6 patients did not attend for nurse review. 3 (50%) did not require referral if the iLFT protocol had been followed.

The median number of times a patient had abnormal LFTs before referral was 5 (min 1; max 33). The median duration of abnormal LFTs before referral was 20 months (min 0; max 87), as shown in figure 1.

Conclusions iLFTs rapidly and safely identify patients with abnormal LFTs requiring referral to secondary care. If iLFTs were used by referrers, 24.7% of patients would not require referral to secondary care. This represents a substantial cost saving in terms of clinic appointments and unnecessary investigations. In addition, patients would be analysed at the time of the first abnormal LFTs, providing referrers information on how to monitor for disease progression and when to refer. This would save significant time in Primary Care, avoiding repeated venepuncture and ensuring patients are referred before they develop end stage liver disease.

PTU-023 INVESTIGATING LIVER FUNCTION IN THE DIABETIC POPULATION OF A DISTRICT GENERAL HOSPITAL

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10.1136/gutjnl-2019-BSGAbstracts.232

Background The rising prevalence of type 2 diabetes (T2DM) and its complications including non-alcoholic fatty liver disease (NAFLD) is now a leading cause of cirrhosis and hepatocellular cancer. Prompt identification of fibrosis in patients with NAFLD is essential for directing specialist care and improving outcomes. Although T2DM is a major risk factor for NAFLD, there are no guidelines on screening in this population.

We aimed to identify the prevalence of liver abnormalities in patients attending secondary care diabetes clinic. In addition we used serum markers to determine degree of fibrosis.

Methods All patients attending a diabetes clinic at a single centre over a 24 month period were analysed. Patients with an elevated ALT or ALP were identified. Patient demographics, clinical, imaging and laboratory characteristics were recorded. We analysed the ability to calculate Fibrosis-4 (FIB4) and NAFLD fibrosis scores (NFS) to risk stratify patients.

Results Of the 1380 patients assessed, 265 (19%) had elevated ALT and/or ALP, after excluding patients with alternative diagnoses causing liver function derangement. The median age was 64 years, 43.8% were male, mean BMI was 31.8 ± 6.8 kg/m².

25.7% of patients underwent an ultrasound scan. 12% of these were suggestive of cirrhosis. Platelet count and AST were available in 85% and 25% respectively. The FIB4 or NFS was calculatable in only 65 patients (25%). FIB4 and NFS scores identified 6% and 14% as indeterminate risk and 2% and 5% as high risk for advanced fibrosis respectively, see figure 1. Of the indeterminate or high risk patients on either score (n=50), all were older than 40, 46% male and 70% had a BMI > 30 kg/m².

Conclusions We highlight minimal rates of recognition of potentially advanced liver fibrosis in a diabetic high risk population. Further education regarding risk stratification using serum markers will identify those patients who need hepatology assessment and monitoring.

PTU-024 DO IRON STUDIES ON ADMISSION PREDICT OUTCOME IN PATIENTS WITH ACUTE LIVER FAILURE?

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10.1136/gutjnl-2019-BSGAbstracts.233

Introduction Acute liver failure (ALF) has a high short term mortality. Current prognostic models lack sensitivity. This study aimed to investigate the prognostic potential of iron metabolism in ALF.

Methods 528 patients admitted to a liver transplant unit with ALF were selected. Aetiology, outcome (spontaneously survived vs. death/transplant) and hepatic encephalopathy (HE) were recorded. Patients without the relevant iron indices recorded were excluded.

Results Paracetamol overdose (POD) patients (n=236) had a significantly higher transferrin if they survived (n=168) compared to those that died or were transplanted (n=68) (1.585 (1.363–1.918) vs. 1.285 (1.000–1.730) (p<0.001))

In non-POD patients (n=88) the median transferrin was also significantly higher in those patients that survived (n=48) compared to those that died or were transplanted (n=40) (1.965 (1.435–2.375) vs. 1.455 (1.078–1.803) (p=0.001))

In POD patients (n=246) the median ferritin was significantly higher in those who died or were transplanted (n=70) compared with survivors (n=176) (36,822(13,054–64,358) vs.

Abstract PTU-023 Figure 1 Breakdown of patients by risk stratification

17,641 (5,120–42,895) ($p=0.002$). There was no statistical difference in the non-POD group.

Transferrin was significantly lower in POD patients with HE ($n=143$) compared to those that never developed HE ($n=93$) (1.420 (1.100–1.720) vs. 1.670 (1.400–1.990) ($p<0.001$)). Levels were also significantly lower in the non-POD group (1.500 (1.085–1.945) vs. 1.985 (1.445–2.365) ($p=0.007$)).

Ferritin was significantly higher in POD patients with HE ($n=146$) compared to those who never developed HE ($n=99$). (24,828 (9,267–61,214) vs. 17,149 (3,153–37,994) ($p=0.009$)). There was no difference in the non-POD group.

No significant results were found when comparing circulating iron levels and transferrin saturations.

ROC curve analysis, however, showed that iron studies are poor at predicting survival in ALF. For example the area under the curve for transferrin to predict survival in POD patients was 0.662. To predict encephalopathy free course of disease in the same group of patients was 0.666.

Conclusions Iron indices, specifically ferritin and transferrin, are significantly different in patients with ALF and relate to aetiology and outcome. However they are poor at predicting survival.

PTU-025 OBESITY IS ASSOCIATED WITH SEVERITY AND OUTCOME OF ALCOHOLIC HEPATITIS

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10.1136/gutjnl-2019-BSGAbstracts.234

Introduction Both sarcopenia and adiposity can influence the prognosis of patients with chronic liver disease. However, little is known of their interaction in patients with alcoholic hepatitis (AH). With the changing demographics of gender, age and body shape of AH patients, it is important to understand how obesity and sarcopenia affect AH severity and outcome. We aimed to determine whether quantity of skeletal muscle and adipose tissue influenced the outcome and severity of AH patients.

Methods We studied a prospective single centre cohort of patients with severe AH, defined as recent onset jaundice (bilirubin >80 $\mu\text{mol/L}$) in heavy alcohol drinkers ($>80\text{g}$ ethanol/day in males; $>60\text{g}$ in females), AST:ALT >2 and discriminant function (DF) >32 . Patients received standard medical care including 28 days of corticosteroids in the absence of active infection. Clinical and laboratory parameters were obtained at baseline and patient survival recorded at 6 months. CT scans from patients taken within 3 months of their index presentation were evaluated for adipose tissue and skeletal muscle area. An image at the level of L3/4 vertebrae was analysed using ImageJ software according to published methodology (Gomez-Perez, JPEN 2016) and total abdominal, intra-peritoneal and skeletal muscle areas were measured.

Results Of a total 28 patients, 21 (mean age 49, DF 74, MELD 19; 11 females) had an eligible CT scan and were included in the final analysis. There were 5 deaths within 6 months (2 females). Total abdominal area was higher in non-survivors vs survivors at 6 months (1010 v 715 cm^2 ; $p=0.003$) and correlated with severity of AH measured by MELD ($r=0.50$; $p=0.04$). Waist circumference and adipose tissue area were higher in non-survivors than survivors at 6

months (126 v 105 cm; $p=0.02$ and 576 v 406 cm^2 ; $p=0.05$, respectively). Skeletal muscle area was no different between non-survivors and survivors (141 v 121 cm^2 ; $p=0.37$). Multivariate analysis with total abdominal area, age, bilirubin, albumin, urea and MELD as co-variables showed that total abdominal area is an independent predictor of 6-month outcome in this dataset ($p=0.003$).

Conclusions These data demonstrate that adiposity but not sarcopenia is associated with severity and predicts outcome of AH. Obesity should be recognised as a risk factor in patients with AH. Greater understanding of the interaction between alcohol and adipose tissue in AH may reveal new targets for treatment.

PTU-026 EVIDENCE OF GUT-LIVER CROSSTALK AND GUT HOMEOSTASIS IN PARACETAMOL TOXICITY: IN VITRO STUDY USING IMPEDANCE-BASED ASSAY

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10.1136/gutjnl-2019-BSGAbstracts.235

Introduction Drug induced liver injury accounts for approximately one half of all acute liver failure cases, with Paracetamol (APAP) being one of the most reported hepatotoxins [1]. We used an *in vitro* approach to model APAP transport from gut to liver where metabolic products (NAPQI) can cause hepatocellular damage. Investigating the mechanisms of a gut-liver axis *in vitro* may contribute to a better understanding of APAP toxicity.

Methods The cellular impedance ECIS Z Θ platform was used to study the gut-liver axis and effects of APAP. Caco-2 and HepaRGs were seeded on separate 8W10+E ibidi arrays. The Caco-2 array was collagen coated. Step 1: Previously established concentrations of APAP [2] were applied to confluent Caco-2s (day 10), and impedance monitored for 24 hrs. Step 2: This 'preconditioned' culture medium was then transferred to HepaRGs (day 11) for 24 hrs. Step 3: preconditioned culture medium was then transferred from HepaRGs to fresh Caco-2s (day 12) for 24 hrs.

Results Step 1: using an impedance based cellular assay, we show a tightening of barrier function in Caco-2s treated with 20 mM APAP. Step 2: only the highest concentration (20 mM APAP) shows substantial loss of impedance on hepatocytes. Step 3: we demonstrate that barrier function has become sensitized on Caco-2s with an increase in impedance at 10 mM

Discussion and Conclusions This study demonstrates a potential cross-talk between hepatocytes and enterocytes and reveals a homeostatic effect of gut barrier function in presence of APAP. Exposing hepatocytes to toxic levels of APAP taken from Caco-2's attenuates toxicity at 5 and 10 mM concentrations compared to previous study [2] where a dose-dependent loss of tight junctions was observed (5–20 mM APAP). Further exposure of preconditioned media on fresh Caco-2s (step 3) shows that tightening of barrier function is now achieved with lower APAP concentration (10 mM) to reduce absorption of APAP. It is possible a liver-gut axis regulates APAP absorption through paracrine signals, though more work is needed. This may differ between population phenotypes and potentially be responsible for variability of toxic response in patients with APAP overdose.