesting that hepatic dysfunction progresses in parallel with heart failure severity. These finding are consistent with the concept of cardiac hepatopathy, where venous congestion and reduced hepatic perfusion contribute to both synthetic and cholestatic liver dysfunction.[18]

Notably, ALP was elevated in 83.1% of the study participants, making it the most frequent abnormal marker. This aligns with previous research identifying ALP as a sensitive marker for cholestasis, which commonly arises in right-sided heart failure due to hepatic venous congestion that impairs bile excretion.[19] Studies emphasize ALP's diagnostic utility in detecting canalicular dysfunction during liver congestion,[20,21] corroborating the elevated frequency observed in this cohort.[22] Similarly, total bilirubin was abnormally high in 78.5% of cases, which is consistent with literature describing bilirubin elevation as a consequence of impaired hepatic clearance caused by reduced hepatic perfusion and backflow pressure.[23] This observation is supported by illustrating how decreased hepatic blood flow in CHF leads to bilirubin accumulation,[24,25] reinforcing its role as a biochemical indicator of hepatic stress in heart failure.[1]

In addition, serum albumin, a marker of hepatic synthetic capacity and nutritional status, was decreased among 73.9% of the patients. Decreased serum albumin is not only a marker of chronic liver dysfunction and malnutrition but also a strong prognostic indicator in CHF patients, due to its association with systemic inflammation and disease severity. In addition, the fluid

retention associated with CHF may also cause artifactual hypoalbuminaemia.[26] Furthermore, GGT, which was elevated in 70.8% of participants, also reflects hepatic congestion. GGT is a cholestatic enzyme that becomes elevated in response to biliary stasis and oxidative stress, frequently present in CHF-related liver injury.[27]

In contrast, ALT and AST activities were less frequently elevated in this cohort, at 49.2% and 36.9%, respectively.[23] These aminotransferases are primarily indicators of hepatocellular injury, but the relatively lower frequency of abnormal results suggest that ischemic hepatitis or acute liver injury might be less common in this CHF population.[28] Serum ALT, being more liver-specific, had a higher prevalence of abnormality than AST, which is also found in other tissues like skeletal muscle and myocardium, reducing its specificity for liver damage in CHF patients. As previously noted, such aminotransferase elevation is more typical in acute decompensated heart failure or when liver perfusion is critically compromised.[29]

Among the six biochemical LFT biomarkers analysed, only albumin and GGT demonstrated statistically significant associations with NYHA class, suggesting that worsening heart failure is closely linked with deteriorating hepatic function.[8] The rising prevalence of hypoalbuminaemia in NYHA classes III and IV reflects impaired hepatic protein synthesis or ongoing systemic inflammation and malnutrition in more advanced CHF a finding strongly supported by other studies that recognise hypoalbuminemia as a powerful prognostic marker in heart failure.[30,31] Additionally, recent insights into GGT as a congestion biomarker are reinforced by emerging evidence,[2] while findings on LFT trajectories and congestion patterns[27] further confirm the clinical relevance of these abnormalities. Finally, the broader pathophysiological context of cardiohepatic congestion in CHF is extensively detailed in comprehensive reviews.[32]

The observed pattern of hepatic biomarker abnormalities has important implications for the management and prognosis of CHF. Hypoalbuminaemia, in particular, reflects impaired hepatic synthetic function and systemic inflammation, serving as a powerful prognostic indicator that may prompt closer follow-up, nutritional optimization, and intensified management.[30] The strong associations between hypoalbuminaemia, elevated GGT, and worsening NYHA class suggest that routine assessment of these markers may assist in identifying patients at higher risk of decompensation.[30] Serum GGT elevation, a sensitive indicator of liver congestion and biliary stasis, was increasingly evident in patients with more severe CHF, consistent with other findings that emphasized its role in detecting hepatic stress due to venous congestion.[2,33] Collectively, these findings highlight that integrating liver function markers into routine assessment may enhance early risk detection and inform personalized therapeutic strategies aimed at improving outcomes in CHF.[27,32] Serum ALT abnormality, while not statistically significant, demonstrated a marginal association (P=.061), suggesting a potential trend where hepatocellular injury may increase with CHF severity, but further research using larger sample sizes may be warranted to clarify this relationship. On the other hand, AST (P=.778), ALP (P=.767), and total bilirubin (P=.323) showed no significant associations with NYHA class, indicating that these markers, while often abnormal in CHF patients, may not be sensitive indicators of disease progression in this study population.[33]

These findings are consistent with prior literature suggesting that AST and ALP elevations are less specific to CHF-related hepatic injury, and that total bilirubin, although frequently elevated in CHF, can be influenced by other non-cardiac factors such as *in-vivo* hemolysis or intrinsic liver disease.[33] The study also examined potential correlates of liver function test abnormalities in patients with CHF, focusing on their relationship with lifestyle/clinical factors, as measured by the NYHA classification. However, no statistically significant associations were observed between these clinical variables and the presence of liver function test abnormalities. This lack of correlation suggests that hepatic biomarker derangements in CHF may be influenced more by pathophysiological mechanisms such as venous congestion and impaired hepatic perfusion than by the duration of disease or functional class alone. These findings underscore the complexity of hepatic involvement in CHF and highlight the need for larger studies to clarify the interplay between clinical severity and biochemical abnormalities.

Our findings contrast somewhat with earlier research indicating that alcohol consumption and smoking may exacerbate hepatic dysfunction in general population due to their hepatotoxic effects and their contribution to oxidative stress and inflammation.[11] However, the absence of significant association in our study may reflect the low proportion of active drinkers (15.38%) and smokers (1.5%) among our CHF cohort, possibly underpowering the statistical tests to detect true differences.

Similarly, no significant association was observed between the presence of comorbidities (e.g., diabetes, hypertension) and LFT abnormalities, which diverge from prior studies that have shown additive hepatic stress in patients with multimorbidity.[34] One possible explanation may be the limited sample size of patients with documented comorbidities (n=10), which restricted subgroup analyses and reduced statistical power. Importantly, we observed no significant correlation between the clinical/lifestyle factors and abnormalities in any LFT marker, including albumin, transaminases, GGT, or bilirubin. This aligns with findings from other studies which reported that liver dysfunction in CHF is more strongly related to the haemodynamic severity (e.g., congestion, low perfusion) than to how long the patient has been diagnosed with heart failure.[27,32,34]

The exceptionally high prevalence of cholestatic abnormalities, particularly elevated ALP and total bilirubin, may reflect population specific factors, including the predominance of hypertensive and post infectious cardiomyopathies, limited access to advanced cardiac diagnostic tools, and background exposures such as aflatoxin contamination and chronic hepatitis B. These contextual influences give the pattern of LFT abnormalities distinctive clinical relevance and make the findings particularly informative for the management of CHF in Rwanda and other similar low resource settings.

Study Strengths and Limitations

A key strength of this work is that it provides important

evidence on liver dysfunction in CHF patients in Rwanda, offering populationspecific insights shaped by local cardiovascular disease patterns and hepatic risk exposures. This study has certain limitations. Its single-centre scope may restrict the generalisability of the findings to other populations or healthcare settings. In addition, the modest sample size may not have been sufficient to detect all potential differences or associations, and the cross-sectional design limits the ability to establish causality or temporal relationships between variables. Nevertheless, the results remain relevant as they provide valuable preliminary insights into the burden and pattern of liver function test abnormalities among patients with CHF in this context. By highlighting associations with disease severity and identifying markers of hepatic involvement, the study contributes important baseline evidence that can inform clinical practice locally and guide the design of larger, multicentre investigations aimed at strengthening external validity and exploring causal pathways.

CONCLUSION AND RECOMMENDATION

This study demonstrates a markedly high prevalence of LFT abnormalities among Rwandan patients with CHF, with cholestatic markers and hypoalbuminaemia being the most common. Albumin and GGT were significantly associated with advanced NYHA class, underscoring their value in identifying worsening disease. These findings highlight the clinical importance of routine LFT assessment in CHF management and contribute novel evidence from an understudied African population. Routine monitoring of liver function should be incorporated into CHF management, with priority given to albumin and GGT, as these biomarkers most reliably reflect worsening cardiac congestion and declining hepatic function. Serum ALP and total bilirubin should also be assessed to detect cholestatic abnormalities, while ALT and AST may be used selectively to evaluate potential hepatocellular injury. Integrating these tests into standard CHF follow-up can support early identification of deterioration and guide timely clinical intervention. Future prospective, multi-centre studies with larger cohorts are warranted to validate these findings and to further investigate their prognostic utility in guiding therapeutic interventions in CHF.

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