the same groups was 20.7, 17.9, and 14.0 months respectively ($P < 0.001$). Competing risk analyses showed that this excess mortality was associated with an increased risk of death due to liver failure for both treatment modalities. These estimates compare with international benchmarks of median survival of 36 months in patients treated with ablation and 20 months for those treated with TACE.

Conclusions Overall survival in patients with HCC and cirrhosis in England are in line with international benchmarks. Survival is negatively associated with increasing cirrhosis severity for the most common treatment modalities. Considering the impact of previous decompensation on outcomes is critical in treatment selection for HCC.

REFERENCE

1. EASL-EORTC clinical practice guidelines: management of hepatocellular carcinoma. *J Hepatol.* 2012;**56**(4).

OTU-11 NON-SEVERE ALCOHOLIC HEPATITIS IS NOT A BENIGN CONDITION: SYSTEMATIC REVIEW AND META-ANALYSIS OF MORTALITY DATA

^{1,2}Kris Bennett, ²Doyo Enki, ³Mark Thursz, ^{1,2}Matthew Cramp, ^{1,2}Ashwin Dhanda*. ¹University Hospitals Plymouth NHS Trust, Plymouth, UK; ²University of Plymouth, Plymouth, UK; ³Imperial College London, London, UK

10.1136/gutjnl-2019-BSGAbstracts.202

Introduction Alcoholic hepatitis (AH) is a serious complication of alcohol misuse characterised by recent-onset jaundice and coagulopathy in long-term heavy alcohol consumers. To date, clinical and academic interest has almost exclusively focused on severe AH (SAH), which has a very high short- and long-term mortality. AH that does not meet SAH criteria, termed 'non-severe alcoholic hepatitis' (N-SAH) is at least as common as SAH but is less well characterised and mortality in this group of patients is unknown. We conducted a systematic review and meta-analysis to determine the 28-day, 90-day and 1-year mortality of patients with N-SAH.

Methods The study protocol was registered prospectively on the PROSPERO database. Embase, Medline and Cochrane Central databases were searched until July 2018. Abstracts from international liver conferences and reference lists of identified manuscripts were also reviewed. All study designs reporting survival data in patients with N-SAH in any language were eligible for inclusion. Studies reporting on identical cohorts and those with missing data were excluded. N-SAH was defined as discriminant function < 32 , Age Bilirubin INR Creatinine (ABIC) < 6.71 , Model for End-stage Liver Disease (MELD) < 15 or bilirubin $< 85 \mu\text{mol/L}$. Mortality data were extracted and meta-analysis performed using a random effects model. Sensitivity or subgroup analyses were performed to determine whether the definition of N-SAH, method of diagnosis (histological or clinical) or study design influenced the mortality.

Results Twenty-five studies ($n=1372$ patients) were eligible (12 prospective studies including 5 interventional trials) of which, 17 reported 28-day mortality ($n=993$), 15, 90-day mortality ($n=755$) and 5, 1-year mortality ($n=234$). Mortality was 6% (95% CI 91–97%; $I^2=67\%$; $p<0.001$) at 28 days, 7% (89–96%, $I^2=64\%$; $p<0.001$) at 90 days and 13% (76–96%; $I^2=72\%$; $p=0.006$) at 1 year. Sensitivity analysis demonstrated that the criteria used to define N-SAH did not affect mortality. Subgroup analyses did not find differences in

mortality proportion depending on method of diagnosis (histological vs clinical) or study design (retrospective vs prospective).

Conclusions This meta-analysis demonstrates that 'N-SAH' is poorly named and not a benign condition as previously assumed. The 28-day mortality of 6% is comparable to that of acute myocardial infarction. Whilst this meta-analysis is limited to some extent by the heterogeneity of studies in terms of design, sample size and population, it shows that there is a paucity of high quality studies reporting outcomes in this group. N-SAH warrants further study to better define outcomes and develop new strategies to improve survival.

OTU-12 SPLEEN STIFFNESS HAS A GOOD PERFORMANCE IN PREDICTING CLINICALLY SIGNIFICANT PORTAL HYPERTENSION IN PSC

¹Davide Roccarina*, ^{1,2}Francesca Saffioti, ¹Matteo Rosselli, ^{1,3}Anna Mantovani, ^{1,4}Roberta Stupia, ¹Aileen Marshall, ¹Massimo Pinzani, ¹Douglas Thorburn. ¹Sheila Sherlock Liver Centre, Royal Free London NHS Foundation Trust and UCL Institute for Liver and Digestive Health, University College of London, London, UK; ²Department of Clinical and Experimental Medicine, Division of Clinical and Molecular Hepatology, University of Messina, Messina, Italy; ³Division of General Medicine and Hypertension, Department of Medicine, Azienda Ospedaliera Universitaria Integrata, University of Verona, Verona, Italy; ⁴Division of Clinical and Molecular Hepatology, University of Messina, Messina, Italy

10.1136/gutjnl-2019-BSGAbstracts.203

Introduction Large efforts have been made to identify non-invasive surrogates as markers of clinically significant portal hypertension (CSPH). The correlation of spleen stiffness measurement (SSM) with portal hypertension (PH) as measured by hepatic venous pressure gradient has been reported, however data regarding its reliability are still controversial. We assessed the performance of liver stiffness measurement (LSM) and SSM as performed by ElastPQ point-shear wave elastography (pSWE) in detecting the presence of CSPH in primary sclerosing cholangitis (PSC).

Methods Predictors of CSPH were investigated in 46 PSC patients who underwent an upper-GI endoscopy within 12 months of the elastographic assessment. Demographics, biochemistry and ultrasonographic data were prospectively collected. Fibroscan transient elastography (F-TE), liver and spleen pSWE were obtained on the visit date. Receiver operating characteristics (ROC) curves were constructed to establish the performance of elastographic techniques in predicting CSPH.

Results Mean age 47 ± 16 y, 74% male, CSPH was detected in 18 cases. On univariate analysis CSPH was associated with: cirrhosis, Child-Pugh, MELD and Mayo risk score, spleen area and length, platelet, albumin, bilirubin, AST, GGT, INR, F-TE, liver and spleen ElastPQ, LSPS (LSM*spleen diameter/platelet). SSM remained the only independent predictor of CSPH on multivariate models (OR 1.125; 95% CI 1.015–1.246; $p<0.025$). F-TE and ElastPQ LSM showed a good performance [AUROC, 95%CI, Sensitivity (Se) and Specificity (Sp)]: 0.82, 0.68–0.96, 92% and 64%, and 0.83, 0.70–0.97, 92% and 68%, respectively. Best cut-off was 18.5 and 21.1 kPa, respectively. However, Sp was $< 70\%$ for both tests. ElastPQ SSM showed a better performance in predicting OVs (0.87, 0.76–0.99, 92% and 77%), cut-off 40.2 kPa. However, the best diagnostic performance was obtained by combining ElastPQ SSM, spleen diameter and platelet count (0.92, 0.83–

1.00, 92% and 81%) which was better than LSPS (0.90, 0.81–0.99, 94% and 67%). ElastPQ SSM was superior to BavenoVI and Expanded BavenoVI criteria (Se 92%, for both; Sp 54% and 59%, respectively).

Conclusions ElastPQ SSM, particularly in combination with spleen diameter and platelet count, can be used as a reliable tool for the diagnosis of CSPH in patients with PSC.

OTU-13

10.1136/gutjnl-2019-BSGAbstracts.204

Licence to publish not granted by author

OTU-14

POSITIVE RESULTS FROM REGENERATE: A PHASE 3 INTERNATIONAL, RANDOMIZED, PLACEBO-CONTROLLED STUDY EVALUATING OBETICHOIC ACID TREATMENT FOR NASH

¹Zobair Younossi, ²Vlad Ratziu, ³Rohit Loomba, ⁴Mary Rinella, ⁵Quentin M Anstee*, ⁶Luna Zaru, ⁶Leigh MacConell, ⁶Reshma Shringarpure, ⁷Stephen Harrison, ⁸Arun Sanyal. ¹Betty and Guy Beatty Center for Integrated Research, Inova Health System, Falls Church, USA; ²Sorbonne Université Hôpital Pitié – Salpêtrière, Paris, France; ³University of California San Diego, San Diego, USA; ⁴Feinberg School of Medicine, Northwestern University, Chicago, USA; ⁵Newcastle upon Tyne Hospitals NHS Foundation Trust, Newcastle, UK; ⁶Intercept Pharmaceuticals, San Diego, USA; ⁷Pinnacle Clinical Research Center, San Antonio, USA; ⁸Virginia Commonwealth University, Richmond, USA

10.1136/gutjnl-2019-BSGAbstracts.205

Introduction Obeticholic acid (OCA), an FXR agonist, improved both fibrosis and histologic features of nonalcoholic steatohepatitis (NASH) in the Ph2 FLINT study. This Month 18 pre-specified interim analysis of the ongoing Ph3 REGENERATE study evaluated the effect of OCA on liver histology in patients (pts) with biopsy-confirmed NASH.

Methods Pts with NASH and fibrosis stages F2–3 (ITT), and an exploratory group of F1 pts with metabolic syndrome, were randomized to placebo (PBO), OCA 10 mg, or OCA 25 mg QD. Primary endpoints were fibrosis improvement (≥ 1 stage) with no worsening of NASH, or NASH resolution with no worsening of liver fibrosis. The safety population included all randomized and dosed pts (F1–3, N=1968). Clinical outcomes will be evaluated at the end-of-study.

Results The ITT population included 931 pts (PBO [n=311], OCA 10 mg [n=312] or OCA 25 mg [n=308]), comprised of 44% F2 and 56% F3. Baseline characteristics were well-balanced across groups. Results in table 1. The primary fibrosis endpoint was met by 11.9% PBO, 17.6% OCA 10 mg ($p=0.0446$), and 23.1% OCA 25 mg ($p=0.0002$) pts (ITT). The primary NASH endpoint was not statistically significant (ITT); however, in a pre-specified analysis that included F1–F3 pts (N=1218), more OCA 25 mg pts achieved NASH. Pruritus was the most common AE (19% PBO, 28% OCA 10 mg, 51% OCA 25 mg) and was predominantly mild to moderate in severity (severe: <1% PBO, <1% OCA 10 mg, 5% OCA 25 mg). More OCA 25 mg pts discontinued due to pruritus (<1% PBO, <1% OCA 10 mg, 9% OCA 25 mg). SAEs occurred in 11% PBO, 11% OCA 10 mg and 14% OCA 25 mg pts. Increases in LDLc with OCA were observed by Wk 4, but approached baseline by M18 (OCA 25 mg: LS mean change Wk4 +22.6 mg/dL, M18 +4.0 mg/dL). Three deaths

occurred; none were considered treatment-related (PBO n=2; OCA 25 mg n=1).

Abstract OTU-14 Table 1

	Placebo	OCA 10 mg	OCA 25 mg
Primary: ITT Population (F2 + F3)	n=311	n=312	n=308
Fibrosis improvement + no worsening of NASH	11.9%	17.6% $p=0.0446$	23.1% $p=0.0002$
NASH resolution + no worsening of fibrosis	8.0%	11.2% $p=0.1814$	11.7% $p=0.1268$
Additional: Full Efficacy Analysis (ITT + F1)	n=407	n=407	n=404
Fibrosis improvement + no worsening of NASH	10.6%	15.7% $p=0.0286$	21.0% $p<0.0001$
NASH resolution + no worsening of fibrosis	7.9%	11.3% $p=0.0903$	14.9% $p=0.0013$

Overall study discontinuations (ITT): 16% PBO, 17% OCA 10 mg, 15% OCA 25 mg.

Conclusion Treatment with OCA 25 mg improved liver fibrosis, key histologic features of steatohepatitis and liver biochemistry, demonstrating consistent efficacy with an overall AE profile similar to previous studies.

OTU-25

TREATING IN CHAOS: OUTCOMES OF HEPATITIS C TREATMENT IN NEWCASTLE'S HOMELESS DRUG USERS

¹Emma Buchanan*, ²Heather Ord. ¹Newcastle Upon Tyne Hospitals, Newcastle Upon Tyne, UK; ²Cgl, Newcastle Upon Tyne, UK

10.1136/gutjnl-2019-BSGAbstracts.206

Introduction There is a high prevalence of Hepatitis C (HCV) among patients leading chaotic lives; this includes patients who are homeless, currently injecting drugs and dependently drinking alcohol. A HCV outreach treatment clinic was set up in the needle exchange in central Newcastle to bring treatment to those who have many barriers to attending treatment service in secondary care. This clinic was offered in conjunction with harm minimisation services.

Methods We retrospectively looked back at the referrals to the clinic and HCV treatment cure rates from September 2017 to November 2018. Data from the needle exchange's harm minimisation team was also collected to look at the number of HCV tests carried out during the first 3 months of the service running and the last 3 months of the study period to assess the impact treating chaotic patients is having on the prevalence of HCV in this community.

Results Between September 2017 and November 2018 54 patients were referred to the HCV outreach clinic following a HCV test. Of these patient 48 attended for treatment. 31 (65%) of the attenders were of no fixed abode (NFA). All the patients who attended for treatment stated intravenous drug use (IVDU) as their main risk factor for HCV and 73% of patients were still using IVDs at assessment for HCV treatment. A further 12 patients were also dependently drinking. 37 of the 48 patients assessed for treatment had commenced HCV treatment at the point of publication. 11 patients did not commence treatment due to 2 dying (both drug related deaths), 3 spontaneous clearance of HCV, 2 moved out of area, 2 being in custody and 2 did not reattend. Of the patients who commenced treatment 18 (49%) have achieved a