

Conclusion This clinical trial postulates decompensated cirrhotics have high evidence of RLS with portal hypertension. Larger trials will validate.

Disclosure of Interest None Declared.

PWE-128 ROMIPLOSTIM'S EFFECT TO OPTIMIZE SVR WITH TELAPRAVIR, RIBAVIRIN, AND PEG INTERFERON-ALFA 2A IN THROMBOCYTOPENIC CIRRHOTICS WITH CHRONIC HEPATITIS C. A PLACEBO CONTROLLED PROSPECTIVE CLINICAL TRIAL: RESTRAINT C TRIAL

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Introduction Treating HCV cirrhotic patients with thrombocytopenia is challenging, often requiring dose reduction/discontinuation to avoid complications. Significant dose reduction affects response guided therapy (RGT); affecting outcomes. Thrombopoietin (TPO) agonists are used to avoid disruption or therapeutic failure to optimise SVR. This study evaluated the use of TPO agonist in thrombocytopenia in cirrhotics with CHC.

Methods Forty five (n = 45) cirrhotic treatment experienced CHC-GT1 patients wererecruited with mean MELD 16, mean platelet count 95. Group A-(n = 15) placebo plus reduced dose of p-IFN with Ribavirin and Telaprevir. Group B (n = 15) Romiplostim 500mcg lead in 1 month prior to initiation of therapy and SOC with Telaprevir. Group C (n = 15) Elthrombopag 50mg orally daily lead in prior 15 days and SOC with Telaprevir for 12 weeks. RGT was analysed with serial platelet counts, haemoglobin/hematocrit, absolute neutrophils count and platelet antibodies. HCV RNA 1ST, 2ND, 4TH, 12TH, 24TH, 36TH and 60TH weeks for SVR.

Results

Conclusion This study demonstrates the efficacy of Romiplostim in thrombocytopenic cirrhotics in optimising SVR (Group A-53%, Group B-67% and Group C-60%). A larger trial is needed to validate.

Disclosure of Interest None Declared.

PWE-129 PEGYLATED INTERFERON ALFA, NITAZOXANIDE, TELAPREVIR, RIBAVIRIN, IN GENOTYPE 1 UNDERGOING PRIOR EXPERIENCED CHC-A RANDOMIZED PLACEBO CONTROL CLINICAL PILOT TRIAL (INTRIGUE-C)

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Introduction Chronic Hepatitis C is a global challenge with End stage liver disease and rising Hepatocellular Carcinoma. Peg Interferon Alfa and Ribavirin was the backbone of therapy. Recently introduced Directly Acting Antivirals (DAAs)-protease inhibitors have escalated Sustained Viral Response (SVR) in Response guided therapy in non responders, partial responders and relapsers. This study utilised NTZ & Telaprevir; with SOC for 24 weeks in treatment experienced patients.

Methods Fifty (n = 50) patients were divided into Group A (n = 12) NTZ 500 mg three times for 12 weeks, Group B (n = 12) NTZ, BID for 12 weeks Group C (n = 26) control. All received Peg Interferon Alfa 2a 180 mcg SQ QOW with fixed dose of Ribavirin 1200 mg daily for 24 weeks and Telaprevir 750 mg three times daily for 12 weeks. Viral load was obtained at day 0, 7th day, 14th day, 4 weeks, 12th, 24 weeks and 48th weeks SVR. Viral kinetics was analysed. In Group A, B and C: 5/12(42%), 5/12(42%), 10/26(38%) Non Responder, 6/12(50%), 6/12 (50%), 4/26(15%) partial responder, and 2/12(16%), 1/12 relapsers (8%), 4/26(15%) relapsers, 2/26(8%) unknown. Use of Growth factors-12% for severe anaemia, 8% for thrombocytopenia and 7% for neutropenia. Skin rash was 29%. Rectalgia was 11%. 3/50(6%) drop out, 2/50(4%) fell in fertility law. Exclusion; Decompensated liver disease. HCC, poor controlled DM, severe CAD, Hemolytic anaemia, Major depression, Renal failure, Prior severe skin rash, active drug and alcohol abuse.

Results

Abstract PWE-129 Table

| | GROUP A | GROUP B | GROUP C |
|--------------|------------|-------------|------------|
| Undetectable | 9/12(75%) | 10/12(83%) | 16/26(62%) |
| NR | 1/12 (8%) | 2/12 (16%) | 4/26 (15%) |
| PR | 1/12 (8%) | 12/12(100%) | 3/26 (11%) |
| AVR | 11/12(92%) | 12/12(100%) | 20/26(77%) |
| VRVR | 11/12(92%) | 10/12(83%) | 22/26(84%) |
| RVR | 9/12(75%) | 10/12(83%) | 18/26(70%) |
| EVR | 9/12(75%) | 10/12(83%) | 16/26(62%) |
| ETVR | 9/12(75%) | 10/12(83%) | 16/26(62%) |
| SVR | 8/12(67%) | 8/12(67%) | 15/26(58%) |

Abstract PWE-128 Table

| | AVR 1 week | VRVR 2 weeks | RVR 4 weeks | EVR 12 weeks | MTVR 24 weeks | ETVR 36 weeks | ETVR 48 weeks | SVR 60 weeks | SVR 72 weeks |
|---|----------------|-----------------|----------------|-----------------|------------------------|-----------------------|-----------------------|-----------------|-----------------|
| Group A R=7 PR= 8 BT=0 PLT 90K | 5/15 (33%) | 7/15 (49%) | 9/15 (60%) | 10/15 (67%) | 10/15 (67%) | | 9/15 (60%) | | 8/15 (53%) |
| | | | 112K | 101K | 93K | 98K BT1/15(7%) | 102K | R1/15 (7%) | 84K |
| Group B R=8 PR= 6 BT=1 PLT 68K | 9/15 (60%) | 10/15 (66%) | 11/15 (77%) | 12/15 (80%) | ETVR 12/15 (80%) | | SVR 11/15 (77%) | | |
| | | | 210K | 90K | 96K | R1/15(7%) PLT 220K | 180K | | 58K |
| Group C R=7, PR= 6 BT=2 PLT 128K | 7/15 (47%) | 8/15 (53%) | 9/15 (60%) | 9/15 (60%) | | 10/15 (67%) | | 9/15 (60%) | |
| | | | 101K | 102K | 90K | 80K | R 1/15(7%) PLT108K | | 131K |

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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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%) 9th (25%), 2/16(6%) died of sepsis] NAC Groups A2[normalised LFTs 3rd (57%) 6th day-(43%) 9th day (25%), (7%)-one died] B2 (CLD)[Normalized LFTs- 3rd day-(63%), 6th day-(25%) 9th 1/16(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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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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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