

## LABORATORY PARAMETERS AND ALBI SCORE FOR ASSESSING SEVERITY OF ALCOHOLIC HEPATITIS IN CIRRHOTIC PATIENTS

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### Abstract

Alcoholic hepatitis (AH) is an acute deterioration of liver and often associated with poor prognosis. The aim of this study was to analyze laboratory parameters and to evaluate the prognostic performance of albumin–bilirubin (ALBI) score for severity stratification in patients with AH and underlying cirrhosis.

This retrospective, single-centre, cross-sectional study included patients with previously undiagnosed alcoholic cirrhosis hospitalized between January 2017 and December 2021 at a tertiary referral centre in Bulgaria. Among 262 patients with alcoholic cirrhosis, 85 fulfilled the NIAAA/AASLD diagnostic criteria for AH. Disease severity was stratified using MELD-Na ( $< 20$  vs.  $\geq 21$ ). Laboratory parameters and prognostic scores were analyzed. Receiver operating characteristic (ROC) curves were constructed to assess the predictive value of ALBI, Child–Pugh score, and total bilirubin for severe AH.

Among patients with AH, 83.5% were classified as ALBI grade 3 and 77.6% as Child–Pugh class C. Severe AH (MELD-Na  $\geq 21$ ) was associated with significantly higher total bilirubin, INR, creatinine, and leukocyte counts, as well as lower serum albumin and sodium levels ( $p < 0.05$ ). ROC analysis demonstrated that an ALBI cut-off of  $-0.675$  showed the highest specificity (94.7%; AUC = 0.806), while total bilirubin  $\geq 101.25$   $\mu\text{mol/L}$  had the highest sensitivity (93.6%; AUC = 0.846). Strong correlations were observed between ALBI and Child–Pugh scores ( $r = 0.883$ ), as well as between ALBI and MELD-Na ( $r = 0.687$ ,  $p < 0.001$ ).

The ALBI score is a simple and objective tool with high specificity for severity assessment in alcoholic hepatitis superimposed on cirrhosis. Its combined use with total bilirubin may improve early risk stratification. Prospective studies are warranted to validate these findings.

**Key words:** ALBI score, Child–Pugh classification, MELD–Na score, hepatic decompensation, alcoholic liver diseases

**Introduction.** Alcoholic hepatitis (AH) represents an acute deterioration of liver function associated with prolonged and excessive alcohol consumption and is considered the most severe clinical form of alcoholic liver disease (ALD) [1]. Its clinical presentation varies widely, ranging from asymptomatic cases to life-threatening liver and multi-organ failure [2]. In 60–80% of cases, AH develops on the background of established liver cirrhosis, which is a key factor contributing to poor prognosis [3]. According to the EASL-CLIF Consortium (European Association for the Study of the Liver – Chronic Liver Failure Consortium), AH is among the three most common causes of cirrhosis decompensation, alongside infections and gastrointestinal bleeding [4]. The true incidence of AH is difficult to establish due to its often oligosymptomatic course and frequent underdiagnosis [5]. In clinical practice, the most commonly used tools for assessing the severity of chronic liver disease are the MELD (Model for End-Stage Liver Disease) and Child–Pugh scores. The ALBI (albumin-bilirubin ratio) score emerges as a promising and convenient tool, although large-scale and systematic studies confirming its role in alcoholic hepatitis superimposed on cirrhosis are still lacking. ALBI is an objective and easy-to-use model that evaluates liver functional status based solely on two laboratory parameters. Originally, the index was developed and validated for assessing liver function in patients with hepatocellular carcinoma [6]. Various studies have demonstrated that ALBI is a reliable prognostic marker for future decompensation [7] and predicted mortality in cirrhosis [8], as well as for assessing the benefits of corticosteroid therapy in severe forms of AH [9]. Additionally, ALBI has been validated as an alternative marker in resource-limited centres for chronic ALD [10]. The present study aims to analyze the laboratory profile of patients with AH superimposed on liver cirrhosis and to assess the degree of hepatic dysfunction using MELD–Na and Child–Pugh scores. Additionally, the study evaluates the applicability of the ALBI score as a complementary tool for severity stratification in this patient population.

**Materials and methods.** This retrospective, single-centre, cross-sectional study was conducted among patients with previously undiagnosed liver cirrhosis who were treated at the University Hospital “Georgi Stranski” in Pleven, Bulgaria between January 1, 2017, and December 31, 2021. Data were extracted from medical records, ensuring strict anonymity and removal of all identifying information, in accordance with the Declaration of Helsinki. Therefore, additional ethical committee approval was not required. A total of 361 cases with clinically confirmed liver cirrhosis were identified in this period. All patients had clinical,

laboratory, and ultrasound findings indicative of advanced liver damage. Of these, 262 (72.6%) had alcoholic aetiology, among whom 85 (32.4%) showed clinical evidence of AH. Histological confirmation of diagnosis was not performed in any of the cases. The diagnosis of AH was established according to the NIAAA/AASLD (National Institute on Alcohol Abuse and Alcoholism/American Association for the Study of Liver Diseases) criteria: onset of jaundice within  $\leq 8$  weeks prior to admission, active heavy alcohol consumption ( $>40$ – $60$  g ethanol daily), abstinence of  $\leq 60$  days before symptom onset, and laboratory parameters (AST  $>$  ALT, both  $< 400$  IU/L; AST/ALT ratio  $> 1.5$ ; total bilirubin  $> 3$  mg/dL or  $51$   $\mu\text{mol/L}$ ), with exclusion of other causes of liver dysfunction [11]. Criteria for acute cirrhosis decompensation were defined following EASL recommendations, including symptom duration  $\leq 4$  weeks and at least one of the following: ascites, jaundice, portal systemic encephalopathy (PSE), coagulopathy, or laboratory markers of liver failure [12]. Exclusion criteria included non-alcoholic cirrhosis, concomitant malignancy, gastrointestinal bleeding, and patients younger than 18 years. The Child–Pugh score was calculated using the point-based system, while MELD–Na and ALBI scores were computed using validated online calculators based on established formulas. Patients were stratified into two subgroups according to MELD–Na severity: MELD–Na  $< 20$  (mild AH) and MELD–Na  $\geq 21$  (severe AH). The ALBI score was calculated using the formula:

$$\text{ALBI} = (\log_{10} \text{bilirubin } [\mu\text{mol/L}] \times 0.66) + (\text{albumin } [\text{g/L}] \times -0.085).$$

Based on ALBI values, cases were classified into three groups: ALBI 1:  $\leq -2.60$  (low risk), ALBI 2:  $> -2.60$  to  $\leq -1.39$  (moderate risk), and ALBI 3:  $> -1.39$  (high risk) [13].

Statistical analysis was performed using IBM SPSS Statistics version 26 (IBM Corp., Armonk, NY, USA) and Microsoft Excel (Microsoft Corp., Redmond, WA, USA). Quantitative variables were presented as mean  $\pm$  standard deviation (SD) and 95% confidence interval (CI). Group comparisons were conducted using the Mann–Whitney U test for non-normally distributed variables. The  $\chi^2$  test and Fisher's exact test were used for categorical variables. Receiver Operating Characteristic (ROC) analysis was employed to determine cut-off values for Child–Pugh, ALBI, and total bilirubin in relation to severe alcoholic hepatitis (defined as MELD–Na  $\geq 21$ ). Correlations among the three scores were examined across the entire cohort. Statistical significance was set at  $p < 0.05$ .

**Results.** Among the 262 patients, 210 (80.15%) were male and 52 (19.85%) were female. At baseline, 94 patients (35.9%) presented with acute decompensation at the time of diagnosis. Of these, 85 (90.4%) had clinical evidence of AH, while the remaining cases presented with other causes of decompensation. Among the AH group, 63 (74.1%) were male and 22 (25.9%) female. Women had a higher proportion of AH at decompensation (42%,  $n = 22$ ) than men (30%,  $n = 63$ ) ( $p = 0.001$ ). Patients with AH were younger than those without, an effect more

pronounced in women ( $p = 0.037$ ). Ascites was common in both groups, but without a significant difference ( $p = 0.316$ ). PSE was more frequent in AH patients (70.6% vs. 52%,  $p = 0.005$ ), and all AH patients had jaundice ( $p = 0.001$ ). Renal dysfunction showed no major difference, though HRS (Hepatorenal Syndrome) with and without AKI (Acute Kidney Injury) was more frequent in AH ( $p = 0.054$ ). Regarding liver disease severity, significantly more AH patients were in Child–Pugh class C (77.6% vs. 22.6%,  $p = 0.001$ ), and 83.5% of AH patients were in ALBI grade 3 ( $p = 0.001$ ), indicating high liver failure risk (Table 1).

Patients with severe AH (MELD-Na  $\geq 21$ ) exhibited significantly higher levels of bilirubin, INR, urea, creatinine, and leukocytes, accompanied by lower serum sodium and albumin levels ( $p < 0.05$ ). Prognostic scores (Child–Pugh, ALBI, MELD-Na) were also significantly elevated. No significant differences were found in AST, ALT, AST/ALT ratio, or ALP, while GGT levels showed borderline statistical significance (Table 2).

ROC curve analysis was used to identify cut-off values for Child–Pugh score, ALBI score, and total bilirubin in detecting severe alcoholic hepatitis. The ALBI

T a b l e 1

Baseline demographic and clinical characteristics in patients with and without AH

Variables	Without AH ( $n = 177$ )	With AH ( $n = 85$ )	$p$ -value
<b>Sex, <math>n</math> (%)</b>			< 0.001
Male	147 (83.1)	63 (74.1)	
Female	30 (16.9)	22 (25.9)	
<b>Age, years (mean <math>\pm</math> SD)</b>			0.037
Male	57.29 $\pm$ 9.99	53.95 $\pm$ 11.49	
Female	56.23 $\pm$ 7.24	48.41 $\pm$ 10.76	
<b>Major complications, <math>n</math> (%)</b>			
Ascites	119 (67.2)	63 (74.1)	0.316
PSE (any grade)	92 (52)	60 (70.6)	0.005
Renal dysfunction Total	32 (18.07)	16 (18.82)	0.054
HRS-non AKI	15 (8.5)	10 (11.8)	
HRS-AKI	6 (3.4)	6 (7.1)	
CKD	11 (6.2)	0 (0.0)	
Jaundice	16 (9.03)	85 (100)	<0.001
<b>Child–Pugh class, <math>n</math> (%)</b>			<0.001
Child A	47 (26.6)	2 (2.4)	
Child B	90 (50.8)	17 (20)	
Child C	40 (22.6)	66 (77.6)	
<b>ALBI grade, <math>n</math> (%)</b>			<0.001
ALBI 1	31 (17.5)	2 (2.4)	
ALBI 2	99 (55.9)	12 (14.1)	
ALBI 3	47 (26.6)	71 (83.5)	

**Note:** Data are presented as mean  $\pm$  standard deviation (SD) for continuous variables and as number ( $n$ ) and percentage (%) for categorical variables;  $p$ -values represent the overall difference between the compared groups.

T a b l e 2

Laboratory parameters in mild (MELD-Na &lt; 20) versus severe (MELD-Na ≥ 21) AH

Parameter	MELD Na < 20		MELD Na ≥ 21		p-value
	n = 38 (44.7%)		n = 47 (55.3%)		
	Mean ± SD (95% CI)		Mean ± SD (95% CI)		
AST (U/L)	187.30 ± 251.52	(107.33–276.27)	174.71 ± 103.23	(145.19–204.22)	0.150
ALT (U/L)	72.03 ± 129.02	(31.00–113.05)	65.61 ± 47.05	(52.15–79.06)	0.086
AST/ALT ratio	3.32 ± 1.69	(2.78–3.85)	3.09 ± 1.73	(2.59–3.58)	0.488
GGT (U/L)	1095.68 ± 1138.99	(733.54–1457.82)	668.47 ± 763.58	(450.17–886.77)	0.052
ALP (U/L)	209.48 ± 128.84	(168.51–250.44)	197.19 ± 102.80	(167.80–226.58)	0.895
Albumin (g/L)	29.36 ± 6.45	(27.30–31.41)	25.49 ± 3.71	(24.42–26.55)	0.008
Total bilirubin (μmol/L)	113.40 ± 77.96	(88.61–138.18)	223.44 ± 119.79	(189.19–257.68)	< 0.001
Direct bilirubin (μmol/L)	90.98 ± 76.52	(66.65–115.30)	176.93 ± 97.03	(149.19–204.67)	< 0.001
Serum sodium (mmol/L)	137.82 ± 3.74	(136.63–139.00)	131.79 ± 5.73	(130.15–133.42)	< 0.001
INR	1.36 ± 0.313	(1.26–1.46)	1.77 ± 0.45	(1.64–1.89)	< 0.001
Blood urea (mmol/L)	4.15 ± 2.81	(3.25–5.04)	9.48 ± 9.71	(6.70–12.56)	0.002
Creatinine (μmol/L)	71.22 ± 50.96	(55.01–87.42)	139.57 ± 141.73	(99.05–180.08)	0.009
Hb (g/L)	110.53 ± 19.51	(104.32–116.73)	104.06 ± 22.82	(97.53–110.58)	0.233
RBC × 10 <sup>12</sup> /L	3.24 ± 0.62	(3.04–3.43)	2.99 ± 0.63	(2.81–3.17)	0.084
MCV (fl)	101.17 ± 5.59	(99.39–102.94)	101.96 ± 10.6	(98.93–104.99)	0.234
Platelets count × 10 <sup>9</sup> /L	161.34 ± 84.14	(134.58–188.09)	155.74 ± 83.51	(103.27–208.20)	0.234
WBC × 10 <sup>9</sup> /L	9.57 ± 4.35	(8.18–10.95)	12.86 ± 6.37	(11.03–14.68)	0.004
Child–Pugh point	9.92 ± 2.12	(9.24–10.59)	11.49 ± 1.55	(11.04–11.93)	< 0.001
ALBI score	−1.20 ± 0.514	(−1.36–−1.03)	−0.68 ± 0.323	(−0.77–−0.58)	< 0.001
MELD Na	16.74 ± 2.59	(15.91–17.56)	27.26 ± 4.77	(25.89–28.62)	< 0.001

score demonstrated the highest specificity, whereas total bilirubin showed the highest sensitivity (Table 3).

A significant positive correlation was observed among the three scoring systems, with the strongest association found between the Child–Pugh and ALBI scores ( $r = 0.883$ ;  $\rho = 0.880$ ;  $p = 0.001$ ) (Table 4).

**Discussion.** According to published data, approximately one-third of cases of acute liver decompensation in patients with cirrhosis are associated with concomitant AH, which is consistent with our findings[4]. Although the number of women in the studied population was significantly lower, they were more likely to present with AH-related decompensation compared to men. These observations support previous research indicating that the harmful effects of alcohol are more pronounced in women, even at lower levels of consumption. Women also ap-

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Predictive cut-off values for severe alcoholic hepatitis (MELD-Na ≥ 21)

Parameter	Cut-off value	AUC	Std. error <sup>b</sup>	95% CI	Sensitivity	Specificity
ALBI score	−0.675	0.806	0.062	0.685–0.927	55.3%	94.7%
Child–Pugh	11.50 points	0.723	0.056	0.615–0.832	57.0%	76.0%
Total bilirubin	101.25 μmol/L	0.846	0.044	0.760–0.932	93.6%	63.2%

T a b l e 4

Correlation coefficients between prognostic scores

Parameters	Pearson $r$	Spearman $\rho$	$p$ -value
Child–Pugh/ALBI	0.883**	0.880**	< 0.001
Child–Pugh/MELD Na	0.728**	0.736**	< 0.001
ALBI/MELD Na	0.687**	0.619**	< 0.001

**Note:**  $n = 262$  (all cases of alcoholic cirrhosis, regardless of the presence or absence of AH);  $p < 0.01$ \*\*

pear more susceptible to developing severe forms of ALD and experiencing more frequent complications [14]. We observed a higher incidence of concomitant hepatic encephalopathy and ascites among patients with AH. In cases of severe AH (defined as MELD-Na  $\geq 21$ ), significantly elevated levels of bilirubin, INR, urea, creatinine, and leukocytes were recorded, along with significantly reduced levels of sodium and albumin ( $p < 0.05$ ). Prognostic scores, including Child–Pugh, ALBI, and MELD-Na, were also significantly higher in this group. Previous studies have demonstrated that these laboratory and clinical parameters are independently associated with increased 90-day mortality [15]. A decrease in albumin and an increase in bilirubin levels are key indicators of impending liver failure, forming the basis for the development of the ALBI score [6]. A systematic review encompassing 118 studies found that serum levels of these markers are among the most important laboratory predictors of survival in liver cirrhosis [13]. Elevated total bilirubin is a principal laboratory marker for AH. Notably, an increase in the direct fraction of bilirubin serves as a prognostic indicator for six-month survival in cirrhotic patients [16] and helps identify acute-on-chronic liver failure [17]. In our study, we observed a significant increase in the ALBI score among severe forms of AH ( $-0.68 \pm 0.323$ ), confirming ALBI's utility in assessing the severity of liver dysfunction. Values above  $-1.36$  (ALBI grade 3) identify patients at high risk with reduced survival [16]. In our cohort, 83.5% ( $n = 71$ ) of patients with AH were classified as ALBI grade 3. Longitudinal follow-up of patients with ALBI grade 3 indicates a higher likelihood of reduced survival [8]. Another study identified an ALBI cut-off of  $-1.01$  with a sensitivity of 94.92% and specificity of 32.5% for increased mortality risk in chronic alcoholic liver disease [10]. Scores above  $-0.6$  are strongly associated with acute-on-chronic liver failure and higher in-hospital mortality [18], while values above  $-1.17$  correlate with more favourable survival outcomes [19]. We found a significant correlation between ALBI and Child–Pugh scores ( $r = 0.880$ ), consistent with previous findings ( $r = 0.853$ ) [20]. Additionally, significant correlations were observed between ALBI and MELD-Na, as well as between Child–Pugh and MELD-Na, which have also been confirmed in patients with variceal bleeding [21].

The present study has several limitations. First, its retrospective and single-centre design may affect the generalisability of the findings. The lack of histologi-

cal confirmation of alcoholic hepatitis is another potential limiting factor, despite the use of established clinical criteria (NIAAA/AASLD). Another limitation of this study is the absence of longitudinal follow-up, which precludes evaluation of survival outcomes or disease progression. Nevertheless, our results emphasize the potential of ALBI score values and serum bilirubin levels to serve as practical and early markers for the initial assessment of liver dysfunction severity in these cases. These findings provide a basis for future prospective studies aimed at validating their prognostic value and evaluating their potential use as alternatives or adjuncts to traditional scoring systems for early clinical assessment.

**Conclusion.** This cross-sectional, retrospective study provides an objective initial evaluation of disease severity in alcoholic hepatitis superimposed on cirrhosis by employing laboratory parameters alongside established scoring systems (MELD-Na and Child–Pugh). The ALBI score demonstrated both a strong correlation with traditional scores and high specificity, whereas total bilirubin proved to be a highly sensitive marker for identifying severe cases. These findings highlight the potential benefit of combining ALBI and bilirubin for rapid and accurate risk stratification.

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