delineate their correlation with HRS reversal (defined as at least 1 SCr level of ≤1.5 mg/dL on treatment), renal replacement therapy (RRT)-free survival, transplant-free survival (TFS), and overall survival (OS). Statistical analyses included the Wald test (logistic regression analysis), Chi-square/Fisher's exact test (HRS reversal), and the log rank test (survival estimates).

Results: SCr levels were significantly associated with HRS reversal in univariate and multivariate logistic regression analyses ($p < 0.001$) (Figure). Among pts with HRS, the incidence of HRS reversal inversely correlated with SCr subgroup: <3 mg/dL, 49.2%; ≥3 to <5 mg/dL, 28.0%; ≥5 mg/dL, 9.1% ($p < 0.001$). At Day 30 follow-up, RRT-free survival was significantly higher for pts with HRS in the lower SCr subgroups versus the highest subgroup (<5 mg/dL vs >5 mg/dL; $p = 0.01$). Terlipressin-treated pts with HRS who had a lower baseline SCr level had higher OS ($p < 0.001$) and TFS at Day 90 ($p = 0.04$).

Baseline parameters	Terlipressin n	Odds ratio	95% CI	p value
SCr	312	0.518	0.381-0.704	<.001
Model for end-stage liver disease score	312	0.939	0.902-0.977	.002
Prior midodrine and octreotide	312	1.849	1.106-3.091	.019

Figure: Multivariate logistic regression of baseline characteristics on HRS reversal (Terlipressin group, pooled intent-to-treat population)

Conclusion: Terlipressin-treated pts with HRS and a lower baseline SCr level experienced higher HRS reversal and survival outcomes. It is important to identify and treat pts with HRS early when they have lower SCr levels and a greater probability of clinical response to terlipressin.

FRI538

Non-invasive tools are suboptimal to predict the presence of varices needing treatment and risk of clinical decompensation in patients with autoimmune hepatitis related cirrhosis

Randeep Rana[1], Samagra Agarwal[1], Sanchit Sharma[1,2], Ujjwal Sonika[3], Syed Ahmed[1], Srikanth Gopi[1], Deepak Gunjan[1], Anoop Saraya[1]. *[1]All India Institute Of Medical Sciences New Delhi, Gastroenterology and Human Nutrition Unit, New Delhi, India; [2]Translational Gastroenterology Unit, John Radcliffe Hospital, Oxford, United Kingdom; [3]Govind Ballabh Pant Hospital, Gastroenterology, New Delhi, India*
Email: ansaraya@yahoo.com

Background and aims: Baveno VII consensus recommend the use of non-invasive tools (NITs) to predict the risk of clinical decompensation (CD) and detecting varices needing treatment (VNTs) in patients with compensated cirrhosis. The present study aimed to validate these recommendations in patients with autoimmune hepatitis (AIH) related cirrhosis.

Method: Diagnostic performance of Baveno-VI (LSM >20 kPa or platelet <1 50 000/mm^3), expanded Baveno-VI (LSM >25 kPa or platelet <1 10 000/mm^3) and platelet-albumin (albumin <3.6 g/dl or platelet <1 20 000/mm^3) criteria for detecting VNTs was assessed against the strategy of endoscopy for all to screen large varices using decision curve analysis (DCA). Patients were stratified using Baveno VII criteria for definite clinically significant portal hypertension (CSPH) (LSM>25 kPa), possible CSPH (LSM>15 kPa and platelet<1 50 000/mm^3) and no CSPH (LSM<15 kPa and platelets >1 50 000/mm^3). Cumulative incidence of clinical decompensations on follow-up were compared across different strata to assess its clinical relevance.

Results: One hundred ten (110) patients with AIH-related cirrhosis were included, with 37 patients having VNTs. At a threshold probability of 10% missed VNTs, platelet-albumin criteria was the most beneficial among NITs to detect VNTs with a potential to avoid 44 additional endoscopies before 1 additional VNT is missed. Rest of the NITs had similar benefit to performing endoscopy in all patients. At lower thresholds (<10% missed VNTs), performing endoscopy in all patients was the most beneficial strategy. Over a median follow-up of 30 months (IQR13–39 months), 30 of 96 patients with available follow-up developed clinical decompensation. The 1-year and 3-year rate of clinical decompensation in patients with definite CSPH, possible CSPH and no CSPH was (29.2%, 29.0%, 19.8%) and (35.6%, 39.5%, 29.0%), respectively (log rank test, $p = 0.75$).

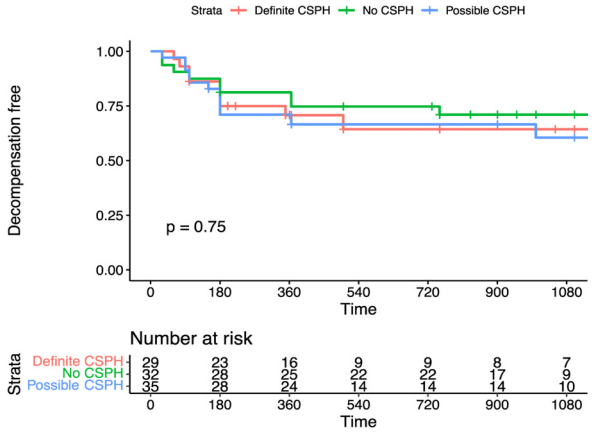

Figure: Figure shows 1-year and 3-year rate of clinical decompensation in patients with definite CSPH, no CSPH and possible CSPH in AIH related cirrhosis.

Conclusion: The strategy of endoscopy for all is superior to all NITs to detect VNTs at lower thresholds for treatment in patients with AIH. The Baveno VII criteria for predicting clinical decompensation is suboptimal to stratify patients of AIH.

FRI539

Improving of hepatic encephalopathy manifestations in cirrhotic patients with clinically significant portal hypertension treated with splenic artery embolization

Sergii Kozlov[1], Pavlo Ivanchov[2], Elina Manzhalii[3], Iryna Kostishyna[4], Oleksanr Kozlov[2], Illia Koshman[5]. *[1]Bogomolets National Medical University, Surgery, Kyyiv, Ukraine; [2]Bogomolets National Medical University, Surgery chair #3, Kyiv, Ukraine; [3]Bogomolets National Medical University, Gastroenterology, Kyiv, Ukraine; [4]Kyiv municipal hospital #12, Surgery, Kyyiv, Ukraine; [5]Kyiv municipal hospital #12, Surgery, Kyyiv*
Email: sergiinikol@gmail.com

Background and aims: In cirrhotic patients, hepatic encephalopathy (HE) is a severe complication in which hyperammonemia plays a leading role. At the same time, clinically significant portal hypertension with spontaneous venous portosystemic shunts is common clinical finding that aggravated the HE manifestation. Endovascular shunt occlusion considered to be a possible treatment option. We studied the efficacy of splenic artery embolization (SAE) in resolution HE symptoms as a perspective alternative RI treatment option for patients with high risk variceal bleeding.

Method: Selective endovascular embolization of the splenic artery was performed as secondary prophylaxis treatment in 120 patients with one or more episodes of variceal bleedings in history. Partial reduction of excessive splenic artery flow (no more than 70% of baseline levels) was achieved by insertion of several (from 2 to 5) flexible coils into the distal portion of the vessel. Serum levels of albumin and ammonia were evaluated before the procedure, 30 days and 12 months after the procedure. Hepatic encephalopathy grade was evaluated according to West Heaven criteria at the same time points. Standard supportive treatment included non-selective beta-blockers (40–60 mg daily) and UDCA (500–1000 mg daily) with dose adjustment according to BMI.

Results: The plasma ammonia levels improved after SAE from 107 ± 34 to 62 ± 25 μmol/l The mean serum albumin pre and post SAE were 29 ± 0.5, 27.8 ± 0.4 (1 month), and 32.5 ± 0.7 g/L (12 months) respectively. West Heaven score mean tended to decrease from 2.2 on the baseline to 1.9 (1 month) and 1.4 (12 months). Thus, basic liver

functions (protein synthesis and ammonia purification) demonstrated considerable improvement in the long-term observation period due to combined treatment. 65 (54.2%) of 120 patients had no bleeding episodes at the endpoint of 12 months, 39 (32.5%) were hospitalized with 1 (28) or more (11) minor bleedings. Fatal bleedings took place in 11 (9.2%) patients and not related to bleeding deaths-in 4 cases.

Conclusion: From our point of view, the improving of HE manifestations after SAE procedure could be explained by synergetic effects of reduction of the excessive splenic flow, restoring of liver synthetic function due to ameliorating splenic artery steal syndrome, and, as a result, liver tissue rearterialization. SAE showed a long-term reliable prophylactic effect in high-risk patients with CSPH and HE.

FRI540

Sequential algorithm of spleen stiffness measured by a dedicated 100 Hz examination and Baveno VII criteria for clinically significant portal hypertension in compensated cirrhosis

Ruiling He[1,2], Chuan Liu[1], Li Jie[3], Jianzhong Ma[4], Ying Guo[4], Xiaoqing Guo[4], Shirong Liu[5], Bo Gao[6], Ning Liu[5], Mingkai Chen[7], Hongwei Zhou[8], Tianyu Tang[9], Yu Shi[10], Shenghong Ju[9], Yan Huadong[11], Xiaolong Qi[1]. *[1]The First Hospital of Lanzhou University, CHESS Center, Institute of Portal Hypertension, China; [2]The First Clinical Medical College of Lanzhou University, China; [3]Nanjing Drum Tower Hospital, The Affiliated Hospital of Nanjing University Medical School, Department of Infectious Diseases, China; [4]The third people's Hospital of Taiyuan, Department of Hepatology, China; [5]Qufu People's Hospital, Department of Infectious Diseases, China; [6]Qufu People's Hospital, Medical Laboratory, China; [7]Renmin Hospital of Wuhan University, Department of Gastroenterology, China; [8]Zhujiang Hospital, Southern Medical University, Department of Laboratory Medicine, China; [9]Zhongda Hospital Affiliated to Southeast University, Department of Radiology, China; [10]Shengjing Hospital, China Medical University, Department of Radiology, China; [11]Shulan (Hangzhou) Hospital Affiliated to Zhejiang Shuren University Shulan International Medical College, Department of Infectious Diseases, China*
Email: qixiaolong@vip.163.com

Background and aims: The renewing Baveno VII consensus proposed that liver stiffness measurement (LSM) and spleen stiffness measurement (SSM) can be used to rule in and rule out clinically significant portal hypertension (CSPH) in patients with compensated cirrhosis; however, the thresholds of SSM should be further validated with spleen-dedicated 100 Hz examination. This study aims to assess the performance of sequential algorithm of SSM measured by a dedicated 100 Hz FibroScan® and Baveno VII criteria for CSPH in patients with well-characterized compensated cirrhosis.

Method: This is a prospective multi-center study (NCT05251272). Patients with well-characterized compensated cirrhosis undergoing a 100 Hz specific FibroScan® to evaluate SSM and LSM were consecutively enrolled between August 2021 and March 2022. CSPH was defined as the threshold of hepatic venous pressure gradient measurement above 10 mmHg.

Results: A total of 101 patients (71 male) with a mean age of 52 ± 9 years were recruited from 5 university centers, and 50 (49.5%) patients had CSPH. Applying the Baveno VII criteria for exclusion of CSPH (LSM ≤15 kPa and platelet count ≥150 × 10^9/L), 24/101 (23.8%) patients could rule out CSPH with 8.3% of patients being missed, and the negative predictive value (NPV) and sensitivity were 91.7% and 96.0%. The sequential algorithm for SSM and Baveno VII criteria [SSM <21 kPa OR (LSM ≤15 kPa and platelet count ≥150 × 10^9/L)] showed that 28/101 (27.7%) patients could rule out CSPH with 7.1% of patients being missed, and the NPV and sensitivity were 92.9% and 96.0%, respectively. Furthermore, the Baveno VII criteria for identification of CSPH (LSM ≥25 kPa) had 100% positive predictive value (PPV) and specificity, yet merely 5/101 (5.0%) patients would enable to identify correctly. Comparing with the former, the sequential algorithm of SSM >50 kPa OR LSM ≥25 kPa improved the proportion of ruling in CSPH (22.8% vs 5.0%, p < 0.001) with only 4.3% of patients misclassified, and the PPV and specificity were 95.7% and 98.0%, respectively.

Table: Performance of different methods for ruling out and ruling in clinically significant portal hypertension.

	CSPH Missed	Rule out CSPH	Non-CSPH Missed	Rule in CSPH
LSM ≤15 kPa and PLT ≥150 × 10^9/L	2/23 (8.3%)	24/101 (23.8%)	-	-
SSM <21 kPa OR (LSM ≤15 kPa and PLT ≥150 × 10^9/L)	2/28 (7.1%)	28/201 (70.5%)	-	-
LSM ≥25 kPa	-	-	0	5/101 (5.0%)
SSM >50 kPa OR LSM ≥25 kPa	-	-	1/23 (4.3%)	23/101 (22.8%)***

Data are presented as n (%) or n/N (%), where N is the total number of related cases. CSPH, clinically significant portal hypertension; LSM, liver stiffness measurement; SSM, spleen stiffness measurement; PLT, platelet count. ***p < 0.001, LSM ≥25 kPa compared to SSM >50 kPa OR LSM ≥25 kPa.

Conclusion: The sequential algorithm of SSM measured by a dedicated 100 Hz examination and Baveno VII criteria is useful to assess CSPH accurately, especially in ruling in CSPH compared to Baveno VII criteria only.

Liver tumours: Experimental and pathophysiology

FRI542

NKG2A hampers tumor surveillance of liver-infiltrating natural killer (NK) cells via TLR4 signaling pathway after liver transplantation

Xinxiang Yang[1,1], Kwan Man[1]. *[1]The University of Hong Kong, Surgery, Hong Kong*
Email: kwanman@hku.hk

Background and aims: Liver transplantation (LT) provides the best option for selected hepatocellular carcinoma (HCC) patients. However, tumor recurrence occurs at a rate of 10–20% within 5 years. Graft injury during LT significantly affects the outcome of tumor recurrence by changing the regional immune microenvironment. However, the immunological mechanism of hepatic natural killer (NK) cells in graft injury was not completely understood.

Method: 349 HCC patients who underwent LT in Queen Mary Hospital, Hong Kong were recruited in this study. The correlation among the number of intra-graft NK cells, liver function, recurrence free survival (RFS) rate was analyzed. The cytotoxic function of NK cells was evaluated by co-culture with primary HCC cells. Hepatic ischemia-reperfusion injury plus major hepatectomy (IRI + Hx) was performed in C57bl/6 mice to mimic the pathological changes during human LT. Tumor cells (Hepa 1–6) were injected into the liver via portal vein after IRI + Hx in TLR4 knockout and wild-type mice.

Results: The frequency of intra-graft NKG2A+NK cells was significantly increased in patients with tumor recurrence after LT, along with increased expressions of its ligand HLA-E and inflammation signatures (IL-1β, IL-6, NLRP3, HMGB1, and TLR4). GWR (the ratio of graft volume to estimated standard liver volume) <60% was an independent risk factor for tumor recurrence after LT. The frequency of circulating NKG2A + NK cells was significantly increased in the in the patients with GWR <60% compared with GWR ≥60% at 3, 6, 9, and 12 months post-LT. The cytotoxic function of NKG2A + NK cells was significantly decreased compared to NKG2A-NK cells when co-cultured with primary HCC cells, characterized by decreased expressions of Granzyme B and CD107a. In IRI+Hx mouse model,