Decompensated liver cirrhosis: assessment of complications and mortality in hospitalised patients


Abstract. Background. Liver cirrhosis is a severe, progressively fatal disease if untreated. Hospitalised patients face high mortality rates, and current methods for assessing prognosis vary widely. The research aims to investigate complications and predictors of mortality in patients admitted for decompensated cirrhosis to a tertiary care centre in Tirana, Albania. Materials and methods. The retrospective study included 212 patients aged (58.67 ± 10.09) years: 174 (82.1 %) men, 38 (17.9 %) women. The Child-Turcotte-Pugh, MELD, MELD-Na, MELD 3.0, iMELD, Meso, and UKELD scales were used to assess the severity of the condition and risk stratification of patients. The number of patients with a fatal outcome was 43 (20.3 %). Results. Among participants with different etiological factors of liver cirrhosis, the mortality rate did not differ significantly (p = 0.873). The presence of hepatic encephalopathy (0.43; p = 0.001), acute-on-chronic liver failure (r = 0.47; p = 0.001) and hepatorenal syndrome (r = 0.49; p = 0.001) and, to a lesser extent, ascites (r = 0.18; p = 0.006) and spontaneous bacterial peritonitis (r = 0.23; p = 0.041) was a marker of unfavourable prognosis of hospitalisation. Also, the risk of death increased in the presence of leukaemia (hazard ratio = 4.21 (1.65; 10.74); p = 0.003). Conclusions. The MELD 3.0 and MELD-Na scores, calculated based on laboratory values obtained within 48–72 hours of hospitalisation, were found to be the prognostically significant (p < 0.05).

Keywords: hepatology; mortality; Child-Turcotte-Pugh score; MELD score; liver failure; liver fibrosis

Introduction

As defined by the European Association for the Study of the Liver [1], liver cirrhosis is a slowly progressive disease, the clinical manifestations of which are initially minimal and then intensify due to an increase in portal hypertension and a decrease in liver function. As the pathology progresses, decompensation of the condition occurs, accompanied by a specific clinical picture. The most common clinical manifestations are ascites, bleeding from the upper gastrointestinal tract, encephalopathy, and jaundice. Further progression of liver cirrhosis leads to the need for liver transplantation and, in its absence, death. Decompensated liver cirrhosis is a common cause of emergency admissions to intensive care units. The management of these patients is complex, and mortality is high [2].

According to the World Health Organization (WHO), in 2010, the number of deaths from liver cirrhosis was 1.3 % of the total, the mortality was estimated at 8.3 per 100,000 people. The initial conditions for the formation of liver cirrhosis in Albanian residents are the high prevalence of hepatitis B virus in the 1990s [3, 4]. In 2005, the role of viral hepatitis as a cause of chronic liver disease slightly decreased compared to 1995, but the number of alcoholic liver diseases increased significantly, from 35 % in 1995 to 57 % in 2005 [5].

About 80 % of people with newly diagnosed hepatocellular carcinoma have a history of liver cirrhosis [1]. According to the results of the study by B. Resuli and A. Sallaku [6], the number of patients with hepatocellular carcinoma in Albania was almost doubled between 2000 and 2014, with more than half of the cases being associated with hepatitis B virus and about a third with excessive alcohol consumption. Thus, the study of liver cirrhosis and the search for optimal ways to stratify risks in these patients is an urgent issue.

In 2012, B. Kraja et al. [7] confirmed the prognostic significance of the model for end-stage liver disease (MELD) for predicting the development of spontaneous bacterial peritonitis and death in Albanian residents hospitalised for cirrhosis. The most common causes of cirrhosis in the participants of
this study were alcohol consumption and, to a lesser extent, hepatitis B virus. In 2017, B. Kraja et al. [8] reported that it’s optimal to determine the risk of bleeding from oesophageal varices, the FIB-4 index, which considers the patient’s age, alanine aminotransferase and aspartate aminotransferase activity, and platelet count. In 2023, L. Cuko et al. [9] found that low platelet and prothrombin counts, and high bilirubin and creatinine levels were predictors of spontaneous bacteraemic peritonitis in hospitalised patients with decompensated liver cirrhosis, and hepatic encephalopathy, hepatorenal syndrome, gastrointestinal bleeding, and jaundice were the most common concomitant complications in these patients.

Thus, although liver cirrhosis is widely studied in Albania, there is a need for further research into the prognosis of decompensated liver cirrhosis in hospitalised patients, especially given the dynamic changes in the aetiology and, accordingly, the course of this pathology. The research aims to predict the course of liver cirrhosis in patients hospitalised for decompensation of the disease, considering complications and clinical and laboratory parameters.

Materials and methods

This research is the result of a retrospective observational study conducted at the University Hospital Centre “Mother Teresa” (Tirana, Albania), in particular, in the conditions of the therapeutic department and the gastro-hepatology service. The study analysed the medical records of patients who were admitted to the intensive care unit or gastro-hepatology service for decompensated liver cirrhosis between January 2022 and April 2023.

The criteria for inclusion in the study were as follows: the presence of liver cirrhosis in the stage of decompensation, regardless of the aetiology of the disease; age over 18 years; written informed consent to participate in the study. The exclusion criteria were as follows: liver cirrhosis in the compensatory stage; hepatocellular carcinoma; portal hypertension caused by etiological factors unrelated to liver cirrhosis; significant deficiencies in the completion of the patient’s medical records identified during the analysis.

Based on the data presented in the medical records, the demographic characteristics of the study participants (gender, age), medical history (aetiology and duration of the disease, treatment received by the patients at the time of hospitalisation), life history (alcohol consumption, significant comorbidities, exposure to harmful factors at work, etc.) and the objective status of patients at the time of hospitalisation, including the presence of liver cirrhosis complications, were analysed. The results of instrumental examination such as abdominal ultrasound and gastroscopy were also considered. Laboratory tests were as follows: a complete blood count, including platelet count, liver complex, coagulation test, including prothrombin index and international normalised ratio (INR), electrolytes, as well as serological tests for hepatitis B and C viruses, cytological and bacterial tests of ascitic fluid. The severity of liver cirrhosis and the prognosis of the disease were determined using the Child-Turcotte-Pugh (CTP) score and the MELD index. All of the above clinical and laboratory characteristics of patients were assessed twice: during the first day of hospitalisation and between the next 48 and 72 hours.

The endpoints evaluated in the study were complications of decompensated cirrhosis and hospital outcomes (discharge or death). The minimum number of patients to be included in the study to ensure representativeness was calculated during the study planning. The Cochrane formula for relative values was used for this purpose. The minimum number of study participants was determined to be 148. Quantitative continuous variables were expressed as arithmetic mean ± standard deviation; categorical variables were expressed in absolute and relative terms; when calculating the hazard ratio (HR), the lower and upper confidence intervals (95 %) were also calculated and indicated in parentheses. Statistical analyses were performed using the Statistical Package for the Social Sciences, version 25. The following statistical methods were used: χ² test, Pearson correlation, multiple logistic regression analysis (Cox regression), and receiver operator characteristic (ROC) analysis. The results were considered statistically significant at p < 0.05. Analytical data were visualised using Microsoft Excel.

Results

The study analysed the medical records of 212 patients. This number of study participants is higher than the minimum number (148) calculated using the Cochrane formula, which indicates that the sample is sufficiently representative. Most people included in the study were men, namely 174 (82.1 %), and only 38 (17.9 %) were women (the ratio of men to women was 4.5 : 1). The minimum age of the study participants was 30 years, the maximum was 80 years, with a mean age in the sample being (58.67 ± 10.09) years. The age of most patients (146 (68.8 %)) was between 50 and 70 years. A detailed description of the distribution of patients by age is shown in Fig. 1.

All patients included in the study had a CTP score of 7 or more, indicating decompensated cirrhosis. In 85 (40.1 %) cases, the disease was classified as class B on the CTP scale (7–9 points, a relatively better prognosis). In 127 (59.9 %) patients, the disease was more severe with a worse prognosis, which was equal to class C on the CTP scale (10–15 points). The severity of liver cirrhosis according to the CTP scale was determined by the researchers during the analysis of medical records based on clinical and laboratory data presented in the medical history. In all cases, the diagnosis made by the researchers coincided with the diagnosis made by the attending physician during hospitalisation. The absence of discrepancies in the formulation of diagnoses indicates the absence of
bias in the researchers, as well as the high qualification level of the medical centre’s doctors. On average, hospitalisation lasted (13.49 ± 9.55) days.

The clinical pattern of the disease at the time of hospitalisation varied widely. The most common complaint in all 212 (100.0 %) patients was generalised weakness, which was also described by them as excessive fatigue and exhaustion. A feeling of discomfort, distension or bloating was reported in 170 (80.2 %) cases, while tension ascites requiring laparocentesis was diagnosed in 171 (80.7 %) patients on objective examination. Jaundice was characteristic of 116 (54.7 %) cases. 77 (36.3 %) patients complained of oedema of various localisations. 70 (33.3 %) people had nausea, which was also accompanied by vomiting.

Almost half of the study participants (107 (50.5 %) people) had significant comorbidities, which was reflected in their complaints. The most common medical conditions in patients were hypertension (46 (21.7 %) cases) and type 2 diabetes mellitus (48 (22.6 %) cases). Accordingly, during hospitalisation, patients complained of high blood pressure, palpitations, dry mouth, frequent urination, and dizziness. Patients with comorbidities, especially if they had more than one comorbidity, had a more severe general condition at the time of hospitalisation, which also harmed their prognosis.

The most common etiological factor of liver cirrhosis was excessive alcohol consumption and alcoholic liver disease that developed in this regard: alcohol was recognised as the cause of the disease in 131 (61.8 %) cases. Infections without concomitant alcoholic liver disease were the cause of cirrhosis in 38 (17.9 %) patients, including 11 (5.2 %) patients with viral hepatitis B and 15 (7.1 %) with viral hepatitis C. Viral hepatitis B and C in these cases were diagnosed earlier and confirmed that are typical for haematological and biochemical parameters of patients with decompensated liver cirrhosis. In 27 people, there was a combination of alcohol and infections: in 24 (11.3 %), alcoholic liver disease was accompanied by viral hepatitis B, and in 3 (1.4 %) cases, by viral hepatitis C.

The number of patients with a fatal outcome in this study was 43 (20.3 %). Among those who died, the period from hospitalisation to death was (35.6 ± 2.9) days. The age and gender of patients who died did not differ statistically from those who were discharged from the hospital due to stabilisation (p > 0.05).

To determine whether a particular aetiology of cirrhosis is associated with a poor prognosis, a statistical analysis was performed using the χ² method. It was found that the incidence of deaths among patients with cirrhosis of alcoholic, infectious (associated with hepatitis B and C), autoimmune and cryptogenic origin did not differ (p = 0.873).

Complications of liver cirrhosis were diagnosed in 197 (92.9 %) patients. Bleeding from oesophageal varices was the most common (81 (38.2 %)). Acute-on-chronic liver failure (ACLF) was almost as common among the study participants, which, according to the Asia Pacific Association for the Study of the Liver, is a case of acute liver failure that develops in the setting of chronic liver damage and is usually manifested by coagulopathy and jaundice [10]. ACLF was observed in 77 (36.3 %) patients. Hepatic encephalopathy occurred in 64 (30.2 %) cases, and spontaneous bacterial peritonitis in 48 (22.6 %). Hepatorenal syndrome and re-active pleurisy were the least common: 26 (13.2 %) and 12 (5.7 %) cases, respectively.

Pearson correlation analysis was used to determine the impact of cirrhosis complications on the lethal outcome during hospitalisation. It was found that complications such as bleeding from oesophageal varices and pleural effusion during hospitalisation were not associated with an increased mortality rate (p = 0.21 and p = 0.25, respectively). However, for other complications, the associations were confirmed. In particular, hepatic encephalopathy (r = 0.43, p = 0.001), ACLF (r = 0.47, p = 0.001) and hepatorenal syndrome (r = 0.49, p = 0.001) had a positive medium-strength association with mortality in cirrhosis. Conditions such as ascites and spontaneous bacterial peritonitis were characterised by a positive correlation of low strength (r = 0.18, p = 0.006 and r = 0.23, p = 0.041, respectively). It was also found that the presence of more than one complication in a patient with cirrhosis is a negative prognostic sign (p < 0.05).

When analysing the laboratory data presented in the medical records, certain common patterns were identified that are typical for haematological and biochemical parameters of patients with decompensated liver cirrhosis. In particular, pancytopenia, decreased serum sodium concentration, increased total bilirubin, decreased serum concentration of total protein, albumin and prothrombin, and increased urea and creatinine levels were characteristic of the liver study participants. To determine whether there was a relationship between laboratory values and disease outcomes, all laboratory data presented in the medical records were systematised and analysed statistically using multiple analysis methods (Cox regression): haematological parameters (red blood cell (RBC) and white blood cell (WBC) counts, haemoglobin concentration, haematocrit level, red cell distribution width (RDW), absolute and relative numbers of neutrophils, lymphocytes, monocytes, eosinophils, and basophils), biochemical parameters (total and direct bilirubin, total serum protein and albumin, creatinine, urea), electrolytes (sodium (Na) and potassium (K)), correlogram (prothrombin and INR). The worst laboratory values were selected for statistical analysis. The results of the analysis are presented in Table 1.

Thus, a decrease in the total leucocyte count below the laboratory reference in the blood of patients with decompensated cirrhosis taken during the first 72 hours of hospital stay is the most important predictor of death during hospitalisation. In participants with leukaemia, the likelihood of death can be up to 10 times higher than in patients without it. The prognostic significance of total bilirubin levels is less pronounced, but statistically significant, with an elevation in this parameter increasing the risk of death by about 1.5 times. The remaining parameters did not demonstrate clinical significance as predictors of adverse outcomes, as even with a low probability of statistical error, the HR for these laboratory data did not exceed 1.
A common approach in hepatology is to use standardised scales to determine the risks of an unfavourable prognosis in patients with cirrhosis. In addition to the CTP scale, which was widely used by doctors at the University Hospital Centre “Mother Teresa”, there are other scales with a high level of sensitivity and specificity for prognosis. In particular, these are various modifications of the MELD scale: the classic one, which assesses the levels of creatinine, bilirubin and INR, as well as the presence of haemodialysis in the history; MELD-Na, which additionally evaluates the level of sodium; iMELD, which includes the level of sodium and the patient’s age; MELD 3.0 (updated MELD), which, in addition to creatinine, bilirubin and INR, also includes sodium, albumin and gender. For risk stratification, there is also the MESO index, which is defined as the ratio of the MELD index to the sodium level expressed in mmol/L; and the UKELD score, which is essentially an analogue of the MELD-Na index without assessing the presence of a recent history of haemodialysis.

It is still uncertain which laboratory values are more informative for the calculation of the above indices: the results of tests obtained within the first 24 hours of hospitalisation or those obtained later, within 48–72 hours of hospitalisation, should be considered. To find the optimal period for laboratory tests to determine the prognosis in hospitalised patients with decompensated cirrhosis, a ROC analysis was performed. The indices calculated based on laboratory characteristics of patients on the first day and from the second to third day after hospitalisation were compared. The results of the analysis are presented in Table 2.

### Table 1 — Predictors of mortality in hospitalised patients with decompensated liver cirrhosis

<table>
<thead>
<tr>
<th>Indicator</th>
<th>B</th>
<th>SE</th>
<th>p</th>
<th>HR</th>
<th>95% CI Lower</th>
<th>Upper</th>
</tr>
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<tbody>
<tr>
<td>RBC</td>
<td>0.202</td>
<td>0.231</td>
<td>0.382</td>
<td>1.224</td>
<td>0.778</td>
<td>1.923</td>
</tr>
<tr>
<td>Haemoglobin</td>
<td>–0.411</td>
<td>0.277</td>
<td>0.138</td>
<td>0.663</td>
<td>0.385</td>
<td>1.142</td>
</tr>
<tr>
<td>Haematocrit</td>
<td>0.108</td>
<td>0.093</td>
<td>0.243</td>
<td>1.114</td>
<td>0.929</td>
<td>1.336</td>
</tr>
<tr>
<td>WBC</td>
<td>1.438</td>
<td>0.478</td>
<td>0.003</td>
<td>4.212</td>
<td>1.651</td>
<td>10.744</td>
</tr>
<tr>
<td>RDW</td>
<td>0.037</td>
<td>0.109</td>
<td>0.73</td>
<td>1.038</td>
<td>0.839</td>
<td>1.285</td>
</tr>
<tr>
<td>Neutrophils, abs.</td>
<td>–1.694</td>
<td>0.566</td>
<td>0.003</td>
<td>0.184</td>
<td>0.061</td>
<td>0.557</td>
</tr>
<tr>
<td>Neutrophils, %</td>
<td>0.064</td>
<td>0.055</td>
<td>0.244</td>
<td>1.066</td>
<td>0.957</td>
<td>1.187</td>
</tr>
<tr>
<td>Lymphocytes, abs.</td>
<td>–0.164</td>
<td>0.469</td>
<td>0.726</td>
<td>0.848</td>
<td>0.339</td>
<td>2.126</td>
</tr>
<tr>
<td>Monocytes, %</td>
<td>–0.08</td>
<td>0.068</td>
<td>0.359</td>
<td>0.923</td>
<td>0.777</td>
<td>1.096</td>
</tr>
<tr>
<td>Eosinophils, abs.</td>
<td>–9.183</td>
<td>3.927</td>
<td>0.19</td>
<td>0</td>
<td>0</td>
<td>0.226</td>
</tr>
<tr>
<td>Eosinophils, %</td>
<td>0.551</td>
<td>0.27</td>
<td>0.041</td>
<td>1.735</td>
<td>1.022</td>
<td>2.944</td>
</tr>
<tr>
<td>Basophils, abs.</td>
<td>9.118</td>
<td>8.676</td>
<td>0.293</td>
<td>9115.109</td>
<td>0</td>
<td>2.212</td>
</tr>
<tr>
<td>Basophils, %</td>
<td>–2.485</td>
<td>1.381</td>
<td>0.072</td>
<td>0.083</td>
<td>0.006</td>
<td>1.248</td>
</tr>
<tr>
<td>Total bilirubin</td>
<td>0.267</td>
<td>0.12</td>
<td>0.026</td>
<td>1.306</td>
<td>1.032</td>
<td>1.653</td>
</tr>
<tr>
<td>Direct bilirubin</td>
<td>–0.31</td>
<td>0.171</td>
<td>0.069</td>
<td>0.734</td>
<td>0.525</td>
<td>1.025</td>
</tr>
<tr>
<td>Albumin</td>
<td>–0.102</td>
<td>0.413</td>
<td>0.805</td>
<td>0.903</td>
<td>0.402</td>
<td>2.031</td>
</tr>
<tr>
<td>Total protein</td>
<td>–0.01</td>
<td>0.308</td>
<td>0.974</td>
<td>0.99</td>
<td>0.541</td>
<td>1.812</td>
</tr>
<tr>
<td>Urea</td>
<td>0.018</td>
<td>0.007</td>
<td>0.006</td>
<td>1.018</td>
<td>1.005</td>
<td>1.031</td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.199</td>
<td>0.238</td>
<td>0.404</td>
<td>1.22</td>
<td>0.765</td>
<td>1.947</td>
</tr>
<tr>
<td>Na</td>
<td>–0.107</td>
<td>0.046</td>
<td>0.02</td>
<td>0.898</td>
<td>0.821</td>
<td>0.983</td>
</tr>
<tr>
<td>K</td>
<td>–0.412</td>
<td>0.291</td>
<td>0.157</td>
<td>0.663</td>
<td>0.375</td>
<td>1.171</td>
</tr>
<tr>
<td>Prothrombin</td>
<td>–0.076</td>
<td>0.04</td>
<td>0.057</td>
<td>0.927</td>
<td>0.857</td>
<td>1.002</td>
</tr>
<tr>
<td>INR</td>
<td>–0.832</td>
<td>0.834</td>
<td>0.318</td>
<td>0.435</td>
<td>0.085</td>
<td>2.229</td>
</tr>
</tbody>
</table>

### Table 2 — Comparison of the informativeness of prognostic models obtained within 24 and 48–72 hours of hospitalisation

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Cut-off</th>
<th>Sensitivity, %</th>
<th>Specificity, %</th>
<th>AUROC</th>
<th>95% CI Lower</th>
<th>Upper</th>
</tr>
</thead>
<tbody>
<tr>
<td>CTP score</td>
<td>10.5</td>
<td>83.7</td>
<td>74</td>
<td>0.859 ± 0.030</td>
<td>0.799–0.919</td>
<td></td>
</tr>
<tr>
<td>MELD score</td>
<td>18.5</td>
<td>86</td>
<td>65.1</td>
<td>0.877 ± 0.028</td>
<td>0.821–0.932</td>
<td></td>
</tr>
<tr>
<td>MELD-Na</td>
<td>24</td>
<td>81.4</td>
<td>80.5</td>
<td>0.889 ± 0.026</td>
<td>0.837–0.940</td>
<td></td>
</tr>
<tr>
<td>MESO</td>
<td>15.75</td>
<td>81.4</td>
<td>79.3</td>
<td>0.884 ± 0.027</td>
<td>0.831–0.936</td>
<td></td>
</tr>
<tr>
<td>iMELD</td>
<td>44.85</td>
<td>79.1</td>
<td>79.9</td>
<td>0.874 ± 0.028</td>
<td>0.820–0.928</td>
<td></td>
</tr>
<tr>
<td>UKELD</td>
<td>54.85</td>
<td>74.4</td>
<td>71.6</td>
<td>0.841 ± 0.031</td>
<td>0.780–0.903</td>
<td></td>
</tr>
<tr>
<td>MELD 3.0</td>
<td>23.5</td>
<td>76.7</td>
<td>75.7</td>
<td>0.887 ± 0.024</td>
<td>0.839–0.935</td>
<td></td>
</tr>
</tbody>
</table>
Higher values of area under the ROC curve (AUROC) were found when comparing the prognostic models (MELD 3.0, MELD-Na) during the first 24 and 48–72 hours of the follow-up period (p > 0.05). When comparing each of the prognostic models in two different time intervals, a significant increase in AUROC for all models was found over the next 48–72 hours. Thus, for the most accurate prediction of the outcome of hospitalisation in patients with decompen-
sated cirrhosis, the MELD 3.0 and MELD-Na scores calculated based on laboratory values obtained during 48–72 hours of hospital stay should be used. If it is not possible to use the MELD 3.0 and MELD-Na scales for any reason (lack of or invalid laboratory data, etc.), it is acceptable to use scales other than the original MELD score, as the results of ROC analysis indicate sufficient sensitivity and specificity. The specificity of the original MELD score is rather low, which can lead to a high number of false-positive results.

Discussion

The demographic and etiological characteristics of the patients included in this study are comparable to the large-scale CANONIC (2010–2011) and PREDICT (2017–2018) studies of liver cirrhosis, which involved a total of about 3,000 participants. This confirms the representativeness of this study [11, 12].

The study results reflect the dynamic changes in the etiology of liver cirrhosis that have occurred in Western countries over the past few decades. In particular, the prevalence of liver cirrhosis caused by viral hepatitis has significantly decreased. According to S.L. Friedman and M. Pinzani [13], the main reasons are the introduction of mass vaccination against hepatitis B and the development of effective antiviral treatment, which is widely used for hepatitis B and C. It is also worth noting the effectiveness of government strategic measures to prevent the spread of sexually transmitted and blood-borne infections. Accordingly, in this study, only one-third of patients had virus-induced cirrhosis.

M. Roerecke et al. [14] emphasise the growing importance of excessive alcohol consumption in the development of liver cirrhosis. The study found that women have a higher risk of developing alcoholic cirrhosis compared to men, even when drinking less alcohol. Scientists note the influence of other factors such as genetic predisposition, weight, and the presence of metabolic disorders. S.L. Friedman and M. Pinzani [13] also emphasise the important role of metabolic disorders and non-alcoholic fatty liver disease in the development of cirrhosis. However, patients with non-alcoholic fatty liver disease were not included in this study, which may indicate both insufficient alertness of physicians to this pathology and its relatively low prevalence in the Albanian population.

P.L. Wang et al. [15] points out that compensated liver cirrhosis is rarely the direct cause of death in patients. In this cohort, concomitant cardiovascular disease and non-liver-related cancers are more common causes of death. However, the convincing epidemiological data published by D.Q. Huang et al. [16] show that the proportion of cases of decompensated cirrhosis is increasing, while the share of compensated cirrhosis is decreasing. The mortality rate in decompensated cirrhosis depends on the severity of the disease and varies from 1 to 57%. This necessitates more active detection of the disease in the early stages. D.L. Shawcross et al. [17] believe that primary care specialists must pay much more attention to the diagnosis of liver disease. In particular, family physicians should identify the initial signs of hepatic encephalopathy in their patients. The present study did not analyse whether there was an association between the poor prognosis of cirrhosis and the timeliness of diagnosis, as this information was not available in the patient’s medical records.

According to G. Sebastiani et al. [18], cirrhosis caused by the hepatitis C virus has the most unfavourable course and leads to death most often compared to other etiological factors, but in this study, the aetiology did not affect the prognosis of the disease. The relationship between the presence of complications and mortality in patients with decompensated cirrhosis is currently being widely studied. M.S. Garcia et al. [19] emphasise the prognostic significance of such complications as ACLF, which is confirmed by this study. Rapid deterioration of the patient’s condition in a short period in the absence of treatment leads to multiorgan failure and death, and therefore requires careful monitoring and constant correction of prescriptions by medical staff.

One of the possible ways to improve the quality of medical care is using machine learning to determine the most optimal approaches to therapy. This is what M.S. Garcia et al. [19] demonstrate in their study.

A few years ago, bleeding from the upper gastrointestinal tract in patients with cirrhosis was a serious predictor of poor prognosis, but the situation has changed in recent years. C.R.A. Lesmana et al. [20] provided a detailed analysis of strategies for managing oesophageal varices that have reduced mortality rates. First of all, it is the introduction of effective screening. Although oesophagogastroscope is still considered the gold standard for diagnosis, non-invasive methods can be used in resource-limited settings, including ultrasound assessment of the spleen diameter, platelet count, and measurement of liver stiffness using FibroScan*. Non-selective beta-blockers should be used as primary prevention, although these drugs have certain limitations in patients with decompensated cirrhosis. Last, but not least, is the availability of endoscopic treatments and radiotherapy interventions. All these factors have led to the effective treatment of bleeding from oesophageal varices in patients with liver cirrhosis, which was reflected in the results of this study.

Following K. Gupta et al. [21], acute kidney injury in patients with liver cirrhosis can be caused not only by hepatorenal syndrome but also by other mechanisms: toxic effects of medications, concomitant renal disease, or pre-renal azotaemia. Scientists also point to the development of new approaches to the treatment of acute renal failure in such patients: the use of vasopressors (terlipressin, norepinephrine, etc.), the formation of a transjugular intrahepatic portosystemic shunt, and haemodialysis with the additional use of albumin are effective. Further implementation of these techniques in medical practice should reduce mortality caused by hepatorenal syndrome.

In addition to the prognostic scales used in this study, there are other scales for patients with liver damage. In particular, these include Fibrosis 4, FibroTest/FibroSure, AST to platelet ratio index, as well as instrumental diagnostic methods: liver elastography, and standard ultrasound. However, as noted by A. Smith et al. [22], the use of these...
methods has no prognostic value. The scope of their use is to answer the question of whether cirrhosis is present and, if so, whether it is minimal or significant. That is why these indicators were not analysed in this study.

The prognostic value of the CTP scale is significantly lower than that of the MELD-Na scale, as reflected in several recent publications [23–25]. The effectiveness of the MELD 3.0 scale has been the subject of fewer scientific articles, so this study contains significant scientific novelty in this area. A topical issue that is being widely studied is the period of laboratory sample collection for the calculation of indices and risk stratification. According to the findings of L. Fayad et al. [26], when comparing the MELD 3.0, MELD-Na, MESO, and iMELD scales with each other, the accuracy of prediction is significantly improved when using indicators obtained 48 hours after hospitalisation compared to the results of admission tests. The same conclusion was made based on the results of this study.

Among the markers that may have prognostic value, but were not analysed in this study, is sarcopenia. Following a study by X. Zeng et al. [27], skeletal muscle wasting in patients with decompensated liver cirrhosis is associated with an unfavourable prognosis. T.H. Tranah et al. [28] also note the relationship between hyperammonaemia and mortality, which occurs even in patients with compensated cirrhosis. In this study, these indicators were not analysed, which may be an area for future research.

Conclusions

The mortality rate in liver cirrhosis during hospitalisation in decompensated patients is high and amounts to almost 21%. The predominance of alcohol-related cirrhosis in study participants (61.3%) indicates the need to develop a strategy to combat alcoholism at the systemic level. Viral hepatitis B and C, detected in about 20% of patients, also require early diagnosis and timely provision of isotropic therapy.

The most common complication of decompensated liver cirrhosis is bleeding from varicose veins of the gastrointestinal tract (36.6% of cases). However, effective approaches to the treatment have been introduced, and the presence of this complication does not increase the likelihood of death. The most dangerous complications of liver cirrhosis at the moment are hepatic encephalopathy, ACLF and hepatorenal syndrome, and to a lesser extent, ascites, and spontaneous bacterial peritonitis. Accordingly, rapid deterioration, sudden onset of jaundice, abdominal pain, uremia, confusion, delirium, and anuria in the emergency department should be considered as symptoms indicating a poor prognosis and requiring a more active approach to patient management.

When assessing laboratory parameters, attention should be paid to the white blood cell count, as the presence of leukaemia significantly worsens the prognosis. However, the other parameters are only relevant in a comprehensive assessment using standardised scales, as no other marker is prognostically relevant alone.

Of all the various indices for risk stratification, the MELD 3.0 and MELD-Na scales provide the most accurate prognosis. MELD 3.0 and MELD-Na are more objective than the CTP scale and also consider the largest number of laboratory parameters, which indicates the importance of a comprehensive assessment of the patient’s condition. To calculate the indices, laboratory values obtained within 48–72 hours of hospitalisation should be used, which is more accurate than tests from the first 24 hours of hospitalisation.

Implementation of this information into practice is a priority for healthcare practitioners while for the scientific community, the direction for future research is to identify optimal treatment strategies to reduce mortality in patients with cirrhosis.

References

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Декомпенсированный цирроз печени: оценка усилений и смертности в госпитализированных пациентов

**Резюме. Актуальность.** Цирроз печени – тяжелое захоронение, которое приводит до смерти, якобы это не ликвидируется. Среди госпитализированных пациентов наивный високий ревень смертности, а сугубо методы оценки прогноза значительно включаются. До следования моря на визионе усиления и предикторов смертности у здоровых, госпитализированных с приводу декомпенсированного цирроза до центра третинной медицинской помощи в Тирания, Альбания. **Материалы и методы.** У ретроспективного исследования было включено 212 пациентов в витом (58,67 ± 10,09) року: 174 (82,1 %) чоловіки та 38 (17,9 %) жінок. Для оцінки тяжкості стану та стратифікації ризик використовували шкали Child-Turcotte-Pugh, MELD, MELD-Na, MELD 3.0, iMELD, MESO, UKELD. Кількість хворих із летальним наслідком становила 43 (20,3 %) особи. **Результати.** Серед пацієнтів із різними епізодами.


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