n=20; no SIRS, 21.6 (18.6–43.1) nmol/l, n=13, p<0.0001), and in PODs who died/required emergency liver transplantation (OLT 72.9 (68.1–119.2) nmol/l, n=14; survived 30.5 (20.7–59.1) nmol/l, n=29, p=0.006; AUROC 78.6% (95% CI 62.2% to 94.9%). An admission neopterin value of 50 nmol/l predicted death/OLT with a sensitivity of 100.0% (95% CI 76.3 to 100.0) and specificity of 63.2% (95% CI 38.4 to 83.7). Admission neopterin levels in PODs correlated with: serum creatinine (Spearman’s r=0.847, p<0.0001); prothrombin time (r=0.495, p=0.011); platelet count (r=-0.463, p=0.007); IL-6 (r=0.650, p=0.0001); IL-10 (r=0.529, p=0.004); serum ferritin (r=0.467, p=0.006); and with organ failure scores (SOFA, r=0.725, p<0.0001; APACHE II, r=0.659, p<0.0001).

Conclusion Serum neopterin levels are significantly elevated following POD and are correlated with adverse outcomes. Serum neopterin may have value as an early marker of prognosis in ALF and should be assessed further with larger prospective cohorts of ALF patients. The correlation of serum neopterin with adverse outcomes provides further support for the importance of macrophage activation in the pathogenesis of multiorgan failure in ALF.

Competing interests None declared.

REFERENCE

PTU-007 CEREBRAL OEDEMA IS RARE IN ACUTE-ON-CHRONIC LIVER FAILURE doi:10.1136/gutjnl-2012-302514c.7

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Introduction Acute-on-chronic liver failure (AoCLF) has a rapidly progressive disease course associated with significant mortality. Hepatic encephalopathy (HE) is common associated with hyper-ammonemia, systemic inflammation and hyponatraemia. The prevalence of cerebral oedema in AoCLF is unknown. We aimed to describe the prevalence of cerebral oedema in a cohort of AoCLF adult (>18 years) patients admitted to the liver intensive treatment unit (LITU) between January 2005 and 2011.

Methods AoCLF was defined using criteria of Sarin et al. Arterial ammonia (NH3), MELD, UKELD, and organ failure (SOFA) scores were collated (results expressed as medians with ranges). Patients who had undergone cranial CT imaging were identified. Neuro-images were reported by consultant neuro-radiologists.

Results During the study period, 1008 patients with chronic liver disease (CLD) were admitted to the LITU. 173 patients (110 male) underwent neuro-imaging. Of these 81 (48 male) fulfilled criteria for AoCLF. Over the same time period 655 patients were admitted with acute liver failure. Variecal bleeding (50%) and sepsis (31%) were the most frequent precipitants of AoCLF. Compared to the CLD group, AoCLF patients were younger (50, 24–71 vs 59, 30–74, p=0.001), serum NH3 (143, 40–305 vs 111, 28–315), grade of HE (3, 1–4 vs 1, 0–4), MELD (25, 8–40 vs 15, 6–34), SOFA (11, 2–17 vs 4, 0–14), UKELD (63, 50–75 vs 55, 44–75) and SIRS score (2, 1–3 vs 1, 0–3) were higher (p<0.0001 for all). Serum sodium was lower in the AoCLF group (132 118–154 vs 116 120–146, p<0.0001). HE (≥grade 3) occurred in 66% of AoCLF patients vs 13% CLD (p<0.0001). In those with neuro-imaging, 26% were normal, 26% demonstrated increased cerebral atrophy for age, 15% small vessel disease and 10% intra-cranial haemorrhage. Cerebral oedema was seen in two patients with AoCLF, 1 post TIPSS (NH3 289 μmol/l) and 1 with septic shock (NH3 268 μmol/l). 72 patients with CLD underwent neuro-imaging with 52% showing radiological evidence of cerebral oedema. Compared to the CLD group, 30 and 90 day survival was poorer in AoCLF (52% and 42% vs 80% and 75%, log rank p<0.0001). The mode of death was that of progressive multi-organ failure (MOF). The two patients with cerebral oedema on CT suffered cerebral deaths with tonsillar herniation. AUROC analysis for survival of AoCLF identified SOFA (0.67, 95% CI 0.54 to 0.8, p=0.02) MELD (0.74, 95% CI 0.61 to 0.87, p<0.0001) and UKELD (0.87, 95% CI 0.8 to 0.97, p<0.0001).

Conclusion Our data demonstrates poor outcome in patients with AoCLF compared to those with CLD requiring admission to LITU. Mortality was attributable to MOF and although deep levels of encephalopathy requiring ventilation were common (66%), the prevalence of cerebral oedema was rare at 2%.

Competing interests None declared.

REFERENCE