P98 PREDICTORS OF EARLY HEPATIC ARTERY THROMBOSIS AFTER PRIMARY LIVER TRANSPLANTATION: A COHORT MULTI-CENTRE STUDY

doi:10.1136/gut.2010.223362.124

M Dawwas, A Gimson. *Liver Unit, Addenbrooke's Hospital, Cambridge, UK*

Introduction Hepatic artery thrombosis (HAT) is one of the most catastrophic complications of liver transplantation. The risk factors for this complication have rarely been assessed in the setting of a large risk-adjusted analysis.
Method Using the United Kingdom and Ireland Liver Transplant Database, we sought to identify the incidence of and independent risk factors for early (=3 months) HAT among 6297 adult first single-organ liver transplant recipients during the period 1 March 1994–31 March 2006. Univariate and multivariable logistic regression models were fitted to examine the association between early HAT and a wide-range of recipient, donor and graft risk factors.
Results The incidence of early HAT in this cohort was 3.6%. Multivariable analysis identified the following independent risk factors for early HAT: recipient diagnosis of hepatocellular carcinoma (OR 1.64 95% CI 1.08 to 2.47), primary sclerosing cholangitis (OR 1.86 95% CI 1.24 to 2.81), recipient requirement for preoperative renal support (OR 1.81 95% CI 1.26 to 2.60), use of reduced vs whole graft (OR 2.53 95% CI 1.40 to 4.57), use of multiple vs single HA anastomosis (OR 1.55 95% CI 1.11 to 2.18), lower donor weight ($p<0.001$), combination of non-white donor and non-white recipient vs white donor and white recipient (OR 4.67 95%CI 1.41 to 15.47), combination of CMV+ donor and CMV- recipient vs CMV+ donor and CMV+ recipient (OR 1.70 95% CI 1.19 to 2.41), and combination of female donor and female recipient vs male donor and male recipient (OR 1.80 95% CI 1.21 to 2.66). Even after adjustment for the above risk factors, the risk of early HAT was significantly higher in four of the eight liver transplant centres in the UK and Ireland compared to the centre with the lowest incidence, the ORs ranging between 3.5 and 4.6 ($p<0.001$).
Conclusion Our findings suggest that early HAT is predominantly a technical complication that is independent of recipient, donor and graft characteristics. HAT risk-stratification based on the results of this analysis could help identify patients who may benefit from increased surveillance for this complication and/or institution of appropriate prophylactic antithrombotic therapy.

P99 RELATION OF DONOR RISK INDEX AND MODEL FOR END-STAGE LIVER DISEASE SCORE TO OUTCOME OF ELECTIVE LIVER TRANSPLANTATION; A SINGLE CENTRE EXPERIENCE

doi:10.1136/gut.2010.223362.125

M Al-Freah, M A B Al-Freah, E Dionigi, A Suddle, V Aluvihare, K Agarwal, J Wendon, G Auzinger, J O'Grady, M Rela, M Heneghan, N Heaton, W Bernal. *King's College Hospital, UK*

Introduction Patients with advanced liver disease, reflected in high model for end-stage liver disease (MELD) scores have the lowest chances of survival without liver transplantation (LT) and yet, may derive greatest survival-benefit from LT. The use of extended criteria (EC) grafts in such patients has been advocated as a means of optimising the chances of successful transplantation.
Aim To determine the effects of graft quality (using Donor Risk Index, DRI) and MELD score at the time of LT on the duration of post-LT intensive care unit (ICU) and hospital length of stay (LOS) and survival.
Method Retrospective analysis of 898 adult patients who underwent elective LT (109 re-do LT) over the period 2000–2008. Pre-LT MELD scores were categorised as low (<15), intermediate (15–25), or High (>25). Graft quality was categorised as low (DRI >1.7) or High (DRI <1.7). Data are presented as median (IQR).
Results Median age was 53 years (44–60) and 64% were male. Median pre-LT MELD was 15 (11–19) and median DRI in the high and low quality groups were 1.5 (1.3–1.6) and 2.0 (1.9–2.1). Increasing MELD score was associated with greater ICU and Hospital LOS ($p<0.0001$, Kruskal–Wallis test). However, within each MELD category there was no significant difference in ICU or hospital LOS between recipients of grafts with DRI < or >1.7. See Abstract P99 table 1.

Abstract P99 Table 1

	DRI <1.7		DRI>1.7	
MELD	ICU	Hospital	ICU	Hospital
Low (<15)				
n	245		190	
	3 (2–4)	17 (13–29)	3 (2–4)	18 (13–29)
Intermediate (15–25)				
n	237		139	
	3 (2–5)	22 (14–40)	3 (2–5)	21 (14–41)
High (>25)				
n	64		22	
	7 (3–22)	33 (21–68)	5 (4–18)	29 (18–46)

On multivariate regression analysis MELD was an independent predictor of LOS with no significant effect of DRI identified. Survival at 1-year in the overall cohort was 90%; in MELD <15 92.6%, 15–25 87.4% and >25 82.7%. There were no significant differences in 1 year survival between recipients with DRI < or >1.7 in the group as a whole or in the MELD sub-categories.
Conclusion In this cohort, length of ICU and hospital stay related to pre-LT MELD score and not graft quality as evaluated by DRI. Hospital and ICU stay and consequently resource use was increased particularly in those patients with MELD of 25 and above, but not further prolonged by the use of ECD grafts in the range of DRI used in this cohort.

P100 INTRAHEPATIC AND SYSTEM CYTOKINE PROFILES IN ACETAMINOPHEN-INDUCED ACUTE LIVER FAILURE: POSSIBLE MECHANISM OF IMMUNEPARESIS

doi:10.1136/gut.2010.223362.126

H Antoniades, Q Anstee, M Bruce, R Mitry, M Hussain, G Auzinger, C Willars, J Wayel, D Concas, N Heaton, R Goldin, W Bernal, A Quaglia, M Thursz, J Wendon. *Hepatology and Transplantation, King's College Hospital, London, UK*

Introduction Activation of systemic inflammatory responses in acetaminophen-induced acute liver failure (AALF) is associated with elevated levels of both pro- and anti-inflammatory cytokines. Functional monocyte deactivation has been described and this is thought to contribute to increased susceptibility to sepsis and a higher mortality rate. Anti-inflammatory cytokines play a major part in the resolution of inflammatory responses and promote tissue repair processes but increase the risk of systemic infections. We hypothesise that the levels of anti-inflammatory cytokines mirror the severity of hepatic necrosis and reflect attempts to resolve inflammation during AALF. It is the excessive production of these mediators that "spill-over" and increase the risk of systemic sepsis. We sought to delineate hepatic and systemic cytokine responses in experimental and human AALF, and, determine whether there is production of anti-inflammatory cytokines in the liver which "spill-over" into the systemic circulatory compartment.
Aim We sought to delineate hepatic and systemic inflammatory responses in experimental and human AALF, and, determine