Introduction B cells classically provide humoral immunity in the form of antibody production as part of the adaptive immune response. Regulatory and antigen presenting functions of B cells have been reported before and autoantibodies are associated with autoimmune liver diseases. B cell depletion in animal models of PBC has highlighted the regulatory roles of B cells in ameliorating disease. Some evidence of efficacy of anti-B cell therapy using rituximab in human autoimmune liver diseases further supports a role for B cells. Mature B cells (Bm) subpopulations had been described in Sjogren's syndrome. However, little is known about the localisation, subsets, phenotype and function of B cells in human liver diseases.
Methods In this study we characterised the frequencies of B cell subsets in the blood and liver of patients with inflammatory and autoimmune liver diseases.
Results Frequencies of naïve mature BM1 cells were reduced in the liver compared to blood (7.5% ± 2.3 vs. 20.2% ±2.8 p = 0.0022) and $IgD^{neg}CD27^{neg}$ subset was increased in diseased livers compared to diseased blood (22.9% ± 6.8 vs. 6.0% ± 1.1 p = 0.0013). B cells localise close to the bile ducts in PBC and reside around hepatocytes in AIH. Frequencies of regulatory B cells ($CD19^{pos}CD24^{hi}CD38^{hi}$) were significantly reduced in diseased blood vs. control blood (1.8% ± 0.4 vs. 3.6% ± 0.5 p = 0.01) similar to recent observation in acute rheumatoid arthritis. However this population is increased in the diseased liver compared with blood (6.2% ± 0.07 vs. 1.8% ± 0.4 p = 0.007), suggesting enrichment of regulatory B cells within the inflamed liver. Liver infiltrating B cells were capable of IL-10 production.
Conclusion We have characterised for the first time the heterogeneity of B cell subsets and presence of regulatory B cells and IL-10 secreting B cells in human diseased livers. We showed that B cells reside close to bile ducts along with other immune cells; thus B cells may play a role in biliary pathology.
Disclosure of Interest None Declared.

PWE-131 FACTORS CONTRIBUTING TO VARIANCE BETWEEN ARFI ELASTOGRAPHY AND LIVER HISTOLOGY: RESULTS OF A LARGE UNSELECTED CONSECUTIVE SERIES WITH SIMULTANEOUS BIOPSY OF ARFI MEASUREMENT SITE

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Introduction ARFI™ (Acoustic Radiation Force Impulse) elastography is a widely applicable technique for the non-invasive assessment of liver fibrosis, which has been well validated in viral hepatitis patients. As with transient elastography, the predictive value of this technique falls in intermediate stages of fibrosis, and shear velocity readings may also be affected by a number of other factors. However, few studies have systematically exam: ined the causes of the observed variance with liver histology. We report the results of a large unselected series, in which liver stiffness and histology have been sampled simultaneously from the same region of liver tissue.
Methods One hundred and eighty six unselected, consecutive secondary care referrals underwent simultaneous elastography and liver biopsy from the same right lobe liver window, both performed or supervised by a single senior radiologist in all cases. ARFI shear velocity measurements were made using a standard 10 observation technique, and biopsies taken using an 18G Biopince™ needle. All biopsies were reviewed by a single specialist histopathologist. Clinical, laboratory, elastographic and histological data were analysed retrospectively. ARFI/histological variance (AHV) was defined as a difference of more than 1 Metavir or 2 Ishak stages from that predicted by ARFI, according to standard calibration.[1]
Results Aetiologies were 99 viral hepatitis, 39 autoimmune (AILD) and other in 48. AHV was seen in 56(30.1%), of which 46(82%) showed a lower histological stage than predicted. AHV was not associated with age, gender, or ARFI measurement depths. Inflammation (ALT, necroinflammation), steatosis (US echogenicity, histology), suboptimal ARFI quality (IQR/median > 0.3) and AILD aetiology were significantly more common in AHV (p = 0.01, 0.007, <0.001 and 0.018, respectively). Two or more of these variables were present in 61% of variants, compared with 26.9% of non-variants (p < 0.001).
Conclusion These simultaneous paired data show that ARFI/ histological variance is common and influenced by aetiology, inflammation, steatosis and technical quality. It is more common in active autoimmune liver disease than in viral hepatitis. Assuming that histology is a true "Gold standard", taking these 4 factors into account when interpreting ARFI scores will assist in assessing the predictive reliability of elastography, and hence in clinical decision making with regard to liver biopsy and treatment. Further prospective studies with paired ARFI/histology sampling are warranted to confirm these findings.

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PWE-132 ASSOCIATION BETWEEN SMOKING AND LIVER FIBROSIS IN PRIMARY BILIARY CIRRHOSIS

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Introduction Conflicting data for the role that cigarette smoking may play in Primary Biliary Cirrhosis (PBC) have been reported. Some studies have suggested an association of smoking with a more advanced fibrotic stage. The aim of the present study therefore was to assess the association between smoking and a) the severity of histological findings at the time of diagnosis, b) the immunological features of a genetically homogeneous and geographically defined population of PBC patients.
Methods Smoking history data were collected from 171 PBC patients of Cretan origin (163 female) using a standardised questionnaire. Diagnosis was based on standard biochemical, Immunological and histological criteria. Liver biopsy was performed in 148 patients at diagnosis. Liver fibrosis and histological inflammatory activity were semi-quantified according to a METAVIR-based classification system. Odds ratios (OR) were assessed using logistic regression analysis.
Results Smoking history prior to diagnosis was reported in 56 patients (32,7%%). Twenty-six patients (15,2%) were active smokers at diagnosis. Male gender (AOR 8.19, 95% CI: 3.014–11.937), alcohol intake >20 g/d (AOR, 2.20, 95% CI: 1.029–4.099), severe steatosis (AOR, 5.31, 95% CI: 2.019–9.919)), and F3–F4 fibrosis stage (AOR 1.21 95% CI: 1.015–3.031), but