Results Overall the diagnostic activity for HCV has increased over the last two decades. More markedly since 2012 when DAAs were becoming available. The standard diagnostic pathways (primary and secondary care) show large volume testing with a low rate of PCR positivity. In contrast testing pathways aimed at high risk individuals show a higher PCR positive rate. See figure.

Conclusions Utilisation of diagnostic pathways targeting populations most at risk of HCV are more effective at yielding new HCV diagnoses than standard pathways. These tailored diagnostic pathways will also resolve some of the health inequalities around drug use and provide methods of ensuring entry to treatment. We believe using targeted testing will find the majority of our undiagnosed population. This will help us to direct resources and achieve our aim of elimination by 2030.

PTU-034 SPLEEN STIFFNESS BY ELASTPQ POINT SHEAR WAVE ELASTOGRAPHY PREDICTS CLINICALLY SIGNIFICANT PORTAL HYPERTENSION IN PBC

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10.1136/gutjnl-2019-BSGAbstracts.243

Introduction There is an ongoing effort to identify non-invasive surrogates for staging liver disease and detecting clinically significant portal hypertension (CSPH), defined as the presence of gastro-oesophageal varices on endoscopy. The correlation of spleen stiffness measurement (SSM) with portal hypertension measured by hepatic venous pressure gradient has been reported, but data regarding its reliability are controversial.

We assessed the ability of liver stiffness measurement (LSM) and SSM performed by point-shear wave elastography (pSWE) to detect CSPH in primary biliary cholangitis (PBC).

Methods Predictors of CSPH were evaluated in 53 PBC patients who had an upper-GI endoscopy within 12 months of the elastographic assessment. Demographics, biochemical and ultrasonographic data were prospectively collected. Transient elastography (Fibroscan, Echosens) [F-TE], liver and spleen pSWE (ElastPQ, Philips Affiniti70G), and the most common clinical fibrosis scores were obtained. ROC curves were constructed to establish the performance of elastographic techniques and fibrosis scores in predicting CSPH.

Results Mean age 57±12 years, 91% female. OVs were detected in 12 (23%) cases (4 at high-risk of bleeding).

Variables significantly associated with CSPH at univariate analysis were: diagnosis of cirrhosis, Child-Pugh, MELD and Mayo risk score, spleen area and longitudinal diameter, platelets, albumin, ALT, INR, F-TE, ElastPQ LSM and SSM, LSPS (LSM*spleen diameter/platelets). SSM remained the only independent predictor of CSPH in all the multivariate models (OR 1.101; 95%CI 1.14–1.20; $p < 0.022$).

F-TE and ElastPQ LSM showed good AUROCs (95%CI): 0.85 (0.72–0.99) and 0.80 (0.62–0.98), with best cut-off 14.3 and 13.8 kPa, respectively. However, specificity (Sp) was <70% for both tests. ElastPQ SSM showed the best performance in predicting CSPH (AUROC 0.95, 95%CI 0.87–1.00, sensitivity (Se) 88%, Sp 97%), with optimal cut-off 50.1 kPa

(PPV 0.89, NPV 0.97), followed by GUCI, King's score and LSPS [AUROC (95%CI): 0.94 (0.87–1.00), 0.94 (0.85–1.00), 0.93 (0.85–1.00), respectively] (fig. 1).

In this cohort, the diagnostic performance of ElastPQ SSM in detecting CSPH was superior to the recently validated Baveno VI and Expanded Baveno VI criteria, which showed good Sp (77% and 87%, respectively) but low Se (67%, for both).

Abstract PTU-034 Figure 1

Conclusions We provide evidence that transient elastography, in particular ElastPQ SSM, can be used as a reliable tool for the detection of CSPH in PBC.

PTU-035 ECONOMIC IMPACT OF NON-RESPONSE TO URSODEOXYCHOLIC ACID IN PRIMARY BILIARY CHOLANGITIS PATIENTS

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10.1136/gutjnl-2019-BSGAbstracts.244

Introduction NICE recommends the use of Obeticholic acid (OCA), as a second-line treatment for failed or intolerant ursodeoxycholic acid (UDCA) drug therapy in primary biliary cholangitis (PBC). This audit aims to determine the proportion of PBC patients, in a tertiary referral hospital experiencing failed UDCA drug therapy, to gauge the potential economic impact of a switch to OCA.

Methods A total of 120 patients with PBC were identified from an existing patient database. 24 patients were excluded due to inappropriate diagnosis, missing data, non-attendance or failure to tolerate UDCA. Baseline characteristics, UDCA dosage and biochemical response were recorded for all patients. For the purpose of this study, failed UDCA drug therapy was defined as an alkaline phosphatase (ALP) level of greater than 1.67 times the upper limit of normal (ULN) (Toronto Criteria)¹.

Results Of the 96 patients included for analysis, 9 were male and the remaining 87 were female. The mean age and weight

were 64.4 ± 11.9 years and 75.9 ± 15.4 kg respectively. The mean blood results for haemoglobin, platelets, bilirubin, ALP, alanine aminotransferase, and creatinine were 126.8 ± 15.0 g/L, $254.5 \pm 94.3 \times 10^9$ /L, 9.4 ± 4.3 μ mol/L, 144.4 ± 75.6 u/L, 30.3 ± 19.3 u/L, and 75.1 ± 28.3 μ mol/L respectively. The mean UDCA dose for the cohort was 13.9 ± 5.9 mg/kg/day. Biochemical failure was documented in 13 patients who had an ALP of greater than 1.67 times the ULN at the end of 1 year of therapy with UDCA (13.5%). Of these 13 patients, 4 were found to be on suboptimal dosage of UDCA (<10 mg/kg/day), leading to a true non-response rate in 9/96 subjects (9.4%).

Conclusions A substantial proportion of PBC patients were found to be biochemically unresponsive to UDCA. Shifting these patients to OCA would lead to significant drug expenditure for NHS Grampian. An intermediary step of shifting non-responsive patients to the cheaper drug, Bezafibrate, may prove to be cost-effective, but this drug has been revealed to have no benefit in a recent Cochrane review².

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PTU-036 SCREENING OF TYPE II DIABETIC PATIENTS FOR LIVER FIBROSIS WITH FIBROSCAN IN PRIMARY CARE

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10.1136/gutjnl-2019-BSGAbstracts.245

Introduction In 2016, European Association for the Study of the Liver (EASL) recommended the presence of non-alcoholic fatty liver disease (NAFLD) should be looked for in Type II diabetic patients as they are at high risk to develop liver fibrosis. Our prospective cohort study aims to examine the practicality of screening Type II diabetic patients for liver fibrosis and to compare different liver fibrosis scores.

Methods For a 10 weeks period, a hepatology specialist nurse from Aberdeen Royal Infirmary attended the Type II Diabetic Clinic at 3 general practices. Unselected patients at the clinics were given information leaflets before consenting for Fibroscan and blood testing. Different liver fibrosis scores including NAFLD fibrosis, Fibrosis-4 (FIB-4) and AST to Platelet Ratio Index (APRI) scores were calculated on the day of Fibroscan. Clinically significant fibrosis ($\geq F2$) was defined as liver stiffness measurement (LSM) > 7 kPa and advanced liver fibrosis/cirrhosis (F3 and F4) was defined as LSM > 12 kPa. Patients with LSM > 7 kPa were given lifestyle modification advices including diet, exercise and alcohol intake. Full 'liver screen' was performed for these patients and they were seen in the hepatology clinic with follow-up Fibroscan.

Results 49 patients (mean age: 67.6 ± 12.2 , mean BMI: 30.5 ± 6.4 and male: 55%) were recruited in the study after excluding 4 patients due to excess alcohol intake ($n=1$), known liver cirrhosis ($n=1$) and body habitus ($n=2$). 22/49 (45%) patients had LSM > 7 kPa. 12/14 (86%) patients who

had follow-up fibroscan had decreased LSM. 37/40 (93%) patients had a score > 1.455 which indicated indeterminate/advanced fibrosis scores based on previous studies. In our study, all liver fibrosis scores performed poorly at diagnosing clinically significant fibrosis (> 7 kPa): NAFLD score (AUROC=0.57), APRI score (AUROC=0.52) and FIB-4 score (AUROC=0.42). NAFLD score (AUROC=0.81) had the best diagnostic accuracy for advanced fibrosis (> 12 kPa) compared to APRI score (AUROC=0.76) and FIB-4 score (AUROC=0.73).

The best NAFLD Cut-off score for advanced fibrosis was ≥ 0.502 with a sensitivity of 100% and specificity of 54%.

Conclusions In this small cohort study, there was a high prevalence of indeterminate/advanced NAFLD fibrosis scores in unselected type II diabetic patients. This strategy could lead to a significant number of hepatology referrals to hospitals if used as a screening test. The NAFLD score was the best performing panel for advanced fibrosis in this cohort. Lifestyle modification advice given by the hepatology specialist nurse led to significant improvement in patients' repeat liver stiffness measurements.

PTU-037 FONTAN ASSOCIATED LIVER DISEASE – EARLY EXPERIENCE AND INSIGHTS FROM LIVER SCREENING

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10.1136/gutjnl-2019-BSGAbstracts.246

Introduction There is growing recognition of Fontan-associated liver disease (FALD), which encompasses a spectrum ranging from liver congestion to cirrhosis and hepatocellular carcinoma. Diagnostic pathways remain poorly defined, with no consensus on optimal screening and management modalities. Aim of this study was to assess prevalence and severity of FALD by implementation of a structured screening programme.

Methods A cohort of 93 Fontan patients aged ≥ 16 years underwent a screening protocol for liver disease at our tertiary Adult Congenital Heart Disease Centre, including blood tests, liver ultrasound with elastography, and fibroscan. Results were discussed in a MDM and patients categorised according to the risk of having liver fibrosis. MRI, CT scan and/or endoscopy were also organised when appropriate. Patients were followed up yearly if the initial tests were reassuring, or started on 6-monthly HCC surveillance if the initial screening suggested advanced fibrosis.

Results The study cohort comprised 55 (59%) males, mean age 25.4 ± 6.2 years, and median 18 years since Fontan surgery. Most patients were asymptomatic (79%), with good haemodynamics. Liver abnormalities on ultrasonography were evident in 51% (41 of 80 scans), including coarse echotexture (41%), hepatomegaly (12%), hyper-echoic nodules (6%) and signs of portal hypertension (15%). Liver stiffness was elevated on both ultrasound elastography (mean 7.8 ± 3.3 kPa, Philips US ElastPQ) and fibroscan (median 20.0 ± 6.7 kPa). Liver MRIs and triple phase liver CTs, performed in a subset of the cohort, further characterised liver abnormalities, most notably focal nodular hyperplasia (FNH) in 65% (13 of 20 MRIs).