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# Pre-emptive TIPS with 8-mm stents reduces hepatic encephalopathy without compromising efficacy in acute variceal bleeding

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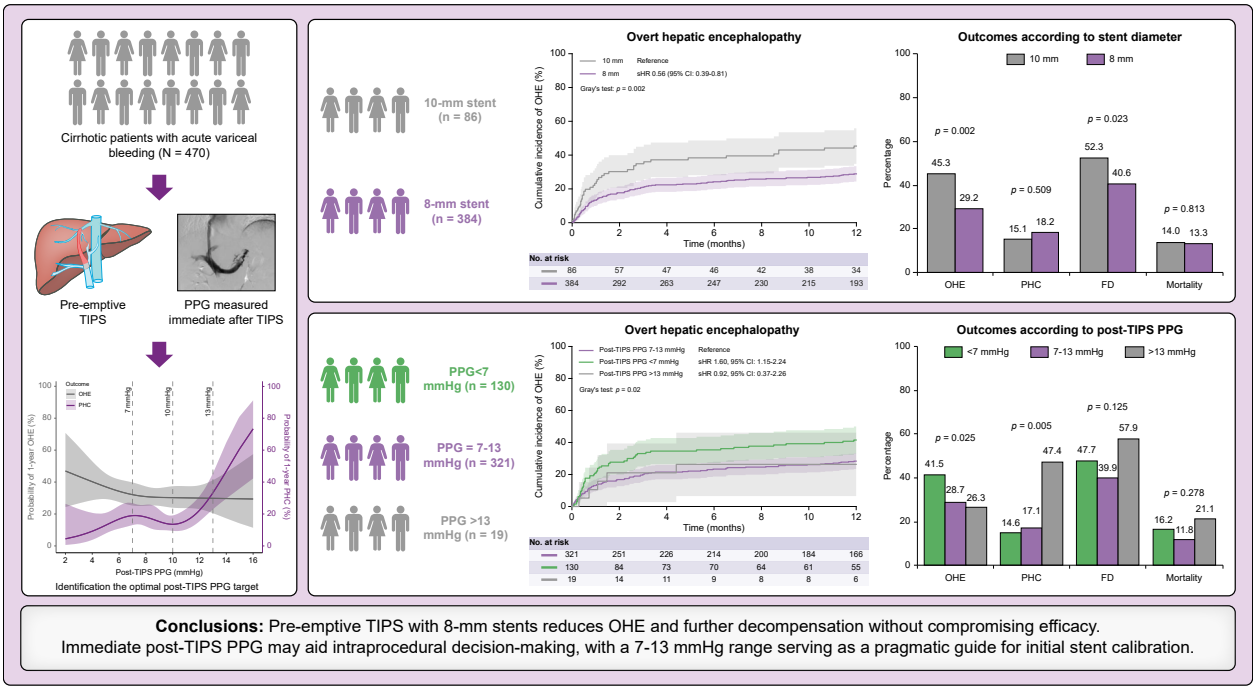
## Graphical abstract




**Conclusions:** Pre-emptive TIPS with 8-mm stents reduces OHE and further decompensation without compromising efficacy. Immediate post-TIPS PPG may aid intraprocedural decision-making, with a 7-13 mmHg range serving as a pragmatic guide for initial stent calibration.

## Highlights:

- 8-mm stents reduce hepatic encephalopathy by 43% compared to 10-mm stents in pre-emptive TIPS for acute variceal bleeding.
- No increased rebleeding or mortality rates were observed with 8-mm stents.
- An immediate post-TIPS PPG range of 7–13 mmHg minimized both encephalopathy and portal hypertension risk.
- 71% of patients achieved PPGs of 7–13 mmHg with 8-mm stents, demonstrating feasibility for intraprocedural calibration.
- Personalized stent sizing guided by hemodynamic targets may improve outcomes in high-risk patients with acute variceal bleeding.

## Impact and implications:

This multicenter study of 470 patients with cirrhosis and acute variceal bleeding shows that pre-emptive transjugular intrahepatic portosystemic shunt (TIPS) placement using 8-mm stents reduces the 1-year incidence of overt hepatic encephalopathy by 43% compared to 10-mm stents, while maintaining similar efficacy in preventing rebleeding. Non-linear analysis identified a post-TIPS portacaval pressure gradient (PPG) range of 7–13 mmHg as an optimal target, minimizing risks of both overt hepatic encephalopathy and portal hypertensive complications. A significantly higher proportion of patients achieved this PPG range with 8-mm stents. These results address a key dilemma in high-risk AVB management, demonstrating that 8-mm stents balance encephalopathy prevention with effective portal decompression. The 7–13 mmHg PPG range provides a practical intraprocedural guide for individualized TIPS calibration, helping interventional radiologists optimize shunt diameter selection.

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# Pre-emptive TIPS with 8-mm stents reduces hepatic encephalopathy without compromising efficacy in acute variceal bleeding

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**Background & Aims:** Pre-emptive transjugular intrahepatic portosystemic shunt (TIPS) improves outcomes in high-risk acute variceal bleeding but its use is limited by hepatic encephalopathy (HE). While stent diameter and post-TIPS portacaval pressure gradient (PPG) targets may influence HE risk, evidence-based standards are lacking. This study aimed to compare 8-mm *vs.* 10-mm diameter stents and evaluate PPG thresholds to balance HE risk and therapeutic efficacy.

**Methods:** In this multicenter observational study, 470 patients with cirrhosis and acute variceal bleeding receiving pre-emptive TIPS (8-mm: n = 384; 10-mm: n = 86) were analyzed. Competing risks regression and restricted cubic splines were used to assess associations between stent diameter, PPG, and clinical outcomes.

**Results:** At 1 year, 8-mm stents reduced overt HE incidence (28.9% *vs.* 45.4%; subdistribution hazard ratio [sHR] 0.57, 95% CI 0.40–0.82) and further decompensation (40.4% *vs.* 52.3%; sHR 0.68, 95% CI 0.48–0.95) compared to 10-mm stents, without increasing the risk of further bleeding (12.0% *vs.* 9.3%; $p = 0.471$) or mortality (13.3% *vs.* 14.0%; $p = 0.813$). Non-linear analysis identified a PPG range of 7–13 mmHg associated with minimized overt HE risks (sHR 1.43 for PPG <7 *vs.* 7–13 mmHg; 95% CI 1.01–2.01) and portal hypertensive complications (sHR 2.76 for PPG >13 *vs.* 7–13 mmHg; 95% CI 2.69–9.39). A significantly greater proportion of patients in the 8-mm group attained the optimal 7–13 mmHg target range compared to the 10-mm group (71.1% *vs.* 55.8%, $p$ <0.001).

**Conclusions:** Pre-emptive TIPS with 8-mm stents reduces HE and further decompensation without compromising efficacy. Immediate post-TIPS PPG measurements may aid intraprocedural decision-making, with a 7–13 mmHg range serving as a pragmatic guide for initial stent calibration in high-risk acute variceal bleeding.



## Introduction

Acute variceal bleeding (AVB), a life-threatening complication of portal hypertension in cirrhosis, carries a mortality rate exceeding 20% within 6 weeks despite advances in pharmacotherapy and endoscopy.[1] Pre-emptive transjugular intrahepatic portosystemic shunt (pre-emptive TIPS), defined as TIPS placement within 72 h of initial hemostasis, has been endorsed by current guidelines as a first-line therapy for AVB in high-risk patients.[2,3] The criteria for pre-emptive TIPS include hepatic venous pressure gradient (HVPG) >20 mmHg, Child-Pugh B >7 points with active bleeding, or Child-Pugh C <14 points.[2–4] While pre-emptive TIPS reduces rebleeding and mortality compared to combined pharmacological and endoscopic therapy, post-TIPS hepatic encephalopathy (HE) remains a significant concern, affecting 20-50% of patients within 1 year.[5–7] This limitation has driven efforts to refine TIPS hemodynamic parameters, particularly through stent diameter optimization, to mitigate HE risk while preserving therapeutic benefits.[8,9]

The balance between therapeutic efficacy and complications hinges on precise hemodynamic control. According to Poiseuille's law, shunt flow is proportional to the fourth power of the stent radius.[8] This principle creates a clinical dilemma: larger stents (*e.g.* 10 mm) maximize portal decompression but risk excessive shunting of gut-derived toxins (*e.g.* ammonia) to the systemic circulation, whereas smaller stents (*e.g.* 8 mm) may preserve hepatic detoxification at the cost of insufficient portal pressure reduction. This trade-off is reflected in current conflicting evidence. A randomized controlled trial from our team in patients with variceal bleeding demonstrated that 8-

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mm stents halved spontaneous HE incidence without increasing rebleeding compared to 10-mm stents,[10] whereas Italian studies in refractory ascites cohorts favored 10-mm stents due to lower ascites recurrence.[11,12] This discrepancy likely reflects indication-specific priorities: variceal bleeding management prioritizes HE prevention, while ascites requires maximal decompression. However, no studies have specifically evaluated stent diameter in pre-emptive TIPS populations – a unique subgroup vulnerable to both rebleeding and HE due to acute hemodynamic instability.

Hemodynamic targets further complicate decision-making. Current guidelines recommend post-TIPS portacaval pressure gradient (PPG) <12 mmHg or >50% reduction from baseline to prevent rebleeding,[2–4] but such aggressive targets risk over-shunting and subsequent HE. Early observational data suggest PPG levels above 10 mmHg may reduce HE,[13] yet these thresholds remain unvalidated in AVB cohorts. Additionally, the non-linear relationship between PPG and clinical outcomes remains uncharacterized, hindering personalized treatment strategies. Recent innovations in controlled-expansion stents, which allow for real-time PPG calibration,[14] now make precision shunting feasible – provided optimal thresholds are established.

This multicenter study aimed to address two unresolved questions: (1) whether 8-mm TIPS reduces HE without compromising efficacy in pre-emptive AVB management, and (2) what post-TIPS PPG values could serve as potential reference points for balancing rebleeding prevention and HE risk.

## Patients and methods

### Study population

This is an ancillary study of the multicenter, observational study that enrolled patients with cirrhosis and AVB treated with either pharmaco-endoscopic therapy or pre-emptive TIPS across 13 Chinese centers between December 2010 and December 2019.[15] Ethical approval for retrospective data analysis was obtained from all participating institutions' review boards, with written informed consent provided by all participants during the original study.

Eligibility criteria were: (i) confirmed cirrhosis diagnosis through histopathology or combined clinical/imaging evidence; (ii) hospitalization for endoscopically verified AVB (spurting/oozing varices, adherent clots, or varices with excluded alternative bleeding sources per Baveno criteria[4]); (iii) pre-emptive TIPS placement within 72 h of admission following initial hemostasis with vasoactive agents and endoscopic therapy. Exclusion criteria were age >75 years, hepatocellular carcinoma beyond Milan criteria, advanced extrahepatic malignancy, bleeding from isolated gastric or ectopic varices, previous liver transplantation and receiving rescue TIPS treatment, Child-Pugh score >13 points, creatinine level ≥3 mg/dl, recurrent HE, heart failure, complete portal vein thrombosis.

### Therapeutic interventions

The treatments of patients have been described in previous study.[6,7,15] Briefly, initial medical therapy included a combination of vasoactive agents (terlipressin, somatostatin, or octreotide administered via continuous infusion for 5 days), antibiotic prophylaxis (norfloxacin 400 mg bid or ceftriaxone 1 g qd for 5 days), and endoscopic band ligation performed within 12 h of admission. Vasoactive therapy was maintained throughout the perioperative period until procedure completion. Pre-emptive TIPS was performed within 72 h of admission using standardized techniques. Briefly, under local anesthesia (1% lidocaine injected subcutaneously at the puncture site, without general anesthesia or deep sedation) and fluoroscopic guidance, an intrahepatic tract was created between a hepatic vein and a portal vein branch, using an 18-gauge RUPS-100 needle. Following successful portal vein puncture, pressures were measured in the main portal vein and inferior vena cava. Portography was then performed. If large varices were identified, collateral embolization with coils was undertaken. The needle tract was then dilated with either an 8-mm or 10-mm balloon angioplasty catheter, followed by deployment of an 8-mm or 10-mm expanded PTFE-covered stent. Only conventional non-controlled-expansion PTFE-covered stents were utilized as controlled-expansion stents were unavailable during this study period. Stent diameter selection (8-mm *vs.* 10-mm) was determined intraprocedurally by the attending interventional radiologist, based on baseline PPG, clinical risk factors for HE, and technical feasibility. Stent dilation was performed immediately after deployment using a standard angioplasty balloon matched to the nominal stent diameter (8-mm balloon for 8-mm stents, 10-mm balloon for 10-mm stents) to ensure full expansion. If necessary, an additional stent was implanted to optimize positioning, aiming to extend the cephalic end to the hepatocaval junction and align the caudal end parallel to the main portal vein wall. Post-dilation hemodynamic measurements were repeated. For patients with portal vein thrombosis, sonographic-guided percutaneous transhepatic or trans-splenic access was utilized to facilitate the TIPS procedure, as clinically indicated. After TIPS, patients with pre-existing encephalopathy received lactulose ± rifaximin as secondary prophylaxis. Patients without prior HE did not receive post-TIPS prophylactic therapy for encephalopathy unless symptoms appeared. Post-procedural surveillance incorporated Doppler ultrasonography to assess shunt functionality, with predefined revision criteria including clinical recurrence of portal hypertension complications or ultrasonographic evidence of shunt dysfunction (a reduction of portal blood flow velocity >50% or <28 cm/s or a reversion of blood flow direction within the intrahepatic branches). Revision strategies encompassed balloon angioplasty or supplemental stent placement as clinically indicated. Patients were followed from admission until death, liver transplantation, one year, the last visit, or the end of the study, whichever occurred first.

### Outcomes

The primary endpoint of the study was incidence of overt hepatic encephalopathy (OHE) within 1 year after TIPS, defined as West-Haven grade II-IV episodes.[16–18] Encephalopathy events were verified by independent adjudicators reviewing clinical records, including outpatient management data. All OHE episodes were recorded, irrespective of hospitalization. The secondary endpoints were portal hypertensive complications (PHCs), further bleeding, new or worsening ascites, and further decompensation. Further bleeding includes failure to control bleeding or rebleeding that were defined according to Baveno VII consensus workshop.[4] New or worsening ascites

**Table 1. Baseline characteristics of patients.**

| Variables | All (N = 470) | Stent diameter | | p values | Post-TIPS PPG | | | p values |
|---|---|---|---|---|---|---|---|---|
| | | 10 mm (n = 86) | 8 mm (n = 384) | | <7 mmHg (n = 130) | 7-13 mmHg (n = 321) | >13 mmHg (n = 19) | |
| Gender, n (%) | | | | 0.854 | | | | 0.741 |
| Male | 291 (61.9%) | 52 (60.5%) | 239 (62.2%) | | 84 (64.6%) | 195 (60.7%) | 12 (63.2%) | |
| Female | 179 (38.1%) | 34 (39.5%) | 145 (37.8%) | | 46 (35.4%) | 126 (39.3%) | 7 (36.8%) | |
| Age (years) | 53.0 (12.0) | 54.9 (12.8) | 52.6 (11.8) | 0.12 | 53.7 (12.5) | 53.0 (11.8) | 47.9 (10.9) | 0.153 |
| Etiology of cirrhosis, n (%) | | | | 0.938 | | | | |
| Chronic HBV infection | 292 (62.1%) | 55 (64.0%) | 237 (61.7%) | | 84 (64.6%) | 196 (61.1%) | 12 (63.2%) | |
| Chronic HCV infection | 31 (6.6%) | 6 (7.0%) | 25 (6.5%) | | 9 (6.9%) | 19 (5.9%) | 3 (15.8%) | |
| Alcohol | 26 (5.5%) | 6 (7.0%) | 20 (5.2%) | | 8 (6.2%) | 18 (5.6%) | 0 (0.0%) | |
| Others | 50 (10.6%) | 9 (10.5%) | 41 (10.7%) | | 8 (6.2%) | 41 (12.8%) | 1 (5.3%) | |
| Miscellaneous | 63 (13.4%) | 9 (10.5%) | 54 (14.1%) | | 20 (15.4%) | 40 (12.5%) | 3 (15.8%) | |
| Cryptogenic | 8 (1.7%) | 1 (1.2%) | 7 (1.8%) | | 1 (0.8%) | 7 (2.2%) | 0 (0.0%) | |
| HBV-DNA detectable, n (%) | 124 (30.0%) | 23 (31.1%) | 101 (29.8%) | 0.937 | 34 (29.1%) | 87 (31.0%) | 3 (20.0%) | 0.709 |
| Comorbidities[‡], n (%) | 106 (22.6%) | 21 (24.4%) | 85 (22.1%) | 0.753 | 31 (23.8%) | 72 (22.4%) | 3 (15.8%) | 0.787 |
| Previous hepatic encephalopathy, n (%) | 44 (9.4%) | 11 (12.8%) | 33 (8.6%) | 0.316 | 12 (9.2%) | 30 (9.3%) | 2 (10.5%) | 0.916 |
| Hepatic encephalopathy at admission, n (%) | 19 (4.0%) | 3 (3.5%) | 16 (4.2%) | 0.951 | 4 (3.1%) | 14 (4.4%) | 1 (5.3%) | 0.603 |
| Ascites at admission, n (%) | 304 (64.7%) | 53 (61.6%) | 251 (65.4%) | 0.596 | 87 (66.9%) | 205 (63.9%) | 12 (63.2%) | 0.819 |
| Mild | 170 (36.2%) | 25 (29.1%) | 145 (37.8%) | | 44 (33.8%) | 118 (36.8%) | 8 (42.1%) | |
| Moderate | 91 (19.4%) | 19 (22.1%) | 72 (18.8%) | | 27 (20.8%) | 61 (19.0%) | 3 (15.8%) | |
| Massive | 43 (9.1%) | 9 (10.5%) | 34 (8.9%) | | 16 (12.3%) | 26 (8.1%) | 1 (5.3%) | |
| Hepatocellular carcinoma, n (%) | 15 (3.2%) | 3 (3.5%) | 12 (3.1%) | 0.744 | 2 (1.5%) | 13 (4.0%) | 0 (0.0%) | 0.38 |
| Portal vein thrombosis, n (%) | 83 (17.7%) | 12 (14.0%) | 71 (18.5%) | 0.401 | 21 (16.2%) | 57 (17.8%) | 5 (26.3%) | 0.502 |
| Infection at admission, n (%) | 36 (7.7%) | 4 (4.7%) | 32 (8.3%) | 0.349 | 9 (6.9%) | 25 (7.8%) | 2 (10.5%) | 0.764 |
| Shock at admission, n (%) | 85 (18.1%) | 17 (19.8%) | 68 (17.7%) | 0.769 | 31 (23.8%) | 49 (15.3%) | 5 (26.3%) | 0.055 |
| Heart rate at admission (beats/min) | 83.9 (15.1) | 84.4 (15.8) | 83.8 (15.0) | 0.758 | 84.7 (15.7) | 83.9 (15.1) | 79.6 (12.0) | 0.374 |
| Systolic blood pressure at admission (mmHg) | 112.3 (15.0) | 113.1 (14.5) | 112.1 (15.1) | 0.541 | 112.0 (15.0) | 113.0 (14.8) | 102.0 (15.2) | 0.008 |
| Diastolic blood pressure at admission (mmHg) | 67.2 (10.2) | 67.4 (9.9) | 67.2 (10.3) | 0.827 | 66.2 (9.8) | 67.8 (10.3) | 65.1 (10.7) | 0.226 |
| White blood cell ($\times 10^9$ cell/L) | 6.2 (4.5) | 5.7 (3.6) | 6.3 (4.7) | 0.141 | 6.0 (4.4) | 6.3 (4.6) | 6.0 (5.4) | 0.855 |
| Hemoglobin (g/L) | 76.5 (19.8) | 77.7 (19.9) | 76.2 (19.8) | 0.54 | 77.7 (21.6) | 76.4 (19.1) | 69.4 (18.5) | 0.235 |
| Platelet count ($\times 10^9$/L) | 80.0 (95.6) | 88.5 (186.7) | 78.1 (58.7) | 0.61 | 85.2 (154.9) | 76.7 (52.4) | 99.8 (129.5) | 0.459 |
| International normalized ratio | 1.5 (0.5) | 1.5 (0.4) | 1.5 (0.5) | 0.621 | 1.6 (0.7) | 1.5 (0.4) | 1.5 (0.3) | 0.091 |
| Albumin (g/L) | 30.2 (5.6) | 29.7 (5.8) | 30.4 (5.5) | 0.335 | 29.9 (6.2) | 30.3 (5.2) | 31.9 (7.4) | 0.315 |
| Bilirubin (mg/dl) | 1.7 (1.4) | 1.7 (1.3) | 1.7 (1.4) | 0.918 | 2.0 (2.0) | 1.5 (1.0) | 1.4 (0.6) | 0.013 |
| Creatinine (mg/dl) | 0.8 (0.3) | 0.8 (0.3) | 0.9 (0.3) | 0.175 | 0.8 (0.2) | 0.8 (0.3) | 0.8 (0.4) | 0.969 |
| Sodium (mmol/L) | 138.0 (5.6) | 137.0 (6.9) | 138.2 (5.2) | 0.135 | 138.0 (5.7) | 138.0 (5.6) | 138.3 (5.7) | 0.964 |
| Child-Pugh score (points) | 7.8 (1.6) | 7.8 (1.7) | 7.8 (1.6) | 0.988 | 8.0 (1.6) | 7.7 (1.6) | 7.9 (1.7) | 0.207 |
| Child-Pugh class, n (%) | | | | 0.998 | | | | 0.687 |
| A | 103 (21.9%) | 19 (22.1%) | 84 (21.9%) | | 24 (18.5%) | 76 (23.7%) | 3 (15.8%) | |
| B | 295 (62.8%) | 54 (62.8%) | 241 (62.8%) | | 83 (63.8%) | 199 (62.0%) | 13 (68.4%) | |
| C | 72 (15.3%) | 13 (15.1%) | 59 (15.4%) | | 23 (17.7%) | 46 (14.3%) | 3 (15.8%) | |
| Early-TIPS criteria[*], n (%) | | | | 0.571 | | | | 0.182 |
| Low risk | 267 (56.8%) | 46 (53.5%) | 221 (57.6%) | | 77 (59.2%) | 183 (57.0%) | 7 (36.8%) | |
| High risk | 203 (43.2%) | 40 (46.5%) | 163 (42.4%) | | 53 (40.8%) | 138 (43.0%) | 12 (63.2%) | |
| Baveno VII clinical criteria [¶], n (%) | | | | 0.773 | | | | 0.965 |
| Low risk | 315 (67.0%) | 56 (65.1%) | 259 (67.4%) | | 86 (66.2%) | 216 (67.3%) | 13 (68.4%) | |
| High risk | 155 (33.0%) | 30 (34.9%) | 125 (32.6%) | | 44 (33.8%) | 105 (32.7%) | 6 (31.6%) | |
| Baveno VII criteria[ſ], n (%) | | | | 0.908 | | | | 0.192 |
| Low risk | 84 (17.9%) | 15 (17.4%) | 69 (18.0%) | | 28 (21.5%) | 55 (17.1%) | 1 (5.3%) | |
| High risk | 386 (82.1%) | 71 (82.6%) | 315 (82.0%) | | 102 (78.5%) | 266 (82.9%) | 18 (94.7%) | |
| MELD score[#] (points) | 12.7 (3.9) | 12.7 (3.7) | 12.7 (3.9) | 0.943 | 13.4 (4.3) | 12.4 (3.8) | 13.0 (3.1) | 0.053 |
| <11 | 177 (37.7%) | 32 (37.2%) | 145 (37.8%) | | 42 (32.3%) | 131 (40.8%) | 4 (21.1%) | |
| 11-19 | 262 (55.7%) | 46 (53.5%) | 216 (56.2%) | | 76 (58.5%) | 171 (53.3%) | 15 (78.9%) | |
| >19 | 31 (6.6%) | 8 (9.3%) | 23 (6.0%) | | 12 (9.2%) | 19 (5.9%) | 0 (0.0%) | |
| MELD-Na score (points) | 14.2 (6.0) | 14.9 (7.5) | 14.0 (5.6) | 0.266 | 14.7 (6.3) | 14.0 (6.0) | 13.7 (4.5) | 0.497 |
| Recalibrated MELD score[$] (points) | -2.7 (0.8) | -2.7 (0.8) | -2.7 (0.8) | 0.943 | -2.5 (0.9) | -2.7 (0.8) | -2.6 (0.6) | 0.053 |
| MELD-HE scores[ʃ] (points) | -2.6 (0.6) | -2.6 (0.5) | -2.6 (0.6) | 0.706 | -2.5 (0.6) | -2.7 (0.6) | -2.6 (0.5) | 0.217 |
| CLIF-C AD score [§] (points) | 45.6 (9.7) | 45.9 (9.1) | 45.6 (9.8) | 0.777 | 46.6 (10.6) | 45.4 (9.2) | 42.6 (10.5) | 0.18 |
| <45 | 223 (47.4%) | 38 (44.2%) | 185 (48.2%) | | 55 (42.3%) | 156 (48.6%) | 12 (63.2%) | |
| 45-60 | 213 (45.3%) | 39 (45.3%) | 174 (45.3%) | | 62 (47.7%) | 145 (45.2%) | 6 (31.6%) | |
| >60 | 34 (7.2%) | 9 (10.5%) | 25 (6.5%) | | 13 (10.0%) | 20 (6.2%) | 1 (5.3%) | |
| FIPS score[❖] (points) | -2.1 (23.4) | -1.0 (1.7) | -2.4 (25.9) | 0.317 | -0.6 (0.8) | -2.8 (28.3) | -1.1 (1.2) | 0.654 |
| CCG-AVB score[#] (points) | 50.8 (8.7) | 51.4 (8.8) | 50.6 (8.7) | 0.469 | 52.3 (10.1) | 50.3 (8.0) | 47.9 (8.3) | 0.149 |
| <48 | 177 (37.7%) | 29 (33.7%) | 148 (38.5%) | | 40 (30.8%) | 128 (39.9%) | 9 (47.4%) | |

(*continued on next page*)

**Table 1.** (*continued*)

| Variables | All (N = 470) | Stent diameter | | p values | Post-TIPS PPG | | | p values |
|---|---|---|---|---|---|---|---|---|
| | | 10 mm (n = 86) | 8 mm (n = 384) | | <7 mmHg (n = 130) | 7-13 mmHg (n = 321) | >13 mmHg (n = 19) | |
| 48-60 | 235 (50.0%) | 57 (66.3%) | 236 (61.5%) | | 70 (53.8%) | 155 (48.3%) | 10 (52.6%) | |
| >60 | 58 (12.3%) | 38 (44.2%) | 165 (43.0%) | | 20 (15.4%) | 38 (11.8%) | 0 (0.0%) | |
| Active bleeding at endoscopy†, n (%) | 203 (43.2%) | 38 (44.2%) | 165 (43.0%) | 0.932 | 53 (40.8%) | 138 (43.0%) | 12 (63.2%) | 0.182 |
| Location of varices at index gastroscopy, n (%) | | | | 0.994 | | | | 0.491 |
| Esophageal varices only | 309 (65.7%) | 57 (66.3%) | 252 (65.6%) | | 80 (61.5%) | 216 (67.3%) | 13 (68.4%) | |
| Esophageal and gastric varices | 161 (34.3%) | 29 (33.7%) | 132 (34.4%) | | 50 (38.5%) | 105 (32.7%) | 6 (31.6%) | |
| Variceal embolization, n (%) | 201 (42.8%) | 36 (41.9%) | 165 (43.0%) | 0.946 | 15 (11.5%) | 169 (52.6%) | 17 (89.5%) | <0.001 |
| Blood transfusion, n (%) | 259 (55.1%) | 51 (59.3%) | 208 (54.2%) | 0.456 | 75 (57.7%) | 171 (53.3%) | 13 (68.4%) | 0.341 |
| Initial endoscopic treatment, n (%) | | | | 0.867 | | | | 0.286 |
| Endoscopic band ligation | 438 (93.2%) | 81 (94.2%) | 357 (93.0%) | | 122 (93.8%) | 300 (93.5%) | 16 (84.2%) | |
| Endoscopic sclerotherapy | 22 (4.7%) | 3 (3.5%) | 19 (4.9%) | | 5 (3.8%) | 14 (4.4%) | 3 (15.8%) | |
| None | 10 (2.1%) | 2 (2.3%) | 8 (2.1%) | | 3 (2.3%) | 7 (2.2%) | 0 (0.0%) | |
| Initial pharmacological therapy, n (%) | | | | 0.085 | | | | 0.608 |
| Octreotide | 342 (72.8%) | 71 (82.6%) | 271 (70.6%) | | 89 (68.5%) | 239 (74.5%) | 14 (73.7%) | |
| Somatostatin | 107 (22.8%) | 13 (15.1%) | 94 (24.5%) | | 33 (25.4%) | 69 (21.5%) | 5 (26.3%) | |
| Terlipressin | 21 (4.5%) | 2 (2.3%) | 19 (4.9%) | | 8 (6.2%) | 13 (4.0%) | 0 (0.0%) | |
| Antibiotherapy, n (%) | | | | 0.226 | | | | 0.836 |
| Norfloxacin | 27 (5.7%) | 8 (9.3%) | 19 (4.9%) | | 9 (6.9%) | 18 (5.6%) | 0 (0.0%) | |
| Ceftriaxone | 385 (81.9%) | 66 (76.7%) | 319 (83.1%) | | 103 (79.2%) | 265 (82.6%) | 17 (89.5%) | |
| None | 58 (12.3%) | 12 (14.0%) | 46 (12.0%) | | 18 (13.8%) | 38 (11.8%) | 2 (10.5%) | |
| Secondary prophylaxis of HE, n (%) | | | | 0.186 | | | | 0.985 |
| Lactulose | 19 (4.0%) | 3 (3.5%) | 16 (4.2%) | | 5 (3.8%) | 13 (4.0%) | 1 (5.3%) | |
| Rifaximin | 16 (3.4%) | 4 (4.7%) | 12 (3.1%) | | 5 (3.8%) | 10 (3.1%) | 1 (5.3%) | |
| Lactulose + rifaximin | 9 (1.9%) | 4 (4.7%) | 5 (1.3%) | | 2 (1.5%) | 7 (2.2%) | 0 (0.0%) | |
| Pre-TIPS portal vein pressure (mmHg) | 30.8 (6.8) | 30.0 (5.9) | 31.0 (7.0) | 0.164 | 29.3 (6.4) | 31.2 (6.9) | 33.7 (5.1) | 0.005 |
| Pre-TIPS inferior vena cava pressure (mmHg) | 7.1 (4.0) | 7.0 (4.0) | 7.1 (4.0) | 0.78 | 6.8 (4.2) | 7.2 (3.8) | 6.6 (4.6) | 0.574 |
| Pre-TIPS PPG★ (mmHg) | 23.7 (5.7) | 23.0 (4.8) | 23.9 (5.8) | 0.139 | 22.5 (4.9) | 24.0 (5.9) | 27.1 (4.4) | 0.001 |
| <20 mmHg | 117 (24.9%) | 22 (25.6%) | 95 (24.7%) | | 42 (32.3%) | 73 (22.7%) | 2 (10.5%) | |
| ≥ 20 mmHg | 353 (75.1%) | 64 (74.4%) | 289 (75.3%) | | 88 (67.7%) | 248 (77.3%) | 17 (89.5%) | |
| Post-TIPS portal vein pressure (mmHg) | 19.7 (5.8) | 18.4 (5.5) | 20.0 (5.9) | 0.019 | 17.5 (6.0) | 20.4 (5.6) | 22.7 (5.1) | <0.001 |
| Post-TIPS inferior vena cava pressure (mmHg) | 11.2 (5.8) | 10.9 (5.0) | 11.2 (6.0) | 0.581 | 12.2 (6.1) | 10.9 (5.6) | 8.3 (5.0) | 0.009 |
| Post-TIPS PPG★ (mmHg) | 8.5 (2.7) | 7.5 (2.6) | 8.8 (2.6) | <0.001 | 5.3 (1.2) | 9.5 (1.6) | 14.4 (1.3) | <0.001 |
| PPG reduction from baseline (%) | 62.4 (14.0) | 66.0 (13.6) | 61.6 (14.0) | 0.008 | 75.3 (7.7) | 58.2 (12.4) | 45.4 (10.0) | <0.001 |

CCG-AVB score, Chinese Collaboration Group-acute variceal bleeding score; CLIF-C AD score, CLIF Consortium Acute Decompensation score; FIPS score, Freiburg index of post-TIPS survival score; HE, hepatic encephalopathy; HVPG, hepatic venous pressure gradient; INR, international normalized ratio; MELD, model for end-stage liver disease; PPG, portacaval pressure gradient; TIPS, transjugular intrahepatic portosystemic shunt; WBC, white blood cell.

Note: Data presented as mean (standard deviation) or number of patients (percentage) where appropriate.

Two-tailed Student's *t* test, one-way ANOVA, or Mann-Whitney *U* test were used to compare continuous variables; two-tailed Pearson's Chi-squared test and Fisher's exact test were used to compare categorical variables.

†Active bleeding was defined as the presence of spurting or oozing from varices on endoscopy.

*Early-TIPS criteria: Low risk: Child-Pugh A and Child-Pugh B without active bleeding at initial endoscopy; High risk: Child-Pugh B with active bleeding at endoscopy and Child-Pugh class C <14.

¶Baveno VII clinical criteria: Low risk: Child-Pugh A, Child-Pugh B without active bleeding at initial endoscopy and Child-Pugh B 7 with active bleeding at endoscopy; High risk: Child B >7 with active bleeding at endoscopy and Child-Pugh class C <14.

ǂBaveno VII criteria: Low risk: Child-Pugh A, Child-Pugh B without active bleeding at initial endoscopy and Child-Pugh B 7 with active bleeding at endoscopy or documented HVPG <20 mmHg at the time of bleeding; High risk: Child B >7 with active bleeding at endoscopy and Child-Pugh class C <14 or documented HVPG ≥20 mmHg at the time of bleeding.

§CLIF-C ADs =10 * ([0.03* age; years] + [0.66* ln {creatinine; mg/dl}] + [1.71 * ln {INR}] + [0.88* ln {WBC; $10^9$ cell/L}] - [0.05*Na; mmol/L] + 8).

ǂǂCCG-AVBs =10 × [0.0289 × (Age, years) -0.0525 × (Albumin, g/L) + 0.3334 × Ln (Bilirubin, mg/dl) + 1.7631 × Ln (INR) + 0.3373 × Ln (WBC, $10^9$/L) + 0.4509 × Ln (Creatinine, mg/dl) - 0.0294 × (Sodium, mmol/L) + 8].

✤FIPS score = 1.43*$\log_{10}$ (bilirubin, mg/dl) - 1.71*1/(creatinine; mg/dl) + 0.02 * age (year) -0.02*albumin (g/L) + 0.81.

#MELD score = 6.43 + 11.2 * ln (INR) + 9.57 * ln (creatinine; mg/dl) + 3.78 * ln (bilirubin; mg/dl).

$Recalibrated MELDs = -5.312 + 0.207*MELD.

¶¶MELD-HE score = -4.265 + 0.1248 * MELD + 1.1506*HE.

★PPG is the difference between the portal vein and inferior vena cava pressures.

‡Comorbidities include hypertension, coronary artery disease, and diabetes.

was defined as new-onset or sustained ascites up to a volume requiring paracentesis despite diuretic use. Further decompensation was defined as the first occurrence of rebleeding, new or worsening ascites, and OHE after TIPS, which was modified based on the Baveno VII workshop definition.[4]

### Statistical analysis

Continuous variables were summarized as mean (SD) or median (IQR) based on data distribution, with between-group comparisons performed using Student's *t* test, one-way ANOVA, or Mann-Whitney *U* test as appropriate. Categorical

variables were presented as counts (proportions) and analyzed using $\chi^2$ or Fisher's exact tests. Pre-TIPS *vs.* post-TIPS PPG changes were evaluated with paired t-tests.

To account for competing mortality and liver transplantation events, we employed competing risks regression analysis using the Fine-Gray subdistribution hazard model. Subdistribution hazard ratios (sHRs) with 95% CIs were calculated to quantify associations between predictors and clinical outcomes. Multivariable competing models were taken to adjust for confounders that were identified through univariable associations ($p$ <0.05) or prior evidence of clinical relevance regardless of statistical significance in our cohort.

Subgroup analyses were conducted across predefined stratification criteria: model for end-stage liver disease (MELD) score (<11/11-19/>19),[19] Chronic Liver Failure-Consortium acute decompensation (CLIF-C AD) score (<45/45-60/>60),[20,21] Child-Pugh class (A/B/C),[22] Chinese Collaboration Group-AVB (CCG-AVB) score (</≥48), Baveno VII criteria (low/high risk),[4] early-TIPS criteria (low/high risk),[23] Baveno VII clinical criteria (low/high risk), pre-TIPS PPG/HVPG (</≥20 mmHg). Heterogeneity of treatment effects was assessed by incorporating interaction terms into adjusted competing risks models. Non-linear relationships between post-TIPS PPG and outcomes were modeled using restricted cubic splines with four knots. To verify the robustness of our findings, we conducted a propensity score matching analysis to evaluate the effect of stent diameter. Patients receiving 8-mm and 10-mm TIPS stents were matched 1:1 without replacement using nearest-neighbor matching with a caliper width of 0.05. Propensity scores were derived from a logistic regression model incorporating the following covariates: age, cirrhosis etiology, active bleeding at endoscopy, ascites, history of HE, bilirubin, albumin, creatinine, sodium, white blood cell count, platelet count, shock at admission, infection at admission, comorbidities, hepatocellular carcinoma status, and history of variceal bleeding. Covariate balance between matched groups was assessed using standardized mean differences, with values <0.1 indicating adequate balance. Additionally, sensitivity analyses were performed, restricting the cohort to guideline-defined high-risk subgroups (Child-Pugh B >7 with active bleeding or Child-Pugh C <14, with or without pre-TIPS PPG ≥20 mmHg). All analyses were performed in R version 4.3.1, with two-tailed $p$ values <0.05 considered statistically significant.

## Results

### Baseline characteristics

From an initial cohort of 3,077 consecutive patients with cirrhosis and AVB, 2,607 were excluded based on predefined inclusion/exclusion criteria (Fig. S1). The final cohort comprised 470 patients undergoing pre-emptive TIPS, including 384 patients (81.7%) receiving 8-mm stents and 86 patients (18.3%) receiving 10-mm stents. Baseline characteristics were summarized in Table 1. The cohort predominantly consisted of males (61.9%) with a mean age of 53.0 ± 12.1 years. Moderate hepatic dysfunction was observed, as reflected by mean Child-Pugh and MELD scores of 7.8 (SD 1.6) and 12.7 (SD 3.9), respectively. HBV-related cirrhosis accounted for 62.1% of cases, and 118 of 124 HBV-DNA–positive patients (95.2%) achieved sustained virologic response, defined as undetectable HBV-DNA, during follow-up. Median follow-up duration was 365 days (IQR 297–365).

### Impact of stent diameter on clinical outcomes

Clinical outcomes are summarized in Table 2. Comparative analysis between stent diameters showed significant advantages for 8-mm stents in reducing OHE risk (Figs 1 and S2-S8). The 8-mm group demonstrated lower cumulative incidence of OHE compared to the 10-mm group (15.6% *vs.* 29.1% at 6 weeks; 28.9% *vs.* 45.4% at 1 year; sHR 0.56, 95% CI 0.39–0.81, $p$ = 0.002; Fig. 1A). This protective effect extended

**Table 2. Summary of outcome measurements.**

| | | Stent diameter | | | Post-TIPS PPG | | | |
|---|---|---|---|---|---|---|---|---|
| **Outcomes** | **All (N = 470)** | **10 mm (n = 86)** | **8 mm (n = 384)** | ***p* values** | **<7 mmHg (n = 130)** | **7-13 mmHg (n = 321)** | **>13 mmHg (n = 19)** | ***p* values** |
| Overt hepatic encephalopathy, n (%) | 151 (32.1%) | 39 (45.3%) | 112 (29.2%) | 0.005 | 54 (41.5%) | 92 (28.7%) | 5 (26.3%) | 0.025 |
| Grade 3-4 | 57 (12.1%) | 16 (18.6%) | 41 (10.7%) | 0.064 | 19 (14.6%) | 36 (11.2%) | 2 (10.5%) | 0.579 |
| More than one episode | 65 (13.8%) | 19 (22.1%) | 46 (12.0%) | 0.022 | 24 (18.5%) | 39 (12.1%) | 2 (10.5%) | 0.215 |
| Portal hypertension complications, n (%) | 83 (17.7%) | 13 (15.1%) | 70 (18.2%) | 0.598 | 19 (14.6%) | 55 (17.1%) | 9 (47.4%) | 0.005 |
| Further bleeding | 49 (10.4%) | 8 (9.3%) | 41 (10.7%) | | 9 (6.9%) | 34 (10.6%) | 6 (31.6%) | |
| Ascites | 29 (6.2%) | 5 (5.8%) | 24 (6.2%) | | 10 (7.7%) | 18 (5.6%) | 1 (5.3%) | |
| Further bleeding + ascites | 5 (1.1%) | 0 (0.0%) | 5 (1.3%) | | 0 (0.0%) | 3 (0.9%) | 2 (10.5%) | |
| Further decompensation, n (%) | 201 (42.8%) | 45 (52.3%) | 156 (40.6%) | 0.063 | 62 (47.7%) | 128 (39.9%) | 11 (57.9%) | 0.125 |
| Further bleeding, n (%) | 54 (11.5%) | 8 (9.3%) | 46 (12.0%) | 0.605 | 9 (6.9%) | 37 (11.5%) | 8 (42.1%) | <0.001 |
| New or worsening ascites, n (%) | 34 (7.2%) | 5 (5.8%) | 29 (7.6%) | 0.74 | 10 (7.7%) | 21 (6.5%) | 3 (15.8%) | 0.234 |
| Mortality, n (%) | 63 (13.4%) | 12 (14.0%) | 51 (13.3%) | 0.993 | 21 (16.2%) | 38 (11.8%) | 4 (21.1%) | 0.278 |
| Causes of death, n (%) | | | | 0.757 | | | | 0.286 |
| Liver failure | 29 (6.2%) | 7 (8.1%) | 22 (5.7%) | | 7 (5.4%) | 20 (6.2%) | 2 (10.5%) | |
| Gastrointestinal bleeding | 5 (1.1%) | 0 (0.0%) | 5 (1.3%) | | 1 (0.8%) | 3 (0.9%) | 1 (5.3%) | |
| Sepsis/pneumonia | 3 (0.6%) | 1 (1.2%) | 2 (0.5%) | | 1 (0.8%) | 2 (0.6%) | 0 (0.0%) | |
| Hepatocellular carcinoma | 4 (0.9%) | 1 (1.2%) | 3 (0.8%) | | 1 (0.8%) | 3 (0.9%) | 0 (0.0%) | |
| Multiorgan failure | 11 (2.3%) | 1 (1.2%) | 10 (2.6%) | | 6 (4.6%) | 4 (1.2%) | 1 (5.3%) | |
| Unrelated with liver disease | 8 (1.7%) | 2 (2.3%) | 6 (1.6%) | | 3 (2.3%) | 5 (1.6%) | 0 (0.0%) | |
| Unknown | 3 (0.6%) | 0 (0.0%) | 3 (0.8%) | | 2 (1.5%) | 1 (0.3%) | 0 (0.0%) | |
| Liver transplantation, n (%) | 23 (4.9%) | 3 (3.5%) | 20 (5.2%) | 0.781 | 7 (5.4%) | 14 (4.4%) | 2 (10.5%) | 0.285 |

PPG, portacaval pressure gradient; TIPS, transjugular intrahepatic portosystemic shunt.
Two-tailed Pearson's Chi-squared test and Fisher's exact test were used to compare categorical variables.

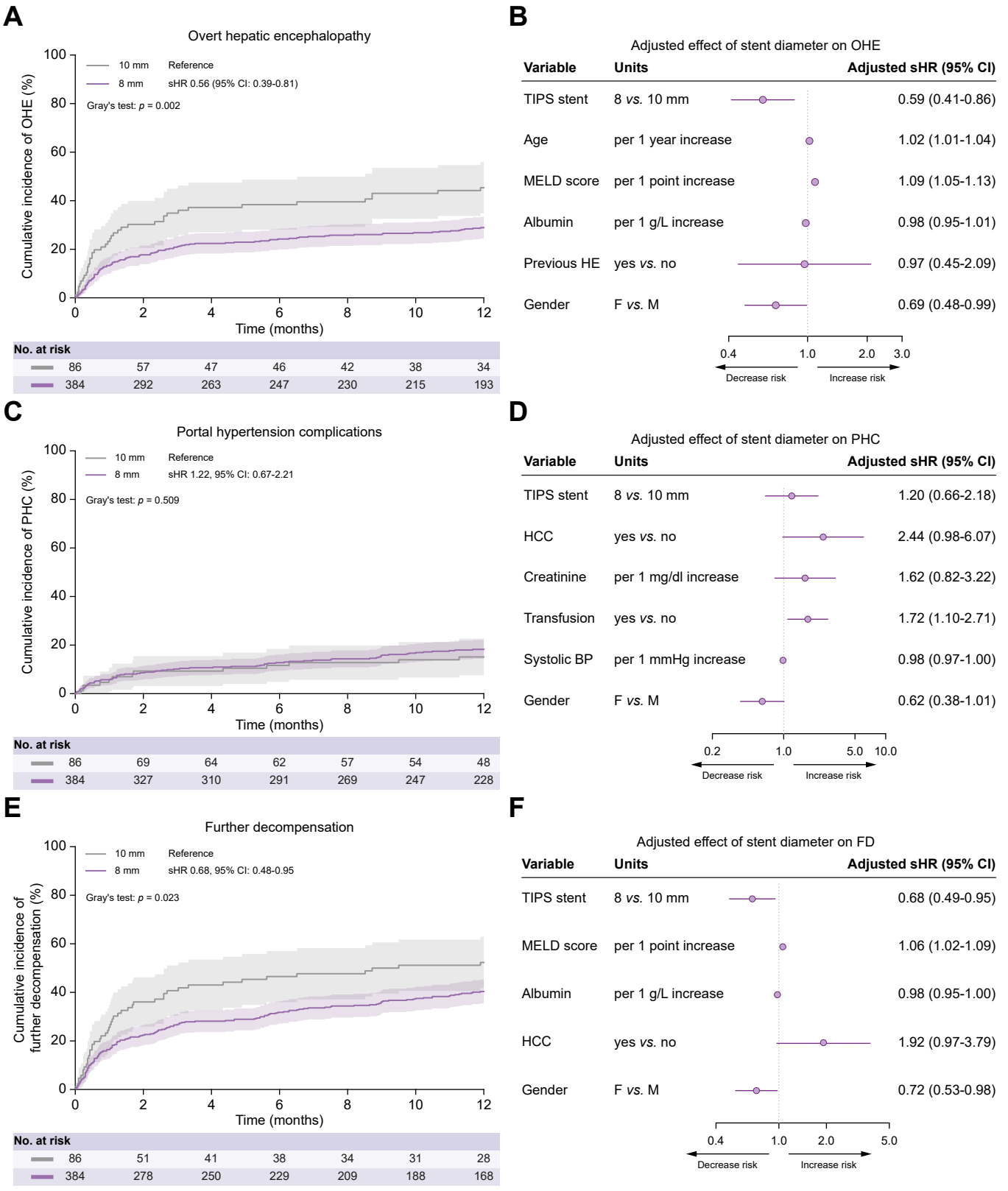


**Fig. 1. Impact of the stent diameter (8 *vs.* 10 mm) on clinical outcomes.** Cumulative incidence curves of (A) overt hepatic encephalopathy, (C) portal hypertension complications, and (E) further decompensation according to the stent diameter (8 *vs.* 10 mm). Forest plot showing the adjusted effect of stent diameter (8 *vs.* 10 mm) on (B) overt hepatic encephalopathy, (D) portal hypertension complications, and (F) further decompensation. Adjusted sHR with 95% CIs are derived from multivariable competing risk regression models. Variables with $p < 0.05$ in univariable analyses and previously reported as potentially influencing the outcomes (regardless of the $p$ value in univariable analysis) were included in the multivariable analysis. sHRs and $p$ values were estimated using competing risks regression analysis of the Fine-Gray subdistribution hazard model. FD, further decompensation; MELD, model for end-stage liver disease; OHE, overt hepatic encephalopathy; PHC, portal hypertension complications; PPG, portacaval pressure gradient; sHR, subdistribution hazard ratios; TIPS, transjugular intrahepatic portosystemic shunt.

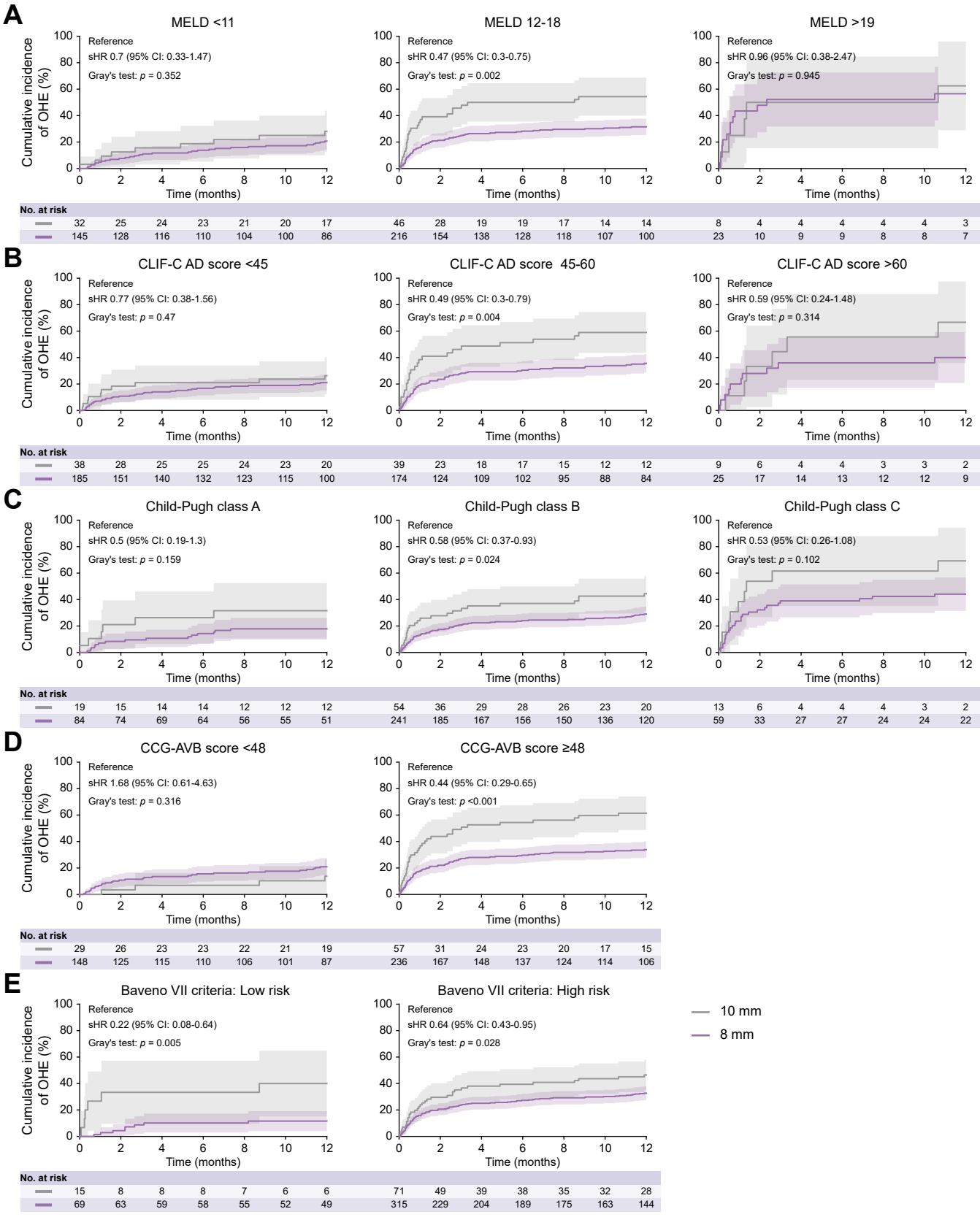


**Fig. 2. Comparative effects of 8-mm *vs.* 10-mm stent diameters on overt hepatic encephalopathy across different risk stratification systems.** Cumulative incidence of overt hepatic encephalopathy at 1 year by stent diameter (8 *vs*. 10 mm) stratified according to (A) MELD 11-19 rules, (B) CLIF-C AD score, (C) Child-Pugh class, (D) CCG-AVB48 criteria, and (E) Baveno VII criteria. Subgroup analyses assessed homogeneity of treatment effects using interaction tests (all *p*-interaction >0.20). Point estimates consistently favor 8-mm stents across subgroups, though statistical significance is not reached in smaller strata due to limited power. Within-

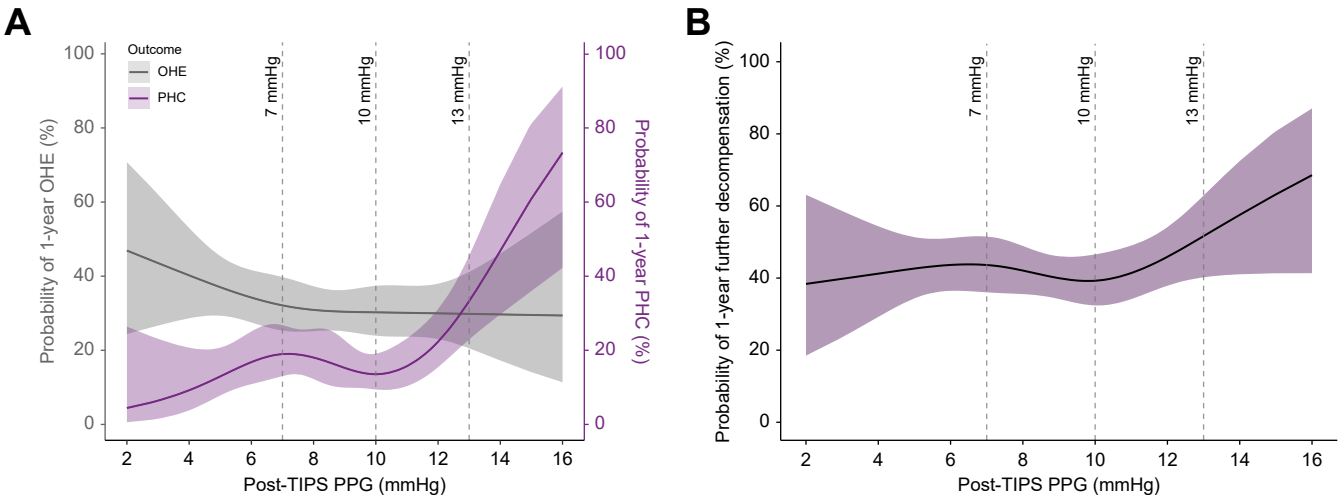


**Fig. 3. Association of post-TIPS PPG with 1-year clinical outcomes.** (A) The spline curves show the probability of 1-year OHE (blue curve, left y-axis), and 1-year PHCs (including variceal bleeding and ascites, red curve, right y-axis) associated with post-TIPS PPG. Shaded regions (light blue for OHE, light red for PHC) indicate 95% confidence intervals. Vertical dashed lines mark clinically relevant PPG thresholds (7, 10, and 13 mmHg). OHE probability decreases with higher PPG while PHC probability increases sharply at PPG >10 mmHg. (B) The spline curve depicts the probability of 1-year further decompensation (further bleeding, new or worsening ascites, hepatic encephalopathy) with post-TIPS PPG. A U-shaped relationship is observed: minimal risk at moderate PPG (10 mmHg), rising at extremes. Gray shading represents 95% confidence intervals. Non-linear relationships between post-TIPS PPG and outcomes were modeled using restricted cubic splines with four knots. OHE, over hepatic encephalopathy; PHC, portal hypertension complications; PPG, portacaval pressure gradient; TIPS, transjugular intrahepatic portosystemic shunt.

to further decompensation, with 6-week and 1-year rates of 20.3% *vs.* 32.6% and 40.4% *vs.* 52.3%, respectively (sHR 0.68, 95% CI 0.48–0.95, $p$ = 0.023; Fig. 1E). The other outcomes, including PHCs (6-week: 7.8% *vs.* 7.0%; 1-year: 18.2% *vs.* 15.1%; sHR 1.22, 95% CI 0.67–2.21, $p$ = 0.509; Fig. 1C), further bleeding (6-week: 6.0% *vs.* 4.7%; 1-year: 12.0% *vs.* 9.3%; sHR 1.31, 95% CI 0.62–2.77, $p$ = 0.471; Fig. S8A), new or worsening ascites (6-week: 2.1% *vs.* 2.3%; 1-year: 7.6% *vs.* 5.8%; sHR 1.29, 95% CI 0.50–3.37, $p$ =0.594; Fig. S8C), and mortality (6-week: 2.9% *vs.* 5.8%; 1-year: 13.3% *vs.* 14.0%; sHR 0.93, 95% CI 0.49–1.75; $p$ = 0.813, Fig. S8E), did not differ significantly between groups. Multivariable analyses adjusted for confounders confirmed these findings (Figs 1 and S8), with adjusted sHR of 0.59 (95% CI 0.41–0.86, $p$ = 0.005) for OHE and 0.68 (95% CI 0.49–0.95, $p$ = 0.023) for further decompensation. Analysis of available ammonia data (n = 132) suggests a trend toward lower levels in patients receiving 8-mm stents (Fig. S9). Subgroup analyses across MELD strata, CLIF-C AD strata, Child-Pugh classes, CCA-AVB score, Baveno VII criteria, early-TIPS criteria, Baveno VII clinical criteria, and pre-TIPS PPG/HVPG demonstrated consistent treatment effects (interaction test $p$ >0.2 for all; Figs 2 and S10–S14).

### Post-TIPS PPG thresholds and clinical outcomes

As shown in Table S1–S2 and Fig. S15–S16, validation of the conventional 10–12 mmHg hemodynamic target confirmed its association with reduced PHCs compared with PPG >12 mmHg (adjusted sHR 4.08, 95% CI 2.07–8.07; $p$ <0.001). However, no protective effect against OHE was observed relative to PPG <10 mmHg (adjusted sHR 1.01, 95% CI 0.70–1.46; $p$ = 0.449). Restricted cubic spline analysis revealed non-linear relationships between post-PPG and outcomes: The risk of PHCs increased with rising PPG (positive correlation), while OHE risk decreased inversely (Fig. 3A). Further decompensation exhibited a U-shaped risk curve, reaching minimal incidence at 10 mmHg (Fig. 3B). Based on inflection points in the spline curves, 7–13 mmHg was identified as a potential reference zone associated with balanced clinical risks. Stratification into PPG categories (<7 mmHg, n = 130; 7–13 mmHg, n = 321; >13 mmHg, n = 19; Table S3–S4) demonstrated superior outcomes in the 7–13 mmHg group (Figs 4 and S17), with reduced OHE risk *vs.* the <7 mmHg group (adjusted sHR 1.43, 95% CI 1.01–2.01) and lower PHC incidence *vs.* the >13 mmHg group (adjusted sHR 2.76, 95% CI 1.34–5.68). The 7–13 mmHg group also showed comparable PHC incidence to the <7 mmHg group (adjusted sHR 0.83,

subgroup $p$ values are provided for transparency but should not be interpreted as evidence for or against differential efficacy. sHRs and $p$ values were estimated using competing risks regression analysis of the Fine-Gray subdistribution hazard model. Note: Baveno VII criteria: Low risk: Child-Pugh A, Child-Pugh B without active bleeding at initial endoscopy or Child-Pugh B7 points with active bleeding at endoscopy, or HVPG <20 mmHg at the time of bleeding; High risk: Child-Pugh B >7 points with active bleeding at endoscopy and Child-Pugh class C <14 points or HVPG ≥20 mmHg at the time of bleeding. CLIF-C ADs =10 * ([0.03* age; years] + [0.66* ln {creatinine; mg/dl}] + [1.71 * ln {INR}] + [0.88* ln {WBC; $10^9$ cell/L}] - [0.05*Na; mmol/L] + 8). CCG-AVBs =10 × [0.0289 × (Age, years) -0.0525 × (Albumin, g/L) + 0.3334 × Ln (Bilirubin, mg/dl) + 1.7631 × Ln (INR) + 0.3373 × Ln (WBC, $10^9$/L) + 0.4509 × Ln (Creatinine, mg/dl) - 0.0294 × (Sodium, mmol/L) + 8]. CCG-AVB score, Chinese Collaboration Group-acute variceal bleeding score; CLIF-C AD, Chronic Liver Failure-Consortium acute decompensation; HVPG, hepatic venous pressure gradient; INR, international normalized ratio; MELD, model for end-stage liver disease; PPG, portacaval pressure gradient; sHR, subdistribution hazard ratio; TIPS, transjugular intrahepatic portosystemic shunt; WBC, white blood cell.

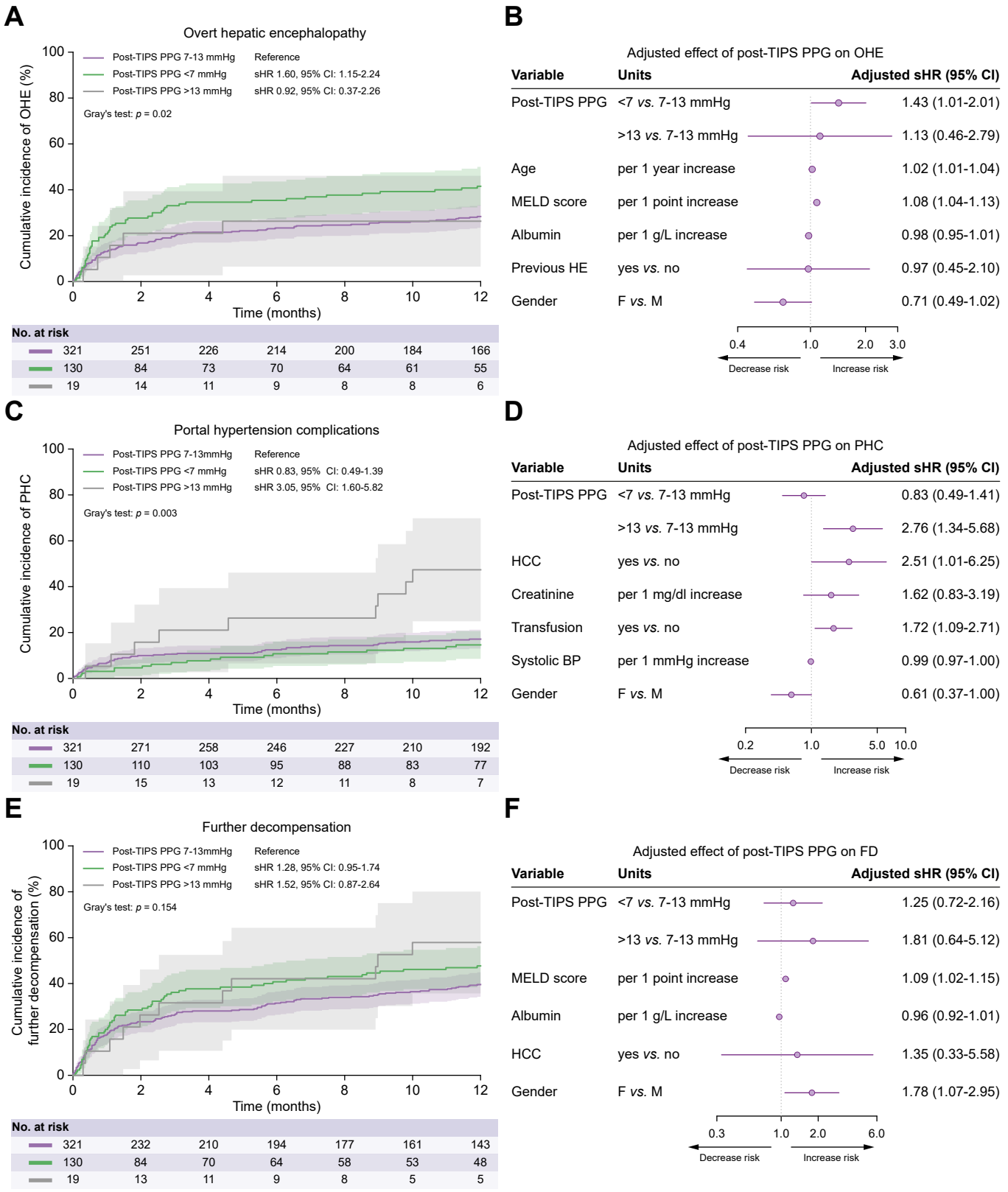


**Fig. 4. Impact of the post-TIPS PPG values, according to the threshold of 7 and 13 mmHg, on clinical outcomes.** Cumulative incidence curves of (A) overt hepatic encephalopathy, (C) portal hypertension complications, and (E) further decompensation according to post-TIPS PPG with the threshold of 7 and 13 mmHg. Forest plot showing the adjusted effect of post-TIPS PPG according to the threshold of 7 and 13 mmHg on (B) overt hepatic encephalopathy, (D) portal hypertension complications, and (F) further decompensation. Adjusted sHR with 95% CIs are derived from multivariable competing risk regression models. Variables with $p$ <0.05 in univariable analyses and previously reported as potentially influencing the outcomes (regardless of the $p$ value in univariable analysis) were included in the multivariable analysis. sHRs and $p$ values were estimated using competing risks regression analysis of the Fine-Gray subdistribution hazard model. FD, further

95% CI 0.49–1.41) and similar OHE incidence to the >13 mmHg group (adjusted sHR 1.13, 95% CI 0.46–2.79). Subgroup analyses (Figs 5 and S15-S22) revealed a lower risk of further decompensation in the 7–13 mmHg group compared to other categories in high-risk patients according to CCA-AVB score or Baveno VII criteria. Evaluation of relative PPG reduction indicated that moderate reductions (30–60% from baseline) optimally balanced PHC prevention and OHE risk mitigation (Fig. S23–S25).

### Association between stent diameter and post-TIPS PPG

Both stent diameters achieved significant PPG reduction post-TIPS (8-mm: 23.9 ± 5.8 to 8.8 ± 2.6 mmHg; 10-mm: 23.0 ± 5.9 to 7.5 ± 2.6 mmHg; both $p$ <0.001; Fig. 6A). Despite comparable pre-TIPS PPG (23.9 *vs.* 23.0 mmHg; $p$ = 0.139), the 8-mm group exhibited higher post-TIPS PPG (8-mm *vs*. 10-mm: 8.8 *vs.* 7.5 mmHg; $p$ <0.001), smaller relative reductions (61.6% *vs.* 66.0%; $p$ = 0.008; Fig. 6B) and fewer patients with PPG <7 mmHg (24.2 % *vs.* 42.0%; $p$ = 0.001; Fig. 6C). A greater proportion of patients in the 8-mm group attained the 7-13 mmHg target range (71.1%) than those in the 10-mm group (55.8%). Univariable and multivariable analysis identified stent diameter (8-mm *vs*. 10-mm: odds ratio [OR] 0.43, 95% CI 0.26–0.71), MELD scores (OR 1.07 per point increase, 95% CI 1.01–1.13), pre-TIPS PPG (OR 0.95 per mmHg increase, 95% CI 0.92–0.98) and shock at admission (OR 1.64, 95% CI 0.98–2.76) as independent predictors of PPG <7 mmHg (Figs 6D and S26). A nomogram incorporating these variables demonstrated satisfactory predictive accuracy (Fig. S27).

### Sensitivity analyses

Similar results were observed in sensitivity analyses, including propensity-matched cohorts (Fig. S28–S29, Table S5–S6) and analyses restricted to guideline-defined high-risk subgroups (Child-Pugh B >7 with active bleeding or Child-Pugh C <14, ± pre-TIPS PPG ≥20 mmHg, Fig. S30–S33, Table S9–S10), demonstrating consistent treatment effects.

## Discussion

This multicenter observational study aimed to optimize stent selection and hemodynamic targets for pre-emptive TIPS in patients with cirrhosis and AVB. The principal findings of the present study are: (1) the use of 8-mm stents significantly reduced OHE incidence and further decompensation risk compared to 10-mm stents, without compromising therapeutic efficacy in portal hypertension control. (2) Exploratory analysis revealed a non-linear relationship between immediate post-procedural PPG values and clinical outcomes, suggesting 7–13 mmHg as a potentially pragmatic intraprocedural target range: values below 7 mmHg were associated with increased encephalopathy risk, while gradients exceeding 13 mmHg correlated with heightened PHCs. (3) Technical feasibility was demonstrated with 71.1% of 8-mm stent recipients achieving this target range, suggesting most patients benefit from standardized small-diameter shunts while preserving options for selective expansion.

Our study included consecutive patients with AVB undergoing pre-emptive TIPS, irrespective of Child-Pugh class, to overcome limitations of conventional high-risk definitions. While guidelines emphasize Child-Pugh B >7 with active bleeding or Child-Pugh C <14 points, significant evidence supports alternative high-risk markers independent of Child-Pugh scores and active bleeding. Crucially, pre-TIPS PPG ≥20 mmHg (a Baveno VII-endorsed criterion strongly linked to mortality) occurred in 68.9% of Child-Pugh A and 74.6% of Child-Pugh B patients, indicating many are hemodynamically high-risk despite their Child-Pugh classification (Fig. S33). Furthermore, other established risk tools (*e.g.* MELD >19, CCG-AVB score >48) can identify high-risk patients missed by Child-Pugh criteria alone (Fig. S34). Finally, sensitivity analyses restricted to guideline-defined high-risk subgroups confirmed the robustness of our primary results across the spectrum of severity. This inclusive design enabled a comprehensive evaluation of risk stratification beyond current recommendations.

The mortality reduction associated with pre-emptive TIPS in high-risk patients with AVB is well-established,[5–7,23] yet post-TIPS hepatic encephalopathy remains a persistent challenge. Our findings extend prior evidence by demonstrating that 8-mm stents reduce OHE incidence by 43% at 1 year compared to 10-mm stents, independent of traditional predictors such as MELD score, age, and HE history.[24] This protective effect aligns with our earlier randomized trial in elective TIPS populations, where 8-mm stents halved spontaneous OHE risk without increasing shunt dysfunction or rebleeding.[10] Mechanistically, the reduced portosystemic shunting with smaller stents likely attenuates systemic exposure to gut-derived toxins (*e.g.* ammonia) while preserving hepatic perfusion – a balance particularly critical in pre-emptive TIPS candidates who inherently face dual risks of rebleeding (most patients have a HVPG >20 mmHg) and encephalopathy (most patients have poor liver function). Notably, our results contrast with studies favoring 10-mm stents in refractory ascites cohorts.[11,12] For instance, Riggio *et al.*[12] reported comparable OHE rates between 8-mm and 10-mm groups but higher ascites recurrence with smaller stents, while Miraglia *et al.*[11] observed increased paracentesis needs in 8-mm recipients. These discrepancies underscore the importance of indication-specific stent selection: variceal bleeding prioritizes encephalopathy prevention, whereas ascites management demands maximal portal decompression.[25] This dichotomy may explain the conflicting outcomes across populations and highlights the need for tailored hemodynamic targets based on TIPS indications. Furthermore, the reduction in further decompensation with 8-mm stents was mainly driven by a lower incidence of HE, without significant effects on PHCs or mortality. This underscores encephalopathy as a key modifiable endpoint in pre-emptive TIPS, particularly for patients prioritizing quality of life. The absence of difference in mortality aligns with the established risk-benefit profile of TIPS, where stent diameter modulates encephalopathy risk but not survival.[10]

The observed association between immediate post-TIPS PPG values and clinical outcomes provides insights for intraprocedural decision-making, though methodological considerations warrant careful interpretation. Our data indicate that aggressive PPG

decompensation; MELD, model for end-stage liver disease; OHE, overt hepatic encephalopathy; PHC, portal hypertension complications; PPG, portacaval pressure gradient; sHR, subdistribution hazard ratios; TIPS, transjugular intrahepatic portosystemic shunt.

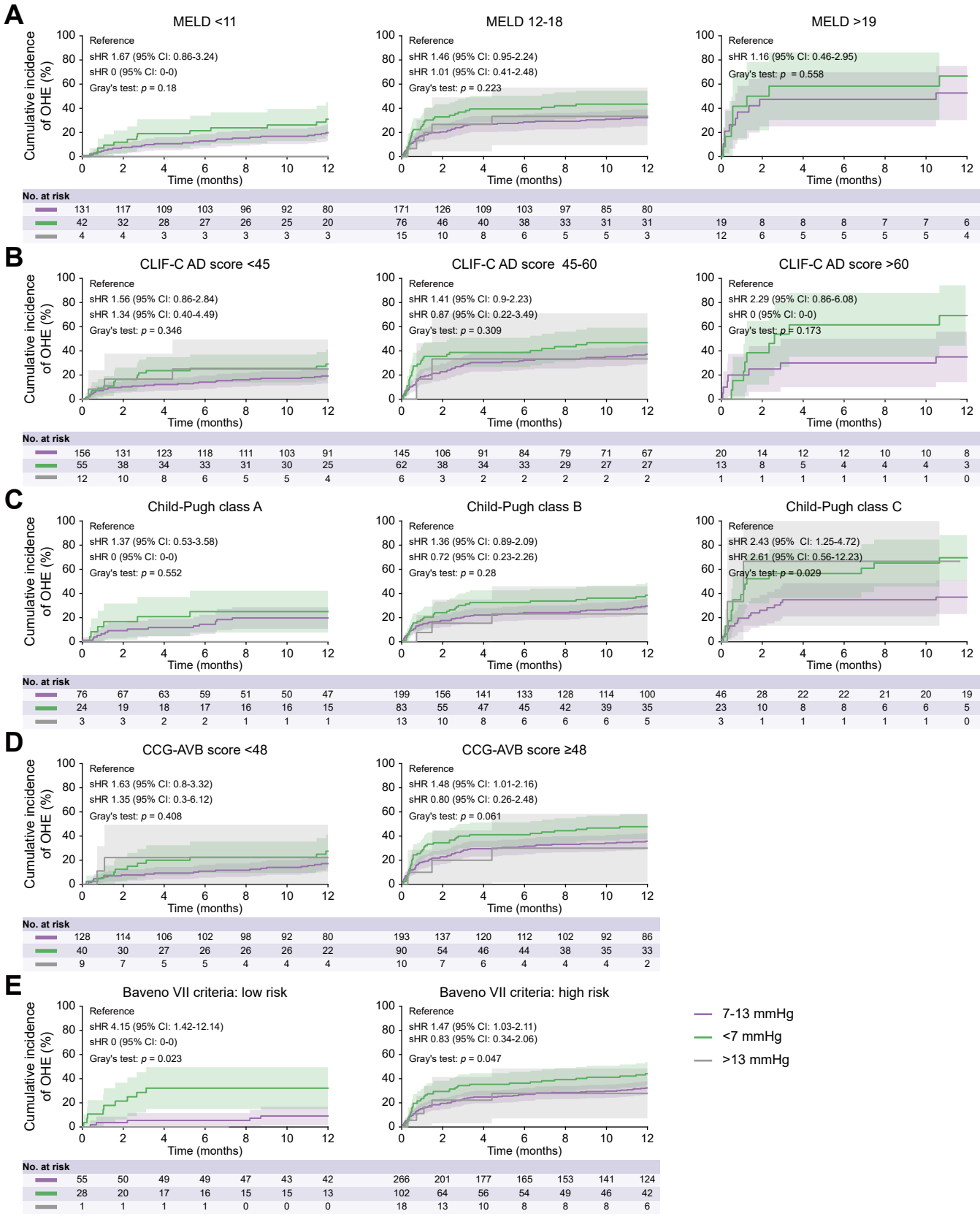


**Fig. 5. Subgroup analysis of the effect of post-TIPS PPG values, according to the threshold of 7 and 13 mmHg, on the overt hepatic encephalopathy according to different risk stratification rules.** Cumulative incidence of overt hepatic encephalopathy at 1 year by post-TIPS PPG values, with threshold of 7 and 13 mmHg, stratified according to (A) MELD 11-19 rules, (B) CLIF-C AD score, (C) Child-Pugh class, (D) CCG-AVB48 criteria, and (E) Baveno VII criteria. Subgroup analyses assessed homogeneity of treatment effects using interaction tests (all *p*-interaction >0.20). Point estimates consistently favor 8-mm stents across

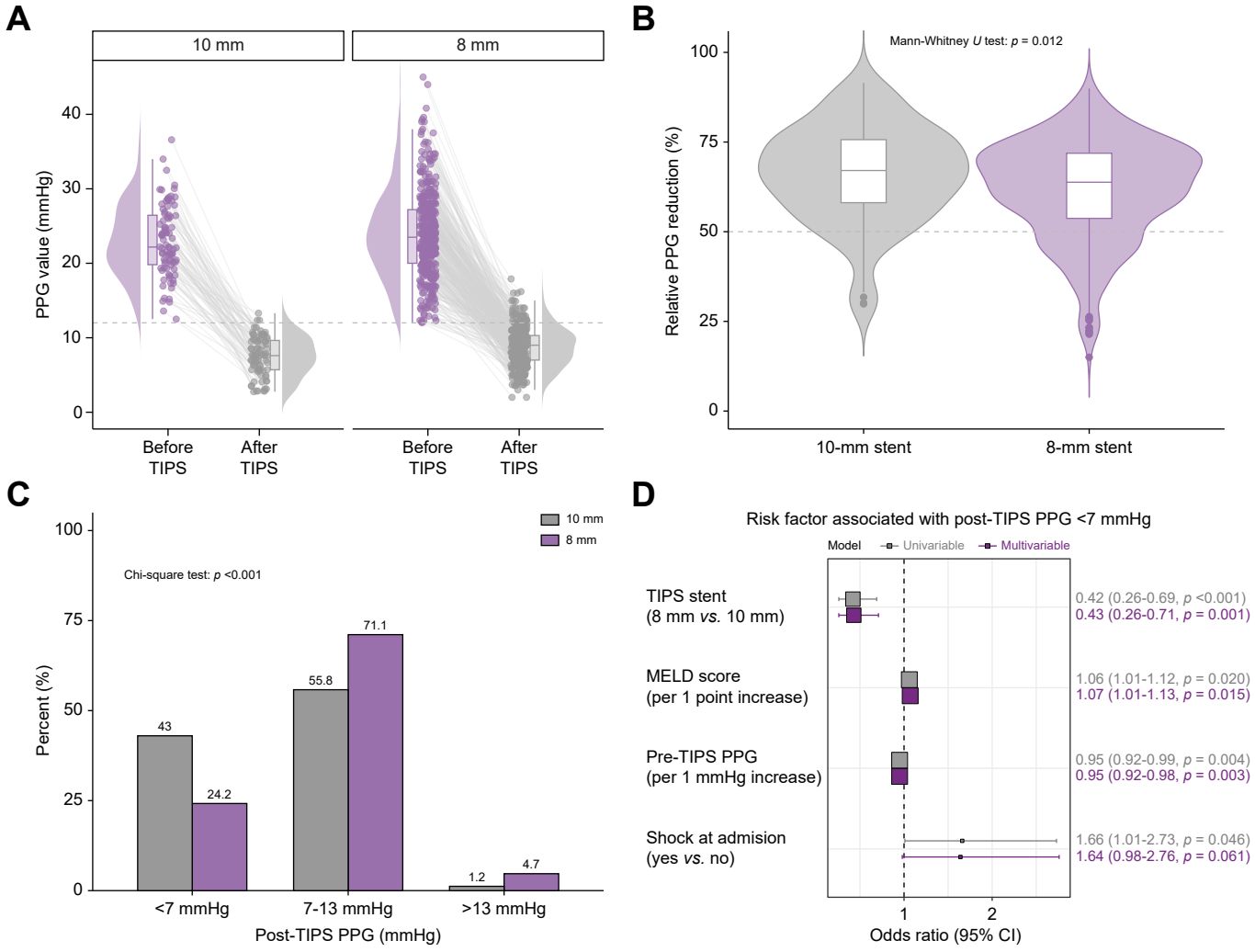


**Fig. 6. Portacaval pressure gradient changes after TIPS placement and predictors of post-TIPS PPG <7 mmHg.** (A) Paired comparison of pre-TIPS and post-TIPS PPG in each group; (B) Comparison of relative pressure reduction between two groups (*p* values were estimated with Mann-Whitney *U* test). (C) Proportion of patients with post-TIPS PPG <7, 7-13, >13 mmHg between groups (*p* values were estimated with two-tailed Pearson's Chi-squared test), (D) Predictors of post-TIPS PPG <7 mmHg. ORs were estimated with Logistic regression analysis. OR, odds ratio; PPG, portacaval pressure gradient; TIPS, transjugular intrahepatic portosystemic shunt.

reduction (<7 mmHg) correlates with increased encephalopathy risk, while conservative targets (>13 mmHg) show reduced efficacy against PHCs. Importantly, the 7–13 mmHg range emerged as a potentially favorable balance point when considering these competing risks. This aligns with physiological principles: sufficient decompression to prevent bleeding while avoiding excessive shunting. However, we acknowledge that immediate PPG measurements may be transiently influenced by procedural factors including sedation and vasoactive medications. Recent evidence also suggests PPG may drift post-procedure.[9,26] Therefore, we emphasize that these findings should be interpreted as providing guidance for intraprocedural calibration rather than definitive prognostic thresholds. Future studies incorporating PPG measurements at least 24 h post-procedure under stable conditions are needed. Furthermore, the 7–13 mmHg PPG target is most robust in the overall cohort, with subgroup-specific nuances warranting further validation in larger prospective studies.

Achieving the 7–13 mmHg target with 8-mm stents proved feasible in 71.1% of patients, suggesting standardized small-diameter shunts suffice for most cases. However, 24.2% of patients still developed PPG <7 mmHg despite 8-mm stents,

subgroups, though statistical significance is not reached in smaller strata due to limited power. Within-subgroup *p* values are provided for transparency but should not be interpreted as evidence for or against differential efficacy. sHRs and *p* values were estimated using competing risks regression analysis of the Fine-Gray subdistribution hazard model. Note: Baveno VII criteria: Low risk: Child-Pugh A, Child-Pugh B without active bleeding at initial endoscopy or Child-Pugh B7 points with active bleeding at endoscopy, or HVPG <20 mmHg at the time of bleeding; High risk: Child-Pugh B >7 points with active bleeding at endoscopy and Child-Pugh class C <14 points or HVPG ≥20 mmHg at the time of bleeding. CLIF-C ADs =10 * ([0.03* age; years] + [0.66* ln {creatinine; mg/dl}] + [1.71 * ln {INR}] + [0.88* ln {WBC; $10^9$ cell/L}] - [0.05*Na; mmol/L] + 8). CCG-AVBs =10 × [0.0289 × (Age, years) -0.0525 × (Albumin, g/L) + 0.3334 × Ln (Bilirubin, mg/dl) + 1.7631 × Ln (INR) + 0.3373 × Ln (WBC, $10^9$/L) + 0.4509 × Ln (Creatinine, mg/dl) - 0.0294 × (Sodium, mmol/L) + 8]. CCG-AVB score, Chinese Collaboration Group-acute variceal bleeding score; CLIF-C AD, Chronic Liver Failure-Consortium acute decompensation; HVPG, hepatic venous pressure gradient; INR, international normalized ratio; MELD, model for end-stage liver disease; PPG, portacaval pressure gradient; sHR, subdistribution hazard ratio; TIPS, transjugular intrahepatic portosystemic shunt; WBC, white blood cell.

underscoring the necessity for individualized calibration in high-risk subgroups. We identified predictors of over-shunting (*e.g.* shock, low pre-TIPS PPG), advocating stepwise dilation starting with 6-mm balloons in these scenarios – a strategy validated by Schepis *et al.*,[27] who demonstrated reduced OHE with under-dilated stents. Conversely, 4.7% of patients with PPG >13 mmHg after 8-mm stents may benefit from incremental expansion, though this must be weighed against encephalopathy risks.

The lower post-TIPS PPG observed with 8-mm stents in our cohort diverges from previous reports showing equivalent gradients across stent sizes.[28] This discrepancy likely reflects our larger sample size and exclusive focus on pre-emptive TIPS populations, where hemodynamic profiles differ from elective TIPS or ascites-driven cohorts. Additionally, the 1-mm gradient difference, though statistically significant, may lack clinical relevance in isolation. However, when contextualized with OHE risk, which increased exponentially below 7 mmHg, this subtle variation underscores the importance of millimeter-level precision in stent calibration. A three-arm design comparing 6-mm, 8-mm, and 10-mm stents with protocolized PPG targets (7–13 mmHg) could definitively establish optimal stent-sizing strategies. Concurrently, technological innovations in adjustable stents and non-invasive PPG monitoring may enable real-time hemodynamic optimization, transforming TIPS from a static intervention into a dynamic therapy.[29]

Our findings must be interpreted within the study's limitations. First, the observational design introduces potential selection bias, though this was minimized by consecutive enrollment and rigorous adjustment for confounders. Second, despite employing multivariable modeling to adjust for recognized potential confounders, unmeasured or unaccounted-for confounding factors (such as sarcopenia, large spontaneous portosystemic shunts) may persist, potentially impacting the validity of our findings. Third, the absence of 6-mm stents precludes direct comparison with ultra-small shunts, which may benefit high-risk subgroups. Fourth, the predominance of viral etiologies of cirrhosis (62% HBV-related) limits generalizability to Western cohorts where alcohol- or metabolic (dysfunction)-associated steatotic liver disease predominate.[30] Fifth, variceal embolization was performed in 42.8% of patients, which exceeds some published series. Nevertheless, it was balanced between stent groups and did not independently predict HE in adjusted models. Sixth, the reliance on immediate post-TIPS PPG (potentially influenced by sedation, vasoactive agents, or acute procedural conditions) leaves uncertainty about long-term hemodynamic stability and limits its utility as a definitive prognostic marker. Finally, while we hypothesize that reduced portosystemic shunting with 8-mm stents attenuates systemic ammonia exposure, this study lacked protocolized ammonia measurements to confirm this mechanism. Future prospective studies should incorporate serial ammonia assessments to directly link hemodynamic targets to toxin clearance.

In conclusion, the present study shows that in patients with AVB receiving pre-emptive TIPS management: (1) 8-mm stents offer advantages in reducing encephalopathy risk while maintaining therapeutic efficacy compared to larger diameters; (2) a 7–13 mmHg PPG range, measured intraprocedurally, helps guide initial stent calibration to balance complication risks, though its validity as a long-term prognostic target requires further validation with delayed measurements; and (3) specific high-risk subgroups (*e.g.* patients with shock or low baseline PPG) might benefit from tailored strategies such as ultra-small stents or staged dilation.


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## Abbreviations

AVB, acute variceal bleeding; CCG-AVB score, Chinese Collaboration Group-AVB score; CLIF-C AD, Chronic Liver Failure-Consortium acute decompensation; HE, hepatic encephalopathy; HVPG, hepatic venous pressure gradient; MELD, model for end-stage liver disease; MELD-HE, model for end-stage liver disease hepatic encephalopathy score; INR, international normalized ratio, OHE, overt hepatic encephalopathy; PHCs, portal hypertensive complications; PPG, portacaval pressure gradient; sHR, subdistribution hazard ratios; TIPS, transjugular intrahepatic portosystemic shunt.


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## Conflict of interests

The authors of this study declare that they do not have any conflict of interest.

Please refer to the accompanying ICMJE disclosure forms for further details.

## Authors' contributions

Study concept and design: Yong Lv, Guohong Han; Acquisition of data: Yong Lv, Junjun Ye, Wei Bai, Yan Zhao, Jianbo Zhao, Xuan Zhu, Zaibo Jiang, Hui Xue, Yuzheng Zhuge, Chunqing Zhang, Pengxu Ding, Xiaoli Zhu, Weixin Ren, Tao Yang, Jun Wang, Wenguang Zhang, Kai Li, Zhengyu Wang, Bohan Luo, Xiaomei Li, Na Zhang, Zhiping Yang, Wengang Guo, Dongdong Xia, Huahong Xie, Yanglin Pan, Yongzhan Nie, Zhanxin Yin, Daiming Fan, Guohong Han; Analysis and interpretation of data: Yong Lv, Guohong Han; Drafting of the manuscript: Yong Lv; Critical revision of the manuscript for important intellectual content: Huahong Xie, Yanglin Pan, Daiming Fan, Guohong Han; Statistical analysis: Yong Lv; Administrative and material support: Daiming Fan.

## Data availability

The data of this study are available under a transfer agreement from the corresponding author based on a reasonable request.

## Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jhepr.2025.101591.

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