

World Journal of *Gastroenterology*

Weekly Volume 31 Number 24 June 28, 2025



REVIEW

Dutt K, Vasudevan A, Hodge A, Nguyen TL, Srinivasan AR. Cardiometabolic diseases in patients with inflammatory bowel disease: An evidence-based review. *World J Gastroenterol* 2025; 31(24): 107661 [DOI: [10.3748/wjg.v31.i24.107661](https://doi.org/10.3748/wjg.v31.i24.107661)]

Chen ZL, Wang C, Wang F. Revolutionizing gastroenterology and hepatology with artificial intelligence: From precision diagnosis to equitable healthcare through interdisciplinary practice. *World J Gastroenterol* 2025; 31(24): 108021 [DOI: [10.3748/wjg.v31.i24.108021](https://doi.org/10.3748/wjg.v31.i24.108021)]

MINIREVIEWS

Wang JY, Yi B, Li CY, Xu HQ, Tang SH. Pay attention to the value of liver regeneration in the re-compensation of decompensated cirrhosis. *World J Gastroenterol* 2025; 31(24): 106564 [DOI: [10.3748/wjg.v31.i24.106564](https://doi.org/10.3748/wjg.v31.i24.106564)]

Wang QC, Jiao J, Zhang CQ. Application of artificial intelligence in portal hypertension and esophagogastric varices. *World J Gastroenterol* 2025; 31(24): 108508 [DOI: [10.3748/wjg.v31.i24.108508](https://doi.org/10.3748/wjg.v31.i24.108508)]

ORIGINAL ARTICLE**Retrospective Study**

Gao Z, Wang XY, Shen ZG, Liu JH, Wang XY, Wu SK, Jin X. Real-world comparison of chemotherapy plus bevacizumab with or without immunotherapy as first-line therapy in colorectal cancer. *World J Gastroenterol* 2025; 31(24): 108298 [DOI: [10.3748/wjg.v31.i24.108298](https://doi.org/10.3748/wjg.v31.i24.108298)]

Clinical Trials Study

Zhu JH, Liu X, Zhou W, Xu XN, Sheng WD, Han YL, Qiu XO, Liu YW, Qian YY, Liao Z, Li ZS. Carbonated soft drink for gastric preparation for magnetically controlled capsule endoscopy: An open-label randomized controlled trial. *World J Gastroenterol* 2025; 31(24): 105823 [DOI: [10.3748/wjg.v31.i24.105823](https://doi.org/10.3748/wjg.v31.i24.105823)]

Basic Study

Baek G, Singh R, Ha SE, Cho H, Padmanabhan S, Vishwanath V, Kim MS, Seon D, You J, Lee MY, Ro S. miR-10a-5p and miR-10b-5p restore colonic motility in aged mice. *World J Gastroenterol* 2025; 31(24): 104437 [DOI: [10.3748/wjg.v31.i24.104437](https://doi.org/10.3748/wjg.v31.i24.104437)]

Schulze S, Keshvari S, Miller GC, Bridle KR, Hume DA, Irvine KM. Perisurgical colony stimulating factor one treatment ameliorates liver ischaemia/reperfusion injury in rats. *World J Gastroenterol* 2025; 31(24): 108234 [DOI: [10.3748/wjg.v31.i24.108234](https://doi.org/10.3748/wjg.v31.i24.108234)]

LETTER TO THE EDITOR

Sonbare DJ. Approaches to laparoscopic anatomic liver resection: Does one size fit all? *World J Gastroenterol* 2025; 31(24): 104907 [DOI: [10.3748/wjg.v31.i24.104907](https://doi.org/10.3748/wjg.v31.i24.104907)]

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INDEXING/ABSTRACTING

The WJG is now abstracted and indexed in Science Citation Index Expanded (SCIE), MEDLINE, PubMed, PubMed Central, Scopus, Reference Citation Analysis, China Science and Technology Journal Database, and Superstar Journals Database. The 2025 edition of Journal Citation Reports® cites the 2024 journal impact factor (JIF) for WJG as 5.4; Quartile: Q1. The WJG's CiteScore for 2024 is 8.1.

RESPONSIBLE EDITORS FOR THIS ISSUE

Production Editor: *Wen-Bo Wang*, Production Department Director: *Xiang Li*, Cover Editor: *Jia-Ru Fan*.

NAME OF JOURNAL

World Journal of Gastroenterology

ISSN

ISSN 1007-9327 (print) ISSN 2219-2840 (online)

LAUNCH DATE

October 1, 1995

FREQUENCY

Weekly

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<http://www.wjgnet.com/1007-9327/editorialboard.htm>

PUBLICATION DATE

June 28, 2025

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PUBLISHING PARTNER

Shanghai Pancreatic Cancer Institute and Pancreatic Cancer Institute, Fudan University
Biliary Tract Disease Institute, Fudan University

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ARTICLE PROCESSING CHARGE

<https://www.wjgnet.com/bpg/gerinfo/242>

STEPS FOR SUBMITTING MANUSCRIPTS

<https://www.wjgnet.com/bpg/GerInfo/239>

ONLINE SUBMISSION

<https://www.f6publishing.com>

PUBLISHING PARTNER'S OFFICIAL WEBSITE

<https://www.shca.org.cn>
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Pay attention to the value of liver regeneration in the re-compensation of decompensated cirrhosis

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Specialty type: Gastroenterology and hepatology

Provenance and peer review: Invited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's classification
Scientific Quality: Grade A, Grade B, Grade B

Novelty: Grade A, Grade B, Grade B

Creativity or Innovation: Grade A, Grade A, Grade B

Scientific Significance: Grade A, Grade A, Grade A

P-Reviewer: Ming RJ; Wang Z

Received: March 3, 2025

Revised: April 25, 2025

Accepted: June 6, 2025

Published online: June 28, 2025

Processing time: 117 Days and 14.9 Hours



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Abstract

Conventional wisdom holds that progression from compensated cirrhosis to decompensated cirrhosis is irreversible in the natural history of the disease. However, in recent years, more and more clinical evidence suggests that liver cirrhosis can achieve re-compensation, that is, after effective etiological treatment and complication management, the liver function of partially decompensated patients with cirrhosis has improved and gradually stabilized, and decompensation no longer occurs for a long time. Liver regeneration, as one of the powerful intrinsic abilities of the liver, is the key to the restoration of the structure and complex physiological functions of the damaged liver. Studies have shown that the restoration of liver regeneration in patients with cirrhosis can promote the occurrence of re-compensation, thereby improving the prognosis of patients. At the same time, monitoring liver regeneration indicators is helpful in assessing patients' re-compensation potential for early selection of appropriate treatment options. Insufficient attention has been paid to the role of liver regeneration in the course of liver cirrhosis. Therefore, this article aims to review the value of liver regeneration in the re-compensation of decompensated cirrhosis.

Key Words: Liver regeneration; Decompensated cirrhosis; Re-compensation; Prognosis; α -fetoprotein

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Core Tip: Regeneration is key to the restoration of the structure and complex physiological functions of the damaged liver. When liver regeneration is stagnant, the deterioration of liver function cannot be reversed. Studies have shown that the restoration of liver regeneration in patients with cirrhosis can promote the occurrence of re-compensation, thereby improving the prognosis of patients. Therefore, we should pay attention to the value of liver regeneration in the re-compensation of decompensated patients with cirrhosis.

Citation: Wang JY, Yi B, Li CY, Xu HQ, Tang SH. Pay attention to the value of liver regeneration in the re-compensation of decompensated cirrhosis. *World J Gastroenterol* 2025; 31(24): 106564

URL: <https://www.wjgnet.com/1007-9327/full/v31/i24/106564.htm>

DOI: <https://dx.doi.org/10.3748/wjg.v31.i24.106564>

INTRODUCTION

Cirrhosis is a wound-healing response (fibrogenesis) to liver injury caused by various causes. As the disease progresses, the normal structure of the liver is gradually replaced by regenerating nodules[1,2]. Clinically, the progression of cirrhosis is usually silent until the portal venous pressure increases and the liver function deteriorates into a clinical phenotype. At this time, complications such as ascites, variceal hemorrhage, and hepatic encephalopathy may occur, marking the occurrence of liver decompensation. The prognosis of decompensated cirrhosis is poor, with a median survival of only 2 to 3 years in patients with decompensated cirrhosis compared with the median survival of patients with compensated cirrhosis of more than 12 years[3].

Therefore, the occurrence of decompensation is an important watershed in the clinical course of liver cirrhosis, which means that the risk of further decompensation events and mortality is significantly increased. Fortunately, studies have shown that after effective etiological treatment, the liver function and tissue structure of some patients with decompensated cirrhosis can gradually return to the compensated phase and remain stable for a long time. In a prospective cohort study by Premkumar *et al*[4], among 1152 patients with hepatitis C virus (HCV)-associated decompensated cirrhosis, 284 (24.7%) achieved re-compensation after treatment with effective direct-acting antivirals. In a multicenter prospective study by Wang *et al*[5], 56.2% (159/283) of patients with hepatitis B virus (HBV)-associated decompensated cirrhosis achieved re-compensation after 120 weeks of antiviral therapy. An observational study by Hofer *et al*[6] on decompensated alcohol-related cirrhosis showed that persistent abstinence resulted in hepatic re-compensation in 18.1% (37/204) of patients. This re-compensation breaks the traditional concept that liver cirrhosis is irreversible and is of great significance for improving the prognosis of patients. However, the current high-quality research and clinical data on liver re-compensation are still limited, and the re-compensation of decompensated cirrhosis of some etiologies has not been fully proven. In addition, not all patients can achieve re-compensation after effective etiologic treatment, which makes us to consider the possible factors that affect the occurrence of re-compensation and adopt an individualized treatment strategy according to the re-compensation situation of different patients. Patients with high re-compensation potential should be encouraged to continue active etiological, anti-inflammatory, and anti-fibrotic therapy, and prevention and treatment of complications if necessary, to promote hepatocyte repair and regeneration, to improve the quality of life of patients. Those who are unlikely to achieve re-compensation should be prepared for liver transplantation in the long term. The liver has a high regenerative potential, which is key to hepatic homeostasis and maintaining liver function and structure after liver injury. The lack of liver tissue regeneration based on cirrhosis may be one of the factors leading to the difficulty of recovery or even continuous deterioration of liver function in decompensated patients, thus affecting the re-compensation and long-term prognosis of patients. At present, some studies have pointed out that gender, severity of liver disease, severity of portal hypertension, systemic inflammatory response, and other factors may affect the re-compensation of patients with decompensated liver cirrhosis. However, there is still a lack of sufficient understanding of the role of liver regeneration. The purpose of this article is to review the potential of liver regeneration in the re-compensation of decompensated cirrhosis to promote the establishment of personalized treatment for such high-risk patients.

LIVER REGENERATION AFTER LIVER INJURY

The liver is a vital organ that has the functions of metabolism, detoxification, excretion, and biotransformation[7]. Liver regeneration is one of the most striking and powerful innate abilities of the liver[8], which allows the liver to regain its original size and function quickly after injury. A variety of pathological factors can lead to liver damage, such as hepatitis virus infection, exposure to hepatotoxic drugs, and after liver resection. Sometimes, these conditions are triggers for liver regeneration, but damage, if severe and persistent, can impair liver regeneration, leading to fatal liver failure[9]. There is a strong association between chronic liver damage and decreased liver regeneration. A retrospective study by Aierken *et al*[10] found that the liver regeneration rate decreased with an increase in the incidence of liver fibrosis, and that liver regeneration in patients with cirrhosis was significantly slower and incomplete compared with the normal liver, which may be related to a significant reduction in the key factors affecting liver regeneration, such as tumor necrosis factor α , interleukin (IL)-6, and hepatocyte growth factor.

The pattern of liver regeneration varies greatly depending on the degree and type of liver injury. The two main pathways of liver regeneration are the proliferation of mature hepatocytes and the expansion of liver progenitor cells (LPCs). The liver has a slower renewal rate under homeostatic conditions. It relies on the self-replication of different hepatocyte subsets to achieve dynamic reproduction and maintain the function and quality of the liver. In partial hepatic resection or other models of acute liver injury, residual hepatocytes not only proliferate to refill the damaged area of the liver but also upregulate their gene expression levels to compensate for lost liver function, resulting in a significantly higher rate of regeneration than hepatocytes during steady state [7,11]. LPCs are proliferative epithelial cells that grow in Hering's ducts, expressing the hepatoblast marker α -fetoprotein (AFP). They are considered dual-potent precursor cells due to their ability to differentiate into hepatocytes and cholangiocytes *in vitro*. LPCs are not observable in the liver of normal adults but appear and dilate in severe or chronic liver injury, and their number depends on the severity of the disease [12]. In severe liver fibrosis/cirrhosis, a large amount of parenchymal tissue is lost, and hepatocyte proliferation is impaired due to excessive inflammation, scarring and epithelial abnormalities. At this time, necrotic hepatocytes and immune cells can produce LPCs proliferation activator to initiate the liver regeneration process induced by LPCs, and the activated LPCs differentiate into hepatocytes and cholangiocytes, refill the damaged epithelial cells and promote the recovery of liver function [13]. LPCs are mainly derived from the dedifferentiation of hepatocytes or cholangiocytes, and it has been suggested that hematopoietic stem cells and myofibroblasts can also transform into LPCs [14]. In some cases, hepatocytes and cholangiocytes can also proliferate as facultative stem cells to each other. When one of the epithelial cells cannot regenerate, an alternative regeneration regimen is activated. Elevated serum AFP levels in patients with active cirrhosis after hepatocellular carcinoma have been excluded, which may indicate hepatic progenitor cell mobilization and proliferation. However, the origin and characteristics of such AFP-expressing cells are not fully understood. The study by Nakano *et al* [15] identified a unique population of AFP-expressing cells induced by Jagged1/Notch2 signaling in mouse fibrotic liver. It was found that in fibrotic liver tissues, the expression of Jagged1 (ligand protein) in myofibroblasts was significantly increased, which stimulated the Notch2 (receptor protein) signaling of adjacent AFP-expressing cells, thereby inducing the mobilization and proliferation of AFP-expressing cells. These AFP-positive cells exhibit the characteristics of immature hepatocytes and have high proliferative capacity, which may contribute to the regeneration of fibrotic liver and are associated with the prolonged survival time after partial hepatectomy in mice with fibrotic liver. Acute-on-chronic liver failure (ACLF) is an acute liver injury and rapid deterioration of liver function based on chronic liver disease, often complicated by bacterial infection, and its short-term mortality rate is high. Xiang *et al* [16] found that liver fibrosis and bacterial infection had significant inhibitory effects on liver regeneration induced by acute liver injury in a mouse model of ACLF, which was achieved by impairing the IL-6/STAT3 pathway, promoting regeneration and enhancing the interferon (IFN)- γ /STAT1 pathway, and inhibiting regeneration. STAT3 and STAT1 are both members of the STAT protein family and are key intracellular signal transduction proteins, which are complementary and often antagonistic. IL-6 and IL-22 predominantly activate STAT3, and IFN- γ predominantly activates STAT1. The balance of the two plays a key role in controlling liver damage and regeneration (Figure 1). IL-22Fc is a recombinant fusion protein consisting of two human IL-22 molecules linked to a constant region of immunoglobulin. IL-22Fc has been found to activate STAT3 and attenuate STAT1 in the liver, promoting liver regeneration and mitigating bacterial infections to improve ACLF survival. It can be seen that effectively restarting liver regeneration in patients with cirrhosis is essential to improve the prognosis, and adjusting the signaling pathways related to liver regeneration is a therapeutic strategy worthy of further consideration.

CONCEPT OF RE-COMPENSATION AND ITS CURRENT RESEARCH PROGRESS

The concept of re-compensation implies that the structural and functional changes in cirrhosis are at least partially restored after the etiology of cirrhosis has been eliminated. In 2021, the Baveno VII consensus proposed for the first time a unified standard for the definition of liver re-compensation, namely: (1) Clearance/inhibition/cure of the primary cause of liver cirrhosis, *i.e.*, elimination of HCV, continuous inhibition of HBV, and continuous abstinence from alcohol for the treatment of alcoholic cirrhosis; (2) Resolution of ascites (discontinuation of diuretics), resolution of hepatic encephalopathy (discontinuation of lactulose/rifaximin) and absence of recurrent variceal bleeding for at least 12 months; and (3) Stable improvement in liver function indicators [albumin (ALB), international normalized ratio (INR) and bilirubin] [17]. Since its inception, the Baveno VII standard has been validated in several studies. However, the generation of new concepts always has to go through a long and iterative process of revision and refinement, and we need to pay attention to the current limitations of the Baveno VII standard.

According to Baveno VII, the core of the concept of hepatic re-compensation is to address the underlying etiology of cirrhosis, which is essential to initiate the re-compensation process. However, most of the existing studies on etiological treatment are based on patients with HBV, HCV, and alcohol-related liver cirrhosis, and there are still insufficient data on the effect of etiological treatments other than alcohol abstinence and antiviral therapy on re-compensation. For liver diseases of different etiologies, particularly less common diseases such as autoimmune hepatitis (AIH), primary biliary cholangitis (PBC), and primary sclerosing cholangitis, there is still a lack of accepted criteria for success in treating the cause. In addition, the definition of successful etiological treatment in patients with multiple overlapping etiologies of liver disease is debatable. In a retrospective study of 42 patients with PBC and decompensated cirrhosis, Hofer *et al* [18] defined the criteria for etiological treatment success as normalization of bilirubin and alkaline phosphatase reduced to ≤ 1.5 times the upper limit of normal (determined to be a biochemical response to UDCA by reference to Paris II criteria) with ursodeoxycholic acid (UDCA, the first-line agent for PBC), and 7 patients (16.7%) achieved re-compensation. Another pilot study demonstrated that decompensated cirrhosis due to AIH can achieve re-compensation in some

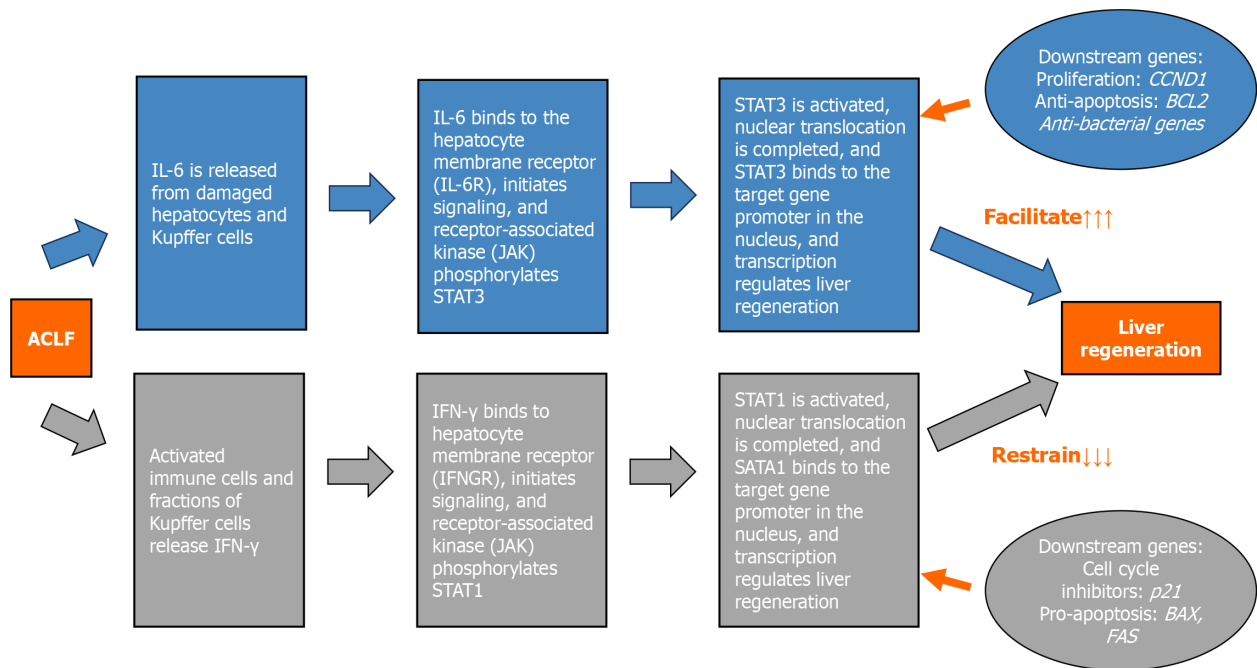


Figure 1 Balance of hepatic regeneration pathways in patients with acute-on-chronic liver failure. ACLF: Acute-on-chronic liver failure; IL-6: Interleukin-6; IFN-γ: Interferon-γ.

patients after successful immunosuppressive therapy (4/21)[19]. These studies have small sample sizes, limited statistical power, and limited extrapolation of results. However, they also suggest the feasibility of re-compensation in cirrhosis of multiple etiologies, which still needs further study.

In addition to successful etiological treatment, the Baveno VII criteria emphasize the resolution of complications related to hepatic decompensation. Transjugular intrahepatic portal system shunt (TIPS) is an effective intervention for variceal hemorrhage or refractory ascites in patients with cirrhosis by reducing the portal pressure gradient and not directly addressing the removal of the cause of cirrhosis[20]. Gao *et al*[21] studied patients with decompensated cirrhosis of different etiologies, including chronic hepatitis B, hepatitis C, alcoholic liver disease, metabolic dysfunction-associated steatotic liver disease, autoimmune liver disease and cholestatic liver disease. Approximately one-third of decompensated patients were found to recompensate for cirrhosis after TIPS, and re-compensation was more likely to occur in younger patients and in patients whose portal venous pressure gradient dropped below 12 mmHg after TIPS. This suggests that therapeutic interventions for portal hypertension may be a key factor in promoting the re-compensation of cirrhosis of different etiologies. Although the Baveno VII consensus emphasizes the importance of addressing the underlying cause to achieve complication re-compensation, there is still a lack of sufficient data to support whether etiological treatment can achieve re-compensation in patients with advanced cirrhosis and severe complications. Gao *et al*[21] concluded that since the occurrence of cirrhosis decompensation is mainly based on the progression of portal hypertension, reversal of portal hypertension by TIPS is essential to achieve re-compensation, and etiological treatment alone is far from sufficient to suppress the recurrence of life-threatening complications of portal hypertension. More data are needed to evaluate the relationship between portal hypertension mitigation and re-compensation to complement and refine the concept of cirrhosis re-compensation.

Although the Baveno VII consensus includes stable improvement in liver function as one of the definitions of re-compensation and provides reference indicators (ALB, INR and bilirubin), it does not establish specific cut-off values for the relevant parameters. Different studies may have chosen different variables and cutoff values for assessing liver function re-compensation. For example, the study of Aravinthan *et al*[22] improved the model for end-stage liver disease (MELD) score to < 15 as the standard for liver function re-compensation in patients with decompensated alcohol-related liver disease, and the study of Kim *et al*[23] defined re-compensation as the recovery of cirrhosis status to a Child-Pugh score of 5 points. Formulating unified and accurate re-compensation standards for liver function will help in future related research and the diagnosis of re-compensation groups in clinical practice. Wang *et al*[5] proposed a precise definition of stable improvement in liver function [MELD score < 10 and/or Child-Pugh class A, *i.e.*, ALB > 35 g/L and INR < 1.50 and total bilirubin (TBIL) < 34 μmol/L]. This was included in China's latest Guidelines for the Prevention and Treatment of Chronic Hepatitis B (version 2022). However, that study only included patients with HBV-related decompensated cirrhosis treated with entecavir, and it is still unclear whether these criteria can be applied to other causes of cirrhosis or other treatments. As for whether liver function indicators other than ALB, INR and bilirubin, or scores other than MELD and Child-Pugh can better evaluate liver function re-compensation, few studies are currently available. Future research needs to continue to improve the definition of liver function improvement to meet the needs of re-compensation.

PAY ATTENTION TO THE VALUE OF LIVER REGENERATION IN RE-COMPENSATION OF DECOMPENSATED LIVER CIRRHOSIS

Role of liver regeneration in the re-compensation of cirrhosis

Although the Baveno VII consensus emphasizes that re-compensation should be accompanied by improvement in liver histological structure, the current clinical judgment of re-compensation is mainly based on the evaluation of complications and liver function, and liver histopathological studies supporting re-compensation of cirrhosis are still lacking. The pathophysiology of hepatic re-compensation is currently unclear, but understanding the mechanisms of cirrhosis decompensation can help identify potential compensatory targets. By reviewing the pathological features of the formation of cirrhosis, Bedossa[24] proposed three significant cirrhosis mechanisms of reversal: Collagen degradation, hepatocyte regeneration, and reconstitution of liver lobular structure. Portal hypertension is an essential driver of decompensation in cirrhosis. Its associated complications are key to determining the prognosis of cirrhosis and develop based on changes in liver structure and function. Severe structural disturbances in the liver, because of progressive fibrosis and vascular occlusion of the liver, followed by parenchymal disappearance and tissue collapse, are the main mechanisms of portal hypertension. Patients with cirrhosis and severe lobular structural disorders lack an appropriate structural framework (basement membrane scaffold), which prevents neohepatocytes from filling collapsed lobules. In this case, hepatocyte regeneration is blocked, resulting in a single regenerative nodule that fails to produce normal liver structure, and the enlarged regenerated nodule continues to compress the hepatic vein, resulting in tissue congestion, hepatocyte necrosis, and a new regeneration cycle, ultimately leading to end-stage irreversible liver disease[25-27]. Successful etiological treatment and antifibrosis can promote collagen degradation, hepatocyte regeneration, and replacement of disappeared fibrotic tissues by newly formed hepatocytes, which can lead to the reconstitution of hepatic lobular structure and translobular blood flow, as well as reduction of portal hypertension and promotion of re-compensation. If the liver lacks fibrosis resolution potential and internal regeneration, cirrhosis may not be reversed, even if the cause is effectively controlled[24]. Many studies have shown that factors such as baseline liver function, gender, age, portal venous pressure gradient, and systemic inflammation may affect the re-compensation of patients. However, there is still insufficient attention to the role of changes in liver regeneration capacity in the re-compensation of cirrhosis.

A few studies have confirmed liver regeneration's effect on the re-compensation of decompensated cirrhosis. Previous studies have demonstrated that AFP is a biomarker of hepatic progenitor cell proliferation. Elevated AFP levels may also indicate the severity of liver destruction and subsequent liver regeneration, which is often observed in patients with a variety of acute and chronic liver diseases, including cirrhosis. Under normal physiological conditions, AFP is produced by the yolk sac and the hepatocytes of the fetus and is gradually replaced by ALB at 2-3 months after birth. After liver injury, serum AFP levels positively correlate with hepatocyte regeneration capacity, possibly because of immature hepatic progenitor cells with characteristics similar to those of fetal hepatocytes. Wen *et al*[28] conducted a study on patients with HBV-associated liver cirrhosis with ascites as the single first decompensation event. They found that AFP was a relevant factor in re-compensation, suggesting that liver regeneration is not negligible in cirrhosis re-compensation. A retrospective study by Kim *et al*[23] found that high AFP level at baseline (≥ 50 ng/mL) was an essential predictor of re-compensation after antiviral therapy in patients with HBV-associated cirrhosis decompensation. A prediction score model for re-compensation, including AFP, was also constructed, namely the BC2AID (Bilirubin-severe Complication-Alpha-fetoprotein-Alanine aminotransferase-INR-Duration of decompensation before NUC therapy) score. The value of this scoring model in predicting re-compensation of decompensated cirrhosis within 1 year after antiviral treatment is better than that of traditional prediction models [Child-Pugh, MELD, MELD-Na, and BE3A (body mass index, encephalopathy, ascites, and serum levels of alanine aminotransferase and ALB) scores]. It can better predict the long-term mortality of patients at 6-36 months. It can be seen that the clinical prediction model that integrates the evaluation of liver regeneration ability has potential research value in predicting the re-compensation of decompensated cirrhosis. Zhang *et al*[29] studied 136 patients with ACLF based on HBV-associated cirrhosis, and found that 56 (41.18%) patients recovered to the compensated phase within 1 year of antiviral therapy. The AFP in the recompensated group was significantly increased, suggesting that a stronger capacity for liver regeneration may be more conducive to re-compensation. Some clinical studies have shown that serum AFP levels are a good prognostic indicator, especially in patients with ACLF. Wang *et al*[30] found that the parameter \log_{10} AFP was a valuable predictor of HBV-ACLF prognosis and that the short-term survival rate of ACLF patients with \log_{10} AFP ≥ 2 was higher. To evaluate the prognosis of patients from both the dimensions of liver regeneration and impaired liver function, Wang *et al*[31] created and validated a new prognostic model for HBV-ACLF with the following mathematical formula: TACIA score = $0.003 \times \text{Tbil} (\mu\text{mol/L}) + 0.036 \times \text{age} + 0.009 \times \text{creatinine} (\mu\text{mol/L}) + 0.525 \times \text{INR} - 0.003 \times \text{AFP} (\text{ng/mL})$. The new scoring system combines the clinical parameters of organ damage and liver regeneration, which can effectively assess the re-compensation of liver function and predict the prognosis of 90-d survival. In addition to AFP, platelets have been reported to predict liver regeneration. A study by Yamazaki *et al*[32] showed a significant delay in liver volume recovery after hepatectomy when platelet recovery rates were low. This may be related to the regeneration of liver parenchyma between platelets and hepatocytes through cytokines such as hepatocyte growth factor, vascular endothelial growth factor, serotonin, and thrombopoietin. Platelet-mediated liver regeneration is inhibited when platelet-hepatocyte interactions are interrupted. Thrombocytopenia is often the first problem in chronic liver disease and early cirrhosis, and the severity of cirrhosis is also an important factor in platelet count. Therefore, focusing on changes in platelet count may help us monitor disease progression in patients with cirrhosis and assess the patient's regeneration ability to predict re-compensation. This is supported by the study of Deng *et al*[33], which explored the predictors of re-compensation after entecavir in treating HBV-related decompensated cirrhosis and found that higher platelets at 24 weeks of treatment were among the best predictors. The construction of a new re-compensation prediction model based on platelet indexes may be worthy of further research in the future, which

Table 1 Multidimensional assessment of cirrhosis re-compensation

Evaluate dimensions		Assessment methods
Liver histopathological evaluation	Degree of liver fibrosis	Traditional semi-quantitative scores (METAVIR, ISHAK, SCHEUER, KNODELL scores), CPA
	Hepatocyte regeneration	Platelet, AFP, precursor cell (LPCs, hematopoietic stem cells, <i>etc.</i>) markers
	Hepatic lobular reconstruction	Semi-quantitative assessment of pathology, GS-index
Liver function assessment	Hepatic synthesis and storage capacity	ALB, prealbumin, ALT, PT, INR, TPO, TC
	Liver biotransformation and detoxification	Bilirubin, blood ammonia, bile acids
	Hepatocyte regeneration and repair ability	Platelet, AFP, precursor cell (LPCs, hematopoietic stem cells, <i>etc.</i>) markers
	Common scoring models	MELD, MELD-Na, iMELD, Child-Pugh, <i>etc.</i>
Assessment of clinical complications	No clinical complications occur for a more extended period (at least one year) after the cause is controlled and the complications are managed	

CPA: Collagen percentage area; AFP: α -fetoprotein; LPCs: Liver progenitor cells; GS-index: Glutamine synthetase index; ALB: Albumin; ALT: Alanine aminotransferase; PT: Prothrombin time; INR: International normalized ratio; TPO: Thrombopoietin; TC: Total cholesterol; MELD: Model for end-stage liver disease.

can comprehensively reflect liver regeneration, inflammatory response, coagulation function, *etc.* However, both the BC2AID score and the TACIA score still lack validation in different large and diverse cohorts, and the scores are all based on patients with HBV-related liver disease. In contrast, patients with liver disease of other causes are not included. In the future, the predictive value of AFP, platelets, and other indicators in re-compensation should be further studied in patients with liver cirrhosis caused by different etiologies (including alcoholic, autoimmune, *etc.*), to build a more versatile clinical prediction model. Liver regeneration capacity is the key to structural reversal and liver function recovery in cirrhosis, and actively maintaining liver regeneration capacity may be a therapeutic strategy to achieve re-compensation.

Different complications also affect the re-compensation and prognosis of patients with cirrhosis. Wen *et al*[28] included only patients with decompensated ascites, and studies on other complications are lacking. He *et al*[34] found that after effective etiological treatment, the re-compensation rate was significantly lower in patients with variceal hemorrhage as the first decompensated event than in patients with ascites as the first decompensated event. In addition, there is a higher risk of further decompensation, hepatocellular carcinoma, death or liver transplantation after re-compensation. This suggests that patients with decompensation who have variceal bleeding as their first decompensated event have a worse prognosis. More and more extensive studies should be conducted to explore the re-compensation of this subset of patients and to pay attention to the role of liver regeneration.

Liver cirrhosis re-compensation was evaluated in combination with the liver regeneration index

The evaluation of liver cirrhosis re-compensation mainly includes liver histopathology (degree of liver fibrosis, hepatocyte regeneration, and liver lobular reconstruction), liver function, and complications, but hepatocyte regeneration evaluation still needs improvement (Table 1). After decompensation of cirrhosis, the restoration of liver function includes the restoration of liver reserve capacity and regeneration. When the ability of liver regeneration is more potent than that of injury, hepatocyte regeneration repairs damaged tissues to rebuild liver lobules, promoting the reversal of liver structure and function, and the prognosis of patients is better. However, when the damage is too extensive or the liver's regeneration ability is weakened, it may be difficult for patients to achieve compensation, and the prognosis is poor. Therefore, evaluation of hepatocyte regeneration deserves our attention.

Clinically, the assessment of liver regeneration ability should be comprehensively judged using multi-dimensional indicators, including laboratory indicators, imaging evaluation, histological examination, *etc.* AFP is an essential indicator for evaluating liver regeneration ability. It can be combined with dynamic monitoring of platelets, liver function indicators (ALB, bilirubin, INR, *etc.*), Child-Pugh scores, and regular abdominal computed tomography (CT) or magnetic resonance imaging (MRI) examinations for patients with decompensated cirrhosis to rule out the existence of hepatocellular carcinoma. On this basis, increased AFP and platelets, and an improvement in liver function may indicate hepatocellular regeneration. Laboratory indicators have low specificity and are susceptible to interference factors. For example, nutritional status affects ALB indicators, and hypersplenism disturbs platelets. Imaging methods such as ultrasound, CT, MRI, *etc.*, are non-invasive and intuitive, which can quantify liver volume changes, but cannot distinguish regenerated liver cells from fibrotic tissue. Hepatic elastography can evaluate dynamic changes in liver fibrosis. A decrease in cirrhosis value (LSM) may indicate that liver regeneration is accompanied by reduced fibrosis, but this examination cannot directly reflect the proliferation activity of hepatocytes. In addition, since hepatocyte regeneration mainly comes from precursor cells, the evaluation value of precursor cell markers such as EpCAM, CK 19, and SOX9, is also worth consideration[27]. Liver biopsy tissue can be used in clinical practice to perform immunohistochemistry, intuitively localize precursor cells expressing specific markers, and evaluate their distribution and number. The increase in marker-

positive cellularity in chronic liver disease/cirrhosis may indicate compensatory activation of liver precursor cells, while sustained high expression may indicate the risk of liver failure or a tendency to cancer. However, this invasive biopsy is unsuitable for dynamic monitoring. Serological markers, such as CYFRA21-1 and EpEX in the serum, can also be detected by ELISA. They can be dynamically monitored to observe peaks and downward trends to evaluate regeneration. However, serum markers have low sensitivity and specificity, and exclusion from other diseases, such as malignant tumors, is required. The clinical application is limited. In general, clinical evaluation of liver regeneration should be multi-dimensional and combined, and need further research in the future.

CONCLUSION

In recent years, increasing clinical evidence indicates the possibility of re-compensation in decompensated cirrhosis, in which structural and functional changes in cirrhosis are at least partially reversed after the cause of cirrhosis has been eliminated. Studies have confirmed that the recovery of liver regeneration ability in patients with cirrhosis can promote the occurrence of re-compensation and improve the patient's prognosis. Patients' liver regeneration ability can be evaluated in multiple dimensions by monitoring the levels of AFP, platelets, and other indicators, detecting precursor cell-specific markers, and establishing reasonable individualized treatment strategies. In short, we should actively maintain the liver regeneration ability of patients with decompensated cirrhosis to promote the occurrence of re-compensation and further improve the clinical evaluation methods in hepatocyte regeneration.

FOOTNOTES

Author contributions: Wang JY wrote the manuscript; Yi B, Li CY and Xu HQ revised the manuscript; Tang SH reviewed the manuscript. All authors have reviewed and approved the final version of the manuscript. Wang JY and Yi B contributed equally to this work as co-first authors.

Conflict-of-interest statement: The authors do not choose to declare any conflict of interest related directly or indirectly to the subject of this article.

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S-Editor: Qu XL

L-Editor: A

P-Editor: Wang WB

REFERENCES

- 1 **Emenena I**, Emenena B, Kweki AG, Aiwuyo HO, Osarenkhoe JO, Iloeje UN, Ilerhunmwuwa N, Torere BE, Akinti O, Akere A, Casimir OE. Model for End Stage Liver Disease (MELD) Score: A Tool for Prognosis and Prediction of Mortality in Patients With Decompensated Liver Cirrhosis. *Cureus* 2023; **15**: e39267 [RCA] [PMID: 37342753 DOI: 10.7759/cureus.39267] [FullText]
- 2 **Tsai FJ**, Yang PY, Chen CJ, Li JP, Li TM, Chiou JS, Cheng CF, Chuang PH, Lin TH, Liao CC, Huang SM, Ban B, Liang WM, Lin YJ. Decreased overall mortality rate with Chinese herbal medicine usage in patients with decompensated liver cirrhosis in Taiwan. *BMC Complement Med Ther* 2020; **20**: 221 [RCA] [PMID: 32664975 DOI: 10.1186/s12906-020-03010-6] [FullText] [Full Text(PDF)]
- 3 **Chandna S**, Zarate ER, Gallegos-Orozco JF. Management of Decompensated Cirrhosis and Associated Syndromes. *Surg Clin North Am* 2022; **102**: 117-137 [RCA] [PMID: 34800381 DOI: 10.1016/j.suc.2021.09.005] [FullText]
- 4 **Premkumar M**, Dhiman RK, Duseja A, Mehtani R, Taneja S, Gupta E, Gupta P, Sandhu A, Sharma P, Rathi S, Verma N, Kulkarni AV, Bhujade H, Chaluvashetty SB, Kalra N, Grover GS, Nain J, Reddy KR. Re-compensation of Chronic Hepatitis C-Related Decompensated Cirrhosis Following Direct-Acting Antiviral Therapy: Prospective Cohort Study From a Hepatitis C Virus Elimination Program. *Gastroenterology* 2024; **167**: 1429-1445 [RCA] [PMID: 39181168 DOI: 10.1053/j.gastro.2024.08.018] [FullText]
- 5 **Wang Q**, Zhao H, Deng Y, Zheng H, Xiang H, Nan Y, Hu J, Meng Q, Xu X, Fang J, Xu J, Wang X, You H, Pan CQ, Xie W, Jia J. Validation of Baveno VII criteria for re-compensation in entecavir-treated patients with hepatitis B-related decompensated cirrhosis. *J Hepatol* 2022; **77**: 1564-1572 [RCA] [PMID: 36038017 DOI: 10.1016/j.jhep.2022.07.037] [FullText]
- 6 **Hofer BS**, Simbrunner B, Hartl L, Jachs M, Balcar L, Paternostro R, Schwabl P, Semmler G, Scheiner B, Trauner M, Mandorfer M, Reiberger T. Hepatic re-compensation according to Baveno VII criteria is linked to a significant survival benefit in decompensated alcohol-related cirrhosis. *Liver Int* 2023; **43**: 2220-2231 [RCA] [PMID: 37469291 DOI: 10.1111/liv.15676] [FullText]
- 7 **Huang R**, Zhang X, Gracia-Sancho J, Xie WF. Liver regeneration: Cellular origin and molecular mechanisms. *Liver Int* 2022; **42**: 1486-1495

- [*RC A*] [PMID: 35107210 DOI: 10.1111/liv.15174] [*FullText*]
- 8 **Qi J**, Dai Y, Sun X, Liu C. Mechanism of liver regeneration: 20-year bibliometric analyses. *Front Pharmacol* 2023; **14**: 1190559 [*RC A*] [PMID: 37383706 DOI: 10.3389/fphar.2023.1190559] [*FullText*]
- 9 **Ozaki M**. Cellular and molecular mechanisms of liver regeneration: Proliferation, growth, death and protection of hepatocytes. *Semin Cell Dev Biol* 2020; **100**: 62-73 [*RC A*] [PMID: 31669133 DOI: 10.1016/j.semcd.2019.10.007] [*FullText*]
- 10 **Aierken Y**, Kong LX, Li B, Liu XJ, Lu S, Yang JY. Liver fibrosis is a major risk factor for liver regeneration: A comparison between healthy and fibrotic liver. *Medicine (Baltimore)* 2020; **99**: e20003 [*RC A*] [PMID: 32481371 DOI: 10.1097/MD.00000000000020003] [*FullText*]
- 11 **Sun T**, Pikirolek M, Orsini V, Bergling S, Holwerda S, Morelli L, Hoppe PS, Planas-Paz L, Yang Y, Ruffner H, Bouwmeester T, Lohmann F, Terracciano LM, Roma G, Cong F, Tchorz JS. AXIN2(+) Pericentral Hepatocytes Have Limited Contributions to Liver Homeostasis and Regeneration. *Cell Stem Cell* 2020; **26**: 97-107.e6 [*RC A*] [PMID: 31866224 DOI: 10.1016/j.stem.2019.10.011] [*FullText*]
- 12 **Yin Y**, Kong D, He K, Xia Q. Regeneration and activation of liver progenitor cells in liver cirrhosis. *Genes Dis* 2021; **8**: 623-628 [*RC A*] [PMID: 34291133 DOI: 10.1016/j.gendis.2020.07.016] [*FullText*] [*Full Text(PDF)*]
- 13 **Qian Y**, Shang Z, Gao Y, Wu H, Kong X. Liver Regeneration in Chronic Liver Injuries: Basic and Clinical Applications Focusing on Macrophages and Natural Killer Cells. *Cell Mol Gastroenterol Hepatol* 2022; **14**: 971-981 [*RC A*] [PMID: 35738473 DOI: 10.1016/j.jcmgh.2022.05.014] [*FullText*] [*Full Text(PDF)*]
- 14 **So J**, Kim A, Lee SH, Shin D. Liver progenitor cell-driven liver regeneration. *Exp Mol Med* 2020; **52**: 1230-1238 [*RC A*] [PMID: 32796957 DOI: 10.1038/s12276-020-0483-0] [*FullText*] [*Full Text(PDF)*]
- 15 **Nakano Y**, Nakao S, Sumiyoshi H, Mikami K, Tanno Y, Sueoka M, Kasahara D, Kimura H, Moro T, Kamiya A, Hozumi K, Inagaki Y. Identification of a novel alpha-fetoprotein-expressing cell population induced by the Jagged1/Notch2 signal in murine fibrotic liver. *Hepatology* 2017; **1**: 215-229 [*RC A*] [PMID: 29404455 DOI: 10.1002/hep4.1026] [*FullText*] [*Full Text(PDF)*]
- 16 **Xiang X**, Feng D, Hwang S, Ren T, Wang X, Trojnar E, Matyas C, Mo R, Shang D, He Y, Seo W, Shah VH, Pacher P, Xie Q, Gao B. Interleukin-22 ameliorates acute-on-chronic liver failure by reprogramming impaired regeneration pathways in mice. *J Hepatol* 2020; **72**: 736-745 [*RC A*] [PMID: 31786256 DOI: 10.1016/j.jhep.2019.11.013] [*FullText*]
- 17 **de Franchis R**, Bosch J, Garcia-Tsao G, Reiberger T, Ripoll C; Baveno VII Faculty. Baveno VII - Renewing consensus in portal hypertension. *J Hepatol* 2022; **76**: 959-974 [*RC A*] [PMID: 35120736 DOI: 10.1016/j.jhep.2021.12.022] [*FullText*]
- 18 **Hofer BS**, Burghart L, Halilbasic E, Simbrunner B, Petrenko O, Mandorfer M, Stättermayer AF, Trauner M, Reiberger T. Evaluation of potential hepatic recompensation criteria in patients with PBC and decompensated cirrhosis. *Aliment Pharmacol Ther* 2024; **59**: 962-972 [*RC A*] [PMID: 38409879 DOI: 10.1111/apt.17908] [*FullText*]
- 19 **Hofer B**, Burghart L, Treiber S, Halilbasic E, Mandorfer M, Trauner M, Reiberger T, Stattermayer AF. THU-183-YI Immunosuppressive treatment facilitates hepatic recompensation in patients with decompensated cirrhosis due to autoimmune hepatitis-a pilot study. *J Hepatol* 24; **80**: S341-S342 [DOI: 10.1016/s0168-8278(24)01151-6] [*FullText*]
- 20 **Ridola L**, Del Cioppo S. Advancing hepatic recompensation: Baveno VII criteria and therapeutic innovations in liver cirrhosis management. *World J Gastroenterol* 2024; **30**: 2954-2958 [*RC A*] [PMID: 38946869 DOI: 10.3748/wjg.v30.i23.2954] [*FullText*] [*Full Text(PDF)*]
- 21 **Gao L**, Li MB, Li JY, Liu Y, Ren C, Feng DP. Impressive recompensation in transjugular intrahepatic portosystemic shunt-treated individuals with complications of decompensated cirrhosis based on Baveno VII criteria. *World J Gastroenterol* 2023; **29**: 5383-5394 [*RC A*] [PMID: 37900585 DOI: 10.3748/wjg.v29.i38.5383] [*FullText*] [*Full Text(PDF)*]
- 22 **Aravinthan AD**, Barbas AS, Doyle AC, Tazari M, Sapisochin G, Cattral MS, Ghanekar A, McGilvray ID, Selzner M, Greig PD, Bhat M, Selzner N, Grant DR, Lilly LB, Renner EL. Characteristics of liver transplant candidates delisted following recompensation and predictors of such delisting in alcohol-related liver disease: a case-control study. *Transpl Int* 2017; **30**: 1140-1149 [*RC A*] [PMID: 28686307 DOI: 10.1111/tri.13008] [*FullText*]
- 23 **Kim TH**, Um SH, Lee YS, Yim SY, Jung YK, Seo YS, Kim JH, An H, Yim HJ, Yeon JE, Byun KS. Determinants of re-compensation in patients with hepatitis B virus-related decompensated cirrhosis starting antiviral therapy. *Aliment Pharmacol Ther* 2022; **55**: 83-96 [*RC A*] [PMID: 34662436 DOI: 10.1111/apt.16658] [*FullText*]
- 24 **Bedossa P**. Reversibility of hepatitis B virus cirrhosis after therapy: who and why? *Liver Int* 2015; **35** Suppl 1: 78-81 [*RC A*] [PMID: 25529091 DOI: 10.1111/liv.12710] [*FullText*]
- 25 **Dezsó K**, Paku S, Kóbori L, Thorgeirsson SS, Nagy P. What Makes Cirrhosis Irreversible?-Consideration on Structural Changes. *Front Med (Lausanne)* 2022; **9**: 876293 [*RC A*] [PMID: 35572980 DOI: 10.3389/fmed.2022.876293] [*FullText*] [*Full Text(PDF)*]
- 26 **Wanless IR**. The Role of Vascular Injury and Congestion in the Pathogenesis of Cirrhosis: the Congestive Escalator and the Parenchymal Extinction Sequence. *Curr Hepatol Rep* 2020; **19**: 40-53 [*RC A*] [DOI: 10.1007/s11901-020-00508-y] [*FullText*]
- 27 **Zhang W**, Sun YM, Chen SY, You H. [Histopathological evaluation of cirrhosis reversal]. *Zhonghua Gan Zang Bing Za Zhi* 2023; **31**: 677-680 [*RC A*] [PMID: 37580246 DOI: 10.3760/cma.j.cn501113-20230421-00185] [*FullText*]
- 28 **Wen S**, Ruan J, Shen J, Wang X, Yang G, Fu J, Li L, Pan X. Development and validation of a nomogram to predict recompensation in HBV-related cirrhosis with ascites as the single first decompensating event. *Scand J Gastroenterol* 2023; **58**: 915-922 [*RC A*] [PMID: 36825324 DOI: 10.1080/00365521.2023.2181037] [*FullText*]
- 29 **Zhang J**, Lin Y, Zhu Y. Recompensation features and prognosis in hepatitis B virus-related acute-on-chronic liver failure patients. *Eur J Gastroenterol Hepatol* 2025; **37**: 337-342 [*RC A*] [PMID: 39589830 DOI: 10.1097/MEG.0000000000002891] [*FullText*]
- 30 **Wang X**, Shen C, Yang J, Yang X, Qin S, Zeng H, Wu X, Tang S, Zeng W. Alpha-Fetoprotein as a Predictive Marker for Patients with Hepatitis B-Related Acute-on-Chronic Liver Failure. *Can J Gastroenterol Hepatol* 2018; **2018**: 1232785 [*RC A*] [PMID: 29854714 DOI: 10.1155/2018/1232785] [*FullText*] [*Full Text(PDF)*]
- 31 **Wang X**, Sun M, Yang X, Gao L, Weng M, Yang D, Li H, Zhou X, Li J, Qin S, Zhou D, Wu X, Tang S, Zeng W. Value of Liver Regeneration in Predicting Short-Term Prognosis for Patients with Hepatitis B-Related Acute-on-Chronic Liver Failure. *Biomed Res Int* 2020; **2020**: 5062873 [*RC A*] [PMID: 32832550 DOI: 10.1155/2020/5062873] [*FullText*] [*Full Text(PDF)*]
- 32 **Yamazaki S**, Takayama T, Mitsuka Y, Aoki M, Midorikawa Y, Moriguchi M, Higaki T. Platelet recovery correlates parenchymal volume recovery after liver resection. *Hepatology* 2020; **50**: 620-628 [*RC A*] [PMID: 31965697 DOI: 10.1111/hepr.13488] [*FullText*]
- 33 **Deng Y**, Kang H, Xiang H, Nan Y, Hu J, Meng Q, Zhao H, Wang Q, Fang J, Xu J, Wang X, Pan CQ, You H, Xu X, Xie W, Jia J. Durability and on-treatment predictors of recompensation in entecavir-treated patients with hepatitis B and decompensated cirrhosis. *JHEP Rep* 2024; **6**: 101091 [*RC A*] [PMID: 39022388 DOI: 10.1016/j.jhepr.2024.101091] [*FullText*] [*Full Text(PDF)*]
- 34 **He Z**, Wang B, Wu X, Hu Z, Zhang C, Hao Y, Yang Y, Huang Y, Rao W, Wang J, Zhou J, Xia S, Ou X, Jia J, You H. Recompensation in

treatment-naïve HBV-related decompensated cirrhosis: a 5-year multi-center observational study comparing patients with ascites and bleeding. *Hepatol Int* 2023; 17: 1368-1377 [RCA] [PMID: 37775724 DOI: 10.1007/s12072-023-10579-w] [FullText]



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