Conclusion This quadruple 24 weeks regimen has excelled the RVR, EVR, ETVT over SOC with DAAs over 11%, with SVR 67%. Needs a larger trial for validation

Disclosure of Interest None Declared.

PWE-130 EFFECT OF N ACETYLCYSTEINE (NAC) IN HYPOXIA INDUCED LIVER INJURY (HILI)–A RANDOMIZED PLACEBO CONTROL CLINICAL TRIAL

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[1,*]P Basu, [2]N J Shah, [3]S Farhat, [1]R Siriki, [1]K Mittimanj, [1]M Rahaman, [1]S Atluri. *[1]Forest Hills Hospital, Hofstra North Shore-LIJ School of Medicine; [2]James J Peters VA Medical Center, Mount Sinai School of Medicine, New York; [3]Columbia School of Physicians and Surgeons, NY, New York, United States*

Introduction HILI is common with a prevalence of 10% in US. Transient shift of intra hepatic hemodynamic compromise leads to tissue hypoxia and induces hypoxia induced protein (HIP), heat shock protein 70 (HSP24.70), Endothelial reticular stress (ER) leading to reperfusion injury (RI). Dramatic rise of transaminases, drastic reversal with restoration of perfusion in weeks follows. In cirrhotics HILI requires liver transplantation. This study evaluated spontaneous recovery and salvage in HILI utilising NAC.

Methods Sixty patients (n = 60) with mean arterial pressure (MAP) < 35% and normal LFTs at base line. Group A (n = 28) chronic liver disease (CLD) [alcohol-11/28 (39%), NASH-9/28 (32%), Hepatitis C-4/28 (14%), hepatitis B-2/28 (7%), PBC-1/28 (3%), AIH-1/28 (3%). Group B (n = 32) [respiratory failure-12/32 (37%), CHF-8/32 (25%), CVA-2/32 (6%), sepsis-6/32 (19%), post op-4/32 (12%)]. Randomized into Placebo group- A1 (14) & B1 (16) and IV NAC for 48 hours - A2 (14) & B2 (16). Serum Transaminases, Bilirubin, INR, Creatinine and MELD score at 0, 3rd, 6th, 9th and 12th days with MAP and modified Sequential Organ Failure Assessment (SOFA) Score. All patients were allowed standard of care (SOC) and resuscitations if needed.

Exclusions: Organ transplant, Septic shock, Hemodialysis, cancer, acute myocardial Infarct, Tylenol injury, acute viral hepatitis and organ trauma.

Results Placebo groups A1, B1: Normalized A1-[LFTs- on 3rd day-(7%), 6th day-(21%), 9th day-(36%) and 12th day-(21%). 1/14(7%) died]. B1(CLD)[LFTs 3rd day-(19%) 6th (44%) 9 th (25%),2/16(6%) died of sepsis] NAC Groups A2[normalised LFTs 3rd (57%)6th day-(43%) 9 th day (25%), (7%)-one died] B2 (CLD)[Normalized LFTs-3rd day-(63%), 6th day-(25%) 9 th1/16(6%), one died]

Conclusion This Study postulates that IV NAC (A2, B2) has efficient spontaneous recovery and salvage in non-CLD sub group B2 (63%) > A2(57%) in day 3, in CLD NAC (A2) > placebo (A1) clinical recovery over placebo at 3rd day, (44%) over (36%) - 6th day. Larger trial need to establish the routine usage of IV NAC in HILI.

Disclosure of Interest None Declared.

PWE-131 DOES CAPSULE ENDOSCOPY HAVE A ROLE IN PATIENTS WITH CHRONIC LIVER DISEASE AND OBSCURE GASTROINTESTINAL BLEEDING

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[1,*]P S Sidhu, [1]M E McAlindon, [1]R Sidhu. *[1]Gastroenterology, Royal Hallamshire Hospital, Sheffield Teaching Hospitals, Sheffield, UK*

Introduction Chronic liver disease (CLD) is commonly associated with anaemia. Whilst varices represent the commonest cause of gastrointestinal bleeding in patients with CLD, in patients where iron deficiency anaemia (IDA) persist, capsule endoscopy (CE) may have a useful role to investigate the small bowel (SB). We conducted a study to evaluate the utility of CE in patients with CLD and obscure gastrointestinal bleeding (OGB) and their subsequent management consequences.

Methods We retrospectively analysed our data set and isolated patients with OGB and CLD. Data collected included demographics, clinical indication (overt bleeding (OB) or IDA) the presence of co-morbidity, diagnostic yield (DY) and subsequent follow up.

Results Of the 1324 patients investigated for OGB using CE, 3%(n = 41) had CLD. The mean age was 61 years (range = 26–88) with 59% males. The indications for CE was IDA in 66%(n = 27) and OB in the remaining 34%. All patients in this cohort had other significant co-morbidity in addition to CLD. Five patients were on non-steroidal anti-inflammatories whilst 2 patients were transfusion dependent. The DY (as defined by lesions responsible for OGB) identified on CE was 49%(n = 20).The commonest finding was SB ulcer and erosions 27%(n = 11) and SB angioectasia (AE) 24%(n = 10). Other findings included SB varices (2), blood without definite source (5), a tumour (metastatic renal tumour) and other miscellaneous lesions (4). In 13 patients (32%), lesions found were within the upper GI tract, which had been underestimated at the index gastroscopy. These included gastric antral vascular ectasia (3), varices (oesophageal and duodenal)(2), blood without definite source (5) and others lesions (erosions, ulcers, portal hypertension and polyps)(9). In 2 patients, colonic lesions were identified (erosions and AE). There was no significant difference in the DY between those with IDA and OB (p = 0.59) and between the sexes (p = 0.41). In our cohort, management was altered in 90%(n = 18) of those with a DY, in the form of further procedures (25%, n = 5) which included repeat OGD (2), colonoscopy (2), double balloon enteroscopy (1) and the patients with renal metastasis avoided surgery. 25%(n = 5) of patients within this cohort also received argon photocoagulation therapy. On logistic regression, factors that were associated with a subsequent change in management included previous transfusions (p = 0.04) and SB AE (p = 0.03).

Conclusion CE is a useful tool for investigation of OGB in patients with chronic liver disease and persistent anaemia. Ulcers and AE were the commonest pathology seen in the SB in patients with CLD, in keeping with the published literature. CE is also useful to pick up pathology in the upper GI tract which may have been underestimated.

Disclosure of Interest None Declared.

PWE-132 THE PREVALENCE AND CLINICAL SIGNIFICANCE OF CRYOGLOBULINAEMIA IN A SCOTTISH COHORT OF HEPATITIS C PATIENTS

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[1,*]R E Swann, [1]M Robinson, [2]S Barclay, [2]M Priest, [2]P Mills, [1]J McLauchlan, [1]A Patel. *[1]MRC University of Glasgow Centre for Virus Research, University of Glasgow; [2]Gastroenterology, NHS Greater Glasgow and Clyde, Glasgow, UK*

Introduction Hepatitis C virus (HCV) infection is the most common cause of cryoglobulinaemia – a clonal B cell disorder characterised by precipitation of antibody aggregates on serum cooling. This can lead to vasculitic symptoms and complications including renal failure [1]. One meta-analysis suggested a prevalence of 44% in HCV infected patients. However, other studies have reported much lower rates [2]. Genotype is thought to influence prevalence, and data from the UK, where 45% of cases are genotype 3, is unknown. This study aimed to determine the prevalence of cryoglobulinaemia in a cohort of HCV infected patients and identify any associated clinical features.

Methods 75 patients with chronic G1 or G3 HCV were prospectively recruited from liver clinics in addition to 20 healthy controls. None had a prior diagnosis of cryoglobulinaemia. Each patient completed a symptom questionnaire and clinical and laboratory details were recorded. A whole blood sample was collected and maintained at 37°C until serum had been separated using a heated centrifuge. Serum was stored at 4°C for 7 days. A patient was recorded as

cryoglobulin positive if a precipitate formed which disappeared on re-warming. Clinical features were correlated with presence of cryoglobulin.

Results Adequate samples were received in 65/75 HCV infected patients (31 G1, 34 G3). Of these, 35.4% (23/65) had detectable cryoglobulin. No cryoglobulin was detected in the healthy control samples. Clinical associations are listed below (p values from Fisher's Exact Test unless otherwise stated).

Abstract PWE-132 Table

Clinical Parameter	Cryoglobulin Positive (n = 23)	Cryoglobulin Negative (n = 42)	P value
Male (%)	13 (57)	34 (81)	0.04
Age (mean yrs)	44.6	45.8	0.73*
Genotype 3 (%)	15 (65)	19 (45)	0.10
Cirrhosis (%)	13 (57)	10 (24)	0.09
Renal Function (mean Cr)	67	72	0.17*
Viral load (mean IU/ml)	$5.2x10^5$	$4.6x10^5$	0.23*
Any symptoms (%) (excluding fatigue)	12 (52)	16 (38)	0.30~
Fatigue (median score/10)	5	5	0.75

*t test
~Wilcoxon rank sum

There was no difference in prevalence of IVDU or Diabetes in those with cryoglobulins. No individual symptom was associated with cryoglobulin detection.

Conclusion Cryoglobulinaemia has a surprisingly high prevalence of 35% within our UK based cohort of HCV patients, being less common in males. Symptoms are non-specific and occur in the absence of detectable cryoglobulin with no association between symptoms and cryoglobulin positivity. There was a non-significant trend to association with cirrhosis and genotype 3 as shown in previous studies.

Cryoglobulinaemia may have been underdiagnosed previously due to practical difficulties with testing and it should be considered in any patients with renal dysfunction and HCV.

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PWE-133 OESOPHAGEAL VARICES SCREENING - ARE WE MEETING THE GUIDELINES?

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[1,*]R Bevan, [1]C Parker, [1]K George, [1]S Panter. *[1]Gastroenterology, South Tyneside District General, South Shields, UK*

Introduction Oesophageal varices develop and enlarge in cirrhotic patients at a rate of 8% per year and haemorrhage occurs at a rate of 5–15% per annum, causing significant morbidity and mortality. Of the 16,000 deaths/year attributed to cirrhosis, 33% are due to variceal bleeding. Despite improvements in therapy and prophylaxis the mortality from bleeding varices has remained static. Current BSG guidelines recommend screening with OGD at diagnosis and then 1–3 yearly depending on endoscopic findings[1].

Methods A retrospective review of clinic letters for all gastroenterologists at STDH from Aug-Oct 2012 was performed. Those eligible for variceal screening i.e. established cirrhosis, decompensated liver disease or evidence of portal hypertension on imaging were identified. Demographic details and liver disease aetiology were recorded. The endoscopy reporting system was reviewed to identify OGDs performed within the last 3 years and the indication for OGD. If an OGD report was absent, the appointment system and case notes were reviewed to establish if the patient refused or failed to attend (FTA) for OGD. Where no evidence of FTA or refusal were found, clinician failure to refer was documented.

Results 84 eligible patients were identified, 64 (76.2%) had an OGD recorded within the last 3 years.

Table 1 shows results according to liver disease aetiology. In the group with a NAFLD/NASH or "other" aetiology, all 16 cases who had not had OGD had not been referred. Of the alcohol aetiology, 7/16 had not been referred for OGD, and 9/16 FTA or were documented to have refused the test.

Abstract PWE-133 Table 1

Aetiology	Number	OGD within 3 years (%)	Indication (%)		
			Screening/ surveillance	Bleeding	Other
Alcohol	67	51 (76.1)	37 (55.2)	9 (13.4)	5 (7.5)
NAFLD/NASH	8	5 (62.5)	3 (37.5)	1 (12.5)	1 (12.5)
Other*	8	7 (87.5)	5 (62.5)	1 (12.5)	1 (12.5)

*autoimmune hepatitis, primary biliary cirrhosis, hepatitis C, cryptogenic cirrhosis, and unknown or still under investigation

Conclusion Three quarters of patients eligible for varices screening have had an OGD within the maximum time frame suggested by the BSG guidelines. However, excluding OGDs performed for acute bleeding or other indications only 45/84 (56.6%) have been appropriately screened.

The majority of cases in this audit are secondary to alcoholic liver disease and their high FTA rate reiterates the known difficulties in engaging this group of patients, although numbers are small. This audit suggests a need to improve rates of screening for oesophageal varices; the main reasons suggested by this audit that could be targeted to improve screening rates are appropriate referral by clinicians and reluctance to attend for the test particularly in liver disease secondary to alcohol.

Disclosure of Interest None Declared.

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PWE-134 OUTCOMES FOR LIVER DISEASE PATIENTS ADMITTED TO A DISTRICT GENERAL HOSPITAL

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[1,*]S Paranahewa, [1]L Allen, [1]C Toms, [2]A Lalloo, [1]V Kulhalli, [1]M Guinane, [1]A Rochford, [1]A Dias. *[1]Gastroenterology, Newham University Hospital, Barts Health; [2]Medicine, Barts and the London School of Medicine and Dentistry, London, UK*

Introduction In the UK, liver disease is the 5th commonest cause of death. A recent BSG commissioning report states that there were 43,694 hospital episodes due to liver disease with a mortality rate of 15.5% and median age of death of 59[1]. The guidelines recommend that all liver disease should be managed by a hepatologist. Newham University Hospital (NUH) serves a socially deprived and ethnically diverse population of 290,000.

Methods All patients with a primary diagnosis of liver disease admitted to NUH from 1 April 2012 to 31 October 2012 were included in this study. Patients were identified from the on-call admission lists and electronic patient records and admission notes were checked for suitability. Patients admitted with alcohol withdrawal but without underlying liver disease were excluded. We examined the outcomes of all liver patients admitted during this time period.

Results 78 patients were admitted, of which 9 had ≥2 admissions. The demographic data and outcomes are listed in Table 1. The ethnic variation reflected that of the local community. The main causes of liver disease were alcoholic liver disease (56%), viral hepatitis