	BL PLT ($\times 10^9/l$)	PreOp PLT ($\times 10^9/l$)	4 wk PostOp ($\times 10^9/l$)	Mean Chg BL to PreOp ($\times 10^9/l$)	Mean Chg PreOp to 4 wk PostOp ($\times 10^9/l$)	Mean Chg BL to 4 wk PostOp ($\times 10^9/l$)	Cost (WAC)
PLT TRF	56.9*	183.8	85.9	126.8	-97.9	28.9	\$7500
Romiplostim	58.7*	232†	366.2‡	173.3†	192.9‡	307.5‡	\$2284
Elthrombopag	54.5*	189.9*	173.6§	135.4*	-16.3§	119.1§	\$2991

*p=ND vs PLT.

†p<0.05 vs PLT TRF and Elthrombopag.

‡p<0.001 vs PLT TRF and Elthrombopag.

§p<0.001 vs PLT TRF.

Romiplostim, a fusion protein TPO, is a hormone that regulates platelet production approved in idiopathic thrombocytopenic purpura (ITP). This study evaluates single use of Romiplostim 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 fibrotic score F4 were randomised in blinded fashion into three groups: Group A (n=18), received seven units of platelet transfusion at night for the morning procedure. Group B (n=23) received Romiplostim 500 µg sc given 2 weeks prior to the procedure, and Group C (n=24) Elthrombopag orally 75 mg/day for 2 weeks. PLT-CT was repeated 2 h prior and Post-biopsy in 4 weeks in all groups. Inclusion criteria: CLD with thrombocytopenia.

Results

Conclusion This pilot study demonstrates that single use of Romiplostim is efficacious, cost-effective, and safe without side effects for liver biopsy with severe thrombocytopenia. Single use of Romiplostim should be considered before Trans jugular intra-hepatic porto-systemic shunts or portal haemodynamic procedures and prior to surgical interventions with severe thrombocytopenia. A large randomised clinical trial is needed for further validation.

Competing interests None declared.

PTU-014 LEVELS OF CARE AND OUTCOMES IN DECOMPENSATED CIRRHOSIS: A DESCRIPTIVE STUDY

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Introduction As hospital admissions due to decompensated cirrhosis increase it is important to characterise the severity of disease and establish “best practice” in secondary care units. While many studies have reported on patients in the ITU setting, we have expanded this evaluation to explore patient outcome according to level of in-patient care provided; ward (level 0–1), medical HDU (2) or ICU (3).

Methods Prospective analysis of consecutive decompensated cirrhosis patients admitted to the liver service of a district general hospital between November 2010 and November 2011).

Results 66 patients accounted for 153 separate presentations. ALD accounted for 54 patients (78%). Of those with ALD 24 (44%) were a first presentation, 22 (41%) continued to drink against advice and eight were abstinent. Alcoholic hepatitis (AH) was diagnosed in 24, with median discriminant function (DF) of 49. 16 (23%) were admitted to ICU (multiple organ failure 12, variceal bleeding without organ failure 4). 88% were mechanically ventilated, 81%

received inotropic support and 56% haemofiltration. 14 (20%) were admitted to medical HDU and the 36 (61%) remained on the ward. Median MELD and UKELD scores were not significantly different between groups; ITU 16 and 57, HDU 20 and 58, Ward 13 and 55 respectively. Overall median length of stay was 11.5 days. LOS was highest in HDU patients (23 d vs ward 11 d and ITU 17 d, p=0.04). In-hospital mortality was 5% in ward patients, 14% in HDU patients and 36% in ITU patients (p=0.01), with 90-day mortality rising to 8%, 21% and 50% respectively. Six patients with extra-hepatic organ dysfunction received a “ceiling of care” decision, whereby active treatment on HDU was offered but organ support on ICU was not; these patients did not differ from ICU patients in terms of age, active drinking, liver failure scores or SOFA score. The natural history of these patients was characterised by rising MELD despite optimum therapy and late development of extra-hepatic organ dysfunction (median period from admission 17 d vs 1 d, p=NS). All six died.

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.

PTU-015 EXPANDED CD14HI/CD16+ MONOCYTES IN ACUTE LIVER INJURY (ALI): ANGELS OR DEMONS?

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Introduction CD14hi/CD16+ monocytes resemble tissue macrophages and normally constitute ~5% of circulating monocytes. Expansion of CD14hi/CD16+ monocytes has been implicated in the perpetuation of liver injury and hepatic fibrosis in chronic liver disease. We therefore wanted to explore whether CD14hi/CD16+ monocytes are implicated in ALI; show features of activated endothelial adhesion (CD11b) and chemotaxis (CCR5); and phenotypically resemble classically activated “M1” or alternatively activated “M2” macrophages. “M1” macrophages secrete pro-inflammatory cytokines, express high levels of HLA-DR and CD86 and are implicated in tissue damage. “M2” macrophages, are immunoregulatory, express scavenger receptors such as CD163 and are involved in tissue repair.

Methods Blood was sampled from patients within 24–48 h of admission consisting of 33 hyper-Acute Liver Failure (hyperALF: