Introduction and Aims: Endothelial dysfunction can be detected at early stages of chronic kidney disease (CKD). Although endothelial functions improve after successful renal transplantation, renal transplant recipients have still worse endothelial functions compared to healthy subjects. Recent trials showed that vitamin D deficiency and high fibroblast growth factor 23 (FGF-23) levels may have a role on endothelial dysfunction in CKD patients besides their well-known effects on calcium and phosphorus metabolism. Aim of this study is to investigate the association between endothelial functions, vitamin D and FGF 23 levels in renal transplant recipients.

Methods: One hundred nine renal transplant recipients (71 male, 38 female) underwent beat to beat measurement of carotid-femoral pulse wave velocity (PWv). serum 25 OH vitamin D and FGF-23 level measurements. Patients were divided into two groups based on endothelial functions. Vitamin D and FGF-23 levels were compared between patients with normal and abnormal endothelial functions. Correlations between amount of FMD, vitamin D level and FGF-23 were also investigated.

Results: Mean ages of the patients was 40.4±11.5 years, mean duration after transplantation date was 74.7±69.5 months. Endothelial functions were abnormal in 79 patients (72.5%). Prevalence of vitamin D deficiency (<15 mcg/L) was 65.1%. Patients with normal endothelial functions and endothelial dysfunction had similar demographic, clinical characteristics and laboratory values. Vitamin D levels were significantly lower in patients with endothelial dysfunction compared to patients with normal endothelial functions (12.6± 6.6 mcg/L vs 17.3±10.0 mcg/L respectively, p=0.02). FGF-23 levels were not different between two groups. Vitamin D levels had a significant positive correlation with arterial stiffness. Arterial stiffness is an important risk factor for adverse cardiovascular outcomes. Observation of this study is that vitamin D replacement could be a potential treatment option for endothelial dysfunction in these patients.

Conclusions: Vitamin D deficiency is one of the causes of endothelial dysfunction in renal transplant recipients. Further studies are needed to clarify whether FGF-23 is a marker or a potential initiation for endothelial dysfunction and the effect of vitamin D replacement on endothelial functions in these patients.

ARTERIAL STIFFNESS AND METABOLIC SYNDROME INDICES IN RENAL TRANSPLANTATION PATIENTS

Orhan Guliyev1, Mehtap Erkmen Uyar1, Emre Tutal1, Zeynep Bal1 and Siren Sezer1
1Baskent University Hospital Ankara Turkey

Introduction and Aims: Although renal transplantation improves survival, cardiovascular morbidity and mortality still remain as a significant problem compared with nonrenal populations. In end stage renal disease metabolic cardiovascular risk factors such as hypertension, hyperuricemia, obesity and diabetes mellitus have been confirmed to be positively correlated with arterial stiffness. Arterial stiffness is an important characteristic of the arterial wall and can be assessed noninvasively by the measurement of carotid-femoral pulse wave velocity (PWV). The aim of this study is to evaluate the risk factors for arterial stiffness in kidney transplant recipients.

Methods: One hundred and forty nine kidney transplant recipients from our renal transplant outpatient clinic were enrolled into the study. All patients were evaluated for their standard clinical (age, gender, duration of hemodialysis, post-transplant time), biochemical parameters. Anthropometric and body composition analyses were performed for all patients. Body compositions were analyzed by using the Body Composition Analyzer (Tanita BC 420MA). PWV was determined from pressure tracing over carotid and femoral arteries using the SphygmoCor system.

Results: PWV of all patients were significantly decreased. In post transplantation period, patients with high PWV levels had significantly higher PWV (>7m/s) than patients with normal PWV levels (p=0.02). FMD, vitamin D level and FGF 23 were also investigated.

Conclusions: High PWV could be a predictor of arterial stiffness in renal transplant recipients. Electrocardiographic evaluation is seen to be a cheap and reliable way to detect arterial stiffness.

VITAMIN D SUPPLEMENTATION CAN SAFELY AND EFFICIENTLY BE ACHIEVED BY MONTHLY ORAL BOLUS CHOLECALCIFEROL IN VITAMIN D DEFICIENT RENAL TRANSPLANT PATIENTS

Anthony O’Rourke-Potowki1, Nathan Gauge1, Hugo Penny1, Antonia Cronin1, Sharon Fram1 and David J. Goldsmith1
1Nephrology and Transplantation Guy’s Campus King’s Health Partners AHSN, London United Kingdom

Introduction and Aims: Vitamin D insufficiency (> 25 < 50 nmol/L) and deficiency (< 25 nmol/L) are common in stable ambulant renal transplant patients (RTx). This has been associated with adverse skeletal, renal, cardiovascular and cancer outcomes in this population, but a formal repletion RCT with hard end-points has never been completed. We undertook vitamin D repletion using oral Dekristol™ cholecalciferol in a group of long-term renal transplant survivors, all of whom had demonstrated sustained vitamin D deficiency, to assess both efficacy and safety of this intervention.

Methods: Out of 360 long-term (> 8 years surviving) RTx patients we found 57 subjects with sustained very low (< 25 nmol/L) serum vitamin D concentrations, and either or both of raised PTH or otherwise unexplained proximal myopathy and bone pain. We prescribed all of these patients 40,000 IU Dekristol™ cholecalciferol per month (total dose 240,000 IU) and then interrogated the biochemical changes in plasma vitamin D, PTH, alkaline phosphatase, calcium, phosphate and creatinine (eGFR) concentrations over the course of the repletion period. Paired t-tests.

Results: Three patients did not complete the course of vitamin D repletion (two died from unrelated causes, and one developed cancer necessitating major surgery). This left 54 completed (per protocol) repletion courses to examine for efficacy and safety outcomes. Mean age 54 +/- 17 years. Mean time post-transplantation 14.4 +/- 3.5 years. Mean eGFR (at start) 58+/- 9 mls/min.
Conclusions: All 54 patients completed their repletion course. In all cases plasma vitamin D concentrations rose to >25 mmol/L and in 80% to >50 mmol/L. Two patients experienced a >20% rise in plasma creatinine (biopsy proven rejection in both cases). The remaining patients had a very modest change in plasma creatinine (proportional to the rise seen in plasma calcium, also modest). Only 5 patients experienced a plasma calcium concentration of >2.60 mmol/L and in no case was it necessary to discontinue vitamin D treatment. The fall in plasma PTH concentration was significant and potentially valuable. The fall in plasma alkaline phosphatase values just missed significance. Monthly bolus oral cholecalciferol seems a safe and effective means by which to render RTX patients vitamin D replete.

**Abstracts**

### SP640

**LONG TERM OUTCOMES OF HIGHLY SENSITIZED KIDNEY TRANSPLANT RECIPIENTS**

Jude A. Yagan1 and Anil Chandraker2

1 Renal Deinstallation Brigham and Women's Hospital Boston MA United States, 2 Renal International Society of Nephrology Brussels Belgium

**Introduction and Aims:** To follow the clinical outcomes of 45 highly sensitized patients who had undergone a desensitization protocol prior to kidney transplantation, and report the incidence of complications, allograft survival, and Patient survival.

**Methods:** We conducted a retrospective review of 45 kidney transplant recipients transplanted between 9/2002 and 10/2011, who had a positive T or B cell complement dependent cytotoxicity (CDC) crossmatch assay. B cell CDC crossmatches were confirmed with a solid-phase assay to determine presence of class II anti-HLA antibodies.

**Results:** All subjects completed a desensitization protocol of plasmapheresis, intravenous immunoglobulin, +/- rituximab to render a negative T cell crossmatch or a negative or weak titer B cell crossmatch 24 hours prior to transplantation. Post-transplant all recipients received antibacterial and antiviral prophylaxis; allograft biopsies were performed when clinically indicated. The mean and median follow-up was 5 years. Thirty-three subjects (73%) suffered acute rejection of the allograft, 30 (67%) occurred in the first year post-transplant, and 27 (60%) occurred in the first month post-transplant.

**Conclusions:** Patients with a positive CDC crossmatch that are transplanted after a plasmapheresis-based desensitization protocol have high rates of acute rejection and infectious complications. Despite increased rate of rejection and over-immunosuppresion, patient and graft survival in the desensitized group is comparable to the 1, 3, 5 year survival (graft: 89%, 78%, 67% respectively; patient: 95%, 90%, 85% respectively) of recipients of repeat transplants from living donors.

### SP641

**CMV AND KIDNEY TRANSPLANT VASCULOPATHY**

Nicoleta Serpieri1, Fabrizio Grosojean2, Giuseppe Sileno2, Massimo Torreggiani2, Vittoria Esposito1, Filippo Mangione1, Massimo Abel1, Francesca Castoldi1, Davide Catucci1, Ciro Esposito1 and Antonio Dal Canton1

1 Nephrology and Hemodialysis Fondazione S. Maugeri, University of Pavia Pavia Italy, 2 Nephrology Fondazione IRCCS Policlinico S. Matteo, University of Pavia Pavia Italy, 3 Surgery Fondazione IRCCS Policlinico S. Matteo, University of Pavia Pavia Italy

**Introduction and Aims:** Cytomegalovirus (CMV) infection is one of the most frequent complications after organ transplantation. CMV seems to exhibit vascular tropism, being observed in atherooclerotic lesions and being able to induce vascular damage in viral lethopasy. Several studies suggested a possible association between CMV infection and accelerated graft vasculopathy in heart transplant recipients. Aim of our study was to investigate a role for CMV in kidney transplant vasculopathy.

**Methods:** We retrospectively selected 2 groups of kidney transplant recipients, group A (N=14) with CMV infection requiring treatment (both pre-emptive and for clinical indication) and group B (N=14) CMV negative. Subjects in both groups had similar comorbidities and similar exposure to calcineurine inhibitors.

**Results:** Average GV was lower in group A than group B, at each time point we considered, as expression of a relative glomerular hyperperfusion (1st month: 4.2±1.1 vs 4.9±1.7; 6th month: 4.3±1.6 vs 5.2±1.1; 12th month: 3.2±1.3 vs 4.3±2.1×10^-3 mm²; A vs B group). Average WLR was increased in group A (1st month: 2.7±1.2 vs 2.3±0.9; 6th month: 3.1±1.3 vs 2.6±0.9; 12th month: 3.6±1.7 vs 2.4±0.4; A vs B group). WLR was evaluated 12 months after transplantation in patients with clinically evident CMV infection was significantly increased compared with CMV negative patients (5.2±1.7 vs 2.3±0.4, p<0.01). We detected few CMV positive cells only in the vessels and interstitium of kidneys from CMV positive subjects with clinically evident disease.
Introduction and Aims: One of the current tasks of transplantation is to overcome “graft-host” immune conflict. Partially this conflict caused by the presence of circulating pre-existing antibodies. Highly sensitized patients are at greater risk of rejection and subsequent graft loss. There are several methods to remove the anti-HLA antibodies, one of which is a double filtration plasmapheresis (DFPF). This report presents our experience of DFPF in recipients of high immunologic risk.

Methods: The study included 18 patients after kidney transplantation. All patients were classified as high-immunologic risk group. The predisposing factors were only one HLA-match (7 patients) re-transplantation (9 patients), the presence of anti-HLA antibodies – (2 patients). These patients DFPF performed before transplantation, in the first days after transplantation, and two days after the transplantation.

Immunosuppressive therapy included calcineurin inhibitors – tacrolimus, mycophenolate, and corticosteroids. Induction therapy was a monoclonal anti-CD25 antibodies and methylprednisolone. We monitored the immune status: total number of lymphocytes, activation markers of humoral immunity – IgG, IgM before and after the DFPF. We used enzyme-linked immunosorbent assay (ELISA) for the analysis of humoral background of the recipient. Procedure performed on the ctoNova (MeSys, Germany) with a plasmfilter and plasma components separator. Protocol biopsies were performed on days 7 and 30.

Results: Three patients had rejection crises, confirmed histologically. One of the patients with acute rejection on postoperative day 7 with laceration of graft and bleeding. In this patient we emergency made 2 DFPF procedures daily. Total IgM antibodies were reduced by 41% of the original level. IgG was reduced 57% after the first treatment and then remained stable. There are no signs of antibody-mediated rejection in biopsy obtained on day 30 after the operation. Within 3 months of follow graft function remained stable.

Conclusions: DFPF can safely and effectively reduce the high titers of antibodies that are responsible for humoral rejection of renal allograft. Reduction of antibodies in sensitized patients immediately after transplantation may improve graft function.

SP645
effect of prednisolone and rejection episodes on granzyme b transcripts levels after kidney transplantation

Christoffer Borst1,2, Ying Liu1,2, Jan Thoning1,2 and Martin Tepel1,2
1Nephrology Odense University Hospital Odense DK Denmark, 2Institute of Molecular Medicine University of Southern Denmark Odense DK Denmark

Introduction and Aims: Expression of main paracrine factors from cytotoxic T lymphocytes, including granzyme B, may be associated with outcome after kidney transplantation. Currently it is unknown whether immunosuppressive therapy or rejection episodes may specifically affect these paracrine factors.

Methods: Peripheral blood samples were repeatedly collected every week after transplantation for 4 consecutive weeks. RNA was extracted from mononuclear cells using established technology. Transcripts of granzyme B, perforin, interferon stimulating glycoprotein 15, and matrix metalloproteinase 9 were analyzed using quantitative real-time PCR.

Results: Transcripts were measured in 99 patients after kidney transplantation (55 living related kidney donors and 44 deceased kidney donor transplantations). Immunosuppressive treatment consisted of basiliximab, tacrolimus and mycophenolate mofetil. Prednisolone was administered to 32 patients, whereas 67 patients were prednisolone-free. Patients in the prednisolone group showed significantly lower granzyme B compared to the prednisolone-free group (0.011±0.003 arbitrary units vs. 0.023±0.005 arbitrary units; mean±SEM; p<0.0005).

Conclusions: These preliminary data suggest that after 6 months, T-reg in patients of group A are significantly increased when compared both to the BAS and group B, while T-reg cells are suppressed by standard immunosuppressive therapy (group B). These results suggest that spertimental immunosuppressive protocol in vivo may improve peripheral tolerance in kidney transplantation recipients.

SP647
Transplant glomerulopathy and de novo thrombotic microangiopathy: different manifestations of humoral rejection

Rui Miguel Costa1, Eduardo Vásquez Martín2, Juan Rebroedo2 and Constantino Rivera2
1Nephrology CHT MAD Vila Real Portugal, 2Pathology CH Juan Canalejo Coruña Spain

Introduction and Aims: This study characterizes clinical and pathological relationships between transplant glomerulopathy (TG) and de novo thrombotic microangiopathy (TMA) in renal allograft biopsies.
Methods: A retrospective comparative analysis was performed during a 10 years period that included demographic, clinical, laboratory, histological and outcome features.

Results: Out of a total of 627 biopsies, a total of 39 TG cases (6.2%) in 33 patients and 14 (2.2%) de novo TMA cases in 12 patients were found. TG was diagnosed later (6.9±5.9 vs 3.5±6.5 years, p=0.01) and presented higher proteinuria (4.0±3.6 vs 2.3±1.6 grams/24h, p=0.02). De novo TMA was associated to worse graft function (4.9±1.8 vs 2.7±0.7 mg/dl, p=0.001) with all allografts provided from older patients (53.8±10.1 vs 41.3±17.9 years, p=0.035) and a trend in TMA to longer cold ischemia time (1439±210 vs 1218±441 min, p=0.141) and previous rejection episodes (21.4% vs 5.3%, p=0.079) was found. No differences in proportion of deceased/dying donor grafts, HLA mismatches, PRA levels, pre-transplant or cyclosporine therapy were reported. In light microscopy, diffuse glomerular lesions were observed in all de novo TMA cases, opposing to a focal presentation in TG (100% vs 45.8% p=0.001). Both groups presented equally glomerular basement membrane (GBM) double contours, glomerulitis, tubulitis, capillaritis and ischemic features. Capillary congestion (100 vs 35.1%, p=0.001), microthrombosis (50% vs 5.4%, p=0.001), schistocytes (42.8% vs 7.7%, p=0.009) and mesangiolysis (85.7% vs 29.7%, p=0.001) were associated to TMA. Most TG cases presented advanced glomerulocapsular, interstitial fibrosis and tubular atrophy and interstitial plasma cells were only seen in these cases (30.8% vs 0%, p=0.018).

Positive C4d staining in de novo TMA cases was similar to TG (74.1% vs 54.8%, p=0.352) but arteriolar C4d deposition was predominantly seen in these cases (35.7 % vs 8.7%, p =0.042). Donor Specific Antibodies (DSA) detection was equally found in both groups (TG: 41.6% ; TMA: 57.1%, p=0.050) and were mainly constituted by anti-HLA Class II (60% and 75%, respectively). After ultrastructural analysis, only TG cases presented GBM multilamination (75% vs 0%, p=0.044) and tended to expressed more frequently peritubular capillary basement membrane thickening (100% vs 60%, p =0.074) with multilamination (75% vs 20%, p=0.060). Considering all follow up time, graft loss occurred similarly in both groups (47.1% vs 45.5%, p=0.936) but de novo MAT cases had lower first year post-transplantal survival (73.3% vs 97.0%, p=0.034).

Conclusions: Similarities in histological features, C4d staining and presence of DSA suggests that TG and de novo TMA may represent different manifestations of humoral endothelial injury. While TG may express a subclinical ongoing antibody mediated injury, presenting chronic cyclic accommodation mechanisms, TMA results from an intense acute manifestation of endothelial lesion.

SP648 CLINICAL RESULTS OF COMBINED AND SEQUENTIAL LIVER-KIDNEY TRANSPLANTATION: A SINGLE CENTER EXPERIENCE

Francesca Simonato1, Giuliana Tognarelli, Germana Daidola1, Ester Gallo1, Manuel Burdese1, Vincenzo Cantaluppi1, Luigi Biancone1 and Giuseppe P. Segoloni1

1Moioferte Hospital Nephrology, Dialysis and Transplantation Unit Turin Italy

Introduction and Aims: Since 2002, when the end-stage liver disease model score (MELD) was adopted, the number of combined liver-kidney transplantsations increased. Between January 1995 and December 2011 we performed 38 combined liver-kidney transplantation (CLKT) and 6 sequential liver-kidney transplantation (SLKT).

Methods: This study compares the outcomes of CLKT and SLKT with the contralateral kidney (31 of the first group and 6 of the second one) used for the kidney alone (SLKT).

Results: The indications for CLKT were: polycystic disease (60.6%), primary Hyperoxaluria type 1 (21%), end-stage kidney disease and cirrhosis (18.4%). In SLKT, the major cause of renal failure was calcineurin inhibitor nephropathy (83.3%) and dialysis started on a 7 years average time after liver transplantation. Delayed renal graft function (DGF) occurred in the 52.6% of CLKT vs. 38.7% in the KTA, despite a minor cold ischemia time and lower donor age in CLKT group. Infections and bleedings were more common in CLKT patients (86.8% vs. 61.5% in KTA1 p=0.034), as well as surgical complications (42% vs. 11.5% in KAT1 p=0.03). The immunosuppressive protocol mostly used was tacrolimus, mycophenolate mofetil and prednisone. In CLKT recipients tacrolimus levels were lower and steroid was stopped earlier than KTA. The acute renal rejection frequency was lower in CLKT (2.8% in CLKT, 7.7% in KTA1 and 16.6% in SLKT, p = not significant) despite a major HLA mismatch, positive X-match, specific anti-donor antibody and lower immunosuppression. Mean creatinine serum levels were more frequent in the first three months after transplantation, the patient and kidney allograft survival rates appeared to be superior than those in SLKT. In addition, in CLKT there were lower serum creatinine levels despite a major HLA mismatch, positive X-match and lower and steroid was stopped earlier than KTA. The acute renal rejection frequency was lower in CLKT (2.8% in CLKT, 7.7% in KTA1 and 16.6% in SLKT, p = not significant) despite a major HLA mismatch, positive X-match, specific anti-donor antibody and lower immunosuppression. Mean creatinine serum levels were more frequent in the first three months after transplantation, the patient and kidney allograft survival rates appeared to be superior than those in SLKT. In addition, in CLKT there were lower serum creatinine levels despite a major HLA mismatch, positive X-match and higher immunosuppression. HLA mismatch (25% in CLKT vs 37.5% in SLKT) and higher creatinine levels (4.9±1.8 vs 2.7±0.7 mg/dl, p=0.001) were associated to KTA. Mean mTOR levels (56.1±10.1 vs 42.3±9.5, p=0.041) were associated to KTA. In conclusion in CLKT recipients, although complications and mortality were more frequent in the first three months after transplantation, the patient and kidney allograft survival rates appeared to be superior than those in SLKT. In addition, in CLKT there were lower serum creatinine levels despite a major HLA mismatch, positive X-match, specific anti-donor antibody and lower immunosuppressive effect on the renal allograft from the same donor.
**Introduction and Aims:** Measurements of glomerular filtration rate (GFR) are frequently interpreted assuming a linear variation with age. However this may be simplistic. Non-linear relationships may give a better representation of the changes associated with “normal aging”. This is a really important consideration in a population of potential living kidney donors generally considered to be even healthier than age-matched controls.

**Methods:** This was a retrospective study of 904 subjects (468 women, 436 men; age range 18–85 years) undergoing assessment as prospective living kidney donors. GFR was evaluated from $^{51}$Cr-EDTA plasma clearance using blood samples taken at 2, 3, and 4 hours. The slope-intercept GFR was corrected for body surface area (BSA) using the Haycock formula and for the fast exponential using the Brochner-Mortensen equation. The relationship between age, gender, and GFR was examined using best-fit curve analysis. Non-linear relationships with age were explored using fractional polynomials.

**Results:** There was no gender difference in BSA corrected GFR over five decades of age (P = 0.40). However, female donors with a body mass index > 30 kg.m$^{-2}$ had a statistically significantly lower GFR than non-obese women (P < 0.01). The best-fit relationship between age and GFR was non-linear and described using a fractional polynomial model of degree 1 (GFR = 103.9 – 0.0061*Age$^2$ mL.min$^{-1}$.1.73 m$^{-2}$) with an RMSE of 12.9 mL.min$^{-1}$.1.73 m$^{-2}$). The residual variance for this model was significantly smaller than for the best-fit linear model (P = 0.006).

**Conclusions:** GFR measurements in prospective healthy living kidney donors are best corrected for age using a non-linear relationship. Our results help to establish potential normative mGFR ranges for this important population, which will crucially inform decisions on potential wisdom of kidney donation.

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**THE EFFECT OF MAGNESIUM SUPPLEMENTATION ON EARLY POST-TRANSPLANTATION GLUCOSE METABOLISM**

**Introduction and Aims:** Our objective was to evaluate the usefulness of three-dimensional contrast enhanced MR angiography (3D CE MRA) for assessment of renal artery anastomosis in the early period after kidney transplantation.

**Methods:** Between January 2010 and February 2012, a consecutive series of 267 KT patients were examined with 3D CE MRA 14 days after transplantation. The study patients were divided into four groups by the degree of renal artery inflow stenosis qualitatively (group I: normal; group II: mild ≤ 50%; group III: moderate 50%–70%; group IV: severe > 70%). The following variables were compared: donor and recipient characteristics, multiplicity of renal arteries, the type of the arterial anastomosis (end-to-end anastomosed to IIA (internal iliac artery) or end-to-side anastomosed to EIA (external iliac artery)).

**Results:** 261 (80.9%) of the 267 patients had normal 3D CE MRA, 29 (10.9%) showed mild, 8 (3.0%) was moderate, and 14 (5.2%) had severe stenosis of renal inflow. 11 patients of severe arterial stenosis on CE MRA underwent selective digital subtraction angiography (DSA). In ten patients, angioplasty or stenting was performed. The mean creatinine value at 14 days post-transplant (1.27 ± 0.48, 1.21 ± 0.48, 1.04 ± 0.32, 1.20 ± 0.27, respectively) did not significantly differ among the four groups (P = 0.495). The prevalence of graft loss (n=2, 14.3%) was high in patients with severe arterial stenosis, but there was no significant differences in these groups (P = 0.118). In group IV, multiplicity of renal arteries (n=8, 57.1%) and the type of end-to-end arterial anastomosis (n=12, 85.7%) were much higher frequency (P = 0.026, P = 0.362, respectively) than other groups.

**Conclusions:** The incidence of arterial flow stenosis is unexpectedly high in the early period after kidney transplantation even if creatinine level was normal. So, 3D CE MRA allows rapid global assessment of renal transplant arterial system. It can also help detect or exclude many of the various causes of renal transplant dysfunction.

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**ASSOCIATION BETWEEN A DECLINE IN DONOR CREATININE CLEARANCE AND ALLOGRAFT OUTCOMES**

**Methods:** The decline in CrCl of donor kidney was calculated by the difference over 30 days after KT. $\Delta$CrCl = (CrCl at post-KT 30 days – CrCl at Pre-KT) / CrCl at Pre-KT x 100 (%). All recipients were divided into 2 groups according to $\Delta$CrCl Group I (n = 69), < 30% and Group II (n = 70), > 30%.

**Results:** A total of 135 recipients were followed for 136 ± 63 months. At baseline, there was no significant difference in baseline donor, recipient, and KT-related characteristics. While there was no difference in recipient eGFR at 3 posttransplant, Group I had higher eGFR at 1 month ($60 \pm 24$ vs. $50 \pm 23$ mL/min/1.73 m$^2$), 3 months ($65 \pm 19$ vs. $53 \pm 22$ mL/min/1.73 m$^2$), 1 year ($63 \pm 19$ vs. $52 \pm 23$ mL/min/1.73 m$^2$) and 3 years ($56 \pm 20$ vs. $45 \pm 21$ mL/min/1.73 m$^2$) posttransplant as compared with Group II. However, eGFR at 10 years follow-up was not different between the groups. In linear regression analysis, donor $\Delta$CrCl was significantly associated with eGFR at 12 months and 3 years posttransplant, but not eGFR at 3 month follow-up of allograft recipient. KM analysis revealed that Group I had a greater dialysis-free survival rate at 10 years’ follow-up as compared with Group II ($79 \pm 7\%$, log rank P = 0.035).

**Conclusions:** In this study, we showed that the change of donor GFR over 30 days after KT measured using CrCl was associated with recipient eGFR at 1- and 3-year posttransplantation. However, $\Delta$CrCl had no association with eGFR at post-KT 10 years and kidney transplant recipients with higher $\Delta$CrCl had no good over-all long-term dialysis-free allograft survival. These results suggest that the short-term allograft survival is longer when the initial decline in donor CrCl is less. Follow-up measurement of donor kidney function may be useful to monitor the patient at risk for allograft loss.

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**TRANSPLANT RENAL ARTERIAL STENOSIS DEFINED BY CONTRAST ENHANCED MR ANGIOGRAPHY IN EARLY POST-Renal TRANSPLANTATION: PREDISPOsING FACTORS AND CLINICAL OUTCOMES**

**Introduction and Aims:** We aimed to prospectively evaluate whether oral magnesium (Mg) supplementation modifies the risk for NODAT.

**Methods:** Single-center, randomized, controlled, 3-month open label trial comparing Mg supplements (Mg oxide 450mg up to thrice daily) aiming at normalizing serum Mg (n=27) vs no supplements (n=27) in adult tacrolimus-treated non-diabetic renal transplant recipients with hypomagnesemia (1.7mg/dL) the first two weeks post-transplantation. Mg response was defined as increase of serum Mg at month 3 vs baseline. The primary outcome was change in glucose metabolism, assessed by OGTT at month 3 with measurement of AUC of glucose and by the change of HOMA-IR 3 months after inclusion (second OGTT).

**Results:** In this population (63% male, age 51.9±11.9, BMI 25.1±3.35), Mg supplementation increased serum Mg (MgA 8.1±0.21mg/dL vs. 8.02±0.23mg/dL, p=0.062). Of the supplemented patients, 25.9% were non-responders vs 48.1% in the control group (p=0.091). Overall incidence of NODAT was 10/54 (18.5%) and not different between supplemented (4/27) vs controls (6/27) (p=0.484). At month 3, fasting glucose levels were lower in the supplemented group vs the controls (92.6±9.6).
vs 104.1±21.9 mg/dL; p=0.022) (p=0.003 adjusting for tacrolimus levels) while AUC of glucose was not (135.9±34.2 vs 145.6±49.5; p=0.271 after adjustment for tacrolimus levels). HOMA-IR rose equally in both groups (3.01±3.16 vs 1.75±5.49 in the control group). Almost half (49.9%) had an abnormal glucose metabolism at 3 months. A fast post-transplantation drop of serum Mg (<1 mg/dL from pre-transplantation to inclusion) was associated with IFG at month 3 (AOR 6.34;95% CI 1.19-33.72; p=0.03 adjusting for baseline tacrolimus levels and HOMA-IR). Mg non-response (n=20) vs response was associated with a higher baseline HOMA-IR (3.05±3.32 vs 2.04±1.31; p=0.037), incidence of NODAT (30% vs 11.8%; p=0.093) and insulin resistance (HOMA2) at month 3 (AOR 8.143;95% CI 1.566-42.334; p=0.013 adjusting for tacrolimus levels). Of the supplemented patients without Mg response, 85.7% vs 38.5% of the controls had abnormal glucose metabolism (p=0.043). No discontinuation due to adverse events.

Conclusions: Post-transplantation hypoglycaemia predicts early NODAT but reverse causation cannot be ruled out. Supplementation of e-icious-might beneficially alter glucose metabolism. Failure to increase serum Mg by supplementation or the degree of post-transplant serum Mg drop both are associated with abnormal glucose metabolism, suggesting that glucose metabolism is more dominant for Mg status than its supplementation.

### SP656

**ASSOCIATION BETWEEN URIC ACID CONCENTRATION AND POSTDONATION KIDNEY FUNCTION AFTER LIVING DONOR NEPHRECTOMY**

Ajin Cho¹, Jung-ho Shin¹, Hye Ryu-jang¹, Jung Eun Lee¹, Woosong Huh¹, Dae Joong Kim Kim¹, Ha Young Oh¹ and Yoon-Goo Kim¹

¹Samsung Medical Center, Sungkyunkwan University School of Medicine Seoul Republic of Korea

**Introduction and Aims:** Elevated serum uric acid (SUA) concentration may contribute to renal damage or its progression via renal vasoconstriction and loss of renal autoregulation. Considering previous experimental and clinical data on SUA concentration we hypothesized that elevated SUA concentration plays a role in renal complications after donor nephrectomy. The aim of this study was to investigate the potential influence of preoperative SUA concentration on the risk of renal function impairment after living donor nephrectomy.

**Methods:** This retrospective observational study investigated 413 living kidney donors. The association between baseline SUA concentration ≥7 mg/dL in men and ≥6 mg/dL in women; odds ratio (OR), 3.78; 95% confidence interval [CI], 1.3–11.0; P = 0.02; older age (OR, 1.06; 95% CI, 1.02–1.09; P = 0.001); and lower baseline eGFR (OR, 0.82; 95% CI, 0.89–0.94; P = 0.001). An SUA concentration in the fourth quartile was significant related to renal function impairment in women (OR, 1.56; 95% CI, 1.10–1.88; p = 0.04) but not in men.

**Conclusions:** Predonation SUA concentration is associated with renal function after nephrectomy in living kidney donors. The association is stronger in women than in men. Further studies with longer follow-up are needed to assess the prognostic value of predonation SUA concentration to long-term renal insufficiency in living kidney donors.

### SP657

**PROGNOSTIC SIGNIFICANCE OF CHANGES IN PROTEINURIA IN EARLY STAGES OF RENAL TRANSPLANTATION**

Asunción Sancho Calabuig¹, Eva Gavela Martínez², Julia Kanter Berga³, Sandra Beltrán Catalán¹, Ana Isabel Avila Bernabeu¹ and Luis Manuel Pallardó Mateu¹

¹Nephrology Hospital Universitari Dr Pesset Valencia Spain

**Introduction and Aims:** Proteinuria is considered the main independent risk factor of end stage renal disease. Some authors consider that changes in proteinuria could be as a surrogate of kidney disease progression. Proteinuria is highly prevalent in renal transplantation and it has been associated to a lower graft and patient survival. There is no information about the relation between changes in proteinuria in early stages of transplantation and long term graft and patient survival. To analyze the effect of the magnitude of proteinuria and its changes from 3rd to 12th month after transplantation on long term graft and patient survival.

**Methods:** Retrospective analysis of 701 kidney transplants from deceased donors. Minimum follow-up: 1 yr. Baseline proteinuria (3rd month) and proteinuria at 12th month after transplantation in 24h urine were analyzed. Proteinuria was categorized depending on its magnitude: (0)144mg/d, (1)150-999mg/d, (2)300-999mg/d, (3)≥1g/d. Relative changes of proteinuria from 3rd to 12th month was calculated and categorized as follows: (0) reduction ≤50%, (1) increase or decrease (Δ) <50%, (2) increase ≥50% from baseline. Associations between baseline and 12th months proteinuria and changes of proteinuria and survival were examined using Kaplan-Meier and Cox analysis. X2 and T-test were used to analyze differences between groups.

**Results:** We studied 593 patients, mean follow-up of 84.5±4.6 months (r: 12±1.91). Proteinuria ≥150mg/d was present in 49.9% at 3 month, 47.0% at 12th month. At 3rd month proteinuria of 0.51%, (1) 17.9%, (2) 28.3%, and (3) 3.7%. Presence of increasing degrees of proteinuria at 3rd and 12th month was associated with long term graft failure (P=0.000), and mortality in case of 12th months (P=0.000). We observed an increasing relative risk of graft failure and mortality from category 2 at 3rd month (HR 2.083, 95%CI 1.362-3.187, P=0.001) and in the 12th month (HR 3.051, 95%CI 1.966-4.733P<0.000) that increases in category 3 (p=0.000). Between 3rd and 12th month we observed an absence or decrease of proteinuria ≥50% in 48.4%, Δ=50% in 26.4% and increase ≥50% in 25.2% of patients. Progression of proteinuria ≥50% was associated with graft failure (HR 6.000) in every category of proteinuria analyzed (P=0.000), not with mortality (P=0.183). Progression ≥50% was an independent risk factor of graft failure (HR 2.378;95%CI 1.540-2.167, P= 0.000). Proteinuria progression ≥50% was related to a higher serum creatinine at 12th month (P=0.000). Month duality in glomerular filtration rate at 12th months (MDRD-4).

**Conclusions:** Risk of graft failure and mortality increases with increasing amounts of proteinuria and with time from transplantation, since early stages of kidney transplant.

Proteinuria progression ≥50% in early stages of renal transplantation could be considered as a marker of graft failure, regardless of baseline proteinuria category.
Introduction and Aims: Persistent secondary hyperparathyroidism (SHPT) and residual proteinuria are important problems in kidney transplant patients. Both conditions can contribute to graft loss. Paricalcitol has shown a beneficial effect on chronic kidney diseases (CKD) related SHPT and decreases proteinuria in diabetic nephropathy. Information about paricalcitol treatment in kidney transplant patients is very scarce. Our objectives were to analyze the influence of paricalcitol on markers of mineral metabolism, residual proteinuria, renal function, and inflammation.

Methods: In a historical cohort of 58 kidney transplant patients with SHPT, we studied the impact of paricalcitol (initial dose 1 μg/48 hr) on the control of intact-parathormone (iPTH) and other markers of mineral metabolism, on the amount of residual proteinuria, renal function, blood pressure and inflammation. We used routine standard deviation, paired t-tests and Wilcoxon test. Remission ≥30% of iPTH and ≥50% of 24 hours proteinuria were calculated by logistic regression.

Results: Baseline characteristics: 26 men (44.8%), 55.7±12.7 years, creatinine 2.1±0.7 mg/dL, 24 hours proteinuria 1.1±0.7 g, calcium 9.3±0.5 mg/dL, phosphorus 3.4±0.5 mg/dL, iPTH 333.1±225.9 pg/ml, 25(OH)D 17.6±3.3 ng/ml. Serum iPTH significantly decreased after paricalcitol treatment (333.1±225.9 to 187.4±88.9 pg/ml) and 44 patients (76%) achieved a decrease of baseline iPTH≥30%. A baseline iPTH ≥500 pg/ml was associated with an iPTH reduction ≥30% (OR: 5.6, 95%CI: 1.3-24.6; p=0.022). Proteinuria decreased from 1.1±0.7 to 0.7±0.7 g/24 hours (p<0.05) (mean reduction 35%) and 26 patients (45%) achieved a ≥50% decrease of baseline proteinuria. Serum CRP <1.2 mg/dl was associated to proteinuria reduction ≥50% (OR: 13.8, 95%CI: 2.9-95.1, p=0.008). Renal function, with a significant decline during the 2-yr period before treatment, remained stable during paricalcitol treatment. C-Reactive Protein (CRP) showed a significant decrease with paricalcitol. Paricalcitol doses were reduced since the third month of treatment until the end of the follow-up (4.1±1.8 μg/week to 3.3±1.2 μg/week, p=0.05). Mild increases of serum calcium (10-10.5 mg/dL) and phosphorus (4.4-5.5 mg/dL) were detected in 4 (6.9%) and 7 (12.1%) patients, respectively, and responded to reduction of doses.

Conclusions: Oral paricalcitol is a safe and efficacious therapy of SHPT in kidney transplant patients, reducing residual proteinuria as well as systemic inflammation.
characteristic disease-specific HDL proteome. Of note, alterations of HDL were virtually identical between patients with CKD I-III and IV-IV and independent of transplant vintage and both were similar to ESRD patients. Furthermore, transplant HDL displayed decreased cholesterol content, but increased cholesteryl ester and free cholesterol in PBMCs of the patients. Finally, impaired anti-inflammatory HDL function could be demonstrated by increased expression of inflammatory cytokines including IL-6, IL-12p40 and TNF-α in monocytes compared to healthy HDL.

Conclusions: We demonstrate unique alterations of HDL from renal transplant recipients at the molecular and functional level. Importantly, remodeling of HDL including enrichment of distinct proteins previously identified from uremic HDL was also observed in patients with excellent graft function independent of the transplant vintage. These data may therefore not only help to unravel the causes of the excessive cardiovascular risk in renal transplant patients, but may also pave the way for novel diagnostic and innovative therapeutic directions.

Introduction and Aims: The utilization of expanded criteria donor (ECD) kidneys needs to be evaluated within the objective perspective of critical organ shortage and graft function and survival. Our objective was to compare the clinical outcomes of expanded criteria (ECD) versus concurrent standard criteria (SCD) deceased donors in adult renal transplantation.

Methods: Between February 2000 and December 2011, we performed 195 deceased donor renal transplants included 31 grafts (15.9%) from ECD and 164 grafts (84.1%) from SCD. ECD kidneys were classified by the UNOS definition. Donor and recipient risk factors were separately analyzed and correlated with recipient graft function and survival (minimum 6-month follow-up).

Results: ECDs were older (56.8±6.3 yrs), showed an increased incidence of hypertension, diabetes and cerebrovascular brain death, and had a higher pre-retrieval serum creatinine level compared with SCD. ECD kidney recipients had a shorter waiting time (P=0.019) but other baseline characteristics (age, gender, BMI, cause of ESRD, type of renal replacement therapy, incidence of Diabetes and hypertension, Number of antigen mismatch, positive of panel reactive antigen and cold ischemic time) were no significant difference from those of SCD kidney recipients. The mean MDRD GFR level at 1month, 6month, 1year and 3 year after transplantation was significantly lower in patients with ECDs but MDRD GFR level at 5, 10 years did not significantly differ (P=0.114, 0.702). The incidence of acute rejection episodes, surgical complications and infectious complication did not significantly differ between two groups, but rate of delayed graft function (DGF) was higher in ECD kidney recipients (P=0.007). Actual patient and graft survival rates were similar between groups with a mean follow-up of 43 months. There were no significant differences between two groups in graft survival (P=0.111) and patient survival (P=0.562).

Conclusions: In our center, 15.9% of deceased kidney transplants are performed from ECD. Although intermediate term renal function followed longitudinally was better in SCD kidney recipients and graft and patient survival of ECD kidney recipients were significantly more often in Tx patients, comparing to the IC population (81.8 vs. 60%). EV-HPV infections occur more frequently in transplant (Tx) patients receiving immunosuppressive therapy than in the immunocompetent (IC) population but exact mechanism of carcinogenesis and types of viruses responsible for that process are still uncertain. We investigated the association between the presence of betaHPV in NMSCs and healthy skin among Tx patients and IC population.

Methods: Skin biopsies (NMSC and healthy skin) were taken from 28 patients (11 Tx and 17 IC). Samples: 22 (8 Tx, 14 IC) from basal cell carcinoma (BCC) and 6 (3 Tx, 3 IC) squamous cell carcinoma (SCC) were tested for the presence of 25 different beta-HPV genotypes, including HPV: 5, 8, 9, 12, 14, 15, 17, 19-25, 36-38, 47, 49, 75, 80, 92, 93, and 96. DNA HPV was detected with the use of PCR with biotinylated PM-primer set and by means of hybridization (RHA skin (beta) HPV assay, Diassay BV, The Netherlands).

Results: In this study, HPV DNA within the area of NMSCs were detected in 9/11 examined Tx patients (81.8%) and in 5/17 IC patients (29.4%) (p=0.016). EV-HPV DNA in the clinically healthy skin was more frequently found in Tx patients (4/9 patients, 44.5%), compared to the IC population (4/14 patients, 28.6%), although the difference was not statistically significant (p=0.84). In patients with BCC, HPV-HPV DNA was significantly more frequent in patients with BCC (75%) and in 4/14 IC patients (28.6%) (p=0.035). In case of SCC, positive results for EV-HPV in the lesions were obtained in 3/3 (100%) whereas in the IC population EV-HPV was found in 1/3 lesions (33.3%) (p=0.8). In the studied samples the most frequent detected types of viruses were: HPV 9, 23 and 93. Non-identified types of EV-HPV were detected in 36.4% of Tx patients and 0% of IC population. In 2 cases of Tx patients 7 different types of HPV were discovered whereas in the IC group the highest number of detected HPV types were 3.

Conclusions: 1. EV-HPV presence in the examined NMSCs lesions occurred more frequently than in Tx patients, comparing to the IC population (81.8% vs. 28.4%). 2. Presence of non-identified types of EV-HPV emphasizes the need for further researches, aiming to determine the HPV role in development of NMSC.
Introduction and Aims: Renal transplant (RT) is burdened by huge cardiovascular (CV) mortality which is 10 to 50 fold higher than general population, due to worsened CV risk factors. Among these, reduced physical activity (PA) is a modifiable major one and it favors diabetes, dyslipidemia, obesity, metabolic syndrome, hypertension. Hence, PA may impact on both graft and patient survival, but no reliable data on PA exist in RT patients. This in an observational, controlled study aimed at measuring duration and intensity of PA in RT patients.

Methods: We recruited 110 stable renal transplanted patients (RTx) from at least 6 months and 110 healthy controls (CON). All of them underwent anthropometric measures and continuous measurements of PA along 3 consequtive days by means of SenseWear Armband® (BodyMedia, Pittsburgh, USA), a clinically-validated accelerometer device able to collect duration and intensity of PA in a free-living context. IPAQ questionnaire was also administrated to evaluate the subjectively reported PA data.

Results: Males were 46% in both groups. RTx-age was 80.6±6.9 y; RTx patients were comparable to CON for age, 48.8±11.9 vs. 48.1±13.5 y, body weight, 74.5±14.8 vs. 75.3±13.7 kg and BMI, 28.0±5.1 vs. 27.2±5.5 kg/m². Number of steps was lower in RTx patients than in CON, 9.27±8.417 vs. 11.108±4.036 steps/day (p<0.001). Intensity of PA, expressed as average METs, resulted similar in RTx and CON (1.5±3.2 vs. 1.5±3.2 METs/day). Duration of moderate PA was comparable in RTx (a2.3 METs) vall similar in Males (166±12 vs. 135±86 min/day), but lower in RTx Females (97.8±4 vs. 1381133 min/day, p<0.05) than CON Females. In RTx the number of steps per day inversely correlated with body weight (r=-0.252; p<0.01) and BMI (r=-0.288; p<0.01); intensity of PA, in addition to BW and BMI, also correlated with age (METs/day; r=0.263; p<0.01) and was inversely associated with F gender (both, METs/day and METs ≥2.3). Even though the actual reduced PA measured by accelerometer, 89% of RTx declared a subjective perception of PA of moderate-high degree by IPAQ.

Conclusions: Renal transplanted patients showed a reduced physical activity, mainly as duration but also as intensity, in female group at least. Reduced physical activity is age/gender related and the major modifiable determinant is increased body weight. Despite the low physical activity, renal transplanted patients has a subjective perception of adequate physical activity.
Methods: KIM-1 and NGAL levels were measured by ELISA in 24-hours urine samples collected within the first year after transplantation but not within a 2-week proximity of the surgery or transplant biopsy. We determined the predictive value for time to 50% and 100% decline in renal function or death-censored graft failure by Cox regression and incident/dynamic (I/D) ROC analyses. We evaluated whether they have a predictive value when eGFR is still within the good range and therefore performed all analyses separately in patients with baseline eGFR >50 mL/min.

Results: Baseline samples of 412 transplant recipients were included with a median of 54 days (IQR 44 – 77 days) and a follow-up of 1054 (554 – 1811 days). The concentration of KIM-1 and NGAL associated with the extent of albuminuria (respectively r0.46 and 0.43, P<0.0001), proteinuria (r0.44 and 0.39, P<0.0001) and the eGFR (r=0.27 and -0.39, P<0.0001). KIM-1 also associated with the urinary quantity of ADMA (r=0.22, P<0.0001). KIM-1 did not associated with 50% (HR per 0.99, 95% CI 0.87-1.12) or 100% (HR 1.01, 95% CI 0.88-1.17) decline in eGFR or graft failure on follow-up. Urinary NGAL neither associated with 50% (HR per 100 ug/24h 1.00, 95% CI 0.95-1.05) and 100% (HR 1.00, 95% CI 0.95-1.06) decline in eGFR or graft failure. The corresponding 1, 3 and 5 year I/D ROC AUCs for 50% and 100% decline in eGFR were comparably low for KIM-1 and NGAL excrution. When only investigating the patients with a baseline eGFR of >50 mL/min, NGAL (not KIM-1) excretion associated with 50% (HR per 100 ug/24h 1.51, 95% CI 0.99 – 2.32, P=0.06) and 100% decline in renal function (HR 1.75, 95% CI 1.22-2.72, P=0.01). However, the corresponding 1, 3 and 5 year I/D ROC AUC for 50% and 100% decline were low for both KIM-1 and NGAL.

Conclusions: Urinary excretion of KIM-1 and NGAL measured within the first year after transplantation have no prognostic value with all calculated incident C-indices below 0.60 indicating limited discriminatory value.

**SP669** KIDNEY TRANSPLANTATION AGGRAVATES UROLOGICAL MALIGNANCY RISK IN ESRD PATIENTS: A POPULATION-BASED STUDY

Jyh-Chang Hwang1, Ming-Yang Jiang1, Yi-Hua Lü1 and Shih-Feng Weng1

1*Nephrology Chi Mei Medical Center Tainan Taiwan Republic of China, 2Division of Medical Research Chi Mei Medical Center Tainan Taiwan Republic of China

**Introduction and Aims:** Post-transplantation is associated with various cancers. High urological malignancy incidence was reported in end-stage renal disease (ESRD) patients on dialysis. This study was undertaken to evaluate whether kidney transplantation (KT) aggravates urological malignancy risk in ESRD patients.

**Methods:** We used claims data of the Bureau of National Health Insurance of Taiwan for analysis. All KT recipients who developed urological malignancy from 1st January, 1999 to 31st December, 2007 (n=2,386) were enrolled for study. A database of 1:2ratio random new ESRD patients with matched age, gender, and cohort entry time was used as control (n=4,772).The longest observation period lasted up to 31st December, 2008.

**Results:** KT recipients had lower prevalence of Diabetes mellitus (p=0.001), C-hepatitis (p=0.0016) and liver cirrhosis (p=0.001). The cumulative incidence rate was significantly higher in the KT patients than those without transplantation (p<0.001). This gap became more prominent about 2 years after transplantation. By Cox proportional hazard regression tests, post-KT (HR:2.24, 95%CI:1.62-3.09), aging (especially after 35 years) and female gender (HR: 2.10, 95%CI:1.49-2.95) were independent factors for the development of urological malignancy after KT. After acquiring this malignancy, transplanted patients were similar to the ESRD counterparts in independent factors for the development of urological malignancy after KT. After 2 years after KT.

**Conclusions:** ESRD patients tended to have a significantly higher urological malignancy incidence after KT, especially for those patients who were older, of female gender, and at 2 years after KT.

**SP670** THE rs17384213 POLYMORPHISM IN THE DDAH1 GENE IS A MARKER OF CHRONIC ALLOGRAFT DYSFUNCTION

Alessandra Testa1, Gaetana Porto1, MCristina Sangueedolce1, Belinda Spotolo1, Rosamaria Parlongo1, Anna Pisano1, Giuseppe Enia1, Giovanni Tripepi1 and Carmine Zoccali1

1IBIM-National Research Council Reggio Calabria Italy

**Introduction and Aims:** Endothelial dysfunction may be involved in the development or progression of vascular lesions in Chronic Allograft Dysfunction (CAD) and Asymmetric Dimethyl arginine (ADMA) an aminoacid degraded by dimethylaminohydrazide (DDAH1 and DDAH2) has been implicated in CAD in these patients. Since Plasma ADMA levels may be confounded by various biological and environmental influences, we used a genetic approach, investigating the impact of variation in the rs17384213 polymorphism in the DDAH1 gene, to establish any causal role for the ADMA pathway in CAD. Application of this genetic approach in a previous study in two CKD cohorts negated a causal role of ADMA in CKD progression and showed an apparently paradoxical risk excess for CKD progression in patients with the allele (G) associated with low ADMA levels (Kidney Int 2010;77:459-67).

**Methods:** The study cohort included 249 patients over a pool of 277 transplant patients on follow-up in a large renal Unit serving a 700.000 residents area. The baseline eGFR (MDRD) was 54±24mL/min/1.73m2 and the median follow-up was 10,7 years (range 1-29 yrs). We used repeated measures of a quantitative trait (eGFR) as an outcome measure to maximize the study power and the average number of eGFR measurements per patient applied to calculate the individual evolution of renal failure over time was 70 (range 7 to 289).

**Results:** The genotype distribution in the rs17384213 polymorphisms in the DDAH1 gene in mediating renal function loss in chronic allograft dysfunction independent of established risk factors and proteinuria. Furthermore, these findings indicate that it is much unlikely that the link between ADMA and allograft dysfunction in previous studies be causal in nature. Full clarification of the role DDAH1 in allograft dysfunction may help the development of treatments aimed at countering chronic allograft dysfunction in transplant patients and at retarding the evolution of renal disease in CKD patients.

**SP671** ANONYMITY IN LIVING KIDNEY DONATION: AN ELTP VIEW

Willi Zuckerd1, Nizam Mamode2, Annette Lennering3, Franco Citterio4, Emma Massley5, Kristof van Assche6, Sigurd Sterck6, Michaela Frenzua6, Harald Jung7, Assaya Pascalev8, Rachel Johnson9, Charlotte Loven3, Willem Weimar10 and Frank Dör11

1Internal Medicine Erasmus MC Rotterdam Rotterdam The Netherlands, 2Department of Transplantation Guys Hospital London United Kingdom, 3The Transplant Institute Sahlgrenska University Hospital Göteborg Göteborg Sweden, 4Department of Surgery Catholic University Rome Italy, 5Research Group on NTS VUB, Ghent University Brussels, Ghent Belgium, 6Department of Systemic Philosophy Babes-Bolyai University Cluj Romania, 7University of Medicine and Pharmacy Târgu Mures Romania, 8Bulgarian Center for Bioethics Sofia Bulgaria, 9Blood and Transplant NHS Bristol United Kingdom, 10Department of Surgery Erasmus MC Rotterdam Rotterdam The Netherlands

**Introduction and Aims:** There is a wide variation in the practice of maintaining anonymity before and after living donor transplantation in both unspeciated donation (altruistic donors) and specified indirect donation (paired exchange programmes). Further debate and clarification of the ethical and normative issues is clearly required.

**Methods:** A multidisciplinary team, including transplant surgeons, physicians, ethicists, philosophers, coordinators, lawyers and psychologists was convened by the working group living donation of ELPAT section of ESOT to consider this issue. A ethicists, philosophers, coordinators, lawyers and psychologists was convened by the working group living donation of ELPAT section of ESOT to consider this issue. A
Conclusions: There was a clear recommendation that anonymity should be maintained prior to either unspecified or specified indirect transplantation. Preservation of anonymity subsequently was considered ideal, and should only be lost under carefully controlled conditions to minimise potential harm to both donor and recipient. Further studies with unspecified donors and their recipients are needed to consider when to offer the opportunity to both donor and recipient to meet each other. Reference: Anonymity and live donor transplantation: An ELI-PAT view. Mamode N. et al. Transplantation in press.

SP672 PROSPECTIVE STUDY OF BKV INFECTION AND NEPHROPATHY IN THE FIRST YEAR POST-RENAL TRANSPLANTATION

Tayebeh Soleymani1, Hossein Keyvani2, Seyed Mohammad Jazayeri3, Zeynab Fazeli4, Shipra Gharun4, Mostafa Mahabadi1, Valiollah Chegeni1, Iraj Najafi1 and Mohammad Reza Ganji1
1Nephrology Tehran University of Medical Sciences Tehran Iran (Islamic Republic of), 2Department of Virology, School of Medicine Tehran University of Medical Sciences Tehran Iran (Islamic Republic of), 3Hepatitis B Molecular Laboratory, Department of Virology, School of Public Health Tehran University of Medical Sciences Tehran Iran (Islamic Republic of)

Introduction and Aims: BK virus nephropathy (BKN) has recognized as an emerging cause of allograft dysfunction in renal transplant recipients. We aimed to assess prospectively the incidence of BKV infection and nephropathy in the first year after transplantation.

Methods: BK virus (BKV) viremia during the first year of renal transplantation was quarterly assessed in 32 consecutive recipients. BKV DNA was determined in all samples by real time PCR according to manufacturer’s instruction.

Results: Mean age of the patients was 33.28±15.29 years. Seventeen (53.1%) patients were male. Sixteen patients (50%) were received antithymocyte globulin (ATG) for induction therapy. Living donor transplant consisted of 75% of kidney donations. Maintenance immunosuppressive therapy covered ciclosporine A in 27 (84.4%) of the patients, tacrolimus in 10 (31.3%) and sirolimus for 3 (9.4%). BK virus viremia was detected in 8 (25%) patients. The highest detected plasma viral load was less than 4 log copies/ml. BK virus was respectively positive in 5 (62.5%), 2 (25%) and one (12.5%) patient during the first 4, 8 and 12 months after transplantation. Biopsy proven rejection and antirejection therapy by methylprednisolone pulses were performed in 5 (62.5%), 2 (25%) and one (12.5%) patient during the first 4, 8 and 12 months after transplantation.

Conclusions: Routine screening of BKV infection particularly in centers with low prevalence of BKV nephritis may not be cost-effective in predicting this disease.

SP673 ESPN DATABASE - ADOLESCENCE AND KIDNEY TRANSPLANTATION; A HAZARDOUS COMBINATION?

Karlín M.E. Meys1, Jaap W. Groothoff2, Kitty Jager3, Franz Schaefer1, Karlien M.E. Meys1, Jaap W. Groothoff2, Kitty Jager3, Franz Schaefer1
1Dept. of Pediatric Nephrology Erasmus MC Sophia Children’s Hospital Rotterdam The Netherlands, 2Dept. of Pediatric Nephrology AMC Emma Children’s Hospital Amsterdam The Netherlands, 3Dept. of Pediatric Nephrology Heidelberg University Hospital Heidelberg Germany, 4Dept. of Pediatric Nephrology Centro Hospitalar do Porto Porto Portugal

Introduction and Aims: It is widely believed but sparsely documented that transplant survival is poorest during adolescent age. We sought to determine the function and survival of kidney allografts in adolescents; compared to both younger children and young adults, across Europe.

Methods: We used information available from the ESPN/ERA-EDTA and ERA-EDTA Registries regarding first time transplant recipients between 1995 and 2010 aged 6 to 30 years.

Results: A total of 6,064 transplant recipients, thereof 1,086 performed pre-emptively, from 30 European countries were analysed. Female patients transplanted between the age of 16 and 20 years had a poorer 10-year graft survival than either younger (HR 2.8, 95%CI 1.5-5.2 p<0.001) or older females (HR 1.7, 95%CI 1.4 – 2.2 p<0.001) and compared to males of the same age (HR 1.4 95%CI 1.0-1.9, p=0.05). Among the males differences were less obvious, although adolescent boys had a higher risk of graft loss than any other age group. Turning 17 highly increased the risk of graft loss among young females when included in a time dependent analyses. This risk is irrespective of age of transplantation, cause of renal failure, donor source and pre-emptive transplantation. Turning 17 was more predictive than the age of transplantation.

Conclusions: Females undergoing transplantation between 16 and 20 years of age are at very high risk of graft loss. We speculate that because of cosmetic or social reasons medication adherence may be lower in this age group.

SP674 KIDNEY TRANSPLANTATION AMELIORATES CARDIAC FUNCTION IN DIABETIC PATIENTS WITH END-STAGE RENAL DISEASE

Encumert Gurülier1, Nazim Güres1, Altan Alim1, Alhan Gurkan1, Ufuk Canik1 and Ibrahim Berber1
1Transplant Center Acibadem University Istanbul Turkey

Introduction and Aims: Cardiac disorders are very common in individuals with chronic kidney disease and are associated with high morbidity and mortality. The purpose of this study was to evaluate the impact of successful kidney transplantation on chronic and diastolic ventricular dysfunction in patients with end-stage renal disease (ESRD).

Methods: We prospectively evaluated 200 patients with ESRD, immediately before and one year after transplantation, using tissue Doppler echocardiographic study.

Results: The mean age was 41.6 years and 40% of patients were diabetic. We observed a reduction in left ventricular diastolic diameter (52.3 to 49.5 mm, p = 0.021) after kidney transplantation. The ejection fraction increased compared to basal assessment (69.7% vs. 45.8%, p < 0.01). The prevalence of diastolic dysfunction decreased 47% during the evaluated period. Amelioration of all of the parameters mentioned above was even better in diabetic patients.

Conclusions: Kidney transplantation is known to lead a considerable improvement in left ventricular systolic and diastolic function of patients with ESRD, and the results seem even more optimistic for the diabetic patients who have higher mortality on dialysis.

SP675 INSULIN HYPOSECRETION PREDICTS NON-RESPONSE TO MAGNESIUM SUPPLEMENTATION IN RENAL TRANSPLANT RECIPIENTS

Steven Van Laecke1, Rogier Caluwé2, Evi Naglier1, Wim Van Biesen1, Patrick Peeters2, Bruno Van Vem2 and Raymond Vanholder1
1Department of Internal Medicine Renal Division Ghent University Hospital Ghent Belgium, 2Department of Internal Medicine Renal Division OLV Aalst Aalst Belgium

Introduction and Aims: Insulin resistance and hyposecretion are common after transplantation. Hypomagnesemia is associated with glucose metabolism disorders in both transplanted and non-transplanted patients. Magnesium (Mg) supplements previously improved insulin sensitivity in non-diabetic overweight persons in the general population. We aimed to prospectively evaluate the effect of magnesium supplementation on insulin resistance and secretion.

Methods: Two-center, randomized, 6-month, open label trial comparing Mg supplementation (magnesium oxide 450mg up to thrice daily) aiming at normalizing serum Mg concentration (n=24) or versus no Mg supplements (n=24) in non-diabetic renal transplant recipients with serial hypomagnesemia (<1.8mg/dL) > 4 months after transplantation after a 2 week washout of Mg supplements. Magnesium non-response was defined as absence of increase of serum Mg at month 3. We performed an OGTT with measurement of AUC of glucose by the trapezoidal rule, insulin resistance (HOMA) and derived insulin secretion indices baseline and 6 months after inclusion (primary outcome) next to Hba1c (secondary outcome).

Results: 52 patients were originally randomized, of which 4 had early drop-out due to dyscompliance. Mean age was 53±12.3 in a 60.4% male population with mean BMI of 23.7±3.9 and a median time of 51(26-86) months after transplantation. Magnesium supplementation (mean dose 688.5±236.9mg) was unable to increase serum Mg (Δ0.08±0.14 vs 0.11±0.18mg/dL) nor intra-erythrocytic magnesium (Δ0.25±0.46 vs 0.18±0.2mg/dL) more than controls. Tacrolimus levels were higher at month 6 in the treatment vs. the control group (0.25±0.46 vs 0.18±0.82mg/dL) more than controls. Tacrolimus levels were higher at month 6 in the treatment vs. the control group (7.92±2.48 vs 6.59±1.75 ng/ml; p=0.042). Alterations in HOMA-IR, first phase insulin secretion (FPIR), second phase insulin secretion (SPIR), deposition index (DI) and Hba1c were non-significant. Magnesium non-responders had lower FPIR (1032±588 vs 1544±982; p=0.049), SPIR (283±141 vs. 388±243; p=0.07) and this difference persisted 6 months after randomization with a lower FPIR (971±1604 vs. 1544±982; p=0.003) and a higher AUC glucose (146±25.1 vs. 122±12.5; p=0.044). Non-responders had no differences in HOMA-IR nor DI. No patients discontinued supplementation due to adverse events.

Conclusions: Ineffectiveness of oral Mg supplementation to alter Mg status pre-emptively of oral Mg supplementation to alter Mg status pre-emptively pre-emptively pre-emptively of oral Mg supplementation to alter Mg status pre-emptively prevents evaluation of potential effects on glucose metabolism. This ‘Mg resistance’ is predicted by insulin hypersecretion, suggesting that hypomagnesemia is a consequence of a disturbed glucose metabolism rather than a cause. Trial registration Clinical Trials NCT01291030.
Patients were diagnosed with multiple neoplasms. In dialyzed group 13 neoplastic therapy were collected.

Results:

Introduction and Aims: The clinical dermatological examination was performed in 484 patients after renal transplantation (early detection of skin changes in RTR program) consisted of 295 man and 189 woman in the mean age 46.1 +/- 13 years with median time after transplantation 74.3 +/-52.1 months. The group of 112 dialyzed patients (57 male and 55 female) aged 57.4 +/- 15.4 years without history of immunosuppressive therapy and systemic underestimation of the glomerular filtration rate (GFR) by the Modified of the new equation for kidney function at 3 months after KT in patients undergoing KT. To analize the relationship that may exist between both (inflammation and oxidation) with mortality.

Methods: 196 KT between 2003 and 2008. 68% were male. Medium age: 51,89±12,54 years old. Time on dialysis: 28,64±24,76 months. 19,8% had diabetes mellitus (DM) and 14,8% had cardiovascular disease (CVD) before KT. We analyze the inflammation markers ([MIP]; C-reactive protein (CRP), interleukin 6 (IL-6), tumoral necrosis factor α (TNFα), soluble receptor interleukin 2 (sR-IL2) and soluble receptor TNFα (sR-TNFα); and oxidation markers: oxidized low-density lipoprotein (oxLDL), anti-oxidized low-density lipoprotein antibodies (oxLDL Abs); before and at 3 months after KT. We calculated glomerular filtration rate by MDRD formula.

Results: Global mortality until April 2012 was 13,8% (27/196). Patients who died had a worse inflammation state at 3 months after KT: IL-6 (8,23±6,45 vs 6,15±5,53; p=0,012), sR-TNFα (6,12±3,57 vs 4,35±3,48; p=0,015), and an impaired renal function: MDRD 34,5±12,47 vs 47,91±15,74;p<0,0001). There were no statistically significant differences between inflammation markers previous KT. 41,7% of KT patients who died had oxLDL Abs higher than P<0,770U/l/ml (p=0,046). The multivariate analysis, using as a dependant variable the mortality and as covariants: age, gender, DM previous KT, CVD previous KT and all these values at 3 months after KT: MDRD, IL-6, sR-TNFα, oxLDL (P<0,05), show as independent risk factors of mortality: age [OR1,068, IC95%: 1,011-1,127; p=0,018], oxLDL Abs [OR3,27, IC95%: 0,980-10,96; p=0,054], oxLDL Abs P<0,05 [OR6,99, IC95%: 2,10-23,28] and MDRD [OR: 0,910, IC95%: 0,863-0,959; p<0,000].

Conclusions: Kidney transplant patients with a worse inflammation state, a worse oxidation state and with an impaired renal function have a higher mortality. Independent predictive variables of mortality were age, oxidation markers and kidney function at 3 months after transplantation.
significantly higher rate in low-GNRI group (P<0.05) after transplantation. There were no significant relationship between GNRI and the episodes of post-operative complications such as DGF, graft loss, infection, and cardiovascular events.

Conclusions: Kidney transplantation promoted the better post-operative recovery in nutritional status and inflammation condition particular in malnourished patients, while the rapid nutritional recovery might induce the new-onset impaired glucose metabolism. GNRI is available for the pre and post-transplant nutritional evaluation and expected to be a predictive marker for post-operative diabetes mellitus (PODM).

**IS BASILIXIMAB INDUCTION, A NOVEL RISK FACTOR FOR NEW ONSET DIABETES AFTER TRANSPLANTATION FOR LIVING DONOR ALLOGRAFT RECIPIENTS?**

Narayan Prasad1, Desraj Gurjer1, Dharmender Bhadouria1, Amit Gupta1, Raj Sharma1 and Anupama Kaul1
1Nephrology Sanjay Gandhi Postgraduate Institute of Medical Sciences Lucknow Uttar Pradesh India

Introduction and Aims: Basiliximab by affecting populations of T lymphocytes indirectly affects β-cell function leading to impaired glucose homeostasis. The study was aimed to prove basiliximab induction, as a novel risk factor for new onset diabetes after transplant (NODAT) in renal transplant recipients.

Methods: In this prospective observational study, we included renal allograft recipients from 1st July 2007 to 31st July 2011 at our tertiary care institute. The overall incidence of hyperglycemia [transient hyperglycemia, impaired fasting tolerance (IFT), impaired glucose tolerance (IGT) and NODAT] was compared between two groups of patients with and without Basiliximab. The risk factors predicting NODAT were also analyzed on multivariable logistic regression analysis. NODAT was labeled when the diagnosis of diabetes mellitus was confirmed with fasting plasma glucose ≥126mg/dl or 2-hour plasma glucose ≥200mg/dl. IGT was defined as 2-hour plasma glucose ≥140mg/dl and IFT was defined by fasting plasma glucose ≥110mg/dl and ≤126mg/dl. The patients with IFG, IGT, and those with occasional rise in blood sugar level on monitoring with glucometer and requiring insulin therapy were leveled as transient hyperglycemia.

Sample size and power of the study calculation: With assuming null hypothesis of no difference in percentage of NODAT between patients with and without Basiliximab induction, with maximum allowable difference between these proportions that still results in equivalence of 30% and the actual difference of the proportions of 15%, 400 sample sizes of the study population were required to achieve 98% power of the study that is the power to reject the null hypothesis. We had 439 eligible patients for data analysis after exclusion in our study.

Results: Of the 439 eligible study patients, 105 patients received Basiliximab and 334 patients did not. Overall hyperglycemia (transient hyperglycemia, IFT, IGT and NODAT) was detected between two groups of patients with and without Basiliximab. The risk factors predicting NODAT were also analyzed on multivariable logistic regression analysis. NODAT was labeled when the diagnosis of diabetes mellitus was confirmed with fasting plasma glucose ≥126mg/dl or 2-hour plasma glucose ≥200mg/dl. IGT was defined as 2-hour plasma glucose ≥140mg/dl and IFT was defined by fasting plasma glucose ≥110mg/dl and ≤126mg/dl. The patients with IFG, IGT, and those with occasional rise in blood sugar level on monitoring with glucometer and requiring insulin therapy were leveled as transient hyperglycemia.

**EFFECTIVENESS AND SAFETY OF ENZYME REPLACEMENT THERAPY (ERT) IN A COHORT OF KIDNEY TRANSPLANT RECIPIENTS WITH FABRY DISEASE**

Markus Cybulski1, Michael West2, Kathy Nicholls3, Joan Torras4, Gere Sunder-Plassmann5 and Sandro Ferruzzi6
1Nephrology and Rheumatology FGM Center of Internal Medicine Muelheim Germany, 2Department of Medicine Dalhousie University Halifax Canada, 3Department of Medicine Royal Melbourne Hospital and University of Melbourne Melbourne Australia, 4Nephrology Hospital of Bellvitge (IDIBELL) Barcelona Spain, 5Department of Nephrology Medical University Vienna Austria, 6Nephrology Bellècote Hospital Viterbo Italy

Introduction and Aims: Enzyme replacement treatment (ERT) was introduced in 2001 as a treatment option for patients with Fabry disease. The Fabry Outcome Survey (POS, sponsored by Shire HG7) is a worldwide outcome database, established to monitor long-term effectiveness and safety of agalsidase alfa ERT. This analysis presents clinical data in kidney transplant recipients (KTRs) with Fabry disease.

Methods: This study presents clinical data for Fabry disease (KTRs) with Fabry disease. The effects of long-term agalsidase alfa (0.2mg/kg every other week; Shire HG7) on renal outcomes were analyzed using Fabry patients' data in POS. Estimated glomerular filtration rate (eGFR) was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation. Hypertension was considered a blood pressure measurement of >120/80 mm Hg.

Results: Renal function was analysed in 93 KTRs (78 ERT treated before or after transplant surgery; 15 untreated). For the 78 treated KTRs, 53 (67.9%) had kidney transplants before start of treatment; the mean (SD) time on ERT until last visit was 3.7 (3.1) years. During the observation time, the average eGFR slope per year declined numerically steeper in untreated versus treated patients in both males and females. Further comparison promoted untreated and treated cohorts should be avoided as the latter were predisposed to more severe disease at baseline, and follow-up measures were not corrected for this difference. Patients with baseline proteinuria had poorer graft function at follow-up compared with patients without baseline proteinuria. Additionally dialysis treatment before transplantation had no negative impact on later graft function. Only minor side effects or infusion-related reactions were reported.

Conclusions: In this study, graft function in KTR Fabry patients remained stable on ERT.

**RISK FACTORS ON SURVIVAL OF RENAL TRANSPLANT WAITING LISTED PATIENTS: A SINGLE CENTRE ANALYSIS FROM DATA OF RENAL REGISTRY**

Stanley Lo1, Patrick Y.H. Wong1, David Ip1, C.K. Wong1, Vincent C.C. Chow1 and Stephen K.L. Mo1
1Department of Medicine Pamela Youde Nethersole Eastern Hospital Hong Kong SAR China

Introduction and Aims: Renal transplantation may offer survival benefit over dialysis for end stage renal failure patients who were registered in transplant waiting list. Hong Kong Renal Registry records data of end stage renal patients who are put on transplant waiting list and who received a renal transplant. We aim at examining the risk factors associated with survival of patients who are put on renal transplant waiting list.

Methods: All the patients who were put on renal transplant waiting list and who received a renal transplant graft between Jan 1994 and Oct 2012 were identified by the Organ Transplant Registry System of a regional hospital in Hong Kong East cluster and were recruited to the analysis. Life table was created for the survival after initiation of renal replacement. Cox regression hazard model was used to identify independent risk factors for survival of patients. Analysis was performed in a intention-to-treat manner.

Results: 813 end stage renal failure patients were identified. After excluding 155 patients who had a pre-empted renal transplant, 658 patients were recruited for the analysis. Three patients received 2 or more renal grafts. 210 (31.9%) patients received a renal transplant while 448 (68.1%) patients continued to have dialysis. 237 (36%) patients died. Mean age of renal replacement therapy was 46.5 ± 11.9 years. Female-to-male ratio was 1.84. At the time of renal replacement, the prevalence of DM, HT, ischemic heart disease and stroke were 42.7%, 80.9%, 24.6% and 3.8%, respectively. Mean duration of renal replacement was 6.5 ± 5.0 years. 1, 5, 10 and 15 year survival (graft failure censored) of renal transplant were 98.6%, 92.2%, 81.8% and 74.4%, while those receiving dialysis were 90.6%, 57.1%, 28.8% and 22.3%, respectively. Cox model showed 3.1% increase in risk of death for every year increment in age at the time of renal replacement (p=0.000). DM status and history of ischemic heart disease increased risk of death by 2.2 fold (p=0.000) and 1.37 folds, respectively. Continuing dialysis was associated with higher risk of death (HR=5.24; p=0.000) compared with renal transplant recipients.

Conclusions: Renal transplant offers significant survival benefit in end stage renal patients who were put on transplant waiting list. Age at the initiation of renal replacement, history of DM and ischemic heart disease were independent risk factors.

**RED CELL DISTRIBUTION WIDTH IS ASSOCIATED WITH MORTALITY IN KIDNEY TRANSPLANT RECIPIENTS**

Miklos Molnar1,2, Akos Ujjasvari1, Maria E. Czira1, Marta Novak1 and Istvan Mucsi1
1Institute of Pathophysiology Semmelweis University Budapest Hungary, 2University of Toronto Toronto ON Canada, McGill University Health Centre Montreal QC Canada

Introduction and Aims: Red cell distribution width is an important laboratory parameter for risk assessment in kidney transplant recipients. Red cell distribution width was associated with mortality in kidney transplant recipients.
Introduction and Aims: Red Cell Distribution Width (RDW), a parameter routinely reported as part of the complete blood count (CBC), is associated with increased morbidity and mortality risk in different patient populations. No published data are available about the association between RDW and mortality in kidney transplant recipients. Methods: We collected socio-demographic, clinical parameters, medical and transplant history and laboratory data at baseline in 723 prevalent kidney transplant recipients between June and October 2008 (mean age 51 ± 13 [SD] years, 56% men, 21% diabetics). Associations between baseline RDW values and all-cause mortality over 3 years were examined in unadjusted and adjusted models (adjusted for: estimated glomerular filtration rate [eGFR], age, gender, iron status markers, hemoglobin, serum albumin, C-reactive protein, abdominal circumference, Charlson Comorbidity index, total time in ESRD, steroid use, mammalian target of rapamycin [mTOR] use, ACEI or ARB use, iron and folic acid supplementation). Results: Of the 723 participants 81 patients died and none were lost to follow-up during a median follow-up of 35 months. The unadjusted mortality rate was significantly higher among patients in the "high" (≥median) RDW group (crude mortality rates in the "high" group: 67.4/1000 patient-years [95%CI: 54.1-84.1]; "low" RDW group: 20.5/1000 patient-years [95%CI: 13.3-31.1]; p=0.001). Increasing RDW was associated with increased mortality in both unadjusted ([HR1% increase = 1.63; 95% CI: 1.41-1.89] and [HRmedian = 2.74; 95% CI: 1.68-4.48]) and fully-adjusted models ([HR1% increase = 1.60; 95% CI: 1.27-1.89] and [HRmedian = 1.33; 95% CI: 0.76-2.35]). The association of RDW with mortality was uniformly increasing when modeled as a continuous variable and using fractional polynomials and cubic splines in our unadjusted (Figure) model. In reclassification analyses RDW improved the predictive value of all-cause mortality prediction models (the net reclassification improvement [NRI]) was (NRI=0.198; p<0.001). Conclusions: Our prospective cohort study demonstrated that higher RDW is a significant predictor of mortality in prevalent kidney transplant recipients. RDW provides additional prognostic information of mortality in addition to known risk factors and co-morbid conditions. RDW should be included in risk prediction models in order to better estimate mortality risk in kidney transplant recipient.

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STERILE LEUKOCYTURIA PREDICTS INTERSTITIAL FIBROSIS AND TUBULAR ATROPHY IN KIDNEY ALLOGRAFT PROTOCOL BIOPSIES

Josep M. Cruzado, Silvia Coeelho, NA Víria Porta, Oriol Bestard, Edoardo Mellili, Omar Tacó, Israel Rivas and Josep Girón

1Nephrology Bellvitge Hospital L’Hospitalit Llobregat Spain, 2Nephrology Hospital Fernando Fonseca Lisbon Portugal, 3Biostatistics IDIBELL L’Hospitalit Llobregat Spain

Introduction and Aims: In kidney allograft protocol biopsies the presence of interstitial fibrosis and tubular atrophy (IFTA) is associated with graft loss. In this study we sought to investigate the association of sterile leukocyturia (Leu+) with Banff criteria histological findings. Methods: In this retrospective study we have evaluated all kidney biopsies performed in our Institution between January 2006 and July 2010 who had an available urinalysis at the time of 6-month protocol biopsy. Patients who had a comitant positive urine culture were excluded. Renal lesions were diagnosed according to the 2007 update of Banff 1997 classification. Results: We identified 189 patients with six-month protocol biopsy. Eleven were excluded because of insufficient histological sample, 47 because of unavailable urinalysis and 12 because of urinary tract infection. From the 119 evaluable cases, 24 displayed Leu+. Risk factors for Leu+ were female gender, deceased donor (DD) and delayed graft function (DGF). Renal function was similar between Leu+ and Leu– (Scr 123±49 vs 127±49 mcmol/L). Regarding kidney graft histology findings, Leu+ was only associated with IFTA (75% vs 54%, P = 0.02). The overall IFTA prevalence in our study population was 54.6%. By univariate analysis risk factors for IFTA were Leu+, donor and recipient age, DGF, retransplantation and acute rejection. By multivariate analysis donor age (OR 1.036; P < 0.01) and Leu+ (OR 2.74; P = 0.05) were the only predictors of IFTA. Sensitivity and specificity of Leu+ for predicting IFTA was 84% and 89% respectively. Nevertheless, the probability of IFTA with Leu+ and donor age ≥ 60 yr was 90% whereas it was 40% with Leu- and donor age < 60 yr. Conclusions: In the current oncoa era our results suggest that classical urinary sediment still provides valuable clinical information for the follow-up of kidney allograft recipients.

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NEPHROLOGISTS’ PERCEPTIONS OF DISPARITIES IN KIDNEY TRANSPLANTATION: AN INTERNATIONAL SURVEY

Nasrollah Ghahramani, Zahrnia Karparvar, Shahrouz Shadrou and Mehrdad Ghahramani

1Medicine Pennsylvania State University College of Medicine Hershey PA United States

Introduction and Aims: Disparities in the rate of kidney transplantation (KT) have been well-recognized. While factors relating to provider perceptions are potentially important contributors to disparities, nephrologists’ perceptions regarding this issue have not been well-explored. Using a web-based survey, we investigated the perceptions of nephrologists, worldwide, about causes of disparities in KT. Methods: The relation between perceptions and demographic characteristics of 1280 nephrologists (age >50: 40% female; 28% rural practice; 11% academic; 63% transplant nephrologist; 51%) from 74 countries were examined by univariate and multivariable analyses. Results: Socioeconomic factors were perceived as the most important causes of disparities in KT among 50% of the respondents. Nephrologists from Africa (OR: 2.36; 95% CI: 1.23 to 4.53; p<0.01) and the Indian Subcontinent (OR: 2.34; 95% CI: 1.23 to 4.30; p<0.01) were more likely, while those from Western Europe (OR: 0.23; 95% CI: 0.15 to 0.35; p<0.001) were least likely to consider socioeconomic factors as the most important factors leading to disparities. Patient age was considered by 27% of the respondents as the most important factor leading to disparities in KT. Nephrologists...
practice in rural areas were less likely (OR: 0.34; 95% Cl: 0.19 to 0.73; p=0.004), while those with >10 years of practice were more likely (OR: 1.36; 95% Cl: 1.01 to 1.83; p=0.04) to perceive patient age as the most important factor in disparities. Nephrologists from Western Europe were more likely (OR: 3.41; 95% Cl: 1.30 to 11.30; p=0.001), while those from North America were less likely (OR: 0.33; 95% Cl: 0.23 to 0.46; p=0.001) to consider age as the most important factor in disparities. Patient race was perceived as the most important factor leading to disparities among 12% of the respondents. North American (OR: 3.36; 95% Cl: 2.37 to 4.88; p<0.001) and Australian (OR: 3.41; 95% Cl: 1.60 to 7.26; p= 0.001) nephrologists were more likely and those from Latin America (OR: 0.30; 95% Cl: 0.13 to 0.70; p<0.01) and the Middle East (OR: 0.19; 95% Cl: 0.05 to 0.80; p=0.02) were less likely to perceive race as the most important factor leading to disparities in KT. Rural residence, gender and urban city residence were perceived as the most important factors by 6%, 3% and 2% of the nephrologists, respectively.

Conclusions: We conclude that socioeconomic factors are considered the most important causes of disparities in transplantation among the majority of nephrologists, worldwide. Other factors perceived as important contributors to disparities include patient age and race. There are significant geographic and demographic differences in perceptions of causes of disparities among nephrologists. Cultural factors, training and demographic backgrounds of nephrologists are likely contributors to these differences in perception.

Introduction and Aims: Kidney transplantation is currently the best treatment for end stage renal diseases. Traditionally survival analyses after kidney transplantation include enrolment like graft loss and death with functioning graft. Several studies investigated risk factors for early death of patients with a functioning kidney allograft. Little is known about the survival after losing graft function and returning to dialysis. For this purpose we analysed 1901 patients who were primarily transplanted between December 1968 and December 2010.

Methods: The primary end-point was death after graft loss (GL). Therefore, we calculated annual mortality rates during transplant period and afterwards. The Kaplan-Meier analysis was used to display the survival curve after GL. A multivariate Cox model was used to identify risk factors for death after GL. For this analysis follow-up started at the time of GL. The web-based patient file „TBase“ was used to retrieve data. If the outcome of patients was uncertain the families were contacted to obtain follow-up information. Only the first transplantation was included in the analysis.

Results: Of the 1901 patients 662 lost their graft. The median follow-up of patients after GL was 83 months (range 0-431 months). Patients without GL (n=1239) had a median follow-up of 98 months (range 0-463). Two hundred twenty patients died during follow-up after GL (23% of cardiovascular disease, 20% of infections, 14% of malignancies and 13% of other causes; in 30% no definite cause of death could be evaluated). Two hundred ninety eight patients died with functioning graft. Annual mortality rate after GL was significantly higher than in patients without GL (3.8 vs. 2.3, respectively). Age > 50 years at transplantation (RR 1.3) and age > 50 years at GL (RR1.8), and diagnosis of hepatitis B (RR 1.4) were associated with a significantly higher risk of death after GL. A shorter transplantation period (<7 years) seems to be protective against death after GL (RR 0.7). Longer time (>2 years) on dialysis and the diagnosis of diabetes mellitus led to a tendency towards increased risk of death (RR 1.3; P=0.054 and RR 1.32; P=0.09, respectively).

Conclusions: Patients who lose their kidney graft have a significantly higher risk of death than patients who do not. Understandably, age at the time of transplantation and age at the time of GL are major risk factors for death after graft loss. Hepatitis B is a negative predictor, too. Interestingly a shorter duration of time being transplanted is associated with better outcome for patients who lose their grafts. It remains unclear whether the combined outcome before and after graft loss in various kidney transplantation over maintenance dialysis. Therefore an adequate dialysis control group is lacking.

Nuria Montero1, Angela C. Webster2, Ana Royuela3, Javier Zamora3, Marta Crespo1 and Julio Pascual1
1Nephrology Hospital del Mar Barcelona Spain, 2Nephrology, Epidemiology Sydney School of Public Health, Sydney Medical School, University of Sydney Sydney NSW Australia, 3Clinical Biostatistics Unit Hospital Ramon y Cajal Madrid Spain

Introduction and Aims: Pancreas or kidney-pancreas transplantation improves outcomes in diabetic patients with kidney failure, but chronic steroid treatment risks adverse events. We aimed to systematically assess safety and efficacy of steroid withdrawal (SW) or avoidance (SA) versus continuing steroid maintenance in patients receiving pancreas transplant alone (PTA), simultaneous pancreas kidney transplantation (SPK) or pancreas after kidney transplantation (PAK).

Methods: We searched the Cochrane Renal Group’s Specialised Register. We included randomised controlled trials or cohort studies of SA (steroid use of less than 14 days) or versus SW (steroid use during more than 14 days) in PTA, SPK or PAK recipients. Two investigators critically appraised methodology and abstracted study data. Metaanalyses used random effects with results expressed as risk ratios (RR) or mean difference (MD) with 95% confidence interval (CI). Cohort studies were not meta-analysed, but their findings summarised descriptively.

Results: Three RCTs enrolling 144 participants met inclusion criteria. Of these, two compared SA versus late SW and one compared late SW versus steroid maintenance. All of the three studies included SPK and only one also included PTA. All of the studies had an overall moderate risk of bias. Two studies (89 participants) that compared SA versus late SW suggested that SA had no impact on mortality (RR 1.64, 95% CI 0.21 to
12.75), risk of kidney loss censored for death (RR 0.35, 95% CI 0.04 to 3.09) or acute kidney rejection (RR 2.08, 95% CI 0.2 to 21.5). The study that compared late SW versus steroid maintenance observed no deaths, no graft losses or acute kidney rejection at 6 months in either group and suggested no difference for acute pancreas rejection (RR 0.88, 95% CI 0.06 to 13.35). We also identified 13 cohort studies. Forest plot of death at 1 year in steroid avoidance versus late steroid withdrawal.

Conclusions: Evidence for the benefits and harms of SW in pancreas or kidney-pancreas transplantation is sparse with only three RCT of 144 patients identified. Overall these demonstrated no difference in mortality, graft survival or rejection in steroid-sparing strategies but firm conclusions are not yet possible. Moreover, the 13 observational studies findings concur with the evidences found in the RCTs. There is not enough evidence to recommend steroids withdrawal in pancreas-kidney transplantation, although studies showed no differences between groups.
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**C1q-FIXING DONOR-SPECIFIC ANTIBODIES DETECTED BEFORE KIDNEY TRANSPLANTATION DO NOT PREDICT REJECTION OR EARLY GRAFT LOSS, BUT HLA CLASS I DO**

Julio Pascual1, Alberto Torio1, Virginia Masí2, María José Perez-Saaz1, Marisa Mir1, Anna Fauna1, Olga Montes-Ares3, María Dolores Checa1 and Marta Crespo1
1Nephrology Hospital del Mar Barcelona Spain, 2Immunochemistry Hospital Insular de las Palmas Las Palmas de Gran Canaria Spain, 3Immunochemistry Laboratory of Referencias de Catalunya Barcelona Spain, 4Nephrology Hospital Insular de las Palmas Las Palmas de Gran Canaria Spain

**Introduction and Aims:** Anti-HLA donor-specific antibodies (DSA) identified by Luminex are questioned for their excess in sensitivity and lack of prediction of clinical events after kidney transplantation (KT). We performed a retrospective study to evaluate if specific types of preformed DSA (class I or II or C1q-fixing) ones, have impact on graft survival.

**Methods:** KT performed between 2006–2011 across a negative mixed-lymphocytes CDC crossmatch were included (n=335). Anti-HLA antibodies were tested using Luminex Lifecodes LifeScreen and LSA Class I/II assays (Gen-probe, Stanford-CT). One Lambda commercial kits were used to confirm DSA before detection of antibodies capable of fixing complement with One Lambda’s Single Antigens Beads and C1q-Luminex Lifecodes LifeScreen and LSA Class I/II assays (Gen-probe, Stanford-CT).

**Results:** Sixty-six patients with positive screening were further tested for DSA; 28 pretransplant DSA. KT with MFI-2000 were selected to assess the capacity to fix C1q. They were 75% female, 64.3% retransplants, with a high rate of biopsy-proven acute rejection (33.8%) and acute antibody-mediated rejection (AMR=28.6%). Early graft loss (<3 months post-KT) was 14.3% and global loss 21.4% at 30 months follow-up. Renal function for surviving grafts was good (median last SCr=1.27 mg/dl). DSA were measured at 1, 3, 6 and 12 mos then yearly, DSA was measured at 1, 3, 6 and 12mos.

**Conclusions:** Sixty-six patients with positive screening were further tested for DSA; 28 pretransplant DSA. KT with MFI-2000 were selected to assess the capacity to fix C1q. They were 75% female, 64.3% retransplants, with a high rate of biopsy-proven acute rejection (33.8%) and acute antibody-mediated rejection (AMR=28.6%). Early graft loss (<3 months post-KT) was 14.3% and global loss 21.4% at 30 months follow-up. Renal function for surviving grafts was good (median last SCr=1.27 mg/dl). DSA were measured at 1, 3, 6 and 12 mos then yearly, DSA was measured at 1, 3, 6 and 12mos.

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**DONOR SPECIFIC ANTIBODIES ARE COMMON IN RENAL TRANSPLANT PATIENTS SCREENED FOR BK VIREMIA**

Deirdre Sawinski1, Jennifer Trofe-Clark1,2, Tracy Sparke2, Priyanka Patel1, Srinir Goral1 and Roy Bloom1
1Renal Medicine University of Pennsylvania Philadelphia PA United States, 2Pharmacy Hospital of the University of Pennsylvania Philadelphia PA United States

**Introduction and Aims:** Early detection of BK viremia (BKV) and donor specific antibodies (DSA) by prospective screening in renal transplant recipients (RTx) may enable prevention of BK nephropathy and rejection respectively. The effect of BKV and DSA screening on long term allograft outcomes is unclear.

**Aim:** To determine the incidence of donor DSA in RTx screened for BKV.

**Methods:** Retrospective analysis of kidney recipients transplanted Jan 2008 – Dec 2011 and prospectively screened for BKV and DSA. BKV, defined by ≥ 2.6 log copies/ml, was screened for at 3, 6 and 12 mos then yearly, DSA was measured at 1, 3, 6 and 12mos.

**Results:** Induction comprised rabbit anti-thymoglobulin (81% pts), or basiliximab (low-immunologic risk pts) and maintenance w/ tacrolimus (TAC), mycophenolate mofetil (MMF) and steroids. Detectable BKV was first treated with MMF discontinuation, followed by TAC reduction if BKV persisted. In contrast, TAC or MMF was increased for positive DSA.

**Conclusions:** BKV was detectable at least once in 106/691 (16.7%) pts. Class I and class II DSA developed in 8% and 12% of pts. Class II, but not class I DSA occurred more frequently in pts w/ than, without BKV (p=0.02). The median duration of detectable BKV was 172 (54-461) days and the number of days with BKV by DSA was strongly correlated with class II DSA (p=0.005). Median graft survival was 837 (469-1239) days. Detectable BKV had no demonstrable effect on allograft (p=0.57) or pt survival (p=0.8), but class II DSA was associated with decreased allograft (p=0.086) survival.

**Conclusion:** Both BKV and DSA were commonly detected in our cohort. BKV may be a risk factor for DSA but BKV did not clearly contribute to graft loss in our cohort. Longer follow up and further investigation are warranted.

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**ASSOCIATION BETWEEN TGFBR2 GENE POLYMORPHISM (rs2228048, Asn389Asn) AND ACUTE REJECTION IN KOREAN KIDNEY TRANSPLANTATION RECIPIENTS**

Hyun Ju Kim1, Seok Ju Park1, Tae Hee Kim2, Yang Wook Kim1, Yeong Hoon Kim1 and Sun Woo Kang1
1Nephrology Jeju University Busan Republic of Korea, 2Nephrology Wallace Memorial Baptist Hospital Busan Republic of Korea

**Introduction and Aims:** Transforming growth factor-β (TGF-β) signaling transduction initiates with TGF-β activation, and the activated TGF-β then binds with the TGF-β receptor II (TGFBR2). Any quantitative and qualitative changes in TGFBR2 will be expected to affect TGF-β signaling pathway occupying a central position in regulating cell growth, differentiation, apoptosis, immune reaction, angiogenesis and extracellular matrix formation. Recent studies have shown that TGF-β gene polymorphisms may confer susceptibility to early acute and chronic allograft rejection in kidney transplantation recipients by enhancing fibrogenesis. In this study, we assessed whether polymorphisms of the TGFBR2 gene were associated with susceptibility to kidney transplantation rejection.

**Methods:** A total of 347 renal allograft recipients transplanted at three centers in Korea were analyzed. We extracted genomic DNA from blood samples and amplified the genomic DNA using the primers for each Single Nucleotide Polymorphism (SNP). Three SNPs (rs764522, rs3087465, rs2228048) of TGFBR2 gene were genotyped from genomic DNA with direct sequencing. SNPStats, SNPAnalyzer, Helixtree, and Haplovip version 4.2 were used to analyze genetic data. Multiple logistic regression models (codominant, dominant, recessive, and log-additive) were performed to evaluate odds ratios (ORs), 95% confidence intervals (CIs), and p values.

**Results:** Acute rejection (AR) developed in 63 patients (18%). There are no significant differences in age, sex, number of HLA mismatches, cause of renal failure, and immunosuppressive regimen between the AR and non-AR group. The synonymous SNP rs2228048 was significantly associated with AR (p = 0.020 in recessive model, and p = 0.036 in log-additive model) and Fisher’s exact test (p = 0.04). Allele frequencies of rs2228048 were different between AR and non-AR groups (p = 0.026).

**Conclusions:** These results suggest that the synonymous SNP rs2228048 of TGFBR2 gene may be associated with development of AR in Korean kidney transplantation recipients.

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**IMPACT OF POST-RENAL TRANSPLANT LEUCOPLANIA ON GRAFT AND PATIENT OUTCOME**

Medhat Abdel Halim1, Osama Gheith1, Torki Al-Otaibi1, Ahmed Mousa2, Wailed Awaddeen1, Tarek Said1, Prasad Nair1 and M.F.R.N. Nampoori1
1Medical Haemolytic Alesa Organ Transplant Center Kuwait Kuwait

**Introduction and Aims:** Post renal transplant (PT) leucopania is a common clinical challenge which needs fine dose adjustment of offending drugs, management of complications and use of granulocyte colony-stimulating factor (G-CSF). Serious infections and drug manipulations may impact patient and graft outcome. Aim: To study incidence of PT leucopania, clinical management and its impact on graft and patient outcome over one year.

**Methods:** We studied PT patients operated during 2010 in our center who received thymoglobulin/or basiliximab induction, maintenance steroid, MMF and tacrolimus/or cyclosporine A; valgancyclovir (VGC) 900mg and septrin daily for 6 months. Significant leucopenia (<4000x10^9) was managed by reduction of VGC then MMF and giving G-CSF according to the response. All patients were screened for CMV infection by CMV-PCR.

**Results:** 79 patients were transplanted and divided into leucopania and non-leucopania groups (group 1 and 2 respectively). In group 1, 34.17% had at least one attack of leucopenia (<4000 x 10^9) which needed fine dose adjustment of offending drugs, management of complications and use of granulocyte colony-stimulating factor (G-CSF). Significant leucopenia (<4000x10^9) was managed by reduction of VGC then MMF and giving G-CSF according to the response. All patients were screened for CMV infection by CMV-PCR.

**Conclusions:** Over the last decade, significant increases in the number of kidney transplants, most of to be from living donors in Turkey. Our findings suggest the organizational, administrative and economic arrangements made by governments have been important contributive factors in that increase. However, further efforts appear to be needed to increase cadaveric organ donation and consequently cadaveric kidney transplants.
964.3±192.7x10^3 in group1 with significant positive correlation with TLC, p<0.009. High doses of G-CSF were given to all patients in group1 with a mean dose of 1466ugm/patient without significant side effects. There were no significant differences in demographic data especially CKD etiology, dialysis type, donor type, co-morbid conditions, induction and maintenance immunosuppression, cases with delayed graft function, BK viremia (0.6), and incidence of associated infections other than CMV. Four cases of CMV infection were detected in group1 while none were in group2 (p<0.01). There was higher number of NODAT in group1 (p<0.03) most likely due to higher maintenance doses of steroids and tacrolimus to compensate for MMF dose reduction. Mean rejection episode/patient was significantly higher in group1, p<0.03). There were no difference in graft and patient outcome at 1year, p<0.4.

**Conclusions:** MMF and VGC dose reduction due to leucopenia resulted in significantly higher rate of rejection episodes, CMV infection and NODAT. High doses of G-CSF were used safely to treat neutropenia without significant side effects.

<table>
<thead>
<tr>
<th></th>
<th>Group1 (Leucopenia)</th>
<th>Group2 (No leucopenia)</th>
<th>p value</th>
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</thead>
<tbody>
<tr>
<td>Number of renal transplant recipients</td>
<td>29 (34.1%)</td>
<td>52 (65.9%)</td>
<td>0.02</td>
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<tr>
<td>Mean total leucocytic count/year</td>
<td>4294±1488 8205±2123</td>
<td>8205±2123</td>
<td>0.0001</td>
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<tr>
<td>Valgancyclovir and septrin discontinuation</td>
<td>79.6%</td>
<td>79.6% 20.4%</td>
<td>0.0001</td>
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<tr>
<td>MMF reduction ≥50%</td>
<td>85.7%</td>
<td>14.3%</td>
<td>0.0001</td>
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<tr>
<td>Granulocyte-CSF use</td>
<td>1466ugm/patient</td>
<td>nil</td>
<td>0.0001</td>
</tr>
<tr>
<td>New onset diabetes after transplant</td>
<td>32.5%</td>
<td>19.2%</td>
<td>0.037</td>
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<tr>
<td>Rejection episode/patient</td>
<td>0.62±0.852</td>
<td>0.28±0.49</td>
<td>0.03</td>
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<tr>
<td>CMV infection</td>
<td>4</td>
<td>nil</td>
<td>0.012</td>
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<tr>
<td>Graft failure</td>
<td>3.7%</td>
<td>7.7%</td>
<td>0.44</td>
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<td>Patient outcome</td>
<td>100%</td>
<td>100%</td>
<td>NS</td>
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