Introduction Patients with liver cirrhosis are immunocompromised and prone to invasive infection where outcomes are usually poor. In particular, cirrhosis is a major risk factor for severe pneumococcal infection which has a mortality rate of up to 20%. Whilst patients awaiting liver transplantation routinely receive the pneumococcal vaccine, it is less clear whether the remainder of patients with cirrhosis do so or not. The present literature suggests that pneumococcal vaccination prophylaxis should be administered in cirrhotic patients and revaccination desirable at 5-yearly intervals.

The aims of this study were to assess whether patients with liver cirrhosis received pneumococcal vaccination in line with current recommendations at a single centre in the UK.

Methods A retrospective analysis of all patients with biopsy-proven liver cirrhosis, irrespective of aetiology, over a 7-year period (2005–2012) at Barnet and Chase Farm Hospitals was performed. We used a database of patients with biopsy-proven cirrhosis created by our histopathology department. Patient's primary care physicians were contacted in writing to assess whether they had received the pneumococcal vaccine.

Results 37 patients had biopsy-proven liver cirrhosis over the audit period. There was no response from the primary care physicians of 6 patients. Data for 31 patients (17 male, 14 female), median age 50 years were analysed. 14 (45%) patients had received the pneumococcal vaccine. 4 (28.5%) of these patients were vaccinated after their histopathological diagnosis of cirrhosis was proven. The median time interval from diagnosis to vaccination was 24 months in this group of patients. Of the 7 patients vaccinated before 2007, none had received a repeat vaccine after 5 years.

Conclusion More than half of patients with biopsy-proven liver cirrhosis in our study did not receive the pneumococcal vaccine placing these patients at risk of life threatening preventable disease. None of the patients who received the vaccine initially were revaccinated at five yearly intervals as present literature would recommend.

We would recommend an increased awareness of the importance of regular pneumococcal vaccination to all healthcare professionals, primary care physicians and those in secondary care, who come into contact with this important and growing cohort of patients.

Disclosure of Interest None Declared.

PTU-138 POPULATION-BASED STUDY OF ETHNICITY AND THE DIAGNOSIS GAP IN LIVER DISEASE

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Introduction Awareness of liver disease as a major cause of morbidity and mortality has led to an increase in liver function tests (LFTs) performed in primary care with abnormal results a common finding. However, we hypothesise that a large gap exists between numbers of patients with abnormal LFTs and those with recorded liver diagnoses. Non-alcoholic fatty liver disease (NAFLD) is the most common cause of chronic liver injury. Metabolic syndrome, common in people from the Indian subcontinent, is an important risk factor for NAFLD. We hypothesise that NAFLD is more common among adults of South Asian ethnicities.

Methods In a cross-sectional study of 690,683 adults, registered in co-terminus general practices in a region with high ethnic diversity, we extracted demographic information and clinical care data including LFTs in the previous two years, liver disease diagnoses and co-morbidites from the EMIS Web computerised medical records. The breakdown of age, gender, deprivation, BMI, smoking status, alcohol consumption, co-morbidities and cholesterol was described for the whole population and for the six main ethnic groups of Bangladeshi, Indian, Pakistani, White, African and Caribbean. STATA 12 was used to conduct multivariate logistic regression analyses.

Results LFTs were performed on 218,032 patients, of whom 31,627 had elevated serum transaminases. Testing varied by age, ethnicity and the presence of co-morbidities. The prevalence of abnormal LFTs was highest among Bangladeshis and independent risk factors for abnormal LFTs included male gender, alcohol consumption and elements of the metabolic syndrome.

The most commonly recorded liver diagnosis was NAFLD, followed by chronic viral infection and alcoholic liver disease. 88.4% (n = 27,985) of patients with abnormal LFTs did not have a coded liver diagnosis.

The prevalence of recorded NAFLD was highest among patients of Bangladeshi ethnicity. In a multivariate analysis, independent risk factors for NAFLD included Bangladeshi ethnicity, diabetes, raised BMI, hypertension and hypercholesterolaemia.

Conclusion Abnormal LFTs are common in the population, but are under-investigated and often remain undiagnosed. Bangladeshi ethnicity is an important independent risk factor for NAFLD. Among the group of patients with abnormal LFTs and no recorded liver diagnosis, many will have a liver disease that is amenable to further management, which may prevent complications. There is a need for evidence-based guidelines for the investigation, referral and management of patients with abnormal liver tests in the community in order to ensure early identification of treatable disease.

Disclosure of Interest None Declared.

PTU-139 OUTCOMES OF PATIENTS WITH LIVER CIRRHOSIS IN A NON LIVER-SPECIALIST INTENSIVE CARE UNIT: DO ADMISSION LACTATE AND APACHE 2 SCORE HELP PREDICT SUCCESSFUL DISCHARGE?

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Introduction Hospitalised patients with cirrhosis often require admission to intensive care (ITU), usually for management of secondary complications such as GI bleeding, sepsis, or organ failure. Prognosis of these patients is poor. Despite demonstration of modest improvements in recent years, overall in-hospital mortality in this group remained 54.6% in a recent study.[1] Our study aimed to explore the characteristics and outcomes of cirrhotic patients admitted to ITU, and whether this was comparable to published data. We also aimed to explore whether parameters such as APACHE 2 score and admission blood lactate differ between those who survived their ITU stay and those that did not, and if this may help predict discharge.

Methods A retrospective analysis was performed of patients admitted to the Whittington hospital ITU from January 2011-June 2013. Information regarding patients with a diagnosis of cirrhosis was gathered from the Intensive Care National Audit and Research unit (ICNARC) database, and discharge summaries.

Results We identified 60 patients with cirrhosis, 3.07% of total ITU admissions, mean age 54.8 years (range 19–78). 49/60

patients had alcoholic cirrhosis, 5/60 had non-alcoholic steatohepatitis related cirrhosis. Primary reason for admission included GI bleed (24/60, 40%), pneumonia (16/60, 26.7%), other sepsis (10/60, 16.7%), encephalopathy (8/60, 13.3%). Overall mortality figures were 41.7% in-ITU, 48.3% at 30 days, and 70% at 1 year. In-ITU mortality for patients requiring only ventilatory support was 48.9%, those requiring inotropic support was 61.2% and those requiring renal support 64.2%. 24/60 patients required all 3 methods of support, 66.7% of those died in ITU and 75% at 30 days. 23 patients were admitted with sepsis and decompensated liver disease, 65% died in ITU and 73.9% at 30 days. Mean APACHE 2 scores for patients that died in ITU vs. those discharged from ITU were 23 (range 9–30) and 20 (range 8–36) respectively, with a statistically significant difference between the two groups ($p = 0.036$). Mean admission serum lactate for patients who died in ITU vs. those discharged from ITU was 7.6 (range 1–23) and 4.6 (range 1–17) respectively, demonstrating a statistically significant difference ($p = 0.015$).

Conclusion As expected for a non liver-specialist unit, most patients had alcohol related cirrhosis. Mortality was high but comparable to other published data. The worst outcomes were seen in patients with sepsis and decompensated liver disease, and those requiring organ support. Admission lactate levels and APACHE 2 scores were significantly lower in patients successfully discharged from ITU; admission lactate could potentially aid prediction of successful discharge. Further study is needed.

REFERENCE

1 Intensive Care Med. Jun;2012;38(6):991–1000

Disclosure of Interest None Declared.

PTU-140 INTRAHEPATIC TREGS ARE PLASTIC BUT FUNCTIONAL AND BILIARY EPITHELIAL CELLS SUPPORT THEIR FATE

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Introduction Regulatory T cells (T_{regs}) are crucial in maintaining peripheral tolerance. T_{regs} control T effector CD8, CD4, Th_1 cells along with other immune cells to maintain hepatic tolerance. They are implicated in both human and murine model of hepatic inflammation including autoimmune hepatitis, viral hepatitis, liver cancer and post-transplantation tolerance. However little is known about the lineage stability, function and fate of human intrahepatic T_{regs} in the inflamed microenvironment.

Methods Human liver infiltrating (LI) lymphocytes were freshly isolated from explanted liver tissues. LIT_{regs} cells surface phenotype, chemokine and cytokine receptor expression, intracellular-cytokine secretion was assessed *ex-vivo* by flow cytometry. Function and plasticity of post-endothelial transmigrated (PEM) T_{regs} in the inflamed microenvironment was assessed by suppression assays and flow cytometry. Distribution and localisation of LIT_{regs} in tissue was determined using dual immunohistochemistry and confocal microscopy. Cytokine expressions by the liver microenvironment were studied *in vitro* using Luminx. Real time PCR was used to study the mRNA expression. Survival and proliferation of PEM T_{regs} in microenvironment was studied *in-vitro* using co-culture assays using primary human biliary epithelial cells.

Results LIT_{regs} highly express CD39 (57 ± 11%), CD95 (83 ± 4%), CD27 (73 ± 3%), CD44 (90 ± 3%) and low expression of CD40 (6.813 ± 3.25%). Cytokine receptors expression was (31 ± 15%) for IL15R, (17 ± 15%) for IL6R-α. Hepatic microenvironment is highly enriched with IL-1β (363 ± 88 pg/ml), IL-6 (8,960±pg/ml), IL-12 (44 ± 35 pg/ml), IFN-γ (21 ± 8.33 pg/ml). Minimal level of IL-2 was detected in inflamed liver supernatant. Post-endothelial migrated (PEM) T_{regs} and T_{regs} in the inflamed microenvironment are functional but suppression capacity was reduced in T_{regs} residing in the inflamed liver. Plasticity to other T cells lineage is minimal for T_{regs} in the inflamed microenvironment. LIT_{regs} reside close to bile ducts at the portal tract. Co-culture experiment of PEM T_{regs} and with biliary epithelial cells suggested that T_{regs} survival depends on FAS-FASL pathway and IL-2.

Conclusion LIT_{regs} are plastic but functional in the inflamed intrahepatic microenvironment and their fate around biliary epithelial cells is supported via IL-2 cytokine and CD95-CD95 ligand pathway.

Disclosure of Interest None Declared.

Oesophagus, stomach, duodenum I

PTU-141 GASTRIC INTESTINAL METAPLASIA: A RETROSPECTIVE ANALYSIS IN A DISTRICT GENERAL HOSPITAL IN THE UNITED KINGDOM

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Introduction Gastric intestinal metaplasia (IM) is part of a carcinogenesis sequence leading to gastric cancer. Recent evidence-based European Society of Gastrointestinal Endoscopy (ESGE) guidelines highlight additional risk factors, such as extensive intragastric distribution of IM and the presence of *Helicobacter pylori*. The former is identified with ≥2 antral and ≥2 corpus biopsies, involving the greater and lesser curvature, and warrants three-yearly surveillance endoscopies. The latter should be eradicated to slow carcinogenesis progression.

Methods Using keywords "intestinal metaplasia", the histology database for the Queen Elizabeth Hospital, South London, was reviewed over 2000–11 to identify patients with IM on gastric biopsy. Gastro-oesophageal junctional IM was excluded. The number and site of biopsies taken and the presence of *H. pylori* was identified. The terminology used, with regards to "extensive" and "focal" IM, was compared with the suggestions from the ESGE guidelines. To investigate the development of cancer in patients with IM, histology and upper gastrointestinal cancer databases were compared.

Results 175 patients with gastric IM were identified. Of these, only one patient developed gastric cancer. *Helicobacter pylori* was associated with 20/175 (11.4%) of gastric IM biopsies. After review of pathology reports, in 37/175 (21.1%) of cases with gastric IM, the pathologist did not receive sufficient clinical information specifying the site of the biopsies. Of those where the biopsy site was specified, only 10/138 (7.2%) had sufficient biopsies. The term "extensive" was used in 27/175 (15.4%) pathology reports, despite either insufficient number or non-specified location of biopsies.

Conclusion This study identified 175 patients with gastric intestinal metaplasia over 2000–2011. One patient developed gastric adenocarcinoma after 8 years. Since surveillance endoscopy is not routine practice in the Trust, all biopsies were incidental