comparison to UW. This effect seemed to be related to a reduction in ischemia-reperfusion injuries rather than to a decrease in rejection.

P/5 HISTOLOGICAL INJURY DETECTED IN BIOPSIES OF EXTRAHEPATIC BILE DUCT OF DONOR LIVERS REPRESENTS INJURY IN THE REST OF THE BILIARY TREE, INCLUDING THE INTRAHEPATIC BILE DUCTS

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Histological examinations of distal end of extrahepatic bile duct of donor livers at time of transplantation have revealed signs of severe injury, characterized by a loss of biliary epithelium, mural stroma necrosis and injury of the peribiliary vasculature. It is, however, unknown whether injury of extrahepatic bile duct is representative for the injury in the rest of the biliary tree, including the intrahepatic bile ducts. Aim of this study was to examine whether the degree of histological injury of the distal extrahepatic bile duct is representative for the rest of the biliary tree. Ten donor livers that were not used for transplantation were included after obtaining informed consent from the relatives. After a median of 6 h of cold ischemia, biopsies were taken from extrahepatic and two different levels of intrahepatic bile ducts: sectoral ducts and segmental ducts. Histological injury was assessed using a systematic histological grading system. Biliary epithelial loss of >50% of the biliary lumen was observed in all the levels of the biliary tree. Minimal injury of peribiliary vascular plexus (<50% vascular changes) was observed in 91.9% of all the biopsies, and there were no significant differences between extrahepatic and intrahepatic bile ducts. There were no signs of microthrombi in the peribiliary vasculature at any level. Mural stroma necrosis was not different in extrahepatic and different levels of intrahepatic bile duct. Minimal intramural bleeding (<50% of the bile duct wall) was found in only 5% of all the biopsies. The degree of injury of periluminal and deep peribiliary glands was similar at all levels of the biliary tree. Injury of periluminal peribiliary glands, however, was more severe than injury of deep peribiliary glands (>50% loss observed in 43% and 6.25%, resp.; p = 0.002). Histological examinations of bile ducts of donor livers after cold preservation reveal extensive biliary injury. The degree of injury detected in extrahepatic bile duct of donor livers is representative for the rest of the biliary tree. Biopsies of extrahepatic bile duct of donor livers are a valuable tool for research focusing on bile duct injury in liver transplantation.

P/6 CARDIOPROTECTIVE EFFECT OF FONDAPARINUX IN A RAT MODEL OF MYOCARDIAL ISCHEMIA-REPERFUSION

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Introduction: Fondaparinux (FDX) was shown to be cardioprotective in a rat model of myocardial ischemia-reperfusion. It is able to reduce infarct size after 2 h of reperfusion, involving the activation of the survival pathway SAFE. Our aim was to study if this cardioprotection could be explained by anti-inflammatory mechanisms and a protective effect on the endothelium.
Methods: Wistar male rats were submitted to 40 min of myocardial ischemia followed by 30 min or 2 h of reperfusion. Rats were randomized in 4 groups: control 30 min (n = 7), FDX 30 min (n = 7), control 2 h (n = 7), FDX 2 h (n = 7). FDX groups received a 10 mg/kg intraperitoneal injection of FDX, 10 min before the beginning of reperfusion. Hearts were collected at the end of reperfusion. We studied the expression of mRNA of endothelial markers (P-Selectin, thrombomodulin, EPCR, tissue factor) and pro-inflammatory markers (IL-1β, IL-6 and ICAM-1). We also studied the proteic expression of ICAM-1, tissue factor and pro-inflammatory signalling proteins (NFκB, IκB and JNK). Leucocyte infiltration was assessed by histochemistry (hematoxylin-eosin stain).
Results: After 30 min of reperfusion, there is a significant increase of the expression of endothelial markers in the FDX group. This difference is not evidenced any more after 2 h of reperfusion, except for the expression of P-Selectin. Regarding the pro-inflammatory markers, there is a significant increase of their expression after 30 min of reperfusion in the FDX group. The same difference was observed after 2 h of reperfusion except for the expression of IL-1β. After 2 h of reperfusion, there is no effect of FDX on the expression of the pro-inflammatory signalling proteins, tissue factor and on leucocyte infiltration in the myocardium.
Conclusion: At early stage of reperfusion, FDX induced cardioprotection was not mediated by an anti-inflammatory effect. Our work suggests that FDX might have a protective effect on the endothelium at 30 min of reperfusion.

P/7 CINACALCET TREATMENT AT THE TIME OF TRANSPLANTATION IS ASSOCIATED WITH A SIGNIFICANT RISK OF DELAYED GRAFT FUNCTION IN KIDNEY TRANSPLANT RECIPIENTS

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The calcium-sensing receptor (CaSR) has been implicated in the ischemia/reperfusion (I/R) cascade in heart, liver and brain. Renal I/R occurs at the time of transplantation (Tx), with a deleterious impact on early graft function. Here, we retrospectively investigated if the use of cinacalcet, a CaSR agonist, in kidney transplant recipients (KTR) influences early graft recovery. All KTR from 2007 to 2012 in our Academic Hospital were prospectively included in a database. Patients actively treated with cinacalcet on the day of Tx were retrospectively identified from this database and matched with controls on (i) type of donor (living [LD], deceased after brain or circulatory death [DCD]); (ii) cold ischemic time (CIT) ± 1 h; (iii) residual diuresis (± 500 ml); and (iv) donor age (± 5 years). Delayed graft function (DGF) was defined as dialysis requirement after Tx. Baseline characteristics were compared between groups with student's *t*-test or Chi-2 as appropriate. The endpoint was the percentage of DGF in both groups. Among 337 KTR, 36 (10.7%) were treated with cinacalcet at Tx. Control group included 61 patients. Characteristics of patients and donors are summarized in the table. DGF occurred in 42 and 23% of cinacalcet-treated and control groups, respectively (p = 0.05). These retrospective observations suggest that CaSR activation at the time of Tx impairs early graft recovery.

	Cinacalcet (n = 36)	Controls (n = 61)	p
Recipient			
Age at Tx (years)	50.2 ± 10.3	49 ± 13.5	0.92
Sex ratio (% female)	47	41	0.55
Dialysis vintage (years)	3.7 ± 2.1	3.3 ± 3.8	0.57
Resting diuresis (ml)	430 ± 655	444 ± 541	0.91
Multi-organ Tx (%)	5.6	1.7	0.28
Donor			
Age (years)	46.8 ± 11.4	47 ± 11.4	0.93
Sex ratio (% female)	42	46	0.67
LD (%)	2.8	1.6	0.70
DCD (%)	30.6	21.3	0.31
Transplantation			
CIT (min)	779 ± 297	825 ± 255	0.43
HLA mismatches			
A	0.8 ± 0.5	0.9 ± 0.5	0.75
B	1.2 ± 0.7	1 ± 0.5	0.08
DR	0.8 ± 0.4	0.8 ± 0.3	0.99

P/8 INCREASED INFLAMMATION AND FIBROSIS CAUSED BY HYPEROXALURIA IN AN EXPERIMENTAL MODEL OF RENAL ISCHEMIA AND REPERFUSION

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Introduction: Acute kidney injury (AKI) is defined as a rapid loss of renal function due to damage to the organ, resulting in the retention of products of metabolism and uremic toxins that are normally excreted by the kidney. AKI caused by ischemia and reperfusion (I/R) induces renal dysfunction associated with specific markers of inflammation such as TNF-α, interleukins and interferons. On the other hand, the injury I/R may contribute to crystal deposition of calcium oxalate (CaOx) renal tubules, causing additional damage in tubular epithelial cells, inducing necrosis and leading to progressive tubular atrophy and interstitial fibrosis.
Objective: The objective was to assess whether the deposition of calcium oxalate crystals increase renal damage in rats with acute kidney injury and to analyze how animals exposed to ischemia and reperfusion evolve when subjected to an overload of CaOx.