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 ^{1}H 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 REALITY

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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/mm^3 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.

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PWE-267 URINARY TLR4: A NOVEL BIOMARKER TO IDENTIFY PATIENTS WITH ACUTE KIDNEY INJURY IN PATIENTS WITH ACUTE ON CHRONIC LIVER FAILURE

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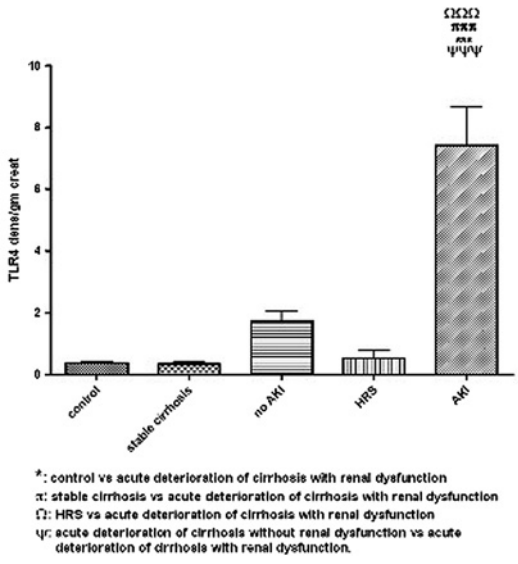
Introduction Patients with stable cirrhosis often present with acute deterioration of cirrhosis secondary to precipitating illness which may progress to organ failure, a condition referred to as acute on chronic Liver failure (ACLF). A proportion of these patients develop renal dysfunction which do not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



Abstract PWE-267 Figure 1

Competing interests None declared.

PWE-268 RECENT TRENDS IN PRIMARY LIVER CANCER IN ENGLAND AND WALES

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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.

Age-standardised/specific mortality and incidence rates (ASMR/ASpMR and ASIR/ASpIR) of PLC subcategories were calculated. Trends in the rates of hepatocellular carcinoma (HCC) and intrahepatic bile duct tumours (IHBD) were evaluated using a regression method in which a least squares regression line was fitted to the natural logarithm of the rates. About 30% of incidence data for PLC included information on the ethnic origin of the cases and thus we were able to analyse the ethnic distribution of HCC and IHBD for this sub-set over the study period.

Results The ASMR for PLC increased in both sexes: from 3.88 and 2.03 per 100 000 in 2001 to 5.10 and 2.63 per 100 000 in 2008, for males and females respectively. Specifically, there was an increase in the ASMR for both HCC and IHBD of between 4% and 7% per year over the study period. ASIR of HCC increased in men (annual % change: 4%) but not in women. In the ethnic sub-set analysis, more than 79% of HCC and IHBD were registered in men and women of white ethnicity. Black Caribbeans and Africans, as well as Indians were the next most affected ethnic populations.

Conclusion Mortality and incidence rates of PLC continue to increase in England and Wales during 2001–2008, with a modest contribution from immigrant ethnic populations to the increasing trend.

Competing interests None declared.

PWE-269 HEAT SHOCK PROTEIN-70 (HSP-70) A NOVEL SURROGATE MARKER FOR HYPOXIA INDUCED LIVER INJURY (HILI)—A PROSPECTIVE OPEN LEVEL CONTROL CLINICAL PILOT STUDY

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Introduction Hypoxic hepatopathy (HH) is a common entity in hospital with impaired hepatic perfusion secondary to transient altered cardio pulmonary haemodynamics, manifesting with sharp rise of transaminases with perfusion injury rarely requiring transplantation. Transaminases are non-specific index of reperfusion injury (IR). HSP are reperfusion signal proteins represent sheer stress. HSP-70 is one of specific signals of IR impacts on specific target. This study evaluates the utility of a novel marker of HILI.

Methods Sixty (n=60) patients were recruited from Hospital. Group A control (n=20) Group B (n=20) HILI, GroupC (n=20) Acute Hepatitis [(Tylenol 8/20 (40%), Acute hepatitis B 8/20 (40%) herpes simplex 1/20 (5%) EBV 1/20 (5%) acute hepatitis A 1/20 (5%) unknown 1/20 (5%)]. All groups underwent serial blood levels of HSP70, Liver and renal functions, MELD score, SOFA score, from day 0, 4, 7 and 10. Exclusion; Cardio respiratory failure, Renal failure, Acute alcoholic hepatitis, sepsis, organ transplant haemolytic Syndromes, CVA, MELD >20, MAP <90.

Results

Day	Group A control HSP level	Group B hypoxic liver injury HSP level	Group C acute hepatitis HSP level	Group C tylenol HSP level
Day 0	0	Intermediate	Low	Normal
Day 4	0	Very high	Low	Intermediate
Day 7	0	Very high	Low	Intermediate
Day 10	0	Low	Low	Normal
MELD (mean)	4	17	8	16
SOFA (mean)	2	8	4	6
MAP (mean)	122	98	102	124

Conclusion Results: Group A; HSP 70 was normal. Group B was intermediate on day 0 and very high on day 4, 7 and reverting back to lower levels on day 10. Group C had a low level (mildly elevated) of HSP-70 on all days with the exception of the Tylenol group where it was intermediate levels. **Conclusion**: HSP-70 is a relevant surrogate marker for hypoxia induced hepatopathy. HSP-70 levels strongly correlated with HILI and poorly with Tylenol induced liver injury. But negatively with other non-vascular liver injuries. Larger study needed to validate this finding.

Competing interests None declared.

PWE-270 UNREVEALING A NOVEL ASSOCIATION OF CHOLESTEROL ESTER STORAGE DISEASE (CESD) AND NON-ALCOHOLIC FATTY LIVER DISEASE (NAFLD)—A SIMILAR CLINICAL SPECTRUM WITH DIFFERENT AETIOLOGY A PROSPECTIVE CLINICAL STUDY

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Introduction NAFLD is the most evolving global morbidity progresses to cirrhosis, liver cancer and Transplantation. Clinical Spectrum is heterogeneous with biochemical and histological diversity. CESD is a part of metabolic storage disease with an intrinsic Lysosomal Acid Lipase deficiency (LAL) mimicking clinical overlap with NAFLD. This clinical pilot study evaluates the clinical overlap of similar metabolic syndromes with diverse aetiology And outcome.

Methods Three hundred (n=300) patients with fatty liver disease with Hepatomegaly, splenomegaly or Both with Mean BMI 27%, Mean (Anthropometric assay W/H ratio mean 0.9, HDL 28, LDL 148, Triglyceride 187, HbA1c 5.9, HOMA Score 2.2, CRP 2.3, ALT 67, RBP 2.3, Homocystein 11, Leptin 3.6, Adiponectin 1.1 TNF-α 1,2, IL10 1.2, IL12 0.8. MELD 4). All under went Abdominal Sonogram and carotid artery Doppler. Serum Fibro sure, NASH score was measured and liver biopsy was performed in NASH group. Patients were divided into Group A (n=100) control with mean BMI 27.8% and no hepato-splenomegaly, Group B (n=100) NAFLD with low BMI <26%, and Group C, NASH (n=100) with NAFLD with BMI >30%.

Results

	Group A	Group B	Group C
LAL levels	0	18/25 (72%)	7/25 (28%)
Heterozygotes	0	18/18 (100%)	6/7 (86%)
Compound heterozygotes	0	0	1/7 (14%)

Conclusion Estimated prevalence of LAL 8.3% compared to the historical data (25 in 1 million) LAL has heterogeneity with an overlap WITH NAFLD and NASH. LAL deficiency has peripheral atherogenic potential with significant clinical morbidities with high Steatotic, Fibrogenic, and inflammatory scores than NAFLA or NASH. CESD is an integral part of Fatty liver disease.

Competing interests None declared.