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Guideline

Chinese Guidelines for Clinical Diagnosis, Treatment, and Management of Cirrhosis (2025)

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Abstract

The Chinese Society of Hepatology of the Chinese Medical Association has invited experts in relevant fields to revise and rename the 2019 "Chinese Guidelines on the Management of Liver Cirrhosis" to "Chinese Guidelines for Clinical Diagnosis, Treatment, and Management of Cirrhosis (2025)". These updated guidelines are aimed at providing recommendations for the clinical diagnosis and management of liver cirrhosis across the compensated, decompensated, and recompensated stages, as well as guidance on cirrhosis reversal and associated complications.

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Introduction

Liver cirrhosis represents the common endpoint of various chronic liver diseases. Regardless of the etiology, chronic (persistent or recurrent) liver inflammation and necrosis can result in diffuse liver fibrosis. Once pseudolobule formation and vascular distortion occur on the basis of liver fibrosis, the condition is classified as liver cirrhosis. Clinically, cirrhosis presents with a wide spectrum of manifestations ranging from mild to severe and complex. In the early stages, it may manifest only through symptoms of the underlying liver disease, often without specific signs, symptoms, or abnormalities in laboratory or imaging findings. As cirrhosis progresses, clinical, laboratory, endoscopic, and imaging manifestations primarily related to impaired hepatic synthetic function and portal hypertension become apparent. In advanced stages, complications such as ascites, esophagogastric variceal bleeding (EVB), hepatic encephalopathy (HE), hepatorenal syndrome (HRS), and progression to hepatocellular carcinoma (HCC) may develop.

The American Association for the Study of Liver Diseases, the European Association for the Study of the Liver, and the Asian Pacific Association for the Study of the Liver have continually developed and updated guidelines and consensus statements on liver cirrhosis and its complications, providing recommendations for their diagnosis and management.[1–3]

In recent years, the Chinese Society of Hepatology has convened a panel of experts from diverse fields, including hepatology, gastroenterology, infectious diseases, surgery, interventional radiology, oncology, traditional Chinese medicine (TCM), and clinical methodology, to update previous guidelines. These updates include the 2022 Guidelines for the Prevention and Treatment of Esophagogastric Variceal Bleeding in Cirrhosis with Portal Hypertension, the 2023 Guidelines for the Diagnosis and Treatment of Ascites in Cirrhosis, and the 2024 Guidelines for the Diagnosis and Treatment of Hepatic Encephalopathy in Cirrhosis.[4–9] In 2025, the Chinese Society of Hepatology revised the 2019 Chinese Guidelines on the Management of Liver Cirrhosis, renaming them the

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Table 1. Quality of evidence and strength of recommendation grading

Evidence quality	
High (A)	Further research is unlikely to change confidence in the estimated effect
Moderate (B)	Further research is likely to impact confidence in the estimated effect and may change the estimate
Low (C)	Further research is highly likely to impact confidence in the estimated effect and is likely to change the estimate.
Very low (D)	Confidence in the observed value is low: the true value is likely to differ from the observed value.
Strength of recommendation	
Strong (1)	Clear evidence that the benefits outweigh the harms or vice versa
Weak (2)	Benefits and harms are uncertain, or evidence of any quality indicates that benefits and harms are balanced

Chinese Guidelines for Clinical Diagnosis, Treatment, and Management of Cirrhosis (2025). This revision incorporated the latest domestic and international research while integrating China's clinical context. The process involved establishing a steering committee, expert panel, and secretariat, and adhered to AGREE II (Appraisal of Guidelines for Research and Evaluation) standards. Clinical research evidence and recommendations in the guidelines were graded according to the GRADE (Grading of Recommendations Assessment, Development, and Evaluation) system (Table 1).

These guidelines are not mandatory standards and cannot address all issues related to the diagnosis and treatment of liver cirrhosis. Therefore, when managing individual patients, clinicians may adhere to the principles of these guidelines, thoroughly assess each patient's condition, carefully consider the patient's perspectives and preferences, and incorporate local medical resources and practical experience to tailor comprehensive and individualized management plans.

Etiology and epidemiology

The most common etiologies of liver cirrhosis include viral hepatitis (hepatitis B and C), alcoholic liver disease, and metabolic dysfunction-associated fatty liver disease (MAFLD). Less common etiologies primarily include autoimmune liver diseases, such as autoimmune hepatitis, primary biliary cholangitis, and primary sclerosing cholangitis. Rare etiologies involve genetic and metabolic disorders such as Wilson's disease, hemochromatosis, hepatic amyloidosis, glycogen storage disease, Alagille syndrome, progressive familial intrahepatic cholestasis, hepatic porphyria, cystic fibrosis, and alpha-1 antitrypsin deficiency. In addition, exposure to drugs or chemical toxins, parasitic infections (e.g., schistosomiasis or clonorchiasis), and chronic circulatory disorders (e.g., Budd–Chiari syndrome or right heart failure) can also lead to liver fibrosis and cirrhosis. Cases with evidence of cirrhosis, albeit with no identifiable cause after systematic evaluation, are classified as cryptogenic cirrhosis (Table 2).

Globally, the incidence of liver cirrhosis is significantly higher in males than in females.[10] From 1990 to 2017, the global age-standardized mortality rate decreased from 21.0 (19.2–22.3) per 100,000 to 16.5 (15.8–18.1) per 100,000. However, the proportion of deaths due to cirrhosis relative to total deaths increased from 1.9% (1.8%–2.0%) to 2.4% (2.3%–2.6%). Notably, the age-standardized prevalence rate of MAFLD-related cirrhosis has significantly increased. Globally, the incidence, prevalence, and mortality rates of NAFLD-related cirrhosis increased by 2.95%, 120.12%, and 76.72%, respectively, with prevalence rising from 12,065.15 per 100,000 to 15,022.90 per 100,000.[11] Given the lack of effective therapies, the burden of MAFLD-related cirrhosis is expected to continue increasing. Importantly, MAFLD has now become one of the leading causes of HCC worldwide.[12]

In a large Chinese cohort study of 5.7 million individuals

Table 2. Common, less common, and rare causes of liver cirrhosis

Viral hepatitis	Genetic and metabolic diseases
Chronic hepatitis B, hepatitis C, hepatitis B with hepatitis D, etc.	Wilson's disease, hemochromatosis, hepatic amyloidosis, glycogen storage disease, Alagille syndrome, progressive familial intrahepatic cholestasis, hepatic porphyria, cystic fibrosis, alpha-1 antitrypsin deficiency, etc.
Alcoholic liver disease	Circulatory disorders
Metabolic dysfunction-associated fatty liver disease	Budd–Chiari syndrome, right heart failure
Drugs or chemical toxins	Autoimmune liver diseases
Acetaminophen, anti-tuberculosis drugs (e.g., isoniazid, rifampin, or pyrazinamide), chemotherapeutic agents, certain Chinese herbal medicines (e.g., *Tripterygium wilfordii*, *Polygonum multiflorum*, or *Gynura segetum*), antirheumatic drugs, toxic mushrooms, carbon tetrachloride, etc.	Primary biliary cholangitis, primary sclerosing cholangitis, autoimmune hepatitis
Parasitic infections	Cryptogenic cirrhosis
Schistosomiasis, clonorchiasis, etc.	

undergoing health examinations, the prevalence of liver cirrhosis was 0.87%.[12] In recent years, the etiological profile of liver cirrhosis in China has shifted. Due to widespread neonatal hepatitis B vaccination, blood donor screening, improved blood transfusion safety, promotion of safe injection practices, and the extensive use of antiviral therapies for hepatitis B and direct-acting antivirals for hepatitis C, the incidence of viral hepatitis-related cirrhosis has significantly declined. In contrast, the proportion of cirrhosis attributed to MAFLD, alcoholic liver disease, and autoimmune liver diseases has been increasing.[13] National data indicate that the incidence, prevalence, and mortality of MAFLD in China have risen by 68%, 119%, and 22%, respectively, with the age-standardized prevalence rate increasing from 18,291.53 to 30,644.26 per 100,000.[14]

Notably, while most patients with liver cirrhosis have a single predominant etiology, many present with multiple contributing factors. For example, hepatitis B may coexist with hepatitis D or hepatitis C, or viral hepatitis (B or C) may be accompanied by excessive alcohol consumption, overweight/obesity, or diabetes.

Pathophysiology

Chronic hepatocyte injury activates hepatic stellate cells, leading to increased synthesis and reduced degradation of extracellular matrix components, such as collagen. This results in diffuse liver fibrosis. Consequently, structural remodeling of hepatic lobules and vascular distortion occur, ultimately culminating in cirrhosis.[15,16] The primary pathophysiological changes in cirrhosis include liver dysfunction and the development of portal hypertension.

1. Liver dysfunction: Chronic liver inflammation induces hepatocyte necrosis. When regeneration of hepatocytes is insufficient to compensate for cell loss, liver dysfunction ensues. This is primarily manifested by reduced synthesis of albumin (ALB) and coagulation factors, impaired metabolism of bilirubin and other toxic substances, and inadequate inactivation of estrogen.

2. Formation of portal hypertension: In cirrhosis, liver fibrosis and pseudolobule formation compress intrahepatic venules and sinusoids, leading to vascular distortion, occlusion, and increased portal vein resistance, the primary cause of elevated portal pressure. Concurrently, widespread visceral vasodilation increases portal venous inflow. In addition, reduced effective arterial blood volume stimulates secondary elevations in circulating vasoactive substances (e.g., norepinephrine, serotonin, and angiotensin) and decreased nitric oxide production, further promoting contraction of intrahepatic portal branches and increasing resistance. The detailed pathophysiology of ascites, EVB, HE, and HRS is described in the relevant guidelines.[4–9]

3. Role of the gut-liver axis in promoting cirrhosis: Patients with chronic liver disease often develop gut microbiota dysbiosis, characterized by reduced beneficial bacteria (e.g., lactobacilli, bifidobacteria) and increased potentially pathogenic bacteria (e.g., Enterobacteriaceae, Streptococcaceae). This imbalance enhances the production of endotoxins (e.g., lipopolysaccharides) and harmful bacterial metabolites (e.g., ammonia, ethanol, short-chain fatty acids). In cirrhosis, downregulation of tight junction proteins and impaired intestinal barrier function further increase mucosal permeability. Moreover, bile acid metabolism disorders associated with cirrhosis exacerbate microbiota dysbiosis and barrier dysfunction. These harmful products enter the liver via the portal vein, triggering inflammatory cytokine release, which in turn promotes hepatic stellate cell activation and extracellular matrix deposition, thereby accelerating liver fibrosis and cirrhosis.[17]

4. Factors contributing to the progression of cirrhosis: Across all etiologies, common drivers of cirrhosis progression include persistent inflammation, alcohol consumption, overweight/obesity, and metabolic syndrome. Patients with cirrhosis also have a markedly increased risk of developing HCC.[18,19]

Assessment of liver function and portal hypertension

(1) Biochemical assessment of liver function

Indicators of hepatic synthetic function primarily include serum ALB, prealbumin, coagulation factors, cholesterol, and cholinesterase.[20] ALB is synthesized by hepatocytes and has a circulating half-life of approximately three weeks; thus, a decrease in ALB indicates that liver disease has persisted for longer than three weeks.[21] Prothrombin time (PT), prothrombin activity, and the international normalized ratio (INR) of PT are sensitive early markers of impaired hepatic synthetic function. In severe liver disease, PT may become prolonged within 24 h, while ALB levels may still remain within normal limits.

(2) Assessment of hepatic reserve function

1. Child–Pugh score: The Child–Pugh score is a method for assessing the severity of liver cirrhosis based on five indicators: HE, ascites, serum ALB, bilirubin, and PT.[22] Hepatic reserve function is classified into three grades: A (5–6 points), B (7–9 points), and C (10–15 points),[23] with one-year liver disease-related mortality rates of <5%, 20%, and 55%, respectively. The Child–Pugh score is a relatively reliable tool for evaluating the prognosis of patients with cirrhosis. However, the inclusion of subjective indicators such as ascites volume and HE grading may introduce variability due to differences in evaluators' interpretations of the criteria.

2. Model for End-Stage Liver Disease (MELD) and MELD-Na Scores: The currently applied MELD scoring system incorporates three indicators: serum bilirubin, creatinine, and INR. The MELD-Na system additionally includes the indicator of serum sodium.[24] By accounting for renal function, the MELD score allows for a more accurate assessment and stratification of cirrhosis severity and prognosis. However, serum creatinine (Scr) levels can be influenced by non–liver-related factors, potentially leading to misjudgments of disease severity. Hyponatremia is an independent risk factor for poor outcomes in cirrhosis, and studies have shown that the MELD-Na score outperforms the MELD score in predicting prognosis for end-stage cirrhosis.[25] In recent years, ongoing refinements of the MELD score have aimed to further improve its predictive value for cirrhosis prognosis.[26]

3. Indocyanine green clearance test: This quantitative test primarily reflects liver blood flow and hepatic reserve function. In cirrhosis or severe liver injury, the 15-min indocyanine green retention rate is significantly increased and correlates with the Child–Pugh score.[27] This test is commonly used preoperatively to assess surgical risk in patients with cirrhosis.

(3) Imaging assessment

1. Abdominal ultrasound: Ultrasound is a convenient method for diagnosing liver cirrhosis and portal hypertension. Cirrhosis is typically characterized by liver atrophy, disproportionate left and right lobe sizes (commonly with left lobe enlargement and right lobe shrinkage), a wavy liver surface, and granular or nodular parenchymal echogenicity. Portal

hypertension is indicated by splenomegaly, portal vein dilation, the presence of portosystemic collaterals, and ascites. Doppler ultrasound can also reveal reduced portal vein blood flow velocity or reversed portal vein flow. A limitation of ultrasound is that its diagnostic accuracy partly depends on the operator's experience.

2. Liver stiffness measurement (LSM): LSM, usually performed with vibration-controlled transient elastography-based devices such as Fibroscan® and Fibrotouch®, is the most widely used non-invasive method for assessing liver fibrosis and early cirrhosis. However, LSM values can be affected by liver inflammation, jaundice, recent food intake, and excessive alcohol consumption. The diagnostic cutoff values vary according to the etiology of liver disease, as outlined in the 2019 Consensus on the Diagnosis and Treatment of Liver Fibrosis.[28]

3. Spleen stiffness measurement (SSM): SSM has similar clinical applications to LSM and can be used to evaluate liver fibrosis, portal hypertension, and the severity of esophageal varices.[29–31] An SSM value < 21 kPa effectively rules out portal hypertension, while an SSM value > 50 kPa strongly suggests significant portal hypertension. When LSM < 20 kPa, platelet count (PLT) > 150 × 10^9/L, and SSM ≤ 46 kPa, high-risk esophageal varices can be ruled out without the need for gastroscopy. The LSM/SSM ratio also provides diagnostic value in distinguishing cirrhosis-related portal hypertension from non-cirrhotic portal hypertension (NCPH).

4. Abdominal computed tomography (CT): Contrast-enhanced CT can evaluate liver cirrhosis and portal hypertension by assessing liver size, morphology, margins, texture, portosystemic collateral circulation, and splenic enlargement.[32] It has high sensitivity and specificity for cirrhosis but lower sensitivity for liver fibrosis. Three-dimensional vascular reconstruction with contrast-enhanced CT can visualize the portal venous system and thrombosis and allows quantification of liver and spleen volumes.[33,34]

5. Abdominal magnetic resonance imaging (MRI) and magnetic resonance elastography (MRE): Abdominal MRI and MRE can be used to diagnose liver fibrosis and portal hypertension. The MRI features of cirrhosis are similar to those observed on CT. MRE, a recently developed non-invasive technique for staging liver fibrosis, assesses the entire liver and provides high accuracy. It is particularly suitable for patients with ascites, obesity, or metabolic syndrome who are not candidates for vibration-controlled transient elastography. However, MRE is expensive, and its utility in diagnosing early cirrhosis and staging liver fibrosis still requires further clinical research.

(4) Non-invasive diagnostic models

Serological markers such as the aspartate aminotransferase-to-platelet ratio index (APRI), FIB-4, and chitinase-3-like protein 1, along with composite models that integrate serological and imaging data, can be used for the diagnosis and staging of liver fibrosis.[35–38]

(5) Histopathological assessment of the liver

Liver biopsy remains the gold standard for diagnosing and assessing early cirrhosis of various etiologies and determining the degree of inflammatory activity. Histologically, cirrhosis is characterized by fibrous septa encircling hepatic lobules, leading to lobular distortion, nodular regeneration of hepatocytes, and pseudolobule formation. Cirrhosis can be classified histologically into active and quiescent phases.[39,40] Elimination or suppression of the underlying etiology and resolution of inflammatory lesions may result in partial histological reversal of cirrhosis in some cases.[41,42] The width of fibrous septa and the size of nodules are independent predictors of portal hypertension.[43,43]

The "Beijing Criteria" represent a novel histopathological standard for evaluating the reversal of liver fibrosis in patients with chronic hepatitis B following antiviral therapy.[45] By assessing the proportion of different types of fibrous septa in liver tissue, liver fibrosis is categorized into three types (PIR classification): progressive type (P-type), characterized by predominant proliferation and expansion of fibrous septa, indicating ongoing fibrosis progression; indeterminate type (I-type), where changes in fibrous septa are not pronounced, making it difficult to determine progression or reversal; and regressive type (R-type), characterized by predominant fragmentation and resorption of fibrous septa, indicating fibrosis reversal.

Histological diagnosis of liver cirrhosis should include both etiological evaluation and assessment of the extent of cirrhotic lesions. However, liver tissue obtained from biopsies in patients with cirrhosis is often fragile and fragmented, which may limit its ability to accurately reflect the full extent of cirrhotic changes. To improve diagnostic accuracy, biopsy samples should ideally measure ≥1.6 cm in length, 1.2–1.8 mm in width, and contain at least 8–10 complete portal tracts.

(6) Assessment of portal hypertension

In addition to abdominal ultrasound, LSM, SSM, CT, MRI, and MRE, which can be used to evaluate the presence of portal hypertension, the following methods are also reliable for assessing its severity:

1. Endoscopy: Endoscopy is the most reliable minimally invasive method for assessing clinically significant portal hypertension in cirrhosis and is considered the gold standard for screening and diagnosing esophagogastric varices as well as evaluating bleeding risk.[46] Refer to the Guidelines on the management of esophagogastric variceal bleeding in cirrhotic portal hypertension.[6,9] Varices occur in the esophagus and/or gastric fundus in approximately 90% of patients with cirrhosis. Gastroscopy allows direct visualization of the presence, extent, and severity of esophageal and gastric varices and can confirm portal hypertensive gastropathy (PHG). Approximately 10% of patients with cirrhosis develop varices in less common sites, such as the duodenum, small intestine, or colon, termed "ectopic varices."

2. Hepatic venous pressure gradient (HVPG) measurement: HVPG measurement indirectly reflects portal vein pressure and is highly valuable for staging cirrhosis, assessing complications, and evaluating treatment response.[6,9] The normal reference range for HVPG is 3–5 mmHg (1 mmHg = 0.133 kPa). An HVPG of 6–10 mmHg indicates mild portal hypertension, typically associated with absent or mild esophagogastric varices and a low risk of decompensation. An HVPG > 10 mmHg denotes clinically significant portal hypertension, usually with evident esophagogastric varices and a high risk of decompensation. An HVPG > 20 mmHg is associated with difficult-to-control or recurrent decompensated cirrhosis complications, such as refractory ascites, uncontrolled EVB, and severe liver dysfunction, with a one-year mortality rate exceeding 60%.[47,48] HVPG measurement is also useful for differentiating NCPH (pre-sinusoidal and post-sinusoidal).

HVPG measurement is, however, invasive, requires specialized equipment and technical expertise, and its high cost limits routine use across all levels of hospitals.

Portal pressure gradient (PPG) measurement is a recently developed clinical method that directly measures portal vein pressure via endoscopic ultrasound-guided transgastric puncture of the portal vein. In sinusoidal cirrhosis, PPG correlates well with HVPG. In NCPH, such as extrahepatic portal

vein thrombosis (PVT) or portal cavernous transformation, PPG is significantly elevated, while HVPG may remain normal or only mildly elevated.[49]

(7) Nutritional risk screening and malnutrition assessment

Malnutrition is a common complication of end-stage liver disease and an independent predictor of poor prognosis in patients with cirrhosis. It is associated with liver failure, ascites, infections, HRS, and HE. Therefore, clinicians should prioritize nutritional risk screening and malnutrition assessment in these patients.

Malnutrition assessment typically involves evaluating body composition, energy metabolism, dietary intake, and the use of comprehensive scoring tools. Commonly assessed indicators include body mass index, hemoglobin, serum ALB, prealbumin, and bone density. Sarcopenia, an important manifestation of malnutrition, encompasses both reduced muscle mass and impaired muscle function. Muscle mass is often assessed using the cross-sectional area of skeletal muscle at the lumbar level on CT or MRI, as well as through measurements of calf circumference, upper arm muscle circumference, and triceps skinfold thickness. Grip strength testing is a widely used method for evaluating muscle function. Dietary intake is commonly assessed using the 24-h dietary recall or food weighing methods. For further details, refer to the Clinical guidelines on nutrition in end-stage liver disease.[50]

Clinical staging, diagnosis, and differential diagnosis of liver cirrhosis

(1) Clinical staging and diagnosis of liver cirrhosis

Currently, the international hepatology community advocates classifying liver cirrhosis into compensated cirrhosis, decompensated cirrhosis, and recompensated cirrhosis. The European Association for the Study of the Liver cirrhosis guidelines further subdivide cirrhosis into six stages: compensated (stages 1 and 2) and decompensated (stages 3, 4, 5, and 6), with annual mortality rates of 1.5%, 2%, 10%, 21%, and 87%, respectively.[1] This staging system provides valuable guidance for clinical research. Diagnosis at each stage requires a comprehensive assessment of etiology, medical history, clinical manifestations, complications, treatment course, laboratory findings, imaging results, and histological features.

1. Definition and diagnostic criteria for compensated cirrhosis: Compensated cirrhosis is defined by evidence of cirrhosis on clinical, laboratory, endoscopic, imaging, or histological assessments, without the occurrence of clinically evident severe complications such as ascites, EVB, or HE. The diagnosis requires meeting one of the following four criteria:

1. Histological confirmation of cirrhosis;
2. Endoscopic evidence of esophagogastric varices or ectopic gastrointestinal varices, with NCPH ruled out;
3. Imaging findings from ultrasound, LSM, SSM, CT, or MRI suggestive of cirrhosis or portal hypertension, such as splenomegaly, portal vein diameter ≥ 1.3 cm, with LSM or SSM values interpreted according to etiology-specific diagnostic cutoffs;
4. In the absence of histological, endoscopic, or imaging evidence, the presence of cirrhosis is suggested by abnormalities in at least two of the following indicators:
   i. PLT <100 × 10^9/L, with no other explanatory causes;
   ii. Serum ALB < 35 g/L, excluding malnutrition, protein-losing enteropathy, or renal disease;
   iii. INR > 1.3 or prolonged PT (after discontinuing thrombolytic or anticoagulant drugs for at least seven days);
   iv. APRI: Adult APRI score > 2[51] (noting that factors such as enzyme-lowering drugs may affect APRI).

2. Definition and diagnostic criteria for decompensated cirrhosis: Decompensated cirrhosis is defined by evidence of cirrhosis on clinical, laboratory, endoscopic, imaging, or histological assessments, accompanied by the occurrence of severe complications such as clinical ascites, EVB, or HE. The diagnosis requires meeting both of the following criteria:

1. Fulfillment of the diagnostic criteria for compensated cirrhosis as described above;
2. Presence of at least one severe complication related to portal hypertension, such as clinical ascites, EVB, HRS, or HE.

Recent studies have described a subclinical form of decompensated cirrhosis, defined by minimal hepatic encephalopathy (MHE), ultrasound-detected minimal ascites (<2 cm), or a positive fecal occult blood test in cirrhosis (with other causes excluded). This subclinical form does not yet meet the criteria for clinical decompensation. Clinicians should recognize subclinical decompensation, first decompensation, stable decompensation, and unstable decompensation as distinct clinical states.[52–54]

3. Definition and diagnostic criteria for recompensated cirrhosis: Recompensated cirrhosis refers to patients with previously decompensated cirrhosis whose underlying etiology has been effectively controlled, resulting in stable improvement in liver function and no occurrence of ascites, EVB, or HE for at least one year. Based on the Baveno VII expert consensus and studies by Chinese scholars, the diagnostic criteria require meeting all three of the following[45,52]:

1. Elimination, suppression, or cure of the etiology of cirrhosis (e.g., clearance of hepatitis C virus (HCV), sustained suppression of hepatitis B virus (HBV), or sustained abstinence in alcoholic cirrhosis);
2. Absence of ascites (after discontinuing diuretics), HE (after discontinuing lactulose/rifaximin), and recurrent variceal bleeding for at least 12 months after stopping primary treatments;
3. Stable improvement in liver function indicators (ALB, INR, bilirubin), with a MELD score < 10 and/or Child–Pugh grade A (ALB > 35 g/L, INR < 1.5, and total bilirubin < 34 μmol/L).

Multiple clinical studies have demonstrated partial functional and histological reversibility of cirrhosis.[45,52,54] In hepatitis B-related cirrhosis, whether compensated or decompensated, effective antiviral therapy can lead to reversal of cirrhosis in some patients, significantly improving the severity of esophageal varices and even reducing portal hypertension. Histological criteria for cirrhosis reversal include: (1) a reduction in the Ishak fibrosis stage by ≥1 level, or (2) a decrease in the PIR classification following treatment.

Based on clinical characteristics and the duration of recompensation, it is recommended to classify recompensated cirrhosis into three stages: temporary recompensation (6–12 months), stable recompensation (12–24 months), and long-term recompensation (>24 months). Temporary recompensation, due to its short duration, carries a higher risk of recurrent complications. Stable recompensation, maintained for >12 months, is associated with a further reduced risk of complications. Long-term recompensation, defined as sustained resolution of complications and stable improvement in liver function indicators for >24 months, has a long-term prognosis approaching that of compensated cirrhosis.

(2) Differential diagnosis

1. Pseudocirrhosis: This term primarily refers to conditions

Table 3. Relationship between HVPG and different causes of portal hypertension

Cause of portal hypertension	Wedged hepatic vein pressure (mmHg)	Free hepatic vein pressure (mmHg)	HVPG (mmHg)
Prehepatic			
Splenic vein thrombosis	Normal	Normal	Normal
Myeloproliferative disorders	Normal	Normal	Normal
Extrahepatic portal vein thrombosis or arteriovenous malformation	Normal	Normal	Normal
Intrahepatic			
Pre-sinusoidal			
Idiopathic non-cirrhotic portal hypertension	Elevated	Normal	Normal or mildly elevated
Congenital hepatic fibrosis	Elevated	Normal	Normal or mildly elevated
Nodular regenerative hyperplasia	Elevated	Normal	Normal or mildly elevated
Sinusoidal			
Chronic liver disease of various etiologies	Elevated	Elevated	Significantly elevated
Post-sinusoidal			
Hepatic veno-occlusive disease	Elevated	Normal	Significantly elevated
Posthepatic			
Budd–Chiari syndrome	Elevated	Elevated	Normal
Constrictive pericarditis	Elevated	Elevated	Normal
Left heart failure	Elevated	Elevated	Normal

such as metastatic liver cancer or hereditary hemorrhagic telangiectasia that mimic cirrhosis, presenting with portal hypertension-related manifestations such as ascites, EVB, and edema. Imaging may or may not reveal intrahepatic nodular changes but often shows segmental liver volume reduction and caudate lobe enlargement.[55,56] The pathogenesis is not fully understood and varies depending on the underlying disease.

2. NCPH: NCPH is not a single disease entity but refers to portal hypertension occurring in the absence of cirrhosis, encompassing pre-sinusoidal, sinusoidal, and post-sinusoidal portal hypertension with diverse etiologies. Its main manifestations include splenomegaly (with or without hypersplenism), esophagogastric varices and bleeding, and ascites, while hepatic synthetic function remains relatively preserved. Approximately 75% of NCPH patients are initially misdiagnosed as having "cirrhosis." Diagnosis primarily relies on imaging and liver histopathology, as HVPG measurement is not suitable for evaluating NCPH (Table 3).[57,58]

Recommendation 1: Liver cirrhosis is classified into compensated, decompensated, and recompensated stages (B1).

Recommendation 2: Diagnosis of compensated cirrhosis: No history of complications such as ascites, EVB, or HE, meeting one of the following four criteria: (1) Histological confirmation of cirrhosis (A1); (2) Endoscopic evidence of esophagogastric or ectopic gastrointestinal varices, with NCPH ruled out (B1); (3) Imaging findings from ultrasound, LSM, SSM, CT, or MRI suggestive of cirrhosis or portal hypertension (B1); (4) In the absence of histological, endoscopic, or imaging evidence, at least two of the following: ① PLT < 100 × 10^9/L with no other explanatory causes; ② ALB < 35 g/L, excluding malnutrition or renal disease; ③ INR > 1.3 or prolonged PT (after discontinuing thrombolytic or anticoagulant drugs for >7 days); ④ APRI > 2 (B1).

Recommendation 3: Diagnosis of decompensated cirrhosis requires meeting both of the following: (1) Fulfillment of the diagnostic criteria for cirrhosis; (2) Presence of portal hypertension-related complications, such as ascites, EVB, HRS, and HE (B1).

Recommendation 4: The subclinical form of decompensated cirrhosis includes MHE, minimal ascites (< 2 cm on ultrasound), and a positive fecal occult blood test in cirrhosis (with other causes excluded). This subclinical form does not yet meet the criteria for clinical decompensation (C2). Clinicians should prioritize management across the spectrum of subclinical decompensated cirrhosis, first decompensation, stable decompensation, and unstable decompensation (B1).

Recommendation 5: The diagnosis of recompensated cirrhosis requires meeting all of the following criteria: (1) Elimination, suppression, or cure of the etiology of cirrhosis (e.g., HCV clearance, sustained HBV suppression, or sustained abstinence in alcohol-related cirrhosis); (2) Absence of ascites (after discontinuation of diuretics), HE (after discontinuation of lactulose/rifaximin), and recurrent variceal bleeding for >12 months after cessation of primary treatments; (3) Sustained improvement in liver function indicators (ALB > 35 g/L, INR < 1.5, total bilirubin < 34 μmol/L), or a MELD score < 10 and/or Child–Pugh grade A (B1).

Recommendation 6: It is suggested to classify

recompensation as temporary recompensation (6–12 months), stable recompensation (12–24 months), and long-term recompensation (>24 months) (C1).

Recommendation 7: Cirrhosis reversal is defined as significant improvement in liver function and esophagogastric varices, reversal of portal hypertension, a reduction in the Ishak fibrosis stage by ≥1, or a decrease in PIR classification following treatment in hepatitis B-related cirrhosis (C1).

Recommendation 8: SSM can be used to assess the degree of liver fibrosis, the severity of portal hypertension, and the severity of esophageal varices. An SSM < 21 kPa effectively rules out portal hypertension, while an SSM > 50 kPa indicates clinically significant portal hypertension. LSM < 20 kPa, PLT >150 × 10^9/L, and SSM ≤ 46 kPa suggest a low probability of high-risk esophageal varices for bleeding, potentially avoiding the need for endoscopic screening.

Complications associated with liver cirrhosis

(1) Serous cavity effusions

Serous cavity effusions in cirrhosis include ascites, hydrothorax, and pericardial effusion.

Ascites: This is the most common complication of cirrhosis. For detailed guidance, refer to the Guidelines on the management of ascites in cirrhosis (2023 version).[4]

Chylous ascites: The incidence of chylous ascites in cirrhosis ranges from 0.5% to 11%. It is characterized by a milky-white appearance. An ascitic fluid triglyceride level > 200 mg/dL (2.26 mmol/L) supports the diagnosis, while a level < 50 mg/dL rules it out. Chylous ascites may occur at any stage of cirrhosis. During diagnosis, other causes must be excluded, including malignancy; abdominal surgery or trauma; thoracic duct injury following endoscopic sclerotherapy for esophageal varices; infections (particularly pulmonary tuberculosis and filariasis); and congenital abnormalities leading to obstruction or destruction of abdominal or thoracic lymphatic vessels.

Hemorrhagic ascites: This is characterized by a venous blood–like or "meat-wash" appearance, or an ascitic red blood cell count > 50,000/mm^3. In patients with cirrhosis and hemorrhagic ascites, malignancy should first be ruled out. Other possible causes include infections (such as tuberculous peritonitis), coagulation disorders, or rupture of peritoneal varices. The appearance may range from pinkish "meat-washing fluid" to frank blood-like fluid.

Hepatic hydrothorax: Other causes, such as tuberculosis, must be ruled out. Hepatic hydrothorax is usually right-sided due to negative intrathoracic pressure during inspiration, which allows ascitic fluid to pass into the pleural cavity through diaphragmatic defects. Severe cases may involve bilateral hydrothorax, while a minority of patients present with isolated left-sided hydrothorax. Diagnosis can be made by chest ultrasound or X-ray. Hydrothorax may also progress to spontaneous bacterial empyema, which carries a poor prognosis, with a median survival of 8–12 months.[59,60]

(2) Gastrointestinal bleeding and other complications

EVB is the most common cause of gastrointestinal bleeding in liver cirrhosis, as detailed in the 2022 Guidelines for the Prevention and Treatment of Esophagogastric Variceal Bleeding in Cirrhosis with Portal Hypertension.[6,9] This section primarily addresses other gastrointestinal complications related to portal hypertension in cirrhosis, including PHG, portal hypertensive enteropathy (PHE), portal hypertensive cholangiopathy (PHC), and internal hemorrhoids.

PHG: PHG results from elevated pressure in the portal vein and its tributaries.[61,62] According to the 1992 Milan Conference definition, it is characterized endoscopically by dilation of gastric mucosal and submucosal vessels, typically presenting as a "snake-skin pattern" or "mosaic sign." PHG is the second most common cause of gastrointestinal bleeding in cirrhosis, following EVB. Gastric antral vascular ectasia may also occur in cirrhosis and is more frequently observed in patients with diabetes and non-alcoholic fatty liver disease.

PHE: A condition characterized by intestinal vascular dilation secondary to portal hypertension, PHE is classified into portal hypertensive colopathy and portal hypertensive small bowel disease (including duodenal, jejunal, and ileal involvement). Most patients are asymptomatic, but some may present with gastrointestinal bleeding, abdominal distension, or pain. Bleeding typically manifests as lower gastrointestinal hemorrhage, often with melena or positive fecal occult blood tests, and occasionally as massive lower gastrointestinal bleeding. International grading standards for PHE are not yet standardized.[63,64] Internal hemorrhoids, a common but frequently overlooked manifestation of cirrhosis, along with PHE, represent important causes of lower gastrointestinal bleeding in these patients.

PHC: Refers to abnormalities in the biliary tree (including the cystic duct and gallbladder) associated with portal hypertension in cirrhosis. Clinical manifestations include gallstones; irregular or thickened bile duct walls; bile duct stenosis; varices of the bile duct or gallbladder wall; chronic cholecystitis; bile duct strictures; and intra- or peribiliary varices.[65] While most patients remain asymptomatic, some may develop fever, upper abdominal pain, jaundice, pruritus, or biliary bleeding. Magnetic resonance cholangiopancreatography is the preferred diagnostic tool, whereas endoscopic retrograde cholangiopancreatography can aid in both diagnosis and treatment.

(3) Hepatic encephalopathy or related neurological damage

HE is a neuropsychiatric syndrome of varying severity, caused by acute or chronic severe liver dysfunction or by portosystemic shunts, with metabolic disorders as the underlying basis. The diagnosis and grading of HE are detailed in the 2024 Guidelines for the Diagnosis and Treatment of Hepatic Encephalopathy in Liver Cirrhosis.[7] Screening for MHE should be emphasized in patients with cirrhosis.

Patients with cirrhosis may develop spontaneous portosystemic shunts (SPSS), leading to elevated peripheral blood ammonia (NH_3) levels. Both SPSS and transjugular intrahepatic portosystemic shunt (TIPS) are important causes of recurrent HE in cirrhosis. The reported incidence of HE after TIPS varies widely, reaching up to approximately 61%. Enhanced management before, during, and after TIPS can help reduce the risk of post-TIPS HE. The occurrence of post-TIPS HE is associated with factors such as advanced age (≥70 years), poor preoperative nutritional status, impaired hepatic reserve function, SPSS, hyponatremia, obesity, diabetes, renal insufficiency, and sedative use.[66–68] Malnutrition and sarcopenia are independent predictors of poor prognosis after TIPS. Patients with these risk factors should be prioritized for HE prevention and treatment.[69]

Hepatic myelopathy: Commonly seen in patients with end-stage liver disease due to various causes, including cirrhosis, HCC, and liver failure. Hepatic myelopathy may be consid-

ered when other neurological disorders have been excluded, and the following features are present in the context of chronic liver disease: progressive bilateral lower-limb weakness, scissor gait, or inability to walk; neurological examination showing spastic paraplegia without significant muscle atrophy or superficial sensory deficits, with increased muscle tone and enhanced plantar extensor reflexes. Lumbar puncture and cerebrospinal fluid analysis are helpful in ruling out inflammatory spinal cord lesions.

Acquired hepatocerebral degeneration: Acquired hepatocerebral degeneration is a rare and mostly irreversible clinical syndrome of neurological impairment caused by chronic liver disease, with an incidence of 0.8%–2% in patients with cirrhosis. Its onset is generally insidious, primarily presenting as psychiatric abnormalities, cognitive decline, and Parkinsonian syndromes. Motor disorders such as ataxia, intention tremor, and chorea may also occur, along with neuropsychological changes such as behavioral abnormalities and intellectual impairment. Functional MRI can assist in differentiation.

In elderly patients, pre-existing cognitive impairment, including declines in memory, attention, learning ability, and sensorimotor function, may overlap with manifestations of HE. Therefore, careful differentiation from other neurological conditions, such as cerebral infarction, Parkinson's disease, and Alzheimer's disease, is essential.

(4) Infections

Spontaneous bacterial peritonitis (SBP): An abdominal infection occurring in the setting of liver cirrhosis, characterized by peritonitis without an identifiable intra-abdominal source (e.g., intestinal perforation or abscess). Pathogenic microorganisms invade the peritoneal cavity, making SBP one of the most common complications in end-stage liver disease, including cirrhosis, with an incidence of 40%–70%. Its diagnosis and differential diagnosis are outlined in the Guidelines for the Diagnosis and Treatment of Ascites in Liver Cirrhosis (2023 Edition).[4]

In addition to SBP, other common infections in patients with cirrhosis include urinary tract infections, biliary tract infections, gastrointestinal infections, respiratory infections, skin and soft tissue infections, and sepsis.[2] The clinical manifestations are diverse, often atypical, and may have an insidious onset, increasing the risk of misdiagnosis. Patients with secondary peritonitis, endocarditis, pneumonia, or sepsis generally have a poorer prognosis. Infections are also frequent triggers of liver failure and HRS in cirrhosis. Risk factors for infection in cirrhosis include hepatic microcirculatory dysfunction, local and systemic inflammatory responses, immune dysregulation, and gut microbiota dysbiosis.

(5) Renal impairment

Renal impairment in patients with cirrhosis includes acute kidney injury (AKI), HRS-AKI, hepatorenal syndrome–non-acute kidney injury (HRS-NAKI), and chronic kidney disease (CKD).

AKI is a serious complication in decompensated cirrhosis. Its incidence in hospitalized patients with cirrhosis ranges from 20% to 80%, with a high risk of progression to renal failure and significant mortality. According to the revised diagnostic criteria by the International Club of Ascites (ICA),[70] AKI is defined as a Scr increase of ≥26.5 μmol/L (0.3 mg/dL) within 48 h of admission compared with baseline, or an increase in Scr of ≥50% within seven days compared with an existing or inferred baseline value (any creatinine measurement within the past three months may serve as the baseline).

Stage 1: Increase in Scr by an absolute value of ≥26.5 μmol/L (0.3 mg/dL), or increase to 1.5–2.0 times the baseline value.

Stage 2: Increase in Scr to 2.0–3.0 times the baseline value.

Stage 3: Increase in Scr to ≥3 times the baseline value, or Scr ≥ 353.6 μmol/L with an acute increase of ≥26.5 μmol/L (0.3 mg/dL), or initiation of renal replacement therapy.

Previously, type 1 HRS corresponded to HRS-AKI, while type 2 HRS encompassed HRS-NAKI and acute kidney disease (AKD).

HRS-AKI diagnostic criteria: (1) Presence of cirrhosis and ascites; (2) Meeting ICA criteria for AKI; (3) No response after 48 h of diuretic withdrawal and volume expansion with ALB at 1 g/kg body weight; (4) Absence of shock; (5) No current or recent use of nephrotoxic drugs; (6) No signs of structural kidney injury, including: ① No proteinuria (<500 mg/day); ② No microhematuria (<50 red blood cells per high-power field); ③ Normal renal ultrasonography.

HRS has traditionally been considered "functional renal failure" in patients with end-stage liver disease. However, with advancing research, it is now recognized that HRS-AKI patients may also exhibit tubular injury, and the absence of significant proteinuria and/or hematuria does not rule out renal lesions, particularly tubular or interstitial damage. HRS-AKI can also occur in patients with underlying CKD. Urinary biomarkers such as α1/β2-microglobulin, the urinary sodium-to-potassium ratio, and cystatin C can help differentiate organic renal injury at an early stage.

HRS-NAKI[71] (including AKD and CKD): Refers to cases other than HRS-AKI, characterized by cirrhosis with or without ascites; estimated glomerular filtration rate < 60 mL/min/1.73 m^2 without other structural kidney lesions; or a Scr increase <50% from the most recent baseline value within three months. Possible causes include cholestatic nephropathy, hypovolemia due to gastrointestinal bleeding, excessive diuretic use, or large-volume paracentesis, as well as acute tubular injury, necrosis, or acute interstitial nephritis. According to the ICA, HRS-NAKI fulfills both CKD or AKD criteria and AKI criteria (i.e., Scr increase ≥50% or absolute increase ≥26.5 μmol/L [0.3 mg/dL] within 48 h). Compared with HRS-AKI, patients with HRS-NAKI typically present with higher organ failure scores, and ALB and vasoactive drugs are less effective.[72] Some overlap may exist between HRS-AKI and HRS-NAKI.

CKD in cirrhosis: CKD is defined as an estimated glomerular filtration rate < 60 mL/min persisting for at least three months, regardless of the presence of organic renal damage (proteinuria, hematuria, or abnormal renal ultrasound findings). The incidence of CKD is higher in patients with chronic liver disease than in the general population and is often accompanied by complications such as malnutrition and infections. Patients with severe or recurrent AKI are at increased risk of progressing to CKD. Those with MAFLD, chronic hepatitis B, hepatitis C, or other glomerular or interstitial nephropathies are more susceptible to developing AKI or CKD.

(6) HCC

In China, approximately 85% of HCC cases occur in the context of liver cirrhosis. Patients with cirrhosis should undergo early prevention, screening, and timely diagnosis to reduce the incidence and mortality of HCC. In the management of chronic liver disease, an LSM > 10.0 kPa indicates an increased risk of HCC, while patients with LSM > 13.0 kPa or SSM > 40 kPa should be considered for HCC surveillance. Cirrhotic patients require close monitoring with stratified

screening every three to six months, using ultrasound combined with alpha-fetoprotein and/or alpha-fetoprotein-L3, and/or protein induced by vitamin K absence or antagonist-II. Multiple early screening models and molecular biology–based detection techniques can improve the early diagnosis of HCC.[73,74] The diagnosis and treatment of HCC should follow the Guidelines for the Diagnosis and Treatment of Primary Liver Cancer (2024 Edition).[75]

(7) Thrombocytopenia

Thrombocytopenia is one of the common hematological abnormalities in early liver cirrhosis. Thrombocytopenia in cirrhosis is classified into four grades: Grade 1 (75×10^9/L ≤ PLT < 100×10^9/L), Grade 2 (50×10^9/L ≤ PLT < 75×10^9/L), Grade 3 (25×10^9/L ≤ PLT < 50×10^9/L), and Grade 4 (PLT < 25×10^9/L). Severe thrombocytopenia is defined as PLT <50×10^9/L. For these patients undergoing high-risk invasive procedures, reference should be made to the Practical Guidelines for the Clinical Management of Thrombocytopenia in Liver Cirrhosis.[76]

(8) PVT

PVT refers to thrombus formation in the main portal vein, its branches, or tributaries, leading to complete or partial vessel occlusion. It is a relatively common complication of liver cirrhosis. The clinical symptoms and prognosis of PVT vary widely, depending on factors such as the presence of cirrhosis, thrombus location, the extent of portal vein occlusion, and thrombus progression. Therefore, PVT evaluation involves staging (based on the time of thrombus formation) and grading/classification (based on the extent and location of occlusion).[77]

Acute portal vein thrombosis (aPVT): Defined as thrombus formation within six months or as thrombus occlusion < 50%. Mild aPVT may be asymptomatic but can present with abdominal pain, distension, nausea, or vomiting. Severe cases may manifest as acute portal hypertension syndrome, potentially leading to intestinal ischemia, obstruction, or necrosis. Anticoagulation with low-molecular-weight heparin, either alone or in combination with warfarin, is effective, with earlier treatment associated with higher rates of portal vein recanalization. Complete occlusion of the main portal vein in aPVT may also result in collateral vessel formation and cavernous transformation.

Chronic PVT: Defined as thrombus persisting for more than six months or as complete occlusion of the main portal vein with collateral vessel formation and cavernous transformation. Clinical manifestations range from asymptomatic to significant worsening of portal hypertension.[77] Contrast-enhanced CT typically shows low-density thrombi and features of cavernous transformation of the portal vein.

Importantly, patients with chronic PVT may experience thrombus progression, with extension into the superior mesenteric vein, splenic vein, or other vessels. This may present as worsening portal hypertension, abdominal pain, or intestinal ischemia/necrosis, and is often described as "acute-on-chronic portal vein thrombosis."

PVT is associated with poor outcomes, including increased three-year mortality and a higher incidence of HCC in patients with cirrhosis. Moreover, patients with complete PVT have a higher one-year post-transplant mortality than those without PVT, underscoring the importance of clinical screening and monitoring in cirrhotic populations. Management of PVT should follow current recommendations, including those from Baveno VII and the 2020 Practice Guidance by the American Association for the Study of Liver Diseases on vascular liver disorders, PVT, and procedural bleeding in patients with liver disease.[52,78]

(9) Hepatic osteodystrophy

Hepatic osteodystrophy refers to metabolic bone diseases characterized by abnormal bone mineral density in patients with chronic liver disease. It primarily manifests as osteoporosis (OP), osteopenia, and, rarely, osteomalacia. The incidence of OP in chronic liver disease ranges from approximately 12% to 55%, with its occurrence positively correlated with the severity of liver disease. OP can be secondary to cirrhosis (approximately 50%), primary biliary cholangitis (PBC)[79] (approximately 20%–44%), and alcoholic cirrhosis (approximately 56.7%). The risk of OP-related fractures in heavy drinkers is two to three times higher than in the general population. The current diagnostic standard for OP is based on dual-energy X-ray absorptiometry. Quantitative CT assessment of bone mineral density at the lumbar spine (L1–L4), left femoral neck, and total hip is highly sensitive.[80] When a fragility fracture occurs, a clinical diagnosis of OP can be made without relying on bone density testing. Vertebral compression fractures often lack symptoms and can easily be missed. Follow-up intervals should be determined based on the severity of OP, typically every three to twelve months. Bone density should be reassessed 12–18 months after initiating glucocorticoid therapy. Multidisciplinary collaboration among hepatologists, endocrinologists, and orthopedists is essential to optimize the diagnosis and management of hepatic osteodystrophy.

(10) Cirrhotic cardiomyopathy (CCM)

CCM: A chronic cardiac dysfunction associated with liver cirrhosis, characterized by impaired myocardial contractility and diastolic function in the absence of other known cardiac diseases.[81,82] CCM may be related to systemic inflammatory responses[83] and portal hypertension. The exact prevalence of CCM in cirrhotic patients is unclear, but it has been reported in approximately 50% of cases. Clinical manifestations are often insidious, with few obvious symptoms in the early stages. In advanced stages, heart failure may develop, presenting with symptoms such as chest tightness, shortness of breath, and peripheral edema. CCM progresses slowly, necessitating regular monitoring with electrocardiography, echocardiography, and cardiac MRI, with echocardiography and electrocardiography being particularly important. Diastolic dysfunction is a hallmark of CCM and may impact the prognosis of cirrhotic patients, regardless of whether they undergo liver transplantation.

CCM diagnostic criteria: ① Systolic dysfunction: Assessed by echocardiography. Dynamic stress testing, either exercise-induced or pharmacological, should be performed. A lack of increased cardiac output following physiological or pharmacological stress (in the absence of β-blocker influence) indicates systolic dysfunction. ② Diastolic dysfunction[84]: Defined by a ratio of early to late ventricular filling velocity (E/A) < 1.0, deceleration time > 200 ms, and isovolumic relaxation time > 80 ms. ③ Supporting criteria: Include electrophysiological abnormalities, impaired myocardial chronotropic response, prolonged QT interval, electromechanical dyssynchrony, left atrial enlargement, myocardial hypertrophy, elevated peripheral blood B-type natriuretic peptide and its precursor, and elevated troponin levels.

Approximately 40%–60% of cirrhotic patients exhibit electrophysiological abnormalities, with prolonged QT intervals more common in alcoholic cirrhosis. Clinicians should exercise caution when prescribing medications, particularly

non-selective beta-blockers (NSBBs), due to their potential adverse effects on cardiac electrophysiology.[85,86]

The existence of a hepato-cardiac syndrome remains controversial among domestic and international scholars, with limited research available.

Additionally, restrictive cardiomyopathy, severe tricuspid regurgitation, severe pulmonary hypertension, constrictive pericarditis, congenital heart disease, advanced cardiomyopathy, atrial fibrillation, and chronic heart failure from various causes can lead to cardiac cirrhosis,[87] which may also present with symptoms of portal hypertension. These conditions should be differentiated from CCM.

(11) Hepatopulmonary syndrome (HPS) and portopulmonary hypertension (PoPH)

HPS: Characterized by oxygenation abnormalities and a range of pathophysiological changes caused by intrapulmonary vascular dilation, primarily due to end-stage liver disease, portal hypertension, or congenital portosystemic shunts. Typical symptoms include exertional dyspnea or dyspnea at rest. Approximately 25% of HPS patients may exhibit platypnea (worsening dyspnea when moving from supine to upright) and orthodeoxia (PaO_2 decrease >5% or >4 mmHg when moving from supine to upright). In patients with severe HPS, mortality can be significantly reduced following liver transplantation.

HPS diagnostic criteria: ① Presence of liver disease, typically cirrhosis with portal hypertension; ② Positive contrast-enhanced transthoracic echocardiography — injection of 10 mL saline via a peripheral arm vein, with microbubbles visible in the left heart ≥3 cardiac cycles after right heart opacification; ③ Abnormal gas exchange in the lungs: alveolar-arterial oxygen gradient ≥ 15 mmHg (or >20 mmHg for patients aged >64 years). For further details, refer to the International Liver Transplantation Society Practice Guidelines: Diagnosis and Management of Hepatopulmonary Syndrome and Portopulmonary Hypertension.[88]

PoPH[89–91]: Refers to a condition characterized by pulmonary arterial hypertension in the context of portal hypertension. Diagnostic criteria: ① Clinical evidence of portal hypertension, such as esophagogastric varices, splenomegaly, ascites, or portal vein pressure measurement confirming portal hypertension; ② Mean pulmonary artery pressure > 25 mmHg; ③ Pulmonary vascular resistance > 2 Wood units; ④ Pulmonary artery wedge pressure < 15 mmHg.

(12) Sarcopenia and malnutrition in cirrhosis

Sarcopenia in cirrhosis, also known as cirrhotic muscle atrophy, is a common complication characterized by both reduced muscle mass and diminished muscle function. Its incidence ranges from approximately 40% to 70% and is closely associated with complications such as HE, infections, and ascites, contributing to a high mortality rate.[92] Measurement of the cross-sectional area of lumbar skeletal muscle via CT is a preferred method for diagnosing sarcopenia in cirrhosis, while objective indicators such as muscle strength and calf circumference can also be used for assessment.[93]

Recommendation 9: Serous cavity effusions in cirrhosis include ascites, hydrothorax, and pericardial effusion. Hydrothorax in cirrhotic patients is commonly right-sided; severe cases may present with bilateral hydrothorax, while a minority of patients have isolated left-sided hydrothorax. Attention should be paid to infections associated with serous cavity effusions in cirrhosis (A1).

Recommendation 10: EVB is the most common cause of gastrointestinal bleeding in cirrhosis. Attention should also be paid to gastrointestinal bleeding caused by PHG, PHE, PHC, and internal hemorrhoids in cirrhotic patients (A1).

Recommendation 11: HE should be differentiated from hepatic myelopathy, acquired hepatocerebral degeneration, and other neurological conditions. MHE and Grade 1 HE should be distinguished from age-related cognitive impairments in elderly patients, such as declines in memory, attention, and sensorimotor function (B1). Management of HE coexisting with neurological disorders in elderly cirrhotic patients requires attention (C1).

Recommendation 12: Renal impairment in cirrhotic patients includes AKI, HRS-AKI, HRS-NAKI, and CKD (A1).

Recommendation 13: Enhanced early prevention, screening, and early diagnosis of HCC should be implemented in all cirrhotic patients to reduce its incidence and mortality (B1).

Recommendation 14: Thrombocytopenia in cirrhosis is classified into four grades: Grade 1: 75×10^9/L ≤ PLT < 100×10^9/L; Grade 2: 50×10^9/L ≤ PLT < 75×10^9/L; Grade 3: 25×10^9/L ≤ PLT < 50×10^9/L; Grade 4: PLT < 25×10^9/L. Grades 3 and 4 (PLT < 50×10^9/L) constitute severe thrombocytopenia (B1).

Recommendation 15: PVT in cirrhosis is classified as acute (<6 months) or chronic (>6 months) (B1). Chronic PVT may progress, with thrombus extension or involvement of the mesenteric or splenic veins, leading to worsened abdominal pain, aggravated portal hypertension-related complications, or even intestinal ischemia and necrosis, a condition termed acute-on-chronic PVT (C1).

Recommendation 16: Hepatic osteodystrophy is a common but often underrecognized complication of end-stage liver disease, frequently presenting as OP and reduced bone mass, with a predisposition to fragility fractures. The severity of OP in cirrhosis correlates positively with the severity of liver disease (B1).

Recommendation 17: Cirrhotic patients exhibit a high incidence of electrophysiological abnormalities. Close attention should be paid to the potential adverse effects of medications, such as NSBBs, on cardiac electrophysiology, and to the screening for CCM (B1).

Recommendation 18: HPS and PoPH represent two distinct pulmonary vascular complications in patients with cirrhosis and/or portal hypertension, requiring careful differential diagnosis (B1).

Treatment of liver cirrhosis

Once cirrhosis is diagnosed, comprehensive treatment should be initiated as early as possible, including etiological therapy, anti-fibrotic therapy, and symptomatic management. Specific treatment goals are as follows: for patients with compensated cirrhosis, to achieve histological reversal of liver fibrosis and early cirrhosis through etiological and/or anti-fibrotic therapy to prevent progression to decompensated cirrhosis; for patients with decompensated cirrhosis, to strive for recompensation through etiological and/or anti-fibrotic therapy combined with management of complications.[94] For

all cirrhotic patients, the objectives include reducing HCC incidence, improving early diagnosis rates, and lowering mortality through etiological and/or anti-fibrotic therapy along with long-term clinical monitoring.

Primary and secondary prevention of cirrhosis and its complications abroad primarily focus on reducing portal vein pressure. In China, prevention strategies include: (1) etiological therapy; (2) interventions to reduce portal vein pressure; and (3) anti-fibrotic and cirrhosis-specific treatment. Etiological therapy and anti-fibrotic therapy can significantly reduce the risk of complications, HCC, and mortality in cirrhotic patients.[95]

For subclinical forms of decompensated cirrhosis, appropriate individualized primary or secondary prevention strategies should be developed based on the patient's clinical characteristics.

(1) Etiological treatment

Antiviral therapy for HBV-related cirrhosis should follow the Guidelines for the Prevention and Treatment of Chronic Hepatitis B (2022 Edition).[96] Patients with HBV-related cirrhosis who achieve HBsAg clearance or seroconversion through nucleos(t)ide analogs and/or pegylated interferon-alpha treatment do not meet the criteria for a "functional (clinical) cure" or recompensation. Despite HBsAg clearance or seroconversion, these patients do not achieve complete histological or liver function recovery and may still develop complications such as ascites or bleeding, with HCC incidence remaining higher than in HBV-infected individuals without cirrhosis. Some patients with HBsAg clearance or seroconversion continue to experience disease progression.[97–99] Therefore, HBV-related cirrhotic patients who achieve HBsAg clearance or seroconversion but do not reach recompensation or reversal require continued antiviral, anti-fibrotic, or anti-inflammatory therapy, with some needing long-term "dual therapy" (antiviral plus anti-fibrotic) until complete recompensation or reversal of cirrhosis is achieved.

Antiviral therapy for HCV-related cirrhosis should follow the Guidelines for the Prevention and Treatment of Hepatitis C (2022 Edition).[100] Treatment for hepatitis E virus-related cirrhosis should follow the Consensus on the Prevention and Treatment of Hepatitis E.[101]

For alcoholic cirrhosis, strict alcohol abstinence is required, as outlined in the Guidelines of Prevention and Treatment for Alcoholic Liver Disease: a 2018 update.[102] Treatment for metabolic-associated (non-alcoholic) fatty liver disease should follow the Guidelines for the Prevention and Treatment of Metabolic Dysfunction–Associated (Non-Alcoholic) Fatty Liver Disease (Version 2024).[103]

Treatment for autoimmune liver disease-related cirrhosis should follow the Guidelines on the Diagnosis and Treatment of Autoimmune Hepatitis (2021).[104] Guidelines on the Diagnosis and Treatment of Primary Biliary Cholangitis (2021).[105] and Guidelines on the Diagnosis and Treatment of Primary Sclerosing Cholangitis (2021),[106] respectively.

Treatment for Wilson's disease-related cirrhosis should follow the Guidelines for the diagnosis and treatment of hepatolenticular degeneration (2022 Edition),[107] while treatment for hemochromatosis-related cirrhosis should follow the Chinese Guidelines for the Diagnosis and Treatment of Hereditary Hemochromatosis.[108]

Treatment for drug- or chemical-induced cirrhosis should follow the Chinese Guidelines for the Diagnosis and Treatment of Drug-Induced Liver Injury (2023 version).[109]

For schistosomiasis- or clonorchiasis-related cirrhosis with active infection, praziquantel or similar agents may be administered after thorough assessment of liver function.[110,111]

For cirrhosis caused by other etiologies, the underlying cause should be identified and treated specifically. For example, in congestive cirrhosis due to right heart failure or constrictive pericarditis, the primary goal is to alleviate right heart overload; for hepatic outflow obstruction such as Budd–Chiari syndrome, the obstruction should be relieved.

(2) Anti-inflammatory and anti-fibrotic therapy

For patients who cannot undergo etiological treatment or who have persistent or progressive liver inflammation and/or fibrosis despite adequate etiological treatment, anti-inflammatory and anti-fibrotic therapies may be considered.

Commonly used anti-inflammatory hepatoprotective drugs include glycyrrhizic acid preparations (magnesium isoglycyrrhizinate, diammonium glycyrrhizinate, compound glycyrrhizin), bicyclol, polyene phosphatidylcholine, silymarin derivatives (silybin meglumine, silybin, silymarin), S-adenosylmethionine, and reduced glutathione. These drugs reduce liver tissue damage, promote hepatocyte repair and regeneration, alleviate intrahepatic cholestasis, and improve liver function by inhibiting inflammatory responses, supporting detoxification, modulating immunity, scavenging reactive oxygen species and free radicals, regulating energy metabolism, and improving hepatocyte membrane stability, integrity, and fluidity.[112]

Currently, no chemical anti-fibrotic drugs have been clinically validated as effective, whereas TCM has played a significant role.[113] Results from the National "12th Five-Year" and "13th Five-Year" Major Infectious Disease Projects indicate that "dual therapy," combining anti-HBV treatment with anti-fibrotic therapy, can improve and reduce liver fibrosis, lower the risk of cirrhosis complications, reduce HCC risk, improve survival, and reverse the course of cirrhosis. TCM medications such as Anluohuaxian pills, Fuzheng Huayu tablets/capsules, and compound Biejia Ruangan tablets exert anti-fibrotic effects, improve liver function, enhance immune function, and reduce portal hypertension by alleviating liver blood circulation disorders through mechanisms such as strengthening and tonifying deficiency, promoting blood circulation to resolve stasis, clearing heat (detoxification), and promoting diuresis.[114–122] These formulations embody the TCM principles of strengthening the body and eliminating pathogens while addressing both symptoms and root causes, with enhanced efficacy when based on TCM syndrome differentiation. Studies suggest that anti-fibrotic therapy should last at least one year, with prolonged treatment associated with reduced portal vein pressure, increased recompensation, and decreased HCC incidence.

(3) Prevention and treatment of complications

1. Ascites: Refer to the Guidelines on the management of ascites in cirrhosis (2023 version).[4]

Grade 1 ascites and mild Grade 2 ascites can be managed on an outpatient basis, while severe Grade 2 or Grade 3 ascites require hospitalization. First-line treatment includes controlling the underlying cause, reasonable salt restriction (4–6 g/day), and diuretics such as spironolactone and/or furosemide. Second-line treatment includes appropriate use of vasoconstrictive drugs (e.g., terlipressin, midodrine hydrochloride), other diuretics (e.g., tolvaptan), large-volume paracentesis with human ALB supplementation, and TIPS.[123] Third-line treatments include liver transplantation and renal replacement therapy.

For refractory ascites, triple therapy is recommended: diuretics, ALB, and vasoconstrictive drugs.[124] Large-volume paracentesis, TIPS, or long-term indwelling drainage catheters may also be considered.[125] Vasodilators, such as dopamine,

are not recommended.

For cirrhosis with chylous ascites, screening for underlying causes (e.g., tumors, tuberculosis, lymphatic obstruction) is necessary. Primary treatment includes a medium-chain triglyceride, high-protein, low-fat diet to reduce chyle production. Portal pressure-lowering drugs (e.g., terlipressin, somatostatin) may have some efficacy. If drugs are ineffective, TIPS or large-volume paracentesis may be attempted.[126]

For cirrhotic patients with hemorrhagic ascites, norepinephrine, terlipressin, or somatostatin may be used. Treatment principles for hydrothorax in cirrhosis are similar to those for ascites.

2. Gastrointestinal bleeding: Refer to the Guidelines on the management of esophagogastric variceal bleeding in cirrhotic portal hypertension.[6]

The primary causes of gastrointestinal bleeding in cirrhosis are EVB, PHG, and PHE. Patients with minor bleeding and stable vital signs can be closely monitored in a general ward, whereas those with massive bleeding should be admitted to the ICU.

1. EVB: Treatment principles include achieving hemostasis, restoring blood volume, reducing portal vein pressure, and preventing complications. During the acute bleeding phase, patients should fast and receive appropriate fluid resuscitation. Terlipressin, somatostatin, or its analogs can be used to lower portal vein pressure.[127] Proton pump inhibitors are recommended to suppress gastric acid and increase pH, thereby aiding hemostasis.[128] Antibiotics, such as third-generation cephalosporins, should be administered for three to five days to prevent infection.[129] Red blood cell transfusion may be necessary, targeting hemoglobin levels > 60–70 g/L.

   If pharmacological treatment is ineffective, endoscopic therapy, vascular interventions (TIPS), or surgical treatment may be considered. Endoscopic treatments include endoscopic variceal ligation (EVL), endoscopic injection sclerotherapy, or tissue adhesive injection, with terlipressin, somatostatin, or its analogs improving efficacy and safety. Anesthetic intubation and ICU support can further enhance the effectiveness and safety of emergency endoscopic treatment. Follow-up endoscopy should be performed two to four weeks after initial treatment to assess efficacy. High-risk patients with acute bleeding should receive early TIPS (within 72 h). For gastric variceal bleeding, balloon-occluded retrograde transvenous obliteration may be selected.[130,131] If pharmacological treatment is ineffective and emergency endoscopy or TIPS is unavailable, a Sengstaken-Blakemore tube may be used as a temporary salvage measure.

   After acute bleeding is controlled, secondary prevention should be initiated promptly. Endoscopy combined with NSBBs, such as carvedilol, is the first-line approach, with a response defined as a HVPG ≤ 12 mmHg or a >10% reduction from baseline. If HVPG cannot be measured, resting heart rate should be reduced to 75% of baseline or 50–60 beats/min.

   For patients with high bleeding risk, including those with EVB and concurrent PVT, TIPS may be considered.

   For moderate-to-severe esophagogastric varices with high bleeding risk (Child–Pugh B or C, or positive red signs), NSBBs (preferably carvedilol) or EVL are recommended to prevent first variceal bleeding.[132,133] For esophagogastric varices with ascites, primary and secondary prevention may cautiously use reduced doses of carvedilol or other NSBBs if blood pressure and pulse are within normal limits. Antiviral therapy combined with carvedilol can delay the progression of esophageal varices in compensated HBV-related cirrhosis.[134] Combining EVL with NSBBs is not recommended for primary prevention. Nitrate monotherapy or combination with NSBBs is not recommended for primary prevention.
2. PHG and PHE bleeding: PHG bleeding often presents as chronic bleeding and iron-deficiency anemia, with carvedilol as the first-line treatment, supplemented by iron therapy. For acute bleeding, pharmacological treatments are similar to those for EVB, including terlipressin or somatostatin analogs, and antibiotics may be administered. For both acute and chronic bleeding, if pharmacological treatment is ineffective or bleeding recurs, endoscopic therapy, TIPS, or surgical shunting may be considered. Secondary prevention may involve carvedilol or similar drugs to reduce portal pressure and the risk of bleeding. Treatment for PHE bleeding is similar to that for PHG, although the level of evidence is relatively lower.[135]

3. Hepatic encephalopathy: Refer to the Chinese guidelines on the management of hepatic encephalopathy in cirrhosis (2024).[7]

Early recognition and timely treatment are essential for improving HE prognosis. Addressing precipitating factors is critical, including common triggers such as infection, gastrointestinal bleeding, and electrolyte disturbances, as well as screening for abnormal portosystemic shunts.

The main treatment approaches focus on promoting ammonia excretion, reducing ammonia production, cleansing the gut, decreasing absorption of gut-derived toxins, and correcting amino acid imbalances, using agents such as lactulose, L-ornithine-L-aspartate, and rifaximin.[136–139] Prevention of HE following TIPS is crucial and involves appropriate patient selection, choosing the optimal stent diameter, and implementing postoperative dietary management and ammonia-lowering therapy.[69] The risk of HE is highest during the first month to one year after TIPS but tends to decrease with improved muscle mass and reduced stress events. Lactulose, L-ornithine-L-aspartate, or rifaximin may be used prophylactically to reduce the risk of HE.

4. Infections: Refer to the Guidelines on the management of ascites in cirrhosis (2023 version)[4] and the Expert consensus on diagnosis and treatment of end-stage liver disease complicated with infections (2021 version).[140]

Cirrhotic patients may develop infections at multiple sites caused by various pathogens, with the peritoneal cavity being the most common site, typically manifesting as SBP caused by gram-negative bacilli. Upon signs of infection, prompt etiological testing and initiation of empirical anti-infective therapy are recommended. Upon obtaining the results of etiological identification and drug susceptibility testing, antimicrobial therapy should be transitioned to a targeted approach as soon as possible. If etiological results are negative, further testing or treatment adjustments should be guided by the response to empirical therapy and disease progression. Attention should also be given to preventing secondary fungal infections.

In cases of sepsis and septic shock, vasoactive drugs can improve visceral organ perfusion and correct tissue ischemia and hypoxia. Norepinephrine is the first-line drug for septic shock. Low-dose vasopressin can effectively increase blood pressure and provide other physiological benefits in patients with septic shock; its use is recommended in combination with norepinephrine for adult patients with persistent hypotension.[141]

Human ALB can reduce systemic inflammation in patients with decompensated cirrhosis.[142] In sepsis and severe infections, ALB supplementation may be used alongside antibiotics.[4] Albumin not only increases colloid osmotic pressure but

also exhibits anti-inflammatory, antioxidant, and endothelial cell-stabilizing effects. For patients with confirmed SBP, ALB infusion should be administered alongside antibiotics, with greater benefit in those with baseline serum bilirubin ≥ 68 μmol/L (4 mg/dL) or creatinine ≥ 88 μmol/L (1 mg/dL).

Cirrhotic patients with ascites may use rifaximin for secondary prevention of SBP and HE.

5. Renal impairment: Refer to the Guidelines on the management of ascites in cirrhosis (2023 version)[4] and the Guidelines for the Diagnosis and Treatment of Liver Failure (2024 version).[143]

Correct hypovolemia, actively control infections, avoid nephrotoxic drugs, and carefully weigh the risks and benefits before using intravenous contrast agents to prevent AKI. If AKI occurs, reduce or discontinue diuretics, nephrotoxic drugs, vasodilators, or non-steroidal anti-inflammatory drugs; use crystalloids, human ALB, or other blood products as appropriate for volume expansion. Low-dose dopamine or other vasodilators are not recommended as renal-protective agents.

Terlipressin combined with ALB is superior to placebo, ALB alone, octreotide, or midodrine plus octreotide plus ALB in reversing HRS-AKI and HRS-NAKI and improving renal function.[144–146] Terlipressin is administered at 1 mg every 4–6 h combined with ALB 20–40 g/day for three days. If Scr decreases by less than 25%, terlipressin may be gradually increased to 2 mg every 4 h. If effective (Scr decreases to <133 μmol/L with increased arterial pressure, urine output, and serum sodium), treatment continues for seven to fourteen days; if ineffective, terlipressin is discontinued. Alternatively, norepinephrine (0.5–3.0 mg/h) combined with ALB (10–20 g/day) may be used.

TIPS can improve renal function in HRS-AKI and HRS-NAKI patients.[147] However, cirrhotic patients with ascites and HRS-AKI often present with severe conditions and may have contraindications to TIPS. Blood purification therapies (e.g., artificial liver support or renal replacement therapy) may improve renal function in some HRS-AKI patients. Liver transplantation remains the preferred treatment for HRS-AKI and HRS-NAKI.[143]

6. HCC: Treatment should follow the Guidelines for the Diagnosis and Treatment of Primary Liver Cancer (2024 Edition).[75] Surgical, ablation, interventional, chemical, targeted, immunological, and radiation therapies should be selected based on the patient's condition.

7. Thrombocytopenia: Refer to the Chinese expert consensus for the management of thrombocytopenia in cirrhosis.[76,148] For invasive procedures, surgery, or antitumor drug therapy, thrombopoietin-class drugs, including recombinant human thrombopoietin and thrombopoietin receptor agonists (e.g., avatrombopag, lusutrombopag), may be used based on the recommended platelet threshold (typically 50 × 10^9/L).[149–151] Compared to controls, recombinant human thrombopoietin treatment significantly increased PLTs from baseline, with elevated serum thrombopoietin and hepatocyte growth factor levels after seven days, and significant improvements in MELD score, ALB levels, and INR after 14 days.[149]

For splenomegaly with hypersplenism, partial splenic artery embolization or splenectomy can increase platelet, white blood cell, and hemoglobin levels[152,153]; however, indications remain controversial, and prophylactic splenectomy is not recommended in patients without a history of gastrointestinal bleeding.

8. PVT: The treatment goal for acute or acute-on-chronic PVT is to recanalize the occluded portal vein, prevent progression from acute to chronic thrombus, and avoid thrombus extension.[154] The primary treatment is pharmacological anticoagulation,[154–156] using non-vitamin K antagonist oral anticoagulants (e.g., rivaroxaban), low-molecular-weight heparin, or warfarin. Anticoagulation typically lasts three to six months, with regular assessment of bleeding and thromboembolism risks during treatment. Other options include TIPS, thrombolysis, and surgical interventions.[157] Chronic PVT requires individualized treatment.

9. Hepatic osteodystrophy: Patients with OP may use calcium and vitamin D.

10. CCM: No specific drugs are available, and pharmacological treatments have limited efficacy.[158] In CCM patients with overt heart failure, volume load restriction is critical. Cardiac glycosides do not effectively improve cardiac contractility in CCM patients. Drugs that prolong the QT interval should be used cautiously. Vasodilators are contraindicated in patients without hypertension, and diuretics should be used cautiously. Liver transplantation may help alleviate CCM.[159]

11. HPS and PoPH: No effective pharmacological treatments are available. For significant hypoxemia, oxygen therapy may be provided, with liver transplantation being the primary intervention to alter disease outcomes.[160,161] When PaO_2 < 80 mmHg, low-flow oxygen (2–4 L/min) may be administered via nasal cannula or mask; for patients with increased oxygen requirements, pressurized masks or endotracheal intubation may be used. Reports indicate that HPS patients receiving oxygen therapy for up to one year showed improved liver function (Child–Pugh C improved to A) and resolution of ascites.[162] Growth hormone replacement therapy may improve HPS in MAFLD cirrhotic patients with hypopituitarism.[163] The benefit of TIPS remains unclear.[147]

For PoPH, drugs to reduce pulmonary artery pressure include endothelin receptor antagonists (e.g., ambrisentan), phosphodiesterase-5 inhibitors (e.g., sildenafil), prostacyclin analogs (e.g., treprostinil), and guanylate cyclase stimulators (e.g., riociguat).[91] Notably, calcium channel blockers are ineffective in reducing pulmonary artery pressure, and NSBBs may exacerbate pulmonary hypertension.

12. Nutritional support: Refer to the Clinical guidelines on nutrition in end-stage liver disease.[50] For malnourished cirrhotic patients, daily energy intake should be 30–35 kcal/kg, and protein intake 1.2–1.5 g/kg, with plant-based proteins preferred. In cases of severe HE, oral protein intake may be reduced or temporarily restricted, with supplementation of branched-chain amino acids, gradually increasing protein intake to the target amount based on patient tolerance. Intravenous ALB infusion can improve liver function and quality of life.[164] Patients with decompensated cirrhosis or at nutritional risk should supplement vitamins and trace elements. Prolonged fasting should be avoided, and four to six small, frequent meals per day are recommended, especially a bedtime snack (e.g., whole-grain products and high-quality proteins such as cheese) to reduce protein catabolism from prolonged overnight fasting.

13. Long-term follow-up management: For cirrhotic patients, liver biochemistry, complete blood count, blood ammonia, coagulation function, alpha-fetoprotein, and abdominal ultrasound should be rechecked every one to three months, depending on the patient's condition, to monitor disease progression, particularly for early HCC. Patients with severe conditions require ongoing monitoring, and long-term clinical management plans should be developed for those with recurrent complications.

14. Nursing and psychological support: During massive gastrointestinal bleeding, maintain a patent airway, position the patient supine with the head turned to one side, promptly clear blood clots to prevent aspiration, closely mon-

itor vital signs, and observe skin and nail bed color and limb temperature. Establish two or more intravenous access lines to ensure effective delivery of blood products and medications, maintaining blood pressure around 90/60 mmHg and hourly urine output ≥ 30 mL.

HE nursing: "Three preventions and three protections." The "three preventions" include preventing patients from wandering off, harming others, or self-harm. The "three protections" include bed rails, restraint belts (with family consent), and gloves. Monitor HE patients' personality, behavior, consciousness, mental status, and neurological signs; pay attention to dietary structure, particularly daily protein intake, and carefully record intake and output.

Cirrhotic patients often experience low mood, anxiety, depression, and fear. Targeted psychological support and interventions can alleviate negative emotions, improve treatment adherence, enhance condition management, and improve quality of life.[165]

Recommendation 19: Primary and secondary prevention strategies for cirrhosis and its complications include: (1) etiological treatment; (2) therapies to reduce portal vein pressure; and (3) anti-fibrotic and anti-cirrhosis treatment. These interventions can significantly reduce the risk of complications, HCC, and mortality in cirrhotic patients (A1).

Recommendation 20: Prevention strategies for subclinical forms of decompensated cirrhosis should be tailored to the individual patient's clinical characteristics, implementing appropriate personalized primary or secondary prevention measures (C1).

Recommendation 21: Actively pursue etiological and anti-fibrotic therapy (dual therapy), using medications such as Anluohuaxian pills, Fuzheng Huayu tablets/capsules, and compound Biejia Ruangan tablets, with a treatment duration exceeding 12 months (A1).

Recommendation 22: Antiviral therapy should be initiated for treatment-naive patients diagnosed with HBsAg-negative hepatitis B cirrhosis. Those who achieve HBsAg loss or seroconversion through nucleos(t)ide analogs and/or pegylated interferon-alpha treatment do not meet the criteria for a "functional (clinical) cure" and should continue antiviral and anti-fibrotic therapy (dual therapy) until complete recompensation or reversal of cirrhosis is achieved (B1).

Recommendation 23: Treatment of refractory ascites includes: salt restriction with sodium intake of 4–6 g/day (B1); use of diuretics, human ALB, and vasoconstrictive drugs; large-volume paracentesis (4,000–5,000 mL per session) combined with human ALB infusion (4 g per liter of ascites removed) (B1); consideration of TIPS if pharmacological therapy is ineffective and portal hypertension is the primary cause; cautious use of ascitic fluid drainage via indwelling peritoneal catheter (C2); and priority listing for liver transplantation (B2).

Recommendation 24: For chylous ascites or hydrothorax, administer a medium-chain triglyceride, high-protein, low-fat diet, along with terlipressin and somatostatin; if ineffective, paracentesis or TIPS may be considered (C1).

Recommendation 25: For hemorrhagic ascites, medications such as norepinephrine, terlipressin, and somatostatin may be used (C1).

Recommendation 26: For upper gastrointestinal bleeding in cirrhosis, terlipressin, somatostatin analogs, proton pump inhibitors, or H2 receptor blockers can be administered (A1).

Recommendation 27: For cirrhosis with EVB unresponsive to pharmacological treatment, a Sengstaken-Blakemore tube may be used as a temporary bridge, followed by EVL, sclerotherapy, or tissue adhesive treatment (B1); interventional therapy (C1); or surgical treatment (C2).

Recommendation 28: Secondary prevention should be initiated five to seven days after cessation of gastrointestinal bleeding in cirrhosis. This may include gastroscopy combined with carvedilol or similar drugs (B1). For primary and secondary prevention of gastrointestinal bleeding in patients with ascites, carvedilol or other NSBBs may be used cautiously at reduced doses, provided blood pressure and pulse are stable (D2).

Recommendation 29: For chronic blood loss due to PHG or PHE, carvedilol and iron supplementation are recommended, with blood transfusion when indicated (B1). For acute bleeding, terlipressin or somatostatin analogs may be used (B2).

Recommendation 30: Actively identify and eliminate HE triggers, promote ammonia excretion, reduce ammonia production, cleanse the gut, reduce absorption of gut-derived toxins, and correct amino acid imbalances. Lactulose (A1), L-ornithine-L-aspartate (A1), and alpha-crystalline rifaximin (B1) may be administered.

Recommendation 31: For HRS, terlipressin (1 mg every 4–6 h) combined with human ALB (20–40 g/day) may be used for seven to fourteen days, with repeat treatment for responders upon recurrence (B1). Vasodilators such as dopamine are not recommended (B1).

Recommendation 32: TIPS may be considered for HRS-NAKI patients with massive ascites who do not respond to vasoconstrictor drug therapy (B1). TIPS is not recommended for HRS-AKI (C2).

Recommendation 33: For HRS-AKI unresponsive to vasoconstrictive drugs and meeting criteria for renal replacement therapy, renal replacement therapy or artificial liver support systems may be considered (B1). Renal replacement therapy is not recommended for HRS-NAKI (C2). Both HRS-AKI and HRS-NAKI patients should be prioritized for liver transplantation (B1).

Recommendation 34: For cirrhotic patients with infection, initiate empirical anti-infective therapy promptly. Transition to targeted anti-infective therapy is recommended as soon as pathogen identification and drug sensitivity results are available (B1).

Recommendation 35: For cirrhosis with sepsis, severe infection, or septic shock, combination therapy with antibiotics, ALB, and vasoactive drugs may be used (B1).

Recommendation 36: The treatment for HCC should be selected based on the patient's condition, including surgical, ablative, interventional, chemotherapeutic, targeted, immunological, radiotherapeutic, or systemic therapy options (B1).

Recommendation 37: In thrombocytopenic patients undergoing invasive procedures, surgery, or antitumor drug therapy, thrombopoietin-class drugs, including recombinant human thrombopoietin and thrombopoietin receptor agonists, may be used according to the required platelet threshold (B1).

Recommendation 38: Acute or acute-on-chronic PVT in cirrhosis should be treated with anticoagulation or thrombolysis (C1), using rivaroxaban, low-molecular-weight heparin, or warfarin. Active bleeding, such as gastrointestinal hemorrhage, is a contraindication for anticoagulation therapy (B1).

Recommendation 39: For hepatic OP, calcium and vitamin D supplementation may be used (C2).

Recommendation 40: For CCM, improve cardiac function, use drugs that may prolong the Q-T interval cautiously, and list patients for liver transplantation (B1).

Recommendation 41: No specific drugs are available for HPS. Long-term oxygen therapy is suggested for patients with HPS and severe hypoxemia (C1), and liver transplantation is recommended (B1). NSBBs may exacerbate PoPH (C1).

Recommendation 42: Nutritional support therapy is essential for cirrhotic patients to prevent and reduce sarcopenia, with four to six small, frequent meals daily (B1).

Recommendation 43: For all cirrhotic patients, liver biochemistry, coagulation function, alpha-fetoprotein, and abdominal ultrasound should be rechecked at least every 3 months, depending on the patient's condition, to monitor disease progression, particularly for early detection of HCC.

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Conflict of interest

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Issues to be addressed

1. Development of intelligent liver pathology interpretation systems.
2. Development of new technologies for non-invasive, dynamic HVPG measurement in cirrhotic patients.
3. Development of next-generation LSM and SSM diagnostic technologies.
4. Development of simple, highly specific, sensitive, and practical detection methods for MHE.
5. Research on the clinical diagnosis and management of metabolic-associated fatty liver cirrhosis.
6. Research on diagnostic criteria and influencing factors for recompensation/reversal of cirrhosis.
7. Research on prevention and clinical management strategies for subclinical forms of decompensated cirrhosis.
8. Clinical research on TCM for anti-fibrotic therapy and recompensation/reversal in various types of cirrhosis.
9. Research on the dosage, duration, and comprehensive effects of human ALB in decompensated cirrhosis.
10. Research on the clinical significance of long-term stable improvement of PLTs in cirrhosis.
11. Clinical research on nutrition and sarcopenia in cirrhosis.
12. Research on comprehensive, multidisciplinary management of cirrhosis using artificial intelligence and big data.

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