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

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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