of this protein lies within its coiled coil region. Two new epitopes, one of which dominant, have being discovered, both in PBC and SS. The fact that the fine epitope specificity of anti-Ro-52 is virtually identical in PBC and SS suggests a common mechanism of tolerance breakdown to this autoantigen in the two conditions.

P06 HEPATIC LUMICAN EXPRESSION IN PAEDIATRIC NON-ALCOHOLIC FATTY LIVER DISEASE

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Introduction Lumican is a glycoprotein involved in collagen cross-linking and modulation of the innate immune system. Over-expression of lumican was recently described in a group of adult patients with histologically progressive non-alcoholic fatty liver disease (NAFLD). It has not yet been evaluated in paediatric NAFLD.
Aim The aim of this study was to determine the degree of lumican expression in the liver of children with varying stages of NAFLD.
Method The study group consisted of 24 children (17 boys), median age 13.1 years, with liver biopsy-proven NAFLD and six children with chronic liver disease other than NAFLD (four with auto-immune hepatitis and two with Wilson disease). Paraffin-embedded biopsy sections were scored according to the NAFLD Activity Score (NAS). Sections were immuno-stained for lumican using HRP-DAB. Quantitative analysis was performed using imageJ (NIH, USA), expressing lumican staining as percentage of the total area. In addition, relative quantification real-time PCR for lumican was undertaken on frozen biopsy specimens.
Results Median BMI z-score of the group with NAFLD was 2.2 and median HOMA-IR; 4.4. 58% had splenomegaly. Thirteen children scored =5 (NASH), 6 scored 3–4 (borderline) and 5 scored=2 (simple steatosis). Fibrosis was minimal in 10 (F<2) and significant in 14 (F=2). Two children had type 1 NASH, the remainder had type 2 or a mixed pattern. Lumican was overexpressed in those with significant fibrosis (F=2) vs those with minimal fibrosis (F<2); (168%, p=0.01). Lumican was also overexpressed in NASH vs simple steatosis (215%, p=0.012). The pattern of lumican staining followed the sinusoidal contour, and marked the portal vascular endothelium and the luminal border of bile ducts. There was no clear staining of hepatocytes. At gene level, lumican was upregulated (compared to normal control liver) in those with F=2 (15.8-fold) and in those with F<2 (10.9-fold). Lumican expression was not related to age, BMI z-score, HOMA-IR, splenomegaly or transaminase levels. There was variable expression of lumican in the biopsies of those with chronic liver disease other than NAFLD. Percentage area stained did not correlate with degree of fibrosis in these patients.
Conclusion Lumican is expressed with increasing severity of paediatric NAFLD. Upregulation at gene level in those with both minimal and histologically more severe disease is also evident. The role of lumican in progression of disease has not yet been elucidated and should be the focus of further investigation.

P07 SPLANCHNIC STEAL IN PATIENTS WITH LIVER DISEASE: A 3T MRI STUDY OF VISCERAL BLOOD FLOW

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Introduction Liver cirrhosis and the development of portal hypertension is associated with significant morbidity and mortality. It is widely accepted that patients with liver disease have a hyperdynamic circulation that is associated with an increased cardiac output. We have previously proposed that, rather than a generalised systemic vasodilatation, there is selective splanchnic vasodilatation with concomitant vasoconstriction in other vascular beds: the so-called "splanchnic steal" phenomenon.
Aim To measure regional visceral blood flow using 3T magnetic resonance imaging in patients with liver disease.
Method Single centre pilot study of 19 subjects (10 healthy controls, nine patients with liver disease). Arterial and venous phase magnetic resonance angiograms were obtained using a Siemens 3T Verio MR scanner with gadolinium contrast. From these MRA's, ECG-gated phase contrast flow measurement MR data were then positioned and measured in the hepatic artery, portal vein, superior mesenteric artery, descending thoracic aorta, distal abdominal aorta, and the renal and carotid arteries.
Results Mean MELD score in patient group was 14 (range 7–21) with a range of aetiologies: alcoholic (6), non-alcoholic fatty (1), autoimmune (1), hepatitis C virus (1). In comparison to controls, flow in the descending thoracic aorta was increased by 43% in patients with liver disease (4.74 vs 3.32 L/min; p=0.021) consistent with an increased cardiac output. Hepatic artery flow showed a trend towards increase in patients (0.47 vs 0.27 L/min; p=0.11) whereas portal vein flow decreased dramatically (0.20 vs 1.20 L/min; p=0.006). Overall, in patients with liver disease, there was a 46% reduction in total liver blood flow (0.67 vs 1.47 L/min; p=0.037) and a reversal of hepatic artery/portal vein flow ratio (4.15 vs 0.33 L/min; p=0.009). Although superior mesenteric artery flow was three times greater in patients (0.54 vs 0.15 L/min; p=0.001), renal blood flow showed a trend towards reduction of 32% (0.42 vs 0.62 L/min; p=0.053), no change in carotid blood flow (0.75 vs 0.62 L/min; p=0.129) and no change in inferior aortic flow (1.45 vs 1.12 L/min; p=0.28).
Conclusion There are marked derangements in regional visceral blood flow in patients with liver cirrhosis. Our findings strongly support the splanchnic steal hypothesis that dysregulated splanchnic vasodilatation and porto-systemic shunting induce a high cardiac output state associated with extra-splanchnic vasoconstriction including the renal circulation.

P08 PEOPLE WITH DIABETES HAVE A HIGHER RISK OF DEATH FROM LIVER DISEASE COMPARED TO THE GENERAL POPULATION

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Introduction The association between liver disease and diabetes has been described, but less information is available regarding mortality from liver disease among people with diabetes from a population perspective.
Aim To compare mortality from liver disease among people with prevalent diabetes in 2001 and incident diabetes between 2001 and 2007 with that of the general population of Scotland.
Method We used a population-based diabetes register derived from primary and secondary care electronic records linked to death records to compare mortality from liver disease among people with prevalent diabetes in 2001 and incident diabetes between 2001 and 2007 with that among the general population of Scotland for people of 35–84 years for the period 2001–2007. There were just over 1 million person years of data for people with diabetes and almost 20 million person years of data for the general population. Mortality rates were estimated for liver disease as underlying (primary) cause of death on death certificates using conventional ICD-10 codes including those for hepatocellular carcinoma (HCC). Standardised