compared to 18 gauge needle. Despite being a larger bore needle, it is not associated with an increased rate of complications. We recommend using 16 gauge co-axial needles routinely for percutaneous liver biopsies.

Disclosure of Interest None Declared.

REFERENCES

1. Guidelines on the use of Liver Biopsy in Clinical Practice: 2004. British Society Of Gastroenterology: (www.bsg.org.uk)
2. Tissue pathways for liver biopsies for the investigation of medical disease and for focal lesions: GO64, May 2008, The Royal College of Pathologists
3. Liver Biopsy :AASLD position statement : Don C. Rockey, Stephen H. Caldwell, Zachary D. Goodman, Rendon C. Nelson, Alastair D. Smith;HEPATOLOGY, Vol. 49, No. 3, 2009

PWE-121 COMPARISON OF KINGS SCORE, APRI AND AST/ALT RATIO IN DETERMINING SEVERITY OF LIVER DISEASE VERSUS LIVER BIOPSY

doi:10.1136/gutjnl-2013-304907.409

[1]C Holdstock, [1,*]M Joseph, [1]N Thomas, [2]T Bracey, [3]T J Cross. *[1]South West Liver Unit; [2]Department of Pathology, Derriford Hospital, Plymouth; [3]Hepatology Department, Royal Liverpool Hospital, Liverpool, UK*

Introduction Background and Aims Non-invasive markers of liver fibrosis are used to stratify the severity of Liver disease. The aim of the study was to compare the accuracy of the AST/ALT ratio, AST-platelet ratio index (APRI), and the Kings score in determining significant liver disease using liver biopsy as the reference standard.

Methods A retrospective analysis of patients presenting for liver biopsy at the South West liver unit was reviewed. All patients had routine demographic, biochemical and haematological parameters collected including: aspartate aminotransferase (AST), alanine aminotransferase (ALT), platelets, international normalised ratio (INR) and patient age. The quality of the liver biopsy specimen was recorded including sample length, fragments, and number of portal tracts. Liver biopsy fibrosis was staged using the Ishak score. Non-invasive tests were assessed in their ability to correctly identify significant fibrosis (Ishak stage ≥ F3) or cirrhosis (Ishak Stage ≥F5). The scores were calculated as follows: AST/ALT; APRI = ((AST/AST upper limit of normal))/(Platelets) x100, and Kings score = (AST x Age x INR)/platelets. The accuracy of each test was compared to the reference standard using area under the receiver operated characteristic curve (AUROC).

Results 170 patients were identified. 130 patients had complete data to calculate the scores. The median age 56 years (IQR 45–65), 55% patients were male. Numbers of patients by disease were: autoimmune hepatitis n = 23 (18%), PBC n = 3 (2.3%), PSC n = 2 (1.6%), Fatty Liver disease n = 24 (19%), ALD n = 26 (20%), HCV n = 12 (9.3%), Others n = 40 (30%). The median biopsy length 20mm (17 – 26), portal tracts 9 (5 – 13), biopsy cores 2 (1 – 2). AUROC for significant fibrosis (≥ F3) : AST/ALT = 0.84 (0.77 – 0.91), p = < 0.0001, Kings Score = 0.73 (0.64 – 0.83), p < 0.0001, APRI = 0.69 (0.60 – 0.79), p < 0.0001. AUROC for cirrhosis (≥ F5): AST/ALT = 0.82 (0.75 – 0.90), p < 0.0001, Kings Score = 0.71 (0.61 – 0.80), p < 0.0001, APRI = 0.66 (0.57 – 0.76), p < 0.0001.

Conclusion Conclusions: The AST/ALT ratio had the greatest diagnostic accuracy in determining significant fibrosis or cirrhosis. The Kings score performed better than APRI. AST/ALT is a simple guide to determine significant fibrosis and cirrhosis in liver disease.

Disclosure of Interest None Declared.

PWE-122 PARALLEL TIPSS FOR THE MANAGEMENT OF SHUNT INSUFFICIENCY IN PATIENTS WITH COMPLICATIONS OF PORTAL HYPERTENSION: A TERTIARY LIVER UNIT 19 YEAR EXPERIENCE

doi:10.1136/gutjnl-2013-304907.410

[1,*]N Rajoriya, [2]H Merzhat, [2]K Mangat, [2]S Oliff, [1]D Tripathi. *[1]Liver Medicine; [2]Radiology, The New Queen Elizabeth Hospital, Birmingham, UK*

Introduction Transjugular Intrahepatic Porto-Systemic Shunts (TIPSS) insufficiency can be addressed with a side placement of another TIPSS beside the original ("parallel" technique) thus improving portosystemic pressure gradient (PPG). There is a paucity of data assessing the efficacy of this technique.

The Aim of this study was to assess the efficacy of parallel TIPSS in a large UK tertiary referral centre.

Methods A retrospective study was performed from patient electronic databases. Parallel TIPSS were performed over a 19 year period.

Results 11 patients (8M:3F) were identified (2% of all TIPSS procedures). Mean age at time of parallel TIPSS was 48.6(+/-13.7). Background aetiology of portal hypertension included: 5 ALD, 2 PSC, 2 PBC, 1 liver graft failure, 1 NCPH**.** Indications for index TIPSS (5 covered stents) were: 4 Oesophageal variceal (OV) haemorrhage, 3 gastric variceal (GV) haemorrhage, 1 stromal variceal haemorrhage and 3 for refractory ascites. At time of 1st TIPSS, documented mean PPG was 16.6(+/-7.71) and post TIPSS 10.8(+/-7.35) mmHg. Median time between index TIPSS and parallel TIPSS insertion was 72 days (IQR 4–1122 days). Prior to parallel stent placement, 7 patients had dilatation of the index TIPSS.

At parallel TIPSS, the mean initial PPG was 16.0 (+/-7.40)/post procedure 6 (+/-2.28) mmHg. 63% had covered stent as the parallel TIPSS. One patient had transient encephalopathy, but no other complications were encountered. Nine patients had a resolution in symptoms. One patient had ongoing GV bleeding requiring Thrombin injection and 1 patient had ascites with no flow in parallel TIPSS 4 days post-procedure. Secondary patency was 82% with a median number of interventions of 1.5 (IQR 1–3).

Median follow-up was 30 months (range 0.5–120). 92% patients were alive at 1 month with 86% 1 year survival. Two patients were transplanted during follow-up.

Conclusion Parallel TIPSS is a safe and effective method to treat TIPSS insufficiency. The majority of patients not only had a good haemodynamic result, but also resolution of symptoms.

Disclosure of Interest None Declared.

PWE-123 PREDICTORS OF HEPATIC ENCEPHALOPATHY AND MORTALITY FOLLOWING TIPS INSERTION FOR REFRACTORY ASCITES

doi:10.1136/gutjnl-2013-304907.411

[1,*]O M Dissanayake, [1]A Young, [1]M Morgan, [1]J MacNaughtan, [2]D Patch. *[1]UCL Institute for Liver & Digestive Health, Department of Medicine, Royal Free Campus, University College London Medical School; [2]The Royal Free Sheila Sherlock Liver Centre, Royal Free Hospital, London, UK*

Introduction Transjugular intrahepatic portosystemic shunt (TIPS) insertion has been used for over twenty years to treat the complications of portal hypertension. TIPS insertion provides better control of refractory ascites than large volume paracentesis but with a higher risk of developing hepatic encephalopathy (HE). In addition, a survival benefit has only been found in carefully selected patients. The aims of this study were to review the use of TIPS for the treatment of refractory ascites, in a single centre, over a twenty-year period with the aim of identifying factors predictive of the development of HE and survival.

Methods All patients who underwent TIPS for refractory ascites in the Royal Free Hospital, London, between 1992 and 2012 were reviewed. All non-transplanted patients still alive in 2012 were recalled for assessment of their neuropsychiatric status using clinical, neuropsychometric and neurophysiological criteria. The factors associated with the development of post–TIPS HE were determined by multivariate analysis using the Cox proportional regression model. Differences in survival were determined by Kaplan-Meier analysis.

Results Of the 169 patients identified, 96 (56.8%) had died, 22 (13.1%) had been transplanted while the remaining 51 were alive. The median survival time was 18.8 months. The factors predictive of death were a higher serum AST ($p < 0.04$), ALP ($p < 0.02$), and sodium ($p < 0.02$) and an increased INR ($p < 0.01$). Survival rates were higher in patients of non-British white ethnic origin ($p < 0.00$). Of the 27 patients available for review, 21 (78%) had some degree of HE although less than 30% were on anti-encephalopathy treatment. The factors predictive of HE were older age ($p < 0.01$), with the risk increasing by 6.5% for every year of age, and white British ethnicity ($p < 0.01$).

Conclusion Older patients and those of white British origin are particularly at risk for developing HE and should be monitored carefully following the TIPS procedure. The finding that patients with hypernatraemia have significantly reduced survival rates is novel; it most likely reflects overdiuresis and should be corrected pre-procedure. Thus, careful assessment and selection of candidates for TIPS insertion for refractory ascites as well as closer and longer-term monitoring may help prevent the development of HE and lead to improved outcomes.

Disclosure of Interest None Declared.

PWE-124 VAP-1 ACTIVITY IS ELEVATED IN PSC AND MODULATES A4B7-DEPENDENT LYMPHOCYTE ADHESION TO HSEC UNDER FLOW

doi:10.1136/gutjnl-2013-304907.412

[1,*]P J Trivedi, [1]C Weston, [1]C Corbett, [1]E Liaskou, [1]D Adams. *[1]Centre for Liver Research and NIHR Biomedical Research Unit, University of Birmingham, Birmingham, UK*

Introduction Vascular adhesion protein (VAP)-1 is an adhesion molecule which possesses potent amine oxidase activity, and deaminates dietary amines resulting in the production of H_2O_2. Through this function, VAP-1 leads to activation of NFκB in hepatic sinusoidal endothelium (HSEC) resulting in the expression of mucosal-vascular cell-adhesion molecule-1 (MAdCAM-1); a mechanism proposed to contribute to the homing of gut-tropic lymphocytes expressing a4b7 to the liver. Given the putative role this pathway has in hepatic diseases complicating inflammatory bowel disease (IBD), we set out to quantify circulating/soluble (sVAP-1) and intrahepatic VAP-1 enzyme activity in primary sclerosing cholangitis (PSC), and evaluate the functional consequence of its inhibition on MAdCAM-1 dependent lymphocyte recruitment to HSEC.

Methods Total VAP-1 concentration was measured by ELISA. VAP-1 amine oxidase activity was quantified in human serum and explanted liver tissue using the amplex red assay. Flow-based adhesion assays were performed using human HSEC isolated from liver explants, activated with TNFa and methylamine (VAP-1 substrate), and treated with VAP-1 antibody or semicarbazide (VAP-1 enzyme inhibitor). FAC-sorted peripheral blood leucocytes expressing a4b7 were perfused over HSEC under flow rates simulating physiological shear (0.05kPa) and adhesion and transmigration quantified.

Results Patients with PSC had significantly higher circulating median VAP-1 enzyme activity (114.5pmol H_2O_2 produced/min/ml serum, IQR 100.6–134.7) than patients with IBD (60.3, IQR 38.5–73.0; $P = 0.006$), normal controls (84.0, IQR 77.7–105.7; $P = 0.020$) and individuals with PBC (53.9, IQR 33.0–90.9; $P = 0.006$), and trended higher than AIH (77.6, IQR 51.0–124.5; $P = 0.200$) (Mann-Whitney). Total sVAP-1 concentration correlated well with sVAP-1 enzyme activity ($R^2 = 0.75$). Intrahepatic median VAP-1 activity was also significantly higher in PSC (97.6pmol H_2O_2/min/mg protein respectively, IQR 69.5–114.5) vs. PBC (24.6, IQR 18.7–27.8; $P = 0.029$) and AIH (32.3, IQR 23.3–35.6; $P = 0.028$) (Mann-Whitney). HSEC pretreatment with semicarbazide but not antibody led to a profound reduction in total $a4b7^+$ lymphocyte adhesion (75%); however, both antibody and enzyme inhibition independently reduced transmigration by ~50% compared to untreated HSEC.

Conclusion sVAP-1 enzyme activity is greater in PSC compared to IBD alone, normal controls, and other immune-mediated liver diseases. Intrahepatic VAP-1 enzyme activity is significantly higher in PSC compared to AIH and PBC. Inhibition of VAP-1 leads to abrogation of a4b7-mediated adhesion to HSEC, representing a putative target for therapeutic intervention in PSC.

Disclosure of Interest None Declared.

PWE-125 STRATIFICATION OF HEPATOCELLULAR CARCINOMA RISK IN PRIMARY BILIARY CIRRHOSIS BY BIOCHEMICAL RESPONSE TO TREATMENT

doi:10.1136/gutjnl-2013-304907.413

[1,*]P Trivedi, [1]K-K Li, [1]T Bruns, [1]H Shah, [2]D Tripathi, [2]T Shah, [2]J Neuberger, [1]G Hirschfield. *[1]Centre for Liver Research and NIHR Biomedical Research Unit, University of Birmingham; [2]Liver Unit, Queen Elizabeth Hospital, University Hospitals Birmingham, Birmingham, UK*

Introduction Aims: To review patterns of hepatocellular carcinoma (HCC) presentation in patients with primary biliary cirrhosis (PBC) in the context of disease severity and biochemical response to treatment.

Methods Patients with confirmed PBC seen at the Queen Elizabeth hospital (Birmingham) between September 1996 and December 2012 were identified, and a systematic retrospective chart review performed. Potential risk factors for the development of HCC were analysed using Fisher's exact test, multivariate logistic regression and Kaplan-Meier estimates (GraphPadv5.03).

Results Of 397 patients with PBC identified, 221 patients had developed cirrhosis, and 30 presented with or developed HCC (median age 68) over a 16 year period. Of all cases of HCC, 21 (70%) were identified through HCC surveillance programmes, and 19 patients presented within Milan criteria. In our practise, HCC was exclusively seen in the presence of cirrhosis. Using the cirrhotic non-HCC group as our comparator, male cirrhotics were more likely to develop HCC than female cirrhotics (OR 3.32; 95% CI 1.34–8.26; $P = 0.012$), and those with HCC were older at the time of diagnosis of cirrhosis (68 years vs. 57 years; $P = 0.001$). 67% (n = 20) in the HCC group and 76% (n = 145) of the non-HCC cirrhotic group were taking UDCA for > 1 year following diagnosis of PBC ($P = 0.21$). Of the patients taking UDCA for > 1 year, 52% (n = 76) of the non-HCC cirrhotic group were biochemical responders according to the original Corpechot criteria compared with 20% (n = 4) of the HCC group ($P < 0.0001$). Not taking or non-response to UDCA was significantly associated with the development of HCC (OR 4.54; 95% CI 2.24–9.20; $P < 0.0001$), and remained a significant risk factor after restricting the analysis to UDCA non-responders (OR 4.33; 95% CI 2.31–8.12; $P < 0.0001$). The cumulative hazard for HCC in cirrhotic UDCA non-responders was 0.18 at 5 years and 0.35 at 10 years after diagnosis of cirrhosis, but < 0.1 after 10 years in UDCA responders ($P = 0.003$). The overall HCC incidence rate for cirrhotic patients with PBC was 3.4 cases/100 patient-years. When stratified by treatment response, UDCA responders had HCC rates of only 1 case/100 patient years, compared to 5 cases/100 patient-years in non-responders.

Conclusion Development of HCC in PBC is associated with a failure to respond to therapy with UDCA, older age at diagnosis of cirrhosis and male gender. The incidence rate for HCC development in cirrhotic patients responding to UDCA is arguably beneath the point at which cost-efficacy is likely met.

Disclosure of Interest P. Trivedi Grant/Research Support from: Wellcome Trust funded clinical research fellow, K.-K. Li: None Declared, T. Bruns: None Declared, H. Shah: None Declared, D. Tripathi: None Declared, T. Shah: None Declared, J. Neuberger: None Declared, G. Hirschfield: None Declared