Patients&Methods: Between 2003-2010, 30 DCD-LTx were performed (6% of all LTx). Donor demographics, LTx indications, post-LTx peak transaminase (AST),%biliary complications and%graft rejection were analyzed. Patient/graft survival were analyzed and compared to outcome using Donation after Brain Death (DBD) donors.
Results: Mean donor age was 47.3 yr (range:13-69). Warm ischemia time (stop ventilation to cold perfusion) was 23±11'. Cold ischemia time was 415±104'. Recipient age was 58 yr (range:24-71). Mean labMELD was 17 (range:8-31). LTx indications were cirrhosis related to post-ethyl (13), HCV (4), NASH (3), unknown (4), PBC (1), PSC (1), acute liver failure (1), congenital disorder (1) or HCC without cirrhosis (2). Eleven recipients (37%) had an associated HCC. Post-LTx AST peak was 1712 IU/L. Reasons for graft loss were: hepatic artery thrombosis (1), ductopenic rejection (1) and diffuse intrahepatic biliary strictures (1). Ten patients (33%) developed non-anastomotic biliary complications requiring conservative treatment (2), endoscopic interventions (7) and re-LTx (1). Three recipients had acute rejection: 2 responded to steroids and 1 developed ductopenic rejection. Follow-up ranged from 1-93 months. Actuarial 1, 3, and 5-yr patient/graft survival after DCD-LTx was 92, 83 and 83%, and 89, 79, and 79%, respectively and was similar after DBD-LTx (1, 3 and 5-yr patient/graft survival of 90, 82 and 75%, p=0.846 and p=0.707, respectively).
Conclusion: Unlike in registry data and despite substantial ischemic injury (high peak-AST), short/long-term survival after DCD-LTx was comparable to DBD-LTx. Rapid donor surgery by experienced surgeons, careful donor/recipient selection, short warm/cold ischemia times are key factors to optimize outcome after DCD-LTx. However, strategies to reduce biliary complications are warranted.

RO-033 EFFEKT OF Cilostazol® ON HEPATIC MICROCIRCULATION AND LIVER REGENERATION AFTER PARTIAL HEPATECTOMY IN A RAT MODEL

Maximilian von Heesen[1], Stefan Dold[1], Pascal Jeanmonod[1], Michael D. Menger[2], Martin K. Schilling[1], Mohammed R. Moussavian[1]. *[1]General-, Visceral-, Vascular- and Pediatric Surgery, Saarland University Hospital, Homburg/Saar, Saarland, Germany; [2]Institute for Clinical & Experimental Surgery, University of Saarland, Homburg/Saar, Saarland, Germany*

Introduction: Major liver resection has the risk of postoperative liver failure. So far no efficient treatment was established to improve patient outcome after extended liver resections. The aim of our study was to elucidate if pretreatment with Cilostazol®, a selective phosphodiesterase (PDE)-III-Inhibitor, was capable to improve hepatic perfusion and liver regeneration after major hepatectomy.
Materials and Methods: Sprague-Dawley rats (n=64) were pretreated with Cilostazol® (5mg/kg KG) or glucose solution for 5 days. After 70% liver resection on day 0 as well as on 1., 3. and 6. postoperative day hepatic arterial and portal venous blood flow were analyzed by ultrasonic flow measurement and microvascular blood flow by laser-Doppler-flowmetrie (LDF). Hepatic function and liver regeneration were characterized by bile excretion and liver histology (PCNA). Cilostazol® or placebo was given until end of experiment. Additional animals (n=16) received PDE-III-inhibitor or placebo and no liver resection. Mean values ± SEM, $p < 0{,}05$.
Results: Pretreatment with the PDE-III-Inhibitor resulted in a significantly impaired portal venous blood flow (2.00±0.07 vs. 1.56±0.11 ml/g*min; $p<0.05$) and improved hepatic microcirculation (642.9±41.1 vs. 493.8±25.0 aU; $p<0.05$) when no liver resection was performed. Portal blood flow and hepatic microcirculation were increased 77% and 32% respectively after 70% hepatectomy, whereas hepatoarterial blood flow was found unchanged. Cilostazol treatement improved hepatic blood flow and microcirculation. Interestingly liver regeneration was found enhanced by cilostazol over the whole observation period, with a maximum on the first day after liver resection (32±4 vs. 20±2 PCNA +cells/HPF $p<0.05$).
Conclusion: We could demonstrate that preconditional PDE-III inhibition can improve hepatic perfusion and liver regeneration after 70% hepatectomy. Thus, PDE-III inhibitors may represent an efficient drug therapy to ameliorate liver regeneration and hepatic function after extended liver resection.

RO-034 TRENDS OF USAGE IN STEATOTIC LIVER GRAFTS FOR ORTHOTOPIC LIVER TRANSPLANTATION OVER A TEN YEAR PERIOD IN A SINGLE INSTITUTION

Jeffrey T. Lordan, B. Gunson, Hynek Mergental, John Isaac, David A. Mayer, Paolo Muiesan, Simon R. Bramhall, Darius F. Mirza, D.A. Neil, Thamara Perera. *Liver and Transplant Surgery, University Hospitals Birmingham, Birmingham, United Kingdom*

Introduction: Macrovesicular steatosis is associated with poor peri-operative and long term outcome after liver transplantation (LT). This study was designed to evaluate the trends of steatosis in transplanted grafts at a single institution.
Patients and methods: Graft steatosis was semi-quantitatively assessed in biopsies after immediate reperfusion (t0), and trends in graft steatosis of a historical control (group A;2001–2005) were compared with those obtained in the recent past (group B;2006 –2011). Outcomes were compared between groups with relevance to the degree of steatosis. Significance was assigned at $p<0.05$ using χ^2, Kruskal-Wallis and Mann-Whitney U tests.
Results: Total of 586 t0 biopsies were available from 1172 LT performed during the study period. (Group A, n=211 (36%); group B, n=374 (64%)). The donor age in groups A and B were 53.1 (16.6–72.1)years vs. 54.1 (18.0–73.4) years (p=0.71) and BMI was 25.7kg/m^2(16.5–50.8kg/m^2) and 25.9kg/m^2(14.7–48.0kg/m^2) respectively (p=0.81). The incidence of moderate (MS) and severe steatosis (SS) was 36 (17.1%) and 10 (4.7%) compared to 53 (14.2%) and 3 (0.8%) in group B respectively (p=0.001;OR=1.21;95%CI,0.63-2.33). For the entire cohort, the peri-operative morbidity for no steatosis, MS and SS was 48 (10%), 14 (16%) and 4 (31%) respectively (p=0.02), and the peri-operative mortality was 28 (6%), 9 (10%) and 4 (31%) respectively (p=0.001).The peri-operative morbidity was 26 (14.0%) and 40 (10.0%) respectively (p=0.16). The incidence of peri-operative mortality in group A was 17 (9.1%) compared with 24 (6.0%) in group B (p=0.12).
Conclusion: There has been a significant trend towards reduced use of steatotic grafts for LT due to the significantly increased peri-operative risk. Novel strategies to increase the safe use of steatotic grafts should be explored.

RO-035 LIPOCALIN-2, A PROMISING INFLAMMATORY MARKER IN ACUTE ALLOGRAFT REJECTION?

Herbert T. Maier[1], Natalie Vallant[1], Markus Kofler[1], Philipp Schumpp[1], Hubert Schwelberger[1], Hanno Ulmer[2], Stefan Schneeberger[1], Jakob Troppmair[1], Johann Pratschke[1], Felix Aigner[1]. *[1]Department of Visceral, Transplant and Thoracic Surgery Innsbruck, Medical University Innsbruck, Innsbruck, Austria; [2]Department of Medical Statistics, Medical University Innsbruck, Innsbruck, Austria*

Purpose: Lipocalin-2 (Lcn-2) is associated with ischemia/reperfusion injury (IRI) in different organs. Data on Lcn-2 expression during allograft rejection have been missing so far. The main focus of this study was to analyze the possible implication of Lcn-2 during acute rejection following liver transplantation.
Material and Methods: Serum of 68 patients undergoing orthotopic liver transplantation was collected preoperatively and postoperatively from day 1 to 15. Lcn-2 was analyzed by ELISA and expression levels were correlated with parameters of allograft rejection.
Results: Six patients (8.8%) experienced acute graft rejection within and 12 patients (17.7%) were diagnosed rejection beyond 20 days post transplantation. Serum levels of Lcn-2 following liver transplantation were elevated 3 to 7-fold immediately after transplantation due to IRI and also increased prior to clinically apparent acute rejection closely related to elevated routine markers (e.g. AST, ALT). Dynamic correlations could be observed between Lcn-2 expression and posttransplant renal function and immunosuppression regimens.
Conclusion: Lcn-2 is an inflammatory marker upregulated during acute graft rejection and its expression prior to clinical parameters of acute rejection might help to identify possible targets for therapeutic intervention. Lcn-2 expression correlates with posttransplant renal function (e.g. delayed graft function) and various immunosuppression regimens and is therefore proposed a monitoring marker in the early posttransplant period.

RO-036 LIVER TRANSPLANTATION FOR HEPATOCELLULAR CARCINOMA RECURRENCE AFTER LIVER RESECTION: WHY DENY THIS CHANCE OF CURE?

Stefano Di Sandro, Plamen Mihaylov, Abdallah Slim, Alessandro Giacomoni, Andrea Lauterio, Iacopo Mangoni, Matteo Tripepi, Cinzia Poli, Luca Lamperti, Caterina Cusumano, Luciano De Carlis. *General Surgery and Transplantation, Niguarda Hospital, Milan, Italy*

Introduction: Liver Transplantation (LT) after Liver Resection (LR) for Hepatocellular Carcinoma recurrence may be associated with poor patients long term results and higher peri-operative patients morbidity and mortality. Despite that, some studies published later have demonstrated opposite results, emphasizing the absence of different outcome between primary LT or LT secondary to prior LR.Thisstudy focused on short- and long-term outcomes of LT recipients due to HCC recurrence after LR in asingle-institution cohort as well as inhighly comparablecase-matched subgroups.
Methods: From 2000 to 2009, 570 consecutive patients with documented HCC were treated our Institute by LR (n = 355, 62.2%) or LT (n = 215, 37.8%). The case-matched analysis between two groups: Group A1, LT recipients whom have received a previous LR (n = 26); Group B1, LT recipients whom have not received a previous LR (n = 26).
Results: Patients morbidity resulted higher among the A1 Group in term of packed red blood cells units transfused (respectively, 4.9 and 2.5 for Group A1 and B1: P-value=.008), fresh frozen plasma units transfused (respectively, 2.0 and 1.3 for Group A1 and B1: P-value=.035), median operative time (respectively, 430 min vs 366 min for Group A1 and B1: P-value=.04), post-operative