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Biochemical Predictors of Clinical Outcome in Liver Failure Associated with Obstructive Jaundice

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Summary

The study of predictors of adverse outcomes in liver failure is driven by the rapid increase in patients with obstructive jaundice (OJ) and the lack of standardized diagnostic criteria for assessing liver functional status.

Aim. To investigate the changes of liver injury biomarkers in liver failure associated with OJ.

Materials and Methods. A prospective observational cohort study was conducted on serum biomarkers of liver injury — L-FABP protein, 5'-nucleotidase, liver arginase, and hyaluronic acid — in patients with liver failure due to benign OJ. The study included 53 patients who underwent biliary decompression. Based on the course of disease, patients were divided into two groups: those with favorable outcomes (group 1, N=27) and those with unfavorable outcomes (group 2, N=26). A control group consisted of 25 healthy donors. Serum biomarker levels were assessed on admission and on days 3, 7 and 11 post-decompression. The study used enzyme-linked immunosorbent assay (ELISA). Statistical analysis was performed using IBM SPSS Statistics 22, including Friedman two-way analysis, Kruskal–Wallis H test, Mann–Whitney U test, and two-sample Kolmogorov–Smirnov test, with significance set at $P<0.05$.

Results. At hospital admission, median biomarker levels were significantly higher in both patient groups than in the comparison group. Group 1 showed a statistically significant decrease in all biomarkers during treatment (P=0.01 for L-FABP, 5'-nucleotidase, liver arginase; P=0.03 for hyaluronic acid). In group 2, only L-FABP levels decreased significantly (P=0.04). Sensitivity and specificity for predicting disease outcome were 89.2–92.3% and 88.9–96.3% for L-FABP, 53.8–69.2% and 81.5–85.2% for 5'-nucleotidase, 57.7–76.9% and 77.8–88.9% for arginase, and 38.5–46.2% and 74.1–81.5% for hyaluronic acid, respectively.

Conclusion. Among the studied biomarkers, L-FABP showed the highest specificity and sensitivity values for prediction of outcome in liver failure associated with OJ, while other biomarkers demonstrated less significant results.

Keywords: obstructive jaundice; liver failure; biomarkers of liver injury; L-FABP; 5'-nucleotidase; liver arginase; hyaluronic acid

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Introduction

In recent years, there has been a marked increase in the number of patients diagnosed with obstructive jaundice (OJ) (ranging from 12% to 25.2%) and liver failure (LF) associated with diseases of the hepatobiliary and pancreatic region [1, 2]. The etiologic spectrum of OJ includes choledocholithiasis in 50% of cases, tumors of the bile ducts, greater duodenal papilla, pancreas, and gallbladder in 40%, and stenosis of the greater duodenal papilla, biliary strictures or atresia, cholangitis, pancreatitis, and hepatic neoplasms in the remaining 10% [1–3]. The initial severity of OJ and the subsequent development of LF are important determinants of mortality, which can reach 20–40% [2, 4]. Endogenous intoxication and liver failure are the leading causes of death in patients with this pathology [1, 4, 5].

Liver dysfunction in the setting of OJ almost invariably leads to the development and progression of LF, although early diagnosis remains challenging. However, the extent of liver dysfunction plays a critical role in determining the outcomes of patients with OJ [1, 2].

Current diagnostic criteria for LF in the context of OJ are based on clinical data assessing the intensity and duration of jaundice, as well as laboratory and instrumental studies. Numerous prognostic scoring systems and assessment tools for hepatocellular dysfunction in various pathologies focus primarily on changes in biochemical markers such as bilirubin fractions, alkaline phosphatase, alanine aminotransferase, aspartate aminotransferase, γ-glutamyltransferase, and lactate dehydrogenase [6-8]. However, several studies have shown that these criteria do not always accurately reflect the severity of liver failure and often provide only indirect or approximate assessments. This highlights the need for additional objective diagnostic criteria to complement standard approaches in patients with OJ [9].

A number of researchers have emphasized the critical importance of identifying and applying biological markers of liver injury that can be used at different stages of the disease [10]. A prognostically relevant marker should have high anatomical specificity, diagnostic accuracy, sensitivity, predictive value for clinical outcome, and the ability for dynamic monitoring [11, 12].

In particular, biomarkers such as liver-type fatty acid binding protein (L-FABP), 5'-nucleotidase (5-NT), hepatic arginase, and hyaluronic acid (HA) are considered promising preclinical indicators that reflect the development of hepatic decompensation. These markers provide valuable insight into key pathomorphological changes within the liver parenchyma and demonstrate adequate topographic specificity, sensitivity, and diagnostic accuracy [11, 12].

However, despite the significant number of potential biological markers under investigation in liver failure, their application in the context of obstructive jaundice remains controversial. Their clinical utility requires further validation through accumulated clinical experience and large-scale studies.

The aim of the study was to investigate the changes in liver injury biomarkers in patients with different outcomes of liver failure associated with obstructive jaundice.

Materials and Methods

An observational prospective cohort study was conducted to quantitatively assess serum levels of liver injury biomarkers — liver-type fatty acid-binding protein (L-FABP), 5'-nucleotidase (5-NT), hepatic arginase, and hyaluronic acid (HA) — in patients with LF associated with benign OJ. Biomarker levels were measured by enzyme-linked immunosorbent assay (ELISA).

The study cohort included patients hospitalized at the Department of Anesthesiology and Intensive Care at the Oryol Regional Clinical Hospital (Oryol, Russia) between June 2019 and March 2021. The study was approved by the Ethics Committee of the People's Friendship University of Russia (Protocol No. 14, dated May 21, 2019).

A total of 53 patients aged 35–75 years were enrolled. The cohort consisted of 26 males (49%) and 27 females (51%).

Inclusion criteria:

- age over 18 years;
- moderate or severe liver failure (corresponding to classes B and C according to the classification of E. Galperin et al., 2012) secondary to benign OJ;
- previous biliary decompression.

Exclusion criteria:

- decompensated comorbidities;
- chronic inflammatory liver diseases;
- mild OJ (class A);
- patient refusal to participate;
- surgical complications related to the intervention (massive bleeding, hemorrhagic shock);
- inability to assess the study variables.

Depending on the clinical course and outcome of the disease, patients were divided into two groups:

- Group 1 — patients with favorable outcome (those who achieved clinical stabilization and were discharged from the hospital, *N*=27)
- Group 2 — patients with unfavorable outcome (those who did not achieve clinical stabilization and died during hospitalization, *N*=26) (Table 1).

The mortality structure in the second group was as follows: in 15% of cases (*N*=4) the adverse outcome occurred in the immediate postoperative period (the first 5 days after surgery), while in 85% of cases (*N*=22) it occurred in the early postoperative period (from the 5th to the 21st day after surgery).

Table 1. Characteristics of patients in the study groups, *N* (%) or *Me* [IQR].

Parameter	Values in groups		*P value*
	Group 1, *N*=27	Group 2, *N*=26	
Age, years (minimal-maximal)	63.5 (37–85)	61.9 (35–88)	0.2
Male/female, *N* (%)	14/13 (51.9/48.1)	12/14 (48.3/51.7)	>0.05
SOFA score, points	7.4 [4–9]	8.8 [6–10]	>0.05
APACHE II score, points	20.1 [9–32]	21.8 [12–32]	>0.05

Note. IQR — interquartile range.

Pathophysiological and morphological abnormalities in LF with the underlying cholestasis, despite biliary decompression, triggered local and systemic complications, including coagulopathy, renal dysfunction, and systemic hypotension. As these complications progressed, they led to multiple organ failure and an unfavorable outcome.

In the patients included in the study, cholestasis was caused by benign biliary strictures (5.5%) and cholelithiasis (94.5%).

The diagnosis of «mechanical jaundice syndrome» was made on the basis of clinical and history data in accordance with the clinical guidelines of the Russian Society of Surgeons, approved by the Ministry of Health of the Russian Federation in 2018.

The number of patients with the severity of OJ corresponding to class B (moderate) was 23 (43.4%), and class C (severe) was 30 (56.6%).

The severity of LF was assessed according to the classification of V. Fedorov and V. Vishnevsky (2004). In addition, the severity of the patient's condition on admission was assessed using the APACHE II scale. On the day of admission and on the 3rd, 7th, and 11th days after decompressive surgery, MELD, Child-Turcotte-Pugh scores were assessed; the probability of developing multiple organ failure was determined for all patients at the aforementioned time points using the SOFA scale.

Comorbidities were assessed using the Charlson Comorbidity Index (CCI), which revealed 12 (22.6%) patients with ischemic heart disease and chronic heart failure, 5 (9.4%) with peripheral vascular disease, 6 (11.3%) with a history of peptic ulcer disease, 4 (7.5%) with severe bronchopulmonary disease, and 11 (20.8%) with diabetes mellitus. The CCI averaged 7.5±2.4 points in the favorable outcome group and 8.7±1.9 points in the unfavorable outcome group, ranging from 6 to 16 points.

The study groups were comparable with respect to sex ($P>0.05$) and age ($P=0.2$) and showed no statistically significant differences in the main assessment scales at baseline: APACHE II ($P>0.05$), SOFA ($P>0.05$), and CCI.

Patients hospitalized for hyperbilirubinemia in the setting of obstructive cholestasis were treated according to the clinical guidelines of the Russian Society of Surgeons, approved by the Ministry of Health of the Russian Federation in 2018, which include both conservative and surgical strategies.

Conservative therapy addressed the following aspects: pain management, detoxification, resolution of cholestasis consequences, hepatorenal failure, gastrointestinal erosions and acute ulcers, and cholangitis. Treatment included intravenous detoxification therapy, hepatoprotective agents, antibiotics (administered empirically in cases of systemic inflammatory response until bacteriologic results were available, with subsequent adjustments), and adequate nutritional support.

Surgical management followed a staged approach. On the first day of hospitalization, all patients underwent a minimally invasive procedure aimed at retrograde or antegrade biliary decompression to relieve the OJ and restore bile flow to the duodenum or establish an external biliary drainage. In some cases (26.49%), this was the definitive treatment.

In the second stage, after gradual resolution of OJ (assessed by monitoring bilirubin levels) and normalization of organ function, definitive (including radical) surgical intervention was performed (27.51%).

For patients with bile duct stones, the definitive treatment (85% of cases) was endoscopic retrograde transpapillary intervention. When this approach was not feasible or effective (15% of cases), alternative methods were used such as choledocholithotomy via mini-laparotomy, laparoscopic choledocholithotomy, or open choledocholithotomy via laparotomy.

In cases of benign biliary strictures, definitive treatment consisted of endoscopic correction (70%) or reconstructive plastic biliary surgery (30%).

The following reagents were used to quantify biological markers: for L-FABP, HBT L-FABP ELISA (BioKhimMak, Russia); for 5-NT, HBT 5-NT-I ELISA (BioKhimMak, Russia); for arginase, HBT Arginase-I ELISA (BioKhimMak, Russia); and for HA, HBT GK-I ELISA (BioKhimMak, Russia). All assays were performed on an automated microplate immunoanalyzer (Immunomat™). Serum levels of liver injury biomarkers in OJ were measured at hospital admission and on days 3, 7, and 11 of hospitalization.

The control group consisted of 25 healthy volunteers. Their biomarker levels were established as reference values for individuals without liver diseases.

Statistical analysis. Sample size was calculated using PS Power and Sample Size Calculations software, version 3.0.11 for MS Windows. To reject the null hypothesis with 80% power at $\alpha=0.05$, the minimum sample size required was 26 participants per group.

Statistical analysis was performed with IBM SPSS Statistics 22. The significance of differences was tested using nonparametric methods: the Mann–Whitney U test for between-group comparisons, supplemented by the Kolmogorov–Smirnov two-sample test. Null hypotheses were rejected at $P<0.05$.

Multivariable logistic regression with stepwise variable selection was used for predictive modeling. Methods recommended for small sample sizes were also used, including two-factor nonparametric (rank) Friedman's analysis of variance and Kruskal–Wallis H test for nonparametric (rank) one-way analysis of variance. The significance of the regression coefficients was evaluated using the Wald statistic, and model fit was assessed using the Hosmer-Lemeshow test. Model performance was compared

using ROC–AUC analysis. Only sensitivity and specificity were reported as predictive characteristics.

Results

Upon hospital admission, the median serum levels of liver injury biomarkers (L-FABP, arginase, HA, 5-NT) were significantly higher in patients of both groups than in healthy volunteers of the control group. The levels were significantly higher in patients of the second group compared to the first group ($P<0.05$), except for HA ($P=0.05$) (Table 2).

The changes in biomarker levels during the different treatment phases are shown in Table 3.

At all time points after the initial measurement, the concentration of liver injury biomarkers remained significantly higher in group 2 compared to group 1 ($P<0.05$), with the exception of HA levels on days 3 ($P=0.15$) and 7 ($P=0.09$) (Table 3).

In group 1, a statistically significant sequential decrease in the concentration of most biomarkers was observed by day 11 of treatment: L-FABP and 5'-nucleotidase ($P=0.01$) and hyaluronic acid ($P=0.03$). An exception was the increase in hepatic arginase concentration on day 3 compared to baseline ($P=0.01$). However, by day 7, arginase levels had fallen below baseline levels and continued to decline through day 11 ($P=0.01$) (Table 3).

In group 2, only the concentration of L-FABP showed a statistically significant decrease ($P=0.04$). Changes in the levels of the other biomarkers during the study were not significant ($P=0.39$–0.68) (Table 3).

At the final time point (day 11), none of the biomarker levels in either group had decreased to the median reference values. The biomarker concentrations closest to the reference medians were those of L-FABP and arginase in Group 1 (control vs. group 1: 12.90 vs. 13.70 ng/mL; 15.40 vs. 18.50 ng/mL, respectively) (Tables 2 and 3).

Area under the ROC curve (AUC) data for each biomarker over the study period are shown in Table 4.

The predictive performance of the models, in terms of sensitivity and specificity, varied depending on the treatment time point and showed the following characteristics:

- L-FABP: sensitivity ranged from 89.2% to 92.3%, specificity from 88.9% to 96.3%. The cutoff ranged from 21.6 to 40.0 ng/mL.
- Arginase: sensitivity ranged from 57.7% to 76.9%, specificity from 77.8% to 88.9%, with a consistent cutoff of 34.0 ng/mL.
- HA: sensitivity ranged from 38.5% to 46.2%, specificity from 74.1% to 81.5%. The cutoff value varied over a wide range; however, due to the low predictive performance of models based on HA, a reliable cutoff value could not be determined.
- 5-NT: sensitivity ranged from 53.8% to 69.2%, specificity from 81.5% to 85.2%. The empirically estimated cutoff was 34.4 IU/L.

The model with the predictor «L-FABP concentration» demonstrated the best performance

Table 2. Levels of liver injury biomarkers upon hospital admission in the study groups, *Me (Q1–Q3).*

Biomarker	Values of parameters in groups			*P value**
	Control group, *N*=25	Group 1, *N*=27	Group 2, *N*=26	
L-FABP, ng/mL	12.90 (12.55; 13.50)	26.40 (23.30; 34.10)	56.79 (39.09; 71.12)	0.01
Arginase, ng/mL	15.40 (13.60; 16.65)	22.40 (21.40; 28.40)	39.05 (32.85; 50.43)	0.01
Hyaluronic acid, ng/mL	41.0 (22.0; 69.0)	175.0 (86.0; 423.0)	290.5 (148.5; 517.0)	0.05
5'-nucleotidase, IU/L	1.56 (1.56; 1.71)	25.56 (19.34; 32.21)	36.60 (26.44; 55.56)	0.02

Note. The reference group represents values considered normal. * — significant difference between Group 1 and Group 2.

Table 3. Changes in liver injury biomarker levels during the study period.

Biomarker	Group	Values during study stages				Significance of changes, *P value**
		At admission, >*Me (Q1–Q3)*	Day 3, *Me (Q1–Q3)*	Day 7, *Me (Q1–Q3)*	Day 11, *Me (Q1–Q3)*	
L-FABP, ng/mL	Group 1	26.40 (23.30; 34.10)	21.40 (17.60; 30.30)	17.30 (14.90; 20.90)	13.70 (12.40; 17.60)	0.01
	Group 2	56.79 (39.09; 71.12)	45.80 (35.68; 78.75)	46.65 (32.90; 82.38)	44.15 (27.15; 84.50)	0.04
Between-group *P value***		0.01	0.01	0.01	0.01	—
Arginase, ng/mL	Group 1	22.40 (21.40; 28.40)	22.80 (20.80; 24.90)	19.90 (17.10; 22.90)	18.50 (16.40; 20.70)	0.01
	Group 2	39.05 (32.85; 50.43)	40.60 (34.53; 49.03)	40.10 (34.43; 49.03)	41.80 (34.93; 50.70)	0.68
Between-group *P value***		0.01	0.01	0.01	0.01	—
Hyaluronic acid, ng/mL	Group 1	175.0 (86.0; 423.0)	147.0 (72.0; 286.0)	135.0 (54.0; 274.0)	110.0 (56.0; 242.0)	0.03
	Group 2	290.5 (148.5; 517.0)	256.0 (138.5; 499.5)	258.5 (130.5; 511.5)	255.5 (131.5; 462.0)	0.58
Between-group *P value***		0.05	0.15	0.09	0.03	—
5′-nucleotidase, IU/L	Group 1	25.56 (19.34; 32.21)	24.43 (18.85; 30.38)	22.67 (15.76; 30.08)	15.90 (13.21; 20.61)	0.01
	Group 2	36.60 (26.44; 55.56)	34.92 (16.35; 56.02)	40.55 (24.31; 63.18)	34.70 (20.31; 63.18)	0.39
Between-group *P value***		0.02	0.02	0.01	0.01	—

Note. **P*— Friedman ANOVA (within-group comparison). ***P*— two-sample Kolmogorov–Smirnov test (between-group comparison).

characteristics, with an AUC ranging from 0.926 to 0.979 (95% CI: 0.851–1.000) (Fig.).

Discussion

To date, the search continues for promising laboratory biomarkers that can objectively assess the condition of patients with LF in the context of OJ and help predict the likelihood of an unfavorable outcome. From this perspective, liver injury biomarkers such as L-FABP, 5-NT, arginase and hyaluronic acid, appear to be relevant indicators of LF severity and prognosis in the setting of OJ.

A number of studies have demonstrated the clinical significance of L-FABP in various liver conditions, including liver allograft rejection [13], hepatocellular carcinoma [14–16], alcohol-induced chronic LF [17], and cirrhosis [14]. According to the literature, L-FABP is a sensitive marker of hepatocyte injury both in vivo and in vitro [14–17]. It is predominantly localized in the cytoplasm of hepatocytes, with smaller amounts found in the nucleus and outer mitochondrial membrane [13, 14].

L-FABP belongs to a family of relatively small (15 kDa) cytosolic lipids that are constitutively expressed in the liver. A distinctive feature of L-FABP is the presence of a β-barrel binding cavity, which enables the capture and transport of bile acids, eicosanoids and heme [16,18] to the mitochondria for oxidation [11].

This biomarker has strong diagnostic properties: it is cytosolic, highly specific for liver tissue, present at high intracellular concentrations, and has a low molecular weight [19]. During treatment, patients with favorable outcomes showed a statistically significant decrease in serum L-FABP levels, while those with poor outcomes maintained persistently elevated levels.

Logistic regression modeling demonstrated the predictive value of L-FABP for patient outcomes in LF associated with OJ. Depending on the time point during hospitalization, sensitivity ranged from 89.2% to 92.3%, specificity from 88.9% to 96.3%, and the cutoff ranged from 21.6 to 40.0 ng/mL. These findings underscore the high sensitivity and specificity of L-FABP in detecting hepatocellular injury in LF secondary to OJ, likely due to its cytoplasmic localization and rapid release into the circulation upon hepatocyte injury.

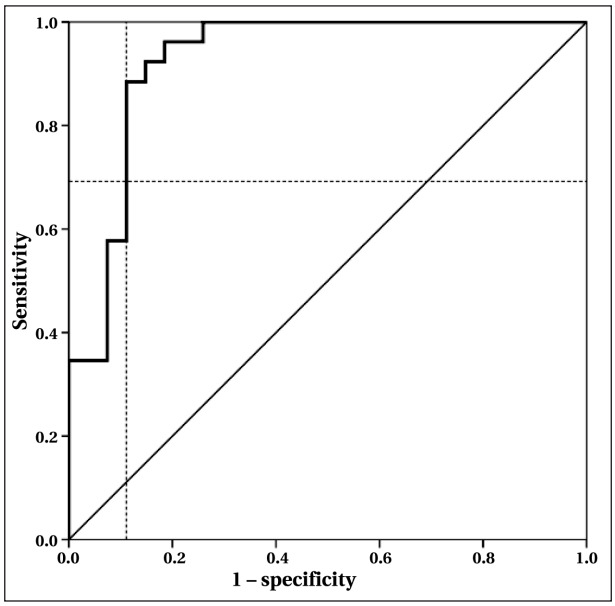

Fig. ROC curve for the logistic model with «L-FABP level» predictor.

5-NT is an integral membrane glycoprotein classified as a phosphatase that catalyzes the hydrolysis of nucleoside 5-phosphates [20]. In the liver, it is localized in the plasma membranes of biliary canalicular cells, sinusoids and Kupffer cells [21, 22]. In clinical practice, 5-NT serves as a highly specific marker for the diagnosis of hepatobiliary pathology in patients with and without obstructive jaundice.

Cholestasis of any etiology is typically associated with a parallel increase in ALP and 5-NT levels [21]. It is considered a reliable marker of both primary and secondary liver tumors, hepatobiliary disease with intrahepatic or extrahepatic bile duct obstruc-

Table 4. Predictive value of liver injury biomarkers according to ROC analysis.

Biomarker	Area under the curve [95% CI]	
	Group 1, *N*=27	Group 2, *N*=26
	L-FABP, ng/mL	
At admission	0.994 [0.982; 1.000]	1.000
Over treatment period	0.926–0.979 [0.851–1.000]	
	Arginase, ng/mL	
At admission	0.748 [0.612–0.884]	0.993 [0.978; 1.000]
Over treatment period	0.812–0.886 [0.048–0.063]	
	Hyaluronic acid, ng/mL	
At admission	0.868 [0.774; 0.963]	0.951 [0.899; 1.000]
Over treatment period	0.685–0.687 [0.542–0.829]	
	5′-nucleotidase, IU/L	
At admission	0.970 [0.913; 1.000]	0.985 [0.953; 1.000]
Over treatment period	0.671–0.781 [0.519–0.911]	

Note. CI — confidence interval. «Over treatment period» represents a range of AUC values observed at different treatment days (Day 3, 7, 11). Group 1: patients with favorable outcomes; Group 2: patients with unfavorable outcomes.

tion [13, 23], viral hepatitis [21, 24], early-stage biliary cirrhosis, third-trimester pregnancy, and graft-versus-host disease [15, 23].

Although 5-NT is a well-established and highly specific biomarker of liver disease, no clear correlation between 5-NT levels and disease severity or outcome in patients with OJ has been reported in the literature. In our study, significantly higher 5-NT levels were observed in patients with unfavorable outcomes, with only a nonsignificant decrease over the treatment period. In contrast, patients with favorable outcomes showed a statistically significant decrease in 5-NT levels, although levels remained above the reference range.

The prognostic value of 5-NT for predicting outcome in patients with LF secondary to OJ was modest. The area under the ROC curve (AUC) for 5-NT-based models ranged from 0.671 to 0.781 (95% CI, 0.519-0.911; *P*=0.02), with sensitivity ranging from 53.8% to 69.2% and specificity from 81.5% to 85.2%, depending on the time point during hospitalization. The cut-off value determined empirically was 34.4 IU/L.

Hepatic arginase catalyzes the hydrolysis of L-arginine to ornithine and urea [25]. Arginase serves two homeostatic purposes: the elimination of ammonia via urea synthesis and the production of ornithine, a precursor for polyamines and proline [25]. Because hepatic arginase activity is higher than in other tissues, an increase in serum arginase levels may be relatively specific to liver pathology. Arginase levels may serve not only as an early marker of liver injury, but also as an indicator of recovery or resolution (e. g., after surgery) [13]. According to the literature, a concurrent increase in serum arginase and gamma-glutamyl transpeptidase may be particularly informative in detecting hepatocellular injury and cholestasis [26].

Our results showed an initial increase followed by a sustained decrease in serum arginase levels from day 7 in patients with favorable outcomes, whereas persistently high concentrations were observed in patients with unfavorable outcomes. The predictive performance of the arginase-based models, as assessed by the area under the ROC curve (AUC), was «good» at baseline (AUC 0.748 [95% CI, 0.612–0.884]) and «very good» on days 3, 7 and 11 of intensive care (AUC 0.812–0.886 [95% CI, 0.048–0.063]), with sensitivity ranging from 57.7% to 76.9% and specificity from 77.8% to 88.9% at a cut-off of 34.0 ng/mL.

Hyaluronic acid (HA) is a glycosaminoglycan, a high molecular weight polysaccharide with a linear, unbranched structure [27]. Under physiological conditions, sinusoidal endothelial cells express specific receptors that facilitate rapid clearance of HA from the circulation (within 5–6 minutes) by the enzyme hyaluronidase. This clearance is impaired in cholestasis, resulting in elevated serum HA levels [28]. HA serves as a biomarker of liver fibrosis, which is clinically relevant in LF associated with OJ, where portal hypertension and cholangitis are common and often lead to fibrosis [13, 29].

There is a documented correlation between serum HA levels and liver disease severity as measured by the Child-Pugh score [28]. In addition, several studies have investigated the use of HA as a tumor marker, including in hepatocellular carcinoma, due to its interaction with CD44 and RHAMM receptors on the cell surface [27, 30].

In our study, patients with favorable outcomes showed a significant initial increase followed by a decrease in HA levels during treatment, while those with poor outcomes maintained consistently high levels. The predictive ability of HA-based models on admission and on day 3 of treatment was determined, with AUC values of 0.685–0.687 [95% CI, 0.542–0.829], sensitivity of 38.5–46.2% and specificity of 74.1–81.5%.

Conclusion

This study highlights the diagnostic and prognostic relevance of several biological markers for the assessment of liver function in the setting of obstructive jaundice.

Among them, dynamic monitoring of L-FABP levels during the overt phase of the disease showed the highest sensitivity and specificity for predicting outcome in patients with liver failure secondary to obstructive jaundice.

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