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КЛИНИКО-ЛАБОРАТОРНАЯ ХАРАКТЕРИСТИКА И ЛЕТАЛЬНОСТЬ У ПАЦИЕНТОВ С ЦИРРОЗОМ ПЕЧЕНИ

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Clinical and Laboratory Characteristics and Mortality in Patients with Liver Cirrhosis

Резюме

Цель исследования: представить клинико-лабораторную характеристику и оценить частоту 28-дневной летальности у пациентов с циррозом печени (ЦП). **Материалы и методы:** в исследование включено 137 пациентов (средний возраст 51±11, 75 (54,7 %) мужчин) с ЦП. Диагноз устанавливался на основе клинических и лабораторно-инструментальных данных. Согласно шкале органной дисфункции CLIF-C OF-score пациентов разделили на две группы: основная группа (n=72, средний возраст 52±11, 37 (51,4 %) мужчин)) — с наличием острой печеночной недостаточности на фоне хронической (ACLF) и группа сравнения (n=65, средний возраст 49±11, 38 (58,5 %) мужчин)) — с декомпенсированным циррозом печени (декЦП) без ACLF. Оценивались клинические показатели, маркеры воспаления, уровень аммиака, лактата, прогностические шкалы, 28-дневная летальность. Статистический анализ данных проводился с использованием программы IBM SPSS 26. **Результаты:** Среди пациентов с ЦП 72 (52,6 %) пациента имели декЦП, а 65 (47,4 %) — ACLF, преобладала алкогольная этиология заболевания (52,8 % и 63,1 % соответственно). Пациенты с ACLF характеризовались значимо более тяжелой печеночной, почечной дисфункцией, системным воспалением и высокими баллами всех прогностических шкал (p <0,001). 28-дневная летальность составила 9,7 % в группе декЦП и 27,7 % — в группе ACLF, достигая 71,4 % при ACLF 3. Анализ выживаемости показал значительные различия между группами (Log-Rank p <0,001). **Заключение:** установлена высокая распространенность ACLF среди госпитализированных пациентов с ЦП. Использование шкалы CLIF-C OFs позволяет эффективно стратифицировать пациентов по степени риска, что имеет важное значение для определения тактики ведения и своевременного направления в специализированные центры.

Ключевые слова: *цирроз печени, острая печеночная недостаточность на фоне хронической (ACLF), декомпенсация цирроза печени*

Конфликт интересов

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Abstract

Background: To present clinical and laboratory characteristics and assess 28-day mortality in patients with liver cirrhosis (LC). **Materials and Methods:** The study included 137 patients (mean age 51±11; 75 (54,7 %) males) with LC. The diagnosis was established based on clinical, laboratory, and instrumental data. According to the CLIF-C OF organ dysfunction score, patients were divided into two groups: the main group (n=72, mean age 52±11; 37 (51,4 %) males) — with acute-on-chronic liver failure (ACLF), and the comparison group (n=65, mean age 49±11; 38 (58,5 %) males) — with decompensated liver cirrhosis (DC) without ACLF. Clinical parameters, inflammatory markers, ammonia and lactate levels, prognostic scores, and 28-day mortality were assessed. Statistical analysis was performed using IBM SPSS 26. **Results:** Among patients with LC, 72 (52,6 %) had DC and 65 (47,4 %) had ACLF. Alcohol-related etiology was predominant (52,8 % and 63,1 %, respectively). Patients with ACLF were characterized by significantly more severe hepatic and renal dysfunction, systemic inflammation, and higher scores on all prognostic scales (p <0,001). The 28-day mortality rate was 9,7 % in the DC group and 27,7 % in the ACLF group, reaching 71,4 % for ACLF grade 3. Survival analysis showed significant differences between the groups (Log-Rank p <0,001). **Conclusion:** A high prevalence of ACLF was established among hospitalized patients with LC. The use of the CLIF-C OFs scale allows for effective risk stratification of patients, which is important for determining management tactics and timely referral to specialized centers.

Key words: *liver cirrhosis, acute-on-chronic liver failure (ACLF), decompensated liver cirrhosis*

Conflict of interests

Co-author of the article Nikitin I.G. is a member of the editorial board of the journal «The Russian Archives of Internal Medicine». The article has passed the peer-review procedure adopted by the journal. Nikitin I.G. did not participate in the decision to publish this article.

Co-author of the article Ilchenko L.Yu. is the editor-in-chief of the journal «The Russian Archives of Internal Medicine». The article has passed the peer-review procedure adopted by the journal. The decision to publish the article was made by the editorial board without the participation of the editor-in-chief.

The authors did not declare any other conflicts of interest

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Conformity with the principles of ethics

The study protocol was approved by the Local Ethics Committee of N.I. Pirogov Russian national research medical university (Approval No. 235, December 18, 2023). Informed consent was obtained from all patients who participated in the study.

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ALD — alcoholic liver disease, ALT — alanine aminotransferase, AST — aspartate aminotransferase, EV — esophageal varices, GGT — gamma-glutamyl transpeptidase, DLC — decompensated liver cirrhosis, EMIAS — Common Medical Information-Analytical System (Russia), GIB — gastrointestinal bleeding, UTI — urinary tract infection, INR — international normalized ratio, NASH — non-alcoholic steatohepatitis, NLI — neutrophil-lymphocyte index, AHF — acute hepatic failure, HE — hepatic encephalopathy, ROPIP — Russian Society for Liver Diseases, CRP — C-reactive protein, SBP — spontaneous bacterial peritonitis, MBP — mean blood pressure, CLD — chronic liver diseases, LC — liver cirrhosis, ALP — alkaline phosphatase

Introduction

Liver cirrhosis (LC) is a common disease representing a terminal stage of the chronic liver disease, associated with high mortality.

The LC-associated mortality is 2.4 % of the global mortality, with two million deaths annually (11th global cause of death) [1, 2].

LC develops over several stages: Stages 0–2 of compensated LC, when the portal pressure gradually increases in the absence of clinical signs or with mild signs; Stage 3 of LC, manifesting with the first episode of bleeding from esophageal varices (EV); Stage 4 of LC, characterized by the emergence of ascites and its worsening, an episode of hepatic encephalopathy (HE); Stage 5 of recurrent decompensated LC (DLC) [3, 4].

These conditions significantly increase the rate of patient hospitalization and intrahospital mortality within the range of 10 to 50 % [5].

However, the patient management with “terminal cirrhosis”, including severe complications (recurrent/refractory ascites, infections, renal or another extrahepatic organ dysfunction) and the possible emergence of acute hepatic failure (AHF) within the setting of the chronic one, or Acute-on-chronic liver failure (ACLF), is an important issue still remaining in clinical practice [6].

Renal and extrahepatic organ dysfunction is observed in ACLF with systemic inflammation and a very high 28-day mortality [7]. In the meta-analysis presented, the global ACLF prevalence among patients hospitalized with LC is 35 % [8].

Despite the generally recognized ACLF diagnostic criteria, data on the prevalence and features of this syndrome in the Russian population remain limited.

Study objective: presenting clinical & laboratory characteristics, determining the 28-day mortality rate in patients with liver cirrhosis.

Study materials and methods

The study was approved by the Local Ethics Committee of FSAEI HE N.I. Pirogov Russian National Research Medical University (Pirogov University) (Protocol No. 235 dated December 18, 2023) and arranged at the Clinical Base of the State Budget Healthcare Institution "V.M. Buyanov City Clinical Hospital" (SBHI V.M. Buyanov CCH), G.I. Storozhakov Department of Hospital Therapy of the Institute of Clinical Medicine, within the period from October 2023 to April 2025.

According to the Clinical Guidelines of Russian Society for Liver Diseases (ROPIP), the diagnosis of LC was established based on the clinical and laboratory-instrumental signs of intrahepatic portal hypertension with signs of hepatic failure or without them [9].

Inclusion criteria: males and females aged 18 to 75 years with LC that signed the informed consent for the study participation and anonymous publication of results.

Non-inclusion criteria: severe decompensated somatic extrahepatic diseases; psychic diseases; a malignancy detected in the patient before or during the hospitalization; patients not signing the informed consent.

This study was a prospective cohort study that enrolled 137 patients (76 males and 61 females, mean age 50 [42–58] years), with predominantly alcoholic liver cirrhosis (79; 57.7 %). The patients were examined within the first three days of hospitalization. The patient's condition severity was determined using the following scales: Child-Turcotte-Pugh (CTP) [10], Model for End-Stage Liver Disease (MELD) [11], Model for End-Stage Liver Disease-Sodium (MELD-Na) [12], Maddrey Discriminant Function (MDF) [13], Sequential Organ Failure Assessment (SOFA) [14].

The organ dysfunction was evaluated using the CLIF Consortium Organ Failure score (CLIF-C OF-score) [15], which helped to divide patients into two groups: ACLF (**main group**) and DLC (**comparison group**) (Table 1).

The ACLF group was divided into ACLF 1, ACLF 2, ACLF 3 subgroups based on the organ dysfunction severity. Such design helped to evaluate differences in mortality and features between patients with the different degrees of the hepatic failure severity.

The following inflammatory response markers were evaluated: C-reactive protein (CRP), neutrophil-lymphocyte index (NLI; a ratio of absolute neutrophil count to absolute lymphocyte count) [16].

HE severity was evaluated based on clinical signs using the West-Haven score and the ammonia levels in capillary blood (microdiffusion method; PocketChem BA PA-4140, Japan) [17].

28-day mortality was selected as a primary endpoint in accordance with the international standards of prognosis evaluation in patients with ACLF established in the CANONIC trial [7]. This period is critical for determining the short-term prognosis and decisions on the liver transplant. 28-day mortality was determined from the moment of the patient hospitalization into the inpatient department. The outcome was evaluated on Day 28 of follow-up. The information about the patient's vital status after discharging was clarified using the Common Medical Information-Analytical System (EMIAS, Russia).

Data were statistically processed using the IBM SPSS 26 software. The distribution normality was evaluated

Table 1. *The CLIF-C OFs for patients with cirrhosis of the liver*

Organ/system		Subscore		
		1	2	3
Liver, bilirubin, μmol/L		<103	103 до 205	≥205,2
Kidneys, creatinine, μmol/L		<177	177 до 309	≥309 or RRT
Brain — West-Haven grade for HE		0	1-2	3-4
Coagulation, INR		<2	2,0 до 2,5	≥2,5
Circulatory, MAP		≥70	<70	Use of vasopressors
Respiratory	PaO2/FiO2	>300	≤300, но >200	≤200
	SpO2/FiO2	>357	≤357, но >214 >214 and ≤357	≤214

Note. RRT- renal replacement therapy; HE — hepatic encephalopathy; INR — International Normalized Ratio; MAP — Mean Arterial Pressure; PaO2, partial pressure of arterial oxygen; FiO2 — fraction of inspired oxygen; SpO2 — pulse oximetric saturation

using the Kolmogorov-Smirnov test. Descriptive statistics were presented using the median and the 25th;75th percentiles for quantitative variables; rates and proportions in the sample for qualitative variables. The Mann-Whitney U-test was used to compare quantitative data in two non-associated samples. The non-parametric Kruskall-Wallis test was used for multiple comparisons of quantitative data in non-linked samples; aposteriori comparisons were provided using the Dunn's test with the Holm's adjustment. Percent proportions during the analysis of quadrupole tables and multipole contingency tables were analyzed using the Pearson's chi-square test. Survival was analyzed using the Kaplan-Meier method. The survival distribution was compared using the log-rank test. Survival diagrams were constructed for graphical data presentation.

The significance level (p) was in general below 0.050 in all comparisons described.

Results

Based on the CLIF-C OF score, 72 (52.6 %) of 137 patients were diagnosed with DLC, while 65 (47.4 %) had AHF within the setting of chronic failure (ACLF). Groups were similar by gender (p=0.856) and age (p=0.128). Among patients with ACLF, those with ACLF 1 predominated (n=41; 63.1 %), while other type — ACLF 2 (n=17; 26.2 %) and ACLF 3 (n=7; 10.7 %) — were less common.

The most common factors provoking the condition worsening in DLC (n=72) were infections (n=20; 27.8 %), alcohol abuse (n=17; 23.6 %), and gastrointestinal bleeding (GIB) (n=9; 12.5 %). The combined effects of several factors were observed in 16 cases (22.2 %); the cause was not determined in 8 patients (11.1 %).

The main ACLF triggers (n=65) included alcohol consumption (n=27; 41.5 %) and the combination of several factors (n=28; 43.1 %); GIB (n=4; 6.2 %) and infections (n=3; 4.6 %) were less common. ACLF cause was not determined in 2 patients (3.1 %). The groups analyzed also did not differ in the severity of edema and ascites as a cause of hospitalization (p=0.238).

The rate of repeated hospitalizations was higher among patients with ACLF (46; 74.2 %) vs. DLC (42; 65.6 %), however, no statistically significant difference was confirmed.

The leading etiological role in LC in both groups belonged to the alcoholic liver disease (ALD): 38 (52.8 %) in DLC and 41 (63.1 %) in ACLF (Table 2). Patients with the mixed etiology of chronic liver diseases (CLDs) formed a significant proportion of cases: ALD + viral infection — 14 (19.4 %) DLC and 8 (12.3 %) ACLF) and ALD + non-alcoholic steatohepatitis (NASH) — 14 (19.4 %) DLC, 15 (23.1 %) ACLF). The "viral infection + NASH" combination was reported only in the DLC group (n=3 [8.3 %]).

See Tables 3 and 4 for specific clinical-morphological and laboratory patient characteristics.

The degree of anemia severity correlated with the increased organ dysfunction severity in ACLF (p=0.05), with the lowest hemoglobin levels observed in ACLF 3.

Patients with ACLF also had significantly larger white blood cell counts (9.8 [7.5–13.2]; 7,9 [5.6–11.2] x10^9/L, p=0.009), CRP levels (34 [16–52.8]; 25 [9.6–48.3] mg/L, p=0.05), and NLI (4.4 [2.5–7.5]; 2.9 [2.1–5.5], p=0.004) vs. the DLC group. The NLI level demonstrated a trend to increasing with the ACLF severity (ACLF 1: 3.8 [2.3–6.7]; ACLF 2: 4.9 [2.2–7.0]; ACLF 3: 4.9 [1.0–7.7]; p=0.023).

ACLF was characterized by significantly higher levels of total bilirubin (p<0.001), AST (p=0.008), and ALP (p=0.036) vs. patients with DLC. The AST/ALT ratio was higher in ACLF. Bilirubin levels significantly increased with ACLF progression (p<0.001).

The protein-synthetic liver function, i.e. the total protein (p=0.05) and albumin (p=0.05) levels, were lower in the ACLF group. The lowest values were reported in ACLF 3 (total protein 56 g/L, albumin 24 g/L, p=0.015).

The capillary blood ammonia level reflecting the detoxifying hepatic function was significantly higher in patients with ACLF vs. the DLC group (p=0.048), increasing in association with the organ dysfunction severity (ACLF 1: 148 [101–195]; ACLF 2: 125 [87–186]; ACLF 3: 196 [172–199] μmol/L; p=0.017).

When evaluating the renal function, higher creatinine levels were reported in ACLF vs. DLC (p=0.016),

Table 2. *Etiology of liver cirrhosis among patients with decompensated cirrhosis (DC) and ACLF*

Etiology of Cirrhosis	DC (n=72), n (%)	ACLF (n=65), n (%)	p-value
ALD	38 (52,8)	41 (63,1)	0,234
ALD + viral infection	14 (19,4)	8 (12,3)	0,267
ALD + NASH	14 (19,4)	15 (23,1)	0,609
Viral infection + NASH	6 (8,3)	0 (0)	0,014

Note: Data are presented as n (%). Group comparison was performed using Fisher's exact test due to expected frequencies <5. ALD — alcoholic liver disease; NASH — non-alcoholic steatohepatitis DC — decompensated cirrhosis; ACLF — acute-on-chronic liver failure.

Table 3. *Comparative clinical and laboratory characteristics of patients with decompensated cirrhosis (DC) and acute-on-chronic liver failure (ACLF)*

Indicator / Reference range	Overall cohort n=137	DC n=72	ACLF n=65	P value
Gender, n (%) males females	 62 (45,3) 75 (54,7)	 38 (52,8) 34 (47,2)	 38 (58,5) 27 (41,5)	0,856
age	50 (42-58)	51,5 (44,3-59,0)	48,0 (51,0-58,0)	0,128
Readmissions, n (%)	88 (69,8 %)	42 (65,6 %)	46 (74,2 %)	0,295
Ascites on admission, n (%)	101 (79, 5 %)	48 (84,2 %)	53 (75,7 %)	0,238
Hemoglobin, 130–170 г/л	109 (90-126)	116 (94-134)	103 (87-118)	**0,010**
Platelets, 150–340×10^9 /л	122 (88-161)	123 (88-164)	121 (76-161)	0,674
Leukocytes, 4–10×10^9 /л	9.0 (6,4-12,3)	7,9 (5,6-11,2)	9,8 (7,5-13,2)	**0,009**
CRP, 0,1–7 мг/л	29 (14-51)	25 (9,6-48,3)	34 (16-52,8)	**0,05**
NLR 1–2	3,6 (2.3-6,4)	2,9 (2,1-5,5)	4,4 (2,5-7,5)	**0,004**
Total protein, 65–85 г/л	67 (59-71)	68 (63-71)	64 (57-72)	**0,05**
Albumin, 35–55 г/л	28 (24-32)	28 (26-33)	27 (23-32)	**0,05**
Total bilirubin, 1,7–20,5 мкмоль/л	108 (36-268)	49 (27-100)	272 (165-467)	**<0,001**
Direct bilirubin, 0,9–5 мкмоль/л	53 (17-142)	27 (16,5-52,5)	143 (79-210)	**<0,001**
ALT, 0–32 МЕ/л	42 (20-73)	35 (19-63)	48 (27,3-77,5)	**0,048**
AST, 5–34 МЕ/л	111 (62-184)	88 (45-173)	140 (82,5-189)	**0,008**
GGT, 0–73 МЕ/л	182 (77-679)	153 (45-643)	249 (96-715)	0,064
ALP, 64–306 МЕ/л	335 (227-501)	295 (201-478)	359 (277-578)	**0,036**
Creatinine, 71–115 мкмоль/л	89 (72-124)	87 (72-108)	103 (71-177)	**0,016**
Lactate, 0,5 до 2,2	2,7 (1,8-4.4)	1,6 (1,5-2,3)	3,4 (2,5-5,4)	**0,001**
INR, 0,85–1,2	1,6 (1,3-2,1)	1,47 (1,29-1,77)	1,86 (1,41-2,46)	**<0,001**
Ammonia, 8–60 мкмоль/л	149 (109-205)	146 (113-219)	149 (101-196)	**0,048**

Note: Data are presented as median (25th; 75th percentile) for continuous and n (%) for categorical variables. Continuous variables were compared using the Mann-Whitney U test. Data for 'Previous hospitalizations' and 'Ascites at admission' were available for 126 and 127 patients, respectively. CRP — C-reactive protein; NLR — neutrophil-to-lymphocyte ratio; ALT — alanine aminotransferase; AST — aspartate aminotransferase; GGT — gamma-glutamyl transferase; ALP — alkaline phosphatase; INR — international normalized ratio.

reaching the maximum in ACLF 3 (p=0.003). Lactate was also significantly higher in the ACLF group (p=0.001), with its peak levels in ACLF 3.

INR was significantly higher in the ACLF group (p<0.001), with its values increasing in correlation with the ACLF severity (p<0.001).

All the analyzed prognostic scores (MELD-Na, MDF, SOFA, CLIF-C OFs, Child-Pugh) presented in Table 5 significantly differed in ACLF vs. patients with DLC (p<0.001 for all patients), as well as in different ACLF stages (ACLF 3 > ACLF 2 > ACLF 1; p<0.001).

Kaplan-Meier curves were constructed for the survival analysis. Statistically significant differences were detected in the 28-day survival between the groups of patients with DLC, ACLF 1, ACLF 2, and ACLF 3 (Log-Rank p<0.001). The features of curve changes demonstrated their significant divergence, which was the clearest in the ACLF 3 group (Fig. 1).

The mean maximum life expectancy in DLC was 26.7±0.5 days; 95 % CI 25.6–27.7; the minimum one was reported in ACLF 3 patients — 13±3.4 days; 95 % CI 5.6–20.4.

The 28-day mortality in patients with DLC did not exceed 9.7 % (7/72), while in patients with ACLF it reached 27.7 % (18/65); (ACLF 1: 24.4 % (10/41), ACLF 2: 17.6 % (3/17), ACLF 3: 71.4 % (5/7).

To detect factors directly associated with 28-day mortality, the comparative analysis of clinical & laboratory parameters and data from prognostic scales was arranged between the survived (n=112) and deceased (n=25) patient groups (Table 6). The proportion of patients with ACLF in the deceased group was significantly higher (18 [72.0 %] vs. 47 [42.0 %] among survived subjects; p=0.007). And, on the contrary, decompensated patients without ACLF predominated among survived patient (65 (58.0 %) vs. 7 (28.0 %) among deceased ones; p=0.007).

Table 4. *Comparative clinical and laboratory characteristics by different stages of ACLF*

Indicator	ACLF 1 (n=41)	ACLF 2 (n=17)	ACLF 3 (n=7)	P value
Gender, n (%)				
males	27 (65,9 %)	8 (47,1 %)	3 (42,9 %)	
females	14 (34,1 %)	9 (52,9 %)	4 (57,1 %)	
age, лет	48 (41-58 %)	47 (41-57 %)	50 (34-57 %)	0,498
Ascites on admission, n (%)	27 (75,0 %)	17 (100,0 %)	4 (100,0 %)	0,090
Readmissions, n (%)	30 (76,9)	13 (81,3)	3 (42,9)	0,181
Hemoglobin, 130–170 г/л	110 (93-123)	93 (83-103)	90 (64-106)	0,005
Platelets, 150–340×10⁹ /л	112 (66-160)	126 (100-162)	132 (70-146)	0,801
Leukocytes, 4–10×10⁹ /л	8,7 (6,8-11,1)	12,1 (9,7-14,8)	12,4 (9,6-25,0)	0,001
CRP, 0,1–7 мг/л	29,7 (16-51)	38,4 (12-48,4)	48 (31-84)	0,106
NLR 1–2	4,2 (2,6-6,5)	5,0 (2,8-6,4)	4,9 (2,0-8,6)	0,023
Total protein, 65–85 г/л	67 (59-73)	64 (57-70)	56 (48-62)	0,015
Albumin, 35–55 г/л	28 (24-34)	27 (23-31)	24 (20-26)	0,015
Total bilirubin, 1,7–20,5 мкмоль/л	260 (117-431)	316 (216-444)	635 (209-640)	<0,001
Direct bilirubin, 0,9–5 мкмоль/л	101 (40-198)	138 (111-206)	199 (145-208)	<0,001
ALT, 0–32 МЕ/л	60 (28-74)	43 (18-80)	62 (43-77)	0,151
AST, 5–34 МЕ/л	147 (71-187)	141 (108-183)	130 (96-195)	0,052
GGT, 0–73 МЕ/л	330 (121-735)	194 (88-517)	156 (74-248)	0,222
ALP, 64–306 МЕ/л	359 (283-580)	381 (340-562)	278 (129-504)	0,044
Creatinine, 71–115 мкмоль/л	89 (69-177)	89 (78-160)	177 (125-349)	0,003
Lactate, 0,5 -2,2	3,0 (2,5-4,4)	3,6 (1,8-7,1)	4,9 (2,6-8,2)	0,008
INR, 0,85–1,2	1,57 (1,32-2,08)	2,30 (1,86-2,83)	2,57 (2,09-3,71)	<0,001
Ammonia, 8–60 мкмоль/л	125 (87-186)	148 (101-195)	196 (172-199)	0,017

Note: Data are presented as median (25th; 75th percentile) for continuous and n (%) for categorical variables. Overall comparison of continuous variables across the three groups was performed using the Kruskal-Wallis test. CRP — C-reactive protein; NLR — neutrophil-to-lymphocyte ratio; ALT — alanine aminotransferase; AST — aspartate aminotransferase; GGT — gamma-glutamyl transferase; ALP — alkaline phosphatase; INR — international normalized ratio.

Table 5. *Comparison of prognostic scales and outcomes*

Scale	DC (n=72)	ACLF (1,2,3) (n=65)	p-value[1]	ACLF1 (n=41)	ACLF2 (n=17)	ACLF3 (n=7)	p-value[2]
MELD-Na	18 (13-23)	28 (24-33)	<0,001	26 (22-29)	30 (26-35)	37 (31-42)	<0,001
MDF	33 (31-45)	58 (40-100)	0,002	43 (35-78)	67 (51-102)	103 (92-134)	0,001
SOFA	4 (2-5)	6 (5-7)	<0,001	5 (4-6)	6 (5-7)	10 (7-12)	<0,001
CLIF-C OFs	7 (7-8)	9 (8-11)	<0,001	9 (9-9)	11 (10-11)	13 (12-14)	<0,001
Child-Pugh class, n (%)							0,012
– A	2 (2,8 %)	0	<0,001	0	0	0	NA
– B	26 (36,1 %)	7 (11,1 %)		7 (17,9 %)	0	0	1-3 =0,023
– C	44 (61,1 %)	56 (88,9 %)		32 (82,1 %)	17 (100 %)	7 (100 %)	1-3 =0,046
28-day mortality, %	7 (9,7 %)	18 (27,7 %)	0,007	10 (24,4 %)	3 (17,6 %)	5 (71,4 %)	0,05

Note: Data are presented as median (25th; 75th percentile) for scores and n (%) for Child-Pugh class. p-value[1] denotes comparison between DC and ACLF (1-3) groups. p-value[2] denotes overall comparison across ACLF 1, 2, and 3 groups (Kruskal-Wallis test for scores and Chi-square for Child-Pugh class). MELD-Na — MELD-Sodium; MDF — Maddrey discriminant function; SOFA — Sequential Organ Failure Assessment; CLIF-C OFs — CLIF Consortium Organ Failure score.

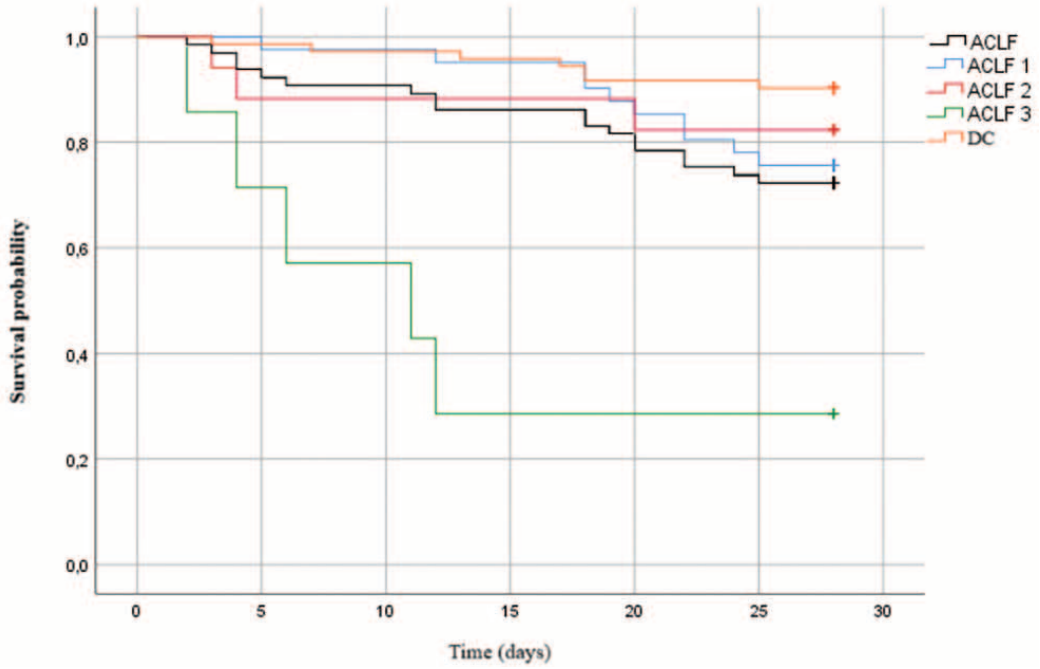

Figure 1. *28-day survival of patients with ACLF and DC*
Abbreviations: ACLF — acute-on-chronic liver failure; DC — decompensated cirrhosis without ACLF.

The age and gender of patients with LC did not differ in the deceased and survived groups (p>0.05). When evaluating oxygenation and hemodynamics parameters among the deceased patients, a decreased SpO_2/FiO_2 index (457.00 [452.00; 466.00] vs. 461.00 [461.00; 466.00]; p=0.001) and a lower mean blood pressure (81.00 [71.50; 91.50] mm Hg vs. 87.00 [80.00; 93.00] mm Hg among survived patients; p<0.001) were reported.

The following laboratory abnormalities were reported: white blood cells (9.70 vs. 8.62×10^9/L; p=0.025), NLI (5.73 vs. 3.26; p=0.034), CRP (48.00 vs. 25.50 mg/L; p<0.001), hyperbilirubinemia (252.0 vs. 97.0 μmol/L; p<0.001), AST (143.0 vs. 108.0 U/L; p=0.046), ALT (47.0 vs. 39.0 U/L; p=0.018); hyperlactatemia (3.40 vs. 2.50 mmol/L; p=0.040) — among the deceased and survived patients with LC, respectively.

One should also underline a larger rate of various infectious complications in the deceased patient group. Thus, urinary tract infections (UTIs) were detected in 16 (69.6 %) deceased vs. 35 (32.1 %) survived patients with LC (p=0.001). However, the rate of pneumonia and spontaneous bacterial peritonitis (SBP) in the groups analyzed did not differ (p>0.05).

Besides, when calculating prognostic scales, significant differences were also reported in the deceased vs. survived group**:** MELD (26.0 vs. 20.0; p=0.001), MELD-Na (26.0 vs. 22.0; p=0.001), Child-Pugh (score: 12.0 vs. 11.0; p=0.001; Class C: 92.0 % vs. 70.0 %; p=0.023), SOFA (5.0 vs. 4.0; p=0.025), CLIF-SOFA (9.0 vs. 8.0; p=0.002), CLIF-C OFs (9.0 vs. 8.0; p=0.001).

Discussion of results

Clinical & laboratory parameters of patients with DLC and ACLF were comparatively analyzed. Patient groups with DLC and ACLF were uniform and did not differ in gender or age.

Among patients hospitalized into the inpatient department with events of decompensated LC, a high rate of ACLF reaching 47 % was confirmed — this exceeds the values obtained in the European population (22.6 %) [7].

Decompensated LC as a cause of inpatient hospitalization is caused by various factors (late hospitalization, untimely referral, alcohol abuse).

The profile of DLC and ACLF triggers significantly differed**:** if isolated causes (infection, alcohol, GIB) were typical for DLC, while ACLF developed mainly due to the combination of these factors.

In both groups ALD was the most common cause of LC, which confirms the importance of alcoholization as a leading factor of CLDs.

Based on the meta-analysis published in 2023, the prevalence of ALD constitutes 3.5 % of the global population, with 5-year mortality rates reaching 50 %. With that, the prevalence of alcoholic LC is 0.3 %, with variable rates in different regions: 3.0 % in Europe, 3.6 % in the Western-Pacific region, 7.0 % in the United States of America [18].

The proportion of deaths caused by alcohol consumption in Russia is approximately 18 %, which corresponds to the 5th place among 180 countries [19].

Table 6. *Clinical and laboratory characteristics of deceased and surviving patients with liver cirrhosis*

Parameter	Deceased (n=25)	Survivors (n=112)	p-value
age, лет	50,0 [46,0; 58,0]	50,0 [42,0; 58,0]	0,085
gender, male (n(%))	14 (56,0 %)	61 (54,5 %)	0,889
Clinical indicators			
ACLF (n(%))	18 (72,0 %)	47 (42,0 %)	**0,007**
DC, (n(%))	7 (28,0 %)	65 (58,0 %)	**0,007**
Readmissions	12 (54,5 %)	76 (73,1 %)	0,085
Ascites	15 (78,9 %)	86 (79,6 %)	0,946
MAP, мм рт.ст.	81,00 [71,50; 91,50]	87,00 [80,00; 93,00]	**<0,001**
SpO_2/FiO_2	457,00 [452,00; 466,00]	461. 00 [461,00; 466,00]	**0,001**
UTI (n(%))	16 (69,6 %)	35 (32,1 %)	**0,001**
Pneumonia (n(%))	4 (16,0 %)	10 (9,1 %)	0,306
SBP (n(%))	2 (10,5 %)	21 (27,6 %)	0,120
Scale			
MELD	26,0 [21,0; 33,0]	20,0 [15,0; 26,0]	**0,001**
MELD-Na	26,0 [24,0; 34,0]	22,0 [16,0; 28,0]	**0,001**
DF	67,0 [43,0; 94,0]	43,0 [32,5; 79,0]	0,114
CPT класс C (n(%))	23 (92,0 %)	77 (70,0 %)	**0,023**
CPT класс B (n(%))	2 (8,0 %)	31 (28,2 %)	**0,034**
SOFA	5,0 [4,0; 7,0]	4,0 [3,0; 6,0]	**0,025**
CLIF-SOFA	9,0 [8,0; 12,0]	8,0 [6,0; 9,0]	**0,002**
CLIF-C OFs	9,0 [8,0; 11,0]	8,0 [7,0; 9,0]	**0,001**
CLIF-C ACLF	43,0 [0,0; 55,0]	0,0 [0,0; 44,0]	**0,001**
Laboratory parameters			
Hemoglobin, г/л	109,00 [88,50; 126,75]	111,00 [92,25; 123,50]	0,895
Leukocytes, $\times 10^9$/л	9,70 [8,00; 12,00]	8,62 [5,90; 12,60]	**0,025**
NLR	5,73 [2,86; 8,71]	3,26 [2,18; 5,75]	**0,034**
Lactate, ммоль/л	3,40 [2,50; 5,60]	2,50 [1,70; 4,23]	**0,040**
Total bilirubin, мкмоль/л	252,0 [135,0; 505,0]	97,0 [34,0; 237,0]	**<0,001**
ALT, Ед/л	47,0 [34,0; 90,0]	39,0 [19,0; 71,0]	**0,018**
AST, Ед/л	143,0 [87,0; 184,0]	108,0 [57,0; 183,0]	**0,046**
CRP, мг/л	48,00 [33,00; 67,00]	25,50 [12,00; 48,00]	**<0,001**
Ammonia	184 [144; 223]	142 [102; 196]	0,068

Note: Data are presented as median (25th; 75th percentile) for continuous and n (%) for categorical variables. Comparisons were made using the Mann-Whitney U test for continuous and Chi-square test for categorical variables. ACLF — acute-on-chronic liver failure; DC — decompensated cirrhosis; MAP — mean arterial pressure; UTI — urinary tract infection; SBP — spontaneous bacterial peritonitis; MELD — Model for End-Stage Liver Disease; MELD-Na — MELD-Sodium; DF — Discriminant Function; SOFA — Sequential Organ Failure Assessment; CLIF-SOFA — CLIF-SOFA score; CLIF-C OFs — CLIF Consortium Organ Failure score; CLIF-C ACLF — CLIF-C ACLF score

With that, chronic alcohol abuse is the most common cause of LC. Less frequently the etiology of CLDs among the patients examined by us was associated with a combination of several factors, i.e. alcohol, viral hepatitis, metabolic disorders.

The key role in the development of ACLF belongs to the systemic inflammatory reaction, which mechanism is still not fully clear. Modern data define the two main pathways leading to the immune response dysregulation and progressive inflammation [20].

The first pathway is associated with bacterial translocation and subsequent activation of Toll-like receptors (TLRs) [21] in immune cells via the interactions with pathogen-associated molecules (PAMPs) [22], e.g. bacterial lipopolysaccharides. This causes the formation of proinflammatory molecules, inducing the systemic inflammatory response.

The second pathway includes the release of damage-associated molecular patterns (DAMPs) by the altered hepatocytes [22], which leads to similar effects, as these molecules also bind to TLRs and other receptors, thus stimulating immune cells. The prolonged activation of these mechanisms leads to the constant proinflammatory response in patients with LC.

Besides, patients with cirrhosis and ACLF demonstrate impaired reactions to PAMPs due to immune exhaustion and endotoxin tolerance, which in turn is also caused by immunodeficiency associated with LC. This imbalance leads to the enhanced susceptibility to infectious complications. Ethanol enhances the sensitivity of hepatocytes to apoptosis, which potentiates their synergistic damaging effects with the bacterial lipopolysaccharide.

UTIs were significantly more common in our study in the deceased patient group, followed by pneumonia and SBP.

Although inflammation plays an important prognostic role in patients with ACLF, the modern laboratory evaluation of the inflammatory response is difficult, mainly due to the absence of a possible routine evaluation of cytokines outside of scientific institutions [23].

Due to the fact that systemic inflammation is a pivotal part of the ACLF pathogenesis, the search for available early markers of this condition is an important objective.

A systemic inflammation parameter, NLI, is one of such markers of the cellular immunity activation. The normal NLI range is 1 to 2; values over 3.0 or below 0.7 are considered abnormal in adults [24].

According to the CANONIC trial, CRP levels in the blood of patients with ACLF are significantly higher than in patients without organ dysfunction. Undoubtedly, the contribution of the infectious trigger is evident, however, when excluding patients with the confirmed infection, statistically significant differences in CRP levels are preserved between groups with or without infectious complications [7], which confirms the importance of immune dysregulation typical for ACLF.

Patients with ACLF demonstrated higher levels of liver dysfunction parameters, including bilirubin, INR, AST, ALP levels, as well as the decreased protein-synthetic function and hemoglobin levels, confirming the progressive organ failure.

Our study results have demonstrated that the mean life expectancy in patients with decompensated LC was 26 days, which significantly exceeded this parameter in the ACLF 3 group (13 days). This underlines the required early detection, timely treatment of cirrhosis complications, and the importance of evaluating disease stages for the outcome prediction.

In our study 28-day mortality in the DLC group was 9.7 %, in ACLF 1 — 24.4 %, while in ACLF 2 — 17.6 %, and ACLF 3 — 71.4 %. These results correlate with parameters in the CANONIC trial arranged based on the analysis of 1343 inpatients with LC from 21 European countries, while the mortality increased correlating with the organ dysfunction severity, i.e. 22.1 % in ACLF 1, 32 % in ACLF 2, 76.7 % in ACLF 3), while it was 4.7 % in the DLC group [7]. Mortality in ACLF was associated with the multi-organ failure, thus transferring patients to the high-risk group. The isolation of this patient group is possible thanks to the timely use of standard scales, which is currently not implemented into routine practice. Besides, limited therapeutic capabilities mostly aimed at symptomatic treatment also promote poor outcomes.

In our study we used scales to evaluate the condition of patients with LC (CTP, MELD, MELD-Na), as well as those evaluating the organ dysfunction (CLIF-SOFA, CLIF-C OFs, CLIF-C ACLF).

Thus, the CANONIC trial helped to develop an organ dysfunction assessment scale CLIF-SOFA [7]. In 2014 Jalan R. et al. showed that the accuracy of the CLIF-C OF simplified scale is similar to that of CLIF-SOFA in predicting the mortality among patients with LC [15]. The CLIF-C ACLF advantage is that the white blood cell count is also checked to assess the level of inflammation. According to multiple studies, CLIF-SOFA, CLIF-C OF, and CLIF-C ACLF are superior to CTP, MELD, and MELD-Na in the accuracy of predicting short-term and long-term mortality among patients with ACLF [7, 15, 25].

Using CLIF-C OFs, CLIF-SOFA scales helps to define a patient group with a high mortality risk.

MELD and SOFA demonstrated their efficacy in the assessment of the patient condition concerning the mortality prediction. However, one should note that each scale has its own limitations and should be used in conjunction with clinical data and individual patient features.

The comparative analysis of patients by the 28-day outcomes (Table 6) has provided significant evidence of the multi-organ failure severity and mortality risk factors in our cohort, which is confirmed by significantly higher scores in all scales assessing the multi-organ failure (SOFA, CLIF-SOFA, CLIF-C OFs, CLIF-C ACLF; $p<0.05$ for all scores).

Conclusion

Our study demonstrated a high rate of ACLF (47.4 %) in patients with LC, which exceeded parameters recorded in the European population (35 %). A combination of provoking factors prevailed among the main triggers leading to ACLF, while infections were the main cause of decompensated LC, which underscores the importance of their prevention, early screening, and timely treatment.

Patients with ACLF were characterized by significantly more severe hepatic failure, renal dysfunction, systemic inflammation, and higher scores in all predictive scales.

In our study, the use of CLIC-C OF as a key tool for ACLF assessment and determining the mortality risk has demonstrated a high predictive value, helping not only to differentiate patients with decompensation and ACLF ($p<0.001$), but also to detect differences between ACLF 1-3. This scale use in clinical practice will promote the timely patient referral to the specialized treatment, including liver transplant centers.

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Contribution of Authors

All the authors made a significant contribution to the preparation of the work, read and approved the final version of the article before publication

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Ilchenko L.Yu.: concept and design of the study, text editing, approval of the final text of the article

Fedorov I.G.: data collection, literature review

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