was 71 ± 18 ml/min. PEs were stopped at month 9 for all, excepted the patient with partial remission which is dependent of PE (two per month). Minimal Change Disease was the only pathological feature on screening biopsies.

This study, limited by the size of the study-population, provides very encouraging results. High rate of remission was rapidly obtained by combined intensive synchronized therapy. Maintenance therapy allowed prolonged remission in the majority of patients despite PEs withdrawal. These preliminary results need to be confirmed in a larger study.

Disclosure of Financial Relationships: nothing to disclose

F-PO1516

Course of Henoch-Schönlein Purpura after Renal Transplantation: A Multicenter Analysis Nada Kanaan,[1] Georges Mourad,[2] Eric Thervet,[3] Patrick Peeters,[4] Maryvonne Hourmant,[5] Yves Vanrenterghem,[6] Martine De Meyer,[1] Jef Struyven,[1] Eric Goffin,[1] Yves Pirson.[1] *[1]Nephrology, Cliniques Universitaires St. Luc, Brussels, Belgium; [2]Nephrology, CHU Montpellier, Montpellier, France; [3]Nephrology, Hopital Necker, Paris, France; [4]Nephrology, U.Z. Gent, Gent, Belgium; [5]Nephrology and Clinical Immunology, CHU Nantes, Nantes, France; [6]Nephrology, U.Z. Gasthuisberg, Leuven, Belgium.*

Aim of the study: In 1994, we found an actuarial risk for clinical recurrence and graft loss due to recurrence of Henoch-Schönlein purpura (HSP) after renal transplantation (TP) as high as 35 and 11% at 5 years. Whether these rates have changed in the current immunosuppressive era is the aim of this study.

Methods: Patients charts from six TP centers were reviewed. Inclusion criteria were (1) either a history of ≥ 2 clinical signs of HSP or a history of ≥ 1 clinical sign together with a kidney biopsy characteristic of HSP; (2) a potential follow-up of ≥ 3 years. Histological recurrence in the graft was defined as IgA deposits. Renal recurrence was defined as histological recurrence with µhematuria. Clinical recurrence was defined as renal recurrence and/or systemic signs of the illness.

Results: Forty-six patients met the inclusion criteria (58 grafts). Patient survival was excellent: 98, 95 and 89% at 5, 10 and 15 years, respectively. Overall graft survival rates were 84, 66 and 53% at 5, 10 and 15 years, respectively. Twenty one grafts were lost from : primary non function (n=1), patient death (n=2), chronic rejection (n=14), HSP recurrence (n=4). Isolated histological recurrence was documented in 7 of the 22 grafts biopsied, a prevalence rate of 32%. Clinical recurrence in a first kidney transplant occurred in 5 patients, a prevalence rate of 11%. Three *first* transplants were lost from HSP recurrence 19 to 157 months after TP. Actuarial risk for clinical recurrence in a first graft is 2.4 and 11.8% at 5 and 10 years. Actuarial risk for graft loss due to recurrence of HSP in a first graft is 2.4 and 7.8% at 5 and 10 years.

Conclusion: Recurrence rates of HSP after TP are lower than in our previous study. The actuarial risk of graft loss from recurrence in a first graft is about 8% at 10 years.

Disclosure of Financial Relationships: nothing to disclose

F-PO1517

Pregnancy after Pancreas-Kidney (PK) Transplantation Serban Constantinescu,[1] Michael Schiraldi,[1] Lisa A. Coscia,[2] Michael J. Moritz,[3] Vincent T. Armenti.[2] *[1]Medicine, Temple University School of Medicine, Philadelphia, PA; [2]Surgery, Thomas Jefferson University, Philadelphia, PA; [3]Surgery, Lehigh Valley Hospital, Allentown, PA.*

The National Transplantation Pregnancy Registry (NTPR) analyzed 75 pregnancies (77 outcomes, two sets of twins) in 43 female PK transplant recipients. Data were collected via questionnaires, phone interviews and hospital records. Of the 77 outcomes there were 54 (70%) livebirths (LB), 18 (23%) spontaneous abortions (SA), 3 (4%, one twin reduction) therapeutic abortions and 2 (3%, one twin) ectopic pregnancies. The mean transplant to conception interval was 4.1±2.8 yrs. Immunosuppression during the 75 pregnancies was cyclosporine based in 46 (Sandimmune® 25, Neoral® 19, Gengraf® 2), tacrolimus based in 28 and mycophenolate mofetil (MMF) monotherapy in 1. Six pregnancies (5 SA, 1 LB no malformations) had MMF exposure and 1 pregnancy (SA) had sirolimus exposure. Comorbid conditions during pregnancy included: hypertension 66%, infection 46%, preeclampsia 33%, and rejection 6%. Four recipients (all with adequate P function prior to pregnancy) reported insulin use during pregnancy. Mean gestational age of the liveborn was 34±3.2 wks; 78% were premature (<37 wks). The mean birthweight was 2087±709 g; 63% were low birthweight (<2500 g). At last follow-up, (mean 7.3±3.7 yrs), 49 (94%) of the children were reported healthy and developing well; three children were medicated for attention deficit hyperactivity disorder and 2 required surgery (undescended testicle 1, and atrial septal defect 1). There was one neonatal death (26 wks) due to sepsis. After 8.3±4.0 yrs of follow-up, adequate function of both grafts was reported by 27 of 43 (63%) recipients; 4 died and 12 had varying degrees of graft dysfunction/loss.

CONCLUSIONS: Pregnancy after PK transplant appears to be well tolerated, with a low incidence of insulin use and rejection during pregnancy, and 70% of pregnancies resulting in a livebirth. The high incidences of hypertension, preeclampsia, and infection underscore the high-risk nature of pregnancy in PK recipients. The impact of pregnancy on long-term maternal graft function is under further investigation.

Disclosure of Financial Relationships: grant/research support: Novartis Pharmaceuticals, Corp., Astellas Pharma, US, Inc., Roche Laboratories Inc., and Wyeth Pharmaceuticals

F-PO1518

Clinical Relevance of Pre-Transplant Adiponectin Levels for Prediction of New-Onset Diabetes after Transplantation Amir Kazory, Cécile Courivaud, Gilles Dumoulin, Jean-Marc Chalopin, Didier Ducloux. *Department of Nephrology, Dialysis, and Renal Transplantation, University of Franche-Comté, Besançon, France.*

Background: Adiponectin is an adipose-specific protein with insulin-sensitizing and anti-atherogenic properties. It is not known if pre-transplant plasma adiponectin levels can be used to predict new onset diabetes after transplantation (NODAT).

Methods: We performed a case-control study to evaluate the potential role of adiponectin as a risk factor for NODAT independent of obesity, and then to find a threshold for identification of high-risk patients. Forty nine patients with NODAT were compared to 49 transplant patients (controls), matched for age, gender, body mass index at the time of transplant, and the calcineurin inhibitor agent used for immunosuppression. Adiponectin levels were also measured in thirty five healthy subjects with normal renal function.

Results: Adiponectin levels were significantly higher in pre-transplant patients compared with healthy subjects (13.9 +/- 5.8 µg/ml vs. 9.5 +/- 4.2 µg/ml; p<0.0001). In multivariate analysis, higher plasma adiponectin level was protective against NODAT (incidence rate ratio 0.86 [95% CI 0.78-0.95], p=0.002). All patients with adiponectin levels ≤ 7 µg/ml (8.2%) developed NODAT. This corresponds to a positive predictive value of 100%. None of the patients with adiponectin values ≥ 22 µg/ml (7.1%) developed NODAT. For a threshold of 10 µg/ml (27.6%), more than one third of patients developed NODAT; the adjusted incidence ratio was 4.29 [95% CI 1.62-11.47] (p=0.004).

Conclusion: This study shows that low pre-transplant plasma adiponectin levels are associated with a higher risk for subsequent development of NODAT. These results suggest that a threshold of 10 µg/ml can identify high-risk patients for whom preventive interventions might be employed.

Disclosure of Financial Relationships: nothing to disclose

F-PO1519

Oxalate Deposits in Kidney Allograft Biopsies within the First Year Post Transplant and Graft Dysfunction Serena M. Bagnasco,[1] Maria Teresa Gandolfo,[1] Basim Mohammed,[1] Haresh Mani,[4] Mark Haas,[1] Lorraine C. Racusen,[1] Robert A. Montgomery,[3] Edward Kraus.[2] *[1]Pathology, Johns Hopkins University, Baltimore, MD; [2]Medicine, Johns Hopkins University, Baltimore, MD; [3]Surgery, Johns Hopkins University, Baltimore, MD; [4]Laboratory of Pathology, NCI-NIH, Bethesda, MD.*

Oxalate deposition in the transplanted kidney can cause acute renal failure in allograft recipients, but little is known about its frequency and long term impact on graft function. Of 1621 renal allograft biopsies reviewed at JHU between 2000 and 2006, oxalate crystal deposits were observed in 76 transplant biopsies from 63 of 680 transplant recipients within the first year from transplantation. Of these 63 recipients, 88% showed acute tubular injury in the early post-transplant period.

In 33 patients oxalate was present beyond the first month post-transplant. The average number of oxalate crystals was 1.3±1.2 per mm^2 of biopsy tissue. A significant inverse correlation between oxalate density and renal function was observed from the first two weeks (P=0.008), up to two years post-transplant (P=0.037). Significant increase in tubular atrophy and interstitial fibrosis (P<0.0001) was noted in repeated biopsy performed about 19 weeks apart in 22 of these patients. Fourteen patients died within three years from transplant, and had a higher oxalate density (P=0.047) than the average in the whole group. Our findings suggest that presence of oxalate crystals in kidney allografts can occur in about 9% of graft recipients, and has a negative impact on renal function of allograft recipients beyond the early post-transplant period.

Disclosure of Financial Relationships: nothing to disclose

F-PO1520

Decreased Expression of Kidney Parenchymal Transcripts Discriminates between Subclinical and Clinical Rejection of Renal Allografts Michael Mengel,[1] Daniel Kayser,[2] Wilfried Gwinner,[2] Jeff Reeve,[1] Anke Schwarz,[2] Hermann Haller,[2] Philip Halloran.[1] *[1]Alberta Transplant Apllied Genomics Centre, University of Alberta, Edmonton, AB, Canada; [2]Department of Nephrology, Medizinische Hochschule Hannover, Hannover, Germany.*

Protocol biopsies (PB) from clinically stable renal allografts are conducted based on the hypothesis that subclinical rejection is the harbinger of more severe, clinical overt rejection and that treatment of subclinical rejection might prevent irreversible allograft damage.

We analyzed by microarrays (Affymetrix HG_U133_Plus_2.0) 36 PB from clinically stable renal allografts taken 6-weeks post transplantation. Expression profiles from PB were compared to 7 biopsies for cause (BFC) taken due to clinical overt T cell mediated rejection (TCMR) episodes.

PB with subclinical TCMR (n=5) had increased expression of cytotoxic T cell associated and gamma-interferon inducible transcripts compared to PB without histological signs of TCMR (n=27), whereas expression values were significantly lower compared to BFC with clinical TCMR (n=7; p<0.01). No differences between subclinical borderline (n=4) and subclinical TCMR were found. In contrast to clinical TCMR subclinical borderline/TCMR showed no decreased expression of kidney parenchymal associated transcripts, i.e. soluble carrier proteins expressed by tubular epithelial cells. Principal component analysis revealed a continuous spectrum of gene expression from normal PB via PB with subclinical borderline/TCMR to BFC with TCMR (=contr_TCMR), whereas the decreased expressed