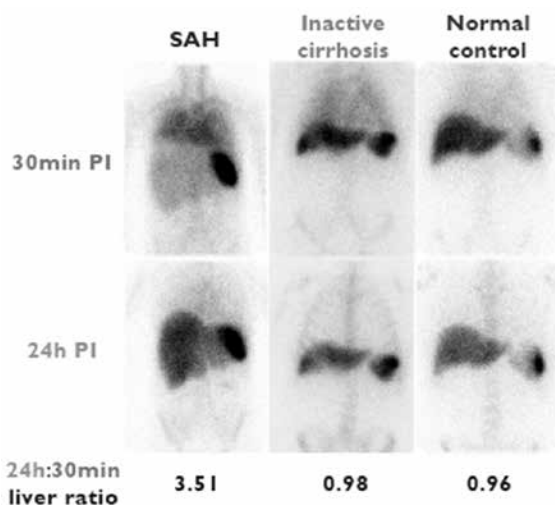


priming. There was no correlation between imaging/histology and CS response, likely due to the small heterogeneous study group.



#### Abstract PTU-117 Figure 1

**Conclusion**  $^{111}\text{In}$ -labelled leucocyte scintigraphy is a novel technique for assessment of hepatic neutrophil migration in SAH. It has potential to be a non-invasive diagnostic tool and may help to prospectively identify those likely to respond to CS.

**Disclosure of Interest** None Declared

#### REFERENCE

1. Mathurin P, Duchatelle V, Ramond MJ, *et al*. Survival and prognostic factors in patients with severe alcoholic hepatitis treated with prednisolone. *Gastroenterology* 1996; **110**:1847–53.

#### PTU-118 LONG-TERM OUTCOME IN SEVERE ALCOHOLIC HEPATITIS

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**Introduction** Although short-term outcome in severe alcoholic hepatitis (SAH) is well described, its long-term course is largely unknown. Our aim was to assess long-term outcome in SAH.

**Methods** Cohort study of patients with SAH (Discriminant Function  $\geq 32$ ) admitted to our institute, identified retrospectively 2007–2009 then prospectively until August 2011. Clinical and laboratory parameters were recorded at accession and subsequent follow-up. Kaplan-Meier (KM) and Cox proportional hazards analyses were performed. Data are presented as mean  $\pm$ SD or median (IQR).

**Results** 109 consecutive patients with SAH were included (63.3% men, age 49.6  $\pm$  9.4yrs) with median follow-up of 40.7 mths (95% CI 37.2–44.3). At accession median DF was 58 (34), MELD 23 (6) and Glasgow Alcoholic Hepatitis Score 8 (2). 55.0% drank spirits, 86.2% had established cirrhosis and 65.1% received corticosteroid and/or pentoxifylline therapy. Prevalence of hepatic encephalopathy (HE), infection and hepatorenal syndrome (HRS) were 38.5%, 47.7% and 18.3% respectively. Median survival was 22.4 mths (95% CI 11.1–33.7), overall mortality being 57.8% (n = 63). All except two deaths were liver related and 33.3% occurred during the index admission.

In univariate analysis, AST, urea, creatinine, white cell count, and the presence of HE, HRS and infection were statistically significantly associated with mortality (p < 0.10 for all), though only HRS was an independent predictor in multivariate analysis (HR 3.842,

95% CI 2.018–7.312, p < 0.0001). However, all baseline factors were associated with short-term mortality and none predicted death beyond 3 mths.

Of those surviving index hospitalisation (n = 87, 86 with available data), 37 (43.0%) were abstinent at last follow-up. Recidivism occurred in the remaining 49 (57.0%), of whom 33 continued to drink and 16 relapsed after initial abstinence. Abstainers were significantly less likely to require hospital readmission than those with any recidivism (median readmissions/patient 1 vs. 5, p = 0.001). In a further univariate analysis, inpatient paracetamol use and abstinence status were associated with mortality after the index hospitalisation (p = 0.032 and 0.010 respectively). Only abstinence remained an independent predictor in multivariate analysis (HR 0.402 (95% CI 0.183–0.883), p = 0.023). KM analysis showed 3 year survival to be significantly higher in abstainers (75.3%) compared to relapsed and continued drinkers (40.2% and 21.0% respectively, p = 0.005).

**Conclusion** Recidivism is common after SAH (~65%) and is the main determinant of the high long-term mortality (~60%), which appears unrelated to the severity of the index episode. Novel strategies to improve abstinence following SAH are urgently needed, especially in view of the increasing numbers of deaths due to alcohol-related liver disease in the UK.

**Disclosure of Interest** None Declared

#### PTU-119 HISTOPATHOLOGY AND LONG TERM CLINICAL PROGNOSIS IN HCV. DOES THE BIOPSY STILL HAVE ANYTHING TO ADD?

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**Introduction** Hepatitis C (HCV) was first characterised in 1989 and most UK sufferers are infected in early adulthood. While a cirrhosis rate of 20% over 20 years is often quoted, life-long outcomes and factors predicting these are not fully defined. With increasing availability of noninvasive markers of fibrosis, liver biopsy is now rarely performed at diagnosis.

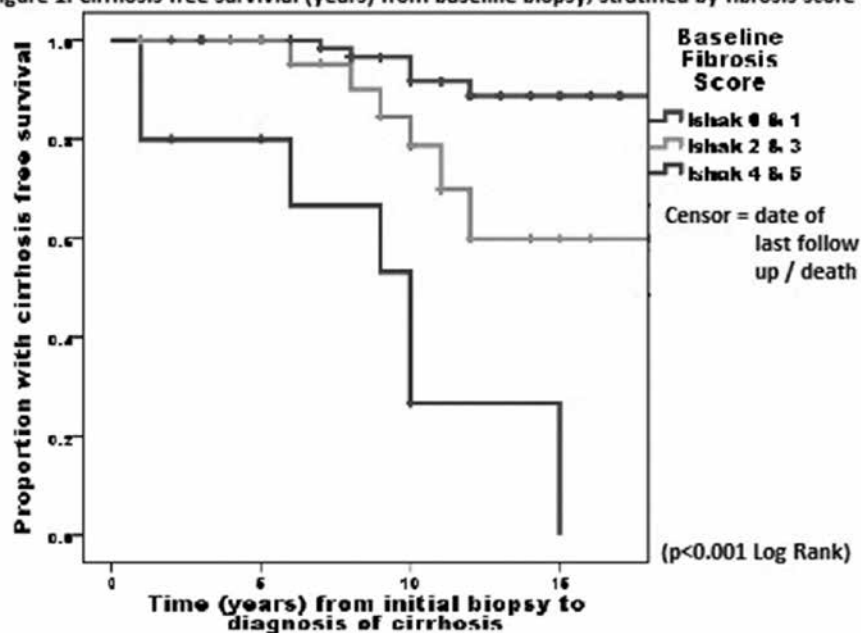
This study aims to explore the value of baseline liver biopsy in determining long-term clinical prognosis.

**Methods** Patients were identified from a historical HCV study cohort (n = 202) at one centre who had a diagnostic liver biopsy at baseline (between 1984 and 2004) and available follow up data. Clinical and histologic data were recorded. Kaplan-Meier plots of time to cirrhosis and multivariate Cox regression were performed.

**Results** 146 patients had adequate follow up data. The mean duration of follow up from presumed date of infection was 22 (SD 8.51) years with a mean follow up from biopsy of 10 years (SD 5.3). The majority of patients were male 90 (62%) and had been infected through IV drug use 81 (55%). 58 patients (40%) had Genotype 1 infection. From baseline biopsies, 44 (30%) had moderate and 12 (8%) severe steatosis. 93 (64%) had Ishak fibrosis scores of 0 or 1 at baseline, 34 (23%) 2–3 and 10 (7%) 4–5. 9 patients (6%) had cirrhosis at baseline.

A total of 31 (21%) developed clinical cirrhosis during follow up. From these, 11 decompensated and 5 developed hepatocellular carcinoma. Factors associated with shorter time to cirrhosis were later age at diagnosis (HR: 1.08, 95%CI 1.03–1.13) and increasing Ishak fibrosis score (HR: 2.13, 95%CI 1.59–2.85). The 10 year cirrhosis free survival from baseline biopsy was 93% for Ishak fibrosis scores of 0 or 1, compared with 78% and 27% for scores 2&3 and 4&5 respectively (see Fig 1.). 95 (65%) patients were treated, of whom 64 (67%) achieved a sustained virological response (SVR) with standard treatment. History of alcohol excess, genotype 1 and severe steatosis were significant negative predictors of SVR.

Figure 1: Cirrhosis free survival (years) from baseline biopsy, stratified by fibrosis score



## Abstract PTU-119 Figure 1

**Conclusion** The rate of cirrhosis was similar to that expected over a 20 year period. Higher baseline fibrosis scores were associated with earlier development of cirrhosis and steatosis was a negative predictor of SVR. Overall, important prognostic information is available from initial diagnostic biopsies and may be useful in determining timing of treatment.

**Disclosure of Interest** None Declared

**PTU-120** QUANTITATIVE MAGNETIC RESONANCE IMAGING (MRI) IN THE EVALUATION OF THE DEGREE OF STEATOSIS, IRON ACCUMULATION AND FIBROSIS IN CHRONIC LIVER DISEASES (MRKER STUDY)

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**Introduction** Half of all the liver biopsies performed are to assess the severity of pathology including grading of fat, iron accumulation as well as fibrosis. Liver biopsies are invasive tests associated with sampling errors; the coefficient of variation for fibrosis measurement is 45% even with 25mm long specimens. We aimed to develop and validate non-contrast, non-breath-holding, quantitative MRI methodology to estimate the amount of fibrosis, fat and iron accumulation within the whole liver.

**Methods** MRI relaxation time data ( $T_1$ ,  $T_2$  and  $T_2^*$ ) were acquired (over 15–20 minutes) using a novel Echo Planar Imaging technique with a respiratory-triggered (r.t.) acquisition method. <sup>1</sup>H MR spectra were acquired (r.t.) using a multiple echo PRESS acquisition which allowed for individual  $T_2$  correction to the spectrum for accurate quantification of the fat fraction in a  $30 \times 30 \times 30 \text{ mm}^3$  voxel.

**Results** 115 patients (67 Training; 48 Validation cohort) with suspected chronic liver disease aged 19 to 72 years [alcoholic (13%), non-alcoholic (56%) fatty liver disease, chronic viral hepatitis (21%)

and haemochromatosis (3%)] who had a liver biopsy  $\geq 25$  mm were included in the study. The diagnostic accuracy of the  $T_1$  parameter in the detection of different histological stages of fibrosis, using receiver operator curves and areas under the curve (AUC), in the training and validation cohort are summarised in Table 1. There were also significant correlations between MR measures of fat fraction and staging of steatosis with a Spearman's correlation coefficient of 0.760 ( $p < 0.001$ ) and  $T_2^*$  with hepatic iron staging with Spearman's correlation coefficient of  $-0.588$  ( $p < 0.001$ ). The  $T_1$  relaxation time of the liver correlated with the percentage of fibrosis measured as a continuous variable on morphometry within the entire study population (Pearson correlation coefficient of 0.712,  $p < 0.001$ ).

## Abstract PTU-120 Table 1

Fibrosis stage (0–4) detected	AUC Training	AUC Validation
Cirrhosis (stage 4 vs. 0–3)	0.91	0.83
Advanced fibrosis (stage 3/4 vs. 0/1/2)	0.81	0.78
Mild fibrosis (stage 2/3/4 vs. 0/1)	0.67	0.70

**Conclusion** Across a range of chronic liver diseases, MR measures of fat fraction, hepatic iron content and fibrosis of the whole liver correlate well with related histological measures.

**Disclosure of Interest** None Declared

## Neurogastroenterology/Motility

**PTU-121** NORMAL VALUES AND REPRODUCIBILITY OF THE REAL TIME BEAT-TO-BEAT INDEX OF CARDIAC VAGAL TONE IN HEALTHY HUMANS

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**Introduction** The vagus nerve is the primary neuroanatomical substrate within the brain gut axis (1). In humans, surrogate measures of vagal tone are most commonly evaluated using heart rate