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Current Research Status and Prospects of Decompensated and Recompensated Hepatitis B Cirrhosis

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ABSTRACT

Decompensated cirrhosis, much like a textile weave that has reached its breaking point under sustained tension, represents the final stage of progressive liver disease. Historically considered an inevitable terminal condition, it is characterized by severe portal hypertension, synthetic dysfunction, and a markedly poor prognosis. However, with the application of highly active antiviral therapy, clinical observations have shown that some patients with decompensated hepatitis B cirrhosis can achieve regression of complications accompanied by stable improvement in liver function. This phenomenon is defined as recompensation. This article reviews recent studies on decompensation and recompensation in hepatitis B cirrhosis, focusing on the definition and evolution of recompensation, its mechanisms, predictive factors, and clinical prognosis. It also discusses research challenges and future directions, aiming to provide references for optimizing individualized management of decompensated hepatitis B cirrhosis patients. Given the high prevalence of chronic liver disease in occupational settings, these insights may have particular relevance for populations with physically demanding occupations, including those in the textile industry.

KEYWORDS

hepatitis B, liver cirrhosis, decompensation, recompensation, textile industry

Hepatitis B virus (HBV) infection remains a global public health issue to this day, significantly impacting the health and labor productivity of workers throughout the textile industry. Chronic HBV infection drives persistent liver inflammation and damage, ultimately progressing from hepatic fibrosis to cirrhosis. Without regular and effective antiviral therapy or long-term poor disease control, the natural course of cirrhosis will significantly accelerate, leading to severe complications such as ascites, variceal bleeding, hepatic encephalopathy, and even hepatocellular carcinoma, with markedly deteriorated prognosis [1,2]. Previous studies suggested that the progression of cirrhosis was irreversible. However, with advances in etiological research, it has been

found that sustained and standardized antiviral therapy can reverse the condition of some decompensated HBV cirrhosis patients, restoring them to the compensated stage, thus introducing the concept of "recompensation." Nevertheless, how to accurately assess the disease status, improve the recompensation rate, and evaluate the clinical outcomes after recompensation urgently requires extensive clinical research for exploration and validation.

DEFINITION OF DECOMPENSATED HBV CIRRHOSIS

Under the influence of long-term HBV infection, the liver exhibits chronic inflammatory manifestations, secreting various cytokines that activate hepatic stellate cells to transform into myofibroblast-like cells. These activated cells extensively synthesize and secrete extracellular matrix, leading to abnormal collagen deposition and disruption of material exchange between hepatocytes and hepatic sinusoidal blood flow [3]. Subsequently, the liver gradually shrinks in volume and distorts in shape, forming multiple hepatocellular nodules separated by broad fibrous bands. This disrupts intrahepatic blood circulation, causing portal hypertension, which eventually progresses to the pathological stage of diffuse hepatic fibrosis, pseudolobule formation, and vascular proliferation both intra-and extrahepatically. This stage is termed cirrhosis [4]. In patients with chronic liver disease, factors such as strenuous physical labor and occupational fatigue commonly observed among workers in the textile industry may further exacerbate disease progression, although the underlying mechanisms remain to be elucidated. During the compensated phase, most patients exhibit no significant clinical symptoms or only manifest as fatigue, poor appetite, and abdominal discomfort. Some patients may develop acute decompensation events (AD) as the disease progresses, defined as the occurrence of ascites, hepatic encephalopathy, gastrointestinal bleeding, bacterial infection, or any combination of these events within two weeks [5,6]. The first occurrence of AD is considered the definitive marker of the transition from the compensated to the decompensated phase. Subsequent recurrent AD episodes significantly impair patients' quality of life.

THE PROPOSAL AND EVOLUTION OF THE CONCEPT OF SECOND COMPENSATION

For patients who have progressed to decompensated cirrhosis, studies have observed that some individuals achieve stable improvement in liver function and no recurrence of AD for at least one year under sustained and effective etiological control. Such patients are currently considered to have entered the recompensated stage [7]. Prior to the clarification of this concept, numerous exploratory studies were conducted domestically

and internationally. In 2017, Aravinthan et al. [8] in Canada observed that some decompensated cirrhosis patients showed clinical improvement or even were removed from the transplant list during waiting for liver transplantation, proposing the hypothesis that cirrhosis may be reversible, though no definitive definition or criteria were provided. The same year, the Chinese Society of Hepatology also introduced this possibility domestically, suggesting that etiological control might reverse the disease course to a non-cirrhotic state. In 2019, domestic guidelines first explicitly mentioned the concept of recompensation but did not specify concrete evaluation criteria. It was not until the 2021 Baveno VII Consensus that the international hepatology community clearly defined the concept of recompensation in decompensated cirrhosis, emphasizing the importance of etiology removal and proposing the discontinuation of medications for complications and other preventive treatments. The consensus identified key indicators of liver function improvement, including serum albumin, total bilirubin, and prothrombin time/international normalized ratio, though specific numerical thresholds were not defined [9]. In 2022, Chinese guidelines further emphasized that the fundamental condition for achieving recompensation in HBV-infected cirrhosis patients is the maintenance of long-term effective antiviral therapy, and based on the Baveno VII Consensus, explicitly defined numerical thresholds for these indicators: serum albumin ≥ 35 g/L, total bilirubin < 34 μ mol/L, and international normalized ratio ≤ 1.5 , sustained for at least 12 months. In the same year, Kim et al. [10] proposed improving the Child-Pugh score to 5 points as the standard for evaluating decompensation. In 2023, the guidelines from the European Association for the Study of the Liver (EASL) and the American Association for the Study of the Liver Disease (AASLD) further explored the threshold and clinical application strategies of novel biomarkers for determining decompensation in cirrhosis, based on refining the liver stiffness measurement (LSM) and the enhanced liver fibrosis score (ELF) for assessing liver fibrosis staging. In 2025, the Chinese Society of Gastroenterology added the recommendation to concurrently administer anti-fibrotic therapy alongside etiology-targeted treatment to actively promote decompensation.

ASSESSMENT TOOLS FOR DECOMPENSATED AND RECOMPENSATED LIVER CIRRHOSIS

Liver Tissue Biopsy

Liver biopsy is the gold standard for pathological diagnosis of liver cirrhosis. The liver tissue obtained through puncture can be observed under microscopy to reveal diffuse hepatic fibrosis with pseudolobule formation, thereby confirming the diagnosis. However, as an invasive procedure, this technique carries potential sampling

errors and the risk of puncture bleeding, resulting in low patient acceptance and limited application in long-term follow-up studies.

Non-Invasive Imaging Assessment Techniques

Ultrasonography Doppler Examination

Ultrasound has become the preferred method for liver cirrhosis screening and long-term follow-up due to its non-invasive, radiation-free, and cost-effective characteristics. Under abdominal color Doppler ultrasound, the liver morphology of cirrhotic patients will exhibit changes, including thickened echoes, widened portal and splenic veins, and slowed portal venous blood flow velocity [11]. However, color Doppler ultrasound relies on operator experience for manual measurements, and further exploration is needed to improve accuracy in assessing the progression of liver cirrhosis and decompensation.

CT Examination

CT examination is widely used in clinical practice due to its high spatial resolution and rapid, stable imaging. In decompensated cirrhosis, contrast-enhanced CT can reveal disproportionate liver volume, nodular surface irregularities, widened hepatic fissures, and heterogeneous parenchymal density [12]. Additionally, contrast-enhanced CT clearly demonstrates the portal venous system and collateral circulation in the esophagogastric fundus and splenorenal regions. Based on portal venous phase images, the Liver Surface Nodularity (LSN) score can be derived to objectively assess portal venous pressure [13]. However, CT examination involves ionizing radiation and is more expensive than color Doppler ultrasound, limiting its application in cases requiring frequent follow-up.

MRI Examination

MRI has become the preferred choice for evaluating liver cirrhosis and screening for hepatocellular carcinoma due to its radiation-free nature and extremely high resolution. T1 and T2-weighted images reveal diffuse signal heterogeneity in the liver parenchyma, as well as regenerative nodules and atypical hyperplastic nodules. Contrast-enhanced scanning further enables precise assessment of hepatic hemodynamics. Due to its prolonged examination time, high cost, and low equipment penetration rate, MRI is primarily used in clinical practice for the diagnosis of complex cases and the precise screening of hepatocellular carcinoma.

Measurement of Liver and Spleen Hardness Values

Vibration-Controlled Transient Elastography (VCTE), point shear-wave elastography (pSWE), two-dimensional shear-wave elastography (2D-SWE), and magnetic resonance elastography (MRE) are non-invasive techniques

capable of measuring patients' liver stiffness (LSM) and spleen stiffness (SSM), serving as crucial tools in recompensation assessment. LSM can be used to determine the criteria for portal hypertension, enabling rapid evaluation of liver cirrhosis severity. The LSM/SSM ratio can differentiate between cirrhotic and non-cirrhotic portal hypertension [14,15]. These elastography techniques, with their non-invasive and reproducible advantages, provide critical evidence for etiological differentiation of portal hypertension and prognostic assessment of liver cirrhosis.

Endoscopic Evaluation

Endoscopy is the most direct tool for evaluating portal hypertension in liver cirrhosis. Endoscopic examination can clearly determine the degree of variceal dilation and high-risk conditions such as the presence of the red sign. The stable regression of varices serves as crucial imaging evidence for the reversal and long-term stabilization of portal hypertension. Additionally, endoscopic hemostasis can be performed for patients with variceal rupture and bleeding. Endoscopy holds significant diagnostic and therapeutic value for liver cirrhosis patients presenting with variceal rupture and bleeding as the initial event.

Serological Models

Serological models are widely applied in clinical and research settings due to their simplicity and readily available data, making the identification of novel models a current research priority. Currently, commonly used clinical models such as the FIB-4 index (Fibrosis-4 index) and the APRI index (Aspartate aminotransferase-to-platelet ratio index) are employed. These models calculate numerical values derived from common clinical indicators for initial screening and assessment of cirrhosis severity, as well as for liver disease risk stratification. However, since they cannot directly reflect changes in liver morphological structure and are influenced by extrahepatic factors, they cannot be used alone as a basis for diagnosing cirrhosis or confirming decompensated status. Instead, in the context of recompensation assessment, serological models serve as complementary tools to imaging modalities. Specifically, a sustained improvement in FIB-4 or APRI index, combined with imaging evidence of reduced portal hypertension such as decreased liver stiffness measurement on transient elastography or resolution of ascites on ultrasound, can collectively support the identification of recompensation.

ANALYSIS OF FACTORS INFLUENCING RECOMPENSATION

Serological Markers

Identifying patients in decompensated cirrhosis and promoting their recompensation, along with screening high-risk populations, has become a critical issue in modern clinical practice. In 2021, Xu et al. [16] identified elevated total protein as a key factor in facilitating recompensation. Subsequently, in 2022, Sharma et al. [17] demonstrated that serum albumin is the most significant survival predictor for advanced compensated cirrhosis. In 2024, Deng et al. [18] further validated the clinical utility of protein as a high-quality prognostic indicator. Current expert consensus holds that both elevated total protein and albumin serve as protective factors for achieving recompensation, with albumin not only predicting recompensation but also indicating better patient outcomes.

Other studies have proposed that serum sodium (Na) is a key indicator reflecting the systemic hemodynamic status in decompensated cirrhosis. In decompensated patients, visceral vasodilation and insufficient effective circulating blood volume often lead to increased non-osmotic secretion of antidiuretic hormone (ADH), resulting in dilutional hyponatremia. Since it is independently associated with short-term mortality, Na has been incorporated into the MELD-Na scoring system to enhance prognostic prediction [19]. In the field of recompensation studies, Wen et al. [20] demonstrated that Na is an independent protective factor for achieving recompensation in HBV cirrhosis patients with ascites as the initial decompensated event. With effective control of the underlying cause, the recovery of Na indicates improvement in neurohumoral regulation. Therefore, some experts consider it one of the earlier and more sensitive indicators of recompensation recovery.

Studies have demonstrated that alpha-fetoprotein (AFP), which reflects hepatic regeneration activity, can serve as a protective factor for achieving regenerative compensation. Kim et al. [10] found that a baseline AFP level ≥ 50 ng/mL is an independent predictor of early regenerative compensation in patients with hepatitis B cirrhosis, with the core mechanism involving the activation and metabolic reprogramming of AFP-positive regenerative hepatocytes [21]. Additionally, in clinical practice, it is essential to combine imaging studies to rule out the possibility of hepatocellular carcinoma, and dynamic monitoring of its trend during treatment is required.

Surgical Treatment

Furthermore, in 2024, Ridola et al. [22] analyzed and concluded that for patients with hepatitis B virus (HBV), hepatitis C, or alcoholic cirrhosis, transjugular intrahepatic portosystemic shunt (TIPS) as first-line therapy can promote decompensation and improve patient survival rates. The following year, Yang Shuofei et al. corroborated this hypothesis, indicating that TIPS significantly alleviates portal hypertension, and early TIPS performed within 72 hours is an independent predictor of postoperative decompensation. Guo Yixuan et al. [23] found that endoscopic treatment combined with partial splenic embolization can also significantly improve variceal conditions, thereby increasing the rate of decompensation.

Others

Finally, clinical studies have identified associations between gender, age, ascites volume, aspartate aminotransferase/alanine aminotransferase (AST/ALT) ratio, and HBV DNA levels with decompensation. However, no definitive consensus has been reached to date, and related basic and clinical research urgently requires updates [24].

RISK OF SECONDARY DECOMPENSATION AFTER RECOMPENSATION

After recompensation, some patients may experience a return to decompensation. Ruan Jiajia et al. reported that 78 cases (39.20%) of patients who had achieved recompensation experienced a recurrence of decompensation. In a retrospective cohort study by Xu Jiajun et al. ,101 cases (49.75%) exhibited recurrent decompensation. Further analysis indicated that factors such as the coexistence of multiple complications, specific biochemical abnormalities (e.g., low serum cholinesterase levels, elevated creatinine levels), a low HBV DNA seroconversion rate at 6 months of antiviral therapy, and a high baseline Child-Pugh score significantly increased the risk of recurrent decompensation. However, there is currently a lack of substantial clinical evidence regarding the long-term clinical outcomes and quality of life in these patients, warranting further investigation.

THE PROFOUND IMPACT OF SECONDARY COMPENSATION ON PATIENT SURVIVAL

For patients with persistent decompensation and recompensation, the quality of life and adverse outcomes remain a focal point in current research. The median survival period for cirrhosis patients after diagnosis is approximately 9 to 12 years. Once the patient enters the decompensated liver function stage, the median survival period can be shortened to 2 years [25]. Studies also indicate that the 5-year survival rate for

decompensated cirrhosis patients without effective intervention is only 14% to 35%, with a grim prognosis [26]. A retrospective study by Chen Anbang et al. revealed that, among patients with hepatitis B virus-related decompensated cirrhosis treated with antiviral therapy combined with carvedilol, those who achieved recompensation within one year had significantly better long-term outcomes. The baseline characteristics between the two groups were generally comparable, although the recompensation group had a lower baseline Child-Pugh score, a lower proportion of portal vein thrombosis, and a higher white blood cell count. The median age was 48.7 years in the recompensation group and 52.6 years in the non-recompensation group. During a median follow-up of 49.6 months, the mortality rate was significantly lower in the recompensation group (3.0%, 1/33) compared to the non-recompensation group (45.9%, 17/37), underscoring the survival benefit associated with achieving recompensation. Zhang et al. [27] found that achieving recompensation can reduce the risk of hepatocellular carcinoma in HBV-related cirrhosis patients, though it remains comparable to that in compensated cirrhosis. However, the survival outcomes for patients who experience recompensation followed by further decompensation still require more clinical research to clarify.

SUMMARY AND OUTLOOK

Currently, as China is a country with a large population of hepatitis B patients, achieving and maintaining effective treatment and long-term management of HBV-induced cirrhosis remains a significant clinical challenge for the health of workers in the textile industry. The risk of progressing to decompensation varies across different populations, evaluation methods differ across hospital levels, and treatment efficacy shows individual variations. This implies the need to intensify efforts to enhance awareness of recompensation among workers in the textile industry, identify universally applicable assessment methods, and update optimal maintenance therapy and follow-up protocols for these laborers. In the future, we should focus on: 1. Exploring the best and clinically accessible indicators to construct non-invasive models for early diagnosis, disease assessment, and survival prediction, and promoting their adoption in primary care hospitals; 2. Conducting extensive clinical studies to clarify the influencing factors of recompensation, identifying relevant high-risk populations, and prioritizing the transition of non-high-risk individuals to high-risk groups while ensuring continuous and effective antiviral therapy for such patients; 3. Monitoring the quality of life and adverse clinical outcomes in patients who experience recompensation followed by decompensation, and establishing the understanding that recompensation is not irreversible; 4. Delving deeper into the pathology of recompensation to investigate mechanisms of liver fibrosis reversal and optimize the best strategies for promotion and maintenance.

Author Contributions

Conceptualization –Li Hongyue, Zhang Qian, Peng Shirui and Xiang Guangming; methodology – Li Hongyue, Peng Shirui and Xiang Guangming; investigation – Li Hongyue, Zhang Qian, Peng Shirui and Xiang Guangming; writing-original draft preparation – Li Hongyue, Zhang Qian, Peng Shirui and Xiang Guangming. All authors have read and agreed to the published version of the manuscript.

Conflicts of Interest

The authors declare no conflict of interest.

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