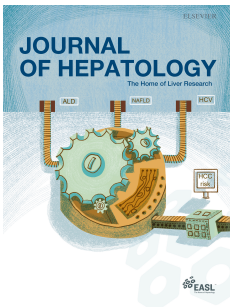

Baveno VIII – Advancing Consensus in Portal Hypertension

Baveno VIII Faculty

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TITLE

Baveno VIII – Advancing Consensus in Portal Hypertension

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Endorsed by

European Association of the Study of the Liver (EASL)

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KEYWORDS

CSPH, advanced chronic liver disease, ACLD, NIT, decompensation, cirrhosis recompensation, hepatocellular carcinoma, HCC, vascular liver disease, BCS, PSVD, NCPF, PVT, steatotic liver disease

ABSTRACT

Since the first Baveno consensus conference in 1990, successive conferences every 5 years have transformed the field of cirrhosis and portal hypertension evolving from a focus on variceal bleeding to a pathophysiology-based framework centered on risk stratification, clinically significant portal hypertension (CSPH), and prevention of decompensation. Building on this progress, the Baveno Cooperation, in collaboration with the Vascular Liver Disease Interest Group (VALDIG), convened an expert faculty to review evidence since Baveno VII and update clinical guidance for patients with advanced chronic liver disease (ACLD), portal hypertension, and vascular liver diseases.

The Baveno VIII Consensus Conference, held in March 2026 in Baveno, Italy, was based on a structured consensus process across nine panels addressing key domains in ACLD and vascular liver diseases and complications related to portal hypertension. Statements and recommendations were developed through iterative review of the evidence and voting, with strong consensus defined as >80% faculty agreement.

A total of 272 statements and recommendations achieved strong consensus. Key areas included non-invasive risk stratification in compensated ACLD (cACLD), modifiers of steatotic liver disease, prevention of first and further decompensation, management of portal hypertensive bleeding, cirrhosis recompensation, and vascular liver diseases, including Budd–Chiari syndrome, portosinusoidal vascular disorder (PSVD) or non-cirrhotic portal fibrosis (NCPF), and portal vein thrombosis (PVT). The key role of CSPH and the expanding use of non-invasive tests was confirmed and cutoffs revised. New to Baveno VIII is the inclusion of selected guidance for paediatric patients. Recommendations from prior conferences that were not specifically revised in Baveno VIII remain valid, such as those on hepatic venous pressure gradient (HVPG) measurement. A research agenda identifying 153 priority topics is proposed.

INTRODUCTION

The Baveno Cooperation, an official consortium of the European Association for the Study of the Liver (EASL) founded in its current framework in 2017, aims to advance knowledge in advanced chronic liver disease (ACLD), vascular liver diseases and portal hypertension. Starting with a very first meeting organized in Groningen, the Netherlands in 1986, the subsequent consensus workshops aimed to harmonize definitions, guide clinical research, and develop consensus-based recommendations, contributing to a shift toward a stage-based, prevention-oriented approach in ACLD/cirrhosis and portal hypertension. The academic rigour and clinical interest has led to the organization of a series of global consensus meetings: Baveno I(1) (1990, Baveno, Italy); Baveno II(2) (1995, Milan, Italy); Baveno III(3) (2000, Stresa, Italy); Baveno IV(4) (2005, Baveno, Italy); Baveno V(5) (2010 (Stresa, Italy); Baveno VI(6) (2015, Baveno, Italy), Baveno VII(7) (2021: virtual conference due to the COVID-19 pandemic), and finally Baveno VIII (2026, Baveno, Italy).

The Baveno VIII Consensus Conference (2026) provides updated statements and recommendations based on evidence accrued since Baveno VII. The statements and recommendations are presented across nine panels: (1) non-invasive test (NIT)-based risk stratification in compensated ACLD (cACLD); (2) modifiers of steatotic liver disease (SLD); (3) prevention of first decompensation; (4) prevention of further decompensation; (5) management of portal hypertension–related gastrointestinal bleeding; (6) management of further decompensation; (7) cirrhosis recompensation; (8) vascular liver diseases, including Budd–Chiari syndrome (BCS), and portosinusoidal vascular disorder (PSVD) also termed non-cirrhotic portal fibrosis (NCPF); and (9) portal vein thrombosis (PVT) both in patients without underlying liver disease and in patients with ACLD. For the first time, paediatric hepatologists were included in some of the panels and the document includes statements related to management of paediatric liver disease and portal hypertension.

Each panel was composed of two chairs and 4–8 panelists (n=79). Proposed statements were iteratively reviewed by the full Baveno faculty (n=84), including chairs, panelists, and invited lecturers (n=5) and were subjected to consensus voting. Those achieving ≥80% agreement, were approved and are presented below,

organized by panel. In total, 272 statements and recommendations achieved consensus, all of which were graded by level of evidence (according to the Oxford Centre for Evidence-based Medicine), strength of the recommendation and whether they were new, modified or unchanged compared to Baveno VII. Each panel also provided a research agenda including a total of 153 priority topics. Finally, a selection of statements and recommendations of previous Baveno consensus reports that remain valid but were not specifically updated for Baveno VIII are shown in a summary box.

Panel 1 – Risk Stratification in cACLD

1.1. The term “compensated advanced chronic liver disease (cACLD)” reflects the continuum of severe fibrosis and cirrhosis. A pragmatic definition of cACLD based on liver stiffness measurement (LSM)* aims to identify those at risk of CSPH, decompensation, and liver-related death at point of care, irrespective of histological stage or the ability of LSM to identify these stages. (LoE 3; modified; Agreement: 100%)

1.2. “Compensated cirrhosis” and “cACLD” are not interchangeable, and the latter is preferred if the diagnosis is established by non-invasive tests (NITs). (LoE 3; modified; Agreement: 97%)

1.3. LSM values <10 kPa in the absence of other known clinical/imaging signs rule out cACLD; values between 10 and 15 kPa are suggestive of cACLD; values >15 kPa are highly suggestive of cACLD. (LoE 3; SoR: strong; modified; Agreement: 100%)

1.4. LSM should always (i.e., for diagnosis of cACLD or CSPH) be performed under fasting conditions. In patients with an index LSM ≥10 kPa, LSM should either be repeated (LoE 2, SoR: strong), or may be complemented by another validated NIT of advanced fibrosis (LoE 3; SoR: weak) to confirm cACLD (modified; Agreement: 97%)

1.5. Patients with chronic liver disease (which does not include vascular liver disease – see respective section) and an LSM <10 kPa have a negligible 3-year risk (≤1%) of decompensation and liver-related death. (LoE 1; modified – minimal; Agreement: 98%)

1.6. LSM values <10 kPa in patients with clinical/imaging signs of portal hypertension should raise the suspicion for vascular liver disease (see respective section). (LoE 3; SoR: strong; new; Agreement: 100%)

1.7. Patients with cACLD should be referred to a liver disease specialist for further work-up. (LoE 3; SoR: strong; unchanged); Agreement: 100%)

1.8. In patients with cACLD, LSM holds prognostic information. (LoE 1) A rule of 5 for LSM (10-15-20-25 kPa) denotes progressively higher relative risks of decompensation and liver-related death, independently of the aetiology of chronic liver disease. (LoE 2; modified; Agreement: 100%)

1.9. In patients with cACLD, NITs for CSPH (see below) should be repeated every 12 months. (LoE 3; SoR: strong; modified Agreement: 97%)

1.10. Changes in LSM confer prognostic information; however, the most recent value should guide clinical decision-making. (LoE 2; SoR: strong; new; Agreement: 97%)

1.11. Although the concept of CSPH is based on HVPG, validated elastography- and validated NITs (see 1.12-1.16) are sufficiently accurate to exclude or identify CSPH and are similarly predictive of hepatic decompensation in cACLD. (LoE 1) The body of evidence for rare aetiologies is more limited. (modified; Agreement: 97%)

1.12. LSM and platelet count (PLT), as well as their derivatives (i.e., ANTICIPATE models) are the mainstay of non-invasive diagnosis of CSPH. (LoE 1; modified; Agreement: 94%)

1.13. Spleen stiffness measurement (SSM) may further improve the classification of CSPH. (LoE 2; new; Agreement: 94%)

1.14. ANTICIPATE (ANTICIPATE-NASH for people with MASLD and obesity [≥30 kg/m^2]) (LoE 1; SoR: strong) or NICER (LoE 2; SoR: strong) should be used to non-invasively estimate the probability of CSPH in cACLD. (New; Agreement: 97%)[1*]

1.15. In patients with cACLD, LSM ≤15 kPa and PLT ≥150×10^9/L rule out CSPH (negative predictive value >90%)[2*]. (LoE 1; SoR: strong; modified; Agreement: 100%)

1.16. In patients with cACLD, the following criteria are recommended to establish the diagnosis of CSPH, while considering their individual limitations: (New)

a. Estimated probability of CSPH ≥75% by ANTICIPATE/ANTICIPATE-NASH (LoE 1; SoR: strong) or NICER. (LoE 2; SoR: strong; Agreement: 93%)

b. In virus- and/or alcohol-related cACLD and non-obese (BMI <30 kg/m^2) MASLD-related cACLD, LSM ≥25 kPa. (LoE 1; SoR: strong; Agreement: 91%)

c. SSM (100 Hz) >55 kPa. (LoE 2; SoR: strong; Agreement: 88%)

1.17. In patients with cACLD and NITs in the indeterminate range (i.e., not meeting rule out or diagnostic criteria for CSPH), re-evaluation at 12 months may be the preferred management strategy. (LoE 4; SoR: weak; new; Agreement: 88%)

(*) throughout this document, liver stiffness measurement (LSM) refers to values obtained by vibration-controlled transient elastography (VCTE), unless otherwise specified

1.18. Given the need to identify patients at substantial risk of decompensation, who benefit the most from preventive treatments (e.g., carvedilol/non-selective beta-blockers [NSBBs] and investigative therapies), the paradigm is shifting from non-invasive estimation of CSPH probability to non-invasive prediction of decompensation as a direct endpoint. Key prognostic indicators for decompensation include presence of CSPH, hepatic function, and aetiological factors. (LoE 2; new; Agreement: 94%)

1.19. Patients with cACLD who are ineligible for NSBB (contraindications or intolerance) to prevent decompensation should undergo oesophago-gastro-duodenoscopy (EGD) to screen for varices, unless LSM is <20 kPa and PLT ≥150 $\times 10^9$/L, or SSM <40 kPa (LoE 1; SoR: strong; modified; Agreement: 100%

1.20. Patients with cACLD who are ineligible for NSBB therapy to prevent decompensation and who do not have varices at index EGD should undergo repeat EGD, unless they subsequently meet criteria specified in 1.19. Time intervals for repeating EGD are 2 years for cACLD patients with an active aetiological factor and 3 years for those in whom the primary aetiological factor has been removed/suppressed. (LoE 4; SoR: strong; new; Agreement: 100%)

Use of NITs in patients with hepatocellular carcinoma (HCC)

1.21. In patients with macrovascular invasion, NITs for CSPH/high-risk varices may not be applicable (LoE 3; SoR: weak; new; Agreement: 94%)

1.22. In patients with cACLD and Barcelona Clinic Liver Cancer (BCLC) stages 0-C, LSM ≤15 kPa and PLT ≥150$\times 10^9$/L rule out CSPH (negative predictive value >90%) and EGD may be avoided. (LoE 3; SoR: weak; new; Agreement: 94%)

1.23. In patients with cACLD and BCLC stages 0-A without large (>5cm) nodules in the right liver lobe, an estimated probability of CSPH ≥75% by ANTICIPATE/ANTICIPATE-NASH may be highly suggestive of CSPH. (LoE 3; SoR: weak; new; Agreement: 95%)

Panel 1 - Paediatric Statements

1.24. In children, extrahepatic portal vein obstruction and advanced chronic liver disease (ACLD) due to biliary atresia are the two leading causes of portal hypertension. (LoE 1; new; Agreement: 95%)

1.25. LSM is a valuable complementary tool that enables the non-invasive identification of children at risk for portal hypertension due to ACLD and helps to monitor disease progression. (LoE 3, SoR: strong; new; Agreement: 97%

1.26. In paediatric patients, management of portal hypertension cannot rely solely on LSM and/or SSM, as cut-offs to identify the presence or absence of varices require further validation. (LoE 3; SoR: strong; new; Agreement: 100%)

1.27. In children, integrating multiple NITs (e.g., the clinical prediction rule based on the age-adjusted Z-score for spleen length, PLT, and serum albumin) may facilitate prediction of high-risk varices and guide endoscopy intervals. (LoE 3; new; Agreement: 100%)

Panel 1 – Research Agenda

RA1.1. *Validation of non-VCTE elastography methods and VITRO for diagnosing CSPH and in prognostication*

RA1.2. *NIT-based diagnosis of CSPH and prognostication in rare liver disease aetiologies*

RA1.3. *Confirmation of the utility of sequential NIT reassessments to guide patient management*

RA1.4. *NIT-based monitoring of changes in HVPG*

RA1.5. *Development of a generally accepted prognostic model for cACLD*

RA1.6. *AI-based data integration for diagnosing CSPH and in prognostication*

RA1.7. *Evaluation of the added value of minimally invasive tests in patients with NITs in the indeterminate range (i.e., not meeting rule out or diagnostic criteria for CSPH)*

RA1.8. *NIT-based prediction of surgical risk (hepatic and extra-hepatic)*

RA1.9. *NIT-based criteria for (ruling-out) varices in paediatric patients*

RA1.10. *NIT-based models for predicting bleeding in paediatric patients*

RA1.11. *AI-based data integration for diagnosing varices and prognostication in paediatric patients*

Panel 2 – Modifiers of steatotic liver disease (SLD)

Alcohol abstinence and liver-related events

2.1. Return to/continuation of alcohol use is a major driver of liver-related events in patients with alcohol-related cACLD. (LoE: 2; new; Agreement: 98%)

2.2. In patients with alcohol-related cACLD, complete alcohol abstinence should be favoured over reduction of alcohol consumption to decrease liver-related events. (LoE: 2; SoR: strong; new; Agreement: 100%)

Interpretation of NIT changes in ALD

2.3. In patients with alcohol-related cACLD, a confirmed ≥30% relative change in LSM is associated with a corresponding change in the risk of liver-related events. (LoE: 5; new; Agreement: 94%)

2.4. In patients with alcohol-related liver disease, FIB-4, LSM, Enhanced Liver Fibrosis (ELF) test and Fibrotest are associated with liver-related events and survival. (LoE: 2; new; Agreement: 100%)

Interpretation of NIT changes in MASLD

2.5. In MASLD-related cACLD, HVPG may underestimate the true portal pressure gradient. However, an HVPG ≥10 mmHg remains associated with a significantly increased risk of decompensation compared with the lower, albeit still present, risk observed with HVPG <10 mmHg. (LoE: 3; new; Agreement: 98%)

2.6. In patients with MASLD-related cACLD without CSPH, a confirmed ≥30% relative reduction in LSM may indicate a ≥1 stage fibrosis regression. (LoE: 3; new; Agreement: 90%

2.7. In patients with MASLD-related cACLD without CSPH, combining a confirmed ≥30% relative reduction in LSM with improvement in an independent non-invasive test (e.g., ≥0.5-unit absolute reduction in ELF) improves the assessment of ≥1 stage fibrosis regression compared with LSM alone. (LoE: 3; new; Agreement: 88%

2.8. NITs and their longitudinal changes should preferentially be calibrated to estimate the risk of liver-related events and its modification over time rather than to assess fibrosis stage or fibrosis stage change. (LoE 4; new; Agreement: 91%)

2.9. In patients with MASLD-related cACLD, a confirmed ≥30% relative change in LSM is associated with a clinically meaningful change in the risk of liver-related events, particularly when it results in crossing the 15 kPa risk threshold. (LoE: 2; new; Agreement: 96%)

2.10. Although the confirmed relative changes in LSM by point-shear wave elastography and 2D shear wave elastography likely carry similar clinical meaning as LSM, the available evidence is currently insufficient to support a specific recommendation on their use as monitoring tools. (LoE: 4; new; Agreement: 97%)

2.11. A ≥20% relative change in LSM by magnetic resonance elastography is associated with a clinically meaningful change in the risk of liver-related events in patients with MASLD-related cACLD. (LoE: 4; new; Agreement: 94%)

2.12. A 0.5-unit absolute change in ELF is associated with a clinically meaningful change in the risk of liver-related events in patients with MASLD. (LoE: 3; new; Agreement: 96%)

2.13. In patients with MASLD-related cACLD, ≥10% weight reduction is associated with a ≥30% relative reduction in LSM, a surrogate of reduced risk of liver-related events. (LoE: 3; new; Agreement: 83%)

Metabolic and bariatric surgery

2.14. In patients with MASLD-related cACLD without CSPH, metabolic and bariatric surgery is associated with only a modest increase in the risk of surgical complications and may improve liver histology and liver-related events. Therefore, MASLD-related cACLD may be considered an obesity-associated morbidity that justifies consideration of metabolic and bariatric surgery at a lower BMI threshold. (LoE: 3; new; Agreement: 96%)

2.15. Surgical complications from metabolic and bariatric surgery are increased in patients with CSPH. Patients with features suggestive of CSPH who are being considered for metabolic and bariatric surgery should be managed by a multidisciplinary team at expert centers. (LoE: 3; SoR: strong; new; Agreement: 100%)

2.16. Metabolic and bariatric surgery is generally contraindicated in patients with decompensated cirrhosis, except in highly specialised settings, such as in conjunction with liver transplantation. These patients should be managed by a

multidisciplinary team at expert centers. (LoE: 3; SoR: strong; new; Agreement: 98%)

Therapeutic agents for steatotic liver disease

2.17. There are currently no regulatory approved pharmacologic therapies for the treatment of patients with MASLD and established cirrhosis. Efficacy data derived from noncirrhotic MASLD populations (moderate or advanced fibrosis) should not be extrapolated to patients with established cirrhosis. (LoE: 2; SoR: strong; new; Agreement: 98%)

2.18. In the absence of approved disease-modifying therapies, management of MASLD-related cirrhosis should focus on optimization of lifestyle and metabolic comorbidities, prevention of hepatic decompensation, and referral for clinical trial participation. (LoE: 3; SoR: strong; new; Agreement: 100%)

2.19. SGLT2 inhibitors and GLP-1–based therapies, including mono- and multi-receptor agonists, can be safely used in patients with cACLD, in the absence of contraindications, for their approved indications, including type 2 diabetes and, for GLP-1–based therapies, obesity. (LoE 3; SoR: strong; new; Agreement: 96%)

Panel 2 – Research Agenda

RA2.1 *Further studies are needed to validate whether the most recent LSM result predicts liver-related events as accurately as longitudinal changes in LSM.*

RA2.2 *Studies are needed to determine the optimal strategy for repeating LSM to improve its reliability, including the timing of repeated examinations (e.g., at a different time on the same day or on a different day, etc.) and whether these LSM examinations should be performed by the same or a different operator.*

RA2.3 *Studies are needed to assess whether calculating longitudinal changes in LSM using confirmed LSM instead of single point measurements can reduce intra-individual variability and allows adoption of a lower threshold for percentage change to define a true LSM response in SLD.*

RA2.4 *Studies are needed to accurately determine the impact of obesity on LSM in SLD as well as across other aetiologies of chronic liver disease.*

RA2.5 *Data are needed on the diagnostic and/or prognostic value of non-VCTE-based modalities to obtain LSM.*

RA2.6 *Further data are needed to determine how, and with what accuracy, longitudinal changes of LSM by different modalities correlate with changes in prognosis in patients with ALD.*

RA2.7 *The impact of weight loss, and the correlation between the magnitude of weight loss and effect on portal hypertension, assessed invasively or non-invasively, should be studied further in MASLD, MetALD and in ALD with overweight/obesity considered separately.*

RA2.8 *Further studies are needed to refine NITs and their thresholds for the diagnosis of CSPH specifically in the context of SLD, for a more precise prediction of the actual portal pressure gradient and/or the prognosis/risk of decompensation in patients with MASLD and obesity, and to assess their sensitivity to change.*

RA2.9 *The performance of NIT-based criteria for the selection of the appropriate patients in MASLD-cACLD/cirrhosis trials (with the aim of enriching the population for the desired endpoint(s) in order to improve trial feasibility) needs further evaluation.*

RA2.10 *A definition of a clinically meaningful change in NIT-based risk estimates in patients with SLD-related cACLD is required to qualify these NITs as surrogate endpoints in clinical trials of SLD-related cACLD/cirrhosis.*

Panel 3 – Prevention of Decompensation

Definition of compensated cirrhosis/cACLD

3.1. Compensated cirrhosis/cACLD is defined by the absence of current or past decompensation of cirrhosis. The transition from compensated to decompensated cirrhosis markedly increases mortality risk. (LoE 2; modified; Agreement: 99%)

3.2.

a) Compensated cirrhosis/cACLD can be divided into two stages, based on the absence or presence of CSPH. (LoE 2)

b) CSPH increases the risk of decompensation, especially in patients with gastro-oesophageal varices. (LoE 2)

c) Patients with compensated cirrhosis/cACLD should be evaluated for the presence of CSPH. (LoE 2)

d) Presence of varices and/or portosystemic collaterals are generally signs of CSPH (LoE 2), but in cases of cured/controlled aetiology they may persist despite resolution of CSPH. (LoE 3)

e) The main therapeutic goal in compensated cirrhosis/cACLD with CSPH is to prevent decompensation. (LoE 2) (modified, Agreement 97%)

3.3. The events that define first decompensation are clinically evident ascites (or hepatic hydrothorax) caused by portal hypertension, variceal bleeding and/or overt hepatic encephalopathy (HE) (West Haven grade ≥II). (LoE 2; modified; Agreement: 99%)

3.4.

a) Minimal ascites or ascites detectable only by imaging (LoE 3), cOHE (LoE 3) and occult bleeding from portal hypertensive gastroenteropathy (LoE 5) cannot be considered as decompensation.

b) Minimal ascites or ascites detectable only by imaging may be associated with decreased survival (LoE 3; modified, Agreement: 93%).

3.5. Jaundice alone (in non-cholestatic aetiologies) may be the first clinical manifestation in a minority of patients with cACLD/cirrhosis (LoE 2), however its definition, association with superimposed liver injury, and prognostic implications require further research. (LoE 4; modified, Agreement: 96%)

Determinants of the natural history of compensated cirrhosis/cACLD

3.6. Development of hepatocellular carcinoma (HCC) in patients with cirrhosis/cACLD increases the risk of decompensation and/or death. (LoE 3; new: Agreement: 99%)

3.7. Superimposed acute liver injury due to alcohol-related hepatitis, acute viral hepatitis (HEV, HAV, HBV), HBV flares, autoimmune hepatitis or drug- induced liver injury (DILI) can precipitate decompensation and/or ACLF in patients with compensated cirrhosis/cACLD. (LoE 2; modified; Agreement: 100%)

3.8. Diabetes mellitus and overweight/obesity are frequent comorbidities in patients with compensated cirrhosis/cACLD that can adversely impact prognosis and should be addressed in clinical trials and managed in clinical practice. (LoE 2; modified; Agreement: 99%)

3.9. There is inconclusive data on the impact of sarcopenia and frailty on the natural history of compensated cirrhosis/cACLD. (LoE 2; modified; Agreement: 94%)

3.10. Bacterial infections in compensated cirrhosis/cACLD patients with CSPH can precipitate decompensation and/or ACLF and, consequently, adversely affect natural history. (LoE 2; modified; Agreement: 100%)

3.11. The prognostic impact of infections in the natural history of compensated cirrhosis/cACLD without CSPH is unclear. (LoE 5; modified; Agreement: 96%)

3.12. Periodontal disease is associated with a higher risk of decompensation in compensated cirrhosis/cACLD (LoE 4; new; Agreement: 87%)

3.13. Surgery, especially major surgery, can precipitate decompensation and/or ACLF in patients with compensated cirrhosis/cACLD and CSPH, with an increasing risk at greater severity of portal hypertension. (LoE 3; new; Agreement: 100%)

Management and therapy

3.14.

a) Successful aetiological treatment can improve portal hypertension and reduce the incidence of decompensation. (LoE 2; new)

b) After aetiological therapy, CSPH can persist and should be reassessed. (LoE 3; SoR: strong; new; Agreement: 99%)

3.15. Treatment with NSBBs, particularly carvedilol, is recommended in patients with compensated cirrhosis/cACLD and CSPH to prevent decompensation and improve survival (LoE 2; SoR: strong; modified; Agreement: 97%)

3.16. In patients with compensated cirrhosis/cACLD without CSPH, NSBB should not be used for the prevention of decompensation/death. (LoE 2; SoR: strong; modified; Agreement: 99%)

3.17. In patients with compensated cirrhosis/cACLD with CSPH and HCC, prevention of decompensation is a clinically relevant endpoint across all stages of the oncological disease, independent of tumour-directed therapy. (LoE 3; new; Agreement: 100%)

3.18. Patients with compensated cirrhosis/cACLD who are on NSBBs do not require screening endoscopy for variceal detection as the results would not impact clinical management. (LoE 2; modified; Agreement: 92%)

3.19. In patients with compensated cirrhosis/cACLD with contraindications or intolerance to NSBB, endoscopy is recommended and if large varices (O V and GOV1) are detected, endoscopic variceal ligation (EVL) is recommended to prevent first variceal bleeding. (LoE 1; SoR: strong; modified). Endoscopic therapy on its own has no effect on prevention of non-bleeding decompensating events. (LoE 1; modified; Agreement: 100%)

3.20.

a) In compensated cirrhosis/cACLD patients with CSPH, TIPS is not recommended for the prevention of decompensation. (LoE 5; SoR: strong)

b) Preoperative TIPS may be considered prior to major surgery to reduce the risk of postoperative decompensation. (LoE 4; SoR: weak; modified; Agreement: 97%)

3.21.

a) Patients with compensated cirrhosis/cACLD with gastric varices have CSPH and benefit from NSBBs, particularly carvedilol, for the prevention of decompensation. (LoE 2; modified; Agreement: 96%)

b) In patients with high risk GOV2, IGV1 (cardiofundal) varices who have contraindication or intolerance to NSBBs, local therapy [i.e. endoscopic injection therapy (LoE 2; SoR: strong), endosonography-guided therapy (LoE 4; SoR: weak) or transvenous obliteration (LoE 3; SoR: weak)] can be

considered to prevent bleeding in centers with expertise. (modified; Agreement: 95%)

c) In patients with compensated cirrhosis/cACLD and GOV2, IGV1 (cardiofundal) varices, there is limited evidence to support the use of endoscopic injection therapy (LoE 3), endosonography-guided therapy (LoE 5) or transvenous obliteration (LoE 5)] in addition to NSBBs to prevent first variceal bleeding and improve survival. (modified; Agreement: 96%)

3.22. There is insufficient evidence to recommend statins or anticoagulation to prevent decompensation (LoE 3) but they should be used or maintained if prescribed for approved indications. (LoE 2; SoR: strong; modified: Agreement: 99%)

3.23. Regular dental and periodontal care may decrease the risk of decompensation in compensated cirrhosis/cACLD. (LoE 3; SoR: weak; new; Agreement: 81%)

Panel 3 – Research Agenda

Definition

RA3.1. *Definition and prognostic impact of jaundice alone as first decompensating event*

RA3.2. *Definition of HE and its differential diagnosis as first decompensating event*

RA3.3. *Determine the prognostic significance of minimal ascites (only detected on imaging), minimal hepatic encephalopathy, or chronic bleeding from PHG in patients with cACLD*

RA3.4. *Evaluation of the impact of different types of first decompensating events and their acuity on mortality*

Determinants of natural history

RA3.5. *Further characterization of the interaction between HCC and portal hypertension on the natural history of cACLD.*

RA3.6. *Evaluation of the impact of comorbidities, including their early detection and treatment, on the risk of liver-related events and on competing non–liver-related mortality (e.g., cardiovascular disease, chronic kidney disease, chronic obstructive pulmonary disease (COPD), non-hepatic malignancies) in patients with cACLD.*

RA3.7. *Evaluation of the impact of multimorbidity in the management of patients with compensated cirrhosis*

RA3.8. *Evaluation of PVT as a precipitant of first decompensation.*

RA3.9. *Determine the impact of sarcopenia and frailty (and of their treatment) on the natural history of cACLD*

RA3.10. *Evaluate the incidence and prognosis of (bacterial) infections in patients with cACLD with and without CSPH.*

RA3.11. *Evaluate whether prevention of infections (i.e., vaccination, oral/dental health) has an impact on the incidence of decompensation.*

RA3.12. *Evaluation of the impact of health measures and dedicated care bundles (including management of sarcopenia, nutritional counseling, physical activity, extrahepatic cancer screening, bone health assessment, vaccination, oral/dental health assessment and counseling on concomitant medication including potential hepatotoxic drugs) on the natural history of compensated cirrhosis/cACLD.*

Management and therapy

RA3.13. *Evaluation of the prevention of decompensation with NSBB in patients with CSPH without varices.*

RA3.14. *Evaluate the role of statins in preventing decompensation.*

RA3.15. *Evaluation of the use of local therapies (endoscopic, endosonographic, or transvenous obliteration), in addition to NSBB, to prevent first gastric variceal bleeding.*

RA3.16. *Evaluation of the role of pre-surgical TIPS to prevent post-surgical decompensation in patients with cACLD*

RA3.17. *Evaluation of the other therapeutic targets (incretin-based drugs, FGF21 analogues, anticoagulation/antiplatelet agents, antifibrotics like galectin-3 inhibitors (e.g. belapectin) or prolonged-release pirfenidone) to prevent decompensation.*

Panel 4 – PREVENTION OF FURTHER DECOMPENSATION

4.1. In patients with decompensated cirrhosis, further decompensation (FD) represents a more advanced disease stage with a higher mortality (LoE 2; modified; Agreement: 86%) and is defined as follows:

(a) In patients with a single decompensation:

- *If ascites*, by the need for ≥ 3 LVP within 1 year, the occurrence of SBP, HRS-AKI, or a second decompensating event (CSPH-related bleeding, OHE, or jaundice);
- *If variceal bleeding*, by the development of CSPH-related re-bleeding or a second decompensating event (ascites, OHE, or jaundice);
- *If OHE, by* the development of recurrent OHE or a second decompensating event (ascites, bleeding, or jaundice);

(b) In patients with ≥2 decompensating events, by any combination of ascites, PH-related bleeding, OHE and/or jaundice.

4.2. After the first decompensating event, an aetiological treatment is recommended to prevent FD. (LoE 2; SoR: strong; new; Agreement: 100%)

4.3. Patients with decompensated cirrhosis should be considered for liver transplantation (LT). (LoE 1 ; SoR: strong; unchanged; Agreement: 98%)

4.4. In patients with ascites and varices, carvedilol/cNSBB should be used to prevent first bleeding (LoE 1; SoR: strong; new), and to lower the risk of ascites-related

complications (LoE 3; SoR: strong; new), with carvedilol being preferred over cNSBB. (LoE 3; SoR: strong; new; Agreement: 81%)

4.5. In patients with ascites and varices, ineligible for carvedilol/cNSBB (intolerance or contraindication), endoscopic therapy alone should be considered to prevent first variceal bleeding. (LoE 3; SoR: strong; modified; Agreement: 92%)

4.6. Carvedilol/cNSBB should be dose-reduced or discontinued in case of persistently low blood pressure (systolic arterial pressure < 90 mmHg or mean arterial pressure < 65 mmHg) and or HRS-AKI. (LoE 2; SoR: modified; Agreement: 95%)

4.7. Carvedilol/cNSBBs can be re-initiated and/or re-titrated once HRS-AKI has resolved and/or blood pressure has recovered. (LoE:2; SoR: strong; Agreement: 95%)

4.8. In patients with a history of bleeding from OV as the first decompensating event, the first line therapy for prevention of recurrent bleeding is the combination of NSBB+EVL (LoE 1; SoR: strong), with carvedilol being preferred over cNSBB (LoE 2; SoR: strong), independently of prior primary prophylaxis with carvedilol/cNSBB (modified; Agreement: 85%)

4.9. In patients recovering from an episode of acute bleeding from OV as a single decompensating event (ineligible for rescue or pre-emptive TIPS [p-TIPS]) who cannot be treated with either EVL or carvedilol/cNSBB, the tolerated therapy should be started/maintained as monotherapy. (LoE 1; SoR: strong; modified; Agreement: 87%)

4.10. In patients recovering from an episode of acute bleeding from OV (ineligible for rescue or p-TIPS) who also experienced a non-bleeding decompensating event, TIPS and LT may be considered. (LoE 3; SoR: weak; modified; Agreement: 92%)

4.11. In patients with decompensated cirrhosis, statins are not recommended to prevent FD, but they should be used or continued if prescribed for approved indications, taking into account the higher risk of statin toxicity in Child-Pugh C patients. (LoE 2; SoR: strong; modified; Agreement: 82%)

4.12. In patients after the first decompensating event, long-term primary antibiotic prophylaxis is not recommended to prevent FD. (LoE 2; SoR: strong; new; Agreement: 99%)

4.13. TIPS should be considered in patients with PH-bleeding despite secondary prophylaxis with carvedilol/cNSBB+EVL (failure of secondary prophylaxis). (LoE 3; SoR: strong; modified; Agreement: 92%)

4.14. The prevention of FD in patients with HCC should be managed in the same way as in patients without HCC, while considering life expectancy and treatment side effects. (LoE 3; SoR: strong; new; Agreement: 95%)

4.15. The presence of HCC does not represent an absolute contraindication to TIPS in patients with recurrent/refractory ascites or hepatic hydrothorax, unless the tumour lies along the TIPS trajectory. An improved control of hepatic decompensation with TIPS may facilitate access to effective oncological treatment. (LoE 3; SoR: weak; new; Agreement 90%)

4.16. Frailty, malnutrition, sarcopenia and myosteatosis have an impact on survival in patients with decompensated cirrhosis or prior history of decompensation (LoE 2; modified). These conditions should be evaluated using available standardised tools. (see Supplementary Table-S1) (LoE 3; SoR: strong; unchanged; Agreement: 90%)

4.17. All patients with decompensated cirrhosis should undergo a structured nutritional assessment, including an individualis ed nutrition plan and counselling on regular physical activity (LoE 2; SoR: strong; modified; Agreement: 94%)

4.18. In patients identified as frail and/or sarcopenic, targeted interventions should be encouraged in addition to standard nutritional and physical activity counselling, as muscle health may improve and lead to improved clinical outcomes (LoE 2; SoR: strong; new), but further evaluation is required regarding their impact on the natural history of further decompensation. (LoE 3; SoR: weak; new; Agreement: 92%)

4.19. Sarcopenia and frailty can improve after TIPS but can also be associated with poor outcomes. Sarcopenia and/or frailty alone should neither indicate nor contraindicate TIPS but should be considered together with other factors in the pre-TIPS work-up. (LoE 3; SoR: strong; modified; Agreement: 91%)

Panel 4 – Research Agenda

RA4.1. *Validate the prognostic weight of any decompensating event occurring within 5 days after acute variceal bleeding*

RA4.2. *Assess the prognostic impact of Grade 1 (mild) ascites as the first decompensating event*

RA4.3. *Investigate the effect of time to FD on prognosis in patients with ascites/hepatic encephalopathy as first decompensation.*

RA4.4. *Determine the role of inflammation biomarkers on predicting the risk of FD*

RA4.5. *Integrate the prognostic concept of FD into clinical decision algorithms to optimize patient selection for more advanced treatments (e.g., TIPS or other interventional radiology, LT)*

RA4.6. *Analyze the positioning of the definition of FD and possible overlap with the concepts and definitions of non-acute, acute decompensation and ACLF.*

RA4.7. *Investigate whether prevention of FD in patients with ascites should be taiSoR ed according to the aetiology of cirrhosis and comorbidities (i.e. patients with MASLD and/or diabetes)*

RA4.8. *Determine how the type and burden of hepatic decompensation at the time of aetiological therapy influences subsequent risk of progression to FD.*

RA4.9. *Identify a priori patients in whom FD cannot be prevented despite effective aetiological therapy.*

RA4.10. *Assess whether carvedilol/cNSSB should be started in patients with ascites as first decompensation regardless of the presence of varices.*

RA4.11. *Investigate the effectiveness of EVL in addition to carvedilol/NSBB for primary prophylaxis of variceal bleeding in patients with a history of first non-bleeding event*

RA4.12. *Confirm the subgroup of patients with ascites who benefit from long-term albumin administration, investigate the optimal baseline serum albumin concentration to initiate long-term albumin administration and monitor patients for continued benefit vs. futility of this treatment*

RA4.13. *RCT comparing efficacy, tolerance and cost between long term albumin administration and TIPS in patients who are candidates for both treatments.*

RA4.14. *Prospective studies should assess if statins, metformin, or GLP-1 receptor agonists prevent FD in certain populations of patients with decompensated cirrhosis.*

RA4.15. *Primary prophylaxis of SBP in high-risk patients requires confirmatory data that carefully balance efficacy against the risk of multidrug-resistant organism infections.*

RA4.16. *Evaluate whether elective TIPS placement can improve outcomes in patients who do not meet criteria for p-TIPS at the time of bleeding but who are nonetheless identified as being at increased risk of FD and death*

RA4.17. *Explore whether elective TIPS placement can improve outcomes in patients who are eligible for p-TIPS but did not receive p-TIPS during the 72h time window or in patients with a first episode of tense ascites*

RA4.18. *Investigate the effects of TIPS on quality of life and on access to HCC treatment according to BCLC stage*

RA4.19. *Assess the additive value of body composition parameters in addition to Child-Pugh/MELD scores*

RA4.20. *Evaluate the impact of sarcopenia and/or frailty and its management on FD*

RA4.21. *Prospective studies regarding the impact of sarcopenia and/or frailty on post-TIPS outcomes.*

RA4.22. *Explore the impact of anticoagulation (type, dose and target population) to prevent further decompensation*

RA4.23. *Investigate the effect of carvedilol/NSBB to prevent FD in patients with ascites and PHG*

Panel 5 – MANAGEMENT OF PORTAL HYPERTENSIVE BLEEDING

5.1. Acute variceal bleeding (AVB) is defined by bleeding from oesophageal, gastric or ectopic varices (blood emanating from a varix: spurting or oozing), or by varices with signs of recent bleeding (clots, white nipple sign), or by varices without other sources of bleeding in the presence of blood in the stomach in a patient with upper gastrointestinal bleeding. (LoE 4; modified; Agreement: 85%)

5.2. Active bleeding at endoscopy is defined as blood coming from a varix (spurting, oozing). However, interobserver agreement for the diagnosis of active bleeding is moderate. (LoE 3; modified; Agreement: 87%)

5.3. The goal of resuscitation is to preserve organ perfusion. Volume restitution should aim to restore and maintain haemodynamic stability. (LoE 5, SoR: strong; modified; Agreement: 97%)

5.4. Restrictive transfusion of packed red blood cell transfusions should be performed with a target haemoglobin level between 7-8 g/dL. However, individualized transfusion strategies should be considered based on the presence of cardiovascular disorders, age, haemodynamic status, and ongoing bleeding. (LoE 2, SoR: strong; modified; Agreement: 92%)

5.5. Intubation is recommended before endoscopy in patients with altered consciousness or active haematemesis (LoE 5, SoR: strong; modified; Agreement: 97%)

5.6. Extubation should be performed as quickly as possible after endoscopy. (LoE 5; SoR: strong; unchanged; Agreement: 96%)

5.7. In suspected variceal bleeding, vasoactive drugs (terlipressin, somatostatin, octreotide) should be started as soon as possible and continued for 2-5 days or until a TIPS (if indicated) is placed. (LoE 1, SoR: 1, SoR: strong; modified, Agreement: 82%)

5.8. A shorter course (even 24 hours) of vasoactive drugs can be considered following successful endoscopic haemostasis, provided NSBB treatment is then initiated. (LoE 2, SoR: strong; modified, Agreement: 82%)

5.9. Sodium levels should be monitored for patients on terlipressin due to risk of hyponatraemia. (LoE 2; SoR: strong; modified; Agreement: 97%)

5.10. Preventive antibiotic therapy is recommended at presentation with AVB (LoE 1; SoR: strong). Duration and need could be individualized based on infection risk

and cirrhosis severity, as Child–Pugh A patients may only require a shorter course or no therapy. (LoE 2; SoR: weak; modified; Agreement: 85%)

5.11. In patients with cirrhosis experiencing AVB 3rd generation cephalosporins are generally preferred for preventive antibiotic therapy; however, local resistance patterns and antimicrobial policies should be considered. (LoE2; SoR: strong; modified; Agreement: 94%)

5.12. Malnutrition increases the risk of adverse outcomes in patients with cirrhosis and AVB. Therefore, oral nutrition should be started as soon as possible. (LoE 5; SoR: strong; modified; Agreement: 93%)

5.13. Airway manipulation and placement of a nasogastric tube should be performed with caution because of the risk of aspiration and pulmonary infection. (LoE 5; SoR: strong; modified; Agreement: 91%)

5.14. Proton pump inhibitors (PPIs), when started before endoscopy, should be discontinued after portal hypertension-related bleeding has been confirmed, unless there is a strong indication to continue them. (LoE 5; SoR: strong; modified; Agreement: 91%)

5.15. Six-week mortality is the recommended primary endpoint for studies on the treatment of AVB. (LoE 5; SoR: strong; modified; Agreement: 97%)

5.16. Treatment failure is defined either by uncontrolled bleeding or by rebleeding within 5 days. (LoE 5; SoR: strong; modified; Agreement: 94%)

5.17. Child-Pugh class C, MELD and variants (e.g. MELD-Na, MELD 3.0) (and its variants), and failure to control bleeding represent key risk factors for 6-week mortality. (LoE 1; modified; Agreement: 87%)

5.18. Following haemodynamic resuscitation, patients with suspected AVB should undergo upper endoscopy within 12 h of presentation (LoE 2, strong recommendation). If the patient is haemodynamically unstable, endoscopy should be performed as soon as possible depending on local availability. (LoE 5; SoR: strong; modified; Agreement: 93%)

5.19. In centers with a hepatology service, 24/7 availability of an on-call GI endoscopist and assistant staff proficient in endoscopic treatment of variceal/GI bleeding is recommended. (LoE 5; SoR: strong; modified; Agreement: 94%)

5.20. In patients with suspected AVB and no contraindications (QT prolongation), a single intravenous dose of erythromycin (250 mg, 30–90 minutes before endoscopy) may improve gastric clearance, enhance endoscopic visualization

and result in a reduced blood transfusion requirement. (LoE 2; SoR: weak; new; Agreement: 94%)

5.21. Patients with AVB should be managed in intensive or intermediate care units. (LoE 5; SoR: strong; unchanged; Agreement: 94%)

5.22. In patients presenting with AVB, lactulose is recommended (orally or by enemas) to prevent or to treat OHE by accelerating blood elimination from the gastro-intestinal tract. (LoE 2; SoR: strong; modified; Agreement: 91%)

5.23. In patients with cirrhosis and AVB, transfusion of fresh frozen plasma is not routinely recommended, as it does not correct coagulopathy and may lead to volume overload and worsening of portal hypertension. (LoE 4; SoR: strong; modified; Agreement: 96%)

5.24. In patients with cirrhosis and AVB, platelet count and fibrinogen levels do not correlate with the risk of failure to control bleeding or rebleeding and their transfusion is not routinely recommended. However, in case of failure to control bleeding, transfusion of platelets or fibrinogen supplementation may be considered. (LoE 5; SoR: weak; modified; Agreement: 90%)

5.25. The use of recombinant factor VIIa and tranexamic acid is not recommended for AVB. (LoE 2; SoR: strong; modified; Agreement: 94%)

5.26. In patients with AVB, anticoagulation should be temporarily discontinued until bleeding is controlled. Timing of re-initiation should be individualized based on the strength of the indication for anticoagulation. (LoE 5; SoR: strong; modified; Agreement: 96%)

5.27. In patients recovering from AVB, iron supplementation should be considered in those with blood loss anaemia. (LoE 3; SoR: strong; new; Agreement: 87%)

5.28. The use of haemoclips, over-the-scope-clips, topical haemostatic agents and fibrin is not recommended as first-line endoscopic therapy for AVB. (LoE 4; SoR: strong; modified; Agreement: 97%)

5.29. In AVB, immediate haemostasis with EVL is recommended for AVB from oesophageal varices and type 1 gastro-oesophageal varices (GOV-1) (LoE 1; SoR: strong; modified; Agreement: 95.6%)

5.30. All patients with AVB should undergo abdominal imaging, preferably contrast-enhanced cross-sectional imaging (CT or MRI) to exclude portal/splanchnic vein thrombosis and HCC, and to map portosystemic collaterals in order to guide treatment. (LoE 5; SoR: strong; modified; Agreement: 87%)

5.31. Pre-emptive TIPS (p-TIPS) with polytetrafluoroethylene (PTFE)-covered stents within 72 h (ideally <24 h) is indicated in patients bleeding from oesophageal varices and type 1 gastric varices (GOV-1) who meet any of the following criteria: Child-Pugh class C 10-13 points or Child-Pugh class B >7 with active bleeding at initial endoscopy or HVPG ≥20 mmHg at the time of AVB. (LoE 1; SoR: strong; modified; Agreement: 93%)

5.32. In patients fulfilling the p-TIPS criteria who did not undergo TIPS within the preferred 72h window, TIPS placement within 2 weeks in patients with Child-Pugh class C 10-13 or within 1 week in patients with Child-Pugh class B >7 with active bleeding may still provide benefit. (LoE 4; SoR: weak; new Agreement: 88.2%)

5.33. In patients fulfilling pTIPS criteria, ACLF, OHE, hyperbilirubinaemia, MELD score, or severe alcohol-related hepatitis should not be considered absolute contraindications. (LoE 3; SoR: strong; new, Agreement: 94%)

5.34. Patients with OHE refractory to medical and interventional therapy after TIPS may be prioritised for liver transplantation. (LoE 4; SoR: weak; new, Agreement: 85%)

5.35. In refractory oesophageal variceal bleeding, a dedicated covered self-expanding metal stent (cSEMS) is preferred over balloon tamponade, as bridge therapy to TIPS or liver transplantation. (LoE 4; SoR: strong; modified; Agreement: 91%)

5.36. Failure to control variceal bleeding despite combined pharmacological and endoscopic therapy is best managed by salvage TIPS. (LoE 2; SoR: strong; Agreement: 97%)

5.37. Salvage TIPS should be discussed for any refractory variceal bleeding, regardless of age, the Child-Pugh and MELD scores, on a case-by-case basis. (LoE3; SoR: strong; modified; Agreement: 96%)

5.38. The Sarin classification is recommended for gastric varices, distinguishing gastroesophageal varices type 1/2 (GOV1/2) and isolated gastric varices type 1/2 (IGV1/2) in order to harmonise reporting, stratify bleeding risk, and inform therapeutic strategy. (LoE 3; SoR: strong; new, Agreement: 97%)

5.39. AVB from GOV-2 or IGV1 is a high-risk event that requires rapid, structured multidisciplinary management. Treatment is bimodal: an acute phase focused on immediate hemostasis and a definitive phase, pursuing durable bleeding control and prevention of further decompensation. Both phases may start in

parallel; if staged - the interval between them should be kept as short as possible. (LoE 5; SoR: strong; new; Agreement: 87%)

5.40. In patients with AVB from GOV2 or IGV1, immediate hemostasis can be achieved with either conventional endoscopic cyanoacrylate injection or EUS-guided therapy, as both appear similarly effective in controlling bleeding and preventing early rebleeding. When available, EUS-guided coil plus cyanoacrylate may be recommended in experienced centre s because it may achieve higher variceal obliteration rates, reduce late rebleeding and need for reintervention. (LoE 2; SoR: weak; new, Agreement: 81%)

5.41. TIPS ± variceal embolisation performed as soon as possible is the preferred definitive treatment option in patients with AVB from GOV2/IGV1. When TIPS is contraindicated, other definitive strategies, including transvenous obliteration, endoscopic therapies (including EUS-guided approaches), or surgical methods—may be utilised depending on available expertise and after multidisciplinary discussion, based on the anatomy and location of the bleeding site. (LoE 2; SoR: strong; new; Agreement: 81%)

5.42. Either endovascular (TIPS or transvenous obliteration) or endoscopic treatment should be considered in patients with ectopic varices. (LoE 4; SoR: strong; modified, Agreement. 91%)

5.43. In patients with gastric antral vascular ectasia (GAVE) requiring serial transfusion, EVL is recommended over argon plasma coagulation, as it is associated with higher eradication rates and fewer treatment sessions, with reductions in recurrent bleeding, hospitalisations, and transfusion requirements. (LoE 1; SoR: strong; modified; Agreement: 90%)

5.44. Management of portal-hypertensive gastropathy focuses primarily on reducing portal pressure with NSBB. In cases of severe or refractory bleeding from PHG, TIPS is the most effective rescue therapy. Since endoscopic therapies do not improve portal hypertension, they provide limited benefit and are reserved for focal bleeding lesions or salvage situations. (LoE 4; SoR: strong; modified; Agreement: 91%)

5.45. In patients with cirrhosis and portal vein thrombosis (PVT), management of AVB should generally follow the recommendations for patients without PVT. In selected patients, TIPS ± portal vein recanalisation may be considered when additional factors related to portal hypertension are present (e.g., ascites or

documented progression of PVT) or LT candidacy. (LoE 5; SoR: strong; modified; Agreement. 85%)

5.46. The presence of HCC alone may not preclude consideration of all potential treatments in the setting of AVB. Treatment decisions should be individualis ed within a multidisciplinary framework and should account for futility criteria related to both liver function and tumour prognosis. (LoE 5; SoR: weak; new; Agreement: 94%)

5.47. Post-EVL ulcer bleeding is a rare but serious complication, the management of which should be individualised and may require vasoactive therapy, endoscopic hemostasis, temporary stenting or tamponade, and rescue TIPS with embolisation when needed—particularly in high-risk patients (MELD >18 or emergency EVL). (LoE 1; SoR: strong, new; Agreement: 88%)

5.48. Current evidence is insufficient to recommend the routine use of PPIs for the prevention of (bleeding from) post-EVL ulcers. (LoE 2; SoR: weak; new; Agreement: 91%)

Panel 5 – Research Agenda

RA5.1. *Evaluation of interobserver agreement for AVB*

RA5.2. *RCT comparing preventive antibiotic therapy vs no antibiotics in Child-Pugh A patients and AVB*

RA5.3. *Validation of short course (under 48hrs) of vasoactive therapy (including type of drug) in Child-Pugh A and B7 patients with AVB*

RA5.4. *Compare continuous vasoactive infusion with bolus administration in terms of AVB control and associated adverse effects.*

RA5.5. *Assess the efficacy and safety of tranexamic acid in the management of AVB.*

RA5.6. *Validation of high-risk criteria for p-TIPS including CLIF-C AD and the Chinese Collaboration Group AVB*

RA5.7. *Prospectively evaluate the impact of a 1- or 2-week delay to pTIPS vs. NSBB+EVL in patients with CTP-C or CTP-B≥8 + active bleeding.*

RA5.8. *Evaluate the impact of portal vein thrombosis on outcomes of pre-emptive TIPS in patients with cirrhosis and AVB*

RA5.9. *Assess whether pre-emptive TIPS confers a survival benefit in patients with AVB and HCC beyond Milan criteria*

RA5.10. *Investigate the role of pre-emptive TIPS in patients with AVB aged > 75 years, incorporating frailty assessment as a stratification variable*

RA5.11. *Rifaximin as primary prophylaxis of OHE before pTIPS*

RA5.12. *Investigate the effectiveness of underdilated/small pTIPS in AVB*

RA5.13. *Build a new futility score for rescue TIPS with external validation*

RA5.14. *TIPS in bleeding from PHG*

RA5.15. *RCT between TIPS/transvenous obliteration and EUS-guided therapies for the definitive (long-term) management of gastric varices (GOV2/IGV1)*

RA5.16. *Prevention and management of bleeding from post-EVL ulcers*

Panel 6 – MANAGEMENT OF FURTHER DECOMPENSATION

6.1. Patients with further decompensation (FD) should be evaluated for liver transplantation (LT) (LoE: 1, SoR: strong; new; Agreement: 97%)

6.2. In patients with FD, aetiological cure/control improves survival and should be pursued (LoE: 2; SoR: strong; new; Agreement: 96%)

6.3. Patients with recurrent or refractory ascites should be considered for TIPS. (LoE: 1; SoR: strong; modified; Agreement: 97%)

6.4. In patients with recurrent or refractory ascites/hydrothorax, TIPS and LT should be discussed together. (LoE: 2; SoR: strong; new;Agreement: 91%)

6.5. In patients with recurrent/refractory ascites, to reduce the risk of post-TIPS overshunting (HE, cardiac failure) while preserving efficacy, the stent may be dilated to the smallest diameter that achieves an adequate response (LoE: 3; SoR: weak; new; Agreement: 97%)

6.6. Stepwise dilation should be considered in patients with no improvement in ascites (LoE: 3; SoR: strong; new; Agreement: 97%)

6.7. In patients with hepatic hydrothorax refractory to standard medical treatment, TIPS should be considered (LoE: 3; SoR: strong; new; Agreement: 90%)

6.8. TIPS may be considered for the management of recurrent/refractory ascites or refractory hepatic hydrothorax in selected older adults (≥70 years) on a case-by-case basis and taking into account the benefit/risk (LoE: 3; SoR: weak; new; Agreement: 90%)

6.9. In patients undergoing evaluation for TIPS for recurrent/refractory ascites, renal function should be systematically assessed, and CKD staged (eGFR) for risk stratification. (LoE: 2; SoR: strong; new; Agreement: 91%)

6.10. In patients with recurrent/refractory ascites who are not TIPS candidates, long-term albumin treatment may be considered as it can improve ascites control and reduce the incidence of ascites-related complications (LoE: 3; SoR: weak; new;Agreement: 83%)

6.11. In patients with refractory ascites who have contraindications or an inadequate response to TIPS and are not candidates for liver transplantation, home-based drainage devices (tunnelled peritoneal catheter or low-flow ascites pump) may be considered as an alternative to repeated large-volume paracentesis (LoE 3; SoR: weak; new; Agreement: 93%)

6.12. In patients with recurrent/persistent HE despite optimal pharmacological therapy, embolisation of large portosystemic shunts (PSS) should be considered, in addition to LT discussion, particularly when liver function is preserved (LoE 3; SoR: strong; new; Agreement: 91%)

6.13. In patients with recurrent/persistent HE treatment with L-ornithine-L-aspartate (LOLA) can be considered (LoE: 3; SoR: weak; new; Agreement: 80%)

6.14. Before elective TIPS placement, history of OHE and use of HE medications should be carefully assessed (LoE: 2; SoR: strong; new; Agreement: 96%)

6.15. A history of prior OHE is not an absolute contraindication for elective TIPS placement, and the decision should be individualised based on the patient's clinical context (LoE: 2; SoR: strong; new; Agreement: 95%)

6.16. Rifaximin should be considered for prophylaxis of HE in patients with cirrhosis who are candidates for elective TIPS placement (LoE: 2; SoR: strong; modified; Agreement: 85%)

6.17. In patients with cirrhosis who develop recurrent or persistent HE after TIPS despite removal of precipitants and standard medical treatment, spontaneous shunt embolization and/or reduction or occlusion of the TIPS could be considered (LoE: 3; SoR: weak; new; Agreement: 87%)

6.18. In patients with HRS-AKI, treatment with terlipressin and albumin should be considered (LoE: 1; SoR: strong; new; Agreement: 93%)

6.19. Terlipressin should preferably be administered as continuous intravenous infusion (starting dose 2-3mg/24hours) to reduce the incidence of adverse events (LoE: 2; SoR: strong; new; Agreement: 93%)

6.20. Norepinephrine can be used as an alternative to terlipressin, if terlipressin is unavailable, contraindicated or not tolerated. (LoE: 1; SoR: strong; new; Agreement: 86%)

6.21. In patients with cirrhosis and a history of SBP, antibiotic prophylaxis should be considered to prevent recurrence of SBP. (LoE: 2; SoR: strong; modified; Agreement: 93%)

6.22. Antibiotic SBP prophylaxis should be discontinued in case of recompensation. (LoE: 4; SoR: strong; new; Agreement: 99%)

6.23. Data are insufficient to recommend rifaximin for secondary prophylaxis of SBP (LoE: 4; new; Agreement: 94%)

6.24. In patients with FD, withdrawal of PPIs is recommended unless there is a clear indication. (LoE: 2; SoR: strong; new; Agreement: 93%)

6.25. In patients with jaundice as the sole manifestation of FD, an immediate and exhaustive search for precipitating events, specifically bacterial infections (even if asymptomatic), alcohol-related hepatitis, flares of viral or autoimmune hepatitis, DILI or biliary obstruction, should be performed. (LoE: 3; SoR: strong; new; Agreement: 86%)

Panel 6 – Research Agenda – Management of Further Decompensation

RA6.1. *Define trajectories of FD according to the type and number of first FD event(s)*

RA6.2. *Define trajectories of FD according to the acute/non-acute presentation,*

RA6.3. *Define trajectories of further decompensation according to cause of chronic liver disease and the possibility of eliminating/controlling the etiological factor*

RA6.4. *Identify the optimal haemodynamic response for patients with recurrent/refractory ascites undergoing TIPS placement*

RA6.5. *Define how to assess haemodynamic response*

RA6.6. *Develop a holistic approach to TIPS in patients with recurrent or refractory ascites, integrating splanchnic and systemic hemodynamic data, sarcopenia and frailty, systemic and local inflammatory status, gut microbiota, and comorbidities.*

RA6.7. *Assess, in an RCT, the efficacy of small diameter TIPS in recurrent/refractory ascites and hydrothorax*

RA6.8. *Evaluate the role of small-diameter TIPS in older adult populations and identify predictors of outcomes.*

RA6.9. *Evaluate the role of prehabilitation and post-TIPS rehabilitation programs in patients undergoing elective TIPS*

RA6.10. *Evaluate strategies to prevent congestive heart failure after TIPS*

RA6.11. *Assess the role of fecal microbiota transplant (FMT) in the prevention of HE*

RA6.12. *Assess the role of FMT in the secondary prevention of HE (phase 3 trial)*

RA6.13. *Assess the role of rifaximin prophylaxis at the time of preemptive/salvage/rescue TIPS placement in the prevention of HE*

RA6.14. *Identify the optimal timing for discontinuation of rifaximin after TIPS*

RA6.15. *Evaluate the potential role of rifaximin in promoting the emergence of multidrug-resistant organism (MDRO) colonization.*

RA6.16. *Assess the use of FMT in patients with recurrent/refractory HE*

RA6.17. *Assess the titration of terlipressin based on mean arterial pressure*

RA6.18. *Evaluate the use of point of care ultrasonography to assess intravascular volume and guide albumin treatment in HRS-AKI*

RA6.19. *Compare the use of balanced crystalloids vs albumin in HRS-AKI,*

RA6.20. *Explore the role of urinary biomarker- guided management of HRS-AKI*

RA6.21. *Define the role of TIPS in the treatment of HRS-AKI*

RA6.22. *Identify strategies of non-antibiotic prophylaxis for SBP*

RA6.23. *Develop stopping rules for secondary prophylaxis of SBP beyond recompensation*

RA6.24. *Define the optimal timing for TIPS placement after an episode of SBP in patients with recurrent or refractory ascites.*

Panel 7 – CIRRHOSIS RECOMPENSATION

7.1. Recompensation defines the stage of cirrhosis that has transitioned from a decompensated stage to a compensated stage after aetiologic therapy. Therefore, the term only applies to patients with cirrhosis who have developed overt decompensating events. (LoE 5; new; Agreement: 96.1%)

7.2. Recompensation should not be confused with cirrhosis regression, which denotes histological regression from a cirrhotic to a non-cirrhotic liver architecture. (LoE 5; new; Agreement: 96%)

7.3. Recompensation is associated with significantly lower risk of death and HCC when compared to patients who remain decompensated. (LoE 1; new; Agreement: 93.4%)

7.4. The definition of recompensation requires fulfilling ALL of the following criteria:

a. Removal, control or suppression of the primary aetiology/aetiologies of cirrhosis. (LoE 2, modified; Agreement: 97%)

b. Resolution of ascites on imaging in the absence of diuretics (unless indicated for non-ascites-related conditions) and no clinical evidence of HE (off lactulose, rifaximin, LOLA; unless indicated for non-HE-related conditions). (LoE 2; modified; Agreement: 93%)

c. After variceal bleeding as a decompensating event, absence of recurrent variceal bleeding, regardless of ongoing therapy with carvedilol/cNSBB. (LoE 2; modified; Agreement: 94.7%)

d. Criteria (a-c) must be fulfilled for > 6 consecutive months to define a state of recompensated cirrhosis (LoE 4; new; Agreement: 88.2%)

e. Improvement in liver function tests, to the range consistent with a Child-Turcotte Pugh (CTP) A5/A6 score (LoE 2; modified; Agreement: 88%).

7.5. Evidence of recompensation and definitions for aetiologic cure/control exist for the following liver disease etiologies:

a. Alcohol-related liver disease: (ALD): sustained alcohol abstinence. (LoE 1, new; Agreement: 94%)

b. Hepatitis B virus (HBV) infection: sustained suppression of HBV-DNA. (LoE 1, new; Agreement: 100%)

c. Hepatitis C virus (HCV) infection: sustained virological response (SVR). (LoE 1, new; Agreement: 100%)

7.6. Resolution of ascites while on diuretics is not considered evidence of recompensation. (LoE 5, modified). Withdrawal of diuretics should be considered in patients who achieve aetiologic cure/control, once ascites has resolved and liver synthetic function has improved to CTP A laboratory criteria. (LoE 5; SoR: strong; new) (Agreement: 88%)

7.7. Absence of HE while on specific HE medication (lactulose/rifaximin/LOLA) is not considered evidence of recompensation. (LoE 5, modified). Withdrawal of HE medication should be considered in patients who achieve aetiologic cure/control once HE has resolved and liver synthetic function has improved to CTP A laboratory criteria. (LoE 5; SoR: strong) (Agreement: 89%)

7.8. Patients with TIPS may also achieve recompensation, provided all criteria listed in statement 7.4 are fulfilled. However, resolution of ascites *alone* or lack of recurrent variceal bleeding *alone* after TIPS are not considered evidence of recompensation (LoE 4; modified: Agreement 87%)

7.9. Severity of liver disease at the time of decompensation is a negative predictor of recompensation, with higher CTP and MELD scores and/or the presence of further decompensation indicating a lower likelihood of recompensation (LoE 2; new; Agreement: 94%)

7.10. Severity of portal hypertension at the time of decompensation seems to be a negative predictor of recompensation, with the presence of high-risk varices indicating a lower likelihood of recompensation. (LoE 3; new; Agreement: 96%)

7.11. A shorter time from decompensation to aetiological therapy may increase the potential for recompensation. (LoE 2; new; Agreement: 90%)

7.12. The type and number of decompensating events may impact the likelihood of recompensation. (LoE 4; new; Agreement: 91%)

7.13. Patients with recompensated cirrhosis remain at risk for liver-related events and should remain under follow up by a hepatologist at 6-monthly intervals. (LoE 3; SoR: strong; new; Agreement 86%)

7.14. Even though the risk of HCC in recompensated patients is lower compared to decompensated patients, they are still at risk of developing HCC and should thus continue to undergo regular HCC surveillance. (LoE 2; SoR: strong; new; Agreement: 99%)

7.15. In patients achieving recompensation, CSPH may resolve over time. (LoE 2; new; Agreement: 93%)

7.16. CSPH resolution in recompensated patients can be confirmed by an HVPG <10 mmHg when measured off carvedilol/cNSBB (LoE 2; new; Agreement: 93%)

7.17. Portosystemic collaterals and varices may persist after recompensation, despite resolution of CSPH. Therefore, they are not reliable indicators of CSPH persistence after recompensation (LoE 3). The risk of bleeding from persisting varices after recompensation seems to be low. (LoE 2; new; Agreement: 96%)

7.18. Carvedilol/cNSBB treatment may be discontinued in recompensated patients only if resolution of CSPH can be confirmed (LoE 3; SoR: weak; new; Agreement: 93%)

7.19. In patients with recompensated cirrhosis, CSPH resolution / persistence may be non-invasively assessed via liver (LSM) and spleen (SSM) stiffness measurements:

a. LSM <10 kPa may rule-out CSPH (LoE 2; SoR: weak; new; Agreement: 90%).
b. LSM <15 kPa plus SSM <25 kPa may rule-out CSPH (LoE 4; SoR: weak; new; Agreement 92%).
c. LSM >25 kPa may rule-in CSPH (LoE 4; SoR: weak; new; Agreement 94%).

Panel 7 – RECOMPENSATION - RESEARCH AGENDA

RA7.1. *Investigations regarding cirrhosis recompensation in MASLD and MetALD should clearly define "adequate control of metabolic comorbidities" to standardise a surrogate for aetiologic control and facilitate research on cirrhosis recompensation in this patient population.*

RA7.2. *Generate robust evidence supporting the occurrence of recompensation and to define removal or suppression of the primary aetiologic factor(s) in patients with primary biliary cholangitis (PBC), autoimmune hepatitis (AIH), hepatitis D virus (HDV) infection, and hereditary liver diseases (e.g., hemochromatosis and Wilson's disease).*

RA7.3. *Investigate whether differences in length of follow-up and/or cohort characteristics (particularly liver disease severity) may explain the wide range of recompensation rates reported in the literature.*

RA7.4. *Investigate predictors of cirrhosis recompensation, including structural or other abnormalities present at index decompensation that may define a point of no return and features that resolve or persist among patients who achieve recompensation*

RA7.5. *Assess whether an increase in serum albumin levels alone and/or a decrease in MELD score – and their optimal cutoff values are sufficient to define recompensation. Investigate the durability of these laboratory improvements to better characterize recompensation and assess whether sustained improvement provides incremental prognostic value*

RA7.6. *Investigate whether baseline hepatic venous pressure gradient (HVPG), LSM, or other noninvasive tests, when performed during decompensation, can predict subsequent recompensation (or lack thereof).*

RA7.7. *Assess whether the presence, severity, and number of metabolic comorbidities (e.g., overweight/obesity or diabetes) impact the likelihood of achieving recompensation and the risk of liver-related events or HCC after aetiologic therapy or cure.*

RA7.8. *Assess whether dynamics soon after initiation of aetiologic therapy such as a trend towards improvement to CTP-A, increases in serum albumin, and/or improvements in LSM and SSM can predict subsequent recompensation.*

RA7.9. *Investigate the ability of NITs, including LSM, SSM, blood-based markers, and/or combined models to monitor resolution or persistence of CSPH in recompensated cirrhosis*

RA7.10. *Aetiology-specific data are needed to determine the durability of recompensation and the residual risk of decompensation, HCC, and liver-related and all-cause mortality.*

RA7.11. *Investigate whether pharmacologic agents (e.g., statins, FXR agonists, anti-fibrotic or anti-inflammatory drugs) or other therapeutic strategies can facilitate the achievement of recompensation in patients with decompensated cirrhosis.*

RA7.12. *Investigate the impact of body composition, nutritional support, physical therapy and rehabilitation on the likelihood of achieving and maintaining cirrhosis recompensation.*

Panel 8: Vascular Liver Diseases (VLD), Budd-Chiari Syndrome (BCS), Portosinusoidal Vascular Disorder (PSVD), Non-Cirrhotic Portal Fibrosis (NCPF)

General Considerations in Vascular Liver Diseases (VLD)

8.1. Patients with VLD should be managed in close collaboration with centres having experience in VLD, particularly when endovascular treatments are being considered. (LoE 4; SoR: strong; new; Agreement 98.5%)

8.2. In patients with VLD, vaccinations (including viral hepatitis and SARS-CoV-2) and a lifestyle including regular physical activity, a healthy diet and alcohol abstinence should be encouraged. Psychological and social aspects are commonly overlooked in clinical care and require systematic assessment and management. (LoE 3, SoR: strong; new; Agreement 90%)

8.3. In adults without cirrhosis, vascular liver diseases are frequently associated with ≥1 aetiology factor(s), which may be occult at presentation and should be systematically investigated with a comprehensive workup (detailed information provided in EASL CPG on Vascular Liver Diseases). (LoE 2, SoR: strong; modified; Agreement 97.3%)

Anticoagulation in VLD

8.4. Current evidence does not support selecting anticoagulant therapy based on the type of vascular liver disease. (LoE4, SoR: weak; new; Agreement 97.5%)

8.5. When anticoagulation is indicated, therapy should be initiated with low-molecular-weight heparin (LMWH) - followed by transition to an oral anticoagulant. (LoE3, SoR: weak). Because of the increased risk of heparin-induced thrombocytopenia, the use of unfractionated heparin is generally not recommended and should be reserved for special situations (e.g. glomerular filtration rate <30 ml/min or pending invasive procedures). (LoE3, SoR: weak; unchanged; Agreement 96%)

8.6. When anticoagulation is indicated, direct oral anticoagulants (DOACs) may be the primary choice for oral anticoagulation in the absence of double/triple positive anti- phospholipid syndrome, pregnancy, severely impaired liver function (equivalent to Child-Pugh class C) or impaired renal function (glomerular filtration rate < 30 mL/min). (LoE3; SoR: weak; modified; Agreement 91%)

8.7. Anticoagulation therapy should be revisited regularly in a multidisciplinary team and adapted according to liver and kidney function, and patient adherence. (LoE3; SoR: strong; new; Agreement 96%)

Management of portal hypertension-related complications in VLD

8.8. There is currently no evidence to favour EVL over NSBB for primary prophylaxis of variceal bleeding in patients with VLD (LoE 5; new; Agreement 95%

8.9. In VLD, current evidence is insufficient to support the preferential use of carvedilol over other cNSBB. (LoE 5; SoR: strong; new; Agreement 95%)

8.10. When anticoagulation is indicated, it should be initiated without delay and should not be postponed pending endoscopic prophylaxis for variceal bleeding. (LoE 3; strong; new; Agreement 92%)

8.11. EVL may be safely performed without discontinuing LMWH or vitamin K antagonists (VKA). (LoE 3; SoR: weak). Data are lacking regarding DOACs in this setting. (modified; Agreement 92%)

8.12. In patients with VLD and acute portal hypertension-related bleeding, initial bleeding management and secondary prophylaxis are similar to those of patients with cirrhosis. (LoE 4; SoR: strong; modified; Agreement 98%)

8.13. TIPS should be considered in patients with refractory or recurrent portal hypertensive bleeding. (LoE 3; SoR : strong; modified; Agreement 98%)

8.14. There is no data to support the use of p-TIPS in VLD. (LoE 5;; new; Agreement 98%)

Contraception and pregnancy

8.15. Early counselling should be provided to all women of childbearing age with vascular liver disease. This should include guidance on non-oestrogen contraceptive options, discussion of the potential for successful pregnancy under the care of a multidisciplinary team experienced in vascular liver disease, and detailed counselling on associated maternal and fetal risks. (LoE2; SoR: strong; new; Agreement: 91%)

8.16. All teratogenic medications, including cytoreductive agents should be discontinued before conception. VKA, and DOACs should be discontinued as soon as the pregnancy is detected. Oral anticoagulants should be replaced with

therapeutic doses of LMWH during pregnancy. (LoE2, SoR: strong; new; Agreement: 93%)

8.17. In patients with VLD, screening for gastroesophageal varices should be performed during the second trimester of pregnancy, unless adequate screening was conducted within the year preceding conception. (LoE 4; SoR: strong; new; Agreement: 86.4%)

8.18. Primary and secondary prophylaxis of variceal haemorrhage during pregnancy should follow the same principles as in non-pregnant patients, including the use of propranolol. (LoE 3; SoR: strong; new; Agreement: 91%)

8.19. In the absence of obstetric contraindications, vaginal delivery is generally preferred over caesarean section in patients with portal hypertension, provided the platelet count is >20 G/L. (LoE3, SoR: weak; new; Agreement: 88%)

8.20. Treatment with NSBBs, heparin and warfarin is safe during breastfeeding, whereas other VKA and DOACs are contraindicated. (LoE 2; new; Agreement: 88%)

BUDD CHIARI SYNDROME (BCS)

8.21. Budd-Chiari syndrome (BCS) is the consequence of an obstruction to the hepatic venous outflow. Obstruction can be located from the level of the small hepatic veins to the level of the entrance of the IVC into the right atrium. (LoE 1;; unchanged; Agreement: 96%)

8.22. BCS is the preferred designation for any hepatic venous outflow tract obstruction. (LoE 5;; unchanged; Agreement: 96%)

8.23. BCS is considered secondary when the mechanism for venous obstruction is an extrinsic compression or endoluminal neoplastic/parasitic obstruction. Otherwise, BCS is considered primary. (LoE 5; modified; Agreement: 97%)

8.24. BCS presentation and manifestations are extremely diverse, so the diagnosis must be considered in any patient with acute, acute-on-chronic, or chronic liver disease. (LoE 1; SoR: strong; unchanged; Agreement: 96%)

8.25. BCS is diagnosed by demonstrating obstruction of the venous lumen, or by the presence of hepatic vein collaterals associated with the absence of patent hepatic veins. (LoE 1; unchanged; Agreement: 97%)

8.26. In patients with radiologically confirmed BCS, a liver biopsy is not necessary to establish the diagnosis. (LoE 2; SoR: strong; modified; Agreement: 93%)

8.27. When BCS is suspected, and cross-sectional imaging fails to demonstrate large hepatic vein obstruction, liver biopsy is necessary to confirm BCS with small hepatic vein involvement. (LoE 2; SoR: strong; modified; Agreement: 96%)

8.28. In patients with chronic BCS, hepatic nodules are frequent and most often benign. As HCC may also occur, patients should be monitored with periodic imaging and alpha-fetoprotein (AFP) measurements. (LoE 2; SoR: strong; modified; Agreement: 93%)

8.29. A 6-month interval can be proposed for periodic imaging and AFP monitoring, similar to HCC surveillance in cirrhosis. An AFP level of >15 ng/mL should raise suspicion for HCC. (LoE 3; SoR: strong; modified; Agreement: 97%)

8.30. The LIRADS classification cannot be applied in BCS. MRI with hepatobiliary contrast agents is recommended to help distinguish between benign and malignant lesions. For lesions suspected of being HCC, histological confirmation is needed. (LoE 3; SoR: strong; modified; Agreement: 91%)

8.31. Treatment of BCS should follow a stepwise approach including anticoagulation, recanalisation via interventional radiology with or without stent, TIPS and LT. (LoE 2; SoR: strong; Agreement: 97%)

8.32. Therapeutic anticoagulation should be initiated as soon as possible after BCS diagnosis and continued long-term, including after LT. (LoE 2; SoR: strong; modified; Agreement: 95%)

8.33. Clinicians should actively assess for venous stenoses that are suitable for percutaneous recanalisation and manage them accordingly. (LoE 2; SoR: strong; modified; Agreement: 95%)

8.34. In cases where medical management alone is insufficient to improve symptoms and percutaneous recanalisation is not an option or has failed, TIPS is the recommended next step. (LoE 2; SoR: strong; modified; Agreement 97%)

8.35. Improvement in BCS should be assessed as a combination of the following outcomes, evaluated periodically (e.g., at 2-weekly intervals): decreasing rate of ascites formation; decreasing serum bilirubin concentration, decreasing serum creatinine concentration, and/or decreasing INR when elevated (and/or increasing factor V in patients receiving VKA). (LoE 4; SoR: strong; modified; Agreement: 95%)

8.36. The currently available prognostic BCS scores should not determine individual patient management alone, but can be used for research purposes. (LoE 3; SoR: strong; modified; Agreement: 90%)

8.37. In patients with BCS presenting with acute liver failure, urgent LT should be considered. Salvage TIPS should be performed, if possible, independent of LT listing. (LoE 3; SoR: strong; unchanged, Agreement: 93%)

RESEARCH AGENDA: Budd-Chiari Syndrome (BCS)

RA8.1. *Provide a definition of a centre of expertise for VLD*

RA8.2. *Definition of treatment responses, predictors of improvement and criteria to step-up in the treatment strategy of BCS*

RA8.3. *Role of LSM in evaluating outcome and response to treatment in BCS*

RA8.4. *Strategies to prevent rethrombosis beyond anticoagulant therapy*

RA8.5. *Long term outcome after TIPS vs no TIPS*

RA8.6. *Impact of treatment of the underlying disease on prognosis or treatment response in patients with BCS*

RA8.7. *Efficacy and safety of long-term anticoagulation for patients with BCS when underlying cause has been treated*

RA8.8. *Radio-pathological characterization of BCS-associated hepatic nodules*

RA8.9. *Incidence of HCC, risk factors and impact of 6-month surveillance for the development of HCC or hepatic nodules in patients BCS.*

RA8.10. *Outcome of HCC in patients with BCS. Applicability of traditional HCC LT listing criteria (e.g. Milan, AFP model, etc) in LT for HCC in patients with BCS.*

RA8.11. *Addressing geographical differences in BCS*

RA8.12. *Role of LSM for the early diagnosis of TIPS dysfunction and/or for identifying non-responders to medical treatment*

Portosinusoidal Vascular Disorder (PSVD) or Non-Cirrhotic Portal Fibrosis (NCPF) All new

8.38. 'Porto-sinusoidal vascular disorder' (PSVD) or 'non-cirrhotic portal fibrosis' (NCPF) represent a broad clinico-pathological entity that can lead to portal hypertension in the absence of cirrhosis, which overlaps with idiopathic portal hypertension or non-cirrhotic intrahepatic portal hypertension and encompasses

various histological lesions (see statement 8.43) (LoE 1; SoR: strong; new; Agreement: 98%)

8.39. Following the 2026 multi-society nomenclature agreement, both terms 'PSVD' and 'NCPF' are accepted, and they share the same definition (statements 8.38 and 8.40) and diagnostic criteria (statements 8.42, 8.43 and 8.44). (LoE 5; SoR: strong; new; Agreement: 96%)

8.40. The absence of portal hypertension does not rule-out PSVD or NCPF. The presence of concomitant causes of cirrhosis (e.g., viral hepatitis, excessive alcohol consumption, metabolic syndrome, etc.) does not rule-out PSVD or NCPF, and both can coexist. The presence of PVT does not rule out PSVD or NCPF, and both can coexist. (see Supplementary Table-S2 summarizing the conditions excluded from the definition of PSVD or NCPF). (LoE 2; SoR: strong; new; Agreement: 93%)

8.41. A liver biopsy specimen of ≥ 15 mm with at least one fragment ≥ 10 mm - or otherwise considered adequate for interpretation by an expert pathologist - is required for the diagnosis of PSVD or NCPF. (LoE 3; SoR: strong; new; Agreement: 94%)

8.42. Diagnosis of PSVD or NCPF is based on a scoring system (Figure-4) that incorporates clinical features of portal hypertension, histological findings, and relevant concomitant conditions. Diagnosis requires ruling-out cirrhosis on an adequate liver biopsy and confirmation that no exclusion criteria are present. (LoE 5; SoR: strong; new; Agreement: 91%)

8.43. Histological PSVD or NCPF lesions are categorised as major and minor criteria. Major histological criteria include: nodular regenerative hyperplasia (NRH), muscularized portal venules, and portal venule stenosis involving 50% or more of portal tracts. Minor histological criteria include: abnormal distribution of vascular structures, or portal venule stenosis involving 25–49% of portal tracts, or regenerative changes without clear NRH. The concomitant presence of all three minor criteria is considered a major criterion. (LoE 3; new; Agreement 84%)

8.44. Specific signs of portal hypertension include: medium/large oesophageal varices, gastric varices, spontaneous portosystemic collaterals, and SSM > 40 kPa in the absence of myeloproliferative neoplasm. Non-specific features of portal hypertension include: clinically overt ascites, small oesophageal varices,

thrombocytopenia with platelet counts <150 x 10^9 /L, and splenomegaly. (LoE 3; SoR: strong; new; Agreement: 92%)

8.45. Once the diagnosis of PSVD or NCPF is made, patients should be screened for associated immune/inflammatory/systemic diseases, haematological disorders or genetic disorders and exposure to specific drugs/toxins (see Supplementary Table-S3). (LoE 4; SoR: strong; modified Agreement: 93%)

8.46. Screening for gastroesophageal varices is recommended at diagnosis of PSVD or NCPF. (LoE 3; SoR: strong) The recommended frequency of endoscopic surveillance for varices is 2 years in case of no varices, and 1 year in case of small varices (LoE 3; SoR: strong; modified, Agreement 95%)

8.47. Both at initial diagnosis and throughout follow-up, endoscopy can be safely avoided if spleen stiffness measurement is < 40 kPa and serum bilirubin is < 1 mg/dL (< 17 µmol/L), as the likelihood of finding varices is low. (LoE 3; SoR: strong; new, Agreement 91%)

8.48. LSM-based criteria for screening of oesophageal varices used in patients with cirrhosis cannot be applied to patients with PSVD or NCPF. (LoE 2; SoR: strong; new, Agreement 95%)

8.49. Contrast-enhanced cross-sectional imaging is recommended at diagnosis of PSVD or NCPF to evaluate the anatomy and patency of the portal venous system, assess for the presence of spontaneous portosystemic collaterals or hepatic veno-venous communications and the presence of nodules. (LoE 4; SoR: strong; new; Agreement 96%)

8.50. Doppler ultrasound every 6 months is suggested for PVT screening in patients with PSVD or NCPF and signs of portal hypertension. The presence of liver nodules may also be assessed during these evaluations. (LoE 3; SoR: strong; new; Agreement 95%)

8.51. There is no data to guide screening for PVT in patients with PSVD or NCPF without features of portal hypertension. (LoE 5; new; Agreement 96%)

8.52. Nodules develop in PSVD or NCPF, less frequently than in BCS. Most are benign, but in case of suspicion of HCC, histological confirmation is recommended (LoE 4; SoR: strong; new; Agreement 94%)

8.53. LT should be considered in patients with PSVD or NCPF and severe and refractory complications of portal hypertension, hepatopulmonary syndrome, HCC, or with advanced liver dysfunction. Indications should be discussed in liver

transplant centers with expertise in VLD (LoE 4; SoR: strong; new; Agreement 96%)

Panel 8 - RESEARCH AGENDA for PSVD or NCPF

RA8.13. *Impact of severe associated comorbidities on overall outcomes, liver-related outcomes, and treatment responses in patients with PSVD or NCPF*

RA8.14. *Natural history of PSVD or NCPF in the absence of portal hypertension*

RA8.15. *Incidence, prognosis and predictors of PVT development in patients with PSVD or NCPF, and the impact of anticoagulation*

RA8.16. *Strategies for PVT prophylaxis in patients with PSVD or NCPF and signs of portal hypertension*

RA8.17. *Development and validation of non-invasive methods for diagnosis, prognosis, or treatment response (e.g., cross-sectional imaging, SSM) in patients with PSVD or NCPF*

RA8.18. *Diagnostic and prognostic value of EUS-guided portal pressure gradient measurement in patients with PSVD or NCPF*

RA8.19. *Benefit of carvedilol over cNSBB in patients with PSVD/NCPF*

RA8.20. *Radio-pathological characterization of PSVD or NCPF-associated hepatic nodules*

RA8.21. *Histological progression or regression of PSVD or NCPF related lesions*

RA8.22. *Characteristics of paediatric PSVD or NCPF*

RA8.23. *Impact of PSVD or NCPF on quality of life*

RA8.24. *Role of EUS-guided portal pressure gradient and liver biopsy in the management of patients with PSVD or NCPF*

Panel 9 – VALDIG 2 - Portal Vein Thrombosis (PVT)

9.1. PVT is characterised by the presence of a thrombus in the portal vein trunk or its branches. In the context of PVT, portal cavernoma corresponds to the presence of multiple dilated peribiliary and/or gallbladder wall veins (≥ 2 mm) irrespective of the patency of the main portal vein, that develops as a consequence of prior portal vein obstruction (LoE 5). Recent PVT is defined by the formation of a thrombus within the last 6 months. Acute PVT refers to symptomatic recent PVT. Chronic PVT refers to persistent portal vein obstruction lasting more than 6 months, with or without cavernoma. (LoE 2; modified; Agreement: 90%)

9.2. Doppler ultrasound is the first-line diagnostic tool, showing solid intraluminal material, suggesting PVT, and/or a network of porto-portal collaterals, suggesting cavernoma. Multiphase CT should be performed to confirm PVT diagnosis, assess local factors, determine the extent of PVT, evaluate involvement of other splanchnic veins, and identify complications and signs of portal hypertension. Contrast enhanced MRI is an alternative. (LoE 2; SoR: strong; modified; Agreement: 92%)

9.3. According to the VALDIG PVT criteria, the degree of occlusion should be assessed as the percentage of remnant portal lumen (RL) compared to its largest diameter. The degree of occlusion should be classified as follows: minimal if RL ≥50%; partial if RL <50%; complete if RL = 0%, by CT/MRI in the portal venous phase, with the plane strictly perpendicular to the main portal vein. (LoE 4; SoR: strong; modified; Agreement: 93%)

PVT in the absence of cirrhosis

Etiological workup

9.4. Cirrhosis/cACLD should be investigated by a comprehensive workup, including liver stiffness measurement. Liver biopsy and HVPG measurement should be considered in inconclusive cases or when PSVD or NCPF is suspected (LoE 4; SoR: strong; modified; Agreement: 93%)

9.5. Aetiological workup should include screening for permanent and reversible risk factors (see **Suppl. Table-S4**). Permanent risk factors can be further classified into major (including thrombophilia and myeloproliferative neoplasm) and non-major risk factors, depending on the risk of thrombosis recurrence. (LoE 3; SoR: strong; new; Agreement: 89%)

Anticoagulation

9.6. Recent PVT rarely resolves spontaneously, and recanalisation correlates with early initiation of anticoagulation. Therefore, anticoagulation should be started immediately at diagnosis with therapeutic dosaging. (LoE 2; SoR: strong; unchanged; Agreement: 92%)

9.7. In patients with PVT and recent extension to the superior mesenteric vein (SMV), clinical and radiological signs of intestinal ischemia should be assessed (LoE 3; SoR: strong; modified; Agreement: 97%)

9.8. In patients with recent PVT, at least 6 months of anticoagulation is recommended to prevent extension and achieve recanalisation (LoE 2; SoR: strong; unchanged). However, discontinuation of anticoagulation may be considered in case of complete recanalisation of the portal vein after 3 months, in patients with complete resolution of a reversible risk factor and in the absence of a permanent risk factor (LoE 5; SoR: weak; new; Agreement: 94%)

9.9. In patients with recent PVT extending into the SMV and/or splenic vein (SV) who fail to achieve recanalisation after 6 months, full dose anticoagulation therapy may be continued for an additional 6 months, as delayed recanalis ation can still occur (LoE4; SoR: weak; modified; Agreement: 93%)

9.10. The indication for long-term anticoagulation depends on the type of risk factor (see Figure 5) (Agreement: 100%; modified):

a. Long-term anticoagulation at therapeutic doses is recommended in patients with a permanent major risk factor for thrombosis (LoE3; SoR: strong).
b. In patients without a permanent major risk factor for thrombosis, long-term DOACs at lower dose can be considered to prevent thrombosis recurrence. (LoE2; SoR: weak).
c. After complete resolution of a reversible risk factor, discontinuation of anticoagulation can be considered in the absence of a permanent risk factor (LoE4; SoR: weak)

Management of portal hypertension

9.11. Upper GI endoscopy should be performed at diagnosis. In patients without high-risk varices, upper GI endoscopy should be repeated at 12 months and every 2 years thereafter. In patients with SSM <40 kPa, endoscopy can be spared

because the risk of high-risk varices is low (LoE 3; SoR: strong; modified; Agreement: 89%)

Interventional radiology

9.12. Endovascular restoration of portal vein flow relies on a range of techniques, including thrombolysis and thrombectomy for acute/recent PVT, and angioplasty (with/without stenting) and/or creation of transjugular intrahepatic portosystemic shunt (TIPS) for chronic PVT. Technical feasibility and treatment selection depend on the age and extent of thrombosis and local expertise (LoE 5; SoR: strong; new; Agreement: 93%)

9.13. Endovascular restoration of portal vein flow includes TIPS placement in patients with increased intrahepatic resistance (PSVD, PVT extension to the intra hepatic branches) (LoE 5; SoR: weak; new; Agreement: 90%)

9.14. In patients with PVT extending to the SMV, if bowel ischemia persists or worsens after 24–48 hours of anticoagulation (by clinical reassessment, blood tests including lactate levels, and repeated CT scan) and the patient does not require immediate intestinal surgical resection, endovascular treatment should be considered in experienced centers to prevent/reduce the need for surgical intestinal resection (LoE 4; SoR: strong; modified; Agreement: 93%)

9.15. In patients with chronic PVT, with or without cavernoma, and in the absence of cirrhosis, portal vein flow restoration should be considered in those with recurrent complications of PH or symptomatic portal cavernoma cholangiopathy (LoE 4; SoR: strong; **modified**; Agreement: 96%)

Panel 9 - Research agenda – PVT without cirrhosis

RA9.1. *Understand the effect of etiological treatment of underlying liver disease on PVT risk and outcomes*

RA9.2. *Evaluate the efficacy of long-term low dose anticoagulation to prevent recurrence of thrombosis in patients with a major risk factor*

RA9.3. *Assess the efficacy of DOACs (other than rivaroxaban 15 mg/day) in patients with chronic PVT and no major risk factor for thrombosis*

RA9.4. *Assess the efficacy and the safety of anticoagulation interruption in patients with low plasma D-dimer concentration after resolution of a transient risk factor for PVT and no persistent risk factor for thrombosis*

RA9.5. *Assess the efficacy and the safety of long-term anticoagulation in patients with elevated plasma factor VIII concentration and high-risk molecular variants detected by NGS and no major risk factors for PVT*

RA9.6. *Assess the safety of EVL in patients treated with DOACs*

RA9.7. *Define the optimal timing and patient selection criteria for endovascular restoration of portal vein flow in recent or acute PVT without bowel ischemia, particularly in patients who do not achieve recanalization after anticoagulation or who have a low likelihood of spontaneous recanalization.*

RA9.8. *Prospective evaluation of the beneficial impact of earlier endovascular intervention on long-term outcomes.*

PVT in patients with cirrhosis

Screening

9.16. Screening for PVT is recommended in patients with cirrhosis simultaneously with radiological surveillance for HCC (LoE 3; SoR: strong; modified; Agreement: 96%)

9.17. Occurrence of PVT in the presence of HCC is not proof of vascular invasion. Contrast-enhanced imaging is required, and use of standardised criteria for assessment is recommended (e.g. A-VENA, LI-RADS) (LoE 3; SoR: strong; modified; Agreement: 97%)

Indications for treatment

9.18. In potential liver transplant candidates, the goal of anticoagulation is to prevent re-thrombosis or progression of thrombosis to facilitate porto-portal anastomosis and reduce post-transplant morbidity and mortality. Anticoagulation is recommended in potential candidates for liver transplantation with any PVT (LoE 3; SoR: strong; modified; Agreement: 93%)

9.19. Anticoagulation should be considered in patients with (i) recent partial (<50% remnant lumen) or complete thrombosis of the main portal vein (LoE 3; SoR: strong), (ii) symptomatic PVT, independently of the extension (LoE 3; SoR: strong), (iii) nontumoral PVT and HCC, especially in those with indication for locoregional therapy (LoE3; SoR: weak) (modified; Agreement: 90%)

9.20. In candidates for liver transplantation, anticoagulation should be continued until surgery; in all other patients, it should be maintained for at least 6 months and considered for long-term use, regardless of recanalisation (LoE 3; SoR: strong; modified; Agreement: 97%)

9.21. If anticoagulation is discontinued, close follow-up with imaging at 1 month, and then at 3 months intervals thereafter is recommended to evaluate recurrence/progression of thrombosis (LoE 5; SoR: strong; new; Agreement: 97%)

9.22. In patients with cirrhosis and PVT who are not treated with anticoagulation, repeated imaging is recommended within 6 weeks of diagnosis to assess for progression of PVT. For patients who do not develop progression, close follow up with imaging at 3 months intervals is recommended, particularly in patients listed for liver transplantation. For patients who develop progression during follow-up, anticoagulation is indicated (LoE 5; SoR: weak; new; Agreement: 94%)

Considerations for anticoagulant therapy in patients with cirrhosis

9.23. Certain characteristics may place patients at higher risk of bleeding complications with anticoagulant therapy, including age, frailty, renal insufficiency (e.g. glomerular filtration rate below 30 mL/min), decompensated liver disease, and severe thrombocytopenia (e.g., PLT <50 G/L). Decisions for anticoagulation should be individualised based on expected benefit to risk balance. LMWH, VKA, and DOAC are accepted anticoagulation therapies for patients with cirrhosis and PVT. Benefit-risk balance should be reassessed routinely for change in risk (LoE 3; SoR: strong; modified; Agreement: 91%)

9.24. DOACs are safe in patients with Child-Pugh class A cirrhosis and may have reduced bleeding risk compared to VKA. DOACs should be used with caution in patients with impaired liver function (equivalent to Child-Pugh class B). The use of DOACs in patients with severe liver dysfunction (equivalent to Child-Pugh C) is not recommended outside of research protocols. (LoE 3; SoR strong; modified; Agreement: 97%)

9.25. DOACs may have different safety-efficacy profiles in patients with cirrhosis. Currently no recommendation can be made on the choice of a specific DOAC. (LoE 3; SoR: weak; modified; Agreement: 96%)

Interventional radiology

9.26. In patients with cirrhosis and PVT, endovascular restoration of portal vein flow using TIPS can be considered in patients with main portal vein thrombosis without improvement during anticoagulation therapy and (i) complications of portal hypertension, (ii) potential liver transplant candidates with the aim of facilitating physiological anastomosis during LT (LoE 3; SoR: strong; modified; Agreement: 97%)

9.27. TIPS may be considered as a first-line treatment in patients with main portal vein thrombosis with complications of portal hypertension and contraindication of anticoagulation therapy (LoE 4; SoR: weak; new; Agreement: 93%)

9.28. In patients with PVT and cirrhosis who undergo TIPS, routine anticoagulation after TIPS is not recommended (LoE: 2; SoR: strong; new; Agreement: 96%)

Panel 9 – Research agenda – PVT in cirrhosis

RA9.9. *Assess the VALDIG PVT criteria as surrogates for hard clinical outcomes in patients with cirrhosis and PVT*

RA9.10. *Standardize future research definitions for bleeding outcomes in studies assessing anticoagulation and PVT*

RA9.11. *Assess the efficacy and the safety of TIPS as first-line treatment in patients with PVT*

RA9.12. *Evaluate the efficacy and safety of DOACs in patients with decompensated cirrhosis*

RA9.13. *Assess the safety of variceal band ligation in patients treated with DOACs*

Chronic PVT in children

9.29. Chronic PVT in paediatrics is defined as the complete obstruction to the normal flow of portal vein blood to the liver. It can be congenital or acquired, and it is typically diagnosed on imaging (LoE3; new; Agreement: 96%)

9.30. Diagnostic work up should include standard evaluations for underlying chronic liver disorders. Liver biopsy may be considered to rule out intrinsic liver disease. Investigation of potential thrombophilia is recommended (LoE 3; SoR: strong; new;Agreement: 98%)

9.31. In the presence of features of portal hypertension, Meso-R ex b ypass should be considered for preprimary (i.e. without the presence of varices) and primary

prophylaxis of variceal haemorrhage and/or other complications of portal hypertension in children. To ensure a >90% success rate, established criteria should be met, which include but are not limited to patency of the intrahepatic portal venous system and surgical expertise in performance of the Meso-Rex bypass (LoE 3; SoR: strong; new; Agreement: 100%)

9.32. The role of other types of primary prophylaxis (i.e. pharmacologic, endoscopic and interventional radiologic) of variceal haemorrhage is not well established. (LoE 4; new; Agreement: 95%)

9.33. The approach to management of varices may need to be individualised for children without ready access to medical care. (LoE 4; SoR: weak; new; Agreement: 90%)

9.34. Meso-Rex bypass is the preferred approach to secondary prophylaxis of variceal haemorrhage in children with chronic PVT. If Meso-Rex bypass is not feasible, portal vein recanalisation, endoscopic treatment with or without NSBB, or distal splenorenal shunting should be considered. (LoE 3; SoR: strong;new; Agreement: 100%)

9.35. Distal splenorenal shunting should be considered for recurrent variceal haemorrhage in the absence of hepatopulmonary syndrome, portopulmonary hypertension and/or HE. (LoE 3; SoR: strong; new; Agreement: 93%)

9.36. In patients with portal hypertension and/or portosytemic shunts (including both spontaneous and interventional), annual surveillance is warranted to monitor for complications related to portosystemic shunting, especially occurrence of portopulmonary hypertension. (LoE 4; SoR: strong; new; Agreement: 93%)

9.37. In the absence of alternative therapeutic approaches, LT should be considered in children with potentially life-threatening and otherwise untreatable portal hypertensive complications. (LoE 3; SoR: strong; new; Agreement: 100%)

Recent PVT and PVT in children with cirrhosis

9.38. Recent PVT has been described in neonates with umbilical catheters. Spontaneous resolution of PVT can occur in neonates and as such the role of anticoagulation in neonates with recent PVT is uncertain. Monitoring for clot extension is warranted. In the presence of an extensive thrombus, anticoagulant therapy may be considered. (LoE 4; SoR: weak; new; Agreement: 100%)

9.39. PVT associated with cirrhosis is uncommon in children. Thus, the evidence supporting the translation from adult recommendations to children with cirrhotic PVT is insufficient. (LoE 5; new; Agreement: 93%)

Panel 9 – Pediatric PVT - Research Agenda

RA9.14. *Standardize future research definitions for bleeding outcomes in studies assessing anticoagulation*

RA9.15. *Determine the prevalence and clinical relevance of inherited and acquired thrombophilia in children with chronic PVT and assess its impact on recurrence risk and long-term management.*

RA9.16. *Assess the comparative effectiveness of different strategies for primary and secondary prophylaxis of variceal bleeding in children with chronic PVT, including endoscopic, pharmacological, interventional radiologic and surgical approaches.*

RA9.17. *Evaluate the feasibility, efficacy, and long-term patency of portal vein recanalization in children with chronic PVT.*

RA9.18. *Determine the role and safety of anticoagulation in pediatric recent PVT, particularly in catheter-related thrombosis.*

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- (VALDIG) Vascular Liver Disease Interest Group

Abbreviations

(AC) anticoagulation

(EGD) oesophago-gastro-duodenoscopy

(ACLD) advanced chronic liver disease

(ACLF) acute on chronic liver failure

(AFP) alpha-fetoprotein

(AI) artificial intelligence

(AIH) autoimmune hepatitis

(AKI) acute kidney injury

(ALD) alcohol-related liver disease

(A-VENA) Arterial enhancement, Venous enhancement, Expansile portal vein, Neovascularity, Adjacent HCC

(AVB) acute variceal bleeding

(BCLC) Barcelona Clinic Liver Cancer

(BCS) Budd-Chiari Syndrome

(BIA) bioelectrical impedance analysis

(BMI) body mass index

(cACLD) compensated advanced chronic liver disease

(CKD) chronic kidney disease

(CLD) chronic liver disease

(CLIF-C AD) Chronic liver failure acute decompensation score

(cNSBB) conventional non-selective beta-blocker

(CSPH) clinically significant portal hypertension

(CT) Computed Tomography

(CTP) Child-Turcotte-Pugh

(cSEMS) covered self-expanding metal stent

(DEXA) dual-energy X-ray absorptiometry

(DILI) drug-induced liver injury

(DOAC) Direct Oral Anticoagulant

(EASL) European Association for the Study of the Liver

(EVL) endoscopic variceal ligation

(ELF) enhanced liver fibrosis (score)

(EUS) endoscopic ultrasound

(FD) further decompensation

(FGF21) fibroblast growth factor 21

(FIB-4) Fibrosis-4 (index)

(FMT) faecal microbiota transplant (

FXR) farnesoid X receptor

(GAVE) gastric antral vascular ectasia

(GLP-1) glucagon-like peptide 1

(GOV) gastro-ooesophageal varices

(HAV) hepatitis A virus

(HBV) hepatitis B virus

(HCC) hepatocellular carcinoma

(HCV) hepatitis C virus

(HDV) hepatitis D virus

(HE) hepatic encephalopathy

(HEV) hepatitis E virus

(HRS-AKI) hepatorenal syndrome-acute kidney injury

(HVPG) hepatic venous pressure gradient

(IGV) isolated gastric varices

(LFI) Liver Frailty Index

(LMWH) low-molecular-weight heparin

(LOLA) L-ornithine-L-aspartate

(LoE) level of evidence

(LSM) liver stiffness measurement

(LT) liver transplantation

(LVP) large-volume paracentesis

(MASLD) metabolic dysfunction-associated steatotic liver disease

(MELD) Model for End-stage Liver Disease

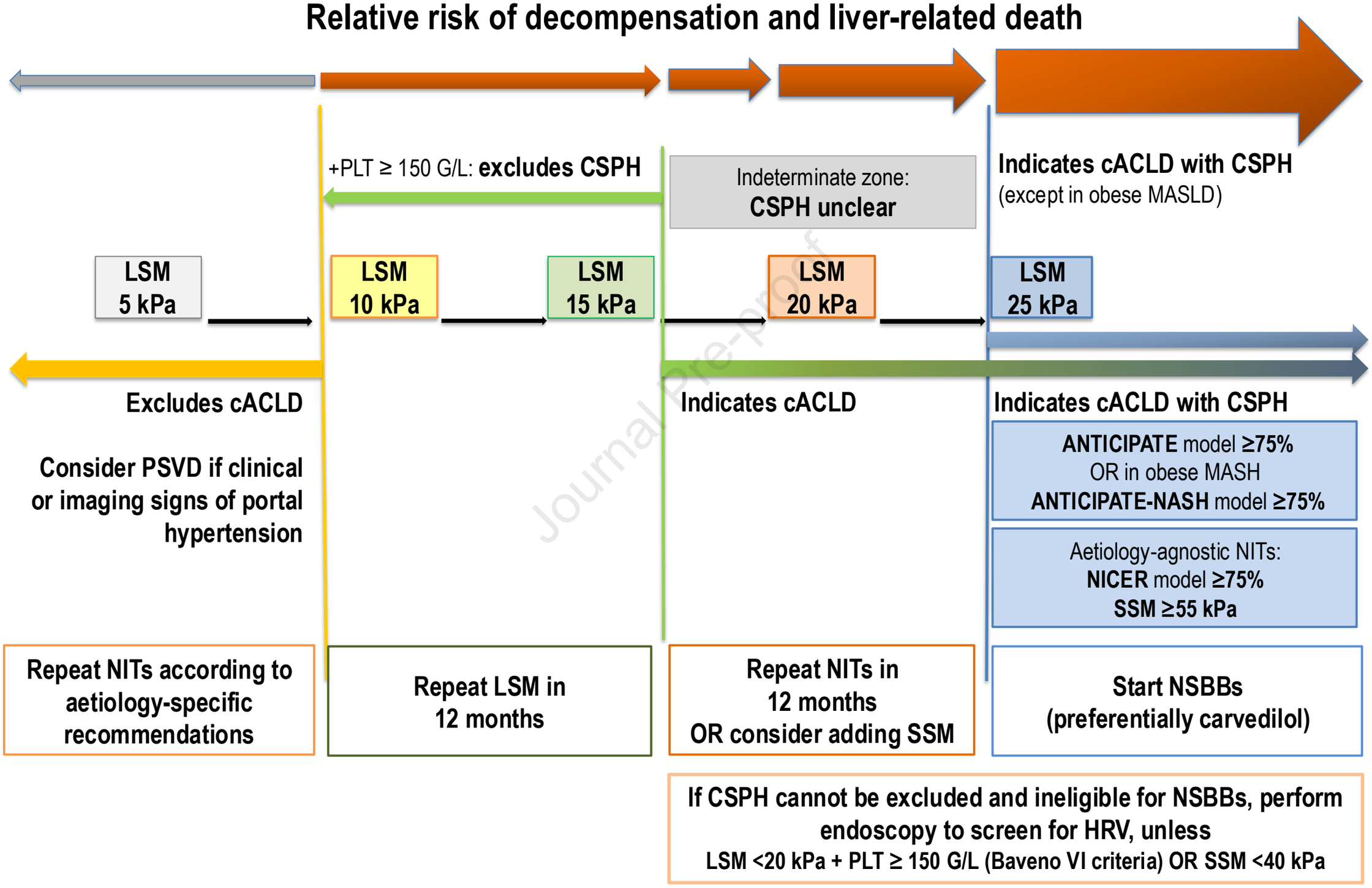
(MetALD) MASLD and increased alcohol intake

(MRI) Magnetic Resonance Imaging

(NCPF) Non Cirrhotic Portal Fibrosis
(NIT) non-invasive test
(NSBB) non-selective beta-blocker
(NRH) nodular regenerative hyperplasia
(OHE) overt hepatic encephalopathy
(OV) ooesophageal varices
(PBC) primary biliary cholangitis
(PH) portal hypertension
(PHG) portal hypertensive gastropathy
(PLT) platelets
(PPI) proton pump inhibitors
(PSS) portosystemic shunts
(PSVD) portosinusoidal vascular disorder
(PTFE) polytetrafluoroethylene
(pTIPS) pre-emptive transjugular intrahepatic portosystemic shunt
(PVT) portal vein thrombosis
(RCT) randomized controlled trial
(SBP) spontaneous bacterial peritonitis
(SGLT 2) sodium-glucose cotransporter 2
(SLD) steatotic liver disease
(SMI) skeletal muscle index
(SMV) superior mesenteric vein
(SoR) strength of recommendation
(SOS) sinusoidal obstruction syndrome
(SSM) spleen stiffness measurement
(SV) splenic vein
(SVR) sustained virological response
(TIPS) transjugular intrahepatic portosystemic shunt
(VALDIG) Vascular Liver Disease Interest Group
(VCTE) vibration-controlled transient elastography
(VITRO) von Willebrand factor antigen to platelet ratio
(VKA) vitamin K antagonist
(VLD) vascular liver disease

REFERENCES

1. de Franchis R, Pascal JP, Ancona E, Burroughs AK, Henderson M, Fleig W, et al. Definitions, methodology and therapeutic strategies in portal hypertension. A Consensus Development Workshop, Baveno, Lake Maggiore, Italy, April 5 and 6, 1990. J Hepatol. 1992;15(1-2):256-61.
2. de Franchis R. Developing consensus in portal hypertension. J Hepatol. 1996;25(3):390-4.
3. de Franchis R. Updating consensus in portal hypertension: report of the Baveno III Consensus Workshop on definitions, methodology and therapeutic strategies in portal hypertension. J Hepatol. 2000;33(5):846-52.
4. de Franchis R. Evolving consensus in portal hypertension. Report of the Baveno IV consensus workshop on methodology of diagnosis and therapy in portal hypertension. J Hepatol. 2005;43(1):167-76.
5. de Franchis R, Baveno VF. Revising consensus in portal hypertension: report of the Baveno V consensus workshop on methodology of diagnosis and therapy in portal hypertension. J Hepatol. 2010;53(4):762-8.
6. de Franchis R, Baveno VIF. Expanding consensus in portal hypertension: Report of the Baveno VI Consensus Workshop: Stratifying risk and individualizing care for portal hypertension. J Hepatol. 2015;63(3):743-52.
7. de Franchis R, Bosch J, Garcia-Tsao G, Reiberger T, Ripoll C, Baveno VIIF. Baveno VII - Renewing consensus in portal hypertension. J Hepatol. 2022;76(4):959-74.



cACLD

- LSM ≥10 kPa suggestive
- LSM ≥15 kPa highly suggestive
- Histology F3/F4

\+ No current or h/o decompensation

cACLD +CSPH

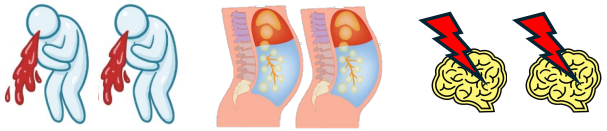
- LSM ≥25kPa
- ANTICIPATE ≥75%
- ANTICIPATE-NASH ≥75%
- SSM ≥55kPa
- NICER ≥75%
- Varices on Endoscopy
- HVPG ≥10 mmHg
- Portosystemic Collaterals

First Decompensation

- Overt Ascites
- Variceal bleeding (AVB)
- Overt hepatic encephalopathy (OHE)

Further Decompensation (DC)

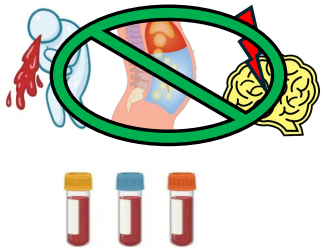
- Any combination of decompensating events
- Recurrent/refractory ascites
- HRS-AKI or SBP
- Recurrent OHE
- Variceal rebleeding
- AVB / ascites / OHE + jaundice



Recompensated Cirrhosis

CSPH resolution

- HVPG <10 mmHg
- LSM <10 kPa
- LSM <15 kPa +SSM <25 kPa



1) Etiologic cure / control / suppression
2) No ascites / no HE (w/o diuretics / HE prophylaxis)
3) No variceal rebleeding
4) Improved liver function: CTP-Score A5/A6

* Criteria 1-3 must be fulfilled for ≥6 months

Point of no Return

Management algorithm for acute variceal bleeding (AVB) in patients with ACLD / cirrhosis — stratified by source of bleeding

ACLD / Cirrhosis — Suspected Upper GI Portal Hypertension–Related Bleeding

INITIAL MANAGEMENT

- Hospitalization: IMCU or ICU per clinical presentation
- Restrictive transfusion pRBC (target Hb 7–8 g/dL)
- Preventive antibiotics — 3rd-gen cephalosporins (Child-Pugh A: shorten/omit)
- Orotracheal intubation if hematemesis or altered consciousness
- No routine FFP, platelets, rFVIIa, or tranexamic acid
- Restore and maintain hemodynamic stability
- Vasoactive therapy — start ASAP, continue 2–5 days or until TIPS
- Erythromycin 250 mg IV, 30–90 min prior to endoscopy
- Lactulose to prevent or treat overt hepatic encephalopathy

Upper Endoscopy within 12 hours after admission

SOURCE OF BLEEDING DETERMINES PATHWAY

Bleeding from EV or GOV1

Endoscopic Band Ligation (EBL)

Bleeding controlled

PRE-EMPTIVE TIPS (pTIPS)*
Within 72 h (ideally <24 h)
· Child-Pugh C10–C13
· Child-Pugh B ≥8 + active bleeding
· HVPG ≥ 20 mmHg

IF CRITERIA NOT MET

No Rebleeding

2° PROPHYLAXIS
NSBB + EBL
Carvedilol preferred over cNSBB
Repeat EBL q2–4 wks until eradication

Rebleeding

Consider TIPS

Failure to control bleeding

SEMS $
Bridge to TIPS or liver transplantation

Salvage TIPS
+ evaluate LT candidacy

Bleeding from GOV2 or IGV1

BIMODAL TREATMENT APPROACH
Both phases may start in parallel · If staged: minimize the interval between them

ACUTE PHASE — IMMEDIATE HEMOSTASIS

PREFERRED — WHEN AVAILABLE
EUS-guided coil + cyanoacrylate
↑ Variceal obliteration rates
↓ Late rebleeding · ↓ Reintervention
Experienced centres only

IF NOT AVAILABLE

ALTERNATIVE
Conventional endoscopic **cyanoacrylate injection**
Equally effective for initial hemostasis

parallel or staged (ASAP)

DEFINITIVE PHASE — DURABLE CONTROL

PREFERRED — AS SOON AS POSSIBLE
TIPS ± variceal embolization
First-line definitive strategy
pTIPS: Child-Pugh C<14 (incl. A and B) age <75 yr · creatinine <3 mg/dL · no heart failure

IF CONTRAINDICATED

ALTERNATIVES — after MDT, per anatomy / bleeding site
› Transvenous obliteration
› Endoscopic therapies (incl. EUS-guided)
› Surgical methods

Consider iron supplementation before discharge in patients with blood loss anaemia (Hb <10 g/dL)

***pTIPS criteria:** *age <75 yr · creatinine <3 mg/dL · no heart failure* **$** *preferred over balloon tamponade* **cNSBB** = *conventional non-selective beta-blocker (propranolol / nadolol)*

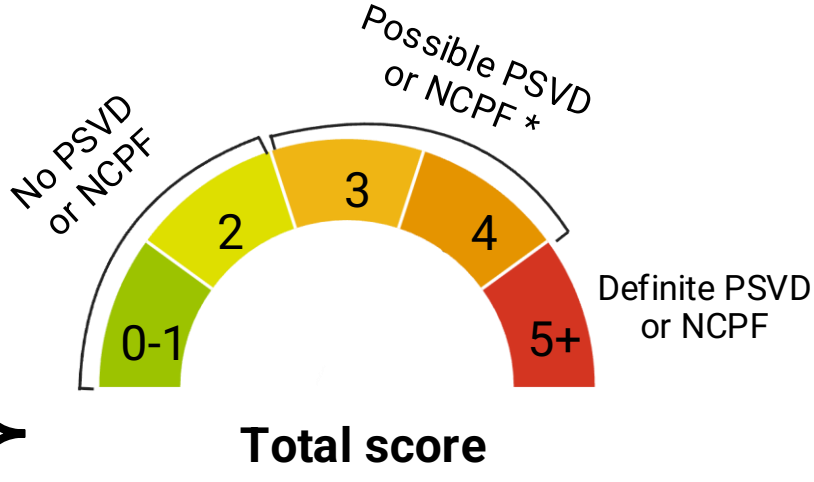
Abbreviations: ACLD = advanced chronic liver disease; AVB = acute variceal bleeding; cNSBB = conventional non-selective beta-blocker (propranolol/nadolol); EBL = endoscopic band ligation; EUS = endoscopic ultrasound; EV = esophageal varices; FFP = fresh frozen plasma; GOV = gastroesophageal varices; Hb = haemoglobin; HVPG = hepatic venous pressure gradient; IGV = isolated gastric varices; IMCU = intermediate care unit; LT = liver transplantation; MDT = multidisciplinary team; NSBB = non-selective beta-blocker; pRBC = packed red blood cells; pTIPS = preemptive TIPS; rFVIIa = recombinant factor VIIa; SEMS = self-expandable metallic stent; TIPS = transjugular intrahepatic portosystemic shunt.

No cirrhosis on adequate biopsy

+

No exclusion criteria

Component	Type	Score
Clinical signs of portal hypertension	Specific	+3
	Non-specific	+2
	No sign	0
Histological lesions	Major	+5
	Minor	+2
	Normal	0
Condition known to be associated with PSVD/NCPF	Present	+1
	Absent	0
Concomitant cause CLD	Present	-1
	Absent	0

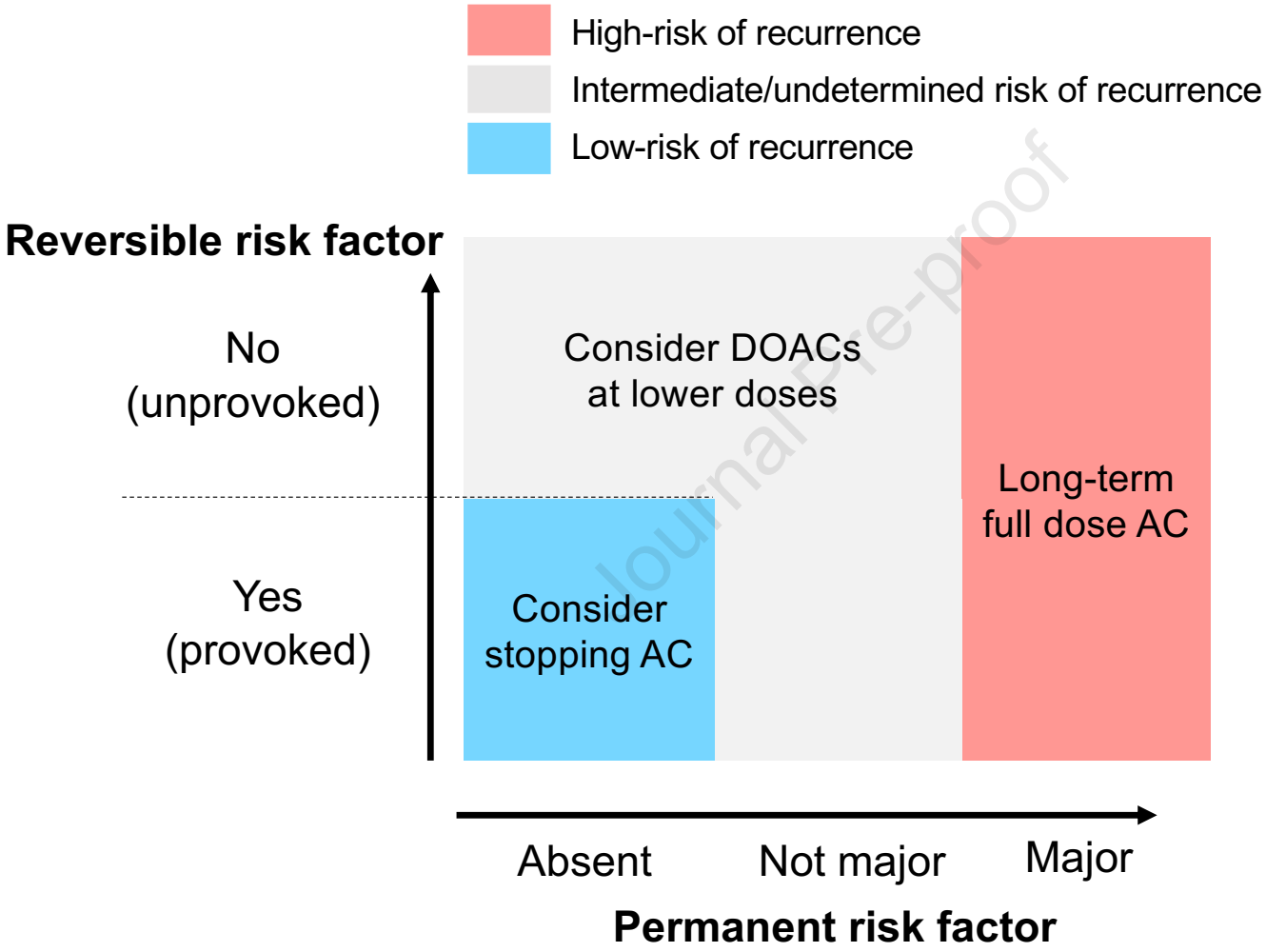


Total score

* Posible PSVD or NCPF (3-4 points):

- Other investigations (e.g. new liver biopsy, LSM)
- refer the patient to a center expert in vascular liver disease
- Follow the patient to detect new signs

Considerations for long-term anticoagulation for PVT in the absence of cirrhosis



Relevant valid statements from prior Baveno Workshops	
Statements on medical management	
The decision to treat with Carvedilol/cNSBB should be taken when indicated, independent of the possibility of measuring HVPG.	Baveno VI, VII
Alcohol abstinence should be encouraged in all patients with cirrhosis irrespective of aetiology.	Baveno VI
The use of aspirin should not be discouraged in patients with cirrhosis and an approved indication for aspirin, since it may reduce the risk of HCC, liver-related complications, and death.	Baveno VII
PHG has to be distinguished from gastric antral vascular ectasia because treatments are different	Baveno VI, VII
Short-term albumin administration is indicated for spontaneous bacterial peritonitis (SBP), acute kidney injury (AKI) >stage 1A, large-volume paracentesis and combined with terlipressin for hepatorenal syndrome (HRS)-AKI.	Baveno VII
TIPS is the treatment of choice in patients who rebleed despite traditional NSBBs or carvedilol and EVL.	Baveno VI, VII
In patients who cannot get/tolerate EVL or carvedilol or traditional NSBBs, any of these therapies can be maintained alone (A1) and TIPS should be considered in patients with recurrent ascites.)	Baveno VII
In patients who bleed despite adherence to traditional NSBBs or carvedilol as primary prophylaxis, the combination of traditional NSBBs or carvedilol and EVL is recommended, and TIPS should be considered in those with recurrent ascites.	Baveno VII
In all patients hospitalized with decompensation, bacterial infections should be ruled out. The minimal work-up for infections should include diagnostic paracentesis, chest-X ray, cultures of blood, ascites and urine and skin examination.	Baveno VII
Patients with bacterial infections should be promptly treated with antibiotics. The empirical antibiotic treatment should be tailored to local epidemiology, risk factors for multidrug-resistant bacteria and severity of infection. If no response to antibiotics is observed, consider viral and fungal infections	Baveno VII
Statement on the value of HVPG*	
HVPG measurement provides prognostic information. It is the gold-standard method to assess the presence of CSPH which is defined as HVPG ≥10 mmHg.	Baveno VI, Baveno VII
In patients with viral- and alcohol-related cirrhosis, HVPG measurement is the gold-standard method to determine the presence of “clinically significant portal hypertension” (CSPH), which is defined as an HVPG ≥10 mmHg.	Baveno VII
In patients with MASLD-related cirrhosis, although an HVPG ≥ 10 mmHg remains strongly associated with the presence of clinical signs of portal	Baveno VII

hypertension, these signs can also be present in a small proportion of patients with HVPG values <10 mmHg.	
The presence of CSPH, determined either by HVPG ≥ 10 mmHg or by clinical manifestations of portal hypertension is associated with a higher risk of decompensation and mortality in patients with cirrhosis undergoing liver resection for hepatocellular carcinoma.	Baveno VII
In candidates for non-hepatic abdominal surgery, a HVPG ≥16 mmHg is associated with an increased risk of short-term mortality after surgery.	Baveno VII
Removal/suppression of the primary aetiological factor leads to potentially meaningful decreases in HVPG in most patients and substantially reduces the risk of hepatic decompensation.	Baveno VII
Absence/resolution of CSPH following removal/suppression of the primary aetiological factor prevents hepatic decompensation	Baveno VII
The optimal percent/absolute decrease in HVPG associated with a reduction in hepatic decompensation following the removal/suppression of the primary aetiological factor in patients with cACLD and CSPH has yet to be established.	Baveno VII
HVPG change is a relevant surrogate outcome. HVPG response to NSBBs is associated with a significant reduction in risk of variceal bleeding and decompensation.	Baveno VI
Measurement of HVPG response to therapy offers additional relevant information: a decrease in HVPG of at least 10% from baseline or to ≤12 mmHg after chronic treatment with NSBB is clinically relevant in the setting of primary prophylaxis. Similarly, acute HVPG response to intravenous propranolol may be used to identify responders to NSBB, specifically a decrease in HVPG of 10% or to ≤12 mmHg may be relevant in this setting.	Baveno V, VI
HVPG measurements should be encouraged in clinical trials investigating novel therapies but are not essential if portal hypertension-associated endpoints are well defined.	Baveno VI
Statements on technical aspects of HVPG and PPG measurement	
The use of an end-hole, compliant balloon occlusion catheter reduces the random error of wedged hepatic vein pressure (WHVP) measurements and is preferred over the use of conventional straight catheter.	Baveno VII
A small volume of contrast medium should be injected when the occlusion balloon is inflated to confirm a satisfactory occluded position and to exclude the presence of hepatic vein-to-vein communications.	Baveno VII
Deep sedation during liver haemodynamic measurement may cause inaccurate HVPG values. If light sedation is required, low dose midazolam (0.02 mg/kg) does not modify the HVPG and is acceptable.	Baveno VII
To properly reflect portal venous pressure, WHVP requires a stabilisation time. Recording of WHVP requires a minimum of 1 minute, with particular attention to stability during the last 20-30 seconds. WHVP should be recorded in triplicate.	Baveno VII

The wedged to free hepatic vein pressure gradient has superior clinical prognostic value than wedged to right atrial pressure gradient and should be used as the standard reference for HVPG calculation. Right atrial pressure (RAP) can be measured to rule out post-hepatic component of portal hypertension.	Baveno VII
The immediate post-TIPS PPG may be influenced by various factors, such as general anaesthesia, use of vasoactive agents or haemodynamic instability and therefore immediate post-TIPS PPG may not represent long-term PPG. PPG measurements in haemodynamically stable, non-sedated patients better reflect post-TIPS PPG values and are recommended.	Baveno VII

**** These statements remain valid but should be interpreted in the context of current Baveno VIII recommendations which favor non-invasive tests over routine HVPG measurements in daily clinical practice***"