Romiprostim, a fusion protein TPO, is a hormone that regulates platelet production approved in idiopathic thrombocytopenic purpura (ITP). This study evaluates single use of Romiprostim 2 week prior to liver biopsy to avoid biopsy related morbidity and mortality.

**Methods** 65 patients (n=65), (mean age: 56 years; M:F-2:1) with Hepatitis C: 37/65 (57%); hepatitis B (HBV) 7 (15.5%), Alcoholic Cirrhosis 10 (15%); Non-Alcoholic steato-hepatitis (NASH) 3 (5%), Primary biliary cirrhosis (PBC) 6 (9%) with pre-biopsy mean platelet count 77k; Mean MELD score 20, mode 16.3
diabetes mellitus (T2D) 14 (22%).

**Primary endpoint** Post-biopsy in 4 weeks in all groups. Inclusion criteria: CLD with thrombocytopaenia.

**Conclusion** This cohort of cirrhotic patients displayed evidence of advanced liver disease. Median UKELD in ward patients was above the limit required for consideration of liver transplant and median DF indicated a high risk of death in those diagnosed with AH. Prolonged hospital stays and ward based convalescence were required, especially in those escalated to HDU, but mortality was lower than that commonly perceived for this patient cohort. Patients who are not offered advanced organ support are identified after gradual deterioration despite optimal ward care. We propose the concept of a “medical HDU” model where hepatologists are able to escalate levels of care locally before the onset of organ dysfunction.

**Competing interests** None declared.
jaundice to encephalopathy (<7 days), 8 Subacute/Acute Liver Failure (S/ALF: jaundice to encephalopathy >7 days), and 13 healthy controls (HC). Flow cytometry was performed to investigate monocyte phenotype (CD14, CD16, HLA-DR, CD86, CD163, CD11b and CCR5).

**Results** Compared to HC, total monocyte count was elevated in S/ALF, but reduced in hyperALF (p<0.001), while CD14hi/CD16+ monocytes were expanded in percentage of total monocytes and absolute numbers in S/ALF (17.4%; 0.14) compared to HC (4%; 0.014) (p<0.001). Although the percentage of CD14hi/CD16+ monocytes in hyperALF was higher (5.6%, p<0.01), the absolute number (0.015) was similar to HC. Though all CD14hi/CD16+ monocytes expressed HLA-DR, the Mean Fluorescence Intensity (MFI) was reduced compared to HC (<0.001). HyperALF CD14hi/CD16+ monocytes had lower HLA-DR MFI compared to S/ALF (p<0.001). A similar pattern was seen for CD86 expression (p<0.01). CD14hi/CD16+ monocytes showed increased expression of CD163 in hyperALF but not in S/ALF compared to HC (<0.01). Compared to HC, CD11b and CCR5 were up-regulated in all ALF groups (p<0.001).

**Conclusion** We have demonstrated an expansion of CD14hi/CD16+ monocytes in ALF with an activated phenotype for adhesion and migration. CD14hi/CD16+ monocytes phenotypically resemble M1 macrophages in S/ALF possibly reflecting a pathogenic role in the perpetuation of liver injury, while they resemble M2 in hyperALF and may be instrumental to the resolution of liver injury. This distinction requires further investigation should therapeutic strategies to target monocyte migration be attempted.

**Competing interests** None declared.

**PTU-016**

**ALBUMIN RESTORES RENAL BLOOD FLOW (RBF) AUTOREGULATION IN PATIENTS WITH REFRACTORY ASCITES AND ACUTE-ON-CHRONIC LIVER FAILURE (ACLF) THROUGH STABILISATION OF ENDOTHELIAL FUNCTION**

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**Introduction** Haemodynamic alterations in liver failure are associated with endothelial dysfunction, a pro-inflammatory state and sympathetic activation which lead to disturbed RBF autoregulation and renal failure. Albumin is a multifunctional protein that has been shown in several studies to prevent and treat renal dysfunction in patients with advanced cirrhosis and liver failure. We hypothesised that the beneficial effects of albumin in cirrhosis is likely to be through mechanisms in addition to volume expansion. The aims of the study were to investigate the effects of albumin on systemic and renal haemodynamics, inflammation and endothelial dysfunction in refractory ascites and patients with acute kidney injury (AKI) in the setting of ACLF.

**Methods** Twenty-two patients were recruited [Group 1, n=12, refractory ascites; Group 2, n=10 patients with AKI] admitted with an acute deterioration of their liver function due to either alcoholic hepatitis or infection]. Both groups were treated with Albumin 60 g/d over 3–4 days. Cardiac output (CO) and renal blood flow (RBF) haemodynamics were measured. Endothelial dysfunction was assessed through measurement of von Willebrand factor (vWF) and serum nitrite (NO) levels. F2α Isoprostanes (F2α) and neutrophil burst and Interleukin (IL)-6 were quantified as markers of oxidative stress, endothoxemia and inflammation respectively.

**Results** Albumin therapy was associated with significant improvements in haemodynamic parameters (increased RBF, MAP, decreased CO, HR; p<0.05) which resulted in a shift in the RBF autoregulation curve towards normalisation (Abstract PTU-016 figure 1). In parallel, improvement of renal dysfunction (creatinine, creatinine clearance and Na+ excretion; p<0.05 each), sympathetic activation (noradrenaline levels; p<0.01), inflammation/oxidative stress (F2α and neutrophil burst; p<0.05), endothelial dysfunction (vWF and NO metabolism p<0.05) and the functional capacity of albumin (IMAR, p<0.005) was observed. Restoration of RBF correlated inversely with change in vWF (r2=0.55, p<0.001).

**PTU-016a**

**FUNCTIONAL DEFECTS IN CIRCULATING MONOCYTES MAY CONTRIBUTE TO SUSCEPTIBILITY TO INFECTION IN ALCOHOLIC HEPATITIS**

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**Introduction** Infection is common in patients with severe alcoholic hepatitis (AH) and a significant contributor to mortality. Monocytes play an important role in bacterial elimination by phagocytosis, using intracellular oxidative killing and antigen presentation. Our study sought to evaluate monocyte phagocytosis and scavenger receptor expression in AH.

**Methods** Monocytes were collected from 14 patients with AH (DF >31, prior to treatment) and 22 healthy controls (HC). Using FACS, monoclonal antibodies to scavenger receptors (CD36, -64, -165, -206, DCIR) and HLA-DR were used for immunophenotyping. Subsequently, ex-vivo monocyte phagocytosis and oxidative burst activity was assessed using FITC-labelled opsonised and non-opsonised Escherichia coli.

**Results** The expression of scavenger receptors was deranged. In CD14+CD16- (classical) monocytes, CD163 MH was reduced in AH compared to controls (SS7 vs 403; p=0.05). CD36, -206 and DCIR expression was similar between HC and AH patients but CD64 MFI was raised (6002 vs 12599; p<0.001). The proportion of monocytes phagocytosing E.coli was lower in AH compared to HC.