VOLUME 84 ISSUE 1, MAY 2026

EASL CONGRESS

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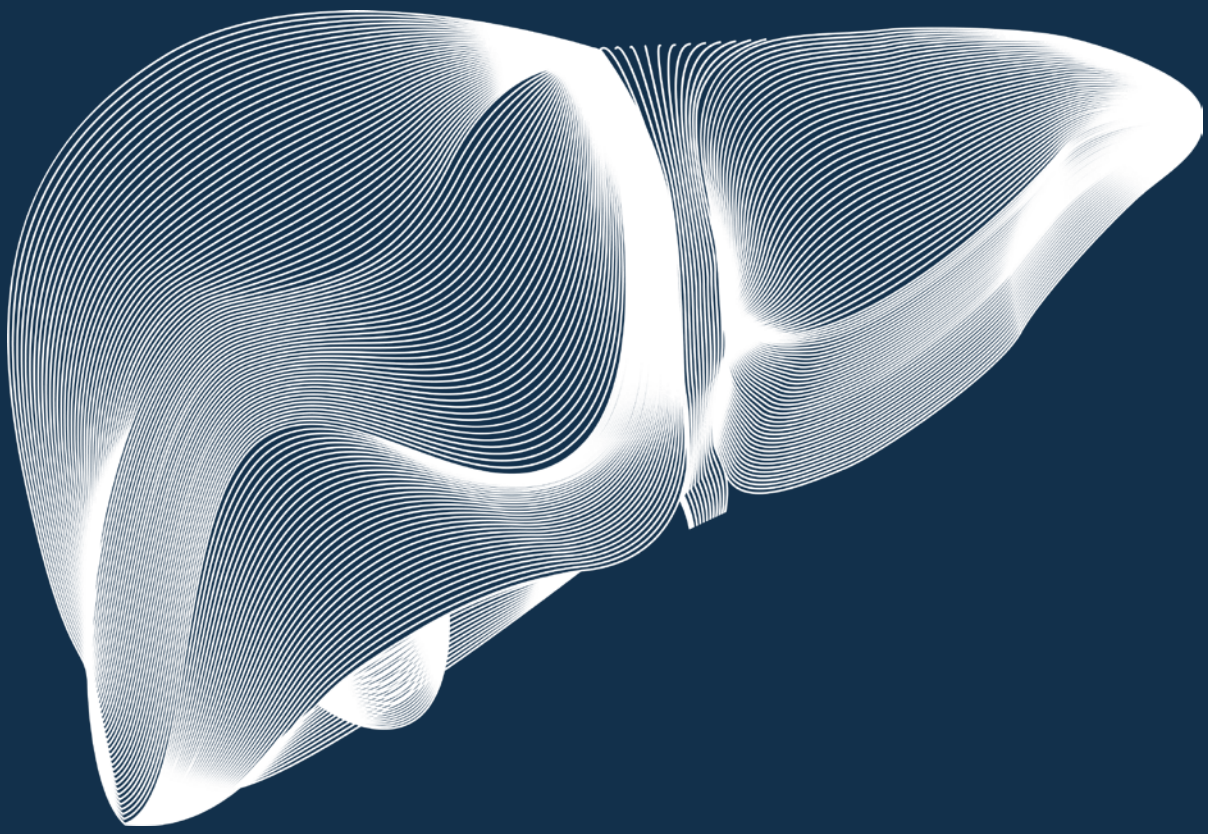

ABSTRACT BOOK

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JOURNAL OF HEPATOLOGY

VOLUME **84**, SUPPLEMENT **S1**, PAGES **S1–S1108**

Abstract Book of EASL Congress 2026
27–30 May 2026, Barcelona, Spain

Publication of this Abstract supplement was supported by the European Association for the Study of the Liver (EASL)

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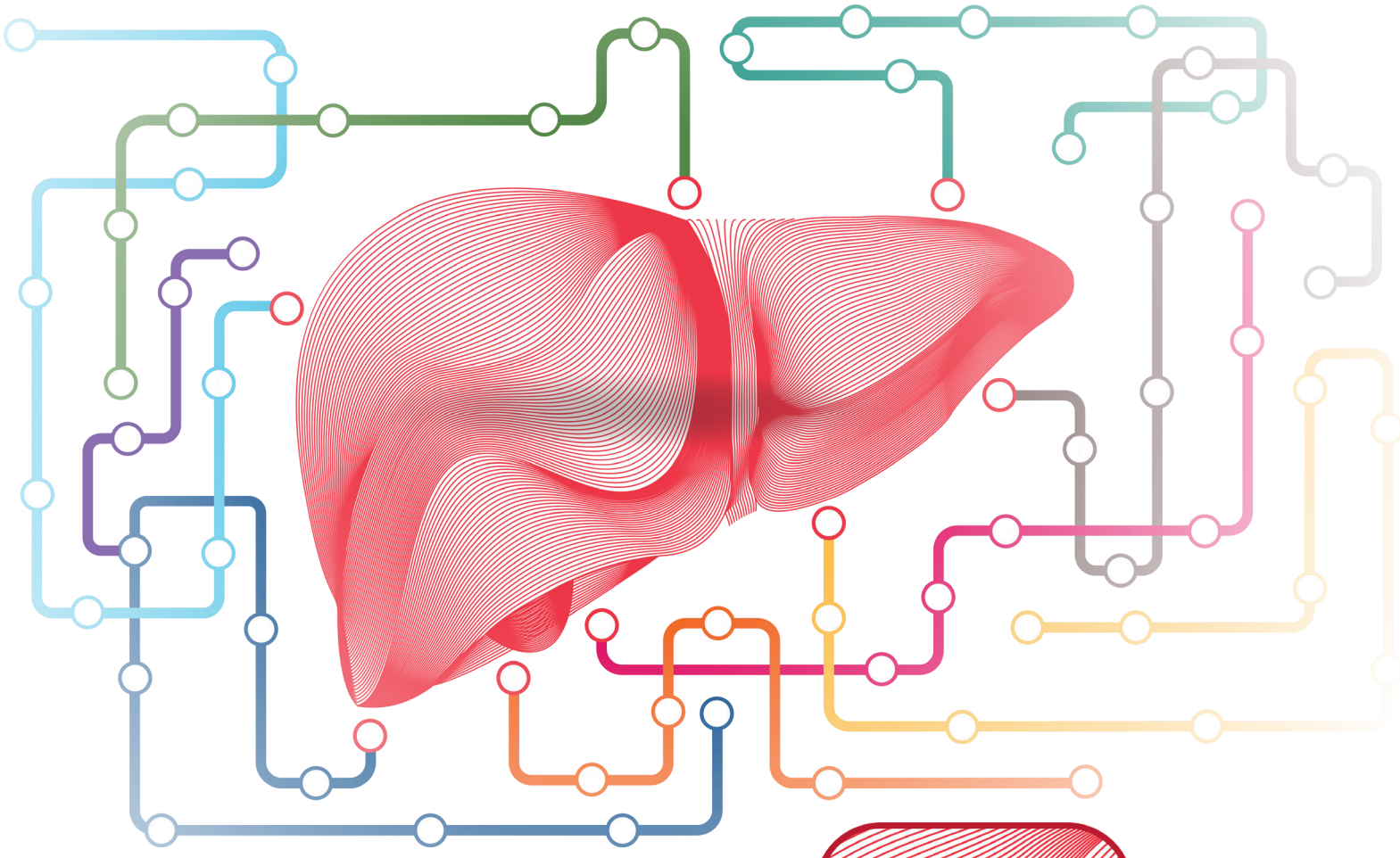

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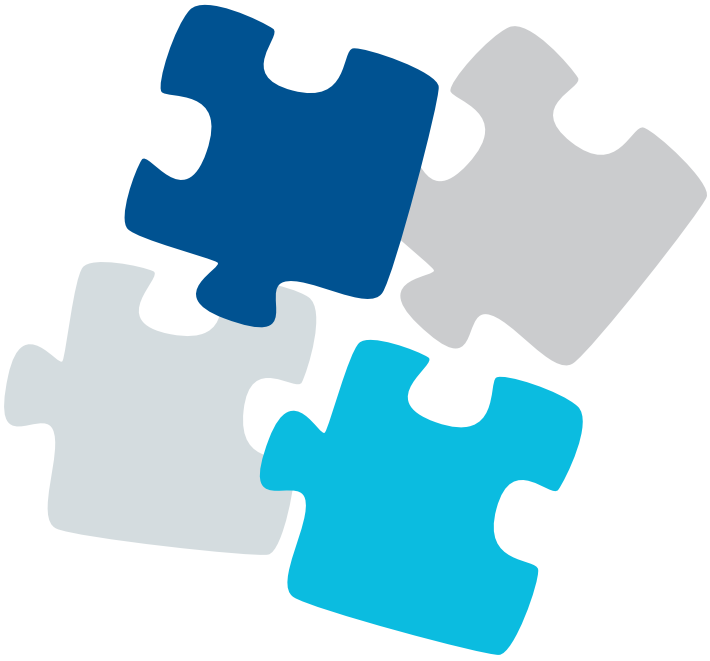

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CONTENTS

Registration of Clinical Trials
The *Journal of Hepatology* endorses the policy of the WHO and the International Committee of Medical Journal Editors (ICMJE) on the registration of clinical trials. Therefore, any trial that starts recruiting on or after July 1, 2005 should be registered in a publicly owned, publicly accessible registry and should satisfy a minimal standard dataset. Trials that started recruiting before that date will be considered for publication if registered before September 13, 2005.
More detailed information regarding clinical trials and registration can be found in New Engl J Med 2004; 351:1250–1251 and New Engl J Med 2005; 352:2437–2438.

early (M1) increase in HA after TIPS may reflect reduced sinusoidal clearance and indicates higher risks of HE, ACLF, and death.

THU-465

Splenic hyperkinetic syndrome: a universal driver of cirrhotic and non-cirrhotic portal hypertension

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Background and aims: Portal hypertension (PH) is attributed to increased intrahepatic resistance. However, the role of splanchnic hyperdynamic circulation, specifically excessive inflow, remains underappreciated. We hypothesized that splenic arterial hyperperfusion acts as a universal hemodynamic driver in PH - regardless of etiology - creating a volume overload that the portal system cannot accommodate. This study aims to evaluate the relations between splenic arterial inflow and portal venous flow in patients with established cirrhotic and non-cirrhotic PH.
Method: The study included three groups: 1) 47 patients with liver cirrhosis and cirrhotic PH (CPH); 2) 7 patients with non-cirrhotic PH (NCPH); 3) a control group of 41 healthy volunteers (CG). All patients of the CPH and NCPH groups presented with esophageal and gastric varices and a history of variceal bleeding(s). All participants underwent a Doppler ultrasound assessment. We analyzed spleen volume, splenic artery parameters (PSV, EDV, RI), and maximum linear flow velocities (V_{max}) in the splenic vein and the main portal vein (PV) trunk.
Results: Patients in the CPH group presented with significant splenomegaly (811.6 ± 342.2 cm/s vs. 146.35 ± 56.27 cm^3 in controls; $p < 0.001$) and a severe hyperdynamic state. PSV in the SA was up to 150.84 ± 43.57 cm/s compared to 85.43 ± 26.72 cm/s in controls ($p < 0.001$), with EDV nearly doubling. This arterial surge led to venous overload (SV velocity was 34.1 ± 15.55 cm/s vs. 23.54 ± 6.16 cm/s in CG, $p < 0.001$). A paradoxical deceleration was observed in the main portal vein (29.06 ± 9.43 cm/s vs. 35.67 ± 10.53 cm/s in controls, $p < 0.01$). This «hemodynamic mismatch» is a result of an existing left gastric vein shunt that feeds gastric/esophageal varices, effectively reducing velocity in the main portal trunk.
Conclusion: Our findings reveal distinct hemodynamic changes in patients with clinically significant portal hypertension: massive splenic artery inflow leads to accelerated venous output, which is then diverted to portosystemic collaterals (gastoesophageal varices). This pattern was consistent across both CPH and NCPH cohorts, suggesting that excessive splenic blood flow is a universal pathogenic driver of portal hypertension development. Targeting splenic inflow (e.g. via NSBB treatment, embolization or both) addresses this fundamental mechanism of collateral filling and may be effective regardless of the portal hypertension etiology.

THU-466

The differential effects of terlipressin on serum sodium trajectories are mediated by ascites status, treatment indication and baseline sodium

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Background and aims: Terlipressin is an established therapy for complications of portal hypertension, however concerns persist regarding its potential to cause hyponatremia. Data describing serum sodium (sNa) trajectories during terlipressin therapy across clinical phenotypes are limited.
Method: We conducted a single-centre, retrospective study of consecutive adult patients treated with terlipressin between 2013 and 2024. Patients receiving treatment for < 2 days, with intrinsic renal disease or on renal replacement therapy were excluded. Linear mixed-effects modelling was used to analyse sNa trajectories for up to 14 days of treatment, stratified by ascites status, treatment indication and baseline sNa. Patients with a ≥ 5% sNa decrease from baseline were also evaluated. Severe hyponatremia was defined as sNa < 125 mmol/L.
Results: Across the entire cohort of 414 patients, sNa was observed to increase during terlipressin therapy (adjusted mean sNa change from baseline to day 14: 4 mmol/L, 95% CI 3 to 6, $p < 0.001$). However, changes in sNa differed according to the presence or absence of ascites, baseline sNa, and the indication for use (acute variceal bleeding [AVB]). Adjusted sNa increased over time in patients with ascites but remained stable in those without ascites. Similarly, adjusted sNa increased over time in baseline hyponatremic patients but remained stable in baseline normonatremic patients. An early decline in adjusted sNa was observed in patients with baseline normonatremia, no ascites, and treated with terlipressin for AVB (adjusted mean sNa change from baseline to day 5: −7 mmol/L, 95% CI −9 to −4, $p < 0.001$). 44 patients (11%) experienced a ≥ 5% decline in sNa relative to baseline and were more likely to have higher baseline sNa, lower baseline serum creatinine, lower Model for End-Stage Liver Disease (MELD) and be treated for AVB ($p < 0.001$). Baseline sNa and MELD were independently associated with a ≥ 5% decline in sNa from baseline (adjusted OR 1.13, $p < 0.001$ and adjusted OR 0.91, $p < 0.004$, respectively). Severe hyponatremia was rare, occurring in 10 patients (2%).
Conclusion: Whilst at a population level terlipressin increased sNa, we observed differential effects on sNa according to ascites status, treatment indication and baseline sNa. Clinically significant hyponatremia was uncommon and occurred mainly in patients without ascites, with higher baseline sNa and AVB as the indication for terlipressin, highlighting the need for closer monitoring in this subgroup during therapy.

THU-467

Liver stiffness measurement as an alternative to liver biopsy to rule-out cirrhosis over PSVD in patients with portal hypertension

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