Orals

BASIC SCIENCE

O01 MELATONIN PROTECTS FROM HEPATIC REPERFUSION INJURY THROUGH INHIBITION OF IKK AND JNK PATHWAYS AND MODIFICATION OF CELL PROLIFERATION

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Background: Reactive oxygen species (ROS) are involved in pathophysiology of ischemia/reperfusion injury. Melatonin is a potent scavenger of ROS. Thus, this study was designed to elucidate its effects in a combined hepatic warm ischemia and resection model.

Methods: The right lateral and caudate lobes (40% of liver) of SD rats underwent warm ischemia for 30 min followed by reperfusion and subsequent resection of the non-ischemic liver tissue (60% of liver). Some rats were gavaged with 50 mg/kg melatonin 2 h before experiments. Controls received the same volume of vehicle. Transaminases were measured serially within 48 h post-reperfusion to assess hepatic injury. H&E staining (to index typical ischemic changes in liver), immunohistochemistry (for iNOS expression in hepatocytes), and flow cytometry (to investigate signal transduction pathways of oxidative stress [JNK, cJUN, NF-κB], and cell proliferation [(PCNA, Ki67)] were performed at 48 h post-reperfusion. One-way ANOVA or Fisher's exact test were used as appropriate. Log rank test of Kaplan–Meier analysis was used for survival. Results are presented as mean ± SEM.

Results: Melatonin significantly improved survival (70% of cases vs. 0% of controls at day 7, $P < 0.05$). Within 48 h post-reperfusion, melatonin lowered the area under the curve for transaminases about 15% ($P < 0.05$). In parallel, melatonin significantly reduced the indices for necrosis, liver damage, leukocyte infiltration, and iNOS expression from 14.7 ± 1.62, 1.6 ± 0.22, 20.9 ± 1.53, and 2.33 ± 0.057 in controls to 6.8 ± 1.20, 1.1 ± 0.07, 17.2 ± 1.02, and 1.55 ± 0.054 in cases, respectively; $P < 0.05$). Furthermore, the expression of IKKα, JNK1, and cJUN was 35%–50% lower in melatonin group ($P < 0.05$). At the same time, significantly reduced expression of both PCNA and Ki67 in liver was documented after melatonin ($P < 0.05$).

Conclusion: Melatonin is hepatoprotective most likely via mechanisms including an inhibition of the IKK and JNK pathways and regulation of cell proliferation.

O02 HUMAN UMBILICAL CORD BLOOD MESENCHYMAL STEM CELLS CAN CONTRIBUTE TO LIVER REGENERATION IN AN ANIMAL MODEL OF CHEMICAL-INDUCED LIVER INJURY

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Background: It is well accepted that human umbilical cord blood (UCB) is an interesting source of mesenchymal stem cells (MSCs) able to differentiate into mesodermal-derived cells and potentially into hepatocyte-like cells. These cells could be employed for cell therapy, in particular for the treatment of acute liver diseases, as an alternative to liver transplantation.

Aim: To evaluate the contribution of MSCs from UCB to liver regeneration in an animal model of acute chemically-induced liver injury.

Methods: Liver injury was induced in 17 male Lewis rats by intraperitoneal injection of CCl4 (1 ml/kg). 48 h after injury, six animals received 106 MSCs isolated from UCB in the peritoneum, five animals received physiological buffer as a control, while six animals died before the cell treatment. Animals were sacrificed at different time points and liver, kidneys and lungs were harvested, fixed and paraffin embedded. Samples were treated with hematoxilyn-eosin staining to evaluate the parenchyma and with immunohistochemical analysis to detect human HLA1 expression. At day 7 after MSCs injection, steatosis and sinusoid dilatation in cell treated animals were lower than the in controls, while at day 14, the liver appeared almost completely regenerated both in cell treated animals and in controls. Immunohistochemical analysis at day 7 after the injection confirmed the presence of HLA1 positive cells only in liver treated with MSCs, but not in any other investigated organ.

Conclusions: MSCs from UCB seem to contribute to liver regeneration by enhancing the early phase of reparative activity following an acute injury that specifically attracts MSCs. However, hepatocytes themselves are able to repair the hepatic damage in a longer time interval. Nevertheless, the modality of MSCs contribution to liver regeneration remains to be investigated.

O03 THE ROLE OF BILE SALT TOXICITY IN THE PATHOGENESIS OF BILE DUCT INJURY AFTER NON–HEART-BEATING PORCINE LIVER TRANSPLANTATION

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Background: Intrahepatic bile duct structures are a serious complication after non-heart-beating (NHB) liver transplantation. Bile salt toxicity has been identified as an important factor in the pathogenesis of bile duct injury and cholangiopathies. The role of bile salt toxicity in the development of biliary strictures after NHB liver transplantation is unclear.

Methods: In a porcine model of NHB liver transplantation, we studied the effect of different periods of warm ischemia in the donor on bile composition and subsequent bile duct injury after transplantation. After induction of cardiac arrest in the donor, liver procurement was delayed for 0 min (group A), 15 min (group B) or > 30 min (group C). Livers were subsequently transplanted after 4 h of cold preservation. In the recepients, bile flow was measured and bile samples were collected daily to determine the bile salt/phospholipid ratio. Severity of bile duct injury was semi-quantified by using a histological grading scale.

Results: Posttransplant survival was directly related to the duration of warm ischemia in the donor. The bile salt/phospholipid ratio in bile produced early after transplantation was significantly higher in group C, compared to group A and B. Histopathology showed the highest degree of bile duct injury in group C.

Conclusion: Prolonged warm ischemia in NHB donors is associated with the formation of toxic bile after transplantation, with a high biliary bile salt/phospholipid ratio.These data suggest that bile salt toxicity contributes to the pathogenesis of bile duct injury after NHB liver transplantation.

O04 PROSPECTIVE STUDY OF NATURAL KILLER (NK) CELL PHENOTYPE IN RECURRING HCV INFECTION FOLLOWING LIVER TRANSPLANTATION

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Background/Aim: Graft reinfection occurs immediately after liver transplantation for HCV-induced cirrhosis but disease progression is variable and largely unpredictable. Several factors may influence the severity of liver disease, including immune responses. Although variable findings had previously been reported for adaptive immunity, the role of innate immunity has never been specifically addressed in this setting. NK cells are the principal effector population involved in innate immune responses to viral infections, and are able to control adaptive immunity via direct interactions between immune cells belonging to both compartments. In this study, we prospectively examined the phenotype of peripheral blood NK cells from 18 patients with recurring hepatitis C followed for 1 year post-transplant and 18 healthy controls.

Methods: Blood mononuclear cells were obtained at baseline and 1,7,30 days and 6 to 12 months after transplantation. Nine patients were treated with cyclosporine, nine with tacrolimus and 12 also received steroids. NK receptors were identified by labelling with monoclonal antibodies specific for inhibitory (NKG2A, CD94, p75, IRP60, iNKRs) and activatory (NKp46, NKp30, NKp44, NKG2D, NKG2C) receptors, as well as the CD69 activation marker.

Results: One day after surgery the activatory NKG2D receptor molecule was downregulated ($P = 0.001$) and, conversely, the inhibitory iNKRs pool was upregulated ($P = 0.042$). The proportion of circulating NK cells was significantly reduced ($P < 0.0001$) 7 days post transplant, probably as a result of migration towards the engrafted liver; however, at the same time, the proportions of CD69- and NKG2D-expressing cells were significantly higher compared with controls ($P = 0.001$ and $P = 0.002$, respectively) attesting to the activated state of NK cells. There was a steady increase in the proportion of cells expressing the activatory NKG2C receptor as a function of time ($P = 0.0067$ at 6 months. and $P = 0.0078$ at 1 year). At baseline, there was a statistically significant negative correlation between the total proportion of circulating NK cells and serum ALT values ($r = 0.497$, $P = 0.042$) suggesting preferential intrahepatic compartmentalization where they may contribute to liver damage. A direct correlation was instead found between expression of the activatory NKp46 receptor and ALT ($r = 0.66$, $P = 0.007$) suggesting that

these cells may eventually participate in the necroinflammatory process. In addition, 7 days post transplant, a direct correlation was noted between HCV RNA levels and NK cells expressing a number of inhibitory receptors suggesting a virus-induced, predominant inhibitory phenotype in the early phase of graft reinfection. There was no correlation with type of immune suppression and viral genotype.
Results: The results of this prospective study of NK cell phenotype in patients with recurring hepatitis C after transplantation show a complex modulation of both activatory and inhibitory receptors, most of which return to baseline levels of expression after significant fluctuations. However, NKG2C-positive cells steadily increase over time similarly to observations in immunocompetent patients with chronic HCV infection in whom the proportion of NK cells expressing activatory receptors is also increased. Moreover, the clinical correlates suggest a role for NK cells in the hepatic inflammatory process.
Conclusions: A low acetaminophen bolus and continuous enteral administration adjusted to blood levels delivers a well reproducible pig model of acute hepatic failure with defined survival times.

O05 HISTOLOGICAL STUDIES IN VARIOUS MODELS OF ACUTE HEPATIC FAILURE

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Objective: Several animal models have been developed for evaluation of acute hepatic failure. A differentiation of liver damage and regeneration is needed to evaluate the different models.
Methods: In 12 German landrace pigs (30–35 kg) hepatic failure was induced by acetaminophen 1g/kg b.w. ($n = 6$), amanitin 0.15 mg/kg b.w. ($n = 2$) or 0.35 mg/kg b.w. ($n = 2$) or extended liver resection ($n = 2$) with 3 h hypoxia of the remaining segments. Hemalum-Eosin and immunohistochemistry staining (Ki67 for regeneration and TUNEL-apoptosis staining) were performed in liver biopsies taken every 24 h. Albumin, ALT, AST and liver depending clotting factors were measured every 8 h.
Results: Liver biopsies of acetaminophen induced liver failure showed massive centrolobular necrosis 24 to 48 h after intoxication which was confirmed by a significant decline of PT and albumin and a worsening clinical course. In the liver of surviving pigs a diffuse Ki67 expression in all over the periportal fields (positive cells per field: 24 h:5, 48 h:27 and 72 h:93) and a focal apoptosis was shown. After amanitin intoxication liver necrosis was seen between 48 and 72 h, 24 h later compared to acetaminophen. In animals with low toxin administration 20% Ki67-positive hepatocytes were observed 96 h after intoxication, which leads to a functional recovery of liver depending clotting factors. In animals with high amanitin dosage, less than 5% Ki67-postive hepatocytes and massive congestion were observed. Disseminated apoptosis was detected. In the resection model, liver architecture in the remaining tissue was restored within 3 days after injury. No signs of liver cell necrosis were observed.
Conclusions: Maximum necrosis and localisation of liver damage and the potential of regeneration is specific to the mechanism of liver injury. This fact must be taken into consideration before therapeutic strategies and liver support devices will be tested.

HEPATOCELLULAR CARCINOMA

O06 PREOPERATIVE FINE NEEDLE ASPIRATION-BIOPSY IN PATIENTS WITH LIVER TRANSPLANTATION FOR HEPATOCELLULAR CARCINOMA. WHY USE IT?

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Introduction: Liver transplantation (OLT) has been advocated for patients with carcinoma hepatocellular (HCC). A preoperative biopsy (fine needle aspiration biopsy) [FNA] facilitates preoperative diagnosis of adverse pathological factors: vascular invasion or histological differentiation. But a biopsy may cause abdominal dissemination and be related to a higher incidence of recurrence.
Patients and Methods: From April 1986 to December 2003, we performed 95 OLT for HCC. We divided them in two groups: group A without FNA-biopsy (67.9%) and group B with FNA-biopsy (32.1%).
Results: We obtained the diagnosis of HCC in only 15 patients (57.6%). In two patients an OLT was avoided due to the presence of abdominal dissemination at the time of transplant. Recurrence incidence was higher among group B patients (5.9% vs. 31.8%; P .003) due to extrahepatic recurrence (2% vs. 27.3%; $P = 0.003$). No differences were observed in morbidity or mortality. The two groups were homogeneous in epidemiological and pathological variables except: sex distribution, Child status, AFP level, tumor size, and pTNM stage. If we compare recurrence rates in the two groups attending to these nonhomogeneous variables, it was significantly higher among patients with tumors larger than 3 cm, pTNM I–III stage, Child B-C, AFP > 200 ng/ml, and males or females.
Conclusions: Preoperative liver biopsy is associated with a larger incidence of tumor recurrence, so we believe that it is not necessary prior to an OLT for HCC.

O07 VALIDATION OF THE BCLC PROGNOSTIC SYSTEM IN SURGICAL HCC PATIENTS. IS IT NECESSARY TO MODIFY THE BCLC TREATMENT ALGORITHM?

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Objectives: To investigate the value of known prognostic systems in 400 Italian patients with hepatocellular carcinoma (HCC) treated with surgical radical therapies.
Background: Prognosis assessment in surgical HCC patients remains controversial. The most widely used HCC prognostic tool is the BCLC classification, but its prognostic ability in surgical patients has not been yet validated.
Methods: A prospective database collection including 400 surgical HCCs observed at a single Institution from 2000 and 2007 was analyzed. By using survival time as the only outcome measure (Kaplan–Meier method and Cox regression), the performance of BCLC classification was compared with that of Okuda, CLIP, UNOS-TNM, and JIS staging systems. A cohort of 315 non surgical patients enrolled in the same period was finally used to test the prognostic power of surgery in each BCLC stage.
Results: Two hundred-twenty five patients underwent to laparotomic resection, 55 to laparoscopic procedures (ablation and/or resection), 120 to liver transplantation In the particular cohort studied, BCLC proved the best HCC prognostic system. Three year survival rates of patients in BCLC A, B, and C stages were respectively 81%, 56%, and 44% ($P < 0.01$), whereas all other tested staging systems did not prove a significant stratification ability. When all 715 HCC patients were considered, surgery proved to be a significant survival predictor in each BCLC stages A, B, and C.
Conclusions: BCLC staging showed the best interpretation of the survival distribution in a surgical HCC population. BCLC treatment algorithm should consider the role of surgery also for intermediate-advanced stages of liver disease.

O08 THE IMPACT OF HCC ON 3-YEAR SURVIVAL FOLLOWING LIVER TRANSPLANTATION IN THE MELD ERA

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Background: Prioritization of patients with liver cirrhosis and HCC in the waiting list (WL) for LT is still a critical issue.
Aim: The aim of this prospective study was to evaluate the impact of MELD and HCC at transplantation, on three-year patient and disease-free survival following LT.
Materials and methods: A mathematical algorithm based on seven patient qualitative and quantitative features (original MELD, CTP, UNOS, HCC, BMI, waiting time, age) was created by a dedicated software to prioritize patients in WL (named MELD-PAD) without extra-MELD points given to HCC patients. Mann-Whitney, Kaplan-Meier tests were used to assess survival.
Results: About 160 consecutive patients with liver cirrhosis (109 M/51 F, mean age 54 years) who underwent LT between June 2004 to February 2007 were enrolled in the study. HCC was diagnosed before LT in 50/160 (31.2%) patients. At LT, the mean CTP and original MELD were eight and 13 respectively in patients with HCC and nine and 17 respectively in patients without HCC ($P < 0.0001$). According to MELD at transplant, the 3-year patient survival was lower in patients with MELD >25 (70%) compared to patients with MELD 0–15 = 88% ($P = 0.03$), and MELD 16–25 = 93% ($P = 0.04$). No different 3-year patient survival and disease-free survival was observed between HCC patients with MELD 0–15 and MELD 16–25 at transplant. The 1,2, and 3 year patient survival in patients with and without HCC was 90%,98%,100% and 91%,98%,99% respectively (P = ns).
Conclusions: Patients with HCC are transplanted with a less severe liver disease compared to patients without HCC, probably justifying the MELD-extra points given for HCC in some centres. A worse outcome is reported in sicker patients transplanted with MELD>25, but survival is not affected by the concomitant presence of HCC.

O09 CALCINEURIN INHIBITORS AND HCC RECURRENCE AFTER LT

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HCC recurrence after LT can be favored by overexposure to cyclosporine. Tacrolimus is now the most widely used main immunosuppressant after LT; its

possible effect on HCC recurrence has never been investigated. We assessed the effect of tacrolimus on recurrence of hepatocellular carcinoma (HCC) after liver transplantation (LT) and compared it with that of the other calcineurin inhibitor, cyclosporine. 139 HCC patients who had LT were reviewed; 60 of them were administered tacrolimus and 79 cyclosporine. The exposure to the drugs was calculated with the trapezoidal rule in each patient, using blood levels measured after transplantation and compared with HCC recurrence together with several clinical and pathological risk factors. HCC recurred in 12 of 60 (20%) patients under tacrolimus versus 9 of 79 (11.4%) patients under cyclosporine; however, the proportion of poorly differentiated and more advanced tumors was significantly higher in the tacrolimus than in the cyclosporine group. Exposure to tacrolimus was 11.6ñ1.5 ng/mL in patients with recurrence and 8.6ñ1.7 in those without ($P < 0.001$). The optimal cut-off values of exposure identified with ROC analysis to categorize the risk of recurrence were 10 ng/mL for tacrolimus (AUC = 0.913) and 220 ng/mL for cyclosporine ((AUC = 0.752). In the tacrolimus group high drug exposure independently predicted recurrence ($P = 0.005$). Multivariate analysis including all patients (tacrolimus + cyclosporine) characterized higher exposure to immunosuppression ($P = 0.01$), alpha-fetoprotein levels ($P = 0.001$), tumor grading ($P = 0.009$) and microvascular invasion ($P = 0.04$) as independent predictors of HCC recurrence. Similarly to cyclosporine, overexposure to tacrolimus increases the risk of HCC recurrence after LT. Careful management of calcineurin inhibitors is recommended in HCC patients.

O10 RECURRENT cccDNA POSITIVE HEPATOCELLULAR CARCINOMA 10 YEARS AFTER LIVER TRANSPLANTATION

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A 60-year-old man underwent liver transplantation (LT) for HBV-related cirrhosis and hepatocellular carcinoma (HCC). He was on cyclosporine 2.5 mg/kg and received 5000 IU of immunoglobulins (HBIg) monthly, as recommended, since LT. No evidence of HBV reinfection occurred. Four years after transplant, the patient started lamivudine (100 mg/day) and HBIg were stopped, aimed to begin a vaccination program. After two vaccination cycles consisting of three intramuscular monthly doses of 40 mg of recombinant S vaccine the patient reached a protective anti-HBs titer and he was maintained without any antiviral treatment for the following 5 years. Booster vaccination doses were repeated occasionally, when the anti-HBs titer dropped below 70 UI/ml. HBsAg and HBV-DNA were repeatedly negative and the patient had an excellent quality of life. Ten years after LT and 5 years after the last vaccine dose, the patient developed a malignant liver tumor extended as an intracaval thrombus. A biopsy performed on the lesion confirmed the diagnosis of HCC. The presence of cccDNA on the same biopsy was evaluated using a sensitive quantitative real-time PCR and a plasmid DNA construct as positive control for quantification. Tumoral tissue was found to be positive for the presence of cccDNA, although staining negative for HBsAg and HBcAg immunohistochemistries. This unusual case confirms recent reports on the persistence of HBV genome as long as 10 years after transplant, despite successful immunoprophylaxis and persistently undetectable viremia post-LT. Although this patient already had HCC before transplant, development of HCC 10 year after transplant strongly suggests de novo tumorigenesis rather then recurrence of the original cancer. In this perspective, this case clearly indicates that persistence of cccDNA is associated with a continuous risk of HCC, which is not blunted by the absence of detectable viremia, nor by mounting a protective anti-HBs titer.

ELTR STUDIES

O11 "RESCUE ALLOCATION" IN LIVER TRANSPLANTATION WITHIN EUROTRANSPLANT: A SINGLE CENTRE EXPERIENCE

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Background: Organ shortage has driven many transplant centers to extend their criteria for organ acceptance. Graft allocation policies have been modified accordingly. This report focuses on the impact of applying the so-called 'rescue allocation' (RA) strategy in liver transplantation (LT) within the Eurotransplant (ET) area.

Methods: Liver grafts are considered for RA when the regular organ allocation is declined by at least three centers or is averted due to donor instability/unfavourable logistical reasons, thus entering a competitive or a single recipient rescue organ offer procedure, respectively. Between January 2004 and December 2006, we transplanted 85 RA livers after a total of 479 registered refusals within ET.

Results: Median cold ischemia time for RA grafts was 10 h (range: 4–17). The indications for LT were: hepatocellular carcinoma (HCC, 44%), chronic liver disease (54%), including viral chronic active hepatitis (15%), and acute liver failure (2%). The MELD score was 13 ± 7 (range: 6–40), and was 12 ± 7 for HCC (P = NS). There were three (3.5%) primary nonfunctions (PNF)in recipients of RA grafts. One-year patient and graft survival was 84% and 75%, respectively. There were no difference between the recipients of RA grafts versus recipients of regularly-allocated grafts regarding initial poor function, PNF, surgical complications, patient survival, and graft survival.

Conclusion: The use of RA organs within ET has increased the donor pool and transplantation dynamics with satisfying results. The unique possibility to match livers with recipients, which is left to the discretion of accepting center, should be judged according to the centers experience to decrease the waiting times for a timely rescue of organs/recipients while avoiding futile transplantations.

O12 PLATELET TRANSFUSIONS DURING LIVER TRANSPLANTATION ARE ASSOCIATED WITH INCREASED POST-OPERATIVE MORTALITY DUE TO ACUTE LUNG INJURY

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Background and Objective: Platelet transfusions have been shown to be a strong independent risk factor for graft failure and patient mortality after orthotopic liver transplantation. However, the mechanisms of patient death and graft failure as a result of platelet transfusions during liver transplantation are unknown. In this study these mechanisms were evaluated.

Methods: A series of 449 adult patients undergoing a first liver transplantation between 1989 and 2005 in our center were included in this study. Reasons for postoperative graft failure and patient death were assessed in patients who did or did not receive perioperative platelet transfusions.

Results: 90-days graft survival was significantly reduced in patients who received platelet transfusions during liver transplantation (75% vs. 90%, $P < 0.001$). The main reason for graft failure in these patients was patient death (20% vs. 5%, $P < 0.001$). 90-days patient survival was also significantly reduced in patients who received platelet transfusions during liver transplantation (79% vs. 94%, $P < 0.001$). This increased mortality could be attributed to an increased occurrence of acute lung injury, which was more frequent in patients receiving platelet transfusions during liver transplantation ($P = 0.004$). Interestingly, liver-related thrombotic complications were equally distributed between patients receiving platelet transfusions and patients who were not transfused.

Conclusions: This study confirms the detrimental effect of platelet transfusions in patients undergoing a liver transplantation. Patient death is the main reason for graft failure in patients who received platelet transfusions during liver transplantation. Acute lung injury is the main reason for patient death in these patients. These findings support previous calls for a cautious use of platelet transfusions in patients undergoing liver transplantation, because of serious adverse events and increased mortality due to acute lung injury.

O13 RISK FACTORS IN FAP TRANSPLANTATION

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Background: FAP disease is a common indication for liver transplantation in Portugal. Timing for the surgery is poorly understood, which prevents clear listing of these patients.

Methods: We analyse a series of FAP liver patients transplanted between November 1990 and March 2001 by several centers and followed in our clinic, and a control group that was not transplanted, and mortality analysed for age onset, length of disease, sex and year of beginning of symptoms.

Results: A series of 242 patients were transplanted(OLT) and compared to 319 patients that were not. Average age at OLT was 36.2y (M/F 34/39.3) with a mean duration of symptoms of 5y (M/F 4.8/5.3). Overall mortality 44(18%), male/female 16.9/20% (n.s.),with mean age of 40.1 for the patients hat died compared to 35.6 of survivors ($P < 0.001$). Mean duration of symptoms of survivors 4.6y compared favourably to non-survivors, 6.7y ($P < 0.001$). Early OLT 5y of symptoms (10% mortality), compared negatively to non-transplanted patients (0%), and compared equal for the following 5y period. After that OLT was significantly superior for survival (36% vs 78%).

Conclusions: Too early FAP patients OLT carries a negative survival impact, but advanced disease patients OLT has a very negative impact. The ideal window still needs further evaluation.

O14 QUALITY CONTROL OF THE EUROPEAN LIVER TRANSPLANT REGISTRY (ELTR). REPORT OF THE LIVING DONOR LIVER TRANSPLANTATION (LDLT) AUDIT

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The ELTR coordinating committee appointed an independent team of five auditors to check the reliability of data contained in ELTR. Centers to be visited and 10% of each center's files were selected at random. The rates of completeness and consistencies of LDLT files were compared to the cadaveric donors (CD) files, for 25 variables common to the two procedures. We studied also the yearly evolution of the quality of variables specific to LDLT (Relationship donor to recipient, early complications, min PT or max INR, max serum bilirubin, cause of reoperation, outcome and outcome date, and cause of death).

Results: Eight hundred twenty files from 35 centers were audited between May 2002 and December 2006. The rate of LDLT completeness was 95.7%, and the rate of consistency between the files and ELTR was 96.7%. These rates were not different from those of CD (95% and 97.8%, respectively). Nevertheless, while the quality of 2005 LDLT data was improved by comparison to 2004, a significant decrease in quality was observed in 2006.

Conclusion: The results of audit visits indicate that ELTR data are reliable. The visits should continue for their undoubted beneficial effect on the quality of ELTR. Before the visit, centers selected for audit show increased motivation associated with an improvement in data quality. The visits are not only used for audit purposes but are also an opportunity to discuss any isssue the center staff may have with the database and to explain how to complete the database more precisely. The meeting with the centers' staff is important for both sides and results in a better collaboration after the visit and the maintenance of a good quality of data in the audited centers.

O15 LIVER TRANSPLANTATION FOR ALCOHOLIC LIVER DISEASE IN ADULTS IN EUROPE

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Introduction: Alcohol related liver disease (ALD) is one of the commonest indications for LT. Although outcome after LT is favourable, there is still reluctance to refer these patients for formal assessment.

Material and methods: All patients transplanted between 1988–2005 in ELTR registry were evaluated. We compared those transplanted for ALD alone with three other groups: Hepatitis Viral Infection (VIR), Cryptogenic Cirrhosis (CRYP), and ALD plus VIR (ALD-VIR). All the ELTR variables related to donor, graft and recipient were analysed. All causes of death/graft failure were analysed, and correlated with discrete variables using uni-multivariate analysis.

Results: Data were available from 10.009 ALD, 11.114 VIR, 1.490 ALD-VIR, and 2.700 cryptogenic transplants. UNOS status 1 or 2 at transplant was reported in 24% of CRYP patients significantly higher than in ALD, VIR and ALD-VIR, but similar to HCV-HBV patients. Graft survival at 1, 3, 5 and 10 years from transplantation was 83%, 74%, 69%, 54% respectively in ALD patients similar to VIR and CRYP patients, but 1 year survival in ALD+HCV group was lower than ALD alone group (80%vs. 86%, $P < 0.05$). Multivariate analysis confirmed the significant role of severity of liver disease at time of transplantation expressed by UNOS status (RR 1.46, $P < 0.0001$) and donor characteristics (donor age > 60 years, RR 1.34; ischaemia time 12 h RR 1.16). Interestingly, non-alcoholic etiology of liver disease was a significant risk factor both at uni-multivariate analysis (RR 1.14). De novo tumours were a major cause of graft failure/death in the ALD (11%) and ALD-VIR groups (14%).

Conclusions: LT for ALD cirrhosis show excellent results compared to other etiologies of liver disease. Attention should be paid to HCV co-infection which is shown to eliminate this advantage and increase long term mortality. Screening and prevention for de novo tumours after transplant is essential and could provide better results in term of survival in this group of patients.

O16 15-YEAR EVOLUTION OF LIVER TRANSPLANTATION IN EUROPE

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From May 1968 to December 2005 the ELTR has accumulated data on 68776 Liver Transplantations (LT) at 137 centers (23 countries). We analysed the evolution of main indications, surgery techniques and results during the last 15 years period comparing three era: (1) 1990–1995; (2) 1995–2000 and (3) 2000–2005. Evolution of indication and surgical technique Era (1) Era (2) Era (3) $n = 11883$ $n = 17939$ $n = 26824$ Indication FUHE 1238 (10%) 1531 (8%) 1989 (7%) ALC 1841 (15%) 3561 (20%) 5402 (20%) VCC 1351 (11%) 2810 (16%) 4265 (16%) VBC 891 (7%) 1139 (6%) 547 (6%) HCC 1042 (9%) 2085 (12%) 4191 (16%) Surgical technique CFS 11094 (93%) 16174 (90%) 22840 (85%) Split 211 (2%) 790 (4%) 1689 (6%) LDLT 81 (0.7%) 299 (2%) 1484 (6%) FUHE: fulminant hepatitis; ALC: alcoholic cirrhosis; VCC: virus C cirrhosis; VBC: virus C cirrhosis; HCC: hepatocellular carcinoma; CFS: cadaveric full size graft; Split: split liver; LDLT: living donor liver transplantation. Patient survival 90–95 95–2000 2000–05 *P* value FUHE 1-year 62% 68% 75% < 0.001 5-year 54% 61% 65% ALD 1-year 81% 86% 88% < 0.001 5-year 68% 73% 71% VHC 1-year 81% 84% 87% 0.167 5-year 64% 67% 62% VHB 1-year 81% 88% 91% < 0.001 5-year 68% 79% 80% HCC 1-year 75% 84% 89% < 0.001 5-year 46% 57% 60% CFS 1-year 78% 84% 87% < 0.001 5-year 64% 71% 69% Split 1-year 64% 77% 85% < 0.001 5-year 55% 70% 73% LDLT 1-year 73% 86% 86% 0.078 5-year 69% 78% 67% With regard to the indication, the results show an increase of ALC and VCC, as of HCC. Regarding the surgical technique, split and LDLT have increased to represent currently each one 6% of all LT. Concerning the outcome, LT is increasingly providing a survival exceeding 80% at 1 year. Survival has significantly improved for all indications including HCC with however the exception of VCC. The same improvement in survival was observed in CFS and split transplants but not for LDLT. In conclusion, improved patient management and surgical technique, and more effective immunosuppression still lead to significant improvements in the outcome of LT.

MY WORST CASE

O17 LIVER RETRANSPLANTATION IN A PEDIATRIC PATIENT: THREE GRAFTS IN A THREE YEAR OLD CHILD. WHAT HAPPENED?

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The patient was a 3 years old male, who had been previously diagnosed of biliary atresia. A liver transplantation was performed in May 1988, using a whole cadaveric graft. Early postoperative course was uneventful, and he received Cyclosporin, ATGAM and steroids as immunosupressive agents. On the 15th postoperative day an acute rejection developed, and afterwards, graft function worsened. Abdominal ultrasonud and CT-Scan showed intrahepatic abscesses and angiography revealed hepatic artery thrombosis, therefore the patient was accepted for retransplantation. On 28th postoperative day, a second liver transplantation was performed, a partial graft including segments I-IV was implanted. An iliac artery graft

from the donor was interposed between infraceliac aorta in the recipient and celic trunk in the graft. During postoperative period, the patient developed two acute rejection episodes, which were treated with steroids; he was also diagnosed of liver abscesses that required surgical drainage. As graft function worsened, a percutaneous liver biopsy was performed, showing a chronic rejection and hepatitis, therefore a third liver transplantation was indicated. In November 1988 a third liver transplantation was performed, using a whole graft. During laparotomy, an aortic aneurism located at the arterial anastomosis, 2.5×2 cm size was diagnosed. Hepatic artery reconstruction was a primary anastomosis without graft interposition (celiac trunk in the recipient and in the liver graft). Postoperative period coursed with the development of an acute rejection with good response to steroids.The patient developed a HVC infection, with normal graft function. AllvAngio-MRI performed have shown a superior mesenteric artery aneurism, which has not changed in size since last liver transplantation. He is now 21 years old, and shows normal liver function.

O18 SURGICAL COMPLICATIONS AFTER LIVER TRANSPLANTATION AND WHIPPLE

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The patient, a 46 year old male, received a liver transplant due to primary sclerosing cholangitis (PSC) in February 2004. Preoperatively bile duct epithelial dysplasia was found in several brush cytological specimens obtained during ERC.The transplant procedure was uncomplicated. Postoperatively cholangiocarcinoma was diagnosed by conventional histology in the choledochus at the plane of resection close to the pancreas while frozen sections taken peroperatively were negative. After 3 months a radical (R0) Whipple's operation was performed with no signs of carcinoma at the plane of resection. Leakage from the pancreatico-enteric anastomosis was diagnosed on the fourth postoperative day and reoperation was subsequently performed with Neopren-occlusion of the pancreatic duct. Later fistula from the stomach developed in addition to multiple intraabdominal abscesses in the remaining pancreatic tail and in the liver graft. Totally eight reoperations was performed during 7 months before the patient was discharged from the hospital. The patient is now in good clinical condition without recurrence of cholangiocarcinoma. Simultaneous liver transplantation and Whipple's resection is possible if the diagnosis of cholangiocarcinoma in PSC patients is established pretransplant. A review of the literature and the Norwegian experience will be presented.

O19 SUCCESSFUL LIVER TRANSPLANTATION IN A PATIENT WITH HBV-RELATED CIRRHOSIS AND PRIMARY YOLK SAC TUMOR OF THE LIVER

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Background: Extragonadal yolk sac tumors (YSTs) are uncommon and primary YST of the liver is extremely rare, with only few reported cases in the literature.
Case Report: A 64-year-old man with HBV-related cirrhosis presented with a liver nodule measuring 2.8 cm revealed by a routine ultrasound and concomitant increased alpha-fetoprotein (AFP) (> 400 UI/l). Contrast-enhanced CT was suggestive of hepatocellular carcinoma (HCC) and the patient underwent laser ablation procedure without complications. Five months later, because of AFP raised up to 1600 UI/l, ultrasonography and abdominal CT were repeated, showing an increased diameter of liver nodule, measuring 3.8 cm. The patient underwent down-staged transarterial chemoembolization (TACE) and then was entered into the active liver transplant (LT) list. Lamivudine was concomitantly started before LT to minimize the risk of HBV recurrence. The patient underwent LT with high priority, showing HBV-DNA serum levels < 103 log/copies at time of surgery. After LT the patient received conventional immunoprophylaxis against HBV recurrence. Pathological analysis performed on the explanted liver showed, instead of the suspected HCC, a picture consistent with an hepatic yolk sac tumor, with the presence of typical ''Schiller-Duval bodies''. The first 12 months of post-operative follow-up were excellent, with no evidence of tumour recurrence. All liver function tests and biochemical analyses were normal.
Conclusion: While the morphologic features of hepatic YST have been extensively described in the literature, this rare neoplasm still pose significant diagnostic problems. Distinguishing preoperatively between HCC and primary YST of the liver has important therapeutic implications, both in terms of chemotherapy and surgical approach. Despite the diagnosis of YST in our case was unexpected and possible only on the explanted liver, LT appeared to be a good therapeutic opportunity, not yet recognized.

O20 REFRACTORY GRAFT VERSUS HOST DISEASE FOLLOWING INTESTINAL TRANSPLANTATION

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Background: Graft-versus-host disease (GVHD) occurs following intestinal transplantation (ITx) with an incidence of 8–10%. GVHD usually responds to increased immune suppression.
Case: A 5-year-old boy received isolated ITx for intestinal failure secondary to gastroschisis associated with multiple bowel atresias. He had an unclassified immunodeficiency characterised by hypogammaglobulinaemia, and persistent T-cell lymphopenia with normal B-cell and NK cell numbers There was a normal response to PHA, PWM and OKT3 stimulation. Immunosuppression regime included primary induction with basiliximab (days 0.4) and immunosuppression with Tacrolimus and prednisolone. He was discharged on full enteral feeds 4 weeks after ITx. He had abnormal liver function tests 10 weeks after ITx, which were thought to be due to a viral infection. Three months post-ISBTx he developed a skin rash, consistent with GVHD, and was confirmed on skin, native liver and native gut biopsies. Initially responsive to steroid, the GVHD progressed to grade IV skin involvement with liver dysfunction and neutropenia and was resistant to further treatment with high dose steroids. A regime of anti-CD25 monoclonal antibodies and anti-tumour necrosis factor blockade was ineffective. Unfortunately he died after developing a cerebellar abscess.
Discussion: Pre-existent immunodeficiency was probably the main factor in the rapid progression and development of refractory GVHD. Primary induction with anti-CD-3 (OKT3)/anti-CD52 should be considered in the immunosuppression regime of pre-existent immunodeficient patients. In refractory GVHD, treatment strategies including use of anti-IL2-receptor, anti-TNF-alpha and OKT3 should probably be used earlier.The role of bone marrow transplantation prior to ITx should be explored in individuals with pre-existing immunodeficiency.
Conclusion: Pre-existent immunodeficiency needs to be characterised before considering any child for ITx. A high index of suspicion and aggressive early treatment is essential for effective management of GVHD.

O21 EARLY PTLD OF THE LIVER AND BILE DUCT AFTER LIVER TRANSPLANTATION FOR HCV RELATED CIRRHOSIS

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Introduction: Post-Transplant Lymphoproliferative Disease (PTLD) after liver transplantation (OLTx) is reported with an incidence ranging between 1 and 10%. Interval from transplant goes from months to years. Case Report: A 38 year old man (EBV EBNA positive, EBV VCA IgM negative, CMV IgG positive CMV IgM negative) underwent OLTx, with a graft from a 34 year old male donor (EBV IgG and CMV IgG negative), for an HCV and alcohol related cirrhosis. On the 2nd postoperative (p.o.) day an ERCP with a naso-biliary tube (NBT) placement was performed for an anastomotic biliary fistula. On 14th p.o. day, due to an increase of the gGT, we performed a liver biopsy that showed a moderate-severe acute rejection (RAI 6/9). A bolus of steroid was administered. The day after, a cholangiography showed a contrast blush at the site of biliary anastomosis without showing the biliary tree. Patient underwent a surgical exploration. Intraoperatively, the wall of the graft?s bile duct was found to be thickened. A sample was taken for histology as well as a liver biopsy and an hepatico-jejunostomy was performed. Histology showed a polymorph PTLD of both the liver and the bile duct. EBV DNA by PCR was 362 Geq/100.000 MN. Genomic research of EBV on a liver sample was negative. Treatment with Rituximab 700 mg once a week for 4 weeks led to a complete stable remission of the PTLD as shown by several liver biopsy. Tacrolimus trough level was maintained between 5 and 7 ng/ml. After 2.7 years patient is alive and well in spite of a recurrence of Hep C.
Conclusion: PTLD can occur even very early after OLTx. Using only routine stained biopsy with Haematossillin-Eosin is unsatisfactory and immunohystochemical lymphocytes phenotyping is a mandatory diagnostic tool.

HEPATITIS C AND QUALITY OF LIFE

O22 HEPATITIS C RECURRENCE: SPLIT LIVER VERSUS WHOLE SIZE GRAFT

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Introduction: Hepatitis C virus is the most common indication for liver transplantation. Recently, reports have suggested that partial liver transplants, in particularly living donor, may be associated with an increased risk for HCV recurrence. The aim of this study is to compare the recurrence of HCV after conventional Split Liver Transplantation (A/P SLT) using right lobe versus Whole Liver Transplantation (WLT).

Patients and methods: From June 1998 to December 2004, in our institution 265 liver transplantation were performed. Among these, 195 (73.58%) were WLT, 59 (22.27%) A/P SLT and 11 (4.15%) Adult to Adult SLT. The HCV recipients enrolled were 80 (30.18%).Among these, 22 (27.50%) received a right liver graft of SLT A/P and 58 (72.50%) received WLT. In the SLT group, the mean of Graft Recipient Weight Ratio (GRWR) was 1.79 ± 0.44 (95% CI: from 1.58 to 1.99).

Results: In our experience, there is no difference in HCV recurrence rate between WLT and SLT groups after a follow-up period of 56.41 months. Reinfection (HCV-RNA title) occurred in 51 recipients (87.93%) in WLT group and in 19 recipients (86.36%) in SLT group ($P = 0.849$). Histologic recurrence in the two groups was 82.75% for WLT and 77.27% for SLT ($P = 0.538$). Hepatic failure for HCV hepatitis recurrence was observed in 22.91% of SLT group vs. 29.41% of WLT population ($P = 0.592$). Retransplantation for hepatic failure post HCV recurrence was needed only for one recipient (4.5%) in SLT group ($P = 0.098$). After comparison of continuous variables between WLT versus SLT patients, a statistical significance ($P = 0.0088$) was found only for shorter cold ischemic times in WL group.

Conclusions: In our experience there is no difference in HCV recurrence rate between WLT and SLT groups after a follow-up period of 56.41 months.

O23 FIBROSIS PROGRESSION ON PROTOCOL LIVER BIOPSIES IN PATIENTS WITH OR WITHOUT ANTIVIRAL TREATMENT FOR HCV RECURRENCE FOLLOWING LIVER TRANSPLANTATION (LT)

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Background: HCV-recurrence following LT is universal and it frequently leads to progressive fibrosis.

Aim: Evaluate the progression of fibrosis on protocol liver biopsies in patients transplanted for HCV cirrhosis.

Materials and Methods: Patients transplanted for HCV cirrhosis who underwent protocol liver biopsies consecutively performed at our Gastroenterology Unit at 6, 12 and 24 months post-LT were included. Histological stage of fibrosis was evaluated according to Scheuer (S 0–4). The fibrosis progression rate (FPR), expressed as fibrosis unit per month (FU/months), was compared over two periods (6–12 months; 12–24 months). In patients selected for antiviral therapy, sustained viral response (SVR) was assessed at 6 months.

Results: 54 patients (mean age 55 ± 7 years, f/u 24–95 months) underwent three serial liver biopsies at 6, 12 and 24 months post-LT. S 3 and 4 was reported in 3.7%, 7.4% and 13% of patients and S (mean value) was 1.17, 1.20, 1.70 respectively; FPR was 0.005 FU/mo (6–12 months) and 0.0416 FU/mo (12–24 months) post-LT. 9/54 (16.7%) patients underwent antiviral therapy, SVR in 4/9 (44.4%) patients. At 6, 12 and 24 months post-LT, S was 1.44, 1.61, 2.11 in treated and 1.12, 1.12, 1.61 in non treated patients ($P = 0.003$); FPR was 0.0283 FU/months and 0.0416 FU/months in treated and 0 and 0.0416 in non treated patients at 6–12 and 12–24 months respectively. According to SVR, at 6, 12 and 24 months post-LT, S 3 and 4 was 0%, 0%, 0%, in SVR+ and 0%, 20%, 40% in SVR- patients, S was 1.25, 1.25 and 1.5 in SVR+ and 1.6, 1.9 and 2.6 in SVR- patients, FPR was 0.00 and 0.02 for SVR+ and 0.05 and 0.06 for SVR- at 6–12 and 12–24 months post-LT respectively (P = NS).

Conclusion: The progression of fibrosis due to HCV recurrence post-LT is accelerated between 12 and 24 months compared to the early period post-LT, and a lower stage is seen in patients not selected for antiviral therapy. In patients with SVR-, pre-cirrhosis or cirrhosis is seen in 40% of cases at 2 years post-LT.

O24 STEROIDS AND HCV RECURRENCE AFTER LT: RESULTS OF A PROSPECTIVE TRIAL HEPATITIS C

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To assess the effect of long-term maintenance of steroids on HCV recurrence after liver transplantation (LT), that is still controversial, a prospective multicentre trial was carried out at the Centres of Bologna and Padua, Italy. From September 2002, 47 HCV positive LT recipients were randomized to receive two different steroid schedules in association with tacrolimus: group A: rapid tapering and withdrawal 91 days after LT group B: slow tapering and withdrawal 25 months after LT Thirty-nine patients were assessable: 23 in group A and 16 in group B. Donor and recipient characteristics were similar in the two groups. Median follow-up was 841 days (130–1376). One hundred liver biopsies were performed, and every patient had a 12-month biopsy. Twenty-two out of 23 (95.65%) patients in group A and 15 out of 16 (93.75%) in group B had histologically-confirmed HCV recurrence. Twelve-month histology showed advanced fibrosis (score 3 or 4) in 42.1% of the patients in group A vs. 7.6% in group B ($P = 0.03$). One- and 2-year advanced fibrosis-free survival were 65.2 and 60.8 in group A and 93.7% in group B ($P = 0.03$ and $= 0.02$ respectively). Slowly tapering off steroids reduced the progression of recurrent hepatitis C after LT.

O25 CADAVER AND LIVING DONOR SPLIT LIVER TRANSPLANTATION FROM CADAVER AND LIVING DONOR IN HCV+ RECIPIENTS: A MULTICENTER EXPERIENCE OF AN ITALIAN TRANSPLANT AGENCY

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Objective: Partial liver transplant has been suggested to be a risk factor for graft failure for hepatitis C recurrence but the vast majority of living donor liver transplantation recipients are HCV-positive. In our organization a specific split liver transplant program from cadaveric donor (CD) started in 1997 while living donor (LD) liver transplantation is active since 2001. Purpose of this work was to compare the outcome of partial transplant from CD or LD in HCV-positive or negative recipients.

Patients and Methods: All HCV-positive or negative adult recipients transplanted with a partial liver from CD or LD from 2000 to 2006 in the centers belonging to our area were included. 3-year graft survival rates were calculated.

Results: From 2000 to 2006, 95 HCV-positive and 129 HCV-negative recipients underwent a partial liver transplant from CD while 28 HCV-positive and 23 HCV-negative underwent a transplant from LD. 3-year graft survival was not statistically different for HCV positive or negative recipients either in CD transplants (71% vs. 74%, respectively) or in LD transplants (66% vs. 72%, respectively). No statistically significant difference was observed among CD and LD HCV-positive or HCV-negative grafts.

Conclusions: In our area partial liver transplant does not negatively affect the short-term outcome of HCV positive recipients. A longer follow-up is needed to confirm these results.

O26 SEXUAL DYSFUNCTION IN MALE BEFORE AND AFTER LIVER TRANSPLANTATION (LT)

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Background: Sexual dysfunction (SD) is highly prevalent in men with liver cirrhosis and few data are reported on the impact of LT on SD. The aim was asses the prevalence of SD in men before and after LT.
Methods: Thirty-five cirrhotic and 44 LT patients were enrolled in a cross sectional study. CPT and sexual hormones (17b-estradiol, PRL, testosterone total and free, SHBG, 17-OHprogesteron) were assessed in all patients. Two validated questionnaires [International Index Erectile Function (IIEF), Beck Depression Inventory (BDI) were used. Statistical analysis: Students t test, data expressed by mean ± SE.
Results: The patients have alcohol-related disease in 57% of cirrhotics and 39% of LT patients. The two groups were age-comparable. A better IIEF score was seen in transplanted versus cirrhotic patients ($P = 0.04$). 80% of cirrhotics and 65% of LT patients presented erectile dysfunctions (ED). The age of cirrhotics with ED was significantly higher compared to patients with no ED ($P < 0.05$), whereas no difference was seen in transplanted. A better BDI score was seen in transplanted versus cirrhotic patients ($P = 0.01$); ED was worse in depressed versus non depressed transplanted patients ($P < 0.05$). The hormone status in cirrhotics demonstrate higher as per 17b-estradiol (156.4 ± 22.6 pmol/l), PRL (17.1 ± 2.9 pg/ml), SHBG (76.9 ± 12.4 nmol/l) and lower DHEAS (1.3 ± 0.6 umol/l); whereas lower DHEAS (1.6 ± 0.5 umol/l), and higher 17b-estradiol (141.9 ± 18.8 pmol/l) were seen in transplanted patients compared to normal values. PRL and SHBG were significantly higher in cirrhotic versus transplanted patients ($P < 0.01$). SD did not correlate with sexual hormones, CPT and etiology of liver disease.
Conclusion: SD is confirmed in men with liver cirrhosis, in whom sexual hormone impairment is evident; the dysfunction is more evident in the older. In liver transplanted patients, unexpectedly, SD is also present, associated with depression, but not with hormonal status or age.

O27 EVALUATION OF PREDICTIVE FACTORS FOR ADHERENCE TO MEDICAL REGIMEN IN PATIENTS BEFORE AND AFTER LIVER TRANSPLANTATION

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Background: Adherence to medical prescription in patients who underwent liver transplantation (LT) is important to ensure patients and grafts short and long term survival.
Aim: To evaluate adherence to medical regimen in a longitudinal study in patients before and after LT.
Materials and methods: 134 patients with liver cirrhosis were enrolled. Liver transplanted patients were evaluated at 6 and 12 months after LT. Before and after LT patients underwent questionnaire on adherence, locus of control test, blood test. Child-Pugh score and MELD were assessed.
Results: before LT 43.3%, 11.9% and 13.4% of patients were non adherent to regular therapy, to outpatient visit and to blood test respectively. Non adherent patients, compared to patients with good adherence, were younger (mean 52.1 vs. 54 ys, p 0.09), married (96.6% vs. 71.3%, p 0.06) and with external LOC (66.7% vs. 40.5%, p 0.04). 23 patients underwent LT. At 6 months after LT 40% of patients presented poor adherence. These patients, compared to patients with good adherence, presented better MELD at time of transplantation (mean 12.7 vs. 16.9, p. 0.04), were drinking alcohol (33.3% vs. 5.9%, p 0.001) and referred > 3 side effects of immunosuppression therapy (66.7% vs. 23.5%, p 0.02). At 12 months after LT 90% of patients were non adherent to medical regimen. These patients, compared to patients with good adherence, were younger (mean 55.3 vs. 61, p 0.05), presented better MELD at time of transplantation (mean 13.3 vs. 17.5), were drinking alcohol (50% vs. 0%, p 0.001) after liver transplantation and referred > 3 side effects of immunosuppression therapy (83.3% vs. 50%, p 0.02). Poor adherence after LT was correlated with poor adherence before transplant in 50% of cases.
Conclusions: 40% of patients before LT present poor adherence. Adherence improves at 6 months and worsens at 12 months after LT. Poor adherence after LT is related to younger age, better clinical condition before LT, side effects of immunosoppressive therapy and poor adherence before LT. Therefore, educational programs are needed.

SMALL BOWEL AND PEDIATRIC TRANSPLANTATION

O28 RISK FACTORS FOR MORTALITY ON THE WAITING LIST FOR COMBINED LIVER AND SMALL BOWEL TRANSPLANTATION

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Since 1990, 242 children have been assessed for intestinal transplant and 92 have been recommended for transplantation, but 30 children died on the transplant waiting list.
Aim: To review risk factors for death on the waiting list.
Methods: 30 children (Group I) who died on transplant waiting list for CLSBTx, were compared with 32 (GROUP 2) received CLSBTx. Statistical analysis was performed by using unpaired T test or Fischers exact test.
Results: The median age at assessment was 9.1 months and 36.5 months in Group 1 and Group 2 respectively ($P = 0.018$). The commonest diagnosis was short bowel syndrome (24 in Group 1 and 16 in Group 2) followed by pseudo-obstruction (Six in Group I and 16 in Group 2). The median bilirubin, plaelet count at assessment were 360 micromol/dl, 79×10^9 in Group 1 and 240 micromol/dl, 115×10^9 in Group 2($P = 0.0001$, $P = 0.035$). The median waiting time in Group 1 was 65 days as compared to 106 days in group 2(not significant).
Discussion: Children who died before transplant were younger, the majority had short bowel syndrome, 66% had platelets below 100×109 and only two had a bilirubin less than 200 mg/dl at the time of assessment.
Conclusion: Infants aged around 9 months with short bowel syndrome (SBS) and evolving intestinal failure associated liver disease (IFALD) are high risk group. Previously we have recommended that patients developing IFALD should be referred with a bilirubin of 100 mmol/l. In view of the rapid deterioration in babies with SBS, shown by the short duration of survival on the waiting list of 65 days, we would recommend that infants with short bowel syndrome be discussed with a multi-disciplinary team and referred at an earlier stage i.e. when the serum bilirubin remains above 70 mmol/l especially in the context of a falling platelet count.

O29 A NEW NATIONAL PAEDIATRIC DONOR ORGAN ALLOCATION POLICY FOR CHILDREN WAITING FOR INTESTINAL TRANSPLANTATION

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Introduction: Graft availability remains a problem in paediatric small bowel transplantation, with a majority of children waiting being <10 kg in weight. In November 2004, UK children listed for intestinal transplantation were prioritised nationally to receive paediatric donor organs to improve donor availability for intestinal transplantation (IT). We aimed to evaluate the effect of this change and its impact on the intestine recipient population.
Methods: Data regarding paediatric donor organ availability and allocation were accessed from the National Transplant database. Recipient demographics and outcomes were recorded from the Liver Unit database. Between 2001 and 2006 there was a total of 243 paediatric donors in the UK (15; 6.2% were non heart beating donors and were excluded). Of the 228 eligible donors 39 (16%) were allocated for emergency super-urgent liver (SUL) candidates. A total of six isolated intestine and 21 liver-intestine transplants were performed.
Results: Since January 2001 there was a progressive reduction in overall paediatric organ donation. Increasing awareness about IT has resulted in a significant increase in number of small bowel organs being offered (19.5% vs. 71.8%), although this has been associated with an increase in referrals for transplantation (20.2 vs. 33.6 mean grafts/year). Despite an increase in number of IT being performed (2.6 vs. 7.7 mean transplants/year), waiting list mortality still remains high in smaller children (66% of the list). No mortality was observed in larger children and in candidates for isolated IT. Median waiting time for isolated IT was longer than for liver-intestine transplant.
Conclusion: The new prioritisation of national paediatric donor allocation favouring IT has resulted in a significant increased number of procedures, without an impact on waiting list mortality, especially for small children. Reduction in the waiting time for isolated IT could be achieved by improved utilisation of paediatric donors allocated for SUL transplantation.

O30 CHRONIC REJECTION AFTER PAEDIATRIC INTESTINAL TRANSPLANTATION: A UK SINGLE CENTRE EXPERIENCE

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Introduction: Chronic rejection (CR) after intestinal transplantation (IT) is reported from busier programmes with increasing number of long-term survivors. We reported a single centre experience with CR in IT.

Methods: We did a retrospective review of the Liver Unit database to identify the children who developed CR. These details were recorded: type of transplant, immunosuppression, pre-disposing factors, presenting symptoms, range of diagnostic tools and outcomes.

Results: Forty nine IT were performed in 46 children (40 combined liver-intestine and nine isolated intestine transplantation). Four children (8.2%) developed CR after a median time of 42.2 months. In case 1 following the episode of severe rejection with Rotavirus gastroenteritis there was a persistent high stomal output and an intermittently low serum albumin with weight loss. The child presented with symptoms of sub-acute intestinal obstruction and the diagnosis was made on laparotomy and confirmed on histopathology of the removed graft. In case 2, the child had unexplained symptoms of diarrhea and weight loss. PTLD was excluded and the diagnosis was made on CT abdomen with oral contrast and later confirmed on histopathology of the explanted graft. Case 3 mirrored the clinical course of case 1 following the episode of severe rejection secondary to severe Rotavirus gastroenteritis, but was in addition associated with development of unexplained multiple intrahepatic strictures and diffuse thickening of the whole small bowel on CT abdomen. Case 4 had repeated episodes of unexplained apneas followed by unexplained diarrhea and presented with sub-acute intestinal obstruction with the diagnosis made on laparotomy. Three patients were re-transplanted, 1 is still on the waiting list.

Conclusions: Following episodes of severe rejection and severe viral enteritis, CR should be suspected in individuals with unexplained diarrhea and persistent low albumin and CT abdomen with oral contrast may be useful to establish an early diagnosis.

O31 PRE-EMPTIVE MANAGEMENT OF ANASTOMOTIC BILIARY STENOSES AFTER PEDIATRIC LIVER TRANSPLANTATION

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Introduction: Biliary stenoses are a frequent complication after liver transplantation (OLTx). Their incidence is higher with partial liver grafts. An aggressive approach in their management has been pursued at our Center.

Methods: Between October 1997 and November 2006, 294 children (median age 1.37 years; weight 9 kg) underwent a primary OLTx (64 whole liver, 230 split liver grafts). A hepatico-jejunostomy was performed in 274 (93%) cases and a duct-to-duct anastomosis in 20 (7%). A biliary stricture was suspected in any case of even slight increase in liver enzymes, even in absence of jaundice, itching or bile duct dilatation at US scan. A liver biopsy was performed in all these cases and when it showed signs of mechanical cholestasis, a percutaneous cholangiogram (PTC) was performed. Any stricture, even mild, was treated by balloon dilatation and temporary stent, repeated until normalization of the liver function test. In case of unsuccess of repeated procedures a surgical re-do of the anastomosis was performed.

Results: 58 biliary stenoses occurred in 57 children. 97% of the stenoses developed in the recipients of a split liver graft. Their incidence was 20%, 33 (57%) occurred early (within 90 days) and 25 (43%) late (after 90 days). Only nine cases were jaundiced. A duct dilatation was found at a US scan in 40 cases (68%). 38 children were asymptomatic. All the patients underwent PTC and balloon dilatation that was successful in 44 cases, a re-do of the anastomosis was required in 14 cases. Patient and graft survival among the 57 children was 98%/91% and 98%/89% respectively at 1 and 5 years. No graft was lost for an isolated biliary stricture after a median follow-up of 5.4 years.

Conclusion: A pre-emptive diagnostic and therapeutic approach allows detecting a number of asymptomatic strictures eliminating any impact on graft survival.

O32 RETRANSPLANTATION RATE IS SIGNIFICANTLY HIGHER IN DONATION AFTER CARDIAC DEATH AND INDEPENDENT FROM THE RECIPIENT STATUS

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Introduction: Donation after cardiac death (DCD) has re-emerged as a potential way to increase organ supply for transplantation. We retrospectively reviewed our experience comparing DCD liver transplants with those performed with donors after brain death (DBD).

Methods: From October 2003 until August 2007, 15 controlled DCD LT and 119 DBD LT were performed in our institution. Splits, LDLT, combined, pediatric LT and retransplantations were excluded from this analysis.

Results: Three year overall graft survival was 53% in DCD recipients vs. 83% in DBD ($P = 0.003$), while patient survival was 70% vs. 84% respectively (0.019). Primary Non Function (PNF), Ischemic Type Biliary Lesions (ITBL), and retransplantation rate was significantly higher in DCD recipient: 33% vs. 7.5% ($P = 0.01$), 27% vs. 1.6% ($P = 0.01$), 20% vs. 1.6% ($P = 0.01$). PNF was 0% and 40% in self procured and shipped DCD grafts respectively. The worst outcome occurred when the graft were shipped by another team or patient-oriented (10). Covariates which individually influenced DCD graft failure included recipient warm ischemia time (rWIT), cumulative WIT (cWIT, donor WIT plus rWIT) and early post-transplant cholestasis. Retransplantation rate and PNF were higher in DCD independently from recipient status.

Conclusion: DCD is still inferior to DBD due to higher rate of PNF and ITBL resulting in more need for retransplantation. Center oriented allocation, self procurement of the graft and shortening of the cWIT may improve the results.