Their expression of CXCR3 provides a potential mechanism for recruitment into the tumour environment.

Competing interests None declared.

BASL plenary session

OC-022 | EMBOLISATION OF INFLOW TO ALLOW SAFER LIVER RESECTION—IS MORE, BETTER?

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Introduction Portal vein embolisation (PVE) is now an established technique to increase the future liver volume/remnant (FLR) prior to liver resection. For those patients where hypertrophy is still considered insufficient complete uni-lateral embolisation incorporating both portal and hepatic artery embolisation (HAE) has been less frequently reported. The aim of this study was to evaluate the feasibility of sequential PV/HA embolisation to increase the FLR prior to liver resection.

Methods All HPB patients are discussed at a weekly MDT meeting to decide on appropriate management decisions including the necessity for FLR augmentation. PVE is performed by initially obtaining a portogram by percutaneous trans-hepatic puncture. Selective embolisation of the necessary portal veins are then performed using a combination of coils and glue etc. Embolisation of Segment 4 PV branches are performed on a selective basis. HA embolisation is performed by mapping arterial inflow and selectively embolising the desired segments planned for resection while carefully preserving the FLR. The aim of this study was to evaluate the feasibility/safety of PVE with sequential HAE over a 5-year period (January 2006–May 2011).

Results 50 patients (M:F = 38:12) underwent a right PVE; 33 (66%) progressed to liver resection. Reasons for inoperability (34%) following PVE (n=17) were (1) Small FLR, (n=6) all underwent HAE (with five proceeding to liver resection) (2) extra-hepatic disease (n=7) (3) progression of hepatic disease (n=4). The median FLR of those who progressed to resection following PVE, by CT volumetry, was 384.5 cc (330–490), significantly more than those who did not 237 cc (110–280) p=0.03. HAE increased the FLR by a further 99.8 cc (range 80.5–130 cc). An R0 resection was achieved in 25 patients (76%), including 4/5 (80%) of sequential patients. Following PVE and sequential embolisation; 9/33 (27%) and 3/5 (60%) suffered serious complications (Clavien-Dindo 3 or 4). There were six post operative deaths including 5/33 (15%) after PVE and 1 (20%) following sequential embolisation respectively.

Conclusion PVE is an increasingly used technique to increase the FLR allowing a significant proportion of patients an R0 resection despite initially being considered inoperable. Nevertheless at least 20% of patients will also have progression of disease. Patients who do not achieve adequate hypertrophy can potentially have HA embolisation to increase the FLR by a further 100 cc but perhaps at the expense of increasing post-operative complications.

Competing interests None declared.

OC-023 | EXTRACORPOREAL LIVER SUPPORT USING UCL-ARSENEL REDUCES INFLAMMATION, IMPROVES HAEMODYNAMIC FUNCTION AND INCREASE SURVIVAL TIME IN A PORCINE PARACETAMOL-INDUCED ACUTE LIVER FAILURE MODEL

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Introduction Though the incidence of liver disease continues to increase, an effective liver support device remains an unmet clinical need. We have demonstrated that in liver failure, albumin function is irreversibly damaged, preventing detoxification processes, and that bacterial endotoxins induce systemic inflammation and neutrophil dysfunction. To date, toxin removal devices have failed to demonstrate clinical efficacy, which may be due to an inability to address albumin damage and/or inflammation. An albumin replacement system with a novel endotoxin ligation (ARSeNEL) component was developed to selectively to adsorb endotoxin and replace damaged albumin in patients' plasma.

Methods We tested the device in an acetominophen model of acute liver failure (ALF). 16 female landrace pigs (eight ALF, five ALF + UCL-ARSeNEL) were studied. Irreversible ALF was induced by acetaminophen administration via a jejunel catheter, confirmed by deranged clotting function (PT >30% normal). Treatment was with UCL-ARSeNEL or CVVH control within 2 h of ALF confirmation. The ARSeNEL device consists of three components; plasmapheresis, endotoxin and high cut-off (100 kDa) filters; with fresh frozen plasma replacing ultrafiltered plasma. Endpoints were: survival; ICP; heamodynamic parameters, standard biochemistry; cytokines; albumin damage; and plasma endotoxin levels.

Results UCL-ARSeNEL significantly increased survival post ALF (ALF 15.8 ± 2.4 h vs UCL-ARSeNEL 23.8 ± 1.9 h; p=0.02). Endotoxin reduced by a quarter (1.99 ± 0.18 Eu/ml vs 1.42 ± 0.21 Eu/ml) in the device group at 16 h. The changes in ICP index (1.7 ± 0.07 vs 1.4 ± 1.58), INR (16.6 ± 6.6 vs 6.8 ± 0.5), ischaemia-modified albumin ratio (0.45 ± 0.166 vs 0.35 ± 0.108), noradrenaline requirement (61.11 ± 15.4 vs 28.7 ± 15.2 μg/Kg), and mean arterial pressure (71 ± 7.6 vs 87 ± 6.0 mm Hg) showed marked improvement in the UCL-ARSeNEL group. Measured inflammatory cytokines IL8, IL6, IL1b, TNFa and neutrophil activation (spontaneous burst p=0.03) were all found to be reduced in the ARSeNEL treated group compared with ALF control.

Conclusion These results confirm that UCL-ARSeNEL improves survival in ALF by addressing key pathophysiological derangements such as albumin dysfunction and endotoxinaemia; which impact upon systemic inflammation and end-organ function. The reduction in inflammation is associated with improved vascular function and reduced inotropic support requirements.

Competing interests None declared.

OC-024 | DEVELOPMENT AND VALIDATION OF A NOVEL CAPTURE-FUSION MODEL FOR HCV REPLICATION

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Introduction HCV replicates poorly in vitro, so testing of novel antiviral therapies currently relies on modified viral replicons, based on genotype (G)1, or the G2 JFH-1 virus. A model allowing patient virions to be cultured would facilitate drug discovery and allow direct sensitivity testing. Here we describe the development of a novel HCV replication assay, its validation using the antiviral agents alisporivir and telaprevir and its value in identifying responses to interferon and ribavirin.

Methods CD14 (+) monocytes derived from patients with chronic HCV infection, or pre-stimulated THP-1 cells infected with serum from G1 and G3 HCV infected donors, were fused with HuH7 cells and treated with antiviral agents at various concentrations. The fused cells were maintained in tissue culture for up to 5 days, before