Posters

animals. Correspondingly, metabolic profiles from both HCC groups with and without norfloxin were similar in character with the norfloxin treated group showing a slightly weaker set of metabolic alterations

Conclusion The spectral profiles of plasma in rats with HCC display marked changes with relation to lipid metabolism and cellular turnover which may indicate a fundamental repression of fatty acid oxidation and cancer cachexia. Norfloxacin appears to abrogate these effects slightly. This is the first animal model plasma ¹H NMR study to report such findings and may both be translational to human disease and allow the study of the effect metabolic modulation upon HCC progression.

Competing interests None declared.

PWE-266 | PROTECTIVE ROLE OF β BLOCKERS IN SBP: MYTH OR

doi:10.1136/gutjnl-2012-302514d.266

¹M Mansoor,* ²H Sherfi, ³Z Rahman, ²J Ramesh. ¹Royal Hallamshire Hospital, Sheffield, UK; ²Wythenshawe Hospital, Manchester, UK; ³Manchester Royal Infirmary, Manchester, UK

Introduction β Blockers may have a protective effect on the development of spontaneous bacterial peritonitis by increasing the intestinal transit time and reducing portal pressure. The aim of this study is to evaluate the significant benefit of β blocker in prevention of spontaneous bacterial peritonitis in patients with chronic liver disease and ascites.

Methods We retrospectively evaluated 332 patients with cirrhosis and ascites admitted over a period of 5 years (males 230, females 102). Diagnosis of spontaneous bacterial peritonitis was based on an ascitic fluid neutrophilic count of >250/mm³ and/or a positive culture without evidence of secondary peritonitis.

Results Spontaneous bacterial peritonitis was diagnosed in 52 of 332 (15.66%) patients. Of the 92 on β-blockers, 6 (6.5%) had SBP and out of 240 patients who were not on β -blockers, 46 (19.2%) had SBP. The patients who were on β -blockers, had a significantly lower incidence of SBP (χ^2 test with continuity correction; p=0.008).

Conclusion Our data indicate that spontaneous bacterial peritonitis significantly increases mortality in patients with cirrhosis. Propranolol therapy was found to be associated with a significantly lower risk for spontaneous bacterial peritonitis, but a Type II statistical error cannot be definitely excluded. The potential protective effect of propranolol on the incidence of spontaneous bacterial peritonitis might deserve evaluation in properly designed prospective studies.

Competing interests None declared.

REFERENCE

Senzolo M. Cholongitas E. Burra P. et al. B-Blockers protect against spontaneous bacterial peritonitis in cirrhotic patients: a meta-analysis. Liver Int 2009; 29:1189-93.

PWE-267

URINARY TLR4: A NOVEL BIOMARKER TO IDENTIFY PATIENTS WITH ACUTE KIDNEY INJURY IN PATIENTS WITH ACUTE ON CHRONIC LIVER FAILURE

doi:10.1136/gutjnl-2012-302514d.267

¹N Shah,* ¹F Mohammed, ¹M Jover-Cobos, ¹J Macnaughtan, ¹N A Davies, ²R Moreau, ²V Paradis, ¹K Moore, ¹R P Mookerjee, ¹R Jalan. ¹Department of Hepatology, UCL Institute of Hepatology, London, UK; ²INSERM, Clichy, France

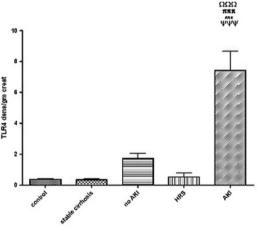
Introduction Patients with stable cirrhosis often present with acute deterioration of cirrhosis secondary to precipitating illness which may progress to organ failure, a condition referred to as acute on chronic Liver failure (ACLF). A proportion of these patients develop renal dysfunction which do not fulfil criteria for the diagnosis of hepatorenal

syndrome (HRS). We hypothesised that the kidneys in patients who develop renal dysfunction in ACLF would exhibit histological and biomarker evidence of acute kidney injury (AKI). Since ACLF is associated with systemic inflammatory response (SIRS) we aimed to look for Toll like receptor (TLR) 4 and 2 which recognise pathogens and when activated lead to apoptosis and production of cytokines.

Methods Study 1: 74 subjects [healthy volunteers (6), compensated alcoholic cirrhosis (11), acute deterioration of alcoholic cirrhosis (57)] were included prospectively. Urinary biomarkers, kidney injury molecule-1 (KIM-1, a marker of renal injury), Glutathione S-Transferase (π GST, α GST; markers of proximal and distal tubular injury) (Commercial ELISA), and urinary TLR4 (Western Blotting) were measured. Study 2: Renal biopsies were available from 8 alcoholic cirrhosis patients (3 AKI; 5 HRS) which were stained for TLR4, TLR2 and, Caspase-3.

Results Study 1: Nine patients developed AKI on the background of acute deterioration of cirrhosis and 3 had HRS. KIM-1, πGST and αGST were higher in patients with acute deterioration of cirrhosis compared with controls but did not differ in those with and without AKI. Urinary TLR4 values were significantly higher in patients with acute deterioration of cirrhosis with AKI (4.7±1.1) compared to controls (0.38 ± 0.04) and stable cirrhosis (0.32 ± 0.08) and patients with acute deterioration of cirrhosis without renal dysfunction (1.6 ± 0.32) (p<0.01) respectively.

Conclusion These data provide evidence for severe tubular injury and apoptosis in patients with AKI and identifies urinary TLR4, as a novel biomarker to identify AKI in Acute deterioration of cirrhosis.



- *: control vs acute deterioration of cirrhosis with renal dysfunction
- α: stable cirrhosis vs acute deterioration of cirrhosis with renal dysfunction
- Ω : HRS vs acute deterioration of cirrhosis with renal dysfunction ecute deterioration of cirrhosis without renal dysfunction vs acute deterioration of dirrhosis with renal dysfunction.

Abstract PWE-267 Figure 1

Competing interests None declared.

PWE-268

RECENT TRENDS IN PRIMARY LIVER CANCER IN **ENGLAND AND WALES**

doi:10.1136/gutjnl-2012-302514d.268

¹N G Ladep,* ¹S A Khan, ¹A V Thillainayagam, ¹S D Taylor-Robinson, ²M B Toledano. ¹Department of Hepatology, Imperial College, London, UK; ²Department of Epidemiology and Biostatistics, Imperial College, London, UK

Introduction Mortality and incidence rates of Primary Liver Cancer (PLC) have been rising in England and Wales towards the end of the last century. The current trend and ethnic distribution of PLC remain unknown.

Methods We obtained mortality and incidence data for PLC for the whole population of England and Wales for the period 2001-2008.

A406