Introduction Alcohol induced liver disease is the predominant cause of alcohol-related mortality in the UK. Therefore abstinence-based treatments are essential. Upto 70% of patients receiving alcohol treatment relapse within 6 months,[1] NICE attribute much of this failure of treatment to underutilisation of pharmacotherapy and recommend this be made available.[2] However, current licensed pharmacotherapies are contraindicated for patients with ALD. Baclofen has shown efficacy in the promotion of abstinence in patients with severe alcohol dependence[3,4] including those with ALD,[5] without exhibiting any of the complications or side effects elicited by current pharmacotherapies. Therefore the primary aim of this study was to measure the effectiveness of Baclofen in maintaining abstinence in this difficult to treat group.
Methods An observational prospective clinical audit was performed. Patients with liver disease and concomitant alcohol use were commenced on Baclofen at 10 mg three times daily (TDS), and titrated according to tolerability and response up to 30 mg TDS. Primary outcome measures were severity of physical dependence, as determined by SADQ score, and weekly alcohol consumption. These were compared at baseline, and 6 months.
Setting Acute Hospital Trust
Participants 149 patients referred to Hepatology for investigation of abnormal liver function and heavy drinking
Results Of the 149 patients commenced on Baclofen 100 (67.1%) remained engaged in treatment for 6 months. There was a significant reduction in alcohol consumption ($P < 0.0001$ 95% CI for difference 18 to 20) with 81 of the 149 patients (54.3%) maintaining total abstinence, 20 (13.4%) continued to drink and 48 (32.2%) were lost to follow-up and assumed to have returned to drinking. There was a significant reduction in the presence of physical dependence ($c^2 = 77.4$ $P < 0.0001$) as categorised by SADQ, and a non-significant improvement of liver biochemistry.
Conclusion Baclofen has a positive impact on alcohol consumption in this very difficult to treat, high risk patient group. A RCT is needed to confirm the benefit of baclofen in this patient group.

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Disclosure of Interest None Declared.

PTU-119 PHENOTYPIC CHARACTERISTICS AND LOCALISATION OF NOVEL HUMAN LIVER INFILTRATING NKP46 SUBSETS

[1]M Ming*, [1]C Thomas, [1]H Jeffery, [1]Y-Y Chen, [1,2]DH Adams, [1,2]DJ Mutimer, [1,2]YH Oo. *[1]Centre for Liver Research and NIHR BRU, University of Birmingham, UK; [2]Liver and Hepatobiliary Unit, UHB NHS Foundation Trust, Birmingham, UK*

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Introduction $CD56^+$Natural killer cells are the principal effector cells of the innate immune system and have a well-established role in tumour surveillance and anti-viral immunity. Expression of NKp46 has been shown to correlate closely with the severity of liver inflammation, viral resistance to IFN treatment and the attenuation of liver fibrosis. $CD56^+$NKp46 cells expressing IL-17 and IL-22 have also been described as a family of innate lymphoid cells in humans. Although the role of intrahepatic NK cells has been well described, little is known about the function and phenotype of intrahepatic NKp46 subsets. Thus, We aim to investigate the phenotypic characteristics of $CD56^+$ NKp46 cells in the inflamed human liver, with a view to exploring their functional role.
Methods Liver infiltrating lymphocytes were freshly isolated from explanted human liver tissue from our transplant program and phenotyped with multicolor flow cytometry. Cellular localization was investigated by immunohistochemistry and confocal microscopy
Results Human liver infiltrating NK cells reside predominantly around biliary epithelial cells at the portal tract close to regulatory T cells. We observed two populations of liver-infiltrating $CD3^{neg}$ $CD19^{neg}$ $CD56^{pos}$ cells distinguished by different levels of NKp46, $NKp46^{mid}$ (15% ±4.8 SD) and $NKp46^{high}$ (11% ±1.2 SD) neither subset expressed NKp44. The chemokine receptor expression of $NKp46^{mid}$ and $NKp46^{high}$ populations was: CCR6 (12% ± 3 vs. 7%± 2.4), CCR9 (20% ± 5.6 vs. 9% ± 0.9), CX3CR1 (18% ± 14 vs. 10% ± 1) CXCR3 (47%±14.4 vs 38%±11.0) and CXCR6 19% ± 4.0 vs. 14% ± 4.6). Both populations expressed IL-18R (42% ± 5.4 vs 7% ± 1.0), IL-23R (19% ± 6.0 vs. 11% ± 2.5), surface receptor CD161 (61% ± 12.1 vs 85% ±4.8) and the integrin receptor CD103 (4% ± 1.35 vs. 16% ± 1.7). The $NKp46^{high}$ population was highly enriched with the activation marker CD69 (77%±18%). NKp46 cells were also shown to express TNF-α (29% ± 7.5), IFN-γ (70% ± 7.0), Granzyme B (23% ± 11.0) and Perforin (23% ±11.1) along with transcription factor Tbet (19% ± 9.1).
Conclusion We hereby report novel subsets of liver infiltrating $CD56^+$NKp46 cells, which localise around the portal tract biliary epithelium in the inflamed human liver. These populations have distinct cytokine, chemokine and CD103 expression, which may explain their recruitment, positioning and effector functions in the inflamed hepatic microenvironment.
Disclosure of Interest None Declared.

PTU-120 EFFECTIVENESS OF NURSE LED HEPATITIS C TREATMENT; A LARGE DISTRICT GENERAL HOSPITAL AUDIT

N Elamin*, S Frayne, J Wadsworth, Y Reddy. *Gastroenterology, Royal Blackburn Hospital, Blackburn, UK*

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Introduction Hepatitis C is the third most common risk factor for liver diseases in the UK. Updated estimates suggest that around 216,000 individuals are chronically infected with hepatitis C. Treatment with combination of pegylated Interferon and ribavarin is well established. Specialist viral hepatitis nurses working collabaratively with clinicians play a major role in delivering excellent clinical outcomes.
Methods Evaluate the safety and clinical effectiveness of chronic hepatitis C treatment that was led by the specialist viral hepatitis nurses under the supervision of gastroenterologists.

Data was obtained from a prospectively maintained hepatitis C database over a 5-year period from September 2008 to date. A retrospective analysis of the database was carried out looking at the treatment outcomes. Patients with liver transplant and/or co-infection with hepatitis B or human immunodeficiency virus (HIV) were excluded. The dedicated viral hepatitis specialist nurses closely followed up all patients.
Results A large database of 437 patients who underwent treatment was analysed. There were 128 (29.2%) females and 309 (70.7%) males ranging between 23–84yrs old (mean age of 42).

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