

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.

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Disclosure of Interest None Declared.

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

¹Y-Y Chen*, ¹J Hannah, ²J Birtwistle, ³I Novitzky Basso, ¹P Lalor, ¹DH Adams, ¹YH Oo. ¹Centre for Liver Research and NIHR BRU, University of Birmingham, UK; ²Clinical Immunology, UHB NHS Foundation Trust, UK; ³MRC Centre for Immune Regulation, University of Birmingham, Birmingham, UK

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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 ($57 \pm 11\%$), CD95 ($83 \pm 4\%$), CD27 ($73 \pm 3\%$), CD44 ($90 \pm 3\%$) and low expression

of CD40 ($6.813 \pm 3.25\%$). Cytokine receptors expression was ($31 \pm 15\%$) for IL15R, ($17 \pm 15\%$) for IL6R- α . Hepatic microenvironment is highly enriched with IL-1 β (363 ± 88 pg/ml), IL-6 ($8,960 \pm$ 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

A Singanayagam*, N Chatrath, L Pee, A Loganayagam. Gastroenterology, Queen Elizabeth Hospital, Woolwich, London, UK

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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 intra-gastric 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

findings. This study suggests that, where biopsy site details were provided, only 7.2% of patients were adequately biopsied. Remaining cases should have repeat biopsies to decide on surveillance. "Extensive metaplasia" refers to a wide intragastric distribution of IM to include the antrum and corpus. We identified discrepant use of nomenclature in pathology reporting in 15.4%. *Helicobacter pylori* was associated in 11.4%, where ESGE advocates its eradication. This study reveals further work is needed to risk stratify and survey this important pre-cancerous condition.

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PTU-142 HIGH PREVALENCE OF GASTROINTESTINAL STROMAL TUMOURS (GISTS): A CASE SERIES IN UK SECONDARY CARE

C Shekhar*, NC Fisher. *Gastroenterology, Russells Hall Hospital, Dudley, UK*

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Introduction Gastrointestinal Stromal Tumours (GISTs) are mesenchymal tumours, predominantly affecting the GI tract. Diagnosis and classification require specialist review and there are few published data on the incidence of GIST in the UK. Reported incidences elsewhere vary between 6.5/ million/ year in Norway and 14.5/ million/year in Sweden.^{1,2} We have analysed our caseload of GISTs in a UK secondary care setting with a population of approx 350,000, in order to estimate incidence and review outcomes.

Methods A retrospective case note reviews of all patients with GIST, as identified from upper GI cancer MDT minutes, from 2008 to 2012 inclusive (5 years). The diagnosis of GIST was considered valid if characteristic imaging and/ or pathological features were verified by CT scanning, endoscopic ultrasound (EUS) needle aspiration/ biopsy and/ or surgical resection.

Results We identified 28 cases with a final diagnosis of GIST. The observed incidence varied between 4 and 8/ year, and estimated annual incidence was calculated at 16/million/year. The age range was 28–91 years (M 12, F16). Nineteen cases (68%) presented with signs or symptoms of GI blood loss; five (18%) with other GI symptoms and remaining cases were found incidentally. GIST size at presentation ranged from 1cm to 20cm in diameter. One case had metastasised at the time of diagnosis. EUS was used for diagnosis and staging in 15 cases; 13 had fine needle aspiration, of which 10/13 were diagnostic. 22 cases underwent resection surgery. 6 cases were treated with Imatinib (Glivec).

Conclusion Our review suggests a higher than expected incidence of GISTs in this population compared with other published series.^{2,3} Most cases present with GI blood loss and surgery is curative in most cases. The incidence of GISTs in the UK is deserving of further study.

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PTU-143 SHOULD WE INVESTIGATE MESENTERIC PANNICULITIS?: UK EXPERIENCE OF 58 PATIENTS

C Ford*, P Patel, K Burney, C Fernandez, R Fisher, P Youd. *Gastroenterology, St Helier Hospital St Helier and Epsom NHS Trust Surrey, London, UK*

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Introduction Mesenteric panniculitis (MP) is an inflammatory condition of the bowel mesentery with characteristic features on CT (computer tomography). Studies suggest MP is associated with malignant pathology, previous abdominal surgery, inflammatory and autoimmune diseases. There is a lack of consensus on the clinical significance of MP and its further investigation.

Methods A retrospective analysis of medical records, imaging, endoscopy reports and histology.

Results 58 patients were identified with mesenteric panniculitis by CT criteria during the study period. 8 patients (13.8%) had undergone previous abdominal surgery. 12 patients (20.7%) had a previous history of malignancy; lymphoma 3, prostate 2, bladder 2, both lymphoma/bladder 1, leukaemia 1, endometrial 1, carcinoid 1, and bronchial 1.

Following index CT a new malignancy was identified in 5 patients (8.6%) and recurrence of a previous cancer in 1 (1.7%). 1 patient was diagnosed with lymphoma, 1 gastric carcinoma, 1 malignant myeloma, 1 bronchial carcinoma and 1 bladder cancer. 1 patient was diagnosed with a recurrence of a previously treated lymphoma. Of these 6 patients, 2 underwent endoscopic investigation; gastric carcinoma/lymphoma was suspected on index CT and endoscopy performed for histological confirmation.

Of the remaining 52 patients with MP on index CT (and no new or recurrent malignancy) 18 (34.6%) underwent further endoscopic investigation. None of these patients were diagnosed with a new malignancy at the time of endoscopy; a new diagnosis of ulcerative colitis was made in 2 (3.8%). 15 patients (36.8%) underwent a follow up CT scan within an 18 month period. None were diagnosed with a new malignancy at the time of follow up CT.

Conclusion This study suggests a high prevalence of malignancy amongst patients with MP on index CT. The diagnosis of MP on CT should alert the physician to the possibility of an undiagnosed malignancy.

MP is poorly understood and inconsistently followed up. Its diagnosis can lead to investigation with poor clinical yield and patient/cost implications. This study suggests a diagnostic strategy for underlying malignancy should focus on close evaluation and scrutiny of index CT prior to consideration of further investigation. A larger study is required to identify the prevalence of associated organ specific malignancies, define the diagnostic yield of further investigation and inform an evidence-based diagnostic approach.

Disclosure of Interest None Declared.

PTU-144 WHEN ARE GASTRIC ULCERS MALIGNANT? PREDICTORS OF BENIGN DISEASE

S Thanaraj, A Sainsbury, R Cochrane, C Selinger*. *Gastroenterology, Leeds Teaching Hospitals NHS Trust, Leeds, UK*

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Introduction Gastric ulcers can harbour malignancy and the National Institute for Health and Care Excellence (NICE) therefore recommends follow-up gastroscopy (FU-OGD). Predictors of