Comparative Performance of ALBI and Child Pugh Scores in Assessing Complications of Portal Hypertension and Decompensated Cirrhosis

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Abstract

Background: Acute variceal hemorrhage is a major decompensating event in cirrhosis and remains associated with substantial morbidity, early rebleeding, organ failure, and mortality. Beyond the immediate consequences of blood loss and hemodynamic instability, gastrointestinal bleeding markedly increases the intestinal nitrogen load, enhances ammonia production, promotes systemic inflammation, and may precipitate hepatic encephalopathy. The development of encephalopathy after variceal bleeding reflects the interaction between impaired hepatic functional reserve, portosystemic shunting, infection, electrolyte disturbances, renal dysfunction, sarcopenia, and altered gut–brain signaling. Early identification of patients at greatest risk is therefore essential for closer monitoring, timely correction of precipitating factors, and consideration of preventive therapy. Conventional prognostic systems, particularly the Child–Pugh and Model for End-Stage Liver Disease scores, are widely used to assess cirrhosis severity; however, their applicability to encephalopathy prediction is limited by subjective variables, categorical thresholds, and incomplete representation of hepatic functional reserve. The albumin–bilirubin score is an objective, continuous model derived exclusively from serum albumin and bilirubin concentrations. By integrating hepatic synthetic capacity and excretory function, it may provide a simple method for identifying patients vulnerable to neurological decompensation after variceal hemorrhage.

Aim: This review evaluates the biological rationale, clinical evidence, and potential prognostic value of the albumin–bilirubin score for predicting hepatic encephalopathy following acute variceal hemorrhage in patients with cirrhosis. It also examines the relationship of albumin and bilirubin to encephalopathy pathogenesis, compares the albumin–bilirubin score with established liver severity models, and discusses its possible role in post-bleeding risk stratification and clinical decision-making.

Conclusion: Available evidence suggests that worsening albumin–bilirubin score is associated with advanced hepatic dysfunction, decompensation, mortality, and a greater likelihood of hepatic encephalopathy. Its objectivity, simplicity, reproducibility, and reliance on routinely available laboratory measurements make it an attractive prognostic tool after variceal bleeding. Nevertheless, current evidence remains limited by heterogeneous populations, variable encephalopathy definitions, retrospective designs, and insufficient external validation. Large prospective multicenter studies are required to establish optimal cutoffs, determine incremental value over conventional scores, and clarify whether albumin–bilirubin-guided preventive strategies can improve patient outcomes.

Keywords: Albumin–bilirubin score; hepatic encephalopathy; acute variceal hemorrhage; liver cirrhosis

Introduction

Cirrhosis represents the advanced stage of progressive hepatic fibrosis in which normal liver architecture is replaced by regenerative nodules surrounded by fibrous septa. These structural changes are accompanied by intrahepatic vascular distortion, portal hypertension, impaired synthetic and metabolic capacity, and increasing susceptibility to systemic complications. The clinical course is commonly divided into compensated and decompensated stages. While compensated cirrhosis may remain clinically silent for years, the development of ascites, variceal hemorrhage, hepatic encephalopathy, or jaundice marks decompensation and is associated with a substantial deterioration in survival. Importantly, decompensating events rarely occur in isolation; one complication may trigger another through hemodynamic instability, inflammation, infection, renal dysfunction, and further loss of hepatic functional reserve.[1]

Portal hypertension is the principal driver of gastroesophageal varix formation in cirrhosis. Increased intrahepatic resistance, together with progressive splanchnic vasodilatation and increased portal venous inflow, promotes the development of portosystemic collaterals that decompress the portal circulation but expose patients to variceal rupture. Acute variceal hemorrhage is therefore not merely a gastrointestinal bleeding episode but a major transition in the natural history of cirrhosis. It can produce hypovolemia, tissue hypoperfusion, renal impairment, bacterial translocation, and systemic inflammatory activation. Although advances in vasoactive therapy, antibiotic prophylaxis, endoscopic variceal ligation, and pre-emptive transjugular intrahepatic portosystemic shunt placement have improved bleeding control, early complications remain frequent and require accurate risk stratification.[2]

Hepatic encephalopathy is a potentially reversible neuropsychiatric syndrome resulting from liver insufficiency, portosystemic shunting, or both. Its clinical spectrum extends from subtle deficits in attention, psychomotor speed, and executive function to disorientation, somnolence, stupor, and coma. Acute gastrointestinal bleeding is a well-established precipitant because digestion of blood within the intestinal lumen generates a large nitrogenous substrate, increasing ammonia production and other potentially neurotoxic metabolites. This effect is amplified by reduced hepatic detoxification, portosystemic shunting, systemic inflammation, electrolyte disturbances, renal dysfunction, and diminished skeletal-muscle ammonia clearance. Consequently, encephalopathy may develop despite successful endoscopic control of hemorrhage, particularly during the early post-bleeding period.[3]

Predicting which patients will develop hepatic encephalopathy after variceal hemorrhage is clinically important because early recognition may justify closer neurological monitoring, prolonged observation, aggressive correction of precipitating abnormalities, and selective use of preventive interventions. Previous investigations have linked post-bleeding encephalopathy to advanced Child–Pugh class, hypokalemia, leukocytosis, severe anemia, infection, and greater transfusion requirements. However, these variables reflect different components of the acute insult and may fluctuate considerably during resuscitation. The Child–Pugh score also incorporates the subjective grading of ascites and encephalopathy, whereas the Model for End-Stage Liver Disease score was primarily developed to predict mortality rather than neurological decompensation. An objective marker of underlying hepatic reserve may therefore complement conventional clinical assessment.[4]

The albumin–bilirubin score was developed as an evidence-based and fully objective measure of liver function using only serum albumin and total bilirubin. Albumin reflects hepatic synthetic capacity but is also influenced by systemic inflammation, nutritional status, vascular permeability, and circulatory dysfunction. Bilirubin reflects hepatic uptake, conjugation, and excretory capacity and generally rises as hepatocellular dysfunction and cholestasis progress. Combining these continuous variables may provide greater discrimination than categorical systems in patients whose liver reserve varies substantially within the same Child–Pugh class. Although initially derived in hepatocellular carcinoma populations, the ALBI score has subsequently been evaluated across several nonmalignant liver diseases and decompensating outcomes.[5]

Nevertheless, evidence specifically addressing ALBI-guided prediction of hepatic encephalopathy after acute variceal hemorrhage remains limited. A prospective single-center study of 250 patients reported that baseline ALBI had greater discriminatory performance for encephalopathy within four weeks than Child–Pugh and MELD scores, with most events occurring during the first two weeks. However, its single-center design, short follow-up, lack of external validation, and absence of detailed assessment across encephalopathy grades restrict widespread clinical adoption. It also remains uncertain whether ALBI adds clinically meaningful information to established bleeding-risk variables or can identify patients who benefit from prophylactic therapy. Therefore, this review aims to critically examine the pathophysiological and clinical basis for using ALBI to predict hepatic encephalopathy after variceal hemorrhage, compare its performance with conventional prognostic scores, identify limitations in the available evidence, and define priorities for future prospective validation.[6]

Liver Cirrhosis

Definition

Liver cirrhosis represents the final stage of chronic liver injury and is characterized by diffuse hepatic fibrosis, distortion of the normal hepatic architecture, and the formation of regenerative nodules. Progressive fibrosis increases intrahepatic vascular resistance, resulting in portal hypertension and progressive deterioration of hepatic synthetic, metabolic, and detoxification functions. Although cirrhosis was traditionally considered an irreversible condition, current evidence demonstrates that early-stage fibrosis may partially regress after successful treatment of the underlying liver disease. Nevertheless, advanced cirrhosis remains a major cause of morbidity and mortality worldwide because of the development of life-threatening complications and progressive liver failure. [7]

Epidemiology

Cirrhosis continues to represent a substantial global health burden. Recent epidemiological studies estimate that more than 120 million individuals are living with cirrhosis worldwide, with the incidence and prevalence steadily increasing over recent decades. This increase is largely attributed to the growing prevalence of metabolic dysfunction-associated steatotic liver disease (MASLD), persistent viral hepatitis in some regions, and alcohol-related liver disease. Although advances in antiviral therapy have reduced hepatitis C-related cirrhosis, the rising incidence of obesity, diabetes mellitus, and metabolic syndrome has shifted the epidemiological landscape toward metabolic liver disease as one of the leading causes of cirrhosis. Despite improvements in medical therapy, decompensated cirrhosis remains associated with poor long-term survival and continues to impose considerable healthcare costs through recurrent hospitalizations and liver transplantation. [8]

Etiology

The causes of cirrhosis vary geographically according to socioeconomic status and disease prevalence. Chronic hepatitis B remains the predominant cause in many Asian and African countries, whereas hepatitis C virus infection, alcohol-associated liver disease, and metabolic dysfunction-associated steatotic liver disease constitute the principal etiologies in many Western countries. Autoimmune hepatitis, primary biliary cholangitis, primary sclerosing cholangitis, hereditary hemochromatosis, Wilson disease, alpha-1 antitrypsin deficiency, and chronic vascular disorders represent less common but clinically important causes. In many patients, multiple etiological factors coexist, such as metabolic dysfunction combined with alcohol consumption or chronic viral hepatitis, accelerating fibrosis progression and increasing the risk of hepatic decompensation. [9]

Clinical Progression and Decompensation

The natural history of cirrhosis evolves from a compensated stage to a decompensated stage. Patients with compensated cirrhosis may remain asymptomatic for years despite significant portal hypertension and progressive fibrosis. Once decompensation develops, however, survival declines dramatically. Major decompensating events include ascites, gastroesophageal variceal hemorrhage, hepatic encephalopathy, spontaneous bacterial peritonitis, hepatorenal syndrome, hepatopulmonary syndrome, portopulmonary hypertension, hepatocellular carcinoma, and acute-on-chronic liver failure. These

complications frequently coexist because each episode of decompensation further impairs hepatic reserve, promotes systemic inflammation, and increases susceptibility to additional organ dysfunction. Consequently, early identification of patients at high risk for subsequent complications has become a major objective in modern hepatology. [10]

Clinical Relevance to the Present Review

Among all complications of cirrhosis, acute variceal hemorrhage and hepatic encephalopathy are closely interconnected. Variceal bleeding not only reflects advanced portal hypertension but also serves as one of the strongest precipitating factors for hepatic encephalopathy through increased intestinal nitrogen load, hyperammonemia, systemic inflammatory activation, and circulatory dysfunction. Because the occurrence of hepatic encephalopathy after variceal hemorrhage markedly worsens prognosis, prolongs hospitalization, and increases mortality, reliable predictors capable of identifying high-risk patients immediately after presentation are of considerable clinical importance. Objective assessment of hepatic functional reserve using the Albumin–Bilirubin (ALBI) score has therefore emerged as a promising strategy for improving risk stratification beyond conventional liver severity scoring systems. [11]

Acute Variceal Hemorrhage

Pathophysiology of Variceal Formation and Bleeding

Portal hypertension is the fundamental mechanism underlying the development of gastroesophageal varices in patients with cirrhosis. Progressive hepatic fibrosis increases resistance to portal venous blood flow, while splanchnic vasodilatation further augments portal venous inflow, leading to sustained elevation of portal pressure. To compensate, collateral vessels develop between the portal and systemic circulations, forming esophageal and gastric varices. These collateral vessels possess thin walls and are continuously exposed to elevated pressure, rendering them highly susceptible to rupture. The likelihood of bleeding increases with larger variceal size, elevated hepatic venous pressure gradient, advanced liver dysfunction, and the presence of red wale markings on endoscopy. [12]

Epidemiology and Clinical Importance

Acute variceal hemorrhage remains one of the most serious complications of portal hypertension and accounts for a substantial proportion of hospital admissions among patients with decompensated cirrhosis. Despite major therapeutic advances over the past two decades, including vasoactive medications, prophylactic antibiotics, endoscopic variceal ligation, and transjugular intrahepatic portosystemic shunt (TIPS) in selected patients, acute variceal bleeding continues to be associated with significant morbidity and mortality. The first six weeks following the bleeding episode represent the period of greatest clinical risk because patients remain vulnerable to rebleeding, infection, renal dysfunction, hepatic encephalopathy, and acute-on-chronic liver failure. [13]

Clinical Presentation and Diagnosis

Patients with acute variceal hemorrhage typically present with hematemesis, melena, or both, frequently accompanied by manifestations of hypovolemia such as hypotension, tachycardia, dizziness, or altered mental status. The severity of presentation depends on the volume and rate of blood loss as well as the underlying hepatic functional reserve. Initial evaluation focuses on airway protection, hemodynamic stabilization, laboratory assessment, and early recognition of associated complications. Upper gastrointestinal endoscopy performed after adequate resuscitation remains the diagnostic gold standard because it confirms the bleeding source while simultaneously allowing therapeutic intervention through endoscopic variceal ligation or sclerotherapy. [14]

Management of Acute Variceal Hemorrhage

Management requires a multidisciplinary approach aimed at controlling hemorrhage, preventing early rebleeding, and minimizing complications. Initial treatment includes restrictive blood transfusion strategies, vasoactive agents such as terlipressin or somatostatin analogues, prophylactic broad-spectrum antibiotics, and prompt endoscopic variceal ligation. In patients with uncontrolled bleeding or those considered at very high risk of treatment failure, early placement of a transjugular intrahepatic portosystemic shunt significantly improves bleeding control and survival. Following successful hemostasis, secondary prophylaxis with non-selective beta-blockers combined with repeated endoscopic

ligation is recommended to reduce recurrent hemorrhage. [15]

Relationship Between Acute Variceal Hemorrhage and Hepatic Encephalopathy

Acute gastrointestinal bleeding is among the strongest recognized precipitants of hepatic encephalopathy in cirrhotic patients. Digestion of intraluminal blood markedly increases intestinal nitrogen availability, resulting in excessive ammonia production by colonic bacteria. Simultaneously, acute blood loss may induce renal hypoperfusion, electrolyte disturbances, systemic inflammation, and bacterial translocation, all of which impair cerebral homeostasis and amplify neurotoxicity. Patients with advanced hepatic dysfunction are particularly vulnerable because reduced hepatic clearance and extensive portosystemic shunting permit ammonia and inflammatory mediators to bypass hepatic metabolism and reach the systemic circulation. Consequently, hepatic encephalopathy frequently develops during the days following successful control of variceal bleeding, emphasizing the importance of early identification of high-risk individuals. [16]

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Albumin–Bilirubin (ALBI) Score

Development of the ALBI Score

The Albumin–Bilirubin (ALBI) score was developed as an objective model for assessing hepatic functional reserve using only two routinely measured biochemical parameters: serum albumin and total bilirubin. Unlike the Child–Pugh classification, which incorporates subjective clinical variables such as ascites and hepatic encephalopathy, the ALBI score relies exclusively on laboratory measurements, thereby minimizing interobserver variability and improving reproducibility. Originally established to evaluate liver function in patients with hepatocellular carcinoma, the ALBI score has subsequently demonstrated clinical utility across a broad spectrum of chronic liver diseases and has emerged as a valuable prognostic tool in cirrhosis. [23]

Calculation and Grading

The Albumin–Bilirubin (ALBI) score is calculated using a mathematical equation incorporating serum albumin and total bilirubin concentrations, generating a continuous numerical value that reflects hepatic functional reserve. Based on predetermined cutoff values, patients are classified into three ALBI grades, with Grade 1 representing the best preserved liver function and Grade 3 indicating the most advanced hepatic impairment. Because the score uses continuous laboratory variables rather than arbitrary categorical thresholds, it provides greater discrimination among patients with similar conventional liver severity classifications and may better detect subtle deterioration in hepatic reserve. [24]

Biological Basis for the ALBI Score

Albumin and bilirubin reflect two fundamental aspects of hepatic physiology. Albumin serves as a marker of hepatic synthetic capacity and contributes to plasma oncotic pressure, antioxidant defense, immune modulation, and transport of endogenous and exogenous compounds. Declining serum albumin concentrations indicate impaired hepatic protein synthesis and are frequently associated with systemic inflammation, malnutrition, and advanced portal hypertension. Conversely, serum bilirubin reflects hepatic uptake, conjugation, and biliary excretion. Elevated bilirubin levels generally indicate worsening hepatocellular dysfunction or cholestasis. Combining these complementary biomarkers provides a practical assessment of overall liver function and biological reserve, explaining the increasing interest in ALBI as an objective prognostic indicator. [25]

Clinical Applications of the ALBI Score

Since its introduction, the Albumin–Bilirubin (ALBI) score has been evaluated in numerous clinical settings beyond hepatocellular carcinoma. Studies have demonstrated its usefulness in predicting hepatic decompensation, postoperative liver failure, mortality following liver resection, survival after locoregional therapies, complications of portal hypertension, and outcomes following liver transplantation. More recently, ALBI has been investigated as a predictor of adverse events in cirrhotic patients presenting with acute variceal hemorrhage, including early mortality, rebleeding, acute kidney injury, and hepatic encephalopathy. These findings suggest that ALBI reflects hepatic reserve sufficiently to identify patients at increased risk for multiple complications during hospitalization and follow-up. [26]

Advantages and Limitations

The principal advantages of the ALBI score include its simplicity, objectivity, reproducibility, and universal availability because albumin and bilirubin are routinely measured in virtually all hospitalized patients with liver disease. The score eliminates subjective clinical assessment and avoids variability associated with grading ascites and hepatic encephalopathy. However, several limitations should also be recognized. Serum albumin may be influenced by acute inflammation, nutritional status, intravenous albumin administration, and renal disease, while bilirubin levels may increase because of hemolysis or biliary obstruction independent of hepatic functional reserve. Furthermore, the ALBI score evaluates liver function but does not directly assess portal hypertension or systemic inflammatory activity, both of which contribute substantially to adverse outcomes in cirrhotic patients. Consequently, ALBI should be interpreted alongside comprehensive clinical assessment rather than as a standalone prognostic tool. [27]

Role of the ALBI Score in Predicting Hepatic Encephalopathy After Acute Variceal Hemorrhage

Pathophysiological Rationale

The relationship between the ALBI score and the development of hepatic encephalopathy after acute variceal hemorrhage is supported by several pathophysiological mechanisms. A higher ALBI score reflects reduced hepatic synthetic function and impaired bilirubin clearance, indicating diminished hepatic reserve. Following variceal bleeding, the liver is exposed to an increased nitrogen load generated by digestion of intraluminal blood. Patients with poor hepatic reserve are less capable of detoxifying ammonia and other neurotoxic substances, resulting in greater cerebral exposure to these metabolites. Simultaneously, systemic inflammation, circulatory dysfunction, renal impairment, and extensive portosystemic shunting further amplify neurological injury, making patients with higher ALBI scores particularly susceptible to hepatic encephalopathy. [28]

Clinical Evidence

Several observational studies have demonstrated a significant association between elevated ALBI scores and the occurrence of hepatic encephalopathy following acute variceal hemorrhage. Patients classified within higher ALBI grades consistently exhibit greater frequencies of neurological complications, prolonged hospitalization, and poorer short-term outcomes compared with those having preserved hepatic function. Available evidence also suggests that most episodes of post-bleeding hepatic encephalopathy develop during the first two to four weeks after the bleeding event, highlighting the importance of early risk assessment using readily available laboratory parameters obtained at admission. [29]

Comparison with Child–Pugh and MELD Scores

The Child–Pugh and MELD scores remain the most widely used tools for assessing disease severity in cirrhotic patients. However, both possess important limitations when predicting hepatic encephalopathy after variceal bleeding. The Child–Pugh classification incorporates subjective variables, including ascites and encephalopathy, which may vary among observers and may already be altered during the acute bleeding episode. Conversely, MELD was primarily designed to estimate mortality and prioritize liver transplantation rather than predict neurological complications. Because ALBI relies exclusively on objective laboratory variables, it provides a reproducible assessment of hepatic functional reserve and may improve discrimination among patients with similar Child–Pugh or MELD scores. Several studies have therefore suggested that ALBI performs comparably or even superiorly to conventional scoring systems for predicting post-bleeding hepatic encephalopathy. [30]

Clinical Implications

Early identification of patients at increased risk for hepatic encephalopathy offers several important clinical advantages. Patients with elevated ALBI scores may benefit from closer neurological observation, more frequent laboratory monitoring, early correction of electrolyte disturbances, prompt treatment of infections, optimization of renal function, and careful avoidance of sedative medications. In selected high-risk patients, clinicians may also consider early initiation of preventive therapies such as lactulose, although management decisions should continue to follow established clinical guidelines

rather than rely solely on prognostic scores. Integrating ALBI into routine assessment of patients admitted with acute variceal hemorrhage may therefore facilitate individualized risk stratification and improve allocation of healthcare resources. [31]

Current Limitations and Future Perspectives

Despite encouraging findings, the current evidence supporting ALBI as a predictor of hepatic encephalopathy remains limited by several factors. Most available studies are retrospective or single-center investigations with relatively small sample sizes and heterogeneous patient populations. Differences in treatment protocols, follow-up duration, diagnostic criteria for hepatic encephalopathy, and ALBI cutoff values further limit direct comparison among studies. Future prospective multicenter studies involving diverse patient populations are required to validate standardized ALBI thresholds, determine its incremental value when combined with established prognostic models, and evaluate whether ALBI-guided management strategies can reduce the incidence of hepatic encephalopathy and improve clinical outcomes after acute variceal hemorrhage. [32]

Conclusion

The Albumin–Bilirubin (ALBI) score is a simple, objective, and readily available tool for assessing hepatic functional reserve in patients with cirrhosis. Current evidence suggests that it has promising value in predicting hepatic encephalopathy following acute variceal hemorrhage and may offer advantages over conventional prognostic models because of its reliance on objective laboratory parameters. Although the available data are encouraging, larger prospective multicenter studies are still needed to validate its predictive accuracy, establish standardized cutoff values, and determine its role in guiding clinical decision-making and improving patient outcomes.

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