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PWE-263 LIVER TO ABDOMINAL AREA RATIO: A NOVEL RADIOLOGY TEST FOR PROGNOSTICATION IN LIVER CIRRHOSIS

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Introduction Prognostication in cirrhotic liver disease is difficult. There are several validated indices which are employed including: Child-Pugh score, MELD and UKELD. There is anecdotal data that liver size is important in determining patient survival and likelihood of re-compensation.

Aims To assess a ratio of liver area and abdominal area on cross-sectional imaging using CT to predict the likelihood of death or need for liver transplantation (LT) in patients with liver cirrhosis.

Methods A retrospective analysis of 280 patients referred to the South West Liver Unit. All patients with cirrhosis were included who had liver CT available. Patients with acute liver failure or hepatoma were excluded from the analysis. Using a webpacs system patient imaging were retrieved and the cross sectional image with the largest area of liver was identified. The liver to abdomen area ration (LAAR) was estimated from the hypothesised ellipses represented by the liver and abdomen using the formula $\frac{a^2}{b^2}$ (where 'a' being half of the long axis and 'b' being half of the short axis). These values were compared against patient survival vs patient death/LT. Accuracy of LAAR in predicting the outcome was assessed using Mann–Whitney U test.

Results 280 patients were identified. Sex was available in 200 patients (61% male). Aetiology was available in 266 patients: ALD=103, HCV=32, NASH=10, PBC=10, PSC=13, HCC=31, ALF=12, Others=51. HCC and ALF patients were excluded from analysis. The median age 54.2 (46.6–61.1). Ascites was present in 79 of 127 patients (62%). Not all patients had a CT. LAAR was calculated in 108 patients, median 0.37 (0.3–0.43) and was shown to be predictive of death/LT ($p=0.035$). The presence of ascites did not predict survival (χ^2 2.5, $p=0.12$, OR 1.9 (95% CI 0.86 to 4.01)).

Conclusion LAAR is a simple, novel imaging based technique to assess prognosis in patients with cirrhosis. It confirms anecdotal data that liver size is important in assessing survival. It is more accurate in determining survival than the presence of ascites. LAAR could be incorporated into existing algorithms for patient selection for LT and in determining patient survival with cirrhosis. Its accuracy should be compared against Childs-Pugh, MELD and UKELD alone or in combination to evaluate its utility in clinical practice.

Competing interests None declared.

PWE-264 BLOOD LIPIDOMIC PROFILING OF HEPATOCELLULAR CARCINOMA IN HUMAN AND ANIMAL STUDIES IDENTIFIES LYSOPHOSPHATIDYLCHOLINE (24, 0, 0), A DISCRIMINATORY BIOMARKER

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Introduction The liver is a hub of lipid metabolism and previous studies have shown liver disease and hepatocellular carcinoma

(HCC) to be associated with altered blood lipid profiles. The primary aim of this study was to examine the lipid profile of HCC in an animal model and to compare findings to changes observed in human populations in an attempt to identify novel lipid tumour biomarkers.

Methods Plasma samples were obtained from a Fisher rat model of HCC (n=7) and healthy controls (n=8). Serum and plasma samples were obtained from patients with HCC and cirrhosis from UK (n=3 and 4) and Nigerian (n=5 and 5) cohorts. All samples were analysed using ultra performance liquid chromatography mass spectrometry (UPLC-MS), optimised using in-house developed dichloromethane lipid extraction protocols. Data were processed using XCMS software followed by multivariate analysis to identify lipids most discriminatory between disease groups.

Results In the rat model, multivariate statistical modelling was robust in classifying animals with HCC from healthy controls. In the human studies, multivariate analyses of lipid profiles were less robust in distinguishing HCC from cirrhosis. Lysophosphatidylcholine (24, 0, 0) (LPC), a major cellular membrane component, was identified as most contributory to all multivariate models.

Conclusion Altered global lipid profiles were robust in discriminating HCC from healthy controls in a Fisher rat model, but less so in parallel human studies. Differences in LPC (24, 0, 0) were present in all studies, which may indicate heightened altered tumour cell turnover as a result of HCC growth. The increased plasma concentrations of LPC in HCC in both species suggests that this molecule may be a robust marker as a lipid tumour biomarker of HCC and requires further validation in larger studies with respect to disease classification and response to therapeutic intervention.

Competing interests None declared.

PWE-265 PLASMA METABOLITE PROFILING IN A RAT MODEL OF HEPATOCELLULAR CARCINOMA AND THE EFFECTS OF CO-ADMINISTERED ANTIBIOTICS

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Introduction The profiling of metabolites, small molecules representing the end points of cellular processes in biofluids, has allowed the detection of novel biomarkers of disease. There are several rat models of hepatocellular carcinoma (HCC), however, there have been no previous reports of ¹H NMR spectroscopy plasma metabolic profiling in animal models of HCC. Quinolone antibiotics, such as norfloxacin, are known to reduce the inflammatory component of liver fibrosis potentially reducing end-stage complications. The primary aim of this study was to identify blood metabolic profile biomarkers of HCC in a rat model of HCC and the secondary aim was to evaluate the effect of the norfloxacin on metabolic profiles.

Methods HCC was induced in 10 Fisher rats by administration of intra-peritoneal diethylnitrosamine (DEN) and oral N-nitrosomorpholine (NMOR) and plasma was collected upon sacrifice. Five rats were concomitantly administered oral norfloxacin. Six Fisher non-treated rats acted as healthy controls. Proton NMR spectra were acquired for all samples using a Bruker 600 MHz NMR system and results were analysed by visual comparison and multivariate analysis.

Results Proton NMR spectra from diseased rats displayed significant decreases in lipoproteins, unsaturated fatty acids, N-acetyl-glycoproteins, acetoacetate, and glucose ($p \leq 0.001$). Plasma citrate and formate levels were increased ($p=0.02$). Although animals treated with norfloxacin also developed tumours, background fibrosis and tumour nodularity was less marked than non-antibiotic treated