quantitative measurement of the molecular features of fibrosis using high-resolution stain-free imaging of collagen 1 and 3. This post-hoc analysis assessed the impact of OCA treatment on collagen morphometry using biopsy samples from the POISE substudy.

Methods Patients included in this analysis had an inadequate response or intolerance of UDCA, biopsies ≤ 1 year from double-blind baseline and after ~ 3 years of OCA treatment. Unstained slides were used to quantify collagen using SHG and 2-photon microscopy. Stained biopsy samples were masked, randomized and scored by consensus of 2 blinded pathologists utilizing Nakanuma Staging for histologic evaluation. Two cohorts were identified: an All Biopsy Cohort, defined as all adequate biopsy samples (determined by the pathologists) with collagen data, and a Paired Biopsy Cohort, defined as patients with both a baseline and follow-up biopsy and collagen data.

Results The All Biopsy Cohort was composed of 31 patients (46 samples total, mean age 56 years, 90% female, 97% received UDCA), and the Paired Biopsy Cohort was composed of 16 patients (59 years, 94% female, 100% received UDCA). In the All Biopsy Cohort, trends were observed between increasing median Collagen Area Ratio (CAR) (fig A), Collagen Fiber Density (CFD), and Collagen Reticulation Index (CRI) and increasing Nakanuma Fibrosis Stage. In the Paired Biopsy Cohort, the majority of patients (12/16) had a reduction in CAR after 3 years of OCA treatment (fig B). The median (Q1,Q3) change and percent change from baseline was CAR -2.1 (-4.6,-0.3) and -31.1% (-46.1,-11.4), CFD -0.8 (-2.5,0.0) and -35.3% (-57.0,-2.5), and CRI -0.1 (-0.3,0.0) and -7.4% (-20.8,-1.1).

Conclusions In this analysis, 3 years of OCA treatment resulted in a reduction in CAR, CFD and CRI, supporting the overall trend of improvement or no progression in the histologic components of PBC.

Posters

PTU-001 DIAGNOSTIC ACCURACY OF NON-INVASIVE LIVER FIBROSIS MARKERS IN YOUNG ADULTS WITHIN THE ALSPAC POPULATION-BASED COHORT

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Introduction Early identification of non-alcoholic fatty liver disease (NAFLD) and alcohol-related liver disease (ARLD) can potentially halt progression to cirrhosis by removing the insult. Concerns remain regarding diagnostic accuracy of non-invasive fibrosis scores in extremes of age. This study aimed to evaluate diagnostic accuracy of non-invasive fibrosis scores in young adults within the Avon Longitudinal Study of Parents and Children (ALSPAC).

Methods 3864 study participants (SPs) (mean age 24 years; SD 0.8) underwent transient elastography (TE) using the Echosens Fibroscan 502 Touch®. Results with interquartile range/median ratio $>30\%$ were excluded. SPs with criteria for alcohol use disorder (AUD) or harmful daily alcohol intake were analysed separately; TE cut-off values assumed ARLD. All other SPs' TE results were interpreted using NAFLD TE cut-offs. Data was collated on TE result, body mass index, serology including alanine transaminase (ALT), aspartate aminotransferase (AST), platelet count, and fibrosis markers pro-collagen-3 N-terminal peptide (P3NP) and hyaluronic acid (HA). To differentiate between fibrosis (TE scores equivalent to $\geq$ METAVIR F2) and normal results, receiver operator characteristics (ROC) were derived for the Southampton Traffic Light Test (STLT), AST/ALT ratio, AST to Platelet Ratio Index

Abstract PTU-001 Figure 1

(APRI) score, Fibrosis (FIB)-4 score, P3NP and HA. Statistical analysis was performed using Stata MP 15.1.

Results 3600 TEs were eligible for analysis. 104 SPs (2.9%) had TE ≥ 7.5 kPa. 8 SPs with suspected NAFLD had TE ≥ 11.7 kPa, 1 SP with suspected ARLD had TE ≥ 12.5 kPa. 472 (13.1%) SPs gave a history of excessive daily alcohol consumption or AUD. Of this group, 5.7% had TE ≥ 7.5 kPa. No non-invasive scores or scores accurately identified $\geq F2$ equivalent TE (≥ 7.5 kPa) in all SPs (area under ROC (AUROC) range 0.37–0.65). These results were reflected when assessing AUROCs to detect $\geq F2$ equivalent fibrosis (≥ 7.9 kPa) in SPs with suspected NAFLD (AUROC range 0.39–0.64). In SPs with suspected ARLD, APRI had a modest diagnostic accuracy (AUROC 0.73); all other markers and scores performed poorly in diagnosing $\geq F2$ equivalent fibrosis (AUROC range 0.24 – 0.67, excluding APRI).

Conclusions Non-invasive fibrosis scores have limited corroboration with TE in diagnosing moderate ARLD or NAFLD fibrosis. A study limitation is fibrosis scores were not compared to gold standard liver biopsy, in the setting of a large population study of young healthy adults; results should be interpreted in this context. The Enhanced Liver Fibrosis (ELF) test was not analysed, however, P3NP and HA – 2 of the ELF constituents – had low AUROCs.

PTU-002 DENDRITIC CELL SUBSETS AND SERUM INTERLEUKIN-12 ARE LINKED TO RENAL INJURY IN SCHISTOSOMAL HEPATIC FIBROSIS

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Introduction Chronic infection with schistosomiasis is associated with down-regulation of T-cell immune responses that require activation by innate immune cells like dendritic cells (DCs). The present work was designed to study the DC subsets (myeloid and plasmacytoid) in peripheral blood and serum levels of interleukin (IL)-12 in patients with schistosomal hepatic fibrosis (SHF) in relation to severity of liver disease and renal injury.

Methods Forty five patients with SHF and 15 healthy subjects were included in the study. The severity of liver disease was assessed using Child-Pugh classification and the Model of End Stage Liver Disease (MELD) score. Renal injury was assessed by measuring urinary albumin excretory rate (UAER) and estimated glomerular filtration rate (eGFR). The percentages of DC subsets in peripheral blood were detected using 3-color flow cytometric assay. Dendritic cells were identified as lineage marker negative (lin-)/HLA-DR positive cells and the differentiation of myeloid DC subset from lymphoid DC subset was based on the expression of CD11c or CD123 on the cell surface respectively. Quantitative determination of IL-12p70 heterodimer in serum was performed using enzyme-linked immunosorbent assay. Renal biopsies from patients with macroalbuminuria were examined by immunohistochemical technique for DCs and angiogenesis using antibodies against OX62 and factor VIII-related antigen respectively.

Results The percentages of circulating CD11c⁺ and CD123⁺ DC cells, CD11c⁺DC/CD123⁺DC ratio and serum IL-12 levels showed significant decreases in patients with micro- and macro-albuminuria compared with patients with normo-albuminuria and healthy subjects ($P < 0.01$). Renal tissues from patients with macroalbuminuria showed significant increases in the number of DCs and angiogenesis compared with normal renal tissues ($P < 0.01$). The percentages of circulating DC subsets and serum IL-12 levels were inversely correlated with Child-Pugh score, MELD score, number of renal DCs, renal angiogenesis and UAER and were positively correlated with eGFR. The number of renal DCs showed positive correlations with UAER and renal angiogenesis and negative correlation with eGFR ($P < 0.05$).

Conclusions DCs and IL12 seems to play a role in the progression of liver disease and renal injury in SHF. DC-based vaccines may provide a potential new goal for immunotherapy in SHF.

PTU-003 MICRORNA-17 HOST GENE PROTEIN AS A POTENTIAL BIOMARKER FOR HEPATIC FIBROSIS IN CHRONIC HCV INFECTION

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Introduction MicroRNAs (miRs) are recognized as major players in various biological processes. Circulating miRs can be used as non-invasive, sensitive biomarkers for detecting disease. The polycistronic miR-17~92 cluster is comprised of six miRs and its primary transcript also encodes for a polypeptide of 70 amino acids designated as the miR-17 host gene (MIR17HG) protein. The present study was designed to evaluate plasma MIR17HG protein as a potential biomarker for hepatic fibrosis in patients with chronic hepatitis C virus (HCV) infection.

Methods Thirty treatment-naïve patients with chronic HCV infection [18 patients with chronic hepatitis C (CHC) and 12 patients with cirrhosis] and 15 healthy subjects were included in the study. Quantitative determination of plasma levels of MIR17HG protein was performed using sandwich enzyme immunoassay. Core liver biopsies were obtained from all patients and were assessed for METAVIR histological activity grade and fibrosis stage as well as steatosis grade. Fibrosis scores including aspartate aminotransferase to platelet ratio index (APRI) and Fibrosis-4 (FIB-4) score were calculated. Receiver operating characteristic (ROC) curve was plotted to assess the performance of plasma MIR17HG protein, APRI and FIB-4 score in discriminating patients with early hepatic fibrosis ($\leq F2$) from patients with advanced fibrosis ($> F2$).

Results Plasma MIR17HG protein levels were significantly higher in patients with chronic HCV infection than in healthy subjects, in patients with cirrhosis than in patients with CHC and in patients with advanced fibrosis than in patients with early fibrosis ($P < 0.001$). Plasma MIR17HG protein levels were positively correlated with serum levels of aminotransferases ($P = 0.014$ and $P = 0.036$ respectively), histological activity grade ($P = 0.002$), fibrosis stage ($P < 0.001$) and