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.

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

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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

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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–