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developed HBV seroconversion. Overall, survival in this small cohort was no different than that for all patients transplanted at our center during this time period. Conclusion: Transplantation of hearts from HBcAb+ donors is associated with a low HBV transmission risk. Transplantation of hearts from donors with intracranial tumors is not associated with tumor transmission. Use of hearts from these marginal donors should be considered safe and may help to augment the available donor pool.

1137-63

Recent Outcomes in Cardiac Transplant Patients Receiving Hepatitis C Allografts: A Single Center Experience

Jaymica P. Patel, Jignesh K. Patel, Brandy T. Cole, Maria Espejo Vassilakis, Lynda Kessler, Bernard Kubak, Jon A. Kobashigawa, The David Geffen School of Medicine at UCLA, Los Angeles, CA

Background: Hepatitis C infection in the non-immunocompromised population is a chronic progressive disease with clinical manifestations after several years. The longterm impact of hepatitis C following heart transplantation remains unclear.

Methods: Medical records pertaining to heart transplant recipients receiving allografts from hepatitis C positive donors between August 1991 and August 2002 were reviewed retrospectively. 23 patients were identified as having received allografts from hepatitis C positive donors. Of these, 7 recipients were hepatitis C positive prior to transplantation and 9 patients were Status I listing. Overall survival was determined. The presence of diabetes mellitus, hypertension, renal function, post-transplant albumin, donor age, pretransplant hepatitis C status and type of immunosuppression used were determined to assess possible contribution to outcomes.

Results: Overall survival in patients receiving hepatitis C positive allografts was 57% at one year and 17% at 5 years. This compared to an overall one-year survival of 82% at one year and 70% at 5 years for all adult heart transplants. Cause of death was due to multisystem organ failure and/or sepsis (5), liver failure (2), transplant coronary disease (2), unexplained sudden death (2), malignancy (2), rejection (1), and pulmonary embolus (1). By multivariate analysis and logistic regression, the only significant predictor for mortality was low post-transplant albumin (p<0.017). Pre-transplant hepatitis C status or type of immunosuppression used did not significantly affect outcome.

Conclusion: Patients receiving hepatitis C positive cardiac allografts have a significantly worse outcome when compared to the outcome for all adult heart transplants in our institution. Their mortality compares to patients with severe heart failure. These data suggest that the use of cardiac allografts from hepatitis C positive donors should be restricted to critically ill patients awaiting transplantation for whom other treatment modalities are unsuitable.

1137-64

The Vagrancy of B-Type Natriuretic Peptide Levels in **Heart Transplant Recipients Receiving Tacrolimus**

Daniel Cruz, Maria Espejo Vassilakis, Jignesh K. Patel, Alan Garfinkel, Gregg Fonarow, Jaime Moriguchi, Jon A. Kobashigawa, The David Geffen School of Medicine at UCLA, Los Angeles, CA

Background: Tacrolimus (FK506) is a potent immunosuppressant often used in place of cyclosporine (CSA) in heart transplant (HT) patients. Prior studies on HT patients on CSA have demonstrated that B-type natriuretic peptide (BNP) levels are elevated and even continue to rise after HT. However, studies utilizing BNP as a marker of filling pressures and rejection have been discordant. It is unknown if the same BNP patterns exist for HT recipients receiving FK506. Methods: We retrospectively analyzed BNP levels in 49 patients on CSA or FK506 who were transplanted between August 2001 and May 2002. 300 BNP levels were drawn at the time that hemodynamic measurements and ventricular biopsies were performed. Mean follow-up was 5.8 months. Recipients with end-stage renal disease and those followed by outside institutions were excluded. Results: Most patients receiving FK506 normalize BNP levels (slope -40.2, p<0.0001). 26/33 patients (78%) reached BNP levels of <100 pg/ml by 4 months. Both the CSA and FK506 groups had similar ejection fraction, pulmonary capillary wedge (PCW) pressure, and creatinine. In FK506 recipients, a weak correlation coefficient (r = 0.4) was observed between PCW and BNP. Utilizing a BNP level of 150 pg/ml and PCW of 14 mmHg, the sensitivity of the assay decreased from 100% during the first two months to 21% during the subsequent months. In contrast, the specificity of the assay increased from 34% to 81% during the same time period. There were 7 episodes of rejection (3 cellular and 3 humoral) in 6 HT recipients. Cases were not preceded by an increase in BNP in the days to weeks prior to presentation. Conclusions: Most patients on FK506 achieve normal BNP levels by 4 months in contrast to those reported HT patients on CSA. In HT patients on FK506, BNP appears to be an inadequate assay to screen for rejection or volume status. The mechanism by which FK506 suppresses BNP levels after OHT deserves further investigation.

1137-65

Effects of Growth Hormone Therapy Following Pediatric **Cardiac Transplantation**

Seema Mital, Aleza Andron, Barney Softness, Daphne T. Hsu, Jacque M. Lamour, Linda J. Addonizio, Columbia University, New York, NY

Background: Growth hormone (GH) is used to treat growth failure following cardiac transplantation (Tx). GH can increase cardiac muscle mass and improve function in patients with cardiomyopathy. We evaluated the safety, efficacy and cardiac effects of GH in children following cardiac Tx.

Methods: Ten pediatric cardiac Tx recipients with growth failure were followed serially for 4 years before and after GH therapy. Parameters followed included growth velocity (GV), cyclosporine levels, frequency of rejection episodes, echocardiographic measures of cardiac function and dimensions, and hemodynamic measurements. To determine if outcomes were related to increased GV or to a direct effect of GH, these data were compared to measurements obtained in 8 age-matched pediatric cardiac Tx recipients

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experiencing a natural growth spurt. Results: The mean age at Tx in the GH group was 7.8±1 yr with mean age at onset of GH therapy of 13±1 yr (mean duration, 2.5±1 yr). The mean GV increased following GH and was not different from controls (8.6±1 vs 7.1±1 cm/yr). Cyclosporine levels were maintained during the growth spurt (312±34 vs 268±28 ng/ml). The frequency of rejection episodes was not different in GH group compared to controls (0.1±0.2/yr vs 0.4±0.3/yr). LV shortening fraction increased in GH group from 36±2% to 44±2%, (p<0.05) and was higher compared to controls (37±2%, p<0.05). The SF returned to baseline levels after discontinuation of GH. Cardiac index increased following GH (3.2±0.3% to 3.9±0.2%, p=0.01) and was higher than controls (2.9±0.3%, p<0.01). Mixed venous saturations were higher following GH (76±1% to 71±2%, p<0.05). The calculated LV mass increased from 95±11 g/m2 to 117±17 g/m2 following GH (p=0.01) and was higher than controls (95±7 g/m2, p<0.05). LV mass/volume ratio was also higher in GH group (0.72±0.02) compared to controls (0.67±0.03, p=0.05). Conclusion: GH is a safe and effective therapy for growth failure in children following cardiac Tx even in the absence of GH deficiency. GH therapy is also associated with short-term improvement in ventricular mass, cardiac function and hemodynamics. Whether these beneficial effects are sustained in the long term remains to be determined.

1137-66

Survival Pre and Post-Heart Transplantation in Patients Listed as UNOS Status 2: Do UNOS Status 2 Patients **Benefit From Transplantation?**

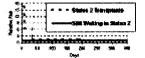
Javier Jimenez, Leah Bennett Edwards, Robert Higgins, Joseph Bauerlein, Si Pham, Stephen Mallon, Jackson Memorial Medical Center, Miami, FL, United Network for Organ Sharing, Richmond, VA

Introduction: Improved outcomes with contemporary medical therapy in patients with advanced heart failure questions the benefit of transplantation (TX) in UNOS status 2 patients.

Methods: Between January 1999 and June 2001: 4,255 patients were listed for heart TX as UNOS status 2. Using a competing risk method, probabilities of events on the waiting list were computed. Additionally, a time dependent proportional hazards model was used to determine predictors of death pre and post TX.

Results: Demographics of this cohort revealed a mean age of 52.3 years, female gender (23%), ischemic etiology (48%), diabetes (21%), white race (85%), and mean time on the waiting list (398.5 days). Relative risks of death (> 1 indicates an increased risk) compared to patients remaining on the waiting list as status 2 were: upgrade to status 1A; RR 14.9 (95%Cl 9.7-22.2), upgrade to status 1B; RR 3.4 (95%Cl 2.4-4.7), 0-7 days following TX as status 2: RR 16.7 (95%Cl 9.3-30.2) and 7-33 days following TX as status 2: RR 6.5 (95%Cl 3.7-11.2). All factor were significant at p<0.0001. The overall relative risk of death following TX (365+ days) for status 2 patients initially listed as status 2 compared to those that continue to wait as status 2 is shown below; RR 0.92 (95%Cl 0.5-1.58. n=0.8)

Conclusion: After accounting for early perioperative mortality, there appears to be little survival benefit at one year in transplanting UNOS status 2 patients. The point of optimal benefit from TX in UNOS status 2 patients may need to be further defined.



1137-67

Cytomegalovirus Infection Negatively Influences Coronary Remodeling Modalities in Heart Transplant Recipients: A Prospective Study

Luciano Potena, Francesco Grigioni, Fabio Coccolo, Gaia Magnani, Paolo Ortolani. Cinzia Marrozzini, Antonio Marzocchi, Simona Sorbello, Anna C. Musuraca, Carlo Magelli, Angelo Branzi, University of Bologna, Bologna, Italy

Background. Graft coronary disease (GVD) is a major determinant of mortality after heart transplantation (HT). This peculiar form of atherosclerosis has been recently identified as the result of the interaction between intimal hyperplasia and vessel wall response (i.e. vascular remodeling). Although immunological and traditional risk factors are known to be implied in GVD pathogenesis, their contribution to remodeling process remains

Methods. 37 consecutive HT recipients were prospectively studied (age 52±11yrs; 75% males; donor age 32±11yrs). Intracoronary ultrasound (IVUS) of proximal-mid left anterior descending was performed at 1 and 12 months after HT. Vessel, lumen and intimal volume changes over this period were analyzed.

Results. 1189 IVUS images were obtained. Overall intimal volume increased (+80%, P<0.001) while vessel volume remained unchanged (P=0.2) and thus, lumen volume decreased (-9%, P=0.01). Among all the clinical and demographic characteristics analyzed (e.g. donor features, immunosuppression, lipid panel, biopsy score), only the occurrence of cytomegalovirus (CMV) infection was associated with coronary lumen loss (P=0.047). Patients who presented CMV infection (n=14) showed an higher increase in intimal volume (118% vs. 59%, P=0.07), but not in vessel volume (+1% vs. +4%, P=0.5). Therefore, CMV infected patients showed a more significant lumen loss (-13% vs -3%, P=0.04). A trend towards an association between LDL and intimal growth was present only in CMV infected recipients (R=0.46, P=0.07).

Conclusions. This prospective study suggests for the first time that occurrence of CMV infection during the first post-HT year negatively affects vascular remodeling, ultimately resulting in coronary lumen loss. LDL serum levels might contribute to lumen loss interacting with CMV infection in stimulating intimal growth.