



## SURGICAL RISK ASSESSMENT IN PATIENTS WITH LIVER CIRRHOSIS – A LITERATURE REVIEW

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### ABSTRACT

Patients with chronic liver injury and liver cirrhosis develop disease-specific pathophysiological abnormalities. These specific findings compromise the functional activity of other organs and systems, leading to poor postoperative outcomes and increased morbidity and mortality rates in this population of patients. This requires a thorough analysis of the aforementioned pathophysiological abnormalities in order to optimize the patient's clinical condition in the preoperative period and to improve their perioperative performance status.

The impaired physiological reserves in patients with liver cirrhosis necessitate a detailed assessment of the surgical risk in each specific patient. For this purpose, there are multiple classification and scoring systems for assessment – Child-Pugh – Turcotte (CPT), Model of end-stage liver disease (MELD), Model of end-stage liver disease – Na (MELD – Na), Albumin – Bilirubin score (ALBI), each with specific indications for clinical use. This literature review on the topic examines the pathophysiological changes in patients with liver cirrhosis and the above-mentioned classification systems for risk assessment, as well as the advantages and disadvantages of each of them.

**Keywords:** liver cirrhosis, risk assessment, Child-Pugh-Turcotte, MELD, MELD – Na, ALBI,

### INTRODUCTION

Chronic liver damage and liver cirrhosis lead to significant changes in the physiology and functioning of different organs and systems in the body, which in turn leads to a deteriorated performance status increased morbidity and mortality rates when cirrhotic patients undergo surgical treatment. Liver dysfunction due to chronic liver damage leads to impaired metabolism and pharmacokinetics of a number of substances and medications, which must be taken into account in the therapeutic approach in the perioperative period of these patients, as well as in relation to the anesthesia used during the surgical intervention.[1] Cirrhotics also develop liver synthetic dysfunction, resulting in hypoproteinemia, malnutrition and decreased synthesis of blood clotting factors, followed by a persistent state of hypocoagulation and an increased risk of bleeding in the intra- and postoperative period. At least 10% of all cirrhotic patients would require surgical treatment in their life.[2] Surgical risk assessment in patients with chronic liver injury and underlying liver cirrhosis is essential due to their impaired physiological functions and the increased risk of developing complications in the perioperative period, and the higher mortality rate if they undergo surgical treatment.

### REVIEW RESULTS

The present article summarizes the clinical practice results of surgical institutions worldwide in treatment of patients with chronic liver injuries and cirrhosis, as well as the reasons for poor perioperative performance and outcomes in that patient cohort. It emphasizes the importance of surgical risk assessment in patients with liver cirrhosis and some of the most commonly used classifications and scoring systems in relation to that. The literature search included 152 articles published between the late '90s and present days. After profound reading and analysis of the literature, a review of 24 articles published between 2002 and 2023 was conducted.

### DISCUSSION

Impaired liver function in chronic liver diseases and liver cirrhosis causes disturbances in the synthesis of vasoactive substances in the macroorganism of cirrhotic

patients and development of a hyperdynamic type of hemodynamics in this population. The imbalance in this synthesis can significantly affect vascular tonus, intravascular volume and overall hemodynamics. Amongst the vasoactive substances responsible for the reorganization of hemodynamics in cirrhotic patients is sodium oxide, synthesized by hepatocytes and endothelial cells. Sodium oxide has a powerful vasodilatory effect, and increased production of the latter is observed in patients with liver cirrhosis. This, in turn, leads to increased splanchnic vasodilation, which increases blood flow to the liver and worsens the existing pulmonary hypertension. Another important vasoactive substance secreted by endothelial cells is the endothelin, which has a potent vasoconstrictive effect and its serum levels in patients with cirrhosis are increased. It is involved in vascular remodeling and the development of increased vascular resistance in the systemic circulation, which leads to dysregulation of arterial blood pressure. In chronic liver dysfunction and the development of cirrhosis, increased levels of serum prostaglandins are observed, which are derivatives of arachidonic acid metabolism and have a vasodilatory effect. They also play an active role in inflammatory pathways in the context of liver cirrhosis. They are involved in splanchnic vasodilation and the worsening of portal hypertension, while at the same time, have a vasodilatory effect on the systemic circulation and lead to decreased peripheral vascular resistance. A negative effect of elevated prostaglandin levels on renal perfusion has also been established, which worsens renal dysfunction in cirrhotic patients and mediates the development of hepatorenal syndrome.[3, 4, 5] Liver cirrhosis is characterized by the development of a new type of hemodynamics due to the development of disorders in blood flow, blood circulation and vascular resistance at the macroorganism level. In the initial stages of the disease, the hemodynamics of patients with cirrhosis is the so-called hyperdynamic type, which is characterized by increased cardiac output and increased heart rate as compensatory mechanisms for the reduced effective circulatory blood volume. The development of liver fibrosis and changes in the architectonics of the liver parenchyma lead to the development of portal hypertension due to the increasing resistance of the parenchyma to venous blood flow to the liver. Portal hypertension with an increase in portal pressure leads to the gradual development of portosystemic collaterals, which forms gastroesophageal varices and increases the risk for bleeding and death, as well as leads to splanchnic vasodilation, which is of essential importance for the accumulation of ascites. Due to systemic vasodilation and reduced peripheral vascular resistance, a persistently decreased arterial blood pressure develops because of the reduced effective volume of circulating blood, which worsens renal perfusion. This, in turn, triggers other compensatory mechanisms such as renal vasoconstriction, which in turn activates the Renin – Angiotensin – Aldosterone system (RAAS) and leads to a persistently increased serum level of aldosterone [3, 4]. The reduced effective volume of cir-

culating blood is interpreted by the macroorganism as a state of hypovolemia, which stimulates the hypothalamus to increase the secretion of antidiuretic hormone in order to retain fluids and increase the volume of circulating blood. The simultaneous increase in antidiuretic hormone and aldosterone values leads to simultaneous sodium and water retention, which determines the development of dilutional hyponatremia in the advanced phases of the disease [3, 4]. Increased antidiuretic hormone values increase the amount of accumulated ascites fluid and worsen peripheral edema of the tissue [6]. As liver dysfunction progresses and cirrhosis decompensates, this condition leads to the development of one of the life-threatening complications of the disease – hepatorenal syndrome, which is mediated by renal hypoperfusion, acute renal dysfunction and volume overload in relation to the increased antidiuretic hormone secretion, in the context of a chronically dysfunctional liver. Hepatorenal syndrome (HRS) can be classified as HRS type 1 and HRS type 2. In the first type, acute renal dysfunction and an increase in serum creatinine values above 250 mmol/l are observed within two weeks, which develops in terms of acute and exacerbated liver disease and has a worse prognosis. In the second type of HRS, there is a gradual development of renal dysfunction with serum creatinine values between 150-250 mmol/l. Symptoms of HRS include the development of oliguria (<400ml/day), hyperazotemia, hyponatremia, ascites, and low levels of sodium in the excreted urine [3, 4, 5]. Respiratory failure in patients with liver cirrhosis is characterized by the development of typical changes, leading to potentially life-threatening complications. First and foremost is the development of hepatopulmonary syndrome (HPS), characterized by the development of pulmonary vasodilation due to the increased serum levels of vasodilators, in particular sodium oxide, and the subsequent development of arteriovenous pulmonary shunts, leading to impaired ventilation/perfusion ratio - the formation of areas in the lung parenchyma that are ventilated but not perfused adequately or vice versa, which is the basis of compromised gas exchange and the development of hypoxemia in affected patients. On the other hand, portopulmonary hypertension develops because of the secondary increased pressure in the pulmonary arteries, characterized by increased pulmonary vascular resistance and increased blood flow in the pulmonary circulation, in terms of existing portal hypertension in patients with liver cirrhosis, which in a number of cases of severe cardiovascular comorbidity can lead to right-sided heart failure and death. Increased levels of vasoconstrictive substances and low levels of vasodilators in the serum of patients with liver cirrhosis lead to pulmonary vascular remodeling, causing pulmonary vascular resistance and the development of pulmonary hypertension. Due to fluid retention, typical for patients with liver cirrhosis, volume overload (including iatrogenic) can develop, which in turn can lead to pulmonary edema, impaired gas exchange and respiratory distress [3, 4, 5]

The accumulation of ascites in the abdominal cavity in patients with chronic liver damage and liver cir-

rhosis is due to several factors. First of all, there is the parenchymal liver remodeling and the hyperdynamic type of hemodynamics that develops in connection with it. The developing portal hypertension, low oncotic blood pressure due to hypoproteinemia and, in particular, hypoalbuminemia, as well as the activation of the rennin-angiotensin-aldosterone system and the state of hyperaldosteronism and water retention, lead to the accumulation of ascites fluid in the abdominal cavity, as well as severe edema of peripheral tissues. Due to splanchnic vasodilation and increased vascular permeability, active transudation of fluids towards the abdominal cavity occurs, which is further deteriorated by the compensatory retention of fluids at the kidney level. The clinical manifestation is expressed in an increase in abdominal circumference, feeling of abdominal heaviness and distension, as well as an increase in body weight [3, 4]. The accumulation of ascitic fluid can lead to the development of severe life-threatening complications. There is a higher incidence of developing spontaneous bacterial peritonitis (SBP) in patients with liver cirrhosis and ascites. Spontaneous bacterial peritonitis is an infection of the accumulated ascitic fluid in the abdominal cavity without an identifiable source of infection in the abdominal cavity, usually caused by Gram-negative microorganisms. Its occurrence is due to the pathological fluid accumulation in the abdominal cavity, increased intestinal permeability and bacterial translocation from the intestines to the systemic circulation and the free abdominal cavity, as well as due to the compromised immune status and increased susceptibility to the development of infectious complications in patients with chronic liver dysfunction and cirrhosis [3, 4]. Risk factors for the development of spontaneous bacterial peritonitis are low protein levels in the ascitic fluid (<10g/L), elevated serum bilirubin levels as a marker of liver dysfunction, a history of previous episodes of spontaneous bacterial peritonitis, a higher class according to the Child-Pugh-Turcotte classification system (B or C) increase the risk of developing spontaneous bacterial peritonitis. Other potentially life-threatening complications associated with the accumulation of ascitic fluid are the development of mono- or multiorgan failure of various organs and systems. Ascites leads to restrictive type of dyspnea due to its compressive effect on the diaphragm and impaired lung mechanics. It also reduces functional residual lung capacity, leading to the development of atelectasis and impaired gas exchange. Measures against ascite accumulation in patients with liver cirrhosis can be divided into two groups - conservative and invasive. The first group includes dietary modifications - limited oral intake of salt and water and the use of loop and potassium-sparing diuretics (furosemide and spironolactone) in appropriate dosage regimens. In case of unsatisfactory therapeutic effect or refractory to the treatment of ascites, invasive treatment methods are used, including decompressing paracentesis, as well as shunt surgery or its modern analogue - transjugular intrahepatic portosystemic shunt (TIPS) [3, 4]. In patients with terminal stage liver disease, liver transplantation is considered

as a definitive method of treatment of the underlying disease and its accompanying complications.

Impaired metabolism in terms of compromised liver function affects brain activity, clinically manifested as complex neurocognitive disorders to the extent of developing hepatic encephalopathy, typical for advanced stages of liver cirrhosis. The accumulation of toxins (especially ammonia), which does not occur in a normally functioning liver parenchyma, leads to neurological changes ranging from mild confusion to coma. Ammonia is an intermediate product of protein metabolism that accumulates in the systemic circulation in patients with chronic liver dysfunction. It, as well as other metabolic waste products, leads to impaired neurotransmission due to an imbalance in the levels of excitatory (glutamate) and inhibitory (GABA) neurotransmitters, which worsens brain function in these patients. When hepatic encephalopathy (HE) develops in liver cirrhotic patients, a wide range of symptoms is observed, ranging from mild to severe. Cognitive changes include confusion, disorientation, and memory impairment. Behavioral changes may include apathy, lethargy, and personality disorders. As the disease progresses and moderate to severe hepatic encephalopathy develops, motor disorders such as asterixis (a characteristic flapping tremor of the upper limbs), slurred speech, impaired coordination of movements, and life-threatening quantitative disorders of consciousness such as stupor and coma are observed [5].

In chronic liver diseases, hormonal imbalance also occurs due to liver dysfunction and compromised metabolic activity of hepatocytes. An imbalance of sex hormones occurs with an increase in estrogen levels and a decrease in testosterone levels, leading to the development of gynecomastia in men, as well as a state of decreased libido, erectile dysfunction and infertility. In female patients, menstrual cycle disorders can be observed. Hormonal imbalance of sex hormones in patients with cirrhosis causes loss of bone density. Vitamin D metabolism, its hepatic hydroxylation and the formation of its active form - calcitriol, are disrupted, which in turn compromises calcium resorption, worsening the state of hypocalcemia in patients with cirrhosis, present due to impaired calcium resorption and reduced dietary intake. As a compensatory mechanism for the state of hypocalcemia, secondary hyperparathyroidism develops, which increases osteoclast activity in order to increase the serum calcium level and contributes to the disruption of bone density and the development of osteoporotic changes and osteomalacia. A common disorder in cirrhotics in relation to impaired insulin metabolism is the development of impaired insulin clearance and increased serum insulin levels. Hepatic gluconeogenesis and glycogen storage in hepatocytes are also impaired, leading to a systemic increase in blood sugar levels. As a result of the metabolic disorders that have occurred, peripheral insulin resistance develops, and the risk of developing type 2 diabetes mellitus significantly increases. A very common and typical hormonal disorder in patients with liver cirrhosis is the condition of hyperaldosteronism. In the context of metabolic dys-

function in cirrhotic patients, deviations in hormonal levels of thyroid hormones, somatostatin and prolactin can occur, as well as disrupted cortisol activity with the development of Cushingoid status - moon face, obesity mainly in the abdominal area, hyperglycemia, metabolic syndrome can be observed.

Another specific change in the physiology of this contingent of patients is the compromised immune function and the development of a state of immunosuppression in patients with liver cirrhosis, which leads to an increased risk of infectious complications other than SBP [3]. The immunosuppressive status is caused by multiple factors. Protein-synthetic liver dysfunction leads to a disrupted synthesis of cytokines and complement factors, which plays a crucial role in the protection of the macroorganism. The phagocytic activity of Kupffer cells is impaired, and an imbalance occurs between the synthesis of pro- and anti-inflammatory cytokines, which impairs the effective immune response to the extent of completely compromised during the progression of liver dysfunction and decompensation of liver cirrhosis. For the development of an optimal immune response, a number of vitamins and biochemical elements are necessary, such as vitamin A and zinc, the levels of which are significantly decreased in the cirrhotic population in relation to the developing status of malnutrition. Portal hypertension and the development of portosystemic collaterals lead to a “bypassing” of the liver as a filter for various pathogenic microorganisms that have entered the systemic circulation from the external environment. On the other hand, a change in the intestinal microbiome, combined with increased permeability of the intestinal wall and pronounced bacterial translocation, is observed in patients with liver cirrhosis, which results in the development of the inability of the macroorganism to mount an adequate immune response and to the development of a systemic inflammatory reaction. A particularly important aspect of chronic liver damage is the development of splenomegaly combined with hypersplenism, leading to increased destruction of blood elements and the typical laboratory abnormalities in cirrhotic patients - leuko- and thrombocytopenia, which significantly contributes to the impaired immune response in these patients.

Coagulopathy in patients with chronic liver injury and cirrhosis is a common and potentially life-threatening complication of the underlying disease, which has a significant impact on perioperative risk in this population. Its genesis is complex, with the crucial role of protein-synthetic liver dysfunction, leading to a severely compromised synthesis of blood clotting factors (severely reduced procoagulant effect) in patients with cirrhosis and increased synthesis of anticoagulant substances (proteins C and S), significantly increasing the risk of bleeding. A common feature of cirrhotic patients is the development of vitamin K deficiency concerning the accompanying cholestasis and impaired gastrointestinal absorption, severe malnutrition and reduced oral intake with food. This leads to impaired synthesis of vitamin K-dependent blood clotting factors (2, 7, 9 and 10), worsening the

hypocoagulant status of the patients. Another important aspect of cirrhotic coagulopathy is the development of splenomegaly with increased functional activity of the spleen. Increased platelet sequestration in the spleen is observed, leading to significant thrombocytopenia in patients with liver cirrhosis. Platelet production can be impaired due to liver dysfunction in cirrhotic patients, as well as due to bone marrow suppression, leading to thrombocytopenia. In patients with liver cirrhosis, impaired platelet activation is observed due to weak expression of receptors on the platelet surface and the inability for subsequent platelet aggregation upon stimulation. Reduced synthesis of thromboxane A2 is observed in patients with liver cirrhosis, which has a significant relation to platelet aggregation and normal platelet activity. All of the above aspects of thrombocytopenia and impaired functional activity of platelets in patients with liver cirrhosis determine the increased risk of bleeding in this population [7].

The assessment of their liver function plays an essential role in surgical risk assessment in this population. It can be carried out using standardized scoring systems such as Child-Pugh–Turcotte (CPT) and MELD. [9] The first of them is based on objective indicators, such as serum levels of bilirubin, albumin and coagulation status, and subjective indicators, such as the presence of ascites and signs of hepatic encephalopathy. (Table 1) The MELD scoring system is considered a more objective method for assessing liver function, and it is used as a predictor of survival in the cirrhotic population, helping in the assessment of perioperative risk, as well as prioritizing patients on the liver transplantation waiting list. It is based only on objective indicators such as laboratory values of bilirubin, INR and creatinine. The advantages of MELD are its objectivity, the possibility of systematic use, and better predictability regarding early mortality in the cirrhotic population [4, 6, 9, 10, 11, 12]. The MELD-Na scoring system represents a modification of the MELD scoring system by incorporating the serum sodium level, which is of essential importance in patients with liver cirrhosis due to the commonly observed hyponatremia as the underlying disease progresses. Taking into account the sodium levels provides a more accurate assessment of the perioperative risk and a better ability to predict adverse outcomes in the postoperative period in cirrhotic patients. This scoring system allows for a good risk assessment in patients with decompensated liver cirrhosis since patients with hyponatremia more often develop severe and life-threatening complications of the underlying disease, such as ascites and hepatorenal syndrome. The main disadvantage is the overestimation of the risk of death in patients with temporarily reduced sodium values. MELD-Na is applicable to a wide range of cirrhotic patients, including those who show low MELD scores, which when used alone may lead to an underestimation of risk in cirrhotic patients with severe hyponatremia [13]. CPT scoring system shows better predictive value in short-term mortality, while MELD and MELD Na score is useful in 30 and 90 days mortality rates [1, 4, 14].

**Table 1.** Child – Pugh – Turcotte classification.

	1point	2 points	3points
Encephalopathy	None	Grade 1or 2	Grade 3or 4
Ascites	None	Mild	Moderate
Bilirubin	1-2mg/dL	2-3mg/dL	>3mg/dL
Albumin	>3.5g/dL	2.8-3.5mg/dL	<2.8g/dL
Prothrombine time	1-4sec	4-6sec	>6sec

CPT A – 5-6 points, CPT B 7-9 points, CPT C 10-15 points

The ALBI score system is a simple and objective method for assessing liver function in patients with chronic liver damage based on liver cirrhosis or hepatocellular carcinoma. When it was created, it was mainly used for risk stratification in patients with hepatocellular carcinoma, but its application has expanded to patients with various liver diseases. This scoring system was created as an alternative to the Child-Pugh – Turcotte score for the purpose of a more accurate and, at the same time, simpler assessment of liver function based on two objective laboratory indicators - serum levels of albumin and bilirubin, with albumin assessing the synthetic activity of the liver, bilirubin assessing its excretory functional activity, unlike the CPT score system, which uses both objective and subjective clinical criteria. The ALBI score is calculated using the following equation:  $ALBI\ score = \log_{10}(\text{bilirubin } \mu\text{mol/L}) \times 0.66 + \text{albumin g/L} \times (-0.085)$ . According to the obtained result, patients are classified into four grades. In ALBI grade 1 with a score  $< -2.60$ , patients have good liver function; in ALBI grade 2, the results are  $> -2.60 < -1.39$  and indicate moderate liver dysfunction; in ALBI grade 3, patients show a score  $> -1.39$ , which indicates severely compromised liver function. Patients classified as Child-Pugh class A can be divided into ALBI grades 1 and 2, respectively. Patients classified as ALBI grade 1 show twice as long survival as those classified as ALBI grade 2, even though both grades are Child-Pugh class A according to the Child-Pugh classification system. The main advantages of the ALBI score over the Child-Pugh-- Turcotte scoring system are the lack of subjective indicators in the calculation of the ALBI score, such as the presence of ascites and hepatic encephalopathy, used in the CPT scoring system, as the ALBI score is based on only two objective laboratory indicators (albumin and bilirubin), which makes it an easy and objective method of assessment. While using the Child-Pugh-Turcotte scoring system, many patients classified as class A (with good liver function) may vary widely from mild to moderate liver dysfunction, using the ALBI score allows better differentiation in this group of cirrhotic patients and has better discriminative capabilities in terms of predicting surgical outcomes. Both systems show equal effectiveness in predicting surgical outcomes in patients with chronic liver damage, but the ALBI score is more sensitive

in detecting small changes in patients with compensated liver cirrhosis. The ALBI score predicts survival and outcome of surgical treatment in patients with liver diseases at different stages, including autoimmune liver diseases, chronic viral hepatitis, chronic liver damage due to other different etiologies and unlike the MELD scoring system shows early detection of worsening liver function in the cirrhotic population. Compared to the MELD and MELD-Na scoring systems, the method also shows an advantage due to the lack of the necessity to use INR, which can be influenced by factors unrelated to liver function, such as systemic anticoagulant intake, and due to the fact that the aforementioned scoring systems do not include the value of albumin, which is an important marker for assessing liver function [15, 16, 17]. This ALBI scoring system finds its main application in terms of predictability of surgical outcomes in patients with hepatocellular carcinoma, allowing better stratification of patients who will undergo transarterial chemoembolization (TACE), radiofrequency ablation (RFA) or hepatic resection. In relation to patients with liver cirrhosis, the ALBI scoring system provides good risk stratification in terms of postoperative outcomes, mortality and survival. This scoring system has shown good predictive value for patients' survival with or without hepatocellular carcinoma undergoing general surgery or transplantation in the context of different clinical scenarios. It can provide a good assessment of liver function and indicate whether a patient with chronic liver damage can tolerate general surgery procedures, as well as predict survival and mortality in the cirrhotic population and in patients with hepatocellular carcinoma. The main disadvantages are its limitation to only two objective parameters (bilirubin and albumin) and the lack of assessment of renal function, and the presence of major complications such as ascites and hepatic encephalopathy [17, 18, 19]. The Possum scoring system is a method of perioperative risk assessment and prediction of morbidity and mortality in patients undergoing surgical treatment. The system was created to assess the risk in patients undergoing general surgical interventions. It consists of two main components – physiological and operative score. The physiological component assesses the physiological condition of the patient preoperatively, including an assessment of 12 objective physiological indi-

cators – age, cardiovascular status, respiratory status, systolic blood pressure, heart rate, Glasgow Coma Scale points, hemoglobin, leukocytes, serum urea, sodium and potassium levels, as well as the patient's ECG findings. Each of the listed indicators is assessed on a scale of 1 – 4, depending on the specific deviations in the values of each indicator. The operative component of the score consists of 6 objective indicators related to the surgical intervention undergone by the patient – the volume of the surgical intervention (small, medium, large and very large), the number of surgical interventions, intraoperative blood loss, the presence of peritoneal contamination, the presence of malignant disease, established pre- or intraoperatively, the type of surgical treatment (emergency or elective). Each indicator is evaluated on a scale of 1-4, depending on its deviations. Once calculated, the physiological and operative scores are used in logarithmic regression to calculate the probability of postoperative death or development of complications in the particular patient. The advantages of the Possum scoring system are that both the physiological status of the patient and the severity of the surgical intervention performed are taken into account; it provides a standardized method for risk assessment that can be used for different patient groups, in various medical institutions and by other surgeons to compare surgical results; has a predictive value in terms of morbidity and mortality, which supports preoperative planning, stratification of high-risk patients and informed consent. The disadvantages of the scoring system are primarily based on its complexity and the need to perform multiple calculations, which is not always applicable in daily practice; there is a tendency to overestimate the risk, especially in patients with a low-risk profile; the system is designed specifically for general surgical interventions, which limits its use in other highly specialized surgical interventions (cardiac surgery, vascular surgery, etc.) [20, 21, 22, 23, 24]. In the cirrhotic population, the Possum scoring system does not include parameters assessing the severity of liver dysfunction, as do the Child-Pugh – Turcotte, MELD and MELD-Na scoring systems, and may often lead to an overestimation of the risk of increased morbidity and mortality in cirrhotic patients when used alone. For this reason, the simultaneous use of the specific Child-Pugh – Turcotte, MELD and MELD-Na scoring systems and a general surgical score such as POSSUM is recommended in order to better predict surgical outcomes in this population due to the simultaneous assessment of liver function and general physiological status, including comorbidities in these patients.

In order to adequately assess perioperative risk in this patient population, it is important to consider the severity of comorbidities, especially concomitant cardiovascular and pulmonary pathology. Concomitant heart failure, arrhythmias, and ischemic cardiomyopathy, as well as pulmonary diseases leading to respiratory restriction and worsening pulmonary ventilation, should be taken into account.

Patients with chronic liver injury and liver cirrhosis often show signs of malnutrition, which can lead to impaired tissue healing and recovery. Taking them into account is fundamental in relation to preoperative correction and improving postoperative outcomes in this population. Monitoring of electrolyte balance and correction of hyponatremia and hypokalemia, typical for the cirrhotic population, as well as correction of other metabolic disorders that worsen postoperative outcomes, play a crucial role in optimizing the approach to such patients. Another important aspect of risk assessment in patients with liver cirrhosis undergoing surgery is taking into account the type of surgical intervention performed. Elective surgery in this population allows optimization of liver functioning and improvement of nutritional status, which is difficult to achieve and limited by the time factor in surgical interventions performed on an emergency basis, which increases the risk of emergency surgery procedures.

## CONCLUSION

The cirrhotic population has a higher incidence of developing postoperative complications and higher mortality rates in the postoperative period, which obliges surgeons to be aware of potentially life-threatening complications such as mono- or multiorgan failure, increased incidence of surgical site infections, coagulopathy and increased risk of intra- and postoperative bleeding, as well as death in the early postoperative period or shortly after dehospitalization.

All of the above requires a multidisciplinary approach to risk assessment in the cirrhotic population undergoing elective or emergency surgery, which includes surgeons, anesthesiologists, and gastroenterologists. Cirrhotic patients require careful and in-depth analysis of their chronic liver dysfunction and assessment of perioperative risk, as well as the development of strategies to minimize the risk of complications and improve intraoperative outcomes in this population. Adequate assessment of liver function, comorbidity, and nutritional status is essential.

## Abbreviations:

CPT – Child – Pugh – Turcotte  
MELD – Model of end-stage liver disease  
MELD Na – Model of end-stage liver disease with incorporated sodium level  
ALBI – Albumin-bilirubin  
RAAS – Renin – angiotensin – aldosterone  
HRS – Hepatorenal syndrome  
HPS – Hepatopulmonary syndrome  
SBP – Spontaneous bacterial peritonitis  
TIPS – Transjugular intrahepatic portosystemic shunt  
GABA – Gama-aminobutyric acid  
HE – Hepatic encephalopathy

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