

stated predominant beverage. Body weight data were available in 216 controls and (pre-fluid overload) in 198 patients.

Results TLA was (median (IQR)) 118 (80–175) $\times 10^3$ Units in patients and 131 (93–183) $\times 10^3$ units in controls ($p=0.041$) by Mann–Whitney: 9 (6–12)-fold and 9 (7–12)-fold higher respectively than the minimum specified by the above inclusion criteria. TLA, corrected for body wt was similar in patients and controls. Females with ALD consumed less total alcohol (absolute 85 (52–11) vs 136 (94–193) $\times 10^3$ units ($p<0.01$) and corrected for wt ($p=0.04$)), and also, less beer ($p=0.001$) but more wine ($p<0.001$) and a trend towards more spirits ($p=0.166$) over their lifetime than did males with ALD. However, female controls showed similar significant differences from male controls: lower TLA (both absolute 82 (69–119) vs 145 (108–193) $\times 10^3$ Units) and corrected for body weight) and also, less beer, more wine and more spirits (all comparisons $p\leq 0.001$).

Conclusion The higher total alcohol consumption in controls than in patients is more consistent with a threshold effect than a dose effect of alcohol for development of ALD. Male-female differences in consumption are independent of the presence of liver disease and therefore, are not evidence per se of increased female susceptibility to ALD, although the data do not exclude this.

Competing interests None declared.

PTU-028 TRANSIENT ELASTOGRAPHY IN THE RISK STRATIFICATION OF PATIENTS WITH ABNORMAL LIVER FUNCTION TESTS

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Introduction Transient elastography (TE) (Fibroscan) is a well validated method for assessing liver fibrosis in a trial setting. Gastroenterology Department at Macclesfield DGH is one of the first centres in the UK to adopt Fibroscan examination into the diagnostic pathway of patients with abnormal LFT. This is the first publication of our data of unselected patients undergoing liver stiffness measurement (LSM) demonstrating practicalities and limitations of Fibroscan.

Methods Data were collected from 366 patients with abnormal LFT attending Gastroenterology Clinic from July 2010 to December 2011. Fibroscan examination was performed by two gastroenterology consultants and one specialist nurse. The cohorts of Fatty liver disease (FLD) and Alcoholic liver disease (ALD) were further analysed comparing fibrosis score from TE with clinical findings, laboratory blood tests, ultrasound imaging and liver biopsy.

Results Patients diagnosis were as follows: FLD 35.8% ($n=131$), ALD 14.8% ($n=54$), Chronic hepatitis C 15.6% ($n=57$), PSC/PBC 7.7% ($n=28$), chronic hepatitis B 4.6% ($n=17$), haemochromatosis 2.2% ($n=8$), AIH 1.4% ($n=5$), chronic usage of Methotrexate 1.9% ($n=7$). Additionally 42 healthy volunteers participated. The mean time duration for each successful scan was 173 (30–1363) seconds with 18% of patients requiring the XL probe due to central obesity. The fibrosis scores recorded for all patients scanned were F0-1 41% ($n=150$), F2 6.3% ($n=23$), F3 7.1% ($n=26$) and F4 26% ($n=95$). 15% of scans were considered to be failed as LSM/IQR was >33 , most prevalent in the FLD group 16.8% ($n=22$) and wasn't operator or probe dependent. Significant proportion of patients with liver fibrosis (F3-4) had no clinical signs of chronic liver disease. 80% of patients F3-F4 fibrosis demonstrated portal hypertension on imaging and 32% of patients in that group had low platelets

(3. The sensitivity and specificity of low platelets for significant fibrosis was 32% and 91% and for portal hypertension on imaging was 64% and 89% respectively. There was significant correlation between liver fibrosis scoring on TE and Metvir fibrosis scoring on biopsy in patients without high degree of inflammation. However, coexisting acute inflammation in some cases contributed to false positive results on Fibroscan.

Conclusion The Fibroscan technology has allowed rapid stratification of patients with chronic liver diseases. Significant proportion of patients (40%) were appropriately reassured without need of undergoing liver biopsy. 20% of patients without stigmata of chronic disease and without conventional laboratory markers of liver fibrosis were diagnosed with liver cirrhosis. Early experience has shown difficulties of performing TE in patients with central obesity and FLD. Fibroscan finds its valuable role in the care pathway of patients with abnormal LFT.

Competing interests None declared.

PTU-029 A NOVEL MRI PROTOCOL TO EXAMINE HAEMODYNAMIC COMPARTMENTS IN COMPENSATED LIVER CIRRHOSIS

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Introduction The hyperdynamic circulation in cirrhosis results from changes in the splanchnic, systemic, cardiac and renal compartments; it underpins the clinical consequences of portal hypertension. We present a novel MRI protocol that provides non-invasive measurement of these different haemodynamic compartments in a single assessment. Phase contrast (PC) MRI is a validated technique for the measurement of velocity (in cm/s), area, and hence flux (=velocity \times area (in ml/s)) in a given vessel.¹ Here, we use PC-MRI, together with MR measures of cardiac function, to assess haemodynamics in early, compensated cirrhosis and matched healthy volunteers.

Methods Patients were studied from a prospective, longitudinal study of compensated cirrhosis (CC) (Child Pugh A) with age and sex matched healthy volunteers (HV). 20 CC patients (13M/7F, age 57 ± 1 y (mean \pm SEM), aetiology 2 ALD, 1 HBV, 7 HCV, 3 NAFLD, 1 PBC, 1 Haemochromatosis, MELD 0–10.6) and 20 HV: 13M/7F, age 57 ± 2 y were included. PC-MRI data were collected for the superior mesenteric (SMA), splenic (SA), hepatic (HA), and renal (RA) artery and portal vein (PV). 15 phases were acquired across the cardiac cycle to calculate the mean area, velocity and flux for each vessel. Cardiac MR consisted of short axis cine images to measure left ventricular ejection fraction (EF) and PC-MRI of the aorta. All data were collected within 20 min, with images acquired during short breath-holding (12–15 s).

Results In CC, the portal vein was dilated and had a reduction in velocity compared to the HV group (Abstract PTU-029 figure 1A). The SMA (Abstract PTU-029 figure 1B) and SA, showed vasodilation and increased flow velocity, and a resulting increase in flux in patients with CC compared to the HV. There was a trend for a reduction in RA velocity (Abstract PTU-029 figure 1C) and an increase in HA velocity in CC. The ejection fraction was significantly higher in CC compared with the HV (Abstract PTU-029 figure 1D). There was no change in aortic flux, velocity or area with CC.