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RESEARCH ARTICLE

CLINICAL PATTERNS AND OUTCOMES IN CIRRHOTIC YEMENI PATIENTS WITH ACUTE-ON-CHRONIC LIVER FAILURE

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Abstract

Acute-on-chronic liver failure (ACLF) is a clinical condition of abrupt hepatic decompensation in chronic liver disease patients that is associated with extra-hepatic organ failures and a higher mortality rate. This study aimed to identify the clinical patterns and outcomes in cirrhotic Yemeni patients with ACLF. This prospective cohort study was carried out on 160 cirrhotic patients admitted between May 2023 and May 2024 to the Internal Medicine Department in Al-Sadaqah General Teaching Hospital in Aden governorate, Yemen. Based on the European Association for Study of Liver's Chronic Liver Failure Consortium EASL-CLIF criteria, patients were divided into Group A: 54 patients with ACLF. Group B: 106 patients without ACLF. The prevalence of ACLF is 33.8%. The mean age was 41.54± 8.11 years in ACLF and 39.22± 8.14 years in non-ACLF patients. The proportion of males in ACLF was 51.9% vs. 57.5% in non-ACLF patients. The most common causes of cirrhosis were cryptogenic (58.8%) and autoimmune hepatitis (25.6%). A significant correlation was found between prior hospitalization, decompensation, and the increased risk of developing ACLF. The most common complications were jaundice, hepatic encephalopathy, renal failure, signs of bacterial infection, ascites, and gastrointestinal bleeding, respectively. The main precipitating events for ACLF were bacterial infection, especially SBP (48.1%), GIT bleeding (24.1%), and no identified precipitating events (24.1%). The most common organ failures were renal (70.4%), liver (42.6%), and cerebral (31.5%). ACLF patients showed higher levels of WBCs, bilirubin, and serum creatinine. Mortality rates were significantly higher in ACLF patients compared to non-ACLF at both 28 days (33.3% vs. 5.7%) and 90 days (685% vs. 23.6%). This study demonstrated that ACLF patients have worse prognoses, higher mortality rates, increased ICU admission, lower survival, and higher rates of organ failure than non-ACLF patients.

Keywords: Acute-on-chronic liver failure, Acute Decompensation, Cirrhosis.

1. Introduction

Acute-on-chronic liver failure (ACLF) is a clinical condition of abrupt hepatic decompensation that is associated with one or more extrahepatic organ failures and higher mortality rates in patients with or without pre-existing chronic liver disease (CLD) [1]. ACLF frequently results in death within a short period in the absence of particular supportive treatment [2,3].

The European Association for Study of Liver's Chronic Liver Failure Consortium (EASL-CLIF) proposes a formal definition and grading system for ACLF, characterized by acute decompensation (ascites, hepatic encephalopathy, GIT bleeding, or bacterial infection), organ failure, and a high 28-day mortality rate, despite fluctuating global definitions [4].

ACLF, a rising global public health issue, affects 20–35% of cirrhosis patients, with mortality rates ranging from 30–50% at 28–90 days, correlated with organ failure rates [1]. ACLF etiologies vary across geographical areas and populations, with HBV infection being the most prevalent in Asia and alcoholism being the most prevalent in Western nations. ACLF development is influenced by various factors such as infections, alcohol consumption, GI bleeding, and drugs, with 50% of the triggers being unknown [5,6,7].

The pathogenesis of ACLF is a complex disease characterized by chronic inflammation, cell death, and

immune response activation triggered by bacterial infections or gut translocations, leading to tissue damage and inflammation [8,9].

ACLF is a complex condition with various clinical symptoms, including HE, ascites, coagulopathy, bacterial infections, and jaundice, with organ failures affecting the kidney, brain, coagulation, circulation, and lungs [1,10,11].

The EASL-CLIF criteria diagnose ACLF using a CLIF-C OF score based on organ dysfunctions. High 28–90-day mortality rates are associated with specific organ dysfunctions, and severity is graded from 0 to 3 [8,12,13].

Researchers developed a CLIF-C ACLF score to predict prognosis and mortality in patients with ACLF, combining the CLIF-C OF score, age, and WBC. A score above 64 has no survival chance, regardless of the precipitating cause [10, 12]. The CLIF-C AD score was developed and validated for patients with AD without ACLF [14].

The management of ACLF is challenging and involves supportive care, early identification and treatment of precipitating factors, and LT [2]. The outcome of ACLF is poor, with a high mortality rate even with appropriate management [4].

Studies in Western and Asian nations show ACLF trends, but Yemeni patients' characteristics remain unexplored. Limited data exists on ACLF epidemiology, pathophysiology, and management in Yemen, a country with a high CLD burden. There's no standardized protocol for diagnosis and treatment, and hospital availability is variable.

The main aim of the study is to evaluate the clinical patterns and outcomes of cirrhotic Yemeni patients with ACLF admitted to the Internal Medicine department in Al-Sadaqa General Teaching Hospital in Aden between May 2023 and May 2024.

2. Methodology

2.1. Study setting

This was a prospective cohort study. Carried out at the internal medicine department in Al-Sadaqa General Teaching Hospital in Aden from May 2023 to May 2024.

2.2. Study Population

A confirmed cirrhotic Yemeni patient was treated for complications while staying in the hospital for more than one day. Cirrhosis is diagnosed by history, a full clinical physical examination, previous laboratory investigation, and abdominal imaging.

2.3. Inclusion Criteria

Patients with AD cirrhotic with ACLF are defined as having the presence of HE, ascites, GIT bleeding, or bacterial infection, according to the criteria of the EASL-CLIF-C OFs scoring system [12].

2.4. Exclusion Criteria

Age ≤ 18 years; patients with HCC; extrahepatic malignancy; fulminant ALF; HIV/AIDS; pregnant mothers. Patients with incomplete medical records or who refused to participate.

2.5. Sample Size

After obtaining consent, all patients diagnosed with ACLF who visited the hospital during the study period were enrolled. (One hundred sixty participants signed up and were counted in the sample population. 54 had ACLF, were 106 had no ACLF).

2.6. Data Collection

Upon admission, eligible patients will be approached for informed consent to participate in the study. Data were obtained from each participant through direct patient interviews and medical record reviews: upon admission, during hospitalization, and follow-up (28-day and 90-day).

2.6.1. Demographic data includes age, sex, place of residence, habits, etc.

2.6.2. Medical history, etiology of cirrhosis, previous episode of acute decompensation, and hospitalization.

2.6.3. Clinical profile: (clinical features, causes of admission, precipitating factors, complications), thorough, comprehensive full history taking, physical examination, and laboratory investigation, CBC, liver function tests, renal function tests, coagulation profile, blood serum electrolytes (Na, K), and CRP; when needed: evaluation of ascitic fluid; urine analysis: arterial blood gases; viral markers (HAV Ig M antibody and HEV Ig M antibody for patients with a three-fold increase in liver enzymes); quantitative PCR for HBV, HCV, HBsAg, and anti-HBc IgG; and abdominal ultrasonography and chest X-rays for all patients.

2.6.4. Scoring systems, at 2-3 days after admission assessment, calculate scores for grading patients using the EASL score (CLIF-C OFs, CLIF-C ACLF, and CLIF-C AD score).[4, 12, 14]

2.7. Outcomes

Recording hospitalization duration, survival status at 28 and 90 days.

2.8. Follow-up

All patients were followed up for three months from inclusion or until mortality, whichever came first. Information on mortality at 28–90 days following

enrollment was recorded for all enrolled patients by mobile phone.

2.9. Statistical analysis

Statistical analysis data were analyzed using the software tool SPSS version 23. Descriptive statistics were employed to summarize demographic and clinical characteristics. A comparative analysis of the ACLF and non-ACLF groups was conducted using the Chi-square test for categorical variables. The Mann-Whitney test is used for continuous variables, depending on the normality of the data distribution. A value of less than 0.05 was considered statistically significant.

2.10. Ethical considerations

The study will be conducted after getting approval for the protocol from the ethics committee of the faculty of medicine at Aden University, the internal medicine department, and patients. All participants signed written releases after receiving full disclosure.

3. Results

3.1. Participant characteristics

This prospective observational study evaluated 160 cirrhotic patients with acute decompensation, split between 54 patients with ACLF and 106 patients without ACLF. The study was conducted at the internal medicine department of Al-Sadaqa General Teaching Hospital in Aden from May 2023 to May 2024.

Table 1. The baseline sociodemographic characteristics of the studied patients

Findings	AD with ACLF (n = 54)		AD without ACLF (n = 106)		P-value
	n	%	n	%	
Sex: Male	28	51.9	61	57.5	0.493
Female	26	48.1	45	42.5	
Age ± SD	41.54 ± 8.11		39.22 ± 8.14		0.098
Khat chewing	36	66.7	70	66.0	0.937
Smoking	13	24.1	34	32.1	0.293
Alcohol	5	9.3	13	12.3	0.569
Etiology of cirrhosis					
Cryptogenic	28	51.9	66	62.3	0.206
autoimmune hepatitis	13	24.1	28	26.4	0.748
Schistosomiasis	7	13.0	7	6.6	0.178
HCV	4	7.4	3	2.8	0.227
Alcohol	3	5.6	2	1.9	0.604
HBV	2	3.7	2	1.9	0.604
NAFLD	1	1.9	0	0.0	0.337
Other	2	3.7	0	0.0	0.113
Previous hospitalization	39	72.2	45	42.5	**<0.001**
Previous decompensation	39	72.2	45	42.5	**<0.001**

P value< 0.05 is significant; P value< 0.01 is highly significant; SD: standard deviation; AIH: autoimmune hepatitis; HCV: hepatitis C virus; HBV: hepatitis B virus; NAFLD: non-alcoholic fatty liver disease.

The mean age was 41.54 ±8.11 years in ACLF vs. 39.22 ±8.14 years in non-ACLF patients. The proportion of males in ACLF was 51.9% **vs.** 57.5% in non-ACLF patients. Regarding habits, ACLF **vs.** non-ACLF (66.7% **vs.** 66%) of patients had the habit of Khat chewing, (24.1% **vs.** 32.1%) of them were smokers, and (9.3% **vs.** 13.3%) were alcoholics. No statistically significant difference was found regarding sex, age, or habit (p-value > 0.05).

The most common cause of cirrhosis was cryptogenic in both ACLF and non-ACLF patients (51.9% **vs.** 62.3%), followed by AIH (24.1% vs. 26.4%), and schistosomiasis (13.0% **vs.** 6.6%). There was no statistically significant difference in the cirrhosis etiology. Patients with ACLF had a significantly higher rate of previous hospitalization and decompensation compared to non-ACLF (72.2% vs. 42.5%, p-value < 0.001).

Table 2. Prevalence of ACLF grade at enrollment for studied patients

ACLF grade	ALL Patients (n= 160)	
	n	%
ACLF 0	106	66.2
ACLF Total	54	33.8
ACLF 1	29	18.1
ACLF 2	15	9.4
ACLF 3	10	6.3

In the current study, the prevalence of ACLF at admission is 33.8%. According to the EASL definition of ACLF grade, 29 (18.1%) of patients were in ACLF 1, while 15 (9.4%) were in ACLF 2, and ACLF 3 was seen in 10 (6.3%) patients**.**

3.2. Clinical patterns and manifestations of the studied patients.

The clinical manifestations and precipitating events observed in the study participants are presented in Table 3.

3.2.1. Clinical manifestations and complications of ACLF vs. non-ACLF patients

Upon admission, patients with ACLF, compared to non-ACLF, presented with more severe clinical features, including a higher likelihood of ICU admission (57.4% vs. 13.2%, p-value <0.001), jaundice (94.4% vs. 79.2%, p-value =0.012), HE (87.0% vs. 58.5%, p-value <0.001), renal failure (70.4% vs. 0%, p-value <0.001), signs of bacterial infection (48.1% vs. 19.8%, p-value <0.001), ascites (37.0% vs. 30.2%, p-value =0.011), and GIT bleeding (24.1% vs. 15.1%, p-value =0.163), respectively. These findings suggest a higher disease burden and multi-organ involvement in patients with ACLF at the time of admission.

Vital signs showed no significant difference, except for axillary temperature, which was significantly higher in the ACLF group (p = 0.001), SpO^2/FiO^2, and urine volume, which showed a significant decline in ACLF

compared to without ACLF patients (p-value <0.001 and p-value <0.001, respectively). The use of vasopressors and supplemental oxygen was significantly higher in ACLF patients than non-ACLF patients (p-value <0.001). Table 3.

Table 3. Clinical Features at Admission among the Studied Patients

Findings	AD with ACLF (n= 54)		AD without ACLF (n= 106)		P-value
	n	%	n	%	
ICU admission	31	57.4	14	13.2	**<0.001**
Jaundice	51	94.4	84	79.2	**0.012**
HE	47	87.0	62	58.5	**<0.001**
Renal failure	38	70.4	0	0.0	**<0.001**
Ascites	20	37.0	32	30.2	**0.011**
Bacterial Infection:	26	48.1	21	19.8	**<0.001**
SBP	14	25.9	5	4.7	**<0.001**
Sepsis	10	18.5	0	0.0	**<0.001**
UTI	8	14.8	12	11.3	0.527
Pneumonia	5	9.3	3	2.8	0.078
GIT bleeding	13	24.1	15	14.2	0.118
MAP	67 ±11		67 ±12		0.816
HR (beats/min)	86 ±14		88 ± 13		0.223
Axillary Temperature	37 ± 0.78		36 ± 0.82		**0.001**
SpO^2	85 ± 12		95 ± 2		**<0.001**
SpO^2 /FiO^2	397 ± 73		454 ± 26		**<0.001**
Urine output: mL/day	989 ± 275		1268±270		**<0.001**
Use vasopressors	13	24.1	0	0.0	**<0.001**
Supplemental O^2	9	16.7	1	0.9	**<0.001**
Precipitating events					
Bacterial Infection	26	48.1	21	19.8	**<0.001**
GIT bleeding	13	24.1	16	15.1	0.163
No precipitating identified*	13	24.1	55	51.9	**0.001**
>2 precipitating events**	12	22.2	10	9.4	0.026
Types of organ failure					
Renal	38	70.4	0	0.00	**<0.001**
Liver	23	42.6	11	10.4	**<0.001**
Cerebral	17	31.5	6	5.6	**<0.001**
Circulatory	13	24.1	3	2.8	**<0.001**
Coagulation	10	18.5	3	2.8	0.002
Respiratory	6	11.1	0	0.0	0.002

P value< 0.05 is significant, and P value< 0.01 is highly significant. HE: hepatic encephalopathy; SBP: spontaneous bacterial peritonitis; UTI: urinary tract infection; GIT bleeding: gastrointestinal bleeding; *MAP: main arterial blood pressure. SpO^2/FiO^2: The ratio of oxygen saturation to fractional inspired oxygen*.

*No precipitating event denotes the absence of bacterial infection, GIT bleeding, or other precipitating event.

** More than one precipitating event denotes the presence of at least two of the following: bacterial infection, GIT bleeding, or another precipitating event.

3.2.2. Precipitating events for hospital admission of ACLF vs. non-ACLF patients

The most common precipitating etiology in ACLF patients **vs.** non-ACLF is bacterial infection, especially SBP (48.1% **vs.** 19.8%), GIT bleeding (24.1% **vs.** 15.1%), and no identified precipitating event (24.1% vs. 51.9%), as shown in Table 3.

3.2.3. Distribution of Organ Failures at Enrollment of ACLF vs. non-ACLF patients

The most common organ failures in ACLF patients at admission are renal failure (70.4%), followed by liver (42.6%), cerebral (31.5%), circulatory (24.1%), coagulation (18.5%), and respiratory (11.1%). Organ dysfunction in AD without ACLF (10.4%) included liver failure, cerebral (5.6%), coagulation (2.8%), and circulatory (2.8%). Table 3.

3.2.4. Laboratory investigation

Table 4. Laboratory data at hospital admission of studied patients

Parameter	AD with ACLF (n = 54)	AD without ACLF (n = 106)	P-value
	(m ± SD)	(m ± SD)	
Hb (g/dl)	9.3 ± 1.4	10.4 ± 1.3	**<0.001**
WBCs ($\times10^3/mm^3$)	11.3 ± 3.7	8.5 ± 3.0	**<0.001**
NLR	5.5 ± 4.4	2.9 ± 1.5	**<0.001**
Platelets($\times10^3/mm^3$)	118 ± 42	155 ± 68	**<0.001**
T. bilirubin (mg/dl)	10 ± 6.0	6 ± 3.8	**<0.001**
Albumin (g/L)	3.11 ± .57	3.36 ± .35	**0.002**
AST (U/L)	183 ± 213	196 ± 261	0.067
ALT (U/L)	215 ± 286	232 ± 313	0.359
ALP (U/L)	315 ± 187	265 ±180	**0.013**
GGT(U/L)	349 ± 177	329 ± 180	0.224
INR	2.2 ± .7	1.7 ± 1.9	**<0.001**
PTT	47 ± 6.5	43 ± 7.4	**0.007**
Creatinine (mg/dl)	2.4 ± 0.72	1.1 ± 0.27	**<0.001**
Urea (mg/dl)	74 ± 24	51 ± 16	**<0.001**
Na^+ (meq/L)	128 ± 6	13 ± 5.88	**<0.001**
K^+ (meq/L)	3.9 ± .59	3.97 ± .64	**0.014**
Ca^{+2} mmol/L	1.84 ± .15	1.92 ± 16	**0.003**
CRP (mg/L)	67 ± 21	36 ± 10	**<0.001**

The P value< 0.05 is significant, and the P value< 0.01 is highly significant. SD: standard deviation; Hb: hemoglobin; WBC: white blood cell, NLR: neutrophil-lymphocyte ratio; T. bilirubin: total. Bilirubin; ALT: alanine aminotransferase; AST: aspartate aminotransferase; ALP: alkaline phosphatase; GGT: gamma-glutamyl transferase; International Normalized Ratio; PPT: Partial Thromboplastin Time. Na+: sodium, K+: potassium, Ca+: calcium, and CRP: C-reactive protein.

In comparison, laboratory investigations such as CBC, LFT between the two groups, hemoglobin, platelets, and serum albumin showed a significant decline in ACLF compared to non-ACLF patients, while WBCs, NLR, bilirubin, ALP, INR, and PTT showed significant elevation in ACLF compared to non-ACLF patients. No significant difference between them was found as regards AST, ALT, and GGT (p-value >0.05). Regarding serum creatinine, urea, and CRP, there was a significant elevation in ACLF compared to non-ACLF patients, while serum electrolytes such as Na, K, and Ca, showed

a significant decline in ACLF compared to non-ACLF patients (Table 4).

3.2.5. Prognostic scores of ACLF vs. non-ACLF patients.

Regarding prognostic scores, at enrolment**,** 28 and 90 days after admission, CLIF-OF, CLIF-C-ACLF, and CLIF-C AD scores were significantly higher in ACLF compared to non-ACLF patients.

Table 5. Comparison of prognostic scores between studied patients

Scores	AD with ACLF (n = 54)	AD without ACLF (n = 106)	P-value
	(m ± SD)	(m ± SD)	
At enrolment			
CLIF-OF	11.13 ± 2.82	8.17 ± 1.22	**<0.001**
CLIF-C ACLF	48.44 ± 10.71	34.92 ± 6.57	**<0.001**
CLIF-C AD	66.59 ± 10.10	48.91 ± 10.09	**<0.001**
At 28 days			
CLIF-OF	9.13 ± 2.82	6.17 ± 1.22	**<0.001**
CLIF-C ACLF	46.44 ± 10.71	32.92 ± 6.57	**<0.001**
CLIF-C AD	64.59 ± 10.10	46.91 ± 10.09	**<0.001**
At 90 days			
CLIF-OF	10.94 ± 2.70	8.44 ± 1.77	**<0.001**
CLIF-C ACLF	47.50 ± 11.6	35.83 ± 8.15	**<0.001**
CLIF-C AD	64.81 ± 10.8	49.03 ± 13.10	**<0.001**

P value< 0.01 is highly significant. CLIF-C ACLF: chronic liver failure c acute-on-chronic liver failure; CLIF-C AD: chronic liver failure c acute decompensated; CLIF-C OF: chronic liver failure c organ failure score.

3.2.6. Outcome at 28 and 90 days for ACLF vs. non-ACLF patients

Table 6. Outcome at 28 and 90 days among Studied Patients

Finding		AD with ACLF (n = 54)		AD without ACLF (n = 106)		P-value
		n	%	n	%	
Outcome at 28 days						
Mortality rate = death%		18	33.3	6	5.7	**<0.001**
Readmission		35	64.8	32	30.2	**<0.001**
ICU admission		24	44.4	21	19.8	**<0.001**
Outcome at 90 days						
Mortality rate = death%		37	68.5	25	23.6	**<0.001**
Readmission		32	59.2	30	28.3	**<0.001**
ICU admission		25	46.2	8	8.0	**<0.001**
Duration of hospital stay (days)	Mean ± SD	11.57 ± 2.81		10.88 ±2.61		**<0.003**
	Median	12.0 (10.0-14.0)		11.0 (9.0-12.0)		
	Range	6.0-18.0		6.0-18.0		

P value< 0.05 is significant, P value< 0.01 is highly significant, SD: standard deviation. ICU; intensive care unit.

The mortality rate was significantly higher in ACLF vs. non-ACLF patients at 28 days (33.3% **vs.** 5.7%, p-value <0.001) and at 90 days (68.5% **vs.** 23.6%, p-value <0.001). Also, regarding readmission, ICU admission, and duration of hospital stay (11.57 ± 2.81 vs. 10.88 ± 2.61), it was significantly higher in ACLF patients than non-ACLF patients (p-value <0.003).

4. Discussion

Patients with AD of cirrhosis who develop one or more OFs, significant short-term mortality (> 15%), and severe systemic inflammation are considered to be in ACLF. AD is the primary cause of hospitalization for cirrhotic patients and is defined by the recent onset of GIT bleeding, bacterial infection, ascites, HE, or any combination of these conditions [3].

Acute events might be extrahepatic (like bacterial infections or GIT bleeding) or intrahepatic (like reactivation of HBV or alcohol consumption) and cause ACLF in many people. However, in as many as 40% of patients with ACLF, no precipitating cause is found [4].

Until now, no universal definition of ACLF has been established. As a result, the majority of the data regarding ACLF came from research that relied on the EASL-CLIF consortium definition, which was the most explored term [15].

The epidemiological characteristics of ACLF were recently studied through a meta-analysis involving 43,206 patients who were drawn from 30 studies using the EASL-CLIF ACLF criteria. The results indicated that the prevalence of ACLF worldwide was 35% and the 90-day mortality rate was 58%, with regional variations. Although there have been reports of geographical differences in alcohol consumption rates, alcohol consumption is the most commonly known cause of liver disease. GIT bleeding and infection were the most common causes (35% and 22%, respectively). Renal failure accounted for 49% of recorded OFs, with respiratory failure being the least common (11%) [5].

While ACLF can improve or perhaps go away entirely in up to half of cases, in the other half, disease progression may lead to a potentially fatal illness [16]. Because of this high death rate, it is imperative to find early predictors of short-term mortality in ACLF to identify at-risk patients who may need immediate LT, targeted treatments, or intermediate care [17].

Though the diagnosis of ACLF as a separate syndrome is growing, most studies have focused on American, European, and Asian cohorts, where chronic HBV pathogenesis and alcoholism are the most common causes of liver damage. In the Middle East, where HCV is the primary cause of CLD, particularly in Yemen, no research has examined the patterns of ACLF [17,18].

The main aim of this study was to evaluate the clinical patterns and outcomes of cirrhotic Yemeni patients with ACLF admitted to Al-Sadaqa General Teaching Hospital, Aden. Using the EASL-CLIF definition, we present the first study to characterize ACLF in Yemen.

4.1. Demographic

The current study regarding demographic data showed that the mean age for ACLF and non-ACLF patients was

41.54 ± 8.11 **vs.** 39.22 ± 8.14 years. The percentage of males in ACLF and non-ACLF patients was 51.9%, compared to 57.5%. No statistically significant differences were observed between the two groups regarding age and sex (p-value > 0.05), consistent with previous research conducted [4,19–24], except for the age ACLF and non-ACLF patients, which was 56 ±11 **vs.** 58 ±12, where the Yemeni cirrhotic patients with ACLF were significantly younger compared to these study patients. The disagreement with the current study may be due to the difference in the definition of ACLF, sample size, and prevalence of ACLF, precipitating events for ACLF, and the difference in etiological causes of cirrhosis and available medical facilities for the management of liver disease, which are not found in Yemen.

Regarding habits among studied patients, it was revealed that in ACLF **vs.** non-ACLF (66.7% **vs.** 66%) of patients had the habit of Kat chewing, (24.1% **vs.** 32.1%) of them were smokers, and (9.3% **vs.** 13.3%) were alcoholics. No statistically significant difference was found between the two groups regarding Khat chewing, smoking, and alcohol (p-value > 0.05).

Regarding Kat chewing, no studies assessed the relationship between Khat chewing and ACLF, but according to Terefe et al. [25], there was a significant association between mortality and Khat chewing among patients with CLD. Regarding alcoholic intake, there was no significant association between ACLF and alcohol intake, consistent with [4, 22, 26].

4.2. Etiology of the underlying cirrhosis

The most common cause of cirrhosis in our patients was cryptogenic (undiagnosable) in both ACLF and non-ACLF patients (51.9% **vs.** 62.3%). followed by AIH (24.1% vs. 26.4%) and schistosomiasis (13.0% **vs.** 6.6%). There was no statistically significant difference in the cirrhosis etiology.

Which aligns with a previous study that showed that the major etiology of the underlying cirrhosis varies according to geographical location [4,19,21–23]. In Europeans, alcohol, followed by HCV, is the most common etiology of cirrhosis [4,19]**.** However, the Indian study showed that alcohol, followed by HBV and cryptogenic [22,23], in Egyptian patients revealed that the major etiology of cirrhosis was HCV [20,24]. The disagreement with the current study type of etiology may be due to the difference in the prevalence of underlying liver disease, geographical and ethnic variation, and habitual characteristics of the population. Also, there are limitations to diagnostic capabilities and practices such as Fibroscan and liver biopsy.

Regarding the history of previous hospitalization and decompensation, the current study showed that patients with ACLF had a significantly higher rate of previous hospitalization and decompensation compared to non-ACLF (72.2% vs. 42.5%, p-value < 0.001). Which aligns with [19,21,22,27]**.** Showed that previous decompensation was significantly associated with the development of ACLF in AD patients.

4.3. Prevalence of ACLF

The prevalence of ACLF in our study was 54 (33.8%) among the 160 admitted patients with AD cirrhosis. According to the EASL definition of ACLF grade, 29 (18%) of patients were in ACLF 1, while 15 (10%) ACLF 2 and ACLF 3 were seen in 10 (6%) patients, which aligns with the range reported in previous studies from different geographical regions [4,19–22,24]. This finding highlights the substantial burden of ACLF among cirrhotic patients admitted for acute decompensation events, underscoring the need for early recognition and appropriate management strategies.

4.4. Clinical presentation

At admission, patients with ACLF, compared to non-ACLF, presented a higher likelihood of ICU admission (57.4% vs. 13.2%, p-value <0.001). In alignment with the previous study, it was shown that ACLF often requires ICU support compared to non-ACLF patients, which aligns with the previous studies [4,19,22,28,29].

Upon admission, patients with ACLF vs. non-ACLF presented with more severe clinical features, including a jaundice (94.4% vs. 79.2%, p-value =0.012), HE (87.0% vs. 58.5%, p-value <0.001), renal failure (70.4% vs. 0%, p-value <0.001), signs of bacterial infection (48.1% vs. 19.8%, p-value <0.001), ascites (37.0% vs. 30.2%, p-value =0.011), and GIT bleeding (24.1% vs. 15.1%, p-value =0.163), respectively, while there was no significant difference between them regarding pneumonia, UTI, or GIT bleeding. These findings suggest a higher disease burden and multi-organ involvement in patients with ACLF at the time of admission, which is consistent with previous studies [4, 19,21–24,26,27,30].

Vital signs showed no significant difference between the two groups except for axillary temperature, which was significantly higher in the ACLF group (p-value = 0.001), which aligns with the previous studies and showed that there was no significant difference between the studied groups regarding HR, BP, and MAP [20, 21, 24].

SpO^2/FiO^2 and urine volume showed a significant decline in ACLF patients compared to non-ACLF (p-value <0.001, p-value <0.001, respectively). The use of vasopressors and supplemental oxygen was significantly higher in ACLF patients than non-ACLF patients (p-value <0.001, p-value <0.001, respectively), which aligns with [20, 21, 24, 31].

4.5. Precipitating events

The current study revealed that the most common etiology of precipitating events for ACLF is bacterial infection (48.1%) and GIT bleeding (24.1%), and no precipitating events were identified (24.1%) in patients with ACLF. These findings are consistent with previous studies highlighting the role of acute precipitating events, such as infections and bleeding, in triggering ACLF [4,5,7,19,21,23,27,32].

Worldwide, the most common causes of ACLF were bacterial infections (35%), followed by GIT bleeding (22%), and alcohol (19%) [5]. Where the precipitating events for ACLF in Europe are bacterial infection (32.6%), alcohol (24.5%), GIT bleeding (13.2%), and no precipitating events identified (43.6%) in patients with ACLF, it was indicated that the most common precipitating events for ACLF were bacterial infections followed by no precipitating events and alcohol [4], It was also demonstrated that one of the most common causes of ACLF in Western countries is bacterial infection [7]. Worldwide, the most common causes of ACLF were bacterial infections (35%), followed by GIT bleeding (22%), and alcohol (19%) [5]. Bacterial infections, particularly SBP, are the most common precipitating events in Asia, while active alcoholism in Europe has been a frequent precursor to ACLF [33,34].

4.6. Distribution of organ failure

The current study shows the most common organ failures prevalent in ACLF patients are renal failure (70.4%), flowed by liver (42.6%), cerebral (31.5%), circulatory (24.1%), coagulation (18.5%), and respiratory failure (11.1%). Organ dysfunction in AD without ACLF includes liver failure (11.3%), cerebral (5.6%), coagulation (2.8%), and circulatory (2.8%); there is no kidney or respiratory failure. Consistent with previous studies [4,19,21,24,29], renal failure (55.8%) was the most common organ failure, followed by liver (43.6%), coagulation (27.7%), cerebral (24.1%), circulation (16.2%), and lungs (9.8%) in ACLF, while in AD without ACLF, liver failure (7.2%) was the most common organ failure, followed by cerebral failure (2.5%), with a p-value<0.001.

4.7. Laboratory investigation

Regarding the comparison of laboratory investigations that revealed significant abnormalities in CBC and LFT at hospital admission between the studied patients, hemoglobin, platelets, and serum albumin showed a significant decline in ACLF compared to non-ACLF patients, while WBCs, NLR, total bilirubin, ALP, INR, and PTT showed significant elevation in ACLF compared to non-ACLF patients. No significant difference between them was found as regards AST, ALT, and GGT (p-value > 0.05). These findings are consistent with the previous studies [4,19–23].

In terms of serum creatinine, urea, and CRP, there is a significant elevation in ACLF compared to non-ACLF patients. While serum Na^{+}, K, Ca^{++}, and RBS showed a significant decline in ACLF compared to non-ACLF patients, these findings are consistent with previous studies [4,19–24], which showed that serum creatinine had a significant elevation in ACLF patients compared to non-ACLF patients (mean: 2.33 **vs.** 1.04 mg/dl, p-value < 0.001). while the serum Na^{+} showed a significant decline in ACLF compared to non-ACLF patients (mean: 133 ± 6 vs. 136 ± 6 mmol/l, p-< 0.001).

4.8. Prognostic Scoring

Regarding prognostic score, our study demonstrated that at the time of study enrolment, which was the second days after admission and 28–90 days, CLIF-OFs, CLIF-C ACLF, and CLIF-C AD scores were significantly higher in ACLF compared to non-ACLF patients. ACLF patients had a significantly higher number of OFs than non-ACLF patients. These findings align with previous studies on the utility of these scoring systems in risk stratification and prognosis prediction in ACLF [12,14, 19,20,35].

Also, consistent with the present study's findings that patients with ACLF had significantly higher CLIF-OF and CLIF-AD scores and a higher number of OFs compared to non-ACLF patients, it is recommended that CLIF-AD is the most effective predictor outcome at 28–90 days for non-ACLF patients [14, 16, 19, 36, 37].

4.9. Mortality rate

The mortality rates were significantly higher in ACLF compared to non-ACLF patients at 28 days (33.3% **vs.** 5.7%, p-value <0.001) and 90 days (68.5% **vs.** 23.6%, p-value <0.001). The mortality rates increase with ACLF grade; these findings align with the range reported in the previous study [4, 19, 20]. Moreau, R. et al. [4] reported rates at 28 days (33% **vs.** 4.7%) and 90 days (56% **vs.** 18%); Trebicka, J. *et al*. [19] reported rates at 90 days (40% **vs.** 15.5%); and Eltaweel et al. [20] reported rates (67.3% **vs.** 29.3%). Bhattacharyya et al. [23] reported rates (67.5% **vs.** 14%); Amarapurkar et al. and Dominguez et al. [22,26] reported similar results. (62.5% vs. 9.0%, p-value <0.001). Chetwood et al. [38] reported rates at 28 days (22.5% **vs.** 10%) and 90 days (55% **vs.** 21%). highlighting the notably higher mortality in ACLF patients compared to those without. Moreover, the ACLF grade exhibited a significant association with increased mortality.

5. Limitations of the Study

The current study was limited by the small sample size, being a single-center study, and the relatively short follow-up period. Further comparative studies with larger sample sizes and longer follow-ups are needed to

confirm our results and identify risk factors for adverse events.

6. Conclusion

ACLF represents a critical turning point in the course of chronic liver disease, characterized by acute decompensation, organ failures, and high short-term mortality. This study aimed to evaluate the clinical patterns and outcomes of cirrhotic Yemeni patients with ACLF admitted to Al-Sadaqa General Teaching Hospital, Aden. Patients with cirrhosis have a high risk of developing ACLF.

The findings of this prospective observational study confirm the severity of ACLF, with significant mortality rates and a high burden of complications. Bacterial infections were identified as the most common precipitating events, highlighting the importance of infection prevention and prompt treatment in cirrhotic patients. The CLIF-C ACLF and CLIF-AD Score were found to be strong predictors of mortality, emphasizing the need for early identification and risk stratification.

The study also identified several other factors associated with a poor prognosis, including infection, a higher CLIF-C ACLF score, and multiorgan failure. These findings underscore the complex interplay of factors that contribute to the development and progression of ACLF.

The management of ACLF requires a multidisciplinary approach, focusing on treating the precipitating event, supporting failing organs, and preventing complications. LT remains a life-saving option for patients with severe ACLF and a poor prognosis, which is not available in our country.

While this study provides valuable insights into the clinical characteristics and outcomes of ACLF, it also highlights the need for further research. Multicenter studies with larger sample sizes are needed to validate the findings and explore potential differences across diverse populations. Additionally, further investigation is warranted to identify novel prognostic factors, develop effective treatment strategies, and improve the long-term outcomes of ACLF survivors.

In conclusion, ACLF remains a significant challenge in the management of patients with cirrhosis. This study contributes to the growing body of knowledge on ACLF and has the potential to inform clinical practice and guide future research efforts. By improving our understanding of this complex syndrome, we can strive to improve the care and outcomes of patients with ACLF.

7. Conflict of Interest

The author declares that he has no competing interests.

8. Funding Information

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مقالة بحثية

الأنماط والنتائج السريرية في مرضى التليف الكبدي لليمنيين الذين يعانون من فشل الكبد الحاد على المزمن

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المُلخّص

فشل الكبد الحاد على المزمن (ACLF) هو حالة سريرية تتمثل في تفاقم مفاجئ لوظائف الكبد لدى مرضى الأمراض الكبدية المزمنة، ويرتبط بفشل الأعضاء خارج الكبد وزيادة معدل الوفيات. هدفت هذه الدراسة إلى تحديد الأنماط السريرية والنتائج المترتبة على مرضى تليّف الكبد اليمنيين المصابين بالفشل الكبدي الحاد على المزمن. أُجريت هذه الدراسة الوصفية المستقبلية على 160 مريضاً بالتليف الكبدي تم إدخالهم بين مايو 2023 ومايو 2024 إلى قسم الطب الباطني في مستشفى الصداقة التعليمي العام في عدن. بناء على معايير الجمعية الأوربية لدراسة أمراض الكبد: تم تقسيم المرضى إلى: المجموعة الأولى شملت 54 مريضًا بتليف كبدي يعانون من الفشل الكبدي الحاد على المزمن، والمجموعة الثانية شملت 106 مريضا بتليف كبدي لا يعانون من الفشل الكبدي الحاد على المزمن. نسبة انتشار الفشل الكبدي الحاد على المزمن كانت 33.8%. كان متوسط العمر 41.54 ±8.11 سنة في مرضى الفشل الكبدي الحاد على المزمن و39.22 ±8.14 سنة في المرضى غير المصابين بالفشل الكبدي الحاد على المزمن .كانت نسبة الذكور في مجموعة الفشل الكبدي الحاد على المزمن 51.9% مقابل 57.5% في المرضى غير المصابين. كانت الأسباب الأكثر شيوعاً للتليف الكبدي هي مجهولة السبب (58.8%) والتهاب الكبد المناعي الذاتي (25.6%). وُجدت علاقة ذات دلالة إحصائية بين سوابق الإدخال للمستشفى وتفاقم الكبد الحاد، في زيادة خطر تطور الفشل الكبدي الحاد على المزمن. كانت المضاعفات الأكثر شيوعاً هي اليرقان، والغيبوبة الكبدية، والقصور الكلوي، وعلامات العدوى البكتيرية، والاستسقاء، ونزيف الجهاز الهضمي. كانت العوامل الرئيسية المسببة للفشل الكبدي الحاد على المزمن هي العدوى البكتيرية، وخاصة التهاب الصفاق البكتيري (48.1%)، نزيف الجهاز الهضمي (24.1%)، وعدم وجود عوامل مسببه معروفة (24.1%). كانت حالات الفشل العضوي الأكثر شيوعاً هي الفشل الكلوي (70.4%)، فشل الكبد (42.6%)، والفشل الدماغي (31.5%). أظهر مرضى الفشل الكبدي الحاد على المزمن مستويات أعلى من كريات الدم البيضاء، البيليروبين، والكرياتينين في المصل. كانت معدلات الوفيات أعلى بشكل ملحوظ في مرضى الفشل الكبدي الحاد على المزمن مقارنة بالمرضى غير المصابين، على كلا الفترتين 28 يوماً (33.3% مقابل 5.7%) و90 يوماً (68.5% مقابل 23.6%). تظهر هذه الدراسة أن مرضى الفشل الكبدي الحاد على المزمن لديهم توقعات أسوأ، معدلات وفيات أعلى، دخول أكبر لوحدات العناية المركزة، بقاء أقل، ومعدلات أعلى لفشل الأعضاء مقارنة بالمرضى غير المصابين بالفشل الكبدي الحاد على المزمن.

الكلمات المفتاحية: فشل الكبد الحاد على المزمن، تفاقم كبدي حاد، تليف الكبد.

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