Abstract PTU026 Figure 1 Total impedance from steps 1–3 showing effect of 24 hr incubation with APAP treated media

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PTU-027 DRIED BLOOD SPOT BLOOD-BORNE VIRUS SCREENING AND LINKAGE TO CARE IN AN URGENT CARE CENTRE

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Background Currently, there are only guidelines for wider HIV screening. Other blood-borne virus (BBV) screening is targeted to ‘at risk’ populations despite high prevalence in recent studies¹.

The NHS provides approximately 110 million urgent same-day patient contacts annually including those seen in Urgent Care Centres (UCC)². UCCs provide access to less defined ‘at risk’ populations and can be a platform to promote HIV, Hepatitis B (HBV) and Hepatitis C (HCV) screening. As the majority of these patients don’t require phlebotomy, dried blood spot (DBS) testing could be used.

Study objectives include 1) Reporting BBV prevalence in an urban UCC;

2) Assessing linkage-to-care of positive patients;

3) Assessing acceptability of DBS testing instead of phlebotomy.

Method A 9 week prospective study was performed at the UCC at Charing Cross Hospital (March-May 2018). Consenting patients were recruited according to the inclusion criteria and tested with DBS test kits for HIV1/HIV2-Ab, HbsAg and HCV-Ab. Demographics and test results were recorded. Positive patients were referred to respective specialist centres and linkage-to-care rates were measured. Ethical approval was obtained.

Results Total number of patients approached: 677; 396 participated (58.5%). Mean age 39.8 (±15.95). Seventeen abnormal DBS results (4.3%): HIV 11.8%; HBV 5.9%; HCV 82.3%; Males 64.7%; females 35.5%; UK-born 47.1%.

Nine patients (52.9%) had confirmatory PCR (HIV 2; HCV 1; HCV 6). Four false positives were identified (HCV-

Ag only). Active BBV prevalence was 0.8%. There was no statistically significant association between BBV risk, gender, age, ethnicity and place of birth. 76.5% were successfully linked to care.

Conclusion This is the first study reporting BBV screening in the UCC. With over 50% participation; active prevalence of 0.8% and excellent linkage-to-care, there is a role for DBS screening in the UCC; assisting WHO’s goal to eliminate viral hepatitis and HIV by 2030³.

There were false positive results for HCV-Ag in contrast to previous studies. True prevalence was lower than expected possibly due to the sample size, selection bias and the opt-in nature of the study. Further studies will need to focus on the cost-effectiveness of routine testing.

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PTU-028 GENOME-WIDE ASSOCIATION STUDY OF MRI LIVER IRON CONTENT IN UK BIOBANK IDENTIFIES 3 SUSCEPTIBILITY VARIANTS

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Background and aims Excess liver iron content is common, affecting approximately 10% of the population, and observationally associates with hepatic and extrahepatic diseases such as liver fibrosis, hepatocellular carcinoma and metabolic syndrome¹. Genome wide association studies (GWAS) on MRI determined liver iron content, compared to circulating iron traits, permit detection of liver-specific susceptibility loci. Here we aim to find genetic variants influencing liver iron content, using data from UK Biobank.

Methods Data was acquired from UK Biobank (application 9914). Liver phenotypes were calculated from MRI data by trained analysts using LiverMultiScanTM. We used GEMMA to perform a GWAS of MRI scan measures of liver iron in 8,289 individuals of European ancestry. We adjusted for age, sex, BMI, genotyping array, and population structure.

Results We identified three independent genetic variants (rs1800562 & rs1799945 in *HFE*, rs855791 in *TM6RS6*) associated with liver iron content at genome-wide significance ($p < 5 \times 10^{-8}$). These variants have been reported previously to be associated with blood markers of iron levels. Mendelian randomisation analysis provided evidence that higher central adiposity plays a causal role in increased liver iron content. Phenome-wide association analysis demonstrated that liver iron increasing alleles in *HFE* were also associated with high blood pressure, liver cirrhosis, hepatic and extrahepatic malignancies, neuro-psychiatric, endocrine and rheumatological conditions, but inversely associated with LDL cholesterol, heart disease and gallbladder diseases. The liver iron increasing allele in *TM6RS6* was inversely associated with lipidaemias and ischaemic heart disease.

Conclusion Our study provides genetic evidence that mechanisms underlying liver iron content are mostly systemic and not organ specific. The identification of loci which affect circulating iron traits provide genetic validation of the utility of MRI for a fast and non-invasive assessment of liver iron content.

Abstract Table 1 Genome wide susceptibility loci for MRI liver iron content reaching $P < 5 \times 10^{-8}$

SNP	Gene	Chr	Effect allele	EA	BETA	SE	P
rs1800562	[HFE]	6	A	0.076	0.41	0.029	5×10^{-42}
rs1799945	[HFE]	6	G	0.153	0.17	0.0216	8.2×10^{-15}
rs855791	[TMPRSS6]	22	G	0.563	0.11	0.0157	1.3×10^{-11}

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PTU-029 DECOMPENSATED CIRRHOSIS IS THE COMMONEST PRESENTATION FOR NAFLD PATIENTS UNDERGOING LIVER TRANSPLANT ASSESSMENT

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Introduction Non-alcoholic fatty liver disease (NAFLD) accounts for 15–20% of orthotopic liver transplants (OLT) in the United Kingdom. Index presentations with cirrhotic decompensation impact morbidity and mortality and represents

missed opportunities for preventive treatment leaving OLT or palliation as the only options. We aimed to determine the proportion of patients undergoing OLT assessment for NAFLD in whom the first presentation was an episode of cirrhotic decompensation by expanding upon our previous audit.

Methods Patient records were interrogated for all NAFLD patients undergoing assessment for OLT at the Royal Free London NHS Foundation Trust liver transplant unit between January 2003 and December 2017. Demographic, clinical, laboratory and outcome data were extracted. Those with an index presentation of jaundice, ascites, variceal bleeding, encephalopathy or HCC at presentation were classified as ‘decompensated’.

Results Data were available for 81 patients with NAFLD as the primary diagnosis. At first presentation to healthcare with chronic liver disease (CLD) 52 patients had decompensated cirrhosis while 29 had compensated cirrhosis. A decompensation event diagnosed in secondary care represented the first presentation with liver disease for 91.7% of patients compared to 52.6% referred from primary care. Cirrhosis was not suspected at the time of referral to hospital in 24.7% of patients subsequently assessed for OLT. OLT was performed in 43 patients. Thirty-one (72.1%) of these patients were decompensated at first presentation compared to 55.3% who were not transplanted. Four deaths occurred in OLT recipients within 6 months of transplantation, all of whom presented for the first time with decompensated cirrhosis. figure 1 illustrates the difference in survival between those patients who did and did not undergo OLT. Patients who underwent OLT had a significantly longer mean survival time of 9.81 years (95% CI 8.51–11.12) compared to those who did not undergo OLT 4.62 years (95% CI 3.35–5.89, $p < 0.001$).

Conclusions Most patients undergoing assessment for OLT for NAFLD had decompensated cirrhosis at their first diagnosis of CLD. These data underline the association between the late diagnosis of CLD in NAFLD with emergency hospitalisation and mortality and reinforce the necessity for greater awareness and earlier diagnosis of cirrhosis in NAFLD.

Abstract PTU-029 Figure 1 Survival Curve Analysis for patients who underwent OLT