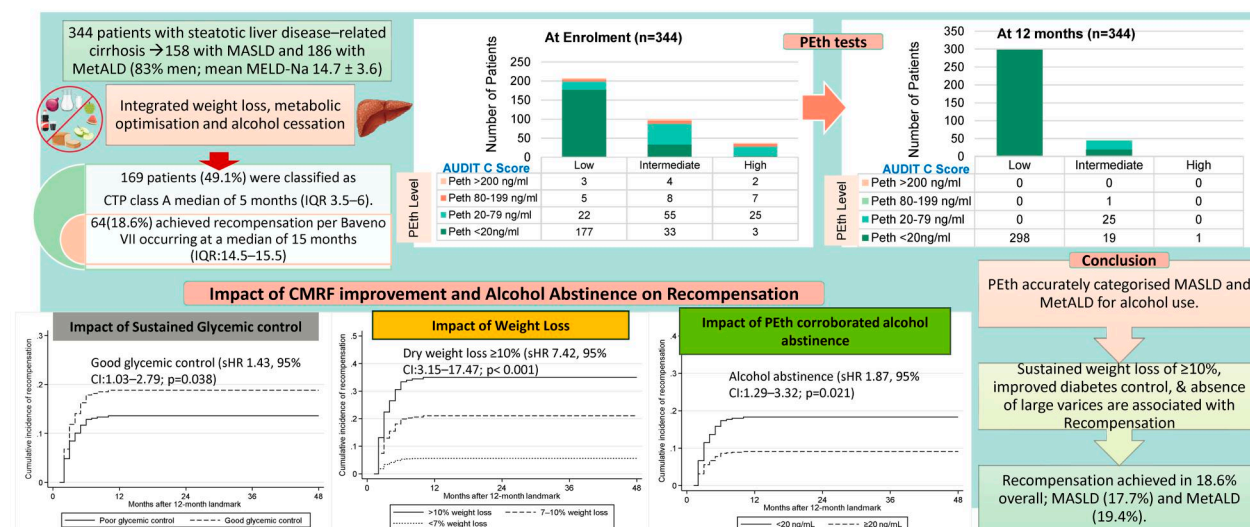


Recompensation of decompensated cirrhosis in a spectrum of metabolic dysfunction–related steatotic liver disease, with PEth-corroborated alcohol abstinence and modification of cardiometabolic risk factors

VISUAL ABSTRACT

Recompensation of decompensated cirrhosis in a spectrum of metabolic dysfunction–related steatotic liver disease, with PEth-corroborated alcohol abstinence and modification of cardiometabolic risk factors



ORIGINAL ARTICLE

Recompensation of decompensated cirrhosis in a spectrum of metabolic dysfunction–related steatotic liver disease, with PEth-corroborated alcohol abstinence and modification of cardiometabolic risk factors

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Abstract

Background and Aims: Recompensation of decompensated metabolic dysfunction–associated steatotic liver disease (MASLD) and metabolic dysfunction–associated steatotic liver disease with alcohol consumption (MetALD) cirrhosis remains unknown.

Approach: We assessed rates and predictors of recompensation (Baveno VII criteria) and outcomes after modifiable interventions, including alcohol abstinence, dry-weight loss, and cardiometabolic risk factor management, in a cohort of patients with decompensated MASLD/MetALD cirrhosis between October 2019 and March 2025.

Results: Among 344 patients with steatotic liver disease-related cirrhosis (mean MELD-Na 14.7 ± 3.6 ; MASLD-158, MetALD-186), 64 (18.6%) achieved recompensation; MASLD (17.7%) and MetALD (19.4%), at a median of 15 months (IQR: 14.5–15.5) over 3.9 years (IQR: 2.8–4) follow-up. Baseline phosphatidylethanol (PEth) was negative in MASLD but positive in

Abbreviations: AASLD, American Association for the Study of Liver Diseases; ACLF, acute-on-chronic liver failure; ALD, alcohol-associated liver disease; ALT, alanine transaminase; ANOVA, analysis of variance; APRI, AST-to-platelet ratio index; AST, aspartate transaminase; AUD, alcohol use disorder; AUDIT-C, Alcohol Use Disorders Identification Test–Consumption; AVB, acute variceal bleeding; BMI, body mass index; CIF, cumulative incidence function; CI, confidence interval; CMRF, cardiometabolic risk factor; CTP, Child–Turcotte–Pugh; EGD, esophagogastroduodenoscopy; EVL, endoscopic variceal ligation; Fib-4, fibrosis-4 index; HBV, hepatitis B virus; HCC, hepatocellular carcinoma; HCV, hepatitis C virus; HDL, high-density lipoprotein; HE, hepatic encephalopathy; HR, hazard ratio; LC–MS/MS, liquid chromatography–tandem mass spectrometry; LDL, low-density lipoprotein; LSM, liver stiffness measurement; MASLD, metabolic dysfunction–associated steatotic liver disease; MELD, Model for End-stage Liver Disease; MetALD, metabolic dysfunction–associated steatotic liver disease with increased alcohol intake; NSBB, non-selective beta blocker; OHE, overt hepatic encephalopathy; PEth, phosphatidylethanol; PGIMER, Postgraduate Institute of Medical Education and Research; POCUS, point-of-care ultrasound; PVR, portal vein recanalization; SGLT-2 inhibitor, sodium–glucose cotransporter 2 inhibitor; sHR, subdistribution hazard ratio; SLD, steatotic liver disease; T2DM, type 2 diabetes mellitus; TIPS, transjugular intrahepatic portosystemic shunt.

The study was presented at a parallel session at The Liver Meeting (TLM) of the American Association for the Study of Liver Diseases in November 2025, in Washington, DC, USA.

Madhumita Premkumar is the first author.

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69.4% of MetALD patients ($p=0.001$). In all, 61 patients (17.7%) died, 11 (3.2%) underwent liver transplantation, and 5 (1.5%) developed hepatocellular carcinoma. Mortality was lower in MASLD than in MetALD (12.0% vs. 22.6%; $p=0.011$). In competing-risk regression adjusted for baseline MELD-Na, absence of large esophageal varices (sHR 1.97, 95% CI: 1.22–3.99; $p=0.021$), weight loss $\geq 10\%$ (sHR 7.42, 95% CI: 3.15–17.47; $p<0.001$), alcohol abstinence (sHR 1.87, 95% CI: 1.29–3.32; $p=0.003$), and glycemic control (sHR 1.43, 95% CI: 1.03–2.79; $p=0.038$) were associated with recompensation. Higher MELD-Na (sHR 1.13, 95% CI: 1.06–1.2; $p<0.001$), MetALD etiology (sHR 1.87, 95% CI: 1.09–3.22; $p=0.023$), lack of recompensation ($p<0.001$), and ongoing alcohol consumption (sHR 5.3, 95% CI: 2.85–9.74; $p<0.001$) were associated with mortality.

Conclusions: PEth improved the ascertainment of alcohol exposure and phenotyping of MASLD and MetALD. Weight loss $\geq 10\%$, achievement of glycemic control, and absence of large varices were independently associated with recompensation, which occurred in 18.6% of patients with decompensated MASLD/MetALD cirrhosis.

Keywords: MASLD, metabolic dysfunction–associated steatotic liver disease cirrhosis, MetALD, PEth, phosphatidylethanol, recompensation in cirrhosis

INTRODUCTION

Recent evidence demonstrates that select patients with decompensated cirrhosis can experience clinically meaningful improvement in their decompensation events following sustained suppression or eradication of the etiology of the underlying disease.^[1–3] This phenomenon of recompensation, as defined by the Baveno VII consensus, involves sustained etiological control, resolution of decompensating events without ongoing prophylactic therapy such as diuretics, lactulose, or rifaximin, and improvement in hepatic synthetic function over time.^[4] While recompensation is associated with improved transplant-free survival, residual risks of portal hypertension progression and hepatocellular carcinoma (HCC) persist.^[1,5]

Alcohol-associated liver disease (ALD) requires sustained alcohol abstinence, which is essential for disease modification, and leads to recompensation in up to 30%.^[2,6] Recompensation in cirrhosis related to the broad spectrum of steatotic liver disease (SLD), including metabolic dysfunction–associated steatotic liver disease (MASLD), remains poorly characterized, while it is the leading cause of chronic liver disease worldwide and is projected to become the dominant indication for liver transplantation.^[7] Unlike ALD or hepatitis B virus (HBV) or hepatitis C virus (HCV)-related liver disease, MASLD is driven by persistent

metabolic dysfunction, including insulin resistance, adipose tissue dysregulation, systemic inflammation, and cardiometabolic risk factors (CMRFs) like type 2 diabetes mellitus (T2DM) and hypertension.^[8] Further, the precise categorization of metabolic dysfunction and alcohol-associated liver disease (MetALD) complicates the assessment of rates and predictors of recompensation.^[9] Even moderate alcohol intake accelerates fibrosis progression and worsens outcomes in metabolically vulnerable patients. In patients with MetALD, recompensation requires both alcohol abstinence and optimization of CMRFs.^[10] Accurate quantification of alcohol intake is central to both MASLD/MetALD disease classification and interpretation of recompensation.^[11] Self-reported alcohol consumption is limited by recall bias and under-reporting. Phosphatidylethanol (PEth), a direct blood-based biomarker formed exclusively in the presence of ethanol, provides highly specific detection of alcohol intake over the preceding 2–4 weeks.^[12] In patients with SLD, PEth has identified under-reported alcohol use, leading to reclassification of a substantial proportion of individuals initially labeled as MASLD into MetALD or ALD categories.^[12] Beyond subclassification, serial PEth measurements enable objective monitoring of abstinence, which is critical when evaluating recompensation, while enabling the use of drugs for alcohol use disorder (AUD).^[13]

Despite growing recognition of MetALD as a distinct steatotic liver disease phenotype, whether patients with prior decompensation can achieve recompensation and whether outcomes differ from those observed in MASLD remain unknown. A recent retrospective analysis of transplant waitlisted patients revealed a 3% recompensation rate among 59 patients with MetALD, but the findings were not corroborated by PETH testing.^[14] At present, pharmacological therapy for MASLD has not been proven to be safe or recommended in patients with decompensated cirrhosis.^[15] Bariatric surgery is contraindicated in most patients with decompensated cirrhosis, and while select interventions may improve metabolic parameters, data on recompensation outcomes are lacking.^[16] In the prospective NASH Clinical Research Network cohort, each 1% reduction in HbA1c and each 5% reduction in body weight were associated with 32% and 27% greater likelihoods of liver stiffness improvement, respectively, supporting the role of cardiometabolic optimization in regression of liver disease severity.^[17]

Recompensation in this setting is likely to depend on sustained metabolic optimization, lifestyle modification, alcohol abstinence, and effective management of portal hypertension. We therefore hypothesized that sustained alcohol abstinence and control of cardiometabolic risk factors could serve as surrogates for etiologic suppression in MASLD and MetALD and be associated with recompensation after prior decompensation. To test this hypothesis, we prospectively evaluated 344 patients with PETH-characterized MASLD/MetALD-related decompensated cirrhosis to determine rates, predictors, and outcomes of recompensation, including mortality.

METHODS

Study design and population

This was a prospective observational cohort study on Cardiometabolic Risk assessment in cirrhosis, conducted between October 2019 and March 2025 at the Postgraduate Institute of Medical Education and Research (PGIMER), Chandigarh, India. Ethical clearance for the prospective cardiometabolic cohort study was obtained from the Institutional Ethical Committee (IEC Number PGI/IEC/2019/002095, dated 25.9.2019) and subsequently modified in view of the change in MASLD nomenclature (vide PGI/INT/IEC/2024/1523, dated 3.12.2024). The research was conducted in accordance with the Declaration of Helsinki and the Declaration of Istanbul. Adult patients with decompensated cirrhosis attributed to MASLD or MetALD were consecutively enrolled and followed at least at planned 6-monthly (with a 1-month window) intervals, with more frequent assessment every 3 months for metabolic

optimization, achievement of alcohol cessation, and management of liver-related complications. All patients were enrolled in an HCC surveillance program with 6-monthly imaging. Planned follow-up assessments included evaluation for recompensation, further decompensation, liver transplantation, or death.

Cirrhosis was diagnosed based on a combination of clinical, radiological, endoscopic, and laboratory criteria, and SLD was classified as per the current consensus definition.^[18] Patients were eligible if they had a prior history of hepatic decompensation, defined as ascites, variceal bleeding, or hepatic encephalopathy.

Patients with ALD, decompensated cirrhosis of other etiologies (autoimmune, Wilson disease, viremic HBV, and HCV-related cirrhosis), patients on hemodialysis, genetic disease-related SLD, drug-induced SLD, active HCC, prior liver transplant, having undergone bariatric surgery, or transjugular intrahepatic portosystemic shunt (TIPS) were excluded from the present analysis.

Study objectives

The objectives of this study were to (1) determine the rates and predictors of recompensation in the overall cohort and in the MASLD and MetALD groups after controlling for cardiometabolic factors and cessation of alcohol; (2) precisely classify the 2 SLDs, that is, MASLD/MetALD, based on PETH testing and AUDIT-C scores; (3) assess and ensure alcohol abstinence based on PETH testing and AUDIT-C scores; (4) assess improvement or deterioration in liver disease severity, that is, achieving Child–Turcotte–Pugh (CTP) class A during follow-up or further decompensation events; and (5) evaluate for differences in clinical outcomes (deaths or liver transplantation) between MASLD and MetALD and transplant-free survival in recompensated versus non-recompensated patients.

Definitions of the steatotic liver disease spectrum

MASLD was defined as hepatic steatosis in combination with at least one cardiometabolic risk factor (overweight or obesity, T2DM, arterial hypertension, or dyslipidemia), in the absence of significant ongoing alcohol consumption above accepted thresholds.^[12] MetALD was defined in patients meeting criteria for MASLD who also reported alcohol intake above MASLD thresholds but below levels traditionally used to define ALD. Alcohol exposure level was defined in MetALD as alcohol intake 140–350 g/week (women) or 210–420 g/week (men), consistent with contemporary nomenclature criteria. In MASLD, alcohol consumption was below MetALD thresholds.^[18] Overweight and

obesity were defined using Asian-specific BMI thresholds: overweight as BMI ≥ 23 kg/m² and obesity as BMI ≥ 27.5 kg/m².^[19]

Patients with ALD, defined by sustained alcohol consumption above these thresholds, were excluded from the present analysis, as they have been reported previously.^[2]

Definition of recompensation

Recompensation was defined according to Baveno VII criteria, requiring:

1. Sustained removal or control of the underlying etiological driver (alcohol abstinence and/or metabolic optimization),
2. Resolution of prior decompensating events without the need for ongoing prophylactic therapy (including diuretics for ascites or lactulose/rifaximin for hepatic encephalopathy) and absence of recurrent acute variceal bleeding (AVB) for at least 12 months.
3. Sustained improvement in hepatic synthetic function, reflected by improvement in CTP class to A and/or Model for End-Stage Liver Disease—Sodium (MELD-Na) to ≤ 10 .

Patients were classified as recompensated only if all criteria were met at the follow-up visit, including the absence of intermittent diuretic, rifaximin, or lactulose use.^[4]

We identified an additional clinical endpoint: patients who reached CTP -class A and/or had a MELD-Na score ≤ 10 without experiencing overt decompensation. These individuals may still require maintenance medications while demonstrating improved liver synthetic function.^[20] In patients who attained CTP-A, an attempt was made to taper and discontinue diuretics and lactulose/rifaximin to assess for recompensation, per Baveno VII criteria. These drugs were restarted if the patient had a recurrence of ascites or HE.

Clinical data

Patient-level data were prospectively collected and included demographics, place of residence, metabolic comorbidities, alcohol exposure, fibrosis and liver disease severity scores, variceal status, type and number of decompensating events, pharmacological therapies, and imaging findings. Data were manually entered into a secure Microsoft Excel database. Changes in liver disease severity were assessed longitudinally, and the CTP and MELD scores were recorded at enrollment and at follow-up.

Assessment of alcohol exposure

To improve the accuracy of MASLD and MetALD phenotyping, alcohol exposure was assessed using self-reported alcohol consumption, the AUDIT-C questionnaire, and PEth testing, with a predefined positive threshold of ≥ 20 ng/mL.^[21] AUDIT-C scores of 0–3 in females and <4 in males were classified as low risk, 3–7 (females) or 4–7 (males) as intermediate risk, and ≥ 8 as high risk for harmful alcohol use. PEth concentrations <20 ng/mL were considered negative, indicating abstinence during the preceding 2–4 weeks.

PEth was measured using a commercially available competitive whole-blood enzyme immunoassay (AFG Scientific, IL, USA; Catalog No. EK240319), with a lower limit of detection of 0.5 ng/mL. Testing was performed at enrollment, 12 months, and whenever alcohol relapse was suspected, at no cost to participants. Sustained alcohol abstinence was defined as repeated PEth concentrations <20 ng/mL during follow-up, while intermittent relapse was defined as any PEth value ≥ 20 ng/mL after an initial negative result. All patients received integrated hepatology, addiction, and endocrinology care through a dedicated multidisciplinary liver clinic (Supplemental Information, <https://links.lww.com/HEP/K517>).

All patients received care in an integrated unit for hepatology, addiction care, and alcohol use disorder, and in an endocrinology clinic, both embedded within the Liver Clinic services, providing a single-window service (Supplemental Information, <https://links.lww.com/HEP/K517>).

Cardiometabolic risk factor management and integrated liver clinic

Metabolic dysfunction was assessed at baseline and follow-up, including body mass index (BMI), waist circumference, presence of T2DM, hypertension, dyslipidemia, and use of metabolic therapies. The patients' corrected BMI was assessed as required for dry-weight estimation. Dry weight was defined as the measured weight, with a 5%, 10%, and 15% subtraction for mild, moderate, and severe ascites, respectively. An additional 5% was subtracted if there was bilateral pedal edema.^[22] Dry body weight was confirmed by point-of-care ultrasound (POCUS) to determine the status of any ascites and minimal or absent subcutaneous edema, as per established criteria.^[23]

Patients received dietitian-supported outpatient medical nutrition therapy for targeted weight loss.^[24] Patients were also advised to engage in a combination of isotonic and isometric exercises in a graded, incremental program, with adaptations based on disease severity and functional status, to improve metabolic health. Adequate protein intake was

emphasized^[25] Glycemic control was optimized through self-monitoring of blood glucose, HbA1C testing, and supervision by an endocrinologist in the integrated hepatology-metabolic clinic. Medications for diabetes included metformin; sodium-glucose cotransporter-2 inhibitors (empagliflozin/dapagliflozin); dipeptidyl peptidase-4 inhibitors (linagliptin or sitagliptin); oral semaglutide; and insulin, either alone or in combination.^[26,27]

Statins (simvastatin or atorvastatin) and/or fenofibrate were prescribed for *dyslipidemia*. Carvedilol was the preferred non-selective beta blocker in this cohort, and other drugs for *hypertension* included ACE inhibitors and calcium channel blockers.

Patients with active alcohol use were offered integrated care at the AUD clinic, which included counseling, pharmacotherapy such as naltrexone and baclofen for reducing craving, and brief supervised courses of lorazepam for alcohol withdrawal^[28] (Supplemental Information, <https://links.lww.com/HEP/K517>).

Alcohol abstinence and CMRF intervention targets

While there is no validated definition of etiological control for MASLD or MetALD, prespecified interventions were evaluated to assess associations between disease modifiers and outcomes. As such, multidisciplinary management included AUDIT-C and Peth-confirmed alcohol abstinence (Peth <20 ng/mL), glycemic control, and dry-weight loss at 12 months.

Glycemic control was assessed in patients with diabetes, prediabetes, or impaired fasting glucose, with HbA1c goals of ~6%–7%. Dry body weight-loss targets of >7% were based on the thresholds proposed by Vilar-Gomez et al^[29] and were categorized as <7%, 7%–10%, and ≥10% dry-weight reduction. Alcohol abstinence, glycemic control, and weight-loss thresholds were prespecified markers of interventions that were subsequently evaluated for their association with recompensation as per Baveno VII criteria.

Statistical analysis

Categorical variables are presented as frequencies and percentages, and continuous variables as mean ± SD or median (IQR), as appropriate. Group comparisons were performed using the Student *t* test or one-way analysis of variance (ANOVA) for continuous variables and the χ^2 or the Fisher exact test for categorical variables. Time to observed recompensation was determined at scheduled study visits and analyzed within a competing-risks framework, treating death and liver transplantation as competing events. Baseline

predictors of recompensation were evaluated using cause-specific Cox proportional hazards regression, with clinically relevant variables prespecified a priori based on biological plausibility, including alcohol exposure, cardiometabolic risk factors, decompensation burden, and liver disease severity. Fine-Gray competing-risk regression was performed as a sensitivity analysis, and cumulative incidence functions (CIFs) were generated from the fitted models.

To evaluate treatment-response variables measured at 12 months (dry-weight loss, Peth-confirmed alcohol abstinence, and glycemic control), landmark Fine-Gray competing-risk analyses were performed among patients alive and transplant-free at the 12-month landmark, with follow-up commencing at 12 months and adjustment for baseline MELD-Na.

For mortality analyses, death was treated as the event of interest and liver transplantation as the competing event. Separate Fine-Gray models were fitted for baseline variables and 12-month landmark variables. Because recompensation is a time-dependent event, its association with mortality was evaluated using cause-specific Cox regression with recompensation modeled as a time-dependent covariate; Simon-Makuch curves were generated for descriptive purposes.

Adjusted models were prespecified and limited to baseline MELD-Na plus one primary exposure of interest to minimize overfitting and collinearity. No imputation of missing data was performed. Baseline analyses were conducted using available-case data. For landmark analyses, only patients alive and transplant-free at the 12-month landmark with available measurements for the exposure of interest were included. Survival time was administratively censored at 48 months. A two-sided *p*-value <0.05 was considered statistically significant. Descriptive analyses were performed using SPSS version 22.0 (IBM Corp., Chicago, IL, USA), and survival and competing-risk analyses using Stata/SE version 16.1 (StataCorp LLC, College Station, TX, USA).

RESULTS

Between October 2019 and March 2025, 1342 patients with decompensated cirrhosis attending the Liver Clinic at PGIMER, Chandigarh, India, were screened for inclusion in a prospective cohort study. After applying the predefined eligibility criteria (Figure 1), 344 patients with MASLD (*n* = 158, 45.9%) or MetALD (*n* = 186, 54.1%) were included. (MASLD patients were older (48.3 ± 13.1 vs. 45.7 ± 10.2 years; *p* = 0.039), whereas MetALD predominantly affected men (99.4% vs. 63.9%; *p* < 0.001). Although both groups had cardiometabolic risk factors, left ventricular diastolic dysfunction (41.7% vs. 19.9%; *p* < 0.001), higher BMI, and overweight/

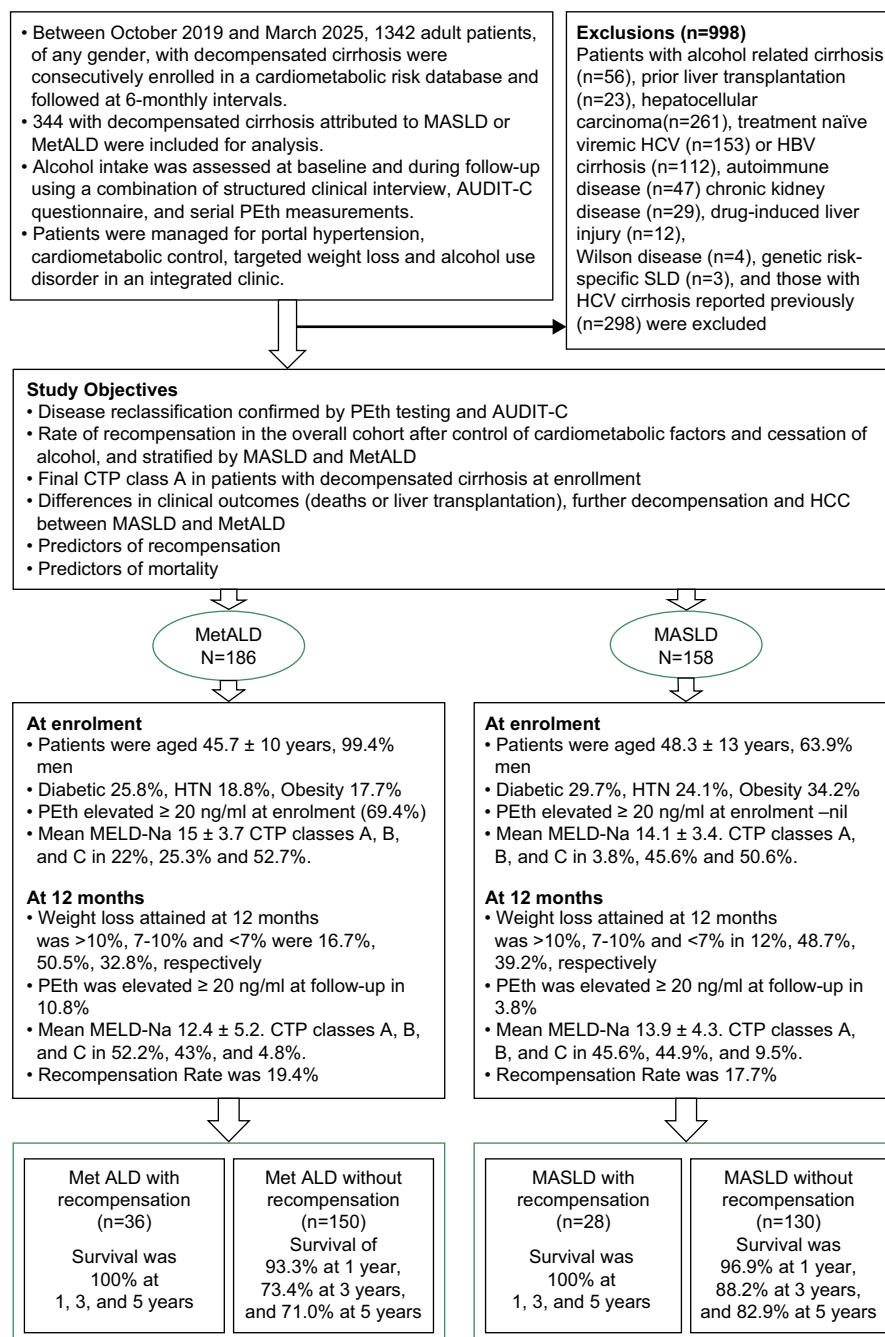


FIGURE 1 Enrollment of the cohort, objectives, and outcomes. Abbreviations: ALD, alcohol-associated liver disease; AUDIT-C, Alcohol Use Disorders Identification Test—Consumption; CTP, Child–Turcotte–Pugh Score; HBV, hepatitis B virus; HCC, hepatocellular carcinoma; HCV, hepatitis C virus; HTN, hypertension; MASLD, metabolic dysfunction–associated steatotic liver disease; MELD-Na, Model for End-Stage Liver Disease—Sodium; MetALD, metabolic dysfunction–associated steatotic liver disease with alcohol consumption; PETH, phosphatidylethanol; SLD, steatotic liver disease.

obesity were more common in MASLD (Table 1). The overall burden of cardiometabolic risk factors was similar between groups ($p=0.251$), although MASLD patients had ≥ 2 risk factors more frequently (Supplemental Table S1, <https://links.lww.com/HEP/K517>).

Ascites requiring diuretic therapy was reported in all patients per history, although clinical ascites was

evident in 86% at enrollment. Overall, 31.1%, 48.5%, and 20.3% of patients had 1, 2, and ≥ 3 decompensation events, respectively. MetALD patients had a greater decompensation burden than MASLD (≥ 3 events: 25.8% vs. 13.9%; $p<0.001$) and more frequently presented with AVB (68% vs. 45%), SBP (37% vs. 16%), large esophageal varices (40% vs.

TABLE 1 Baseline characteristics (demographic, clinical, biochemical, liver disease severity, decompensation events, and therapeutics) of patients with MASLD and MetALD at enrollment

Parameter	Total N = 344	MASLD N = 158	MetALD N = 186	p ^a
Age (y)	46.9 ± 11.7	48.3 ± 13.1	45.7 ± 10.2	0.039
Sex (men)	286 (83.1)	101 (63.9)	185 (99.4)	<0.001
Comorbid ailments				
Diabetes mellitus, n (%)	95 (27.6)	47 (29.7)	48 (25.8)	0.415
Hypertension, n (%)	73 (21.2)	38 (24.1)	35 (18.8)	0.237
Thyroid disorders, n (%)	22 (6.4)	14 (8.9)	8 (4.3)	0.085
Left ventricular diastolic dysfunction, n (%)	103 (29.9)	66 (41.7)	37 (19.9)	<0.001
Coronary artery disease, n (%)	3 (0.9)	2 (1.3)	1 (0.5)	0.469
Chronic kidney disease (not on dialysis)	21 (6.1)	7 (4.4)	14 (7.5)	0.232
Hepatitis C virus infection (post-SVR), n (%)	47 (13.6)	31 (19.6)	16 (8.6)	0.003
Hepatitis B virus (on antivirals), n (%)	10 (2.9)	6 (3.8)	4 (2.1)	0.364
Anthropometry				
Height (cm)	168.6 ± 7.3	166.1 ± 8.1	170.7 ± 5.8	<0.001
Waist circumference (cm) ^b	97.5 (95.3–100.1)	96.5 (92.7–100.1)		0.377
Wet weight (kg)	70.7 ± 12.7	72.6 ± 13.2	69.2 ± 12.1	0.015
Dry weight (kg) ^c	68.1 ± 12.6	70 ± 13.2	66.3 ± 11.8	0.009
BMI (kg/m ²)	24 ± 4.6	25.4 ± 4.9	22.8 ± 4	<0.001
Overweight, n (%)	202 (58.7)	109 (68.9)	93 (50)	<0.001
Obese, n (%)	87 (25.3)	54 (34.2)	33 (17.7)	<0.001
Decompensation events				
Median time from first decompensation to enrollment in protocol (days)	15 (5–25)	15(10–25)	16(5–25)	0.400
Current or history of clinically apparent ascites necessitating diuretics	344 (100%)	158 (100%)	186 (100%)	—
Clinical ascites at enrollment, n (%)	297 (86.3)	136 (86.1)	161 (86.6)	0.897
Hepatic encephalopathy, n (%)	127 (36.9)	38 (24)	89 (47.8)	<0.001
Acute variceal bleed, n (%)	197 (57.3)	71 (44.9)	126 (67.7)	<0.001
Spontaneous bacterial peritonitis, n (%)	94 (27.3)	25 (15.8)	69 (37.1)	<0.001
Acute kidney injury	25 (7.3)	7 (4.4)	18 (9.7)	0.062
Variceal status at enrollment				<0.001
Small esophageal varices (EV) ± PHG	176 (51.2)	109 (69)	67 (36)	
Large EV ± PHG	93 (27)	18 (11.4)	75 (40.3)	
Gastric varices ± EV ± PHG	45 (13.1)	23 (14.5)	22 (11.8)	
PHG alone without EV	20 (5.8)	5 (3.2)	15 (8.1)	
Eradicated varices	10 (2.9)	3 (1.9)	7 (3.8)	
Severity scores				
CTP score at enrollment	9.2 ± 1.9	9.4 ± 1.7	9.1 ± 2.2	0.145
MELD-Na	14.7 ± 3.6	14.1 ± 3.4	15 ± 3.7	0.013
MELD-Na category (baseline), n (%)				0.016
10–15	231 (67.1)	117 (74.1)	114 (61.3)	
16–20	85 (24.7)	34 (21.5)	51 (27.4)	
> 21	28 (8.1)	7 (4.4)	21 (11.3)	
Child–Turcotte–Pugh class at enrollment				<0.001
A	47 (13.7)	6 (3.8)	41 (22)	
B	119 (34.6)	72 (45.6)	47 (25.3)	
C	178 (51.7)	80 (50.6)	98 (52.7)	
Liver stiffness measurement (LSM) 2D SWE, kPa ^d	27.8 ± 11.4	27.9 ± 11.7	27.7 ± 11.2	0.832

TABLE 1 (continued)

Parameter	Total N = 344	MASLD N = 158	MetALD N = 186	p ^a
Non-selective beta blockers (NSBB)				
Carvedilol started at enrollment and continued for the duration of the study ^e	328 (95.3)	151 (95.6)	177 (95.2)	0.858
Propranolol ongoing at enrollment ^f	16 (4.7)	7 (4.4)	9 (4.8)	0.857
NSBB intolerance	18 (5.2)	7 (4.4)	11 (5.9)	0.538
Management of decompensation				
Diuretics started at enrollment	302 (87.8)	152 (96.2)	150 (80.6)	<0.001
Lactulose started at enrollment (as primary or secondary prophylaxis)	281 (81.7%)	118 (74.7)	163 (87.6)	0.002
Rifaximin	164 (47.7)	57 (37.6)	107 (57.5)	<0.001
Medications for dyslipidemia				
Statins	45 (13.1)	29 (18.4)	16 (8.6)	0.010
Simvastatin	25 (55.6)	18 (62.1)	7 (43.8)	
Atorvastatin	13 (28.9)	9 (31)	4 (25)	
Rosuvastatin	7 (15.6)	2 (6.9)	5 (31.3)	
Fenofibrate	15 (4.4)	7 (4.4)	8 (4.3)	0.579
Additional medications for hypertension ^g	n = 73	n = 13	n = 62	
Telmisartan	23 (31.5)	7 (53.8)	16 (25.8)	0.135
Amlodipine	7 (9.6)	5 (38.5)	2 (3.2)	0.254
Medications for diabetes Prescription at enrollment ^h	n = 95	n = 47	n = 48	0.903
Metformin	89 (93.7)	44 (93.6)	45 (93.8)	0.978
Linagliptin	42 (44.2)	21 (44.7)	21 (43.8)	0.982
Empagliflozin	23 (24.2)	7 (14.9)	16 (33.3)	0.035
Dapagliflozin	7 (7.4)	5 (10.6)	2 (4.2)	0.250
Oral semaglutide	2 (2.1)	1 (2.1)	1 (2.1)	0.987
Insulin	18 (18.9)	9 (19.1)	9 (18.8)	0.960

^ap-values in bold indicate significance at <0.05.

^bMedian with interquartile range.

^cDry weight was defined as measured weight with subtraction based on the severity of ascites: 5%, 10%, and 15% for mild, moderate, and severe ascites, respectively. An additional 5% was subtracted if there was bilateral pedal edema. Weight is reported as "dry" unless otherwise specified. Dry body weight was confirmed by point-of-care ultrasound (POCUS) to determine the status of any ascites and minimal or absent subcutaneous edema, as per established criteria.

^dLSM was measured by ElastPQ 2D shear wave elastography in patients at baseline and at follow-up (Philips Affiniti 70). Follow-up LSM was also measured using transient elastography (FibroScan, Echosens) and reported separately.

^eCarvedilol was the preferred non-selective beta blocker.

^fPatients who were well controlled on propranolol were continued on the same regimen.

^gMedications for hypertension were prescribed if needed, besides non-selective beta blockers, for the maintenance of blood pressure, and monitored using automated devices.

^hPatients received ≥ 1 diabetes medication. Self-monitoring of blood glucose was advised for all subjects.

Abbreviations: 2D SWE, 2-dimensional shear wave elastography; ALT, alanine transaminase; APRI, AST-to-platelet ratio index; AST, aspartate transaminase; BMI, body mass index; CTP, Child–Turcotte–Pugh; EV, esophageal varices; Fib-4, fibrosis-4 index; LSM, liver stiffness measurement; MASLD, metabolic dysfunction–associated steatotic liver disease; MELD, Model for End-stage Liver Disease; MetALD, metabolic dysfunction–associated steatotic liver disease with alcohol consumption; NSBB, non-selective beta blocker; PHG, portal hypertensive gastropathy; POCUS, point-of-care ultrasound; SVR, sustained virologic response; TIPS, transjugular intrahepatic portosystemic shunt.

11%), and hepatic encephalopathy (48% vs. 24%) (all $p < 0.001$).

Mean MELD-Na was higher in MetALD than MASLD (15.0 ± 3.7 vs. 14.1 ± 3.4 ; $p = 0.013$), whereas overall CTP scores were similar despite differences in class distribution ($p < 0.001$). All patients classified as CTP class A had presented with AVB at enrollment (Supplemental Tables S1 and S2, <https://links.lww.com/HEP/K517>). The median interval from first decompensation to enrollment was 15 days (IQR 5–25).

Alcohol abstinence and PEth-confirmed outcomes

Alcohol exposure differed markedly between MASLD and MetALD. Any alcohol consumption was reported in 54.1% of the cohort, occurring predominantly in MetALD (93.0% vs. 8.2% in MASLD; $p = 0.001$), while intermittent alcohol relapse and tobacco use were observed almost exclusively in MetALD (11.8% and 13.4% vs. 1.3%, respectively; $p = 0.001$ for both) (Supplemental Table S3, <https://links.lww.com/HEP/K517>). AUDIT-C scores were

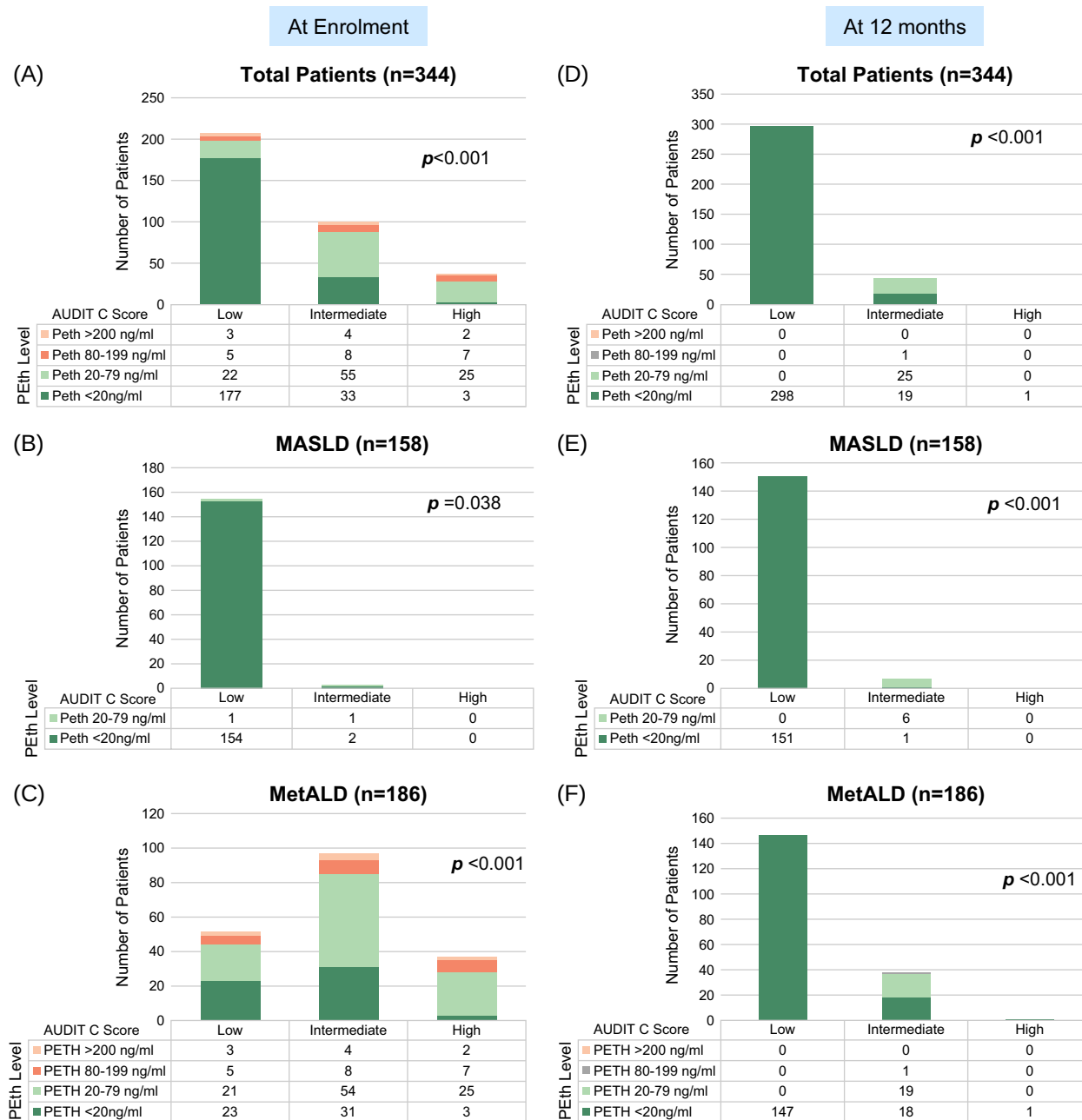


FIGURE 2 Classification of patients with SLD (n = 344) based on AUDIT-C and PETH testing at enrollment (A). Reclassification of patients with SLD as MASLD (B) and MetALD (C) based on PETH test and AUDIT-C testing at enrollment. Panels D, E, and F show the changes in AUDIT-C and PETH test results at 12-month follow-up in the same groups: full cohort, MASLD, and MetALD, respectively. Abbreviations: AUDIT-C, Alcohol Use Disorders Identification Test—Consumption; MASLD, metabolic dysfunction–associated steatotic liver disease; MetALD, metabolic dysfunction–associated steatotic liver disease with alcohol consumption; PETH, phosphatidylethanol; SLD, steatotic liver disease.

higher in MetALD (4.7 vs. 3.0; $p = 0.001$), with intermediate-risk and high-risk categories predominating, whereas 98.1% of MASLD patients were classified as low risk ($p < 0.001$).

PETH testing corroborated self-reported alcohol exposure (Figure 2). At enrollment, 98.7% of MASLD patients had PETH < 20 ng/mL, compared with 30.6% of MetALD patients, whereas higher PETH categories (20–79, 80–199, and > 200 ng/mL) were observed

almost exclusively in MetALD. Two patients initially classified as MASLD demonstrated transiently positive PETH that was not confirmed on repeat testing. Overall, baseline PETH positivity was observed only in MetALD patients (69.4%; $p = 0.001$). At follow-up, most patients reported cessation of alcohol consumption, reflected by low AUDIT-C scores and a low prevalence of positive PETH tests. However, PETH positivity was more frequent in MetALD than in MASLD patients (10.8% vs. 3.8%;

$p=0.017$) (Supplemental Table S3, <https://links.lww.com/HEP/K517>).

Recompensation as per Baveno VII criteria

During follow-up, 64 patients (18.6%) achieved recompensation according to the Baveno VII criteria, with similar rates in MASLD and MetALD (17.7% vs. 19.4%; $p=0.698$). The first documented observation of recompensation occurred at a median of 15 months (IQR 14.5–15.5) after enrollment, reflecting the scheduled follow-up assessment intervals during a median follow-up of 3.9 years (IQR 2.8–4.0). At 12 months, MetALD patients achieved marginally greater weight loss than MASLD patients (−8.0% vs. −7.6%; $p=0.045$) (Table 2).

Baseline demographic characteristics and alcohol exposure were similar between patients who did and did not achieve recompensation. Among patients receiving diuretics at enrollment, 46.4% discontinued therapy during follow-up (MASLD: 39.4% vs. MetALD: 53.5%; $p=0.015$). Ascites recurrence was more frequent in MASLD, whereas hospital readmissions were more common in MetALD (Table 2). Recompensated patients showed greater improvement in liver disease severity and cardiometabolic risk modification, with lower follow-up CTP and MELD-Na scores (Supplemental Tables S4–S6, <https://links.lww.com/HEP/K517>).

During follow-up, 8 patients underwent TIPS (4 for refractory ascites, 3 for refractory variceal bleeding, and 1 for portal vein recanalization); 5 (1.5%) developed HCC, 1 underwent liver transplantation, and 4 died. Only 1 patient who achieved recompensation experienced re-decompensation, with transient ascites following an alcohol binge that subsequently resolved (Table 2).

Cardiometabolic profile at enrollment and follow-up

Cardiometabolic profiles varied by etiology. Patients who subsequently recompensated had a higher prevalence of T2DM (40.6% vs. 24.6%; $p=0.013$).

Patients who subsequently achieved recompensation had a higher prevalence of type 2 diabetes mellitus at enrollment than those who did not (40.6% vs. 24.6%; $p=0.013$) (Table 1). Overall, HbA1c values were similar between MASLD and MetALD, but were lower at 12 months among recompensated patients ($p=0.008$) (Supplemental Table S5, <https://links.lww.com/HEP/K517>). Antidiabetic therapies prescribed at enrollment included metformin ($n=89$), linagliptin ($n=42$), empagliflozin ($n=23$), dapagliflozin ($n=7$), and oral semaglutide ($n=2$) (Table 1).

Weight loss was strongly associated with recompensation. Patients who recompensated more frequently achieved >10% dry-weight loss (31.3% vs. 10.7%), whereas <7% dry-weight loss was more common among those without recompensation (10.9% vs. 41.4%; $p<0.001$). Overall weight loss at 12 months was also greater in the recompensated group (−8.7% vs. −7.6%; $p<0.001$). MetALD patients achieved marginally greater weight loss than MASLD patients (−8.0% vs. −7.6%; $p=0.045$) (Supplemental Table S6, <https://links.lww.com/HEP/K517>).

MASLD patients had higher total, HDL, and LDL cholesterol levels and were more frequently treated with statins, whereas recompensated patients had higher baseline HDL cholesterol and lower triglyceride levels at 6 months (Supplemental Table S5, <https://links.lww.com/HEP/K517>, and Figure 3). Sankey plots illustrating alcohol exposure, PEth status, and weight-loss trajectories are shown in Supplemental Figures S1 and S2, <https://links.lww.com/HEP/K517>.

Attainment of the Child–Turcotte–Pugh class A

At follow-up, 169 patients (49.1%) were CTP class A at a median of 5 months, while continuing diuretics or hepatic encephalopathy prophylaxis. Rates were similar between MASLD (42.9%) and MetALD (57.4%) (Supplemental Table S7, <https://links.lww.com/HEP/K517>). Those who reached CTP-A had fewer episodes of ascites and encephalopathy, lower baseline MELD-Na, and lower PEth positivity.

Clinical course and changes in CMRF control, stratified by MELD-Na categories (≤ 15 , 16–20, ≥ 21) at baseline, are presented in Supplemental Table S8, <https://links.lww.com/HEP/K517>.

Predictors of recompensation as per Baveno VII consensus

Baseline predictors of recompensation were evaluated using cause-specific Cox regression with death and liver transplantation treated as competing events, with Fine–Gray competing-risk regression performed as a sensitivity analysis (Table 3). The 2 approaches yielded concordant results. Diabetes mellitus at enrollment (adjusted csHR 1.84, 95% CI: 1.14–2.97; adjusted sHR 1.89, 95% CI: 1.14–3.15) and the absence of large varices (adjusted csHR 1.68, 95% CI: 1.43–2.00; adjusted sHR 1.97, 95% CI: 1.22–4.00) were independently associated with a higher likelihood of recompensation. Conversely, patients with ≥ 3 decompensation events at enrollment (csHR 0.37, 95% CI: 0.14–0.90) and those with 2 cardiometabolic risk factors (csHR 0.54, 95% CI: 0.32–0.89) were less

TABLE 2 Changes following multidisciplinary management (clinical, biochemical, liver disease severity, decompensation events, and therapeutics) in the cohort with MASLD spectrum

Parameter	Total N = 344	MASLD N = 158	MetALD N = 186	p ^a
Change in weight				
Dry weight 6 months (kg)	65 ± 11.2	65.8 ± 12.5	64.2 ± 9.7	0.445
Dry weight 12 months (kg)	63.2 ± 12.7	65 ± 13.2	61.6 ± 12.1	0.015
% Weight loss at 12 months ^b	-7.8 (-7.9 to -7.6)	-7.6 (-7.9 to -7.4)	-8 (-8.2 to -7.7)	0.045
Dry-weight loss at 12 months				0.314
> 10%	50 (14.5)	19 (12)	31 (16.7)	
7%–10%	171 (49.7)	77 (48.7)	94 (50.5)	
< 7%	123 (35.8)	62 (39.2)	61 (32.8)	
BMI (follow-up at 12 months)	22.2 ± 4.6	23.6 ± 4.8	21.1 ± 4	<0.001
Overweight at follow-up at 12 months, n (%)	200 (58.1)	107 (67.7)	93 (50)	0.001
Obese at follow-up at 12 months, n (%)	51 (14.8)	37 (23.4)	14 (7.5)	<0.001
Decompensation events at follow-up				
Episode of ascites, n (%)	131 (38.1)	75 (47.4)	56 (30.1)	0.001
Episode of hepatic encephalopathy, n (%)	23 (6.7)	11 (7)	12 (6.4)	0.840
Episode of AVB, n (%)	18 (5.2)	15 (9.5)	3 (1.6)	0.001
Spontaneous bacterial peritonitis, n (%)	19 (5.5)	13 (8.2)	6 (3.2)	0.043
Acute kidney injury, n(%)	12 (3.4)	5 (3.1)	7 (3.7)	0.750
Variceal status at 12 months follow-up				<0.001
Small varices ± PHG	111 (32.3)	52 (32.9)	59 (31.7)	
Eradicated varices ± PHG	175 (50.9)	69 (43.7)	106 (57)	
PHG alone	20 (5.8)	5 (3.2)	15 (8.1)	
No varices	38 (11)	32 (20.2)	6 (3.2)	
Severity scores				
CTP score at 12 months	7.3 ± 1.5	7.3 ± 1.5	7.3 ± 1.5	0.802
MELD-Na at 12 months	13 ± 4.9	13.9 ± 4.3	12.4 ± 5.2	0.003
MELD-Na category (follow-up), n (%)				<0.001
< 10	158 (45.9)	52 (32.9)	106 (57)	
10–15	99 (28.8)	54 (34.2)	45 (24.2)	
16–20	52 (15.1)	37 (23.4)	15 (8.1)	
> 21	35 (10.2)	15 (9.5)	20 (10.8)	
CTP (follow-up)				0.176
A	169 (49.1)	72 (45.6)	97 (52.2)	
B	151 (43.9)	71 (44.9)	80 (43)	
C	24 (7)	15 (9.5)	9 (4.8)	
Liver stiffness measurement at follow-up				
LSM, 2D SWE, kPa	16.9 ± 11.5	16.9 ± 11.8	16.9 ± 11.2	0.949
LSM at follow-up (kPa, FibroScan)	23.5 ± 10	24.5 ± 10	22 ± 10	0.996
Management of portal hypertension and complications				
Non-selective beta-blocker intolerance ^c	18 (5.2)	7 (4.4)	11 (5.9)	0.538
Intermittent diuretics taken during the study ^d	42 (12.2)	6 (3.8)	36 (19.4)	0.001
Stopped diuretics on follow-up	140/302 (46.4)	60/152 (39.4)	80/150 (53.5)	0.015
Remained off diuretics for ≥ 1 year	64 (18.6)	28 (17.7)	36 (19.4)	0.698
Stopped lactulose	207/281 (73.7%)	87/118 (73.7)	120/163 (73.6)	0.983
Remained off lactulose for ≥ 1 year ^e	64 (18.6)	28 (17.7)	36 (19.4)	0.698
Stopped rifaximin	108/164 (65.4)	38/57 (66.7)	70/107 (65.4)	0.872
Medications for diabetes prescription at 12 months ^f				
Metformin	n = 91 75 (82.4%)	n = 45 44 (97.8)	n = 46 45 (97.8)	0.784 0.987

TABLE 2 (continued)

Parameter	Total N = 344	MASLD N = 158	MetALD N = 186	p ^a
Linagliptin	46 (50.5)	26 (57.8)	20 (43.5)	0.172
Empagliflozin	45 (49.4)	25 (55.6)	20 (43.5)	0.249
Dapagliflozin	7 (7.7)	5 (11.1)	2 (4.3)	0.226
Oral semaglutide	5 (5.5)	3 (6.7)	2 (4.3)	0.645
Insulin	12 (13.2)	7 (15.6)	5 (10.9)	0.508
Outcomes	N = 344	N = 158	N = 186	
Attained CTP-A status, n (%)	169 (49.1)	72 (45.6)	97 (52.2)	0.060
Recompensated as per Baveno VII, n (%)	64 (18.6)	28 (17.7)	36 (19.4)	0.698
Median duration of follow-up (y)	3.9 (2.8–4)	3.9 (3–4.3)	3.6 (2.4–4)	0.766
Median time to observed recompensation (mo) ^b	15 (14.5–15.5)	15.5 (15–16)	15 (14.5–16.5)	0.262
Readmissions, n (%)	90 (26.2)	28 (17.7)	62 (33.3)	0.003
Deaths, n (%)	61 (17.7)	19 (12)	42 (22.6)	0.011
TIPS placement, n (%)	8 (2.3)	6 (3.8)	2 (1.1)	0.095
Liver transplantation, n (%)	11 (3.2)	3 (1.9)	8 (4.3)	0.207
Hepatocellular carcinoma, n (%)	5 (1.5)	3 (1.9)	2 (1.1)	0.525

^ap-values in bold indicate significance at <0.05.

^bMedian with interquartile range.

^cPatients with NSBB intolerance were tapered off the medication.

^dDiuretics may have been continued intermittently for ascites and pedal edema, and such subjects were not counted as recompensated per Baveno VII.

^eLactulose was also given for constipation and minimal hepatic encephalopathy in patients without overt hepatic encephalopathy. However, patients on even intermittent lactulose were not counted as recompensated per Baveno VII.

^fPatients received ≥ 1 medication for the management of diabetes. Self-monitoring of blood glucose was advised for all subjects. In all, 4 patients with diabetes either died or were lost to follow-up at 12 months.

Abbreviations: AVB, acute variceal bleed; BMI, body mass index; CTP, Child–Turcotte–Pugh; LSM, liver stiffness measurement; MASLD, metabolic dysfunction–associated steatotic liver disease; MELD, Model for End-stage Liver Disease; MELD–Na, Model for End-stage Liver Disease–Sodium; MetALD, metabolic dysfunction–associated steatotic liver disease with alcohol consumption; NSBB, non-selective beta blocker; PHG, portal hypertensive gastropathy; SWE, shear wave elastography; TIPS, transjugular intrahepatic portosystemic shunt.

likely to achieve recompensation. Baseline etiology, age, sex, BMI, PEth positivity, hypertension, and liver stiffness were not associated with recompensation.

The CIF curves for predictors of recompensation, based on the number of CMRF and the decompensation burden at enrollment, are shown in Supplemental Figure S3, <https://links.lww.com/HEP/K517>.

Predictors measured at 12 months were evaluated using a landmark Fine–Gray competing-risk analysis adjusted for baseline MELD–Na. Compared with <7% dry-weight loss, weight loss of 7%–10% (sHR 4.06, 95% CI: 1.84–8.97; $p=0.001$) and ≥ 10% (sHR 7.42, 95% CI: 3.15–17.47; $p<0.001$) were associated with progressively higher probabilities of recompensation (Supplemental Table S9, <https://links.lww.com/HEP/K517>). Alcohol abstinence (sHR 1.87, 95% CI: 1.29–3.32; $p=0.003$) and achievement of glycemic control (sHR 1.43, 95% CI: 1.03–2.79; $p=0.038$) were also independently associated with recompensation (Figure 4).

Among the 329 patients alive and transplant-free at the 12-month landmark, exploratory analyses demonstrated a graded association between the number of prespecified intervention targets achieved (PEth–

confirmed alcohol abstinence, good glycemic control, and ≥ 7% dry-weight loss) and recompensation. Recompensation occurred in 0/38 (0%) patients achieving ≤ 1 target, 19/133 (14.3%) achieving 2 targets, and 45/158 (28.5%) achieving all 3 targets (p for trend <0.001), with similar patterns in MASLD and MetALD (Supplemental Tables S10 and S11, <https://links.lww.com/HEP/K517>).

Among patients with diabetes ($n=95$), baseline glycemic control was not associated with recompensation, whereas achievement of glycemic control at 12 months was independently associated with recompensation (adjusted sHR 4.41, 95% CI: 1.94–7.19; $p<0.001$) (Supplemental Table S12, <https://links.lww.com/HEP/K517>).

Predictors of mortality

During a median follow-up of 3.5 years, 61 patients (17.7%) died. The causes of death were predominantly liver failure, sepsis, or hepatocellular carcinoma (Supplemental Table S13, <https://links.lww.com/HEP/K517>). A higher baseline MELD–Na score was independently associated with an increased risk of death (HR 1.08 per

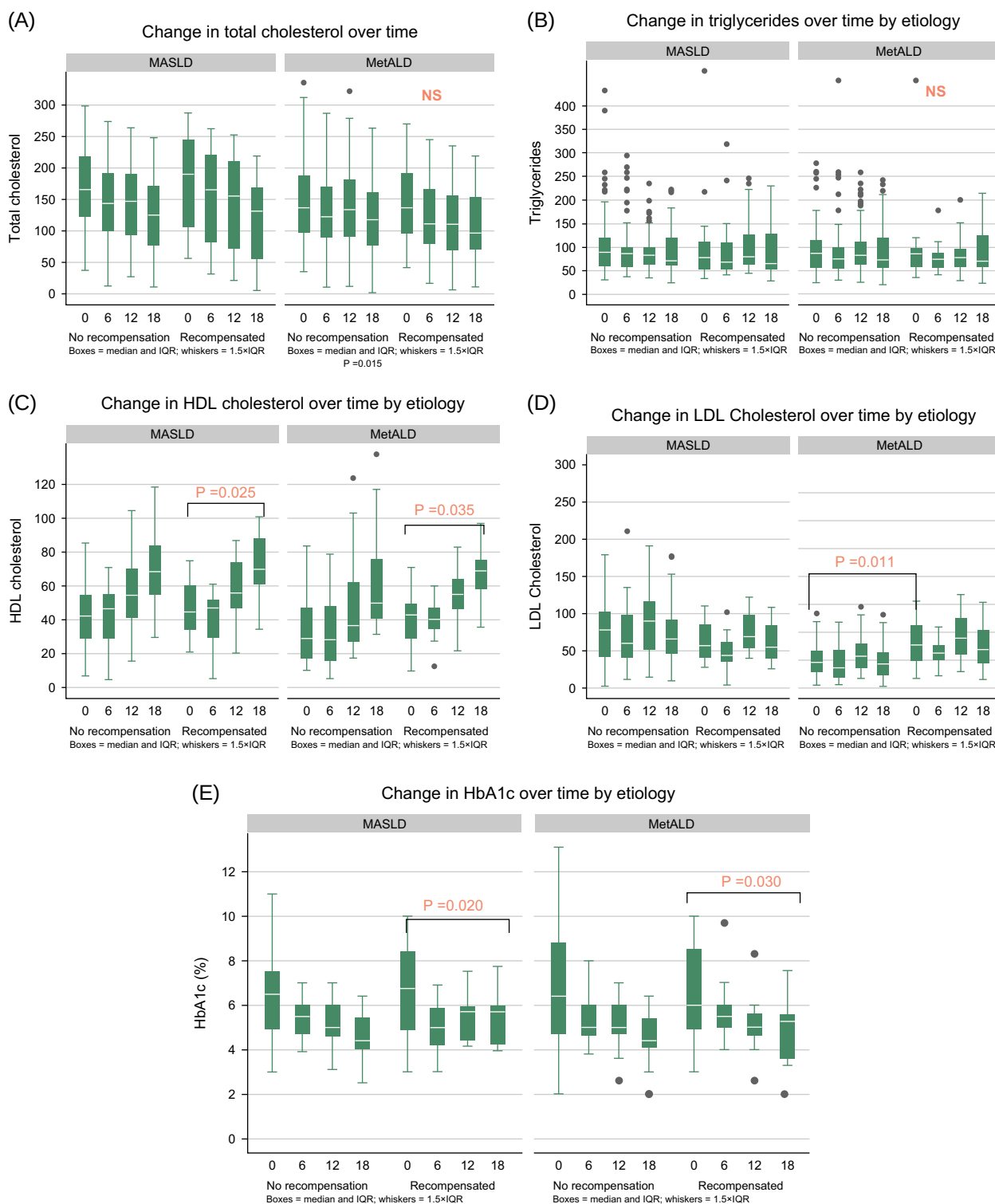


FIGURE 3 Changes in cardiometabolic risk factors over time stratified by etiology (MASLD and MetALD) and recompensation status. Box-and-whisker plots show the median and interquartile range at baseline and at 6, 12, and 18 months to illustrate cardiometabolic intervention targets. Whiskers represent 1.5× the interquartile range. Abbreviations: HDL, high-density lipoprotein; IQR, interquartile range; LDL, low-density lipoprotein; MASLD, metabolic dysfunction–associated steatotic liver disease; MetALD, metabolic dysfunction–associated steatotic liver disease with alcohol consumption.

point, 95% CI: 1.03–1.14; $p=0.001$). In competing-risk regression adjusted for baseline MELD-Na, weight loss was not significantly associated with the cumulative

incidence of death (Figure 5). Among the 64 patients who achieved recompensation, median follow-up after recompensation was 2.13 years (IQR: 1.57–2.75),

TABLE 3 Baseline predictors of recompensation using cause-specific Cox regression (primary analysis) and Fine–Gray competing-risk regression (sensitivity analysis), adjusted for baseline MELD-Na

Parameters	Cause-specific hazard ratio (csHR)	95% confidence interval (CI)	<i>p</i>	Subdistribution hazard ratio (sHR)	95% confidence interval (CI)	<i>p</i> ^a
Etiology						
MASLD	Reference			Reference		
MetALD	1.22	0.75–1.99	0.423	1.19	0.85–1.67	0.317
Age	1.01	0.99–1.03	0.194	1.01	1.00–1.02	0.167
Male sex	1.04	0.57–1.90	0.891	1.02	0.68–1.54	0.913
PEth ≥ 20 ng/mL at enrollment	1.06	0.75–1.35	0.813	1.04	0.63–1.71	0.876
BMI	0.98	0.93–1.03	0.464	0.99	0.94–1.04	0.580
Diabetes mellitus at enrollment	1.82	1.12–2.93	0.015	1.69	1.20–2.36	0.002
Hypertension	0.92	0.51–1.66	0.786	0.93	0.62–1.39	0.717
Absence of large varices	1.32	1.13–1.79	0.033	1.91	1.77–3.09	0.028
Baseline LSM (2D SWE) ^b	1.00	0.98–1.02	0.864	1.00	0.98–1.01	0.826
Number of decompensation events at enrollment						
1 event	Reference			Reference		
2 events	0.98	0.64–1.42	0.767	0.94	0.57–1.58	0.828
≥ 3 events	0.37	0.14–0.90	0.014	0.31	0.12–0.81	0.016
Number of CMRF at enrollment						
1 CMRF	Reference			Reference		
2 CMRF	0.54	0.32–0.89	0.017	0.51	0.31–0.86	0.012
≥ 3 CMRF	0.71	0.32–1.56	0.392	0.67	0.30–1.47	0.316
Multivariable adjusted csHR	Adjusted csHR	95% CI	<i>p</i>	Adjusted sHR	95% CI	<i>p</i>
Model 1 ^c (n = 344)						
Diabetes mellitus	1.84	1.14–2.97	0.013	1.89	1.14–3.15	0.015
Absence of large varices	1.68	1.43–2.00	0.027	1.97	1.22–4.00	0.021

Note: All models were adjusted for baseline MELD-Na.

^a*p*-values in bold indicate significance at <0.05 .

^bLSM was measured using ElastPQ 2D shear wave elastography at baseline and follow-up.

^cBecause only 64 recompensation events occurred, parsimonious models were prespecified, with baseline MELD-Na included a priori and selected clinically relevant predictors evaluated individually to minimize overfitting.

Abbreviations: 2D SWE, two-dimensional shear wave elastography; BMI, body mass index; CI, confidence interval; CMRF, cardiometabolic risk factor; csHR, cause-specific hazard ratio; LSM, liver stiffness measurement; MASLD, metabolic dysfunction–associated steatotic liver disease; MELD-Na, Model for End-Stage Liver Disease–Sodium; MetALD, metabolic dysfunction–associated steatotic liver disease with alcohol consumption; PEth, phosphatidylethanol; sHR, subdistribution hazard ratio.

corresponding to 137.9 person-years of post-recompensation observation, during which no deaths were noted.

Because no deaths occurred after recompensation, the hazard ratio for recompensation could not be reliably estimated in the time-dependent model (Figure 5A). In competing-risk regression adjusted for baseline MELD-Na and accounting for liver transplantation as a competing event, MetALD etiology was associated with a significantly higher cumulative incidence of death compared with MASLD (sHR 1.87, 95% CI: 1.09–3.22; $p = 0.023$) (Figure 5B).

At the 12-month landmark, 92.4% of patients had PEth <20 ng/mL, while 7.6% had detectable PEth ≥ 20 ng/mL. In Fine–Gray competing-risk regression adjusted for baseline MELD-Na, detectable PEth was strongly associated with increased mortality (sHR 5.27, 95% CI: 2.85–9.74; $p < 0.001$). Compared with patients achieving $<7\%$ weight loss, those with 7%–10% weight loss (sHR 1.52, 95% CI: 0.65–3.56; $p = 0.631$) and those with $\geq 10\%$ weight loss (sHR 1.45, 95% CI: 0.76–2.76; $p = 0.944$) did not demonstrate a significantly different risk of death (Supplemental Table S14, <https://links.lww.com/HEP/K517>).

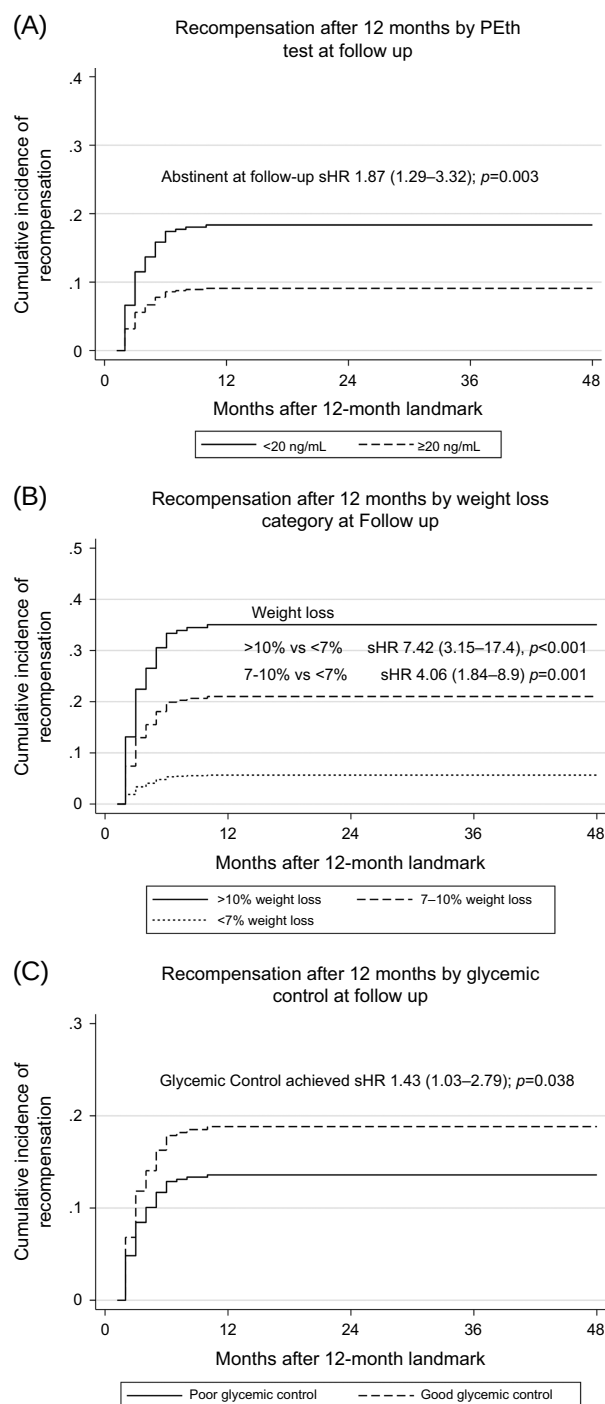


FIGURE 4 Adjusted cumulative incidence functions for recompensation derived from Fine–Gray competing-risk regression using a 12-month landmark approach, with death treated as a competing event and adjustment for baseline MELD-Na. Panels show results by alcohol abstinence (A), weight-loss category (B), and glycemic control (C). Of 344 enrolled patients, 329 were alive and transplant-free at the 12-month landmark and were eligible for analysis. Complete data were available for the weight-loss category, PEth testing, and glycemic control assessment in all eligible patients. No imputation of missing values was performed. Abbreviations: MELD-Na, Model for End-stage Liver Disease—Sodium; PEth, phosphatidylethanol; sHR, subdistribution hazard ratio.

Among patients without recompensation, survival declined over time and was lower in MetALD than MASLD. In MASLD, survival was 96.9%, 88.2%, and 82.9% at 1, 3, and 5 years, respectively, compared with 93.3%, 73.4%, and 71.0% in MetALD.

DISCUSSION

In this prospective cohort of patients with decompensated MASLD and MetALD cirrhosis, recompensation according to the Baveno VII criteria was achieved in approximately one-fifth of patients, with comparable rates across both etiologies. These findings extend the concept of recompensation beyond viral and alcohol-associated cirrhosis and suggest that sustained modification of metabolic and alcohol-associated disease drivers may serve as a surrogate for etiologic control in SLD.^[5,30–32]

Despite marked differences in alcohol exposure, MASLD and MetALD achieved similar rates of recompensation. Sustained alcohol abstinence, confirmed by serial PEth testing and interpreted alongside clinical history, was associated with comparable outcomes, underscoring the importance of modifying disease drivers rather than baseline disease classification.^[33]

Beyond alcohol abstinence, weight loss has been reported as a key modifiable factor in Asian cohorts with MASLD/MetALD.^[34] The mean BMI of MASLD patients in our cohort (25.4 ± 4.9 kg/m²) was lower than that reported in many Western cohorts, likely reflecting advanced decompensated cirrhosis with sarcopenia, frailty, malnutrition, and catabolic changes, as well as dry-weight estimation and the use of Asian-specific BMI thresholds.^[35]

Exploratory analyses demonstrated a graded association between achievement of prespecified alcohol abstinence and metabolic targets and recompensation. No recompensation occurred among patients achieving ≤ 1 target, whereas the highest rates were observed among those achieving all 3 targets, a pattern consistent across both MASLD and MetALD. These findings support the concept that simultaneous modification of multiple disease drivers, including alcohol abstinence, substantial dry-weight loss, and good glycemic control, is associated with recompensation and functional hepatic recovery.^[36]

Notably, while recompensation rates were similar across aetiologies, clinical outcomes differed between MASLD and MetALD, with higher rates of re-admission and mortality observed in MetALD. Lower MELD-Na scores and the absence of large varices at enrollment were associated with recompensation, consistent with prior observations in other etiologies.^[37] Importantly, recompensation was associated with favorable outcomes, supporting its relevance as a clinically

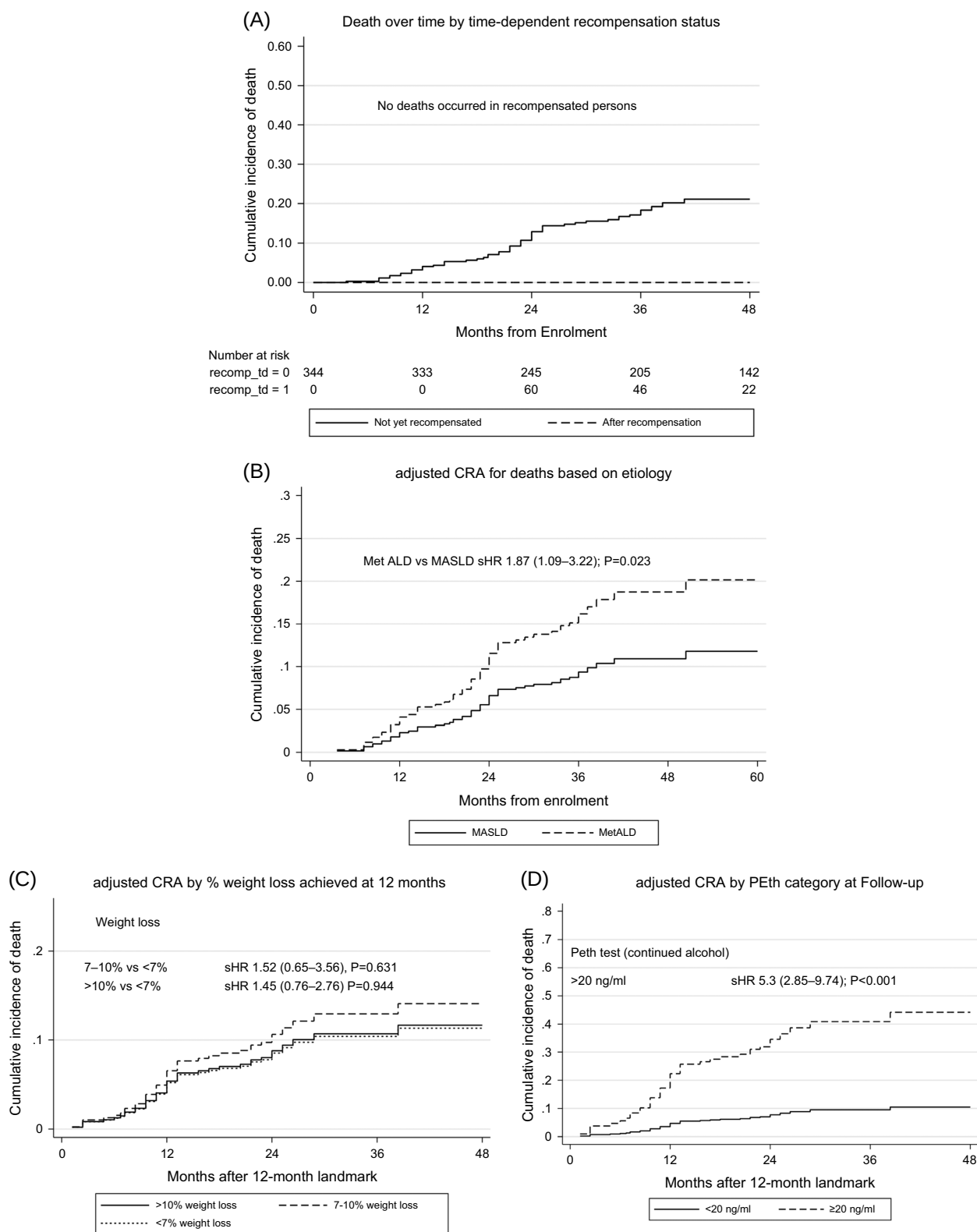


FIGURE 5 (A) Simon–Makuch-type cumulative incidence curves for death following baseline decompensated cirrhosis according to time-dependent recompensation status. Recompensation was modeled as a time-dependent exposure; patients contributed person-time to the “not yet recompensated” group until the time of recompensation and to the “after recompensation” group thereafter. Follow-up was administratively censored at 48 months. Numbers at risk are shown below the x-axis. Because no deaths occurred after recompensation, the hazard ratio for recompensation could not be reliably estimated and is therefore not emphasized. (B–D) Adjusted cumulative incidence functions (CIFs) for death derived from Fine–Gray competing-risk regression, accounting for liver transplantation as a competing event and adjusted for baseline MELD-Na. CIFs were evaluated at the median MELD-Na value and truncated at 4 years of follow-up. (B) Etiology (MetALD vs. MASLD). (C) Weight-loss

category at 12 months ($< 7\%$, $7\%–10\%$, $\geq 10\%$). (D) PEth category at 12 months. Abbreviations: CIF, cumulative incidence function; HR, hazard ratio; MASLD, metabolic dysfunction–associated steatotic liver disease; MELD-Na, Model for End-stage Liver Disease—Sodium; MetALD, metabolic dysfunction–associated steatotic liver disease with alcohol consumption; PEth, phosphatidylethanol; sHR, subdistribution hazard ratio.

meaningful endpoint.^[4] Although no deaths occurred after recompensation, this observation should be interpreted cautiously because recompensation is a post-baseline event, and survivor selection cannot be ruled out. However, no deaths were observed over 137.9 person-years of follow-up after recompensation, suggesting that recompensation identifies a subgroup with a particularly favorable prognosis.

The current Baveno VII criteria have limitations, including the absence of validated time thresholds for recompensation and the requirement for sustained withdrawal of therapies used to control decompensation. Although our prospective design allowed systematic assessment of diuretic and lactulose withdrawal, with reintroduction only if ascites or hepatic encephalopathy recurred, attainment of CTP-A status remained more common than formal recompensation because many patients continued to require low-dose therapy to prevent portal hypertension-related complications.^[3]

A major strength of this study is the accurate phenotyping of MASLD and MetALD using complementary AUDIT-C and PEth assessments, together with a structured multidisciplinary model integrating hepatology, addiction medicine, and metabolic care.^[15,38] In the absence of approved disease-modifying therapies for decompensated MASLD/MetALD cirrhosis, our findings suggest that recompensation is associated with sustained alcohol abstinence, optimization of CMRF, and effective management of portal hypertension complications rather than pharmacotherapy alone.^[39] Our findings also extend those of a recent Spanish cohort, in which 24% of patients with MASLD achieved recompensation over 2 years, and lower MELD-Na, higher albumin, absence of ascites, and fewer decompensation events predicted recompensation. Unlike that study, where the survival benefit diminished after adjustment for liver function, recompensation in our cohort remained associated with improved survival after adjustment for MELD-Na, possibly reflecting prospective follow-up with objective PEth-guided monitoring and structured cardiometabolic management.^[40]

Several limitations should be acknowledged. First, this was an observational study; therefore, causal relationships between treatment-response variables and recompensation cannot be established. Second, unlike viral hepatitis or alcohol-associated liver disease, there is no validated definition of etiologic control in MASLD or MetALD. Accordingly, the prespecified targets used in this study, that is, PEth-confirmed alcohol abstinence (PEth < 20 ng/mL), sustained

glycemic control (HbA1c $< 7\%$), and $\geq 7\%$ dry-weight loss at 12 months, should be interpreted as treatment-response markers associated with recompensation rather than definitive measures of etiologic control. Furthermore, weight loss in decompensated cirrhosis may reflect resolution of ascites as well as sarcopenia, catabolism, or frailty, and should not be considered a specific marker of metabolic improvement. Finally, the 12-month landmark analyses are subject to temporal overlap between achievement of treatment-response targets and subsequent recompensation; therefore, these findings should be interpreted as associative rather than causal.

Recompensation was assessed at scheduled study visits rather than at the exact time of its occurrence; therefore, the narrow interquartile range for time to recompensation likely reflects the protocol visit schedule rather than biological variability. The adjusted regression models were intentionally parsimonious because of the limited number of recompensation events and potential collinearity among metabolic, portal hypertension, and liver disease severity variables. Consequently, residual confounding cannot be excluded, and the observed associations should not be interpreted as causal. PEth was measured using a modified whole-blood immunoassay because commercial assays were not widely available in India. Although PEth correlated with AUDIT-C scores, immunoassays may be less sensitive than LC-MS/MS, the reference standard, for detecting this membrane-associated biomarker.^[41] Finally, while PEth provides high specificity for recent alcohol exposure, it reflects only the preceding few weeks and should be interpreted alongside clinical history, particularly as patients may transition between MASLD, MetALD, and ALD phenotypes over time.^[30,42] Finally, longer-term follow-up is required to assess the durability of recompensation and its impact on HCC risk, re-decompensation, and other liver-related outcomes in MASLD and MetALD.

In conclusion, recompensation according to the Baveno VII criteria is achievable in patients with decompensated MASLD and MetALD. Objective assessment of alcohol exposure using PEth, together with sustained alcohol abstinence, weight loss, and glycemic control, was associated with recompensation. These findings support integrated management of metabolic dysfunction and alcohol exposure as a practical strategy to achieve recompensation in advanced steatotic liver disease.

DATA AVAILABILITY STATEMENT

Deidentified data are available from the corresponding author on reasonable request.

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CONFLICTS OF INTEREST

K. Rajender Reddy advises and received grants paid to his institution from BD Instruments, Mallinckrodt, and Sequana. He advises OrganOx, Ipsen, and Novo Nordisk. He received grants paid to his institution from BMS, Exact Sciences, Intercept, GSK, Camurus, TARGET-HCC, TARGET-NASH, Atea, BioVie, and Grifols. He serves on the DSMB for Pfizer, Novartis, Arbutus, Mirum, Vir, nChroma Bio, and AstraZeneca. He receives royalties from UpToDate. The remaining authors have no conflicts to report.

ETHICAL CLEARANCE AND CONSENT TO PARTICIPATE

Ethical clearance for the prospective cardiometabolic cohort study was obtained from the Institutional Ethical Committee (IEC Number PGI/IEC/2019/002095, dated 25.9.2019) and subsequently modified in view of the change in MASLD nomenclature (vide PGI/INT/IEC/2024/1523, dated 3.12.2024). The study was registered on NCT06386094, available from <https://clinicaltrials.gov/study/NCT06386094>. Patients provided written informed consent for the use of their data and for academic publication, and their participation adhered to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement.

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