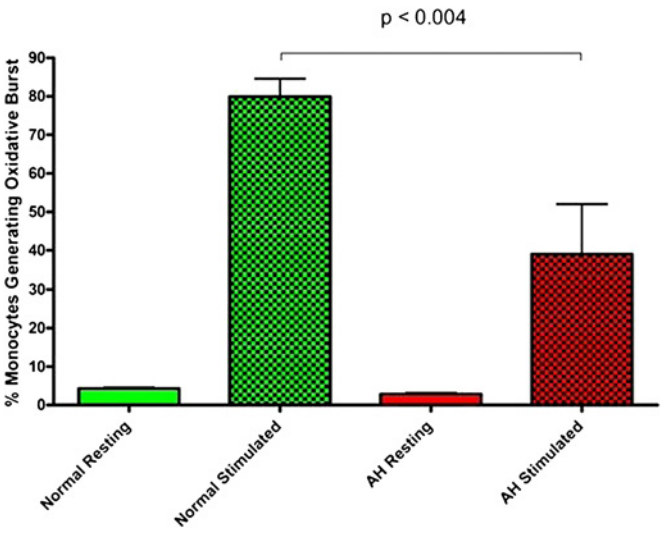
(77% vs 87%; p<0.03) but this was not a result of complement deficiency as phagocytosis did not depend on whether the bacteria were opsonised or not (73% vs 80%; p=0.9). The proportion of monocytes capable of generating an oxidative killing burst in response to phagocytosed *E coli* was markedly reduced (84% vs 47%; p<0.004) in AH patients compared to HC [Abstract PTU-016a figure 1]. Antigen presentation was also impaired: classical monocytes had significantly lower HLA-DR expression in AH compared to controls (73% vs 35%; p=0.002), with similar levels of HLA-DR expression detected in the CD14+CD16+ monocyte subset (94% vs 74%; p=0.4).



Abstract PTU-016a Figure 1

Conclusion It appears that there are a number of functional defects in circulating monocytes in patients with AH. The marked impairment of phagocytosis and intracellular killing may contribute to the increased susceptibility to infection in this group of patients.

Competing interests None declared.

General Liver I

PTU-017 IGG4 +VE AUTOIMMUNE HEPATITIS IS NOT OBSERVED AMONG PATIENTS OF NON-ASIAN ORIGIN

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Introduction IgG4 mediated autoimmune disease has recently been described in relation to pancreatitis and sclerosing cholangitis. Furthermore recent reports, mainly from Japan, have also identified cases of autoimmune hepatitis (AIH) in which an IgG4 positive hepatic inflammatory infiltrate predominates.

Methods We set out to establish the frequency with which IgG4 +ve plasma cells are observed in a cohort of patients with AIH without pancreatitis or sclerosing cholangitis. Immuno-histochemical analysis via application of a monoclonal antibody to IgG4 was undertaken on archived liver tissue specimens. Three fields per biopsy specimen were analysed and the number of IgG4+ve plasma cells per high powered field (HPF) and the proportion of these as a total of the plasma cell infiltrate were recorded. As per recent publications[1 2] a biopsy specimen was determined to be IgG4 +ve if more than 10 IgG4 +ve plasma cells were seen per high powered filed (HPF) AND where this number equated to >40% of the total plasma cell infiltrate. Immunohistochemical and histopathological analysis was undertaken by a single, experienced, hepato-pathologist (YZ).

Results Sixty-three liver tissue specimens underwent immunohistochemical analysis. These specimens derived from 53 Caucasian and 10 Afro-Caribbean patients. All patients met the revised International Autoimmune Hepatitis Group diagnostic criteria for probable or definite AIH. The median age of patients at diagnosis was 31 years and 78% were female. Among this cohort 44 patients presented with chronic disease and histological evidence of chronic active hepatitis while 19 presented with acute disease in whom histology demonstrated hepatic collapse in 16 and severe lobular hepatitis in three patients. Only 7 of 63 samples (11%) demonstrated >10 IgG4 +ve cells/HPF. While there was a greater number of biopsy specimens with >10 IgG4 +ve plasma cells per HPF in acute vs chronic presentations this did not reach statistical significance (16% vs 9%, p=0.44). Additionally, there was no difference in the frequency of this finding between males and females (males 21% females 8% p=0.4) or between different ethnic groups (11% in Caucasians vs 10% in Afro-Caribbeans, p=0.99). Importantly, in none of the seven cases in which >10 IgG4 +ve plasma cells/HPF were noted did the proportion of IgG4 +ve plasma cells equate to >40% of the total plasma cell infiltrate.

Conclusion While a small proportion of non-Asian AIH patients demonstrate >10 IgG4 +ve plasma cells/HPF, in no individual did this represent >40% of the total hepatic plasma cell infiltrate. Consequently, significant IgG4 +ve plasma cell infiltrates are not observed among non-Asian patients with AIH presenting to our institution.

Competing interests None declared.

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PTU-018 CYSTATIN C AND PROTEIN: CREATININE RATIO; POTENTIAL PREDICTORS OF EARLY ACUTE KIDNEY INJURY, RENAL REPLACEMENT THERAPY AND IN-HOSPITAL DEATH IN PATIENTS WITH CIRRHOSIS

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Introduction Acute kidney injury (AKI) is common, but difficult to predict in patients with cirrhosis as lower baseline serum creatinine can mask significant chronic kidney dysfunction. Cystatin C is a biomarker of glomerular filtration rate (GFR), which may overcome this weakness. Quantifying proteinuria using the widely available protein:creatinine ratio (PCR) may better the degree of structural glomerular damage.

Methods 34 patients with cirrhosis and mean (SD) age 51 (14) years and, median (range) Child-Pugh Turcotte (CPT) score 11 (9–11) were prospectively assessed for 10 days or until AKI, developed. Baseline iohexol clearance was performed to calculate GFR and urine underwent PCR analysis. Daily urine and serum samples were collected for determination of novel serum and urine biomarkers of kidney injury, including Cystatin C. Biomarkers were assessed by area under the receiver operating curve (AUROC) for predicting AKI stage 1, renal replacement therapy (RRT) and death.

Results 16 (47%) patients developed AKI defined by an increase of >26.4 umol/l from baseline serum creatinine (median 73 range (37–120 umol/l)). Estimated GFR overestimated GFR with median eGFR 95 (47–181) ml/min/1.73 m^2 compared to a median iohexol