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AKI - CLINICAL

SAP101 IS THE CONTRAST-ENHANCED MAGNETIC RESONANCE IMAGING OR MAGNETIC FIELD SAFE FOR THE PATIENTS WITH HIGH-RISK FOR ACUTE KIDNEY INJURY?

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Introduction and Aims: Gadolinium chelate (GC)s using in magnetic resonance imaging (MRI) have been traditionally considered as non-nephrotoxic contrast materials. But, in some recent articles it has been suggested that GCs may have a nephrotoxic potential. Nevertheless, most of these reports are retrospective, and evaluated contrast agents and their doses were not homogenous. To investigate the effect of gadopentetate dimeglumine (GD) and magnetic field on renal function in patients with high-risk for acute kidney injury (AKI).
Methods: We designed a prospective case control study, and age and sex-matched two groups of patients were included the study. Both of groups were consisted of the patients with high-risk for AKI (diabetes mellitus, hypotension, chronic renal failure, using nephrotoxic material, i.e.) (n=40, for each group). While contrast (gadopentetate dimeglumin)-enhanced non-vascular MRI was performed to group 1 patients, MRI without conrast agent was performed in goup 2 patients. Fixed dose of GD (0.2 mmol/kg) were administered to group 1 patients. All patients were followed up 72 hours. Before and at the 6, 24 and 72 hours after the MRI; biochemical markers, urinalysis, microalbumin/creatinine ratio in spot urine, serum creatinine, and glomerular filtration rate were measured.
Results: Baseline serum creatinine, microalbumin/creatinine ratio, and GFR was not different between group 1 and group 2 ($p>0.05$). We did not observe adverse effect related to procedures. There were no significant changes in renal functional tests (? serum creatinine, ?microalbumin/creatinin ratio, and ?GFR) in both groups after 6, 24 or 72 hours of the procedures ($p>0,05$).
Conclusions: Non-vascular contrast-enhanced (GD, 0.2 mmol/kg) MRI is a safe procedure for patients with high-risk for AKI. Key Words: MRI, Gadopentetate dimeglumine, contrast nephropathy, acute kidney injury.

SAP102 NEURAL NETWORKS FOR THE DIAGNOSIS OF ACUTE KIDNEY INJURY

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Introduction and Aims: Treatment of acute kidney injury (AKI) is hampered by the lack of an accurate tool for early diagnosis. Prediction of AKI in septic patients is cumbersome because of the many influencing factors and the heterogeneity of the underlying disease process. Neural networks (NN) have a great capacity to recognise in a robust way patterns in apparently unstructured data, which theoretically would make them optimal for medical decision making, especially when the exact relation between input (clinical and biochemical data) and output (disease state) is unknown. We therefore hypothesized that NN would be suited for early prediction of AKI in septic patients.
Methods: in the first 100 consecutive patients admitted to a tertiary care ICU and with the diagnosis of sepsis, we collected biochemical data (urinary NGAL, KIM-1, IL18 and Screa) on arrival and clinical information (diuresis, fluid balance, CVP, volume status) during the first 6 hours. AKI was defined as RIFLE class I or F on any of the first five days after admission. Multilayer perceptron NN's (based on SPSS 19) were constructed with sigmoid decision functions for the hidden and output layer, an initial sigma and lambda of 0.00005. For each NN, 70% of data were used for training and 30% for testing. NN's were once fed with only clinical data (NN1), once with only biochemical data (NN2) and once with the combination (NN3). For each condition, 10 NN's were trained during max 1000 epochs each, for the early prediction of occurrence of AKI. Results are provided on the testing cases only.
Results: 54/100 patients developed AKI. Sensitivity for prediction of AKI in the next 5 days ranged between 61.5-80.0% (NN1), 58.3-80.0% (NN2) and 56.3-95% (NN3), and specificity between 64-81% (NN1), 71-76% (NN2) and 55-72% (NN3). For the clinical model NN1, the biochemical model NN2 and the mixed model NN3, positive predictive value (PPV) varied between 46 and 80%, 58 and 88%, and 54 and 86% respectively. The negative predictive value (NPV) varied between 50 and 80%, 50 and 64% , and 50 and 77% resp.
Conclusions: Despite their ability to adequately categorise data with unknown relation, neural networks perform at best moderately in predicting AKI in the 5 days following ICU admission. Most likely, accurate prediction of AKI will remain complicated, as underlying pathophysiology and clinical conditions differ substantially between each individual case, making generalisations nearly impossible. NNs performed equally well when trained with only clinical or only biochemical data. Combination did not add to the prognostic power. As NNs train in a process similar to how experience works in humans, it can be concluded that clinical data are probably as good in predicting AKI as biomarkers in experienced physicians.

SAP103 ASSOCIATION OF ACE D ALLELE WITH ACUTE KIDNEY INJURY IN NON-CHINESE PATIENTS AFTER CARDIAC SURGERY IN A MULTI-ETHNIC SOUTH ASIAN POPULATION

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Introduction and Aims: Postoperative acute kidney injury (AKI) after cardiac surgery is a frequent, serious, multifactorial complication with interpatient variability predicted poorly by preoperative clinical and procedural markers1. In our preliminary study, we noted that 56% of patients presenting for cardiac surgery developed AKI and apart from common known risk factors, ethnicity was independently associated with the risk of AKI, with Indians and Malays having a higher risk of developing AKI after cardiac surgery. The ACE (Angiotensin converting enzyme) D allele has been implicated in kidney injury in African Americans and we postulate that the D allele is associated with the increased incidence of AKI in the non Chinese after cardiac surgery in a multi-ethnic South Asian population.
Methods: 991 consenting patients who underwent cardiac surgery were studied. Clinical covariates were recorded. The primary outcome was AKI, defined as a 25% or greater increase in preoperative to maximum postoperative serum creatinine level within 3 days after surgery. DNA was isolated from preoperative blood and PCR was used to detect the deletion (D) allele and insertion (I) allele of the ACE gene.
Results: 49.5% patients have a creatinine rise of 25% post cardiac surgery. Out of 491 patients who develop AKI, 60.9% carry the D allele. A race effect was seen with Indians and Malays having a higher risk of developing AKI compared to Chinese ($p=0.002$). In addition, non- Chinese with the D allele have a marginally higher risk compared to Chinese of developing AKI (OR 1.037, CI 0.949-1.134)
Conclusions: Indians and Malays who have the D allele have a higher risk of developing AKI compared to Chinese. The ACE D allele is linked to increased renal vasoconstriction and this susceptibility in the non Chinese may be unmasked during cardiac surgery which is associated with problems of atheroembolism and ischaemia-reperfusion injury2. This is the first local report linking the D allele in the non Chinese with development of AKI after cardiac surgery.

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SAP104 PREOPERATIVE SERUM URIC ACID IS THE MOST PREDICTIVE MARKER FOR THE INCIDENCE OF ACUTE KIDNEY INJURY FOLLOWING CARDIAC SURGERY

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Introduction and Aims: Acute kidney injury (AKI) following cardiac surgery is a frequent complication and several risk factors increasing its incidence have already been characterized. This study evaluates the influence of preoperative increased serum uric acid (SUA) levels in comparison with other known risk factors on the incidence of AKI following cardiac surgery.
Methods: 247 patients who underwent elective coronary artery bypass grafting, valve replacement/ repair or combined bypass and valve surgery between October 2010 and February 2011 were prospectively analyzed. Primary endpoint was the incidence of AKI as defined by the AKIN criteria comparing patients with preoperative serum

uric acid (SUA) levels below versus above the median. Multivariate logistic regression analysis was used to identify independent predictors of postoperative AKI.
Results: 30 (12.1%) of the 247 patients developed postoperative AKI, 24 of 30 (80%) had preoperative SUA- levels above the median (=373 μmol/l) (OR: 4.680, CI 95% 1.840; 11.904, p=0.001). In the multivariate analysis SUA levels above the median (OR: 5.497, CI 95% 1.772; 17.054, p=0.003), cardiopulmonary bypass (CPB) time >90 minutes (OR: 4.595, CI 95% 1.587; 13.305, p=0.005), body mass index (BMI) >30 kg/m^2 (OR: 3.208, CI 95% 1.202; 8.562; p=0.02), and preoperative elevated serum-creatinine levels (OR: 1.015, CI 95% 1.001; 1.029, p=0.04) were independently associated with postoperative AKI.
Conclusions: Serum uric acid is an independent risk marker for AKI after cardiac surgery. From all evaluated factors it showed the highest odds ratio.

SAP105 DEVELOPMENT AND VALIDATION OF A SEVERE A ACUTE KIDNEY INJURY RISK INDEX FOR CHINESE ELDERLY PATIENTS WITH CARDIAC SURGERY

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Introduction and Aims: Severe acute kidney injury (AKI) is an important health problem and associated with substantial morbidity and mortality in cardiac surgery patients. It is well known that elderly patients are at risk for severe AKI and this segment of population is rapidly growing. But, there is not a specific predictive risk index for elderly patient who underwent cardiac surgery. Thus, the objective of present study was to develop and validate a risk index to predict severe AKI in Chinese elderly patients with cardiac surgery.
Methods: A consecutive sample of 848 elderly patients (age=60 years old) who underwent cardiac surgery with cardiopulmonary bypass in the Guangdong general hospital between January 1, 2005 and July 31, 2010 was retrospectively evaluated. The clinical outcome was severe AKI according to the serum creatinine criteria of the AKIN clssification. Patients were excluded if they had an end stage renal disease, or experienced renal replacement therapy. Those who had missing data were also excluded. In randomly selected 682 patients of the total cohorts, multivariate logistic regression analysis was used to develop a new prediction score based on clinical characteristics and perioperative variables of patients. The new score was validated on the remaining patients.
Results: The incidence of severe AKI in the derivation cohort which consisted of 682 patients was 10.1% (n=69), while in the test cohort which consisted of 166 patients was 6.6% (n=11). Six variables were included in the predictive index, and each was assigned a number of points proportional to its standard regression coefficient: cerebrovascular disease (2 points), chronic heart failure New York Heart Association above stage 2 (2 points), cardiopulmonary bypass time above 113 minutes (2 points), emergency surgery (1 points), use of intra-aortic ballon pump during the first 24 hours after surgery (1 points), duration of ventilator-assisted respiration during postoperative above 24 hours (3 points). The area under the receiver operating characteristic curve, judging the discrimination of the score, was 0.845 (95%CI 0.798 to 0.893) in the derivation, which in the validation set was 0.865 (95%CI 0.794 to 0.936). The calibration of the score assessed using the Hosmer-Lemeshow statistic in the derivation and validation were 0.608, 0.580, respectively.
Conclusions: A risk index based on Chinese information was valid and accurate in predicting severe AKI after cardiac surgery in elderly patients. The predictive index may improve clinical decision making, prognosis of cardiac surgery patients and research design.

SAP106 LONG TERM RENAL OUTCOMES IN PATIENTS WITH ACUTE KIDNEY INJURY REQUIRING RENAL REPLACEMENT THERAPY AFTER CORONARY ARTERY BYPASS GRAFT SURGERY

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Introduction and Aims: Acute kidney injury (AKI) is a common complication post-cardiac surgery and AKI severity predicts for in-hospital and long-term mortality. However, there is little data on renal outcomes in patients with post-cardiac surgery AKI. We report the long-term renal outcomes in patients requiring Acute Renal Replacement Therapy (ARRT) after coronary artery bypass graft (CABG) surgery.
Methods: This was a single-center retrospective study which included all adult patients with CABG and post-operative AKI requiring ARRT from January 2009 to December 2010. Patients who were dialysis-dependent prior to surgery were excluded. Patients' demographic, co-morbidity and peri-operative data were retrieved from electronic medical records. Primary end-point was defined as dialysis dependence or doubling of serum creatinine at 12 months.
Results: Fifty-five patients (mean age 66 years, M:F 39:16) required ARRT post-CABG. In-hospital mortality was 34.5% (n = 19). Thirty-three patients (60.0%) had renal recovery at discharge with mean creatinine 198 ± 20 μmol/L, mean eGFR 41.5 ± 5.3 ml/min/1.73 m2. Mean follow- up was 19 ± 2 months. Comparing survivors who did not reach the primary end-point (n=30) versus those did (n=6): pre-operative serum creatinine = 250 μmol/L (OR 8.33, 95% confidence interval 1.12-61.50) was associated with poorer long term renal outcome. There was no difference in age (mean 65±2 vs. 68±4, p=1.00); hypertension (p=1.00); diabetes mellitus (p=0.61); peripheral vascular disease (p=0.33); systolic ejection fraction < 35% (p=0.06); concurrent valve repair (p=0.55); cardiopulmonary bypass duration (mean 107±8 min vs. 83±30 min, p=0.72); re-exploration (p=0.31); interval from surgery to ARRT (mean 4±1 vs. 2±1 days, p=0.25); oliguria at time of ARRT initiation (p=0.08) or ARRT modality (p=1.00).
Conclusions: Pre-operative renal function is a predictor for long term renal outcomes in post-cardiac surgery AKI. Larger prospective studies are required to further our knowledge of these patients.

SAP107 EARLY POSTOPERATIVE ANGIOTENSIN COVERTING ENZYME INHIBITORS/ANGIOTENSIN RECEPTOR INHIBITORS OR DIURETICS USE IS ASSOCIATED WITH A LOWER INCIDENCE OF ACUTE KIDNEY INJURY AFTER CARDIAC SURGERY IN ELDERLY PATIENTS

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Introduction and Aims: Acute kidney injury (AKI) is a common complication after cardiac surgery and associated with increases utilization of health care resources, extends intensive care unit length of stay, and independently predicts death. Angiotensin converting enzyme inhibitors/angiote- nsin receptor inhibitors (ACEI/ ARB) and diuretics are used frequently in patients with cardiac surgery. But, littler is known about the relationship between early postoperative use of ACEI/ARB or diuretics and AKI. Thus, we embark on this study with a view to explore the relationship between early postoperative use of ACEI/ARB or diuretics and acute kidney injury (AKI) after cardiac surgery with cardiopulmonary bypass in elderly patients.
Methods: Data from 457 consecutive elderly patients (age=60 years old) who underwent cardiac surgery with cardiopulmonary bypass in the Guangdong general hospital between January 1, 2007 and December 31, 2009 were analyzed in this retrospective research. The primary endpoint was AKI according to the serum creatinine criteria of the RIFLE. The baseline serum creatinine was defined as the latest serum creatinine before cardiac surgery. Multivariate analysis by logistic regression was used to obtain the independent risk factors for AKI.
Results: Among 457 elderly patients, 340(74.4%) patients used diuretics during early postoperative (the first 24 hours after surgery), 55(12.0%) patients taken ACEI/ARB during early postoperative, 313(68.5%) patients were complicated by postoperative AKI. The incidences of AKI between patients taking ACEI/ARB and patients who not were 24(43.6%) vs 289(71.9%), χ^2=17.90, P<0.001, respectively, which between patients taking diuretics and patients who not were 207(60.9%) vs 106(90.6%), χ^2=35.62, P<0.001, respectively. No significant differences were found between patients who taken ACEI/ARB and those who not in age, sex, hypertension, diabetes, glomerular filtration rate (modified MDRD equation) <60ml/min, preoperative medication use, hypertension, mean blood pressure < 60 mmHg after surgery. Patients taking diuretics postoperatively were more likely to have hypertension or glomerular filtration rate <60ml/min. After adjustment other potential factors of postoperative AKI, early postoperative use of ACEI/ARB (OR 0.329, 95%CI 0.156-0.691), and use of diuretics (OR 0.149, 95%CI 0.068-0.326) were independent factors of AKI after cardiac surgery with cardiopulmonary bypass.
Conclusions: Early postoperative ACEI/ARB or diuretics use is associated with a lower incidence of AKI after cardiac surgery in elderly patients.

SAP108 ASSOCIATION BETWEEN THE LOWEST HEMATOCRIT DURING CARDIOPULMONARY BYPASS AND ACUTE KIDNEY INJURY

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Introduction and Aims: Postoperative acute kidney injury (AKI) after cardiopulmonary bypass (CPB) is common and increases patients' morbidity and mortality.1,2 Hemodilution is induced during CPB to reduce blood viscosity and improve regional blood flow. However, recent studies suggest that hemodilution is associated with an increased AKI risk and other organ dysfunction post-CPB.2,3 We therefore tested the hypothesis that hemodilution is independently associated with AKI in our multi-ethnic population
Methods: Data from 1658 patients who underwent CPB between December 2008 and December 2010 at two main national heart centers was extracted. Patients with preoperative renal failure were excluded. Preoperative and daily postoperative creatinine were measured until hospital discharge, following institutional protocol.

Linear regression analysis was performed to determine the independent association of the lowest hematocrit with postoperative creatinine rise. AKI was defined as =25% increase in preoperative to maximum postoperative serum creatinine level within 3 days post-surgery
Results: We observed 36.24% incidence of AKI. Univariate analysis identified age, hypertension, diabetes, preoperative effort tolerance of New York Heart Association (NYHA) Class III or IV, lowest hematocrit in CPB, use of intraaortic balloon pump (IABP) perioperatively and EuroSCORE (logistic) as statistically significant risk factors of AKI development after CPB. After correcting for propensity score and covariates, lowest hematocrit during CPB was independently associated with AKI. Other factors with increased odds ratios include age, hypertension, IABP use perioperatively.
Conclusions: Effects of hemodilution during CPB on various organ systems are well described, with post-operative AKI largely emphasized.2,3,4 Our CPB lowest hematocrit range also corroborates with that of previous studies.4,5 As hematocrit is directly associated with tissue oxygen delivery, the explanation of our proven hypothesis is that excessive hemodilution (indicated with lowest hematocrit range) will contribute to development of AKI by disturbing renal microcirculation with lowered blood viscosity and disrupted viscous layer in capillaries, thereby causing hypoxia, particularly in the renal medulla.3 This may be one of the reasons for the high incidence of AKI in our group of patients. This association is important to note as mortality and morbidity rates are elevated with AKI post-CPB.2
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SAP109 ANAEMIA PREVIOUS CARDIAC SURGERY PREDICTS ACUTE KIDNEY INJURY

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Introduction and Aims: Acute kidney injury (AKI) is a serious complication after cardiac surgery and is associated with higher mortality. Anaemia is a known risk factor of AKI due to contrast nephrotoxicity. However, it has hardly been evaluated in clinical studies of AKI after cardiac and it is not included in Cleveland and SRI (Simplified Renal Index) scores, which are clinical scores to predict AKI after cardiac surgery. The objective of the present study is to evaluate the incidence of AKI defined by AKIN classification and to identify risk factors for AKI.
Methods: Cohort study of prospectively collected data from 601 patients who underwent cardiac surgery in a University Hospital in Madrid during 2 years. AKI was defined according to AKIN classification. The association between preoperative and intraoperative variables and the development of AKI was assessed by multivariate logistic regression analysis.
Results: AKI incidence is 16.6% (n=100). In the univariate analysis patients who developed AKI had lower baseline haemoglobin levels (p<0.001, OR 0.69, 95%CI 0.61-0.78) and a significant lower postoperative haemoglobin level (p=0.016, OR 0.87, 95%CI 0.77-0.97).
Multivariate analysis of risk factors associated with AKI.
Independent risk factors for AKI are: old age (OR 1.05 for each year), male sex (OR 2.7), decreased baseline haemoglobin levels (OR 0,65), valve surgery (OR 4,8) and longer bypass time (OR 1,003 for each minute).
Conclusions: Anaemia before surgery predicts the development of AKI. It is a modifiable risk factor and should be optimised. It is not included in Cleveland and SRI scores, that predict AKI after cardiac surgery. In the clinical practice, to know anaemia as a risk factor for AKI allows the identification of patients at high risk, treat the anaemia and therefore decrease the incidence of AKI and mortality after cardiac surgery.

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Variables	beta	Standard error	Wald	df	*p*	OR (95% CI)
Age	0.048	0.150	10.672	1	0.001	1.05 (1.02-1.08)
Sex (male)	1.011	0.318	10.113	1	0.001	2.7 (1.5 –5.1)
Baseline haemoglobin	-0.432	0.085	25.980	1	<0.001	0.65 (0.55 – 0.77)
Type of surgery (valve)	1.563	0.575	7.398	1	0.007	4.8 (1.6 – 14)
Bypass time	0.003	0.002	3.391	1	0.066	1.003 (1.000 – 1.007)
Constant	-0.863	1.792	0.232	1	0.630	0.422

SAP110 RENIN-ANGIOTENSIN-ALDOSTERONE SYSTEM BLOCKADE EXACERBATES CONTRAST-INDUCED NEPHROPATHY IN PATIENTS UNDERGOING CORONARY ANGIOGRAPHY: PROPENSITY-MATCHED COMPARISON

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Introduction and Aims: The role of the angiotensin converting enzyme ACE inhibitor (ACEI) and angiotensin receptor blocker (ARB) in the pathophysiology of contrast induced nephropathy (CIN) remains controversial, and the available literature is contradictory.
Methods: This study was a retrospective propensity score (PS)-matched study to analyze the effect of ACEI/ARB therapy on the development of CIN. Using PS matching, 1,305 ACEI/ARB recipients and non-recipients were paired for analysis from 11,447 patients with receiving coronary angiography (CAG) or percutaneous coronary intervention (PCI). The predictors in this study were CIN and risk factors.
Results: ACEI/ARB was prescribed for 64.0% of patients with receiving CAG. ACEI/ARB users showed an increased incidence of CIN after PS matching (9.7 vs. 7.4%, P = 0.035). In multivariate analysis, the use of ACEI/ARB remained an independent and significant predictor of CIN in unmatched cohort [odds ratio (OR) = 1.305, 95% confidence interval (CI) = 1.042 – 1.633, P = 0.020]. In the matched cohort, the use of ACEI/ARB was also associated with a higher adjusted OR of CIN (OR = 1.356, 95% CI = 1.012 – 1.816, P = 0.041).
Conclusions: The use of ACEI/ARB was associated with an increased risk of CIN. Further randomized clinical trials are warranted to confirm the effect of ACEI/ARB therapy on the development of CIN.

SAP111 URINARY NGAL AT ADMISSION DOES NOT PROVIDE ADDITIONAL INFORMATION THAN SERUM CREATININE, URINE OUTPUT AND FIRST 24H FLUID BALANCE, FOR PREDICTION OF AKI IN SEPTIC PATIENTS AT ICU

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Introduction and Aims: The search for new urinary and serum biomarkers for early prediction of acute kidney injury (AKI) has been a top priority. One of the most promising biomarkers is NGAL (neutrophil- gelatinase-associated lipocalin). However, most studies so far did not assess NGAL as additional factor on top of easily available clinical information. It should especially be evaluated against urinary volume, the only online biomarker for AKI we have for now, and fluid balance.
Methods: 107 consecutive patients presenting with sepsis at a tertiary care ICU centre were studied. Patients with RIFLE I or F on any of the 5 following days were considered to have AKI. A logistic regression model to predict AKI based on admission data was built. Age, gender and sepsis severity were forced into the model, and then a step-forward and step-backward analysis was performed with the following variables included in the model: urinary volume during the first 6 hours, fluid balance during the first 24 hours, serum creatinine at ICU admission and urinary NGAL at admission.
Results: 107 patients were included of whom 2 did not have NGAL measurements because of anuria. Of the remaining 105 patients, 13, 26 and 66 were diagnosed with sepsis, severe sepsis or septic shock respectively. Median urinary NGAL levels in patients with sepsis, severe sepsis or septic shock were 184 ng/ml (IQR 307), 175 ng/ml (IQR 392) and 560 ng/ml (IQR 1498) (p 0.01). Fifty-eight patients (54.2%) developed AKI over the next 5 days. Both in the step-forward and the step-backward model, serum creatinine at ICU admission, fluid balance during the first 24 hours and urinary volume during the first 6 hours after admission were retained as independent predictors for developing AKI in the next 5 days (p 0.001, p 0.03 and p 0.02; R2 40%), whereas urinary NGAL was not a significant predictor.
Area under the curve (AUC) of RoC curves were 0.82 for a score based on age, gender, sepsis severity, serum creatinine at ICU admission, urinary volume, and fluid balance. Adding urinary NGAL as information did not alter the AUC (0.82).
Conclusions: Urinary NGAL does not provide additional information for prediction of AKI on top of the information provided by serum creatinine at ICU admission, fluid balance during the first 24 hours and urine output during the first 6 hours after admission.

SAP112 RENAL RECOVERY OF ACUTE KIDNEY INJURY (AKI) AFTER CARDIAC SURGERY

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Introduction and Aims: Acute kidney injury (AKI) is a common syndrome complicating several clinical settings. AKI is generally defined as a sudden decrease of renal function, measured by changes in serum creatinine (sCr) and/or urine output. AKI may develop in up to 30 % of patients (pts) who undergo cardiac surgery. Data are still limited about renal recovery in this setting. In our study, we analyzed the incidence of AKI and subsequent recovery in pts undergoing coronary artery bypass, looking for factors related to both.
Methods: We observed 1016 pts, 843 male and 172 female who underwent CABG from May 2004 to August 2010. AKI was classified according to the AKIN (Acute Kidney Injury Network) criteria. Complete renal recovery at discharge was defined as return to pre-morbid renal function; partial renal recovery as an improving AKIN classification. For the analysis of recovery, we excluded patients who underwent Continuous Renal Replacement Therapy (CRRT) after surgery. Statistical analysis was performed by the SPSS, version 17.
Results: AKI developed in 372 pts (36,6%): AKI1 in 300 pts (80,6%), AKI2 in 27 pts (7.2%) and AKI3 in 45 pts (12 %). 37 (9,9 % of AKI population) pts required postoperative CRRT. Significant risk factors associated with occurrence of AKI were: age, pre-existent chronic kidney disease, EUROSCORE risk index, aortic clamping (Ao Clamp) and mechanical ventilation (MV) . Mortality and ICU stay were significantly higher in AKI population. Complete renal recovery at discharge was noted in 188 pts (58,4%), partial renal recovery in 110 (34.2%), no recovery in 24 pts (7,5%). No statistical significant associations were found between renal recovery and none of the variables analyzed (age, sex, comorbid condition, pre-existent renal disease, EUROSCORE index, extracorporeal circulation, Ao Clamp, MV, CVVH intraoperative)
Conclusions: Small changes in sCr have been shown to be associated with adverse short and long-term outcomes. Some Authors observed that early renal recovery, defined as withdrawn of renal replacement therapy, offers a significant survival advantage after cardiac surgery, but no studies were performed on AKI recovery in population not needing CRRT. In our study, renal recovery rate were high, mainly within the AKI1 population. Surprisingly, even after stratifying pts for AKI stages, none of the risk factors observed significantly influenced recovery of renal function. This could be related to the high percentage of AKI1 population in which alteration of sCr are mild and cannot always be considerate a reliable marker of AKI. In our study AKI2 and AKI3 were small groups; this, together with its retrospective nature, limits our observation. Further studies are necessary to confirm these preliminary results.

SAP113 URINARY NGAL (NEUTROPHIL GELATINASE-ASSOCIATED LIPOCALIN) FOR EARLY DETECTION OF ACUTE KIDNEY INJURY (AKI) IN GERIATRIC PATIENTS WITH URINARY TRACT INFECTION (UTI) TREATED BY COLISTIN

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Introduction and Aims: Colistin (polymyxin E) was developed about 60 years ago but was rarely used in clinical practice during the last 20 years because of concerns related to high rates of nephrotoxicity. However, it was recently reintroduced to clinical practice in many parts of the world for the treatment of multi-drug resistant gram-negative bacilli. In the current study, we evaluated the predictive capacity of urine NGAL for early diagnosis of acute kidney injury (AKI) in geriatric patients with UTI receiving colistin therapy.
Methods: We studied 116 patients aged 80.7 ± 12 treated with colistin who suffered from UTI. Urinary NGAL was measured at baseline and 1-2 hours after the second dose of colistin. The primary outcome was AKI. Secondary outcome was in hospital morbidity and mortality.
Results: 52 patients (44.8%) developed ATN (14% of these had underlying CKD), 8 (7%) had prerenal azotemia, 8 (7%) had stable CKD without changes in renal function during hospitalization and the remaining 48 patients (41%) had normal kidney function. The mean duration of colistin therapy was 9.1 ± 4.8 days. At baseline, urine NGAL was 405 ± 452 g/L in ATN, 285 ± 256 g/L in prerenal azotemia, 390 ± 468 g/L in CKD and 347 ± 877 g/L in normal kidney function patients (difference non- significant). We were unable to demonstrate statistically significant increments of urine NGAL following colistin administration in either ATN or non ATN patients groups. Urine NGAL was not correlated with urinary leukocyte or erythrocyte counts or baseline comorbidities such as CKD, heart failure or diabetes. For primary outcome (ATN), receiver operating characteristics curve revealed AUC 0.59 (95% CI 0.49-0.7) sensitivity 0.65, and specificity 0.62 for a cutoff value of urinary NGAL 140 g/L. Similar results were obtained for secondary outcomes.
Conclusions: Our data suggest limited predictive capacity of urinary NGAL for early diagnosis of AKI in a large clinical setting of geriatric patients hospitalized for UTI and receiving the potentially nephrotoxic colistin. This finding is likely due to the powerful influence of UTI on NGAL levels in both patients with normal kidney function and those with a wide spectrum of acute or chronic kidney diseases.

SAP114 THE COMBINATION OF URINE NGAL AND SERUM CREATININE IS A GOOD PREDICTOR FOR DIALYSIS REQUIREMENT IN ICU POPULATION

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Introduction and Aims: The prediction of renal replacement therapy (RRT) requirement by novel biomarkers (or their combination) could lead to timely interventions, among patients admitted to a general Intensive Care Unit (ICU) of a tertiary hospital
Methods: A hundred and six unselected, consecutive critically ill patients (68M/38F, mean age: 64±18 years, APACHE II 19±8, SOFA 9±3) were evaluated daily for urine Neutrophil Gelatinase Assosiated Lipocalin (uNGAL), serum cystatin C (CysC) and serum creatinine (Cr). Peak values of uNGAL, CysC and Cr within the first three days of admission were also recorded. RRT according to traditional indications was required for forty nine patients (46%). In order to find the better predictor for RRT initiation, ROC curves were constructed for each parameter and multiple variables were combined to build a logistic regression model and thus generate a combined predictor. The comparison of ROC curves revealed the better predictor of RRT requirement.
Results: Admission and peak values of uNGAL, CysC and Cr, predicted RRT requirement. Areas under the ROC curve (AUCs) were: 0.697 (95% CI:0.593-0.802 p<0.0001), 0.764 (95% CI: 0.67-0.858 p<0.0001) ?a? 0.763 (95% CI: 0.666-0.859 p<0.0001), for uNGAL, CysC and Cr on admission and: 0.716 (95% CI: 0.613-0.819 p<0.0001), 0.781 (95% CI: 0.699-0.876 p<0.0001) and 0.794 (95% CI: 0.703-0.885 p<0.0001) for peak values of uNGAL, CysC and Cr respectively. Cut-off points of uNGAL =190 ng/ml, Cr =1.17 mg/dl, Cys C=1.45 mg/l on admission and peak values of uNGAL =190 ng/ml, Cr =1.49 mg/dl, Cys C=1.85 mg/l independently predicted RRT need after 7 days. The logistic regression model that was built, included only uNGAL =190 ng/ml on admission [OR: 2,007 (95% CI: 1.096-3.674)] and peak Cr =1.49 mg/l [OR: 3.692 (95% CI: 1.980-6.886)]. A new ROC combining those two parameters revealed higher AUC: 0.843 (95% CI: 0.765-0.921 p<0.0001) compared to AUCs for each separate parameter (p<0.003).
Conclusions: The combination of uNGAL =190 ng/ml on admission and peak Cr =1.49 mg/dl is a good predictor for RRT initiation in a general ICU population.

SAP115 ACUTE KIDNEY INJURY AFTER CARDIAC SURGERY: ROLE OF CYSTATIN C AND NGAL

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Introduction and Aims: Acute renal failure is a frequent complication and an independent predictor of mortality in patients undergoing cardiac surgery. Even today one of the main limitations in the prevention and treatment of acute kidney injury (AKI) is the lack of a reliable early marker. In early past, thanks to the studies of genomics and proteomics, several promising markers of AKI have emerged, including NGAL is Cystatin C, both with the potential for high sensitivity and specificity. The purpose of this study was to examine the association between elevation of Cystatin C plasma levels and urinary NGAL levels and AKI occurrence (and its eventual precocity compared to serum creatinine) in patients undergoing cardiac surgery.
Methods: In 32 consecutive patients without chronic renal failure and candidates for heart surgery on cardiopulmonary bypass, we measured serum creatinine, plasma cystatin C and urinary Ngal in the following ways: at admission (t0); 6 hours (t1), 12 hours (t2), 24 hours (t3) and 48 hours (T4) after surgery.
Results: Of the 32 patients evaluated, 12 had a rise in plasma levels of cystatin C and/or urinary Ngal and among these 7 also showed a rise in serum creatinine postoperatively. Notwithstanding the earliness than the serum creatinine of the two markers analyzed, Cystatin C seems to be more specific (higher in 6 of 7 cases when there is also a later increase in serum creatinine levels) and Ngal more sensitive (high early in the 12 patients with alleged AKI, even those who do not develop then an increase in serum creatinine).
Conclusions: Despite the limitations of the small sample analyzed, indicating that emerges is that the diagnosis of AKI, rather than from a single marker, to date the most reliable information in terms of sensitivity and specificity, results from the execution of an integrated panel of renal biomarkers.

SAP116 PLASMA NEUTROPHIL GELATINASE-ASSOCIATED LIPOCALIN (NGAL) AS AN EARLY PREDICTIVE MARKER OF CONTRAST-INDUCED NEPHROPATHY IN HOSPITALIZED LOW-RISK PATIENTS UNDERGOING COMPUTED TOMOGRAPHY (CT)

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Introduction and Aims: Contrast-induced nephropathy (CIN) is a common cause of hospital-acquired acute kidney injury (AKI). Serum creatinine (SCr) is a delayed and unreliable indicator of CIN. Neutrophil Gelatinase-Associated Lipocalin (NGAL) represents a promising, non-invasive biomarker for AKI. Its role in the early diagnosis of CIN has already been examined in adults and children undergoing coronary angiography. The present study was designed to prospectively evaluate plasma NGAL compared to SCr for early CIN detection among hospitalized low-risk patients undergoing conventional contrast-enhanced computed tomography (CT).
Methods: We prospectively enrolled consecutive hospitalized patients undergoing elective CT with intravenous (IV), low-osmolar contrast administration. Patients with pre-procedure SCr>2.0 mg/dl, overt congestive heart failure, hemodynamic instability of any cause, sepsis or urinary tract infection were excluded. All subjects were in euvolemic state prior to the procedure. Plasma NGAL was measured by using standardized ?riage® NGAL test (Biosite Incorporated, San Diego, CA) at baseline and 6 h after contrast administration. SCr, serum urea, albumin and sodium (Na) were measured and eGFR MDRD4 was calculated at the same intervals, as well as at 24 and 48 h post-procedure. CIN was defined as an increase in SCr of > 25% or > 0.5 mg/dl from baseline within 24-48 h after contrast exposure, in the absence of other obvious causes.
Results: 47 patients, male/female 27/20, median age 68 (31-88) years, 16/47 diabetics, with well- preserved baseline renal function (SCr 1.04±0.23 mg/dl, eGFR MDRD4 68.40±18.22 ml/min/1.73m2) were enrolled. Contrast volume of 120 ml (range 100-150 ml) was administered. CIN was found in 4 subjects (8.51%), but detection by SCr was only possible 24h in 1 and 48 h post-procedure in 3. In contrast, significant elevation of plasma NGAL was found at 6 h post-procedure in those with vs. those without CIN (779.25±361.49 vs. 82.30±40.64 ng/ml, p<0.001). Using a cutoff value of 150 ng/ml, sensitivity, specificity, and area under the receiver-operating characteristic (ROC) curve of 6-h plasma NGAL for CIN prediction were excellent (100%, 90.70%, and 1.00, respectively). Subjects with CIN did not differ in baseline demographics, renal function and diabetes status compared to those without CIN. No differences in any variable were noted between diabetics and non-diabetics. Plasma NGAL at 6 h (R2=0.24, p<0.001) and serum Na at baseline (R2=0.16, p=0.005) were found to be independent predictors of CIN.
Conclusions: Plasma NGAL 6 h after contrast administration measured by the rapid, point-of-care ?riage® NGAL test showed a good performance in the early prediction of CIN among hospitalized low-risk patients undergoing conventional contrast-enhanced CT. CIN prevalence in this well- controlled population underlines the importance of early detection by an adequate and simple procedure as the 6-h plasma NGAL test we evaluated.

SAP117 EVALUATION OF NOVEL BIOMARKERS IN PATIENTS WITH ACUTE KIDNEY INJURY

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Introduction and Aims: Placental growth factor (PlGF), pregnancy-associated plasma protein-A (PAPP-A), soluble receptor for advanced glycation end products (sRAGE) and calcium binding protein S100A12 (EN-RAGE) have been implicated as novel biomarkers in chronic kidney disease (CKD). However, their clinical significance in acute kidney injury (AKI) is unknown. The aim of this cross-sectional study was to determine whether selected biomarkers are elevated in AKI patients.
Methods: The studied groups were categorized as follows: 40 patients with AKI at the inception of renal replacement therapy, 42 patients starting dialysis defined as CKD 5, 31 long-term haemodialysis patients (HD) and 39 age-matched healthy controls. PlGF, sRAGE and EN- RAGE levels were assessed using enzyme linked immunosorbent assay, PAPP-A levels were determined using time-resolved amplified cryptate emission, and routine biochemical parameters were measured using standard methods. Kruskal –Wallis test with Dunn's post-tests was used to compare groups and Pearson correlation test was used for continuous variables.
Results: PAPP-A was significantly (P < 0.001) elevated in AKI (20.6 ± 16.8 mIU/L), CKD 5 (27.8 ± 39.3) and HD (20.8 ± 9.9) compared with controls (9.1 ± 2.3); PlGF was elevated (P < 0.05) in HD (11.5 ±3.3 pg/mL) versus controls (8.5 ± 2.4), but not in the other groups. sRAGE was increased (P < 0.001) in CKD 5 (3227 ± 1458 pg/mL) and HD (2746 ± 1218) versus controls (1765 ± 721), but not in AKI group; EN-RAGE was (P < 0.001) elevated in AKI (492.4 ±. 449.5 ng/mL) compared with controls (60.1 ± 61.8), but not in the other groups. In AKI group PAPP–A levels were inversely correlated with albumin (r = -0.4; P = 0.01), and prealbumin ((r = -0.45; P = 0.004); PlGF positively correlated with C reactive protein (CRP) (r = 0.47; P = 0.002), fibrinogen (r = 0.47; P = 0.002), and inversely with prealbumin (r = -0.5; P = 0.001); sRAGE negatively correlated with haemoglobin (r = -0.39, P = 0.01), and EN-RAGE correlated with CRP (r = 0.33, P = 0.03), orosomucoid (r = 0.46, P = 0.002), leukocyte count (r = 0.43, P = 0.006) and inversely with glomerular filtration rate (r = - 0.33, P = 0.043).
Conclusions: PAPP-A and EN-RAGE levels are significantly elevated, but sRAGE and PlGF levels are not increased in AKI patients. sRAGE has a reverse relation to haemoglobin. Whereas PAPP-A correlates with markers of malnutrition; PlGF and EN-RAGE are related to markers of inflammation. In addition, EN-RAGE levels correlate with renal function. Future studies may delineate whether the above studied biomarkers are also markers of disease activity and severity as well as predictors of outcome in AKI.

SAP118 NOVEL BIOMARKERS FOR PREDICTION OF RENAL REPLACEMENT THERAPY IN ACUTE KIDNEY INJURY: A SYSTEMATIC REVIEW

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Introduction and Aims: Novel biomarkers (BM) may aid in early diagnosis of acute kidney injury (AKI), prediction of AKI severity and adverse outcomes. As such, one potential clinical application is early identification of AKI patients who will need renal replacement therapy (RRT). Our aim was to evaluate the performance of novel BMs for the prediction of RRT.
Methods: We performed a systematic review of Medline/PubMed to identify novel BM studies which reported RRT as an outcome, whether separate or part of a composite. Studies which reported area under the receiver operating characteristic curve (AuROC) and/or sensitivity/ specificity for any outcome which included RRT were eligible for full review; we excluded those related to renal transplant. Data were extracted on study design and quality, population characteristics, and incidence of RRT and outcomes on standardized case report forms. Primary outcome was prediction of RRT. Whenever possible, data on RRT alone was abstracted from composite outcomes.
Results: We identified 30 studies (27 prospective cohort, 2 retrospective, 1 RCT), representing a total of 6631 patients (RRT, n= 791 patients). Studies were performed in diverse clinical settings: cardiac surgery (CS, n=4 studies), critical illness (ICU, n=9), CS&ICU (n=2), hospital AKI (n=9), other (n=6); sample sizes ranged from 19-635. Urinary and blood BMs were evaluated in 20 and 17 studies, respectively; 10 evaluated multiple BMs. The most commonly studied BMs were NGAL (n=18), cystatin C (n=11), KIM-1 (n=5) and IL-18 (n=4). AuROC for RRT or the composite of RRT/death were reported in 12 and 3 studies, respectively. The range of AuROC was <0.66-0.95 for NGAL, 0.62-0.99 for cystatin C (Table 1), 0.62-0.65 for KIM-1 and 0.73 for IL-18. Specific biomarker cut-offs for RRT decisions were poorly reported.
Conclusions: Evidence regarding novel BM performance for predicting RRT is

SAP118 Table 1.

Biomarker	Study	Sample	Setting	RRT / Total pts	AuROC (95%CI)
NGAL	Constantin 2009	Plasma	ICU	7 / 88	0.78 (0.68 - 0.86)
	Bagshaw 2010	Plasma	ICU	13 /83	0.78 (0.61 - 0.95)
	Cruz 2010	Plasma	ICU	15 / 301	0.82 (0.70 - 0.95)
	de Geus 2011	Plasma	ICU	28 / 632	0.86 (0.80 - 0.92)
	Royakkers 2011	Plasma	ICU	14 / 151	<0.66 (NR)
	Haase-Fielitz 2009	Plasma	CS	5* / 100	0.95 (0.86 - 0.99)
	Haase-Fielitz 2009	Plasma	CS	4 / 100	0.83 (0.60 - 0.98)
	Portal 2010	Plasma	Liver Txp	NR / 95	0.84 (0.74 - 0.94)
	Bagshaw 2010	Urine	ICU	13 / 83	0.7 (0.58 - 0.82)
	deGeus 2011	Urine	ICU	28 / 632	0.88 (0.84 - 0.92)
	Endre 2011	Urine	ICU	19 / 528	0.79 (0.65 - 0.94)
	Bennett 2008	Urine	CS	4 / 196	0.86 (NR)
	Koyner 2010	Urine	CS	8 / 123	0.78 (0.59 - 0.98)
Cystatin C	Nejat 2010	Plasma	ICU / CS	71** / 444	0.62 (0.54 - 0.70)
	Rosenthal 2004	Serum	CS	12 / 85	0.76 (0.69 - 0.85)
	Haase-Fielitz 2009	Plasma	CS	5* / 100	0.99 (0.98 - 0.99)
	Perianayagam 2009	Serum	Hospital AKI	84*** / 200	0.65 (0.57 - 0.73
	Endre 2011	Urine	ICU	19 / 528	0.71 (0.57 - 0.84)
	Royakkers 2011	Urine	CS	14 / 151	<0.66 (NR)
	Koyner 2010	Urine	CS	8 / 123	0.77 (0.55 - 0.98)
	Rosenthal 2004	Urine	Hospital AKI	26 / 73	0.92 (0.86 - 0.92)

AuROC reported for composite of RRT/Death. *RRT=4/5 pts. ** RRT=14 /71pts; *** RRT=64/84

highly heterogeneous. The reported AuROCs exhibit a wide range, even within a single BM. This is likely related to multiple factors including clinical setting, comorbidities, timing of specimen collection, type of specimen and assay, among others. Further studies are needed to decipher the utility of these BMs in predicting RRT, and to clarify the optimal biomarker values for informing clinical management decisions.

SAP119 EXPRESSION OF NESTIN IDENTIFIED BY PROTEOMICS: AS A URINARY BIOMARKER OF ACUTE KIDNEY INJURY

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Introduction and Aims: This work explores various markers expressed in rat urines.
Methods: After Ischemia Reperfusion Injury (45 min) by proteomic methods iTRAQ labeled, MALDI- TOF-TOF, and the identified candidate biomarkers were validated by western blotting in individual urine samples from rats subjected to bilateral ischemia and reperfusion (I/R) injury, ciaplant injection. Groups of experimental animals (n=20) were sacrificed 6h, 24h, 48h and 72h post-ischemia. Mean while we collected patients' urine samples from Cardiac Intensive care unit (CCU) patients with and without AKI (n=40) for further study
Results: We identified many proteins that were increased or decreased 6 h after IR injury/cisplant injection. Most of the candidates could not be validated by Western blotting. However, nestin increased at 6h,and peaked at 24h.(long before serum creatinine increase and tubule damage at day3). By HE, PAS, MASSON we clearly found the morphology change of ATN .By immunochemistry and confocal studies, we found nestin was only located in podocytes in normal kidney and expressed on renal tubular cells as ATN progressed. Urinary nestin also increased in ICU Patients with AKI (n=9) compared to the patients without AKI (n=31) by Elisa. In vitro, we employed the human renal proximal tubular epithelial cell line HK-2 under 1% O2 for 12,24,48h and then H2O2 under normal conditions for 8h ,which is for mimicking the ischemia reperfusion in vivo. The western blotting results said nestin was induced to go peak at 24h by hypoxia/H202, the further mechanism of nestin's expression is studying.
Conclusions: We conclude that (1) proteomic analysis of urinary can provide biomarker candidates for the diagnosis of AKI and (2) Urinary nestin might be a predictive biomarker of Structural renal injury.

SAP120 COMPARISON OF SERUM URIC ACID AND BIOMARKERS TO PREDICT ACUTE KIDNEY INJURY

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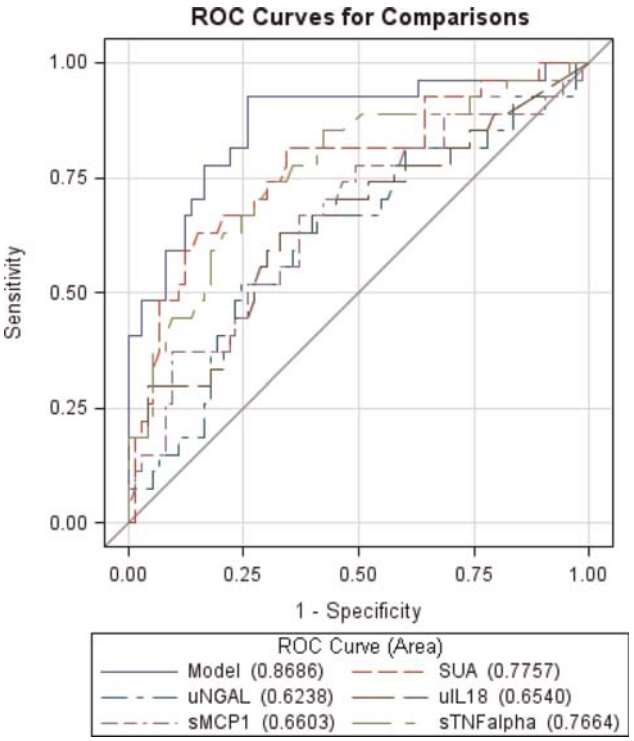
Introduction and Aims: Based on clinical observations, we hypothesized that postoperative serum uric acid (SUA) may be a good predictor of acute kidney injury (AKI) and that it may be comparable to that of biomarkers measured at 24hours from start of surgery.
Methods: Data collected in a prospective, observational study in an academic medical center were utilized for analyses. We investigated the association of SUA with AKI and compared it with novel biomarkers such as urine NGAL (uNGAL) and interleukin-18 (uIL-18) and serum monocyte chemoattractant protein-1 (MCP-1) and tumor necrosis factor-alpha (sTNF-alpha) to predict AKI. SUA and biomarkers were measured at 24hours from start of surgery, i.e. at postoperative day 1.
Results: 100 consecutive cardiac surgery patients were included for analyses. The OR of SUA as a continuous variable for the whole cohort to predict AKI was 0.49 CI95% 0.35-0.71, p<0.001). Since the proinflammatory effects of SUA are manifested at higher serum concentrations, we divided SUA into tertiles and investigated their association with AKI. The 1st, 2nd and 3rd tertiles were associated 15.1%, 11.7% and 54.5% incidence of AKI, respectively. The adjusted ORs for AKI of the 2nd and 3rd SUA tertiles compared to the referent 1st tertile were 0.64 (CI95% 0.12-3.30, p=0.597) and 8.38 (CI95% 2.13-33.05, p=0.002) respectively. Similar graded response were also noted for urine NGAL and IL-18 and serum MCP-1 and TNF- alpha. The Area Under Curve (AUC) of SUA and biomarkers were as follows: SUA 0.777 (CI95% 1.33-3.14, p= 0.001), uNGAL 0.623 (CI95% 0.49-0.75, p=0.211), uIL-18 0.654 (CI95% 0.52-0.78, p=0.010), sMCP-1 0.660 (CI95% 0.53-0.78, p=0.019) and sTNF-alpha .766 (CI95% 0.65-0.87, p<0.001). The diagnostic performance to predict AKI improved when clinical biomarkers, plasma and urine biomarkers were included in the model with AUC of 0.86 (SEM 0.07).
Conclusions: Postoperative SUA was associated with an increased incidence of AKI and graded risk for AKI. The AUC of SUA were comparable to that of novel biomarkers in the time period tested.



SAP120

SAP121 ACUTE KIDNEY INJURY BIOMARKERS FOR PATIENTS IN A CORONARY CARE UNIT: A PROSPECTIVE COHORT STUDY

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Introduction and Aims: Renal dysfunction is an established predictor of all-cause mortality in intensive care units. This study analyzed the outcomes of coronary care unit (CCU) patients and evaluated several biomarkers of acute kidney injury (AKI), including neutrophil gelatinase-associated lipocalin (NGAL), interleukin-18 (IL-18) and cystatin C (CysC).
Methods: Serum and urinary samples collected from 150 patients in the coronary care unit of a tertiary care university hospital between September 2009 and August 2010 were tested for NGAL, IL- 18 and CysC. Prospective demographic, clinical and laboratory data were evaluated as predictors of survival in this patient group.
Results: The most common cause of CCU admission was acute myocardial infarction (80%). According to Acute Kidney Injury Network criteria, 28.7% (43/150) of CCU patients had AKI of varying severity. Cumulative survival rates at 6-month follow-up following hospital discharge differed significantly ($p<0.05$) between patients with AKI versus those without AKI. For predicting AKI, serum CysC displayed an excellent areas under the receiver operating characteristic curve (AUROC) (0.895±0.031, p<0.001). The overall 180-day survival rate was 88.7% (133/150). Multiple Cox logistic regression hazard analysis revealed that urinary NGAL, serum IL-18 and sodium on CCU admission day one were independent risk factors for 6-month mortality. In terms of 6-month mortality, urinary NGAL had the best discriminatory power, the best Youden index, and the highest overall correctness of prediction.
Conclusions: Our data showed that serum CysC has the best discriminative power for predicting AKI in CCU patients. However, urinary NGAL and serum IL-18 are associated with short-term mortality in these critically ill patients.

SAP122 RETROSPECTIVE 10-YEAR ANALYSIS OF SEPSIS-ASSOCIATED ACUTE KIDNEY INJURY : INCIDENCE, IMPACT ON OUTCOME , IDENTIFICATION OF PLASMA MICROVESICLES AS POTENTIAL MEDIATORS AND BIOMARKERS OF DISEASE

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Introduction and Aims: Severe sepsis and septic shock are the predominant causes of acute kidney injury (AKI) in critically ill patients admitted to intensive care units (ICUs). Septic AKI is associated with microvascular dysfunction, multiple organ failures and high mortality rates. The concentration of circulating plasma factors including cytokines correlated with mortality. Microvesicles (MVs) are small particles released from different activated cell types: MVs are present in plasma playing a pivotal role in cell-to-communication. During sepsis, MVs can be released by activated leukocytes and platelets playing a role in tissue injury. The aims of this study were: 1) to evaluate the incidence of sepsis as cause of AKI in the period 2001-2010; 2) to identify the impact of sepsis on outcome of AKI patients 3) to define the role of plasma MVs as potential mediators of tissue injury and biomarkers of disease state.
Methods: We analyzed all patients admitted to ICUs and treated by RRT for AKI in the period 2001- 2010. For all patients RIFLE, SOFA and ATN_ISS scores were calculated. Diagnosis of sepsis/septic shock was performed according to published. Patients' outcome was assessed 28 days after ICU admission. Statistical analysis was performed using the Hemer-Lemeshow test. Plasma samples were collected at the start of RRT to analyze the presence of MVs by FACS analysis.
Results: In the period 2001-2010, 1833 patients with AKI were treated by RRT (9061 total sessions performed). Patients characteristics were: 64,7% males; age 66,4±11,5 yrs; serum creatinine at the start of RRT 3,8±1,9 mg/dl; RIFLE Failure 56.8%, Injury 28.4%, Risk 14.8%; SOFA 10.6±1.3; number of organ failures: 3,4±1,38; ATN_ISS 0.738±0.192. At day 28, mortality was 1257/1833 (68,6%). The observed mortality was significantly lower than that expected by the ATN_ISS score (72%) ($p<0.05$). Sepsis was the main cause of AKI: indeed, septic AKI patients were 415/1833 (22.6%). We distinguished 2 AKI groups: the septic group (S) and the non-septic group (NS, 1418/1833: 77.4%). In the S group, mortality rate at day 28 was 302/415 (72.9%), whereas in NS group was 804/1418 (56,7%), ($p<0.05$). The expected mortality in the S group was 73.8%, whereas in the NS group was 72%. The hours of RRT sessions were higher in the S group than in the NS group, suggesting that S group is subjected to a more intense dialytic treatment to limit fluid overload and to remove circulating inflammatory mediators. Analysis of plasma samples drawn at the start of RRT revealed a 6- fold increase of MV concentration in the S group (n=15) in comparison to NS group (n=15). Last, MVs derived from plasma of S group showed an enhanced expression of molecules involved in inflammation and apoptosis such as Fas-ligand and CD40-ligand and are enriched for different RNA subsets including microRNAs.
Conclusions: The results of our retrospective study showed that: 1) sepsis is the most relevant cause of AKI ; 2) the mortality rate in the S group is significantly higher than in the NS group; 3) In the NS group but not in the S group the observed mortality is lower than the expected mortality calculated; 4) the concentration of plasma MVs was significantly higher in the S group than in the NS group, suggesting a potential role of MVs derived from activated leukocytes and platelets as mediators of sepsis-associated AKI and as biomarkers of disease.

SAP123 THE ROLE OF PLASMA BIOMARKERS IN THE CHARACTERIZATION OF ICU PATIENTS WITH SEPSIS AND WITH AKI

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Introduction and Aims: Sepsis is a primary cause of morbidity and mortality in intensive care unit (ICU) and critically ill patients. Gram-negative bacteria are implicated in 50-60% of cases of sepsis in ICU and endotoxin is considered to play an important role in the pathogenesis of septic shock. Sepsis is also a contributing factor in more than 20% of cases of acute kidney injury (AKI) in ICU patients, with cases severe enough to require renal replacement therapy. AKI occurs in 35-65% of ICU admissions and most studies show a threefold to fivefold increase in the risk of death among patients with AKI compared to patients without AKI. Given the higher mortality rate of ICU patients with sepsis and AKI, we decided to investigate the possible correlation between serum biochemical markers of organ damage, such as Neutrophil Gelatinase-Associated Lipocalin (NGAL), Advanced Oxidation Protein Products (AOPP) and Brain Natriuretic Peptide (BNP) and endotoxin activity in ICU septic patients. Moreover, comparisons of the levels of these biomarkers were made between septic and non septic patients, septic patients with or without AKI and between patients who developed AKI with or without sepsis.
Methods: Ninety-eight consecutive adult patients admitted to ICU of San Bortolo Hospital, Vicenza, Italy, between October 2008 and August 2010, were enrolled in this study. Patients were divided in two groups depending on the presence of sepsis, defined as Systemic Inflammatory Response Syndrome (SIRS) associated with an infectious process. Fifty-six patients had sepsis, while forty-two patients were non septic. Among septic patients, twenty-four subjects developed AKI, defined by RIFLE criteria, while thirty-two did not. AKI occurred in fourteen patients without sepsis as well.
Results: A significant correlation ($p=0.02$) was found only between endotoxin activity and BNP levels of septic patients. The levels of NGAL, BNP and AOPP were significantly higher among septic patients compared with non septic subjects ($p<0.001$). Among septic patients, subjects who developed AKI showed significant higher levels of NGAL and AOPP ($p=0.0425$), and BNP ($p=0.0327$). Among patients who developed AKI, a significant difference was found only in terms of AOPP levels between septic and non septic patients.
Conclusions: The correlation between endotoxin activity and BNP in septic patients and the increase in the levels of NGAL, BNP and AOPP in case of sepsis and AKI, in particular if they are associated, indicate a multiorgan involvement in these two conditions. Their evaluation can allow clinicians to individualize earlier patients at higher risk of morbidity and mortality.

SAP124 CONTRAST MEDIA, NEPHROTOXICITY AND NEUTROPHIL-GELATINASE ASSOCIATED LIPOCALIN: BETWEEN DOUBTS AND CERTAINTIES

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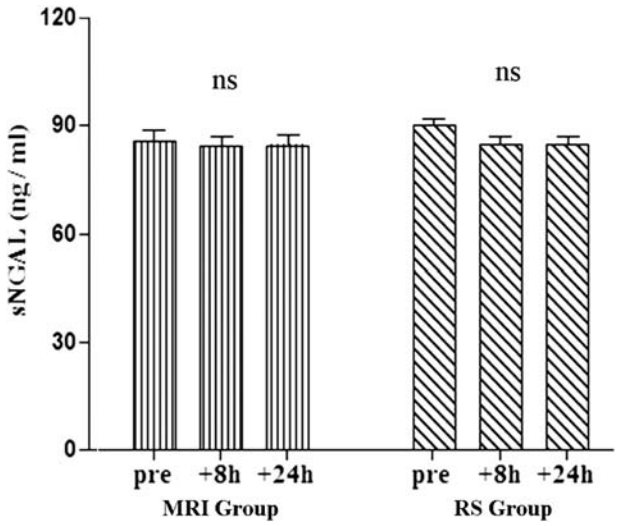
Introduction and Aims: There are no early predictive biomarkers of CIN, although great progress have been made in identifying patients at risk and in studying pathophysiological mechanisms. Neutrophil gelatinase-associated lipocalin (NGAL) is widely considered an excellent predictor of AKI. In the last decade, it has been conducted studies not only for the early detection of biomarkers, but also to assess several therapeutic strategies to prevent CIN. Unfortunately, there are not accepted guidelines. The main aim of the present study was to evaluate the diagnostic power of NGAL in detecting a possible event of CIN in a cohort of patients who received iodinate contrast media, gadoterate meglumine or radiopharmaceutical technetium-99. We also evaluated, in the same cohort, the potential protective effect exerted by the volume expansion with an isotonic saline infusion, with sodium bicarbonate or by N-acetylcysteine administration.
Methods: 120 patients were enrolled. 60 patients underwent computerized tomography (CT group) with administration of Iomeprol, 30 patients underwent magnetic resonance imaging (MRI group) with gadoterate meglumine administration and 30 patients underwent renal scintigraphy (RS group) with the use of the radiopharmaceutical 99mTc-DTPA. Biomarkers were determined from blood samples obtained before examination, 8h and 24h after administration of contrast agent. All the patients were randomly divided into three arms based on the type of drug infusion: N- acetylcisteine (NAC), physiological saline (SF) and sodium bicarbonate (BIC).
Results: In the MRI and RS groups, compared to baseline, changes in NGAL are not found statistically significant at the time +8 h and + 24 h. In these two groups, there were no cases of CIN.
In the CT group, we found a significant rise of NGAL after 8 and 24 h after iomeprol administration. Creatinine did not show statistically significant changes except that after 24 hours to the administration of contrast medium.
The area under the ROC curve demonstrated that the diagnosis of CIN within 8 h after the iomeprol administration is possible by using NGAL as early markers with high sensitivity and specificity.



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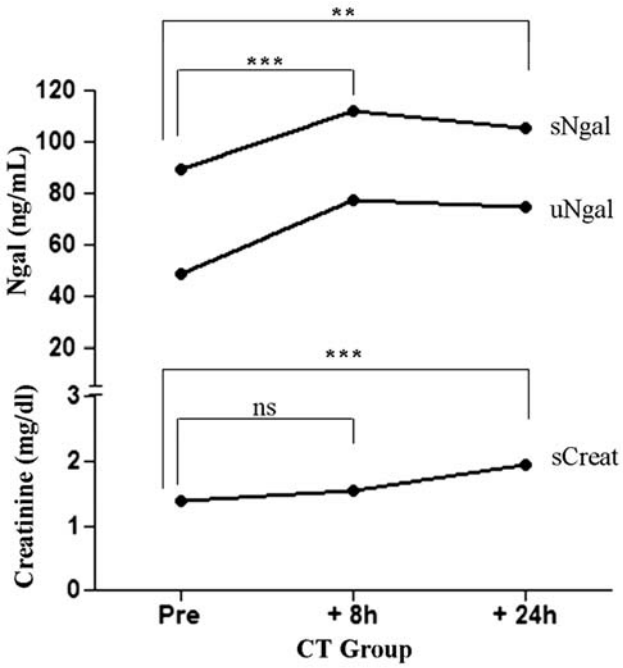

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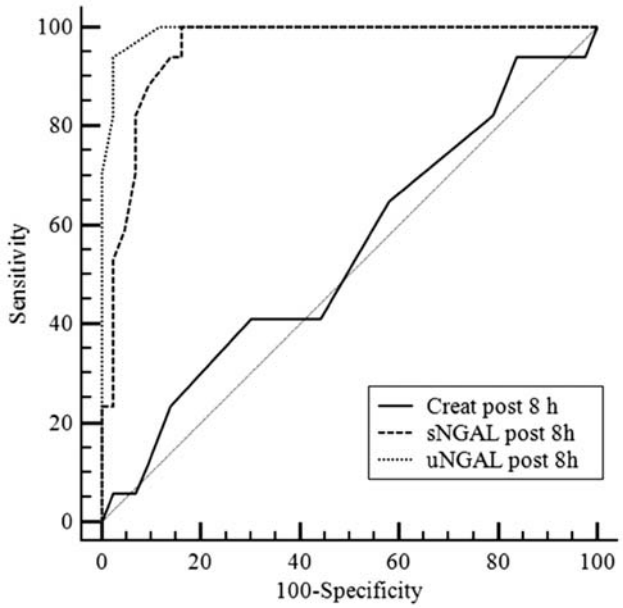

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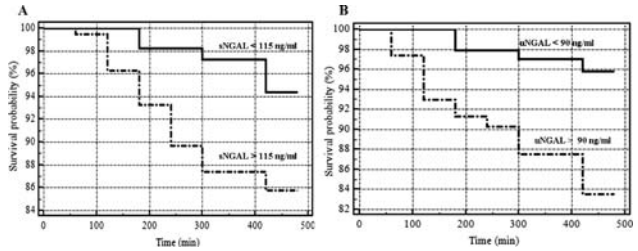
Subjects with NGAL values above 115 ng/ml experienced a significantly faster evolution to endpoint (p= 0.002), with a hazard ratio of 3.70 (95% CI, 1.62 to 8.45). A multiple Cox regression was also constructed.
All patients were also randomly divided into three arms based on the type of drug infusion: NAC, SF and BIC. We do not found any statistical differences of NGAL in the three groups.
Conclusions: This is the first research that utilizes NGAL to identify renal damage after administration of a radiopharmaceutical such as 99mTc-DTPA and meglumine gadoterate. In both cases, we found no events of CIN. NGAL levels did not show any statistically significant change, indicating a non-renal involvement. In condition of CIN, sCreat levels showed a statistically significant increase only 24 hours after administration of contrast medium. In contrast, NGAL was an early marker, with its



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Univariate and Multivariate Cox proportional hazards regression model for incidence of CIN

	Univariate Analysis			Multivariate Analysis		
	HR	95 % CI	p value	HR	95 % CI	p value
Age	1.01	0.95 – 1.07	0.22	1.00	0.98–1.02	0.48
GFR	0.86	0.79 – 0.94	0.002	0.96	0.94–0.98	0.05
sNGAL	1.03	1.03 – 1.09	0.0001	1.02	1.01–1.03	0.003
uNGAL	1.11	1.06 – 1.17	< 0.0001	1.03	1.02–1.04	0.0001
Contrast amount	0.98	0.96 – 1.00	0.15			

HR: hazard ratio; CI: confidence interval; sNGAL: serum NGAL; uNGAL: urinary NGAL; GFR: glomerular filtration rate.

variations observed just 8 hours after drug administration. We have also shown NGAL possesses a high diagnostic sensitivity and specificity, an excellent prognostic power representing the most valuable independent factor for detecting CIN. This study suggests no significant difference in the development of CIN between groups treated with preventive drugs, emphasizing the importance of hydration alone in the prevention of CIN.

SAP125 COMMUNITY ACQUIRED ACUTE KIDNEY INJURY IN THE ELDERLY AND VERY ELDERLY PATIENTS: A SINGLE CENTER EXPERIENCE

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Introduction and Aims: Data about the community-acquired acute kidney injury (CAKI) around the world including our country seems to be insufficient. In this sense there might be differences not only between countries, between age groups as well. It is important to exhibit the features of CAKI to develop preventive medicine strategies. Our aim is to evaluate the application reasons, etiologies, clinical complications, renal replacement treatment (RRT) needs, clinical outcomes and cost analysis of patients above 65 years of age with CAKI who admitted to emergency department.
Methods: Totally 3290 subjects with ages above 65 admitted to emergency department between May 2010 and May 2011. CAKI prevalence was 7.17% (n=236) in the elderly. The patients diagnosed as CAKI were evaluated retrospectively. The admission reasons, medical histories, co-morbidities, using medications, clinical symptoms and signs, etiologies, complications, treatment modalities, RRT necessities and features, clinical outcomes and cost analysis were investigated and recorded. Patients were divided into two groups with respect to age 65-75 yrs (n=136) (group 1) and >75 yrs (n=100) (group 2).
Results: The mean age of the patients was found to be 69,6±3,2 years in group 1 (n=136, male/female: 78/58), and 81,2±4,2 years in group 2 (n=101, male/female: 58/43). The most seen admission reason was vomiting in group 1 (n=54, 39,7%) and group 2 (n=43, 43%). Prerenal AKI was the most common CAKI type in group 1 (% 57,4, n=78), and also in group 2 (%63, n=63) subjects (p>0,05). Nephrotoxic agent use was found in % 88 (n=120) in group 1 and %91 (n=91) in group 2 (p>0,05). 57 (41,9%) of the patients in group 1 and 36 (36%) of the patients in group 2 had treatment history of ACE inhibitors, respectively (p<0,05). The intensive care unit, mechanical ventilator and RRT needs were found to be 51,4% (n=70), 22% (n=22) and 38,2% (n=52) in group 1 and 64% (n=64), 31% (n=31), 48% (n=48) in group 2,

respectively (p<0,05). While %90 (n=27) of the subjects needing mechanical ventilator died, overall mortality was %34,5 (n=47) in group 1, %95,6 (n=22) of the subject needing mechanical ventilator died, overall mortality was %55 (n=55) in group 2 (p<0,05). While the mean cost was 1366.25± 1119.58 Euro (€)/patient in group 1, the mean cost was 1583.75±1014.58 €/patient in group 2 (p<0,05).
Conc lusions: Even though CAKI which can often be seen among the elderly and very elderly subjects is a hinderable disease, and it still has a high morbidity and mortality rates. The most important factors for cost and mortality are older age, along with intensive care unit, mechanical ventilator needing, and RRT needs.
Key Words: Acute renal injury, elderly, cost, mortality.

SAP126 CLINICAL PRESENTATION, COURSE AND OUTCOME OF ACUTE KIDNEY INJURY DUE TO VITAMIN D INTOXICATION

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Introduction and Aims: Most of the Kashmiri population is Vitamin D deficient. Overall vitamin replacement, including vitamin D is very common in this part of the world either as self medication or malpractice related, with most of the elderly being given oral or injectable forms. At times doses prescribed are far above the permissible limit. This has resulted in many cases of vitamin D toxicity, some reported in literature. Its incidence has been on the rise in Kashmir valley recently and more cases are reporting to hospitals with complications. Vitamin D toxicity is a known cause of hypercalcemia and reversible acute kidney injury (AKI). We report 32 patients who had evidence of malpractice-related vitamin D intoxication, presenting with hypercalcemia and AKI. This is perhaps the largest case series ever reported.
Methods: 40 cases of Vitamin D toxicity were admitted in Department of Nephrology over last 18 months. Detailed investigations and follow up was available in 32 cases. The diagnosis of vitamin D intoxication was made on basis of history of excessive vitamin D injection intake (600,000 IU/injection), toxic levels of 25 OH Vitamin D and after ruling out common causes of hypercalcemia (malignancy and hyperparathyroidism). Their presentation was either AKI (Group 1) or acute on top of chronic kidney disease (Group 2).
Results:) In Group 1, there were 21 patients, whose mean age was 61.33±14.48 years, with a male predominance (12:7). The average in-patient days were 7.05± 3.03. The number of vitamin D injections received ranged from 2 to 28. Their creatinine at presentation was 2.95±.96 mg/dl, which decreased to 1.41 ± .27 mg/dl on follow up of 5.2± .6 months. Serum calcium on admission was 13.76 ±1.47 mg/dl and it decreased to 10.79 ±1.23 mg/dl on follow up. The vitamin D level was 313.33 ± 54.84 nmol/L and PTH was 18.13 ± 9.62 pg/ml. In Group 2, 11 patients were studied; their mean age was 64.11±13.01 years, with a female predominance (4:5). The average admissions days were 7.77± 3.86. The number of injections received ranged from 3 to 24. Their creatinine at presentation was 4.03±1.17 mg/dl, which decreased to 3.32 ± 1.09 mg/dl on follow up. Calcium on admission was 13.68 ±2.12 mg/dl and it decreased to 11.11 ±1.08 mg/dl. The vitamin D level was 303.73 ± 48.41nmol/L and PTH was 22.31 ± 12.69 pg/ml. The clinical presentation was weakness in 100%, constipation in 80%, abdominal pain in 60%, nausea and vomiting in 60%, anorexia in 50%, oliguria in 20%, altered sensorium in 20%, hearing impairment in 2%. The treatment received was intravenous fluids in all, normal saline and steroids (short course) in 28 and bisphosphonates in 5.
Conclusions: This case series elucidates the increasing incidence of vitamin D toxicity in Kashmiri population. It is an important cause of reversible AKI which responds to conservative measures. It is necessary to educate the people about vitamin D deficiency, its appropriate treatment, and as well inform the caregivers in the peripheries about the symptoms of acute vitamin D intoxication, and stress about the possible dangers of mega doses vitamin D.

SAP127 RENAL ARTERY STENOSIS: THE COMPARISON OF THE DOPPLER USG, CONTRAST-ENHANCED MAGNETIC RESONANCE ANGIOGRAPHY AND SELECTIVE RENAL ARTERIOGRAPHY

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Introduction and Aims: Conventional selective renal arteriography (SRA) is the gold standard diagnostic method for renal artery stenosis (RAS). SRA is an invasive procedure and has puncture-site related and systemic complications including contrast-mediated nephropathy. Therefore, there is a need for a diagnostic method that is both accurate and safe. Contrast-enhanced 3D magnetic resonance angiography (CEMRA) and renal artery Doppler ultrasonography (DUSG) have been used increasingly for RAS. But, diagnostic utility of these methods is still controversial. To assessment of diagnostic specificity and sensitivity of CEMRA and DUSG.
Methods: Sixty-five consecutive patients who have been investigated for resistant hypertension were assessed. The patients were divided into two group with respect the age, <60 yr group 1, and >60 yr group 2. DUSG was performed to 12 of group 1 and 8 of group 2 patients. CEMRA was performed to 12 of group 1 and 11 of group 2 patients. Both DUSG and CEMRA were performed to 12 of group 1 and 10 of group 2 patients. After these methods, SRA was performed to all patients.
Results: There were 36 patients (12 male, 24 female) in group 1, and 29 patients (13 male, 16 female) in group 2. Mean age was 42,15±12,1 (range, 18-59) years in group 1 and 68,75±22,34 (range, 60-86) years in group 2. SRA was used as the standard of reference. Total of 132 renal arteries were evaluated. DUSG and SRA were concordant in 82,60% and 56,25% of the arteries in group 1 and 2, respectively. CEMRA and SRA were concordant in 66,66% and 90,47% of the arteries in group 1 and 2, respectively. In the evaluation of clinically significant renal artery stenosis (=50%) with DUSG, the overall sensitivity, specificity, positive predictive value, and negative predictive value were 83,33%, 81,82%, 83,33%, 81,82% in group 1 and were 69,23%, 0%, 75%, 0% in group 2 respectively when compared with SRA. In the evaluation of clinically significant renal artery stenosis (=50%) with CEMRA, the overall sensitivity, specificity, positive predictive value, and negative predictive value were 92,31%, 36,36%, 63,16%, 80,00% in group 1 and were 100,00%, 33,33%, 90,00%, 100,00% in group 2 respectively when compared with SRA.
Conclusion: CEMRA and DUSG are the accurate non-invasive techniques for identifying RAS in patients above 60 years of age and under 60 years of age, respectively.
Key Words: Renal artery stenosis, MR angiography, selective renal arteriography, Doppler USG.

SAP128 THYROID FUNCTION TESTS IN ACUTE KIDNEY INJURY

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Introduction and Aims: Little is known about thyroid function in the course of an acute kidney injury (AKI). The aim of our study is to define these changes in thyroid function.
Methods: A prospective study in 35 patients hospitalized for AKI for 2 consecutive years was carried out. TFT (serum thyrotropin, TSH; free thyroxine, FT4; and total triiodothyronine, T3 concentrations) were measured in each patient on three occasions: at admission, at hospital discharge and at their first outpatient visit.
Results: Total prevalence of alterations in TFT was 82.9% (n=29). Of those, euthyroid sick syndrome (ESS) with low T3 only was the most common (n= 13, 37.1%) derangement. In the whole group of patients TSH [0.93 (0.35-2.27) µU/ml] and FT4 (1.2 ± 0.3 ng/dl) were normal, whereas T3 was low (0.7 ± 0.1 ng/ml). TSH, FT4 and T3 were similar in different types of AKI. In the simple regression analysis we only found a negative correlation between TSH and serum urea concentrations (r=-0.382; p=0.024). At hospital discharge [median hospital stay 6 days (2-10)], TFT showed significant changes only in T3 concentrations (0.8 ± 0.3 ng/ml,p=0.013). At this point, the percentage of patients with normal TFT increased from 17.1% at baseline to 40% at discharge and then to 66.7% at their first outpatient visit. We found no association between the presence and type of alterations in TFT and clinical (sex, age, personal history of diabetes and/or hypertension, number and type of drugs used, signs and symptoms, and degree, type and etiology) and prognostic (hospital stay, recovery of renal function, need of renal replacement therapy, residual chronic renal failure and mortality) factors associated to AKI.
Conclusions: Over 80% of AKI patients exhibit alterations in TFT. The commonest derangement is ESS (~70%), mainly low T3 syndrome, which is present in about one third of the patients with altered TFT. ESS recovers spontaneously as renal function improves. The presence of TFT alterations seems not to be associated with clinical and prognostic implications in AKI patients.

SAP129 THE KIDNEY-LUNG CROSSTALK AND MORTALITY IN A COHORT OF PATIENTS WITH SEVERE LEPTOSPIROSIS (WEIL SYNDROME) IN BRAZIL

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Introduction and Aims: Leptospirosis is the most important zoonosis in the world. The severe form (Weil syndrome) is characterized by acute kidney injury (AKI), jaundice and pulmonary hemorrhage, with high mortality rates. The aim of this study is to investigate the kidney-lung interactions and its impact in mortality among patients with severe leptospirosis.
Methods: This is a retrospective study conducted at a tertiary infectious diseases-specialized hospital in Fortaleza city, Northeast Brazil, including 45 patients with confirmed diagnosis of severe leptospirosis admitted to the intensive care unit. AKI was defined according to the RIFLE criteria, and it was compared the results

between patients in "Failure" (RIFLE-F) and "Risk"/"Injury" (RIFLE-R/I). Severity was assessed through APACHE II and SOFA criteria. A comparison between survivors and non-survivors was also done. Statistical analysis was done with SPSS program, version 16.0, considering as significant p<0.05.
Results: Patient`s average age was 42±15 years, and 82% were male. According to the RIFLE criteria patients were at "Risk" (12%), "Injury" (20%) and "Failure" (68%), and dialysis was required for 33 patients (73.3%). Patients in RIFLE-F were older than those in RIFLE-R/I (43±15 vs. 32±13 years, p=0.02). Mean APACHE II score at admission was 20.1±8, and SOFA was 14.8±4.8. APACHE II scores were higher in patients in RIFLE-F (22±6.2 vs. 14±8.6 in RIFLE-R/I, p=0.001), as well was SOFA (RIFLE-F: 16±4.2 vs. RIFLE-R/I: 11±3.9, p=0.0005). Invasive mechanical ventilation was required for 30 patients (66%), and it was more frequently required for patients with RIFLE-F (77.4% vs. 42.8%, p=0.03). The arterial oxygen tension/fractional inspired oxygen ratio (PaO2/FiO2) at admission was lower in the RIFLE-F group (160±100mmHg vs. 183±87), but the difference was not statistically significant (p=0.46). The comparison between patients who required dialysis with those who did not required evidenced a higher frequency of mechanical ventilation (81% vs. 25%, p=0.0008) and lower PaO2/FiO2 ratio (156±97mmHg vs. 196±88mmHg), but with no significant difference (p=0.21). Death occurred in 20 cases (44.4%), and it was higher in patients with RIFLE-F (58% vs. 14.2%, p=0.0001) and in those who required dialysis (57% vs. 8.3%, p=0.005).
Conclusions: AKI in Weil syndrome is associated with severe lung involvement, as evidenced by a higher requirement for mechanical ventilation and higher mortality. Requirement of dialysis is also associated with poor respiratory function and outcome. RIFLE criteria is a good predictor of severity in this group of patients, as demonstrated by the higher APACHE II and SOFA scores in patients with RIFLE-F. Efforts should be made to provide early and adequate lung support for patients with severe leptospirosis.

SAP130 REDUCED SYSTEMIC OXYGEN DELIVERY AND LOW BLOOD PRESSURE ON DAY OF EARLY AKI INCREASE THE RISK OF PROGRESSION TO SEVERE AKI IN CRITICALLY ILL PATIENTS

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Introduction and Aims: Acute kidney injury (AKI) is common in critically ill patients and associated with increased mortality, morbidity and financial costs. Research in high risk surgical patients has shown that pre-emptive strategies of perioperative haemodynamic monitoring and optimisation can reduce the incidence and mortality of postoperative AKI. There are no data on the role of haemodynamic monitoring in critically ill patients with early AKI. Our objective was to investigate the association between haemodynamic parameters at time of early AKI and risk of progression to severe AKI in critically ill patients in the Intensive Care Unit (ICU).
Methods: We retrospectively reviewed the electronic medical records of all patients admitted to the ICU in a tertiary care centre between June 2007 - June 2009 and identified patients with AKI according to the AKI Network criteria who underwent advanced haemodynamic monitoring within 12 hours of diagnosis of AKI stage I. Patients who died or were discharged on the day of AKI diagnosis and patients with AKI stage III on admission to ICU were excluded. Haemodynamic parameters were recorded on the day of AKI stage I and daily until progression to AKI stage III or return to baseline renal function. Logistic regression analysis was employed to determine independent predictors of progression to AKI stage III.
Results: 205 patients (mean age: 66 years; 138 male) underwent haemodynamic monitoring within 12 hours of the diagnosis of AKI I of whom 85 (41.46%) progressed to AKI III. Median time to progression was 2 days (range 1-23). Patients who progressed to AKI III had a lower cardiac index (median 2.82 vs 3.35 l/min/m2; p=0.004), lower indexed systemic oxygen delivery (DO2I) (median 342.3 vs 405.0 ml/min/m2; p=0.006), lower mean arterial blood pressure (MAP) (median 71 vs 74 mmHg; p=0.011) and a higher central venous pressure (median 13 vs 11 mmH2O, p=0.043) on the day of AKI I, compared to patients who did not develop AKI III. After adjusting for demographic factors, severity scores on admission and other covariates, multivariate analysis showed that a higher DO2I [Odds ratio (OR) per 50 ml/min/m2 increase 0.86 (95% CI 0.82 – 0.95)], and a higher MAP (OR per 5 mmHg increase 0.80, 95% CI 0.66 – 0.985) on the day of AKI I were independently associated with a reduced risk of progression to AKI III. A positive fluid balance after the diagnosis of AKI I was associated with an increased risk of AKI progression in multivariate analysis including adjustment for urine output (OR 1.60 per 1000ml/day increase; 95% CI 1.06 – 2.40; p = 0.024). Rises in DO2I and MAP after the diagnosis of AKI I were not independently associated with reduced risk of progression to AKI III. Mean fluid gain/day after the diagnosis of AKI I was the strongest predictor of progression (area under the receiver operating characteristics curve 0.776).
Conclusions: A higher DO2I and MAP on the day of AKI I are associated with a lower risk of progression to AKI III. Once AKI is established, efforts to improve these hemodynamic parameters are not effective and may even be deleterious if associated with excessive fluid administration.

SAP131 ACUTE KIDNEY INJURY ACCORDING TO RIFLE CLASSIFICATION IN THE INTENSIVE CARE UNIT: OCURRENCE, RISK FACTORS, OUTCOME AND PREDICTIVE FACTORS

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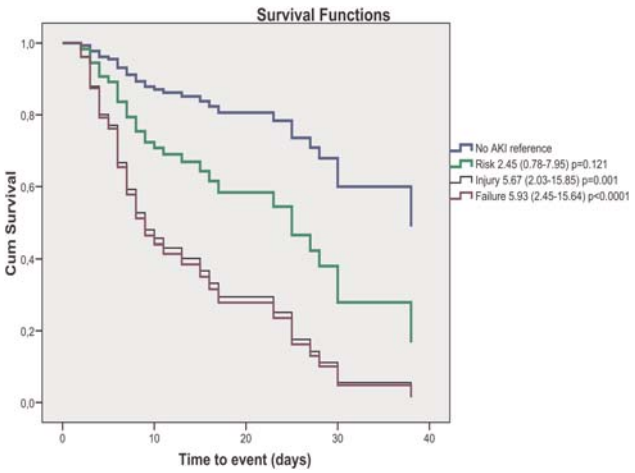
Introduction and Aims: Aim of this study was to evaluate incidence, risk factors, outcome of acute kidney injury (AKI) in the intensive care unit (ICU), based on the RIFLE classification, and predictive factors for ICU mortality.
Methods: Adult patients (<14 years) admitted in two medical surgical ICUs, tertiary care center, between January 2007 and December 2007 were reviewed retrospectively. Patients who stayed more than 24 hours were included. Transplanted and chronic dialysis patients before admission to the ICU were excluded. AKI was classified according to the maximum RIFLE criteria using both serum creatinine and urine output (UO) criterion during the first week of stay. Demographic, severity scores of illness (SOFA score) on admission and outcome data, were studied and compared between non AKI and AKI patients.
Results: 382 ICU patients were included for the study. According to the RIFLE classification, 192 (50.3%) patients met criteria for AKI during the study period and were classified as non AKI 190 (49.7%), Risk 73 (19.1%), Injury 69 (18.1%), Failure 50 (13.1%) patients. There was concordance between the serum creatinine and urine output criteria in 59,4% of cases. In 35,6% of cases, the creatinine criteria led to a worse RIFLE class, whereas in 5% of cases, it was the urine output criteria that led to a worse RIFLE class. ? was 0.368 (P < 0.001). Diuretics (mainly furosemid) were used in 73,3% AKI patients. On the first day of ICU admission 46 patients (12%) had AKI, defined by the RIFLE criteria. During the entire ICU stay 146 patients (38,3%) had an episode of AKI defined by RIFLE criteria. 30 (29,1%) of the patients with initial RIFLE class R progressed to maximum RIFLE class I or class F, and 10 (17,2%) of the patients with initial RIFLE class I progressed to maximum class F. Male AKI patients were 66,1%. AKI patients were aged, median [IQR] 60 (52-68) vs. non AKI patients 55 (31-73); p=0,008 and had higher SOFA score: AKI 5 (3-8) vs. non AKI 3 (1-4), p<0.001. Multivariate logistic regression analysis showed age (OR: 1.02, 95% CI: 1.004-1.043, p=0.019), SOFA score (OR: 1.62, 95% CI: 1.42-1.85, p<0.001) as independent factors associated to AKI development. Renal replacement therapy was in 1,56% AKI ICU patients. Complete recovery of renal function at the time of death or hospital discharge was 52,9%. Mortality increased linearly from non AKI 5 (2,6%), Risk 7 (9,6%), Injury 14 (20,3%), Failure 29 (58,0%). Sepsis was in 15,1% AKI patients and oliguria (UO<0.3 ml/kg per h 24 h or anuriá12 h) in 12,5%. Multivariate logistic regression analysis, for independent risk factors associated to mortality showed, Risk (OR: 0.57, 95% CI: 0.25-1.33, p=0.198); Injury (OR: 1.68, 95% CI: 0.86-3.30, p=0.127); Failure (OR: 16.25, 95% CI: 8.15-32.39, p<0.001); age (OR: 1.17, 95% CI: 1.08-1.27, p<0.001), sepsis (OR: 11.03, 95% CI: 3.46-31.42, p= 0.004); sCr-max (OR: 1.17, 95% CI: 0.67-2.02, p= 0.580); oliguria (OR: 7.60, 95% CI: 5.31- 15.40, p= 0.002). Kaplan-Meier curve for ICU survival by RIFLE class with Cox regression analysis was statistically significant (p<0,0001, fig 1).
Conclusions: According to RIFLE criteria high incidence of AKI in ICU patients is associated with worse outcome. Advanced age and higher SOFA score are independently associated to AKI development. Age, sepsis, oliguria and failure of renal function according to RIFLE were found independent factors associated to mortality of ICU patients. Urine output criterion was found to be less sensitive but high predictive.



SAP131 Figure 1:

SAP132 REDUCED BLOOD PRESSURE AND FLUID ACCUMULATION IN CRITICALLY ILL PATIENTS WITH EARLY AKI ARE ASSOCIATED WITH INCREASED MORTALITY

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Introduction and Aims: Acute kidney injury (AKI) is associated with significantly increased short and long-term morbidity and mortality, especially in critically ill patients. Studies in high risk surgical patients have shown that haemodynamic monitoring and optimization have a beneficial effect on outcome, including risk of AKI, complications and mortality. The objective of this study was to evaluate the association between haemodynamic parameters on day of diagnosis of AKI and hospital outcome in critically ill patients with early AKI.
Methods: We retrospectively reviewed the electronic medical records of all patients admitted to the Intensive Care Unit (ICU) in a tertiary care centre between June 2007 - June 2009 and identified patients with AKI as per AKI Network criteria. We analysed the data of patients who had undergone advanced haemodynamic monitoring within 12 hours of diagnosis of AKI. Patients who died or were discharged on the day of AKI diagnosis and patients with AKI stage III on admission were excluded. We recorded haemodynamic parameters [systemic oxygen delivery index (DO2I), cardiac index, central venous pressure (CVP), mean arterial blood pressure (MAP)], hemoglobin, oxygen saturation, fluid balance (FB) and urinary output (UO) on the day of AKI stage I diagnosis and daily until progression to AKI stage III or return to baseline renal function. Logistic regression was employed to determine independent predictors for hospital mortality.
Results: During the 24 months period, 210 patients (mean age: 67 years; 142 male) underwent haemodynamic monitoring on the day of AKI I of whom 68 (32.4%) died in hospital. The hospital mortality of patients who progressed from AKI I to AKI III was 57.6% compared to 11.7% in patients who did not progress ($p < 0.001$). Multivariate analysis showed that of all haemodynamic parameters recorded on day of AKI I, only MAP in the first 12 hours after the diagnosis of AKI was independently associated with hospital mortality [Odds ratio (OR) per 1 mmHg increase: 0.95, 95% confidence interval (CI) 0.91-0.99, p=0.022]. An increase in MAP by =5mmHg between AKI I and AKI III was independently associated with a decreased risk of death (OR 0.44, 95% CI 0.23-0.85, p=0.014), whereas an increase in cumulative FB by >1000ml was independently associated with an increased risk of mortality (OR 1.90, 95% CI 1.05-3.44, p=0.034). No association was found between the trends in other haemodynamic parameters and hospital outcome. Mean fluid gain/day after the diagnosis of AKI I was 663ml (SD 993 ml) among survivors and 1239ml (SD 1545ml) in non-survivors ($p = 0.0055$). In multivariate analysis, each 1000ml/day increase in FB after the diagnosis of AKI I was associated with 46% increased risk of death after adjustment for demographic factors, severity scores, other haemodynamic variables and UO (OR 1.46, 95% CI 1.10-1.92, $p = 0.008$).
Conclusions: Strategies to reduce mortality in critically ill patients with early AKI should focus on achieving and maintaining an adequate MAP, while avoiding excessive fluid administration. Clinical trials are necessary to establish the best type of haemodynamic optimization for patients at risk of AKI or with early AKI.

SAP133 ACUTE KIDNEY INJURY IN PATENTS HOSPITALIZED WITH ACUTE DECOMPENSATED HEART FAILURE DEPENDING ON HYDRATION STATUS

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Introduction and Aims: Patients with acute decompensated heart failure (ADHF) are usually admitted with severe systemic congestion. Volume overload it the known main driver for morbidity, mortality and readmission to the hospital. Bioimpedance vector analysis (BIVA) is a non-invasive, accurate technique for hydration status evaluation. The aim of the study was to determine the hydration status in patients with ADHF by BIVA and to evaluate the association of fluid status with clinical characteristics and worsening of renal function, short-term and long-term outcomes.
Methods: in 55 patients admitted with ADHF (38 male, 68.7±10.2 years (M±SD), BMI 31.1±6.9 kg/m2, 96% arterial hypertension, 81% ischemic heart disease, 64% myocardial infarction, 63% atrial fibrillation, 59% diabetes mellitus, known chronic kidney disease 32%, ejection fraction (EF) 36.8±11.2%) fluid status was assessed by BIVA. Hydration status was expressed using absolute values of total body water (TBW), extracellular body water (EBW). Worsening of renal function was defined using AKIN and RIFLE criteria of acute kidney injury (AKI). Mann-Whitney and Spearman tests were performed. P <0.05 was considered statistically significant.
Results: 28% of patients developed AKI. Patients with AKI compared with patients without AKI had on admission higher BP levels (146.3±11.5 vs 135.0±10.9 mmHg, p<0.05), respiratory rate (24.3±3.5 vs 20.1±2.5, p=0.009), serum creatinine (156±21 vs 112±17 mkmol/l, p<0.05), lower EF (35.6±5.0 vs 37.6±4.5%, p<0.05), eGFR (50.2 ±15.2 vs 66.7±14.3 ml/min/1.73m2, p<0.05), resistance R (355.3±77.8 vs 376.2±91.4 Om, p<0.05) and reactance Xc (28.4±16.2 vs 29.5± 13.4 Om, p<0.05). The patients with AKI demonstrated higher volume overload compared with patients without worsening of renal function (TBW 51.4±6.6 vs 49.9±7.5 kg, p<0.05; EBW 24.2±2,9 vs 20.9±3.4 kg, p=0.04). Patients without AKI compared with patients with AKI demonstrated better short-term and long-term outcomes: the 30-days mortality 0% vs 12.5%, readmission to the hospital 13% vs 22%. Died patients had significantly lower resistance R (286.3±37.5 Om vs 381.3±84.4 Om, p=0.04). Readmitted patients compared with patients without rehospitalization had lower resistance (349.0±51.4 vs 374.4±93.5 Om, p<0.05) and reactance (20.8±8.1 vs 31.4±15.1 Om, p<0.05).
Conclusions: 28% of patients admitted to the hospital with ADHF developed AKI. Patients with AKI had worse prognosis compared with patients without renal deterioration. Evaluating hydration status by BIVA added useful information to standard clinical parameters and could help to determine the patient population with higher risk of development of AKI.

SAP134 ACUTE KIDNEY INJURY IN PATIENTS WITH NON-ST-ELEVATION ACUTE CORONARY SYNDROME

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Introduction and Aims: The aim of the study was to evaluate the prevalence and predictors of acute kidney injury (AKI) in patients with non-ST-elevation ACS (NSTE-ACS) and to determine the impact of worsening of renal function on in-hospital mortality.
Methods: 218 patients were examined (33% male, 73±12 years (M±SD), BMI 30.0 ±4.4 kg/m2, 8% smokers, type 2 diabetes mellitus (T2DM) 21%, previous MI 40%, arterial hypertension 91%, heart failure 32%, atrial fibrillation 23%, confirmed chronic kidney disease 16%, BP 142±29/81±16 mmHg, serum creatinine (SCr) 106 ±50 mmol/l, GFRMDRD 60±22 ml/min/1.73m2). AKI was diagnosed and staged according Acute Kidney Injury Network (AKIN) criteria (an absolute increase in the SCr =0.3 mg/dl, an increase =50% (1.5 times) from baseline in a 48-hour time frame, or a reduction in urine output with documented oliguria of <0.5 ml/kg per hour for >6 hours). Mann-Whitney and Spearman tests were performed. P <0.05 was considered statistically significant.
Results: Based on the measurement of troponins NSTE-ACS was further qualified as non-ST-elevation myocardial infarction (NSTEMI) in 145 patients (66.5%). Prevalence of AKI in NSTE-ACS was 32.2%. Prevalence of AKI in NSTEMI was 42%, in unstable angina – 15% (p<0.05). Prevalence of AKI stage 1, 2, and 3 was 25, 3.6 and 3.6%, respectively. 39% of AKI occurred within 1 day and 79% of AKI – within 3 days of hospital admission. Patients with AKI compared with patients without AKI had on admission higher SCr (129±73 vs 95±29 mmol/l, p<0.001), urea (11.1±7.1 vs 7.8±3.8 mmol/l, p<0.001), GFR (52±22 vs 64±21 ml/min/1.73m2). AKI was transient (resolved during hospital stay) in 54% and persistent (elevations of SCr that persist at patient discharge) – in 46%. Patients with AKI compared with patients without worsening of renal function demonstrated worse short-term outcomes: in-hospital mortality 17% vs 5% (c2=8.4; ?=0.003).
Conclusions: 32.2% of patients admitted to the hospital with NSTE-ACS developed AKI. AKI stage 1 (AKIN classification) was prevalent and mostly occurred within 3 days of admission. Serum creatinine on admission was found as a predictor of AKI. AKI has been associated with an increased risk of in-hospital death in NSTE-ACS.

SAP135 DISTANT EFFECTS OF AKI ON LUNG INJURIES BY SOLUBLE MEDIATORS IN BRONCHO-ALVEOLAR LAVAGE FLUID IN PATIENTS WITH ACUTE LUNG INJURY / ACUTE RESPIRATORY DISTRESS SYNDROME

Hiroki Hayashi[1], Hiroki Hayashi[2], Sachiyo Suzuki[3], Kensuke Kataoka[4], Yasuhiro Kondoh[4], Hiroyuki Taniguchi[4], Daisuke Sugiyama[5], Kunihiro Nishimura[5], Waichi Sato[3], Shoichi Maruyama[3], Seiichi Matsuo[3] and Yukio Yuzawa[6]

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Introduction and Aims: Recent experimental studies have provided evidence for kidney-lung crosstalk in AKI, and have highlighted the pathophysiological significance of inflammatory dysregulation induced by cellular and soluble mediators. However, this association is not clearly shown in human studies. The aim of our study was determine the impact of AKI on the prognosis of the patients who develop acute lung injury/ acute respiratory distress syndrome (ALI/ARDS) and to determine the progonostic value of various cellular and soluble mediators in the borchoalveolar lavage fluid (BALF).
Methods: We performed a retrospective analysis of 52 patients who developed ALI/ ARDS and were controlled by the noninvasive positive pressure ventilation (NPPV)

with borchoalveolar lavage in our hospital.
AKI was defined by the increases of serum creatinine level 50 % after induction for NPPV over 7days. Primary end point was NPPV failure (the induction for endotracheal ventilation or the death in hospital). The association between AKI and biologic markers was also investigated.
Total fifteen biologic markers that reflect endothelial and epithelial injury, inflammation, coagulation and capillary permeability including thrombomudulin [TM], surfactant protein D [SP-D], tumor necrosis factor [TNF]-a, interleukin [IL]-1b, IL-6, IL-8, IL-10, IL-18, interferon [INF]-g, monocyte chemoattractant protein [MCP]-1, intercellular adhesion molecule [sICAM]-1, plasminogen activator inhibitor [PAI]-1, neutrophil, macrophage and Albumin were measured in BALF. We conducted cox-proportional hazard model analysis for the evaluation of AKI and biologic markers for the risk of NPPV failure.
Results: A total of 52 patients were studied: 24 with AKI and 28 without AKI (non-AKI group). The groups were similar in regard to age, sex, etiology of ALI/ARDS and the serum creatinine at the baseline. NPPV failure in AKI group is significantly higher than in non-AKI (20 (83%) vs. 13 (46%); P=0.009).
The levels of biologic markers in AKI and non-AKI group were summarized in Table1. AKI group show the higher level of biomarkers. Furthermore,IL-6, IL-8, IL-10, IIFN-?, MCP-1 and PAI-1 were associated with NPPV failure statistically in univariate analyses. After adjustment for clinical risk factors, the best model for predicting NPPV failure included AKI, APACHE II score excluding kidney, IL-10 and SP-D as independent predictors (Table2). Since IL-6 in highest quintiles showed significantly higher hazard ratios(HR= 6.8*108, 95%CI 2.2-21*108) for the risk of NPPV failure in AKI group compared to non AKI group , the interaction between AKI and IL-6 in ALI/ARDS lung existed.
Conclusions: AKI was associated with the elevation of various biologic markers in BALF of ALI/ARDS patients and these biomarkers clearly predicted the NPPV failures. This association suggested the cross-talk between kidney and lung in ALI/ARDS

SAP135 Table 2 Best Predictive Model for the risk of NPPV failure selected from backward elimination

Predictors	HR	P-value	95% CI
AKI	2.63	0.02	1.17-5.93
Female	0.24	0.06	0.056-1.06
APACHE II score excluding kidney	1.11	0.03	1.01-1.22
log(SP-D)	2.86	0.03	1.13-7.26
log(IL-10)	1.59	0.01	1.11-2.26
log(ICAM-1)	0.39	0.07	0.13-1.09

SAP136 COMPARED ANALYSIS OF THE MORTALITY IN PATIENTS WITH ACUTE RENAL FAILURE VS MULTI-ORGAN FAILURE IN FRENCH ICU

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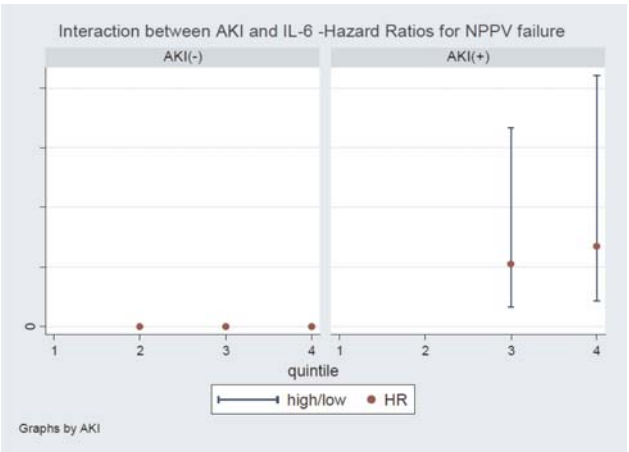
Introduction and Aims: The exact incidence of death as an independent complication of acute renal failure (ARF) is poorly known. The reason for this is that most clinical studies addressing the issue of mortality in the context of ARF come from intensive care unit (ICU) departments; the majority of ICU patients did or will develop acute kidney injury (AKI) in the setting of multiple organ failure (MOF), which by definition is life-threatening. Our aim here was to compare the mortality of isolated vs MOF-associated AKI.
Methods: During a six months period (from June to December 2009), we performed a prospective and bicentric study which included 197 consecutive patients with AKI, stratified according to the RIFLE classification, and admitted in two different french ICUs located in the same hospital in Paris: one that is a polyvalent ICU and another one that is a "renal" ICU, actually dedicated to patients with severe but isolated kidney failure. The aim was to compare the mortality at day 28 and month 3 in two categories of patients: those with AKI/MOF (i.e. AKI associated with at least one other organ failure: hemodynamic, respiratory or neurologic) and those with isolated AKI (iAKI). The secondary objective was to determine risk factors for death in patients with AKI.
Results: 108 patients had iAKI, vs 89 who had AKI/MOF. Both population study groups were comparable for the age and sex distribution, and for comorbidities. As expected, the Sequential Organ Failure Assessment (SOFA) and the New Simplified Acute Physiology (SAPS2) admission scores were higher in AKI/MOF patients. In iAKI patients, the distribution of the maximum RIFLE class was: Risk (R) in 18,5%, Injury (I) in 25,9% and Failure (F) in 55,6%; 25.9% of them required renal replacement therapy (RRT). This should be compared to R 33.7%, I 28.1%, F 38.2% in AKI/MOF patients, 22.5% of whom required RRT. AKI severity was thus higher in iAKI patients; the maximum serum creatinine was 351 mmol/l (range 207.0-537.5) in iAKI patients vs 198mmol/l (138.0-401.0) in AKI/MOF patients (p<0,01). Despite that, mortality at 28 days and 3 months was significantly lower in iAKI patients (4.6% and 13.0%, respectively) than in AKI/MOF patients (27.0% and 40.4%, p<0.01). The existence of a malignant tumour was the most significant risk factor associated with death in iAKI patients, both at day 28 (OR=15; 95%IC[2.48; 90.9] p<0,01) and at 3 months (OR=14.9; 95%IC[3.8;58.6], p<0,01). Incidentally, the RIFLE class, and the need for RRT were not found to be significantly associated with death.
Conclusions: The impact of iAKI on vital prognosis is relatively low, with 4.6% and 13% of deaths at one and three months, respectively, in patients admitted in a renal ICU. A malignancy is the strongest risk factor for a fatal outcome in this population.



SAP135 Figure 1: Interaction between AKI and IL-6 -Hazard Ratios for NPPV failure

SAP137 HEMOPERFUSION PLUS CONTINUOUS VENO-VENOUS HEMOFILTRATION ON PATIENTS WITH MULTIPLE ORGAN DYSFUNCTION SYNDROME

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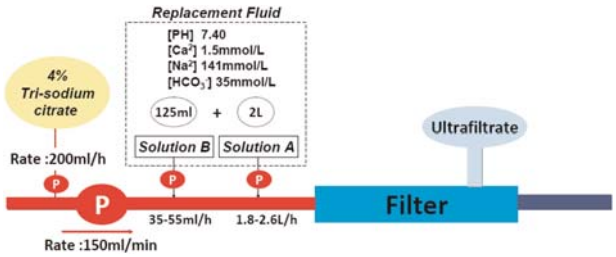
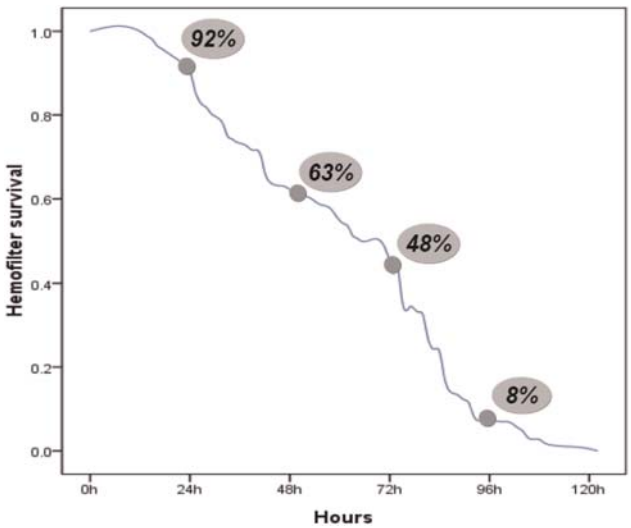
Introduction and Aims: In this randomized controlled clinical trail, we aimed to compare the effect of hemoperfusion (HP) plus continuous veno-venous hemofiltration (CVVH) with CVVH only, on the 60-day mortality among patients with multiple organ dysfunction syndrome (MODS) complicated with acute kidney injury (AKI).
Methods: 42 patients with MODS complicated with AKI were randomly assigned into study group (HP plus CVVH) and control group (CVVH). In study group, hemoperfusion was carried out immediately when CVVH was started (0h), with hemoperfutor installed along the extracorporeal pipeline right after the hemofilter. Hemoperfusion was performed for 2 hours (2h) and repeated in the next two days at the same moment for the same time. Patients in control group received the same treatment except hemoperfusion. Blood samples were drawed at 0h, 2h, 24h, 26h, 48h, 50h, and 72h respectively to test tumor necrosis factor-a (TNF-a), interleukin-1β (IL-1β), interleukin-6 (IL-6),and interleukin-10 (IL-10), ultrafiltrate was collected at the same time to test TNF-a, IL-1β, IL-6, and IL-10 level. Additionally, acute physiology and chronic health evaluation II score (APACHE II), oxygenation index, hospitalization time, and some other lab tests results were documented.
Results: 22 patients were randomly assigned into study group while 20 patients in control group. Non-significant higher 60-day survival rate was found in study group (59.1%) than in control group (45.5%) (P>0.05). APACHE II score was dramatically decreased in both groups, but the decrement of APACHE II score from 0h to 72h was significantly higher in study group than control group (P<0.05). Plasma level of TNF-a, IL-1β, IL-6, IL-10 at 72h were significantly lower than 0h in both groups (P<0.05). Besides, serial plasma levels of TNF-a, IL-1β, IL-6, and IL-10 in study group were significantly lower than those in control group at the same time (P<0.05).
Conclusions: HP combined with CVVH may improve the 60-day survival rate of MODS patients with AKI comparedwith CVVH only, which may be explained by more effective clearance of cytokines.

SAP138 A NEW CITRATE ANTICOAGULATION PROTOCOL IN EXTRACORPOREAL TREATMENT FOR SEPTIC SHOCK PATIENTS WITH COUPLED PLASMA FILTRATION ADSORPTION (CPFA)

Marco Pozzato[1], Fiorenza Ferrari[2], Pasqualina Cecere[1], Paola Mesiano[1], Antonella Vallero[1], Sergio Livigni[2] and Francesco Quarello[1]



SAP137 Figure 1: Simplified protocol using calcium-containing replacement solution in the RCA-based CVVH



SAP137 Figure 2: Kaplan-Meier curve for hemofilter life

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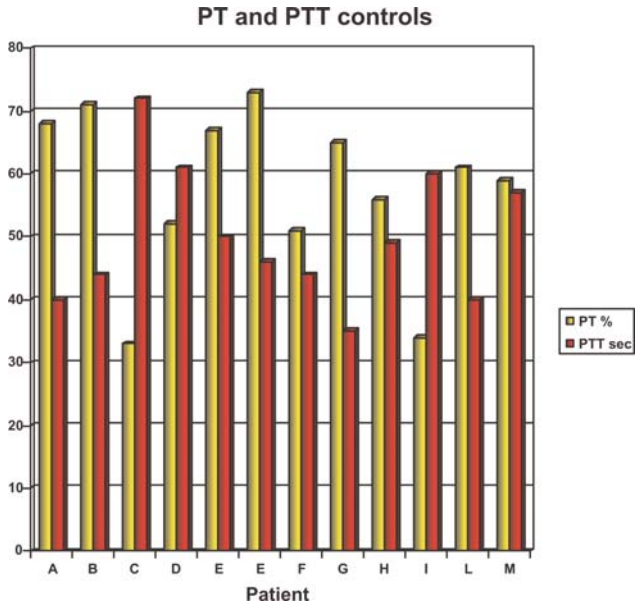
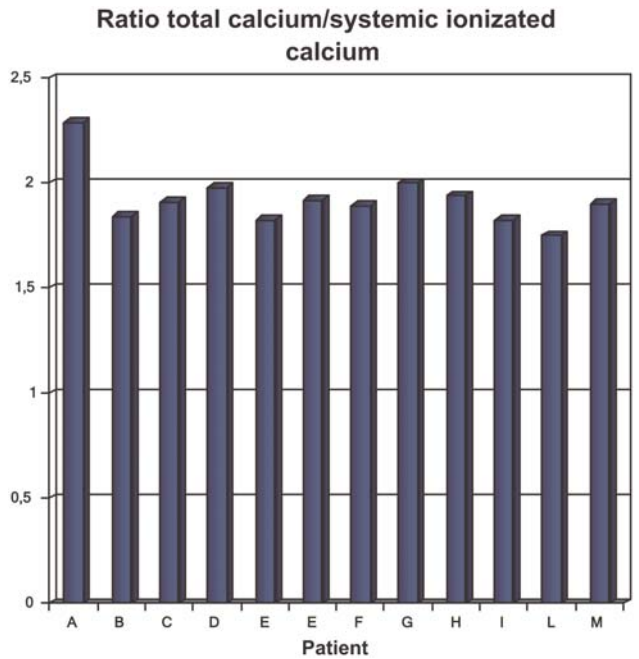
Introduction and Aims: From 2001 to 2011 94 patients in septic shock were treated by us with CPFA, an extracorporeal therapy that combines unselective plasma adsorption resins (MediaSorb) with continuous hemofiltration. CPFA proved to be an effective treatment of septic shock with or without acute kidney injury (AKI), improving hemodynamics, amine reduction and, potentially, survival. The aim of the study was to evaluate a citrate anticoagulation in CPFA to improve the treatment efficiency and simplifying its management. We evaluated the treatment duration, coagulative parameters, bicarbonate and ionized plasma calcium.
Methods: 11 consecutive mechanical ventilated patients (6 M, 5 F) with septic shock and multiorgan dysfunction (4/11 had AKI) were treated. Prescribed CPFA parameters were: Qb 150 ml/min, plasma flow rate (Qp) 30 ml/min, predilution solution (Na+ 136 mmol/l, citrate 10 mmol/l and citric acid 2 mmol/l) infused to keep inlet citratemia at 3 mmol/l, postdilution solution (Na+ 139 mmol/l, K+ 1.5, mmol/l, Ca++ 2 mmol/l, Mg++ 0.75 mmol/l, HCO3 - 35 mmol/l and glucose 5.55 mmol/l) and postdilution CaCl2 at a rate restoring the plasma Ca++ to 1.1 mmol/l (tab. 1) and adjusted according to the patients' need.
Results: 73 treatments were performed accounting for 733 hours, mean duration 8.35 ± 1.0 hours, mean plasma volume of 12.20 ± 2.8 L, Qb 143 ± 12 ml/min, Qp 24.6 ± 3.83 ml/min, a treated plasma dose/kg body weight of 1 ± 0.37 l/kg. Mean CaCl2 10% infusion of 4.5 ± 1.3 ml/h, with a citratemia, evaluated as total Ca++/iCa++ ratio (Fig. 1), always < 2.5 (mean 1.92 ± 0.14), also in 4 patients with liver dysfunction (mean 2 ± 0.20). Bicarbonates HCO3 - was 25.3 ± 4.8 mmol/l, pH 7.42 ± 0.05, ATIII (median 61%), PTT (50 ± 10.7 sec) (Fig. 2) and Ca++ 1.1 ± 0.1 mmol/l.
Conclusions: Our protocol allowed a high plasma dose, with a safe coagulative status, acid-base balance and calcemia correction. Survival rates at 28 and 90 days were 72.7% and 63.6%, respectively (Fig. 3). Citrate CPFA seems a viable and safe treatment for the septic shock patients.

SAP138 Table 1 Protocol scheme citrate-calcium chloride infusion

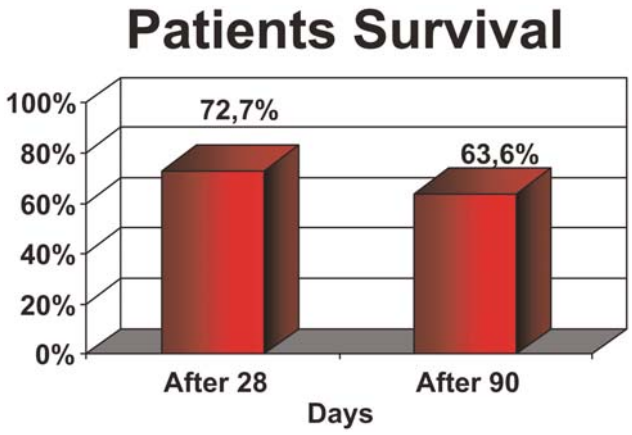
Kg	50	55	60	65	70	75	80	85	90	95	100
PRE ml/h	2250	2250	2250	2250	2250	2250	2250	2250	2250	2250	2250
POST ml/h	100	150	200	250	300	350	400	450	500	550	600
CaCl 10% ml/h	4	4	4	3	3	3	3	3	2	2	2



SAP138 Fig. 1: Average PT and PTT for each patient: lack of significant changes in buoyancy of the coagulation



SAP138 Fig. 2: Average ca^{++}/ica^{++} for each patient to verify the non-toxicity of the citrate at the end of the CPFA cycle



SAP138 Fig. 3: Elevated survival at 28 and 90 days related to clinical status of patients

SAP139 IMPACT OF INTENSIVE DAILY HAEODIALYSIS ON RENAL RECOVERY OF CAST NEPHROPATHIES: A RETROSPECTIVE STUDY

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Introduction and Aims: The main cause of renal failure in multiple myeloma (MM) is cast nephropathy (CN), which is directly due to free light chain (FLC) precipitation. Treatment is based on Bortezomib-Dexamethasone (VD) chemotherapy in association to supportive care. Previous data show some interests for FLC removal (Hutchinson 2009; Leung 2008). PolyMethylMethAcrylate (PMMA) membranes with adsorption properties (BKF, Toray®) have shown their ability to remove FLC in single dialysis sessions and have been used empirically by nephrologists in daily haemodialysis (HD) schedules. The aim of this study was to compare the impact of intensive daily dialysis on PMMA membranes to standard dialysis, on recovery rate of acute renal failure (ARF) due to CN.
Methods: We retrospectively included from 8 french hospitals MM patients who presented between 2007 and 2011, dialysis-dependent ARF most likely due to CN and who have received VD based chemotherapy. They were classified into two groups: intensive or standard, according to the initial dialysis schedule. The main outcome was dialysis-independence at 3 months.
Results: Fourty one patients were included: 21 patients in the "intensive" dialysis schedule (11,3 sessions on PMMA membranes over 13,2 days, and a mean session duration of 4,5 hours) and 20 patients in the "standard" dialysis schedule (mostly 3 times a week, adjusted to individual needs, on various membranes). Both groups were similar in terms of MM characteristics and complications but the patients from the standard group were older (median age of 77 vs 68 years, p = 0,0179) and had a higher median initial serum creatinin (sCr) (723 vs 477 µmol/l, p = 0,05). At 3 months, 15 patients were able to stop dialysis: 8 (38,1%) in the intensive group and 7 (35%) in the standard group (p = 1). Haematological response (HR) (defined by a 50% reduction of M-protein) was observed in 15 patients (71,4%) from the intensive group and 10 (50%) from the standard group (p = 0,2082). All but one patients (14) who were able to stop dialysis had a HR while only 11 (50%) of the 22 still dialysis-dependent patients had HR (p = 0,0053).
Conclusions: This study do not show any benefit on renal recovery of an intensive daily dialysis schedule using PMMA membrane compared to usual HD in context of CN, even considering the differences (age and sCr) between groups. However, the probability to become dialysis-independent seems more likely to be linked to the response to chemotherapy. The limitations are the limited number of patients, the retrospective method and the lack of histological confirmation of CN (only 13 patients had renal biopsy). The place of FLC removal in the treatment of ARF due CN is not well established. If our study fails to demonstrate a clinical benefit of daily dialysis on PMMA membranes, other strategies are actually under investigations. Preliminary encouraging results have recently been published using high-efficiency on-line hemodiafiltration (Granger-Vallée 2011) and we are looking for results of prospective trials on High cut-off HD.

SAP140 URINE OUTPUT AS A FACTOR IN INITIATING CONTINUOUS RENAL REPLACEMENT THERAPY FOR ACUTE KIDNEY INJURY

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Introduction and Aims: Although some studies have found that early initiation of continuous renal replacement therapy (CRRT) is associated with better prognosis, no consensus exists on the best timing to start CRRT to improve patient outcome. We investigated whether the timing of CRRT initiation was relevant to overall mortality and explored which factors at the time of CRRT initiation were associated with better outcomes in critically ill acute kidney injury (AKI) patients.
Methods: A total of 361 patients, who received CRRT for AKI between 2009 and 2011, were collected, and divided into two groups based on median BUN values and 2-hour urine immediately before CRRT start. The impact of the timing of CRRT initiation on 28-day all-cause mortality was compared between the groups and determination of early CRRT initiation as an independent risk factor was by the Cox proportional hazards model.
Results: When the timing of CRRT initiation was stratified by 2-hour urine output, crude 28-day mortality rates were significantly lower in the early CRRT group than the late CRRT group. In contrast, clinical outcomes were not different between early and late CRRT groups classified by BUN levels. Cox regression analysis revealed that the crude 28-day mortality risk was significantly lower in the early CRRT group stratified by 2-hour urine output, even after adjusting for age, gender, mean arterial pressure, serum biomarkers, and APACHE II and SOFA scores.
Conclusions: Urine output might be more useful than BUN concentration in making decisions about the timing of CRRT initiation in critically ill AKI patients.

SAP141 ECONOMIC IMPACT OF TWO MODALITIES OF CVVHDF ON NURSES WORKING LOAD

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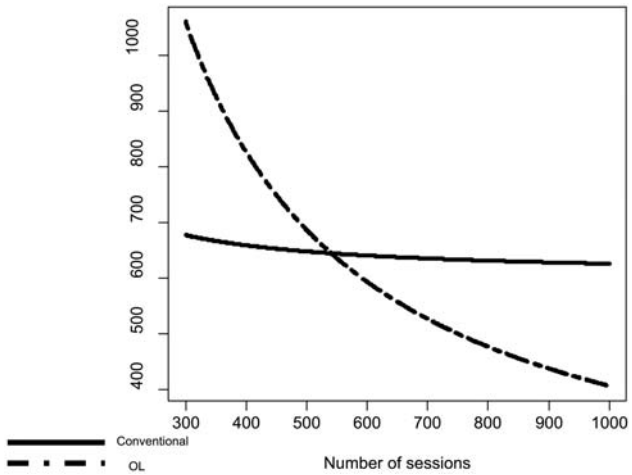
Introduction and Aims: Continuous veno-venous hemodiafiltration (CVVHDF) is a supportive therapy of choice for the critically ill patients with acute kidney injury (AKI). Implementation of this technique in the intensive care units (ICU) remains however, limited by a high cost, inherent to its continuous nature. Two modalities of CVVHDF exist in terms of dialysate/filtrate supply: 1-the conventional CVVHDF, which uses pre-conditioned dialysate/filtrate bags. For this technique, repetitive manual handling by the nurse is required to replace the bags and to provide the appropriate fluid volume throughout the session; 2- the on line (OL)-CVVDHF for which dialysate/filtrate are generated directly from a water loop. While no additional nursing work is specifically required, the installation in the ICU of a water circuit dedicated to this technique is mandatory. We aimed to compare the cost-effectiveness of these 2 techniques of CVVHDF.
Methods: From march to September 2011, every patient admitted in our renal ICU with the diagnosis of AKI and requiring continuous renal replacement therapy were eligible to participate to this prospective, monocentric, cross-over, randomized study. Patients were randomly assigned to 2 consecutive 12 hours-sequences of conventional (HOSPAL Prismaflex® with AN69 ST filter) and on-line (Fresenius® 5008 monitor with Xenium® 2.1 filter) CVVHDF. The different flow rates were kept constant between each sequence and set up at 200 ml/min, 6000 ml/hour, and 2000 ml/hour for blood, dialysate and filtrate flow, respectively. Number and duration of nurse's interventions specifically related to the management of the CVVHDF was monitored for each sequence. Economic evaluation included direct and indirect costs related to: - the dialysis machine and its maintenance, - the consumables used for each sequence, - the nurse workload, and – the water treatment unit installation and its maintenance for the OL sequence. Dialysis dose was evaluated by the urea reduction ratio (RR), the urea kT/V, the phosphorus RR and β2-microglobulin RR. A wilcoxon was used to compare the cost of each sequence.
Results: 15 patients (12 men) were randomized with a median (range) age of 71 (35-88) years and a median IGS II of 55(33-99). No difference in terms of dialysis dose between the 2 CVVHDF modalities was observed. Total duration of nurse intervention was significantly longer for the conventional CVVHDF with a mean (±SD) time of 27 (±26.7) as compared to 83 (±32.8) minutes for the OL modality (p=0.00013). This, along with consumable-related cost, translates into a mean over cost of 450 euros per session of conventional CVVHDF largely compensated by the cost related to the water circuit necessary for the OL-modality. By modelling the cost of each modality according to the number of sessions performed, we observed that the respective costs of the 2 techniques of CVVHDF were equilibrated after 550 sessions (figure).
Conclusions: As compared to conventional CVVHDF, the OL modality is less



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time-consuming and may help reducing nursing workload. The over cost induced by the installation and the maintenance of a specific water treatment unit has to be considered but will be rapidly compensated in most ICUs.

SAP142 OUTCOME AND PROGNOSTIC FACTORS OF MALARIA-ASSOCIATED ACUTE KIDNEY INJURY

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Introduction and Aims: Acute kidney injury (AKI) is one of the most dreaded complications of severe malaria.
Methods: We carried out prospective study in 2010, to describe clinical characteristics, laboratory parameters, prognostic factors, and outcome in 59 (44 males, 15 females) smear-positive malaria patients with AKI. The severity of illness was assessed using Acute Physiology and Chronic Health Evaluation (APACHE) II, Sequential Organ Failure Assessment (SOFA) score, Multiple Organ Dysfunction Score (MODS), and Glasgow Coma Scale (GCS) scores. All patients received artesunate and hemodialysis (HD).
Results: Mean age of patients was 33.63 ± 14 years.Plasmodium falciparum malaria was seen in 76.3% (n = 45), Plasmodium vivax in 16.9% (n = 10), and mixed infection in 6.8% (n= 4) patients. Presenting clinical features were fever (100%), nausea-vomiting (85%), oliguria (61%), abdominal pain/tenderness (50.8%), and jaundice (74.5%). Mean APACHE II, SOFA, MODS, and GCS scores were 18.1 ± 3, 10.16 ± 3.09, 9.71 ± 2.69, and 14.15 ± 1.67, respectively, all were higher among patients who died than among those who survived. APACHE II =20, SOFA and MODS scores =12 were associated with higher mortality ($P < 0.05$). 34% patients received blood component transfusion and exchange transfusion was done in 15%. Mean number of HD sessions required was 4.59 ± 3.03. Renal biopsies were performed in five patients (three with patchy cortical necrosis and two with acute tubular necrosis). 81.3% of patients had complete renal recovery and 11.8% succumbed to malaria.
Conclusions: Prompt diagnosis, timely HD, and supportive therapy were associated with improved survival and recovery of kidney functions in malarial with AKI. Mortality was associated with higher APACHE II, SOFA, MODS, GCS scores, requirement of inotrope, and ventilator support.

SAP143 MICROCIRCULATION CORRELATES WITH RIFLE SCORE ON ADMISSION AND PRIOR TO CRRT INITIATION

Chrysoula Pipili[1], Chrysoula Pipili[2], Christos Manetos[3], Charikleia S. Vrettou[3], Stefania Poulaki[4], Elli-Sophia Tripodaki[4], Adroula Papastylianou[4], Christina Routsi[4] and Serafeim Nanas[3]
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Introduction and Aims: Near Infrared Spectroscopy (NIRS) combined with vascular occlusion technique (VOT) allows evaluation of peripheral tissue oxygen utilization and restoration mainly depending on integrity and functionality of vascular endothelium. Our purpose was to evaluate if microcirculation (MC), as assessed by NIRS and VOT, before initiation of Continuous Renal Replacement Therapy (CRRT) correlates with RIFLE score for acute renal injury.
Methods: Fifty seven (36 males, mean age 64.5 ± 1.8 years, APACHE II score: 19 ±6.9, SOFA: 9.3±2.5, SAPS 65 ± 13) consecutive critically ill patients who underwent CRRT were eligible to participate in the study. RIFLE on admission were R 30% (17), I 23% (13) and F 11% (6), while RIFLE at time of CRRT initiation were R 25% (14), I 44% (25) and F 2% (1). Tissue oxygen saturation (StO2%) defined as the percentage of hemoglobin saturation in the microvasculature compartments was measured on the thenar muscle by NIRS before, during and after 3-minute occlusion of the brachial artery. Measurements included StO2%, oxygen consumption rate (OCR %/min) as the first degree slope of the desaturation of haemoglobin. StO2% on admission was 76.5±12.7% and OCR was -11±6.8 %/min respectively.
Results: StO2 correlated with RIFLE on admission and at the time of CRRT initiation ($r=0.283$, $p=0.03$ and $r=0.45$, $p<0.0001$ respectively) while there was a trend for OCR to correlate with RIFLE on admission ($r=0.263$, $p=0.048$).
Conclusions: Microcirculatory parameters before CRRT initiation, as assessed by NIRS and VOT, might provide helpful information about the disease severity and the degree of kidney injury in the critically ill patient. Further research in this field is promising.

SAP144 DOES ACUTE KIDNEY INJURY LEADS TO OR WORSEN CHRONIC KIDNEY DISEASE? RISK FACTORS FOR CHRONIC KIDNEY DISEASE AFTER ACUTE KIDNEY INJURY

Kensuke Uchida[1], Uchida Kensuke[2], Kunihiro Yamagata[3], Chie Saitou[4] and Masashi Okada[4]
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Introduction and Aims: Acute kidney injury (AKI) has been regarded as a risk factor in chronic kidney disease (CKD). As patients with CKD show a higher incidence of AKI, we elucidated the effect of AKI on the progression of CKD.
Methods: This retrospective study was performed in patients hospitalized between the 1st January and 31st December of 2008 in our hospital. Only those fulfilling the AKIN category (i.e. registering the standard serum creatinine (Cre) content) were defined as having AKI. Outcomes and changes of their renal function after AKI onset were monitored for a 1-year period. The age, gender, underlying disease, AKI cause, AKI stage, therapeutic drugs used, and the CKD stage during the observation period were studied. Incidence of new CKD after AKI onset or the progress of pre-existing CKD stage was evaluated as well. Of a total of 12,758 patients enrolled in the study, 277 (2.1% of total hospitalized) patients had AKI. Of these AKI patients, 61 (22%) patients had pre-existing CKD: a higher prevalence rate than the 10.4% of the general Japanese population with CKD. During the observation period, 111 patients died. The remaining patients were divided into cases with and without CKD before AKI onset, and the risk factors related to development of new CKD onsets and progress of CKD were analysed.
Results: Risk factors were calculated using simple linear regression analysis. Pre-existing hypertension (relative risk [RR]: 1.32; 95% confidence interval [CI]: 1.06-1.64), pre-existing urinary tract disease (RR 1.39, 95% CI: 1.13-1.71), and AKI due to glomerular disease (RR: 1.41; 95% CI: 1.22-1.61) were indicated as significant risk factors affecting the incidence of CKD after AKI. Among the subjects with CKD superimposed AKI, patients complicated with malignant neoplasm showed a significant risk in the progression of CKD stage (RR: 3.36; 95% CI: 1.66- 6.83), while CKD due to glomerular disease showed less progression in CKD stage (RR: 0.26; 95% CI: 0.03-0.40). No significant risk factors were derived between age, gender and AKI-stage in all analyses.
Conclusions: The present study demonstrated that proper follow-up of kidney function and thorough management of the blood pressure after AKI onset may prevent subsequent development of CKD. Furthermore, recovery may be promptly established in cases showing acute exacerbation in chronic glomerular disease with early intervention by nephrologists.

SAP145 THE THROMBOTIC MICROANGIOPATHIES IN THE ERA OF HIV - AN AFRICAN PERSPECTIVE

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Introduction and Aims: The thrombotic microangiopathies (TMA) are a group of disorders of heterogenous aetiology but shared histopathology which clinically present as TTP or HUS. The prevalence of HIV infection in TMA varies between 0 – 83% and is associated with advanced infection. TMA in HIV infection is reported to have a more severe presentation and poorer prognosis. D-dimer levels have been reported to be higher in HIV-associated TMA. South Africa has a high prevalence of HIV infection; however, little is known regarding the presentation and outcomes of TMA in our setting. This study was undertaken:
1. To evaluate the role of HIV infection in the aetiology of TMA in the South

African population
2. To characterize the presentation of HIV-associated TMA in this context
3. To determine whether HIV infection affects severity of presentation, response to treatment, or outcomes of TMA.
Methods: A retrospective review was undertaken of all patients diagnosed with TMA at the Helen Joseph Hospital between 1/1/2001 – 1/12/2009. The clinical parameters at presentation of oral temperature, haemoglobin, lactate dehydrogenase (LDH) activity, platelet count, creatinine, and d-dimer levels were compared between HIV positive (HIV+) and negative (HIV-) patients as was response to plasma exchange, mortality outcomes and recurrences. Presenting clinical parameters were compared in survivor and mortality subgroups. The Mann-Whitney U test and Fisher Exact test were used to compare continuous and categorical variables respectively and the Spearman Rank Order method was used to evaluate correlation. A $p < 0.05$ was considered significant.
Results: 40 patients with 47 separately diagnosed episodes of TMA were included. HIV-associated TMA, systemic lupus erythematosus (SLE), and idiopathic TMA accounted for 81%, 15%, and 2% of cases respectively. Presenting creatinine and D-dimer levels were higher in HIV+ patients (127 mmol/l vs. 92 mmol/l, $p = 0.022$ and 3.53mg/dl vs. 1.10mg/dl, $p = 0.024$ respectively). HIV+ patients showed more rapid response to plasma exchange than HIV- patients (11 exchanges vs. 20, $p = 0.015$). CD4 count correlated to plasma exchange response in HIV+ patients ($R = -0.41$, $p = 0.018$). Despite a higher creatinine at presentation in HIV+ vs. HIV- patients, creatinine following successful therapy was similar between the 2 groups (79 mmol/l vs. 82mmol/l, $p = 0.763$) and HIV+ patients showed a larger improvement in creatinine (37mmol/l vs. 5mmol/l $p = 0.057$). Mortalities were non-significantly higher in HIV+ patients (9 vs. 0, $p = 0.317$). In such patients, mortality was associated with a higher presenting creatinine (298mmol/l vs. 117mmol/l, $p = 0.0004$), and possibly a lower CD4 count (133x106/l vs. 42x106/l, $p = 0.0501$). Recurrences were more frequent in HIV- patients (50% vs. 11.54%, $p = 0.012$).
Conclusions: HIV infection is the dominant cause of TMA in our population. HIV-associated TMA occurs in the context of advanced infection and has a more severe clinical presentation (particularly regarding renal dysfunction). Mortality and renal function outcomes following therapy are similar between HIV+ and HIV- patients. Severe renal dysfunction at presentation may influence mortality in HIV-associated TMA, whilst severity of HIV infection (as indicated by CD4 count) may influence response times to plasma exchange and mortality outcomes.

SAP146 SURROGATE PREDICTORS OF SNAKEBITE MEDIATED ACUTE KIDNEY INJURY AND CARBONYL STRESS – A PROSPECTIVE STUDY

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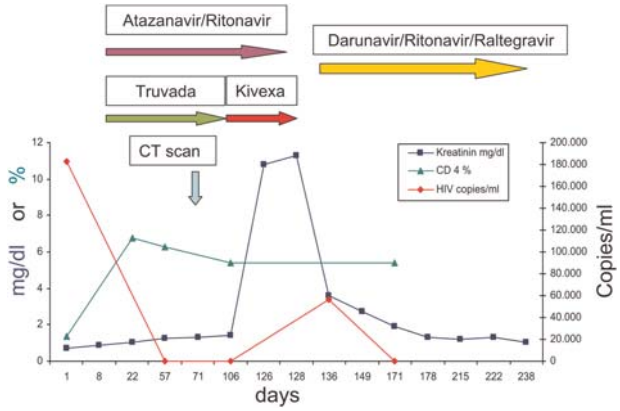
Introduction and Aims: Venomous snakebites are an important medical problem and occupational hazard in Southeast Asia with high morbidity and mortality. 1) To determine the prognostic predictors of snakebite induced acute kidney injury (SAKI) required renal replacement therapy. 2) To measure the oxidative and carbonyl stress level in SAKI patient and correlate with surrogate outcome.
Methods: All SAKI patients admitted from April2010 to July2011 and received hemodialysis were included. Demographical, clinical and biochemical data were analyzed and they are followed from hospitalization to discharge or death. Oxidative and carbonyl stress markers [Advanced oxidation protein product (AOPP), Advanced Glycation End product (AGE), Pentosidine, Dityrosine, Thioberbituric acid reactive substance (TBARS) and Methyl glyoxal (MG)] were measured consecutively in 48 SAKI patient according to standard protocol. All data were analyzed with appropriate statistical methods.
Results: Among 155 SAKI patients received hemodialys Male: Female was2.2:1. The mean age was 36.2 years (4-74 years). Commonest site of bite was lower limb (88.7%).About 74.2% received primary treatment. Oliguria and bleeding manifestation were the common presentation. Hypotension was found in 52(33.5%) cases, cellulites and inflammation was found in about 63% patients. About 42 (27.1 %) had disseminated intravascular coagulation (DIC). Anti snake venom (ASV) was used 21.5±1.94 vial. Median HD was required 3 session .Mean hospital stay was 11 days (2-34 days). Bite to HD initiation time was 3.2 days. Out of 46 (29.7%) patients died 10 (34.48%) were less than 18 years. About 36(78.2%) had cellulites, 24(52.2%) had shock/hypotension at initial presentation ($p<0.05$), bleeding manifestation was found in 37(80.4%) and 22(47.8%) had DIC ($p<0.05$).DIC and shock/hypotension at initial presentation was came out as independent predictor of death at multivariate level. Among the biochemical markers measured for oxidative and carbonyl stress (n=48) AOPP and MG came out as independent predictor ($p<0.05$) of adverse outcom
Conclusions: DIC, shock/hypotension, advanced oxidation protein product (AOPP) and methyl glyoxal (MG) was surrogate prognostic marker in SAKI patients requiring dialysis leading to death.

SAP147 ACUTE TUBULOINTERSTITIAL NEPHRITIS CAUSED BY ABACAVIR AND LAMIVUDINE (KIVEXA ®)



SAP147

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Introduction and Aims: Highly active antiretroviral therapy (HAART) is associated with nephrotoxicity. Pathomechanisms include acute interstitial nephritis, crystal nephropathy, acute tubular necrosis, and renal tubular disorders. We report a 44 year old female HIV positive patient developing acute renal failure most likely caused by abacavir and lamivudine (Kivexa ®). Kivexa is not considered to be nephrotoxic but may cause hypersensitivity reactions in patients testing positive for the presence of the HLA-B*5701.
Methods: Initially the patient received atazanavir and ritonavir in combination with emtricitabin and tenofovir (Truvada®). After a slight increase of creatinine and exclusion of HLA-B-5701-positivity she was switched from Truvada to Kivexa. Twenty days later she presented to the outpatient clinic with edema, shortness of breath, vomiting and anorexia. The patient reported that symptoms had started about ten days ago. Laboratory values revealed a dramatic rise in creatinine with a GFR of 4 ml/Min/1.73 m^2.
Results: The patient was started on a hemodialysis regimen after implantation of a tunnelled catheter. Kidney biopsy revealed tubulointerstitial nephritis with moderate tubulitis and a cell infiltrate consisting of eosinophil granulocytes and plasma cells. After termination of antiretroviral therapy renal function recovered completely. Later, the patient received a Kivexa-free regimen consisting of Darunavir, Ritonavir and Raltegravir; renal function remained stable.
Conclusions: Our patient developed drug-induced, tubulointerstitial nephritis most likely caused by Kivexa. The underlying pathomechanism seemed to be independent of HLA-B*5701. Renal function recovered completely after discontinuation of Kivexa.

SAP148 PATHOGENIC MECHANISMS INFLUENCING THE RECOVERY OF RENAL FUNCTION AFTER MACROHEMATURIA-INDUCED ACUTE KIDNEY INJURY (AKI) IN IGA NEPHROPATHY

Eduardo Gutierrez*[1], Jesús Egido[2], Alfonso Rubio-Navarro[2], Irene Buendía[2], Luis Miguel Blanco-Colío[3], Oscar Toldos[1], Félix Manzarbeitia[4], Alberto De Lorenzo[1], Rosa Sánchez[5], Manuel Praga^[1] and Juan Antonio Moreno^[6]
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Introduction and Aims: Macroscopic hematuria (MH) may cause AKI in IgA nephropathy. Up to 25% of patients with MH-associated AKI do not recover baseline renal function.
Methods: In order to identify subjects at high risk for an adverse renal function outcome, we immunohistochemically examined macrophages, oxidative stress markers (NADPH-p22 and HO-1) and the hemoglobin scavenger receptor (CD163) in renal biopsy specimens from 33 MH-AKI patients with complete (CR, n=17) or incomplete (IR, n=16) recovery of renal function six month after cessation of MH. We then evaluated the correlation between the expression of these markers and clinical parameters of renal function and histological findings from renal biopsies.
Results: IR patients were significantly older and had a higher frequency of a previous MH episode or need for hemodialysis than CR patients. IR patients also showed higher initial, peak and final serum creatinine, highest final proteinuria, longer duration of MH and lower eGFR, as compared with CR patients. The percentage of

tubules with both red blood cells (RBC) casts and with tubular necrosis was significantly higher in the IR group. No significant differences in the rest of histological parameters were observed between patients classified according to recovery of renal function. CD163-expressing macrophages and heme oxygenase-1 (HO-1) and NADPH-p22 expression were located in areas surrounding tubules with iron deposits and filled with erythrocyte casts. CD163-positive macrophages score (r=0,48, p 0,014 and r=0,54, p 0,003) and HO-1 (r=0,43, p 0,029 and r=0,49, p 0,01) and p22-positive staining (r=0,42, p 0,03 and r=0,68, p 0,001) correlated positively with percentage of tubules with erythrocyte casts and tubular necrosis, respectively. Macrophage infiltration, CD163-positive macrophage score, NADPH-p22 and HO-1 positive areas were significantly greater in IR-patients. CD163-positive macrophages score (r=-0,72, p 0,001) and oxidative stress markers (p22, r=-0,61, p 0,001; HO-1, r=-0,59, p 0,001) were negatively correlated with renal function outcome assessed as final eGFR. In multivariate analysis CD163 macrophage score, but not conventional histological parameters, was a significant independent predictor of final eGFR and proteinuria.
Conclusions: In conclusion, patients with IR of renal function show a high proportion of tubules with iron deposits and filled by RBC casts which in turn are associated with evidence of higher oxidative stress via p22-NADPH that may aggravate acute tubular necrosis. These patients also exhibit an enhanced compensatory protective response characterized by increased CD163 and HO-1 expression. These molecular pathways may be involved in the renal response to injury and could be useful to improve diagnosis and therapeutics.

SAP149 HIGHER INFUSION RATE OF MANNITOL INCREASES THE RISK OF ACUTE KIDNEY INJURY IN PATIENTS WITH INTRACRANIAL HEMORRHAGE

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Introduction and Aims: Mannitol, an osmotic agent used to decrease intracranial pressure, has been reported to increase the risk of acute kidney injury (AKI). However, the risk factors of AKI in patients receiving mannitol are yet to be determined. The objective of this study was to assess the impact of mannitol on the incidence and severity of acute kidney injury (AKI), and identify risk factors for AKI in patients with intracranial hemorrhage (ICH).
Methods: A total of 537 adult patients who received mannitol infusion after ICH between January 2005 and December 2009 in the neurosurgical intensive care unit were analyzed retrospectively. After excluding 384 patients due to no measurements of serum creatinine before, during, and after mannitol therapy (n = 179), administration interval of mannitol of more than two days (n = 93),mannitol therapy started at outside hospitals (n = 63), underlying malignancies (n = 32), AKI (an increase of serum creatinine 0.3mg/dL from baseline) on admission (n = 14), and renal failure requiring dialysis (n = 3), 153 patients were included in the study. Multivariate analysis was used to evaluate the risk factors for AKI after ICH. Based on the odds ratio, weighted scores were assigned to predictors of AKI.
Results: The overall incidence of AKI was 10.5% (n=16). AKI was more frequent in patients who received mannitol infusion at a rate 1.34 g/kg/24hr compared to patients who received mannitol infusion at a rate 1.34 g/kg/24hr (75.0% versus 33.6%; P = 0.003). Higher mannitol infusion rate was also associated with more severe AKI.
Risk factors for AKI in patients treated with mannitol for ICH (obtained by multivariate analysis)
Notes: OR, odds ratio; CI, confidence interval; DBP, diastolic blood pressure; GFR, glomerular filtration rate; ACEI/ARB, angiotensin converting enzyme inhibitor/ angiotensin receptor blocker
Using the risk model for AKI, patients with a higher score showed a graded association with a higher incidence of AKI. In the risk model, the area under the receiver operating characteristics curve for AKI was 0.917
Conclusions: The incidence of AKI following mannitol infusion in patients with ICH was 10.5%. Higher infusion rate of mannitol was associated with more frequent and more severe AKI. Additionally, age over 70, DBP above 110 mmHg, and established renal dysfunction before starting mannitol were associated with development of AKI.

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Covariate	OR	95% CI	p-value
Rate of mannitol infusion 1.34 g/kg/24hr	67.627	2.903-1575.310	0.009
Age 70 yr	14.652	1.397 153.634	0.025
DBP 110 mmHg	53.307	2.812 1010.620	0.008
Diabetes mellitus	2.278	0.154 33.621	0.549
Ischemic heart disease	13.041	0.278 610.822	0.191
GFR 60 mL/min/1.73m²	129.786	3.864 4359.858	0.007
ACEI/ARB	4.884	0.363 65.628	0.232
Inotropics	6.093	0.650 57.086	0.113
Furosemide	4.562	0.386 53.962	0.229
Peak serum osmolality 320 mOSM/kg	2.728	0.251-29.697	0.410

SAP150 GADOLINIUM-CONTRAST NEPHROTOXICITY IN MAGNETIC RESONANCE IMAGING

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Introduction and Aims: Gadolinium (Gd)-contrast medium administration for magnetic resonance imaging (MRI) has been considered safe, although a 3 to 5% occurrence of Gd-contrast medium nephrotoxicity has been reported for the period between 1999 and 2011. In this study, we investigated the effect of Gd-contrast medium on renal function during MRI.
Methods: Patients aged 20 to 80 years, weighing 45 to 70 kg and with normal or less than 1.6 mg/dl of serum creatinine (S-Cr) in the 3 months prior to receiving an MRI were enrolled. They were randomly divided into an ionic contrast medium (Omniscan) administration group (group O) and a nonionic contrast medium (Magnevist) administration group (group M). Gd-contrast medium (0.2 ml/kg) was administered to all patients. S-Cr, serum cystatin C (S-Cys), eGFR using MDRD and eCCr using the Cockcroft-Gault formula were measured just before and 24–72 hrs after the MRI. The Mann-Whitney U-test and Wilcoxon signed-ranks test were employed for statistical analysis.
Results: There were no significant differences in the clinical background such as age, sex, serum concentrations of albumin, S-Cr, S-Cys, eGFR and eCCr between group O (n=43) and group M (n=59). There were no significant differences in S-Cr, eGFR and eCCr between pre-MRI and 24–72 hrs post-MRI measurements of both groups. S-Cys increased significantly 24–72 hours after MRI only in group O.
Conclusions: A nonionic contrast medium (Magnevist) had no effect on renal function during MRI, while an ionic contrast medium (Omniscan) affected renal function transiently.

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Serum component	Omniscan (group O)			Magnevist (group M)		
	before	after	p value	before	after	p value
S-Cr (mg/dl)	0.69±0.14	0.71±0.13	0.09	0.69±0.18	0.71±0.20	0.26
S-Cys (mg/l)	0.74±0.14	0.79±0.21	0.03	0.77±0.19	0.77±0.21	0.21
eGFR (ml/min/1.73m²)	90±20	87±20	0.09	95±26	93±28	0.48
eCCr (ml/min)	88±26	86±26	0.11	95±27	93±28	0.33

SAP151 EARLY INITIATION OF CONTINUOUS RENAL REPLACEMENT THERAPY IN CRITICALLY ILL PATIENTS WITH ACUTE KIDNEY INJURY IS ASSOCIATED WITH A BETTER RENAL RECOVERY - A PROSPECTIVE OBSERVATIONAL TRIAL

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Introduction and Aims: Although acute kidney injury (AKI) is a frequently observed complication in intensive care units (ICU) and has been defined by AKIN-criteria, the optimal time point at which to initiate renal replacement therapy (RRT) in AKI is widely debated. The long-term impact of AKI on mortality and morbidity is well described. The aim of this study was to assess influence of RRT initiation timing on long-term renal recovery.
Methods: Observational trial of 9,343 ICU admissions to our tertiary university hospital revealed 583 cases of AKI, receiving overall 6,789 dialysis treatments. Patients requiring RRT before admission were excluded. Prospective observation post-discharge of the 222 surviving AKI patients was performed for a median follow-up period of 850 days (105-1673 days). Complete and partial recovery of renal function as well as further adherence to RRT were assessed at discharge from hospital and in the follow-up period.
Results: Of 583 patients with RRT requiring AKI, 361 patients (61.9 %) died during hospitalization. In 65.8 % of the survived patients, renal function recovered completely. In 17.9 % of these patients, only a partial recovery of renal function was observed and 15.8 % patients required RRT after discharge. Multinominal logistic regression analysis identified the increase of creatinine (DCrea) between hospital admission and start of RRT as independent predictor for a further need of RRT at

discharge (p=0.009) as well as during follow-up (p=0.002). Patients requiring permanent RRT after discharge had a significantly higher DCrea (3.2 ± 0.1 mg/dl) as compared to patients with partial (2.3 ± 0.2 mg/dl) or complete (1.7 ± 0.1 mg/dl) renal recovery after adjustment for all other variables using a general linear model. Consistently, DCrea was significantly higher in patients requiring RRT during follow-up (3.7 ± 0.1 mg/dl) as compared to those with complete or partial recovery of renal function (1.8 ± 0.1 mg/dl; 2.1 ± 0.2 mg/dl).
Conclusions: In critically ill patients with AKI, DCrea was indentified as useful prognostic marker for recovery of renal function during and after hospitalization. We therefore conclude that early initiation of RRT in critically ill patients with AKI may be associated with an improved long-term recovery of renal function.

SAP152 IN-HOSPITAL MORTALITY AND LONG-TERM OUTCOMES OF NON-DIALYSIS REQUIRING ACUTE KIDNEY INJURY IN CRITICALLY-ILL PATIENTS

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Introduction and Aims: The adverse consequences of non-dialysis requiring acute kidney injury (AKI) are not well- clarified. This study aimed to assess the in-hospital mortality and long-term outcomes in critically-ill patients experiencing non-dialysis requiring AKI.
Methods: A total of 922 AKI patients that did not receive dialysis during hospitalization in surgical intensive care units between January 2002 and June 2010 were enrolled. All of the eligible subjects were analyzed for in-hospital mortality; while only those who survived up until the hospital discharge from the index admission were analyzed for long-term outcomes, including stage 3-5 chronic kidney disease (CKD)-entries, end-stage renal disease (ESRD), as well as long-term mortality. All longitudinal serum creatinine measurements after discharge were collected and then calculated into eGFR via the MDRD equation.
Results: The in-hospital mortality rate was 31.2%. At the peak of AKI, mean arterial pressure (OR: 0.97 per mmHg; 95% CI: 1.12-2.68), body weight change (OR: 1.08 per 1% increase; 95% CI: 1.04-1.13), APACHE II score (OR: 1.14; 95% CI: 1.09-1.18), SOFA score (OR: 1.24; 95% CI: 1.15-1.32), and a maximum RIFLE stage of "failure" (OR: 1.73 compared to the "risk" and "injury" stages; 95% CI: 1.12-2.68) were independently associated with hospital death. Among 634 hospital survivors, the median intervals between the onset of AKI and the composite endpoints of "stage 3 CKD or death", "stage 4 CKD or death", "stage 5 CKD or death", and "ESRD or death" were 685, 1319, 1743, and 2048 days, respectively. Using the multivariate Cox's proportional hazards model, it was found that for every 1 mL/min/1.73m2 increase in baseline estimated glomerular filtration rate of individuals that progressed to stage 3-5 CKD during follow-up increased the risk of long-term mortality by 0.8%, 2.7%, and 4.3%, respectively (all p<0.01).
Conclusions: There was a high in-hospital mortality rate and long-term decline in kidney function among critically-ill patients with non-dialysis requiring AKI. Furthermore, there appeared to be an association between deterioration of kidney function and long-term mortality.

SAP153 ACUTE KIDNEY INJURY (AKI): AN ACCEPTABLE RISK OF TREATMENT WITH ACE INHIBITORS AND ANGIOTENSIN RECEPTOR BLOCKERS?

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Introduction and Aims: Results of large randomised controlled trials of the use of renin-angiotensin-aldosterone system (RAAS) blocking drugs in both renoprotection and the treatment of hypertension and heart failure have contributed to clinical guideline recommendations for their use. Importantly the inclusion of RAAS blockade in the clinical indicators of the UK primary care pay for performance Quality and Outcomes Framework (QOF) have led to the widespread use of RAAS blocking drugs in the general population. However, despite their widespread use, and caveats from clinical trials highlighted in the literature, little is known about the risk and risk benefit ratio of these agents in the general population. Our aim was to describe the use of RAAS blocking drugs in primary care, and firstly document the appropriateness of their use with respect to indication, and secondly to describe their effect on acute kidney injury (AKI).
Methods: Data were extracted from our system for early intervention in kidney disease (SEIK), which aids primary care with management of chronic kidney disease (CKD) through a system of data extraction and subsequent reporting. We included all adults who had a serum creatinine (SCr) and blood pressure recorded in the time period 2004-2006. We then split the follow up time into 3 time periods; 2004-2006, 2006-2008 and 2008-2010. The average eGFR was calculated for each time period. To identify AKI we used AKIN criteria and SCr baseline within 12 months [1]. We also extracted recorded co-morbidity, proteinuria testing, all prescription medicines, age and mortality data. Patients were stratified by CKD stage. Patients lost to follow up during the period 2004-2010 were removed from the analysis of AKI.
Results: There were 68,180 adults with an eGFR and blood pressure recorded in the time period 2004-2006. After exclusion of those lost to follow up, there were 20,396 on a RAAS blocking drug in the time period 2004-2006, of whom 16.3% had at least 1 episode of AKI between 2004-2010 (average of 0.44 AKI episodes per person). By comparison, of the 23,366 people who were not on an RAAS blocking drug at any point during the study period (2004-2010) 4.3% had at least 1 episode of AKI (average of 0.08 AKI episodes per person). This difference was observed across all age bands, stages of CKD (excluding stage 5), and co-morbidities. Importantly of the 20,396 people on a RAAS blocking drug, in 15,311 the only indication was simple hypertension (which did not comply with British Hypertension Society guidelines). 16.7% of these had at least 1 episode of AKI (average 0.44 AKI episodes per person), by comparison of 20,641 people who were appropriately not prescribed a RAAS blocking drug 4.5% had at least 1 episode of AKI (average 0.09 AKI episodes per person).
Conclusions: This study highlights the fact that a large number of people in primary care are treated with RAAS blockade, a large proportion of which have no evidence-based indication. These people have an increased risk of AKI. These data dictate the need for further analysis of the risk benefit of RAAS blockade. The agents should only be prescribed where there is a clear evidence-based indication.
1. Lafrance JP, Miller DR. Defining acute kidney injury in database studies: the effects of varying the baseline kidney function assessment period and considering CKD status. Am J Kidney Dis. 2010 Oct;56(4):651-60.

SAP154 CHANGES IN THE INCIDENCE OF ACUTE KIDNEY INJURY REQUIRING DIALYSIS (AKI-D): A FIVE YEARS SINGLE-CENTER REGISTER

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Introduction and Aims: The incidence of AKI-D is dramatically increased in the last years although national registers of the disease are not available. The consequences of AKI-D consist on high mortality, progression to ESRD, economical/social costs. Aim of this study is the evaluation of changes in the incidence and characteristics of AKI-D during five years observation performed in a university-hospital which serves 340.000 inhabitants.
Methods: Cohort study prospectively performed on 520 inpatients from 2007 to 2011 who developed AKI-D. AKI was defined in accord to AKIN classification.
Results: The incidence of AKI-D increased yearly, from 217,65 to 441,20 pmp in 2011. The five years increase was 103%. Gender (M = 66.9%, F = 33.1%), age (70.3 ±13.7; 71.4±14.8), and presence of diabetes did not change, but the co-morbid presence of ischemic heart disease increased from 34%(2007) to 66%(2011). CKD was present in 52.7%. Critically-ill patients, admitted in Intensive Care Units (ICU), increased from 20 to 26%, but significant changes of in-hospital admittance were recorded for non-ICU pts. Those admitted to Nephrology dept decreased from 38 to 25% whereas those who referred to non-nephrology dept. increased from 41 to 52%. AKIN class at the start of dialysis was grade 3 in 81.9%, grade 2 in 13.7%, and grade 1 in 4.4%. The main cause of AKI was pre-renal (59.8%). Heart failure was responsible of 13.9% of AKI. A toxic cause was recognized in 14.3%. Glomerulonephrities/vasculities represent only a marginal cause of AKI, 3.3%. Causes of AKI did not differ during these years. In-hospital mortality, 47.5%, did not change during the observation. Patients who survived without the need of dialysis were 40.8%, but those who were dialysis dependent at hospital discharge were 11.7%. Adjusted mortality was higher in non-critically ill patients admitted to non-Nephrology dept (HR = 2.27) when compared to nephrology dept. By matching AKI-D register data with Regional Register of Chronic Dialysis Treatment for yrs. 2007-2009, a significant change was reported on the percentage of incident chronic dialysis patients who entered in ESRD after AKI-D: 11% in year 2007, 18% in year 2009 (64% increase). AKI-D patients represented 21% of the dialysis population on 2007, 25% on 2009, and with a tendency for 2011 of 33%.
Conclusions: This study provides the evidence that the incidence of AKI-D is continuously increasing, with the consequent the burden of in-hospital and post-hospital resource utilization. In-Hospital mortality rate is extremely high although techniques improvement. Referral to nephrologists is low and a higher utilization of specialists is mandatory particularly in the contest of non-critically ill patients.

SAP155 THE URINARY CYSTATIN C LEVEL AS A PROGNOSTIC BIOMARKER OF MORTALITY IN PATIENTS WITH ACUTE KIDNEY INJURY

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Introduction and Aims: Acute kidney injury(AKI) is a frequent complication in critically ill patients and is associated with high mortality. Clinicians have limited tools to predict the course of AKI at the time of serum creatinine increase. We evaluated the prognostic utility of urinary cystatin C(uCysC) in patients with AKI.
Methods: In this study, serum and uCysC and urinary creatinine (uCr) were measured in patients, presenting with acute kidney injury. AKI was defined and classified according to acute kidney injury network (AKIN) criteria. We excluded the patients with prerenal azotemia and post renal AKI and chronic kidney disease. Prerenal azotemia was defined as a new-onset increase in serum creatinine (sCr) that resolved within 72 hours and returned to the baseline normal kidney function level.
Results: The 126 patients were included in this study. According to AKIN criteria, 39 (31.0%), 36 (28.6%) and 51 (40.5%) patients assigned to stage 1, 2 and 3, respectively. In 126 patients, the mean value of uCysC was 4.0±4.2 mg/L. Urinary CysC concentration rose according to the severity of AKI (mean values 2.65, 3.98 and 5.05 mg/L, corresponding to AKIN stage, p=0.027). During the course of AKI, 35 patients (27.8%) requiring renal replacement therapy (RRT). Thirty-three patients (26.2%) died with comobidities. In 3 patients (2.4%) with AKI did not recovered and underwent dialysis after hospital discharge. In the group of RRT, uCysC/uCr ratio was significantly higher than non-RRT group (0.15 vs. 0.08, respectively, p=0.035). Urinary CysC and uCystC/uCr ratio were not related with recovery of AKI. In multivariated analysis, uCysC and uCysC/uCr ratio related with hospital mortality (p=0.045, p=0.009, respectively). Age, sex, hypertension, diabetes, infection and sCr were not related with hospital mortality.
Conclusions: Urinary CysC and uCysC/uCr ratio were useful for predicting the course of AKI and hospital mortality.

SAP156 CLINDAMYCIN RELATED ACUTE KIDNEY INJURY IN 22 CASES

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Introduction and Aims: Clindamycin is a commonly used antibiotics in current medical practice. Few report has been made on serious adverse effects associated with this drug in abroad. We retrospectively investigate the clinical and pathological manifestation of acute kidney injury (AKI) following infusion of clindamycin in 22 cases.
Methods: From Aug, 2008 to Mar, 2011,37 patients were diagnosed as AKI after infusion of of clindamycin, while 14 patients were excluded from this study for conjunction with other suspected nephrotoxic drugs, and 1 patient with history of chronic kidney disese was also excluded. All the other 22 patients meets the following three criteria: 1)No previous history of underlying chronic kidney disease; 2)Meet the AKIN critieria of AKI soon after the infusion of clindamycin; 3)No obvious other cause of AKI, e.g. volume insufficency, septic shock, urinary obstruction, etc.
Results: 13 males and 9 females was included in this study,with an average of 44.46 ±11.53(20~70)years. The reasons of clindmycin therapy were upper respiratory infection, toothache, and routine anti-infection therapy after minor operation with an usual dosage of 1.0-1.5/d. The median time between adminstration of drug and onset of AKI was 1day (0.5h~4d). The most frequent chief complains were nausea, vomiting(54.55%), lumbodynia(22.73%),abdominal pain(22.73%)and edema (13.64%).13 patients(59.09%)were oliguria and 7 patients(31.82%)were anuria, Sixteen patients(72.32%) had episodes of gross hematuria, while only 3 patients (13.64%)encountered fever and 1 patient(4.55%)had skin rash. Laboratory examination revealed anemia in 16(72.32%)patients ,but eosinophilia was not detected. Nineteen(90.91%)patients were diagnosed as AKI 3 stage, the other 2 cases were in AKI 1 stage on admission . Urine analysis revealed mild proteinuria(0.44 ±0.35g/24h)and severe tubular function injury. Urine eosinophilic cell was positive in only one of them, and uniform microscopic hematuria was positive in 3 patients. Clindamycin lymphocyte transformation assay was positive in 13/16(81.25%) of the patients. Renal biopsy was performed in 18 of them, immunofluorescence were negative with mild glomerular lesions, 10 patients(55.56%)were diagnosed as acute interstitial nephritides(AIN), 2(11.11%)were acute tubular necrosis(ATN), while the other 6 patients(33.33%)had the characters of both AIN and ATN. Continuous renal replacement therapy(CRRT) were delivered to 16 patients(72.32%), and prednisone (30mg/d)was prescribed to 19 patients(86.36%). Urine volume increased 7-14 days later, all the patients discharged from the hospital and free of renal replacement therapy. Serum creatinine was elevated in only one patients 1 month after discharge, and decreased to normal 6 months later.
Conclusions: Most of the AKI associated with clindamycin was oliguria with episodes of gross hematuria, while fever, skin rash and eosinophilia was uncommon. Renal biopsy proven revealed AIN in most of the patients. The recent prognosis is relatively good but the long-term prognosis should be followed.

SAP157 CYSTATIN C PREDICT MORTALITY BUT NOT AKI OCCURRENCE AMONG ELDERLY CRITICALLY ILL PATIENTS

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Introduction and Aims: It has been reported that elderly critically ill patients in the intensive care unit (ICU) have higher risk of developing acute kidney injury (AKI). Despite significant improvements in therapeutics, AKI remains one of the main risk factors that contribute to morbidity and the increase in mortality rate in this population. Thus, in clinical practice, early detection of AKI is crucial in improving the patient's outcome. Aims: To prospectively evaluate whether serum cystatin C can predict AKI occurrence and whether its levels are associated with mortality in elderly critically ill patients in the intensive care unit (ICU).
Methods: This was a prospective cohort study of elderly critically ill patients (> 60 years old) without AKI at admission and with initially normal serum creatinine levels. We enrolled 234 ICU patients, 45 of whom (19%) developed AKI according to AKIN criteria. We also evaluated the demographic and clinical characteristics of patients according to normality for serum cystatin C (= 0.96 and > 0.96 mg/dL). We measured serum creatinine (automated Jaffé method (CREA - Hitachi 912, Roche Diagnostics) and cystatin C in the first 24 hours of ICU admission (by nephelometry).
Results: The mean age of the cohort studied was 74 years and 70% were men. We observed that those patients on mechanical ventilation at admission were more prone to develop AKI (35.5%). Eight (17.8%) of the AKI patients needed renal replacement therapy (RRT). Higher serum levels of cystatin C were more prevalent in patients with more ICU hospitalization days (6±16 vs. 4±6; p = 0.04). Besides, a higher serum level of cystatin C at admission was associated with the development of sepsis (34.8% vs. 13.8%, p < 0.001) and vasopressor drug use (23% vs. 15%, p = 0.01). Interestingly, higher levels of cystatin C did not discriminate AKI occurrence (1.05±0.48 vs 0.94 ±0.36; p = 0.1). However, in logistic regression analysis, we observed that a higher cystatin C level was an independent predictor of mortality (H.R. = 6.16; (95% CI 1.46 – 26.00; p = 0.01). In contrast, AKI was not associated with death.
Conclusion: We demonstrated that a higher cystatin C level is an independent predictor of mortality in elderly critically ill patients in the ICU, indicating that it could be used as a marker of poor prognosis in this population.

SAP158 RENAL EXPRESSION OF MONOCYTE CHEMOTACTIC PROTEIN-1 IN PATIENTS WITH SCHISTOSOMIASIS MANSONI IN AN AREA OF LOW ENDEMICITY IN NORTHEAST BRAZIL

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Introduction and Aims: Schistosomiasis is one of the most important tropical parasitic diseases, affecting more than 200 million people in the developing world. Renal involvement in schistosomiasis mansoni is seldom described, and it may be characterized mainly by glomerular changes. The aim of this study is to investigate renal abnormalities in patients with schistosomiasis in an area of low endemicity in Brazil using traditional and new biomarkers.
Methods: This is a prospective study including 85 patients with parasitologic diagnosis of schistosomiais mansoni (Kato-Katz) followed at primary care centers in Maranguape city, Northeast Brazil. The patients were divided in three groups: Group I (G-I) – control group with 24 non-infected individuals; Group II (G-II) – group with 30 infected individuals by S. mansoni; and Group III (G-III) – group with 31 infected individuals by S. mansoni, treated and evaluated after treatment. Renal function was evaluated with traditional tests, including dosage of urine osmolality, sodium, potassium, creatinine, glomerular filtration rate (GFR), microalbuminuria and the biomarker MCP-1 (monocyte chemotactic protein-1).
Results: There was no difference between the three groups regarding age and gender. Urine osmolality was higher in G-II (664±237mmol/kg H2O), when compared to G-I (538±203mmol/kg H20; p=0,41) and G-III (552 ± 171mmol/kg H2O; p=0.04), being significant when compared to G-III. Urinary sodium was also higher in G-II (154±50mEq/L) than in G-I (113±35mEq/L; p=0.001) and G-III (129±50mEq/L; p=0.05). Urinary potassium was higher in G-I (98±53mEq/L) when compared to G-II (21±15mEq/L; p=0.001) and G-III (31±36mEq/L; p=0.001). Regarding GFR and microalbuminuria there was no significant difference between the three groups. MCP-1 was higher in G-II (178±97pg/ml-Cr) and G-III (175±87pg/ml-Cr), when compared to G-I (123±48pg/ml-Cr), p=0.009 and p=0.007, respectively, but there was no significant difference when comparing G-II and G-III (p=0.892). This evidence that the glomerular abnormality in the infected group did not revert after treatment. Despite not having a significant difference between the three groups

regarding microalbuminuria, there was a significant correlation between the levels of MCP-1 and microalbuminuria (r=0.463, p=0.01).
Conclusions: There was no significant difference regarding GFR, which shows that GFR is preserved in schistosomiasis, predominating electrolyte abnormalities, with increased loss of sodium and retention of potassium. There was a significant increase in the urinary levels of MCP-1 in patients with schistosomiasis mansoni, evidencing its role as a renal function marker. As this protein plays a role in the recruitment of monocytes to sites of injury and infection, its increase in the urine suggests that there is a significant kidney involvement in schistosomiasis and this new biomarker seem to be better than the traditional ones for the evaluation of renal function in this parasitic disease.

SAP159 SNAKEBITES-INDUCED ACUTE KIDNEY INJURY IN NORTHEAST BRAZIL

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Introduction and Aims: Acute kidney injury (AKI) is a common complication of snakebites and is a public health problem in tropical countries. There are four venomous snakes genus involved in this complication: Bothrops, Crotalus, Lachesis e Micrurus. The aim of this study is to investigate the occurrence of AKI after snakebites in a reference hospital in Brazil.
Methods: This is a retrospective study including all patients victims of venomous snakebites admitted to the José Frota Institute, a reference emergency hospital in Fortaleza city, Northeast Brazil, from January 2003 to December 2010. Patients with AKI (group I) were compared to those without AKI (group II). Statistical analysis was done by the SPSS program, and p values<0.05 were considered significant.
Results: A total of 233 patients were included. The majority of patients were male, precedent of rural areas (85.4%) and the most frequently affected body area was the lower limbs (62.2%). The prevalence of AKI was 10.3%. The main involved snake was Bothrops sp (62% of cases). The mean age of group I was 42±20 years, while in the group II it was 33±21 years (p=0.04). The time between the accident and medical care was higher in group I (23±24 hours) than in group II (14±17 hours), p=0.02. The time between the accident and the administration of the antiophidic sera was also higher in group I (24±24 hours) than in group II (13±15 hours), p=0.001. Serum sodium in group I was 134±6.9mEq/l, while in group II it was 139±4.8mEq/l (p=0.0001). The length of hospital stay was higher in group I (14±12.5 days) than in group II (3.3±2.2 days), p=0.0001. Factors associated to the development of AKI in the multivariate analysis were time between the accident and the administration of antiophidic sera, the dose of antiophidic sera and length of hospital stay. AKI was predominantly oliguric (54.2%), with a mean creatinine of 3.3±3mg/dl, need of dialysis in 29.1% of cases and complete renal function recovery in 50% of cases. There was no death in this cohort.
Conclusions: AKI is an important complication of snakebites, being characterized by its severe course, in which half of the patients do not present complete recovery of renal function. The delayed administration of antiophidic sera is an independent risk factor for the development of AKI, which should guide preventive measures for providing these sera in the areas where accidents occur.

SAP160 AKIN (ACUTE KIDNEY INJURY NETWORK) SCORE DID NOT PREDICT SURVIVAL AFTER ORTHOTOPIC LIVER TRANSPLANTATION (OLT)

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Introduction and Aims: Previous studies have shown that perioperative renal failure exerts a significant negative impact on liver transplantation outcomes. However, serum creatinine cut-offs used in those studies tends to underestimate renal failure in patients with cirrhosis. The aim of the present study was to determine the impact of preoperative kidney dysfunction assessed by the AKIN score on survival at 28 days and at one year after OLT.
Methods: This retrospective study evaluated 283 adult patients with advanced chronic liver disease who were consecutively admitted in an urban tertiary medical center to undergo OLT from June 1st, 2005, to December 31, 2009. Subjects with end-of-stage kidney disease diagnosed prior to OLT and/or submitted to liver-kidney transplantation were excluded. Acute kidney injury was defined according to the AKIN criteria and the score was determined at admission and immediately before transplantation.
Results: From the 283 included patients (52.4±11.7 yrs, 72.8% males), 54.4% had viral-related disease, with a MELD of 19.4±17.5, and 21.6% had pre-OLT diabetes mellitus. Acute renal injury at admission and on the day of OLT was observed in 83 (29.3%) and 81 (28.6%) subjects, respectively, with the following AKIN scores: 1=48.2%, 2=22.9%, 3=28.9% at admission; and 1=37.0%, 2=13.6%, 3=49.4% on the day of transplantation. During hospitalization, RRT (conventional or continuous) was required in 77 cases (27.2%). Mortality rate at 28 days was 6.4%, with similar rates across AKIN scores (1=7.5%, 2=5.3%, 3=4.2% at admission, p=0.692; and 1=3.3%, 2=9.1%, 3=7.5% on the day of OLT, p=0.811). Likewise, the mortality rate 1 year after OLT (13.8%) was not influenced by pretransplant AKIN scores at admission (1=17.5%, 2=26.3%, 3=16.7%, p=0.129). However, mortality rate 1 year after OLT was associated with higher AKIN on the day of OLT (1=6.7%, 2=18.2%, 3=30%, p=0.004). After adjustment for MELD score and pre-OLT diabetes mellitus, RRT (OR 4.116, CI95% 1.830–9.261, p=0.001) and older age (OR 1.037, CI95% 1.000–1.076, p=0.049), but not AKIN score, were independently associated with death within 1 year of OLT.
Conclusions: Unexpectedly, the AKIN score did not predict the survival rate at 28 days and within 1 year of OLT. The most important prognostic factor for survival was the need for peritransplant RRT.

SAP161 SNAKE BITES, OBSTETRIC COMPLICATIONS AND INDIGENOUS MEDICINES ACCOUNT FOR HALF THE CASES OF BIOPSIED ACUTE KIDNEY INJURY IN SOUTHERN INDIA

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Introduction and Aims: The etiology of acute kidney injury (AKI) in Southern India is vastly different from that in more developed countries. A working knowledge of these etiologies is imperative for the western world as there are increasing opportunities for travel and work to various parts of the world. There is also continued need to understand its etiology, not only to guide in the management of these patients, but also to encourage advances in understanding its pathophysiology. The aim of this study is to evaluate the etiology and histopathology of AKI in a tertiary centre in southern India.
Methods: Ours is a tertiary care referral centre in southern India where we receive biopsies from rural as well as urban regions. All native kidney biopsies reported in the past 8 months (May 2011 to December 2011) in the renal pathology division of our institute were analyzed. Patients who had a clinical presentation of acute kidney injury were included in this study. Clinical details as specified by the referring clinician, along with the histopathological (light and immunofluorescence microscopy in all cases and immunohistochemistry for myoglobin, when applicable) findings were evaluated.
Results: Of the 857 native kidney biopsies, acute kidney injury was the indication for biopsy in 95 (11.1%) patients. This included 57 men and 38 women. The mean age was 37.5 years (ranging 13 years to 85 years)ATN- Acute tubular necrosis, AIN- Acute interstitial nephritis, TMA- Thrombotic microangiopathy, PIGN- Post infectious glomerulonephritis Among the 857 native kidney biopsies evaluated, there were 62 cases of post infectious glomerulonephritis (PIGN). Amongst these, 11 adult patients presented as acute kidney injury and were included in this evaluation.
Conclusions: This study highlights some causes of AKI which are unusual to the Western world. 1) AKI was the indication for renal biopsy in 11% of the patients. 2) Snake bites, obstetric complications and indigenous medicines account for half the cases of biopsied AKI in Southern India 3) Unsupervised consumption of traditional /indigenous medicines causes acute interstitial nephritis. 4) PIGN is still an important cause of AKI in adults.

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Clinical history	Number	Histopathological findings
Snake bite	17	ATN (9) AIN(4) Cortical necrosis(2) Rhabdomyolysis(2)
Obstetric complications	15	Cortical necrosis(6) TMA(5) ATN(4)
Traditional/indigenous medicines	15	AIN(10) ATN(5)
AKI of unknown etiology	11	ATN(4) PIGN (3) AIN (2) Cortical necrosis(1) TMA(1)
Bacterial infections/sepsis	10	ATN(5) PIGN(4) AIN (1)
Pyrexia of unknown origin	9	PIGN(4) Acute pyelonephritis with microabscess (2) Rhabdomyolysis(1) AIN(1) ATN (1)
Falciparum malaria	5	ATN(2) AIN (2) TMA (1)
Leptospirosis	4	Rhabdomyolysis (2) ATN(2)
Diarrhoea/vomiting	4	ATN(4)
Wasp sting	3	AIN(2) Rhabdomyolysis(1)
Mismatched blood transfusion	1	ATN(1)
Poisoning	1	ATN(1)

SAP162 ACUTE KIDNEY INJURY AFTER AUTOLOGOUS BONE MARROW TRANSPLANTATION IN PACIENTS USING LOWER DOSAGE OF DIMETHYL SULFOXIDE

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Introduction and Aims: Acute kidney injury (AKI) is being increasingly recognized as a common complication of bone marrow transplantation (BMT). The aim of this study is to investigate if lower, but still effective, dosage of dimethyl sulfoxide (DMSO) can reduce the occurrence of AKI after BMT.
Methods: This is a prospective observational study with 20 patients treated with autologous BMT at the Walter Cantidio University Hospital, Fortaleza city, Northeast Brazil, between August 2008 and June 2011. The development of AKI was evaluated based on RIFLE and AKIN criteria. Statistical analysis was done through the SPSS program. The usual dosage in literature of Dimethyl sulfoxide (DMSO) is 10%. The bone marrow transplant's center of HUWC establishes a DMSO's concentration lower than 10% in the infused cells, lower than the literature, and never exceed the dose of 1,5g/1kg. The DMSO usage time was limited to the very moment of the cells infusion.
Results: A total of 20 patients were treated with BMT in the study period. The average age was 41±12 years, and 60% were male. The main indications for BMT were Hodgkin lymphoma (40%), multiple myeloma (35%) and non-Hodgkin lymphoma (20%). The mean creatinine at admission was 0.7±0.1mg/dL. No patient had AKI at admission. AKI was observed in 6 patients (30%) according to RIFLE criteria, 2 in RIFLE-R and 4 in RIFLE-F. According to the AKIN criteria AKI was seen in 7 cases (35%), being 3 in AKIN-1 and 4 in AKIN-3. Febrile neutropenia was seen in 13 cases (65%), and 12 of them used vancomycin. Among patients using vancomycin, 5 developed AKI according to the RIFLE criteria (41%) and 6 according to AKIN (50%) and 9 patients with the use of DMSO alone do not developed AKI. Renal function recovery was observed in almost all cases. Only one patient had elevated creatinine at the time of hospital discharge. There was only one death in this cohort, a 24 years old patient due to a severe sepsis that did not respond to antibiotic regimen. The low dosage of DMSO, and the fact that the majority of patients who had AKI had also used vancomycin, lead us to the conclusion that they had AKI due this medication.
Conclusions: AKI is a frequent complication after BMT and is related to DMSO dosage. The incidence of AKI in our cohort was similar to that reported in literature (25-30%) but was due to vancomycin and not DMSO. A potential risk factor is the misuse of DMSO, wich can cause nephrotoxicity when it exceeds the maximum concentration established. We recommend low and effective dosage of DMSO to avoid AKI.

SAP163 PREDICTIVE FACTORS FOR AKI DEVELOPMENT IN OLDER PATIENTS

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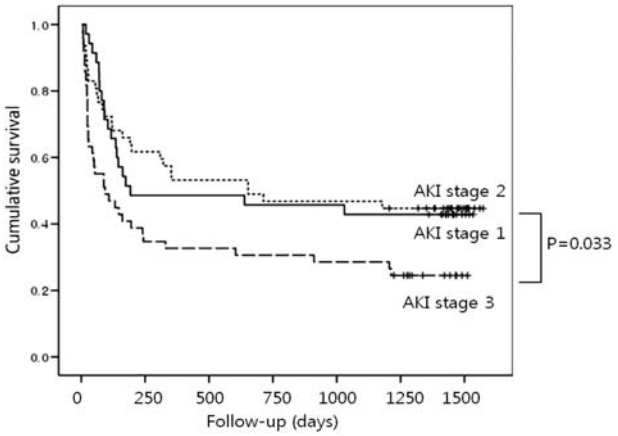
Introduction and Aims: Acute Kidney Injury (AKI) in older patients (pts) is a complication that impacts morbidity and mortality. Furthermore, its association with acuity of illness at Intensive Care Unit (ICU) admission, presence of comorbidities and pre-hospital functional status has not been previously described.
Methods: A total of 100 elderly pts. admitted to the ICU between September/2011 and January/2012 were prospectively evaluated for AKI, defined as an increase in serum creatinine (SCr) > 0.3 mg/dL over baseline value in 48 hours, according to AKIN definition. Univariate analysis was used to study ICU admission parameters associated with AKI development during ICU stay. The severity of acute physiological derangement at ICU admission was assessed with SAPS 3 Score, the presence of co-morbidities with Charlson Comorbidity Index and functional status with VES-13 Score. Goodness of fit and discrimination of the three severity scores evaluated for AKI were calculated with Hosmer-Lemeshow (HL) and area under the ROC curve (AUC ROC), respectively.
Results: AKI incidence was 28 % (n=28), with a mortality rate of 35,7% (n=10), compared to 11% (n=8) for non-AKI pts. The mean age of all pts. was 75,4 ± 8.2 y/o and 48% (n=48) were male. Post-operative (36%) and sepsis (23%) were the major reasons for admission at the ICU. At univariate analysis, the following variables at ICU admission were significantly associated with AKI development during ICU stay: serum potassium (4.4 mEq/L, 3.6-5 vs 3.9 mEq/L, 3.6-4.3; p=0.02), baseline SCr (2 mg/dL, 0.9-3.2 vs 1.1 mg/dL, 0.9–1.4; p=0.001), hemoglobin (10.2 g/dL, 9.2-12.8 vs 12 g/dL, 10.9-13; p=0.03), age (78.5 y/o, 72.2-84.7 vs 74 y/o, 67-81; p=0.03), systolic blood pressure (140 mmHg, 121-155 vs 121 mmHg, 92.5-148; p=0.01), unplanned ICU admission (38% vs 17.4%; p=0.02), diabetes (52% vs 19%; p=0.001), low cardiac output (53.6% vs 18.3%; p=0.0001), heart failure (83.3% vs 24.7%; p=0.002) and acute infection (57% vs 23.5%; p=0.01). The AUC ROC for SAPS III, Charlson and VES-13 Scores were 0.73, 0.68 and 0.66, respectively and the HL p values were 0.73, 0.68 and 0.66, respectively. Variables previously thought to be indicative of a poor prognosis for the elderly, such as functional status, institutionalization and frailty were not predictive of AKI.
Conclusions: AKI is frequent in critically ill elderly pts. and is associated with a high mortality. AKI incidence can be better estimated by evaluation of the acute organ dysfunctions at ICU admission than by the presence of comorbidities or pre-hospital functional status.



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SAP164 OUTCOME OF HOSPITAL-ACQUIRED ACUTE KIDNEY INJURY ACCORDING TO AKIN CRITERIA: LONG-TERM FOLLOW UP

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Introduction and Aims: Assessment of short-term outcome in hospital-acquired acute kidney injury (AKI) may underestimate the true burden of disease. It is important to focus to on long term survival. We investigate the long term outcome of hospital acquired AKI according to the Acute Kidney Injury Network (AKIN) criteria stage.
Methods: This is a prospective, observational, single center study. All hospital acquired AKI patients were included. We monitored serum creatinine everyday for all patients using a hospital data survey system during the study period from Sep. 2007 to Aug. 2008. We calculated the survival time with Korean national health insurance data system.
Results: The baseline population consisted of 131 hospital acquired AKI patients. Hospital acquired AKI occurred in 1.2 % of all hospitalized patients. Among patients with AKI, 29.2 % were stage 1, 36.5% were in stage 2 and 34.4% were in stage 3. Median follow up days is 197 (53-1422) days. The hospital mortality rate was 32.3%, and the long term follow-up mortality was 63%. Cumulative mortality for patients with stage 3 was significantly higher than stage 1 and 2 ($p<0.033$) (figure 1).
Conclusions: Long term outcome of hospital acquired AKI consist of a high additional mortality. AKIN criteria is useful to predict long term outcome of hospital acquired AKI. Figure1. Kaplan Meier survival curve of hospital acquired AKI according to AKIN criteria

SAP165 THE VALIDITY OF THE URINE OUTPUT AND SERUM CREATININE AS CRITERIA OF RIFLE CLASSIFICATION IN OUTCOME OF AKI

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Introduction and Aims: Urine output (UO) and serum creatinine (s-Cr) are valuable markers of kidney function and diagnosing criteria for acute kidney injury (AKI) staging according RIFLE classification. It is important to assess their ability to forecast outcome of the AKI. To study this issue further we analyzed whether these two criteria are sensitive, specific, and early measure for outcome in community acquired AKI (CA-AKI).
Methods: We retrospectively analyzed 7 parameters (age, blood urea nitrogen (BUN), pH, APACHE II score, s-Cr, UO and duration of oliguria/days) in 112 pts with CA-AKI. The outcome was defined as survived and died and the survived were divided as total recovery and survived with some degree of renal failure (RF) at the

discharge from hospital.
Results: Almost all patients at the admission were in RIFLE 3 stage. The mean values were: age 45.51±18.08 (14-87), UO 593.44 ± 661.39 (0-3000), s-Cr 716.14 ± 436.01 (133-2356), BUN 34.94 ± 15.72 (7 -102), pH=7.32 ± 0.11 (6.95 -7.55), APACHE II score 17.32 ± 7.44 (0-38), days of oliguria 5.08±7.12 (0-45). The patients who died were significantly older than those alive (53.96±17.30 vs. 43.09±17.66; p=0.007), had significantly lower levels of s-Cr (553.57±231.26 vs. 762.86±469.63, p=0.033), pH (7.28±0.13 vs. 7.34±0.10, p=0.030) and higher APACHE II score (22.64±10.03 vs. 15.79 ± 5.72, p=0.000). The group of patients with complete restitution were significantly younger than those with some degree of RF (39.04 ± 17.79 vs. 50.06 ± 15.32, p=0.004), had significantly higher pH (7.36±0.08 vs. 7.31 ± 0.12, p=0.041) and less days of oliguria (3.82 ± 4.99 vs. 7.09 ± 7.98, p=0.021). Analysis of variance test between patients who died, patients with complete restitution and patients with some degree of RF showed that the patients were significantly different between age (53.96 ±17.30 vs. 39.04±17.78 vs. 50.06±15.32, p=0.000), urine output (367.40±459.31 vs. 748.73±718.19 vs. 503.13±641.94, p=0.036), sCr (553.57±231.26 vs. 659.29 ± 394.09 vs. 940.87 ± 538.14, p=0.001), pH (7.28 ± 0.13 vs. 7.36±0.08 vs. 7.31 ± 0.12, p=0.015) and APACHE II score (22.64 ± 10.03 vs. 14.93 ± 5.23 vs. 17.28 ± 6.31, p= 0.000).
Conclusions: Values of sCr are controversial in relation to the ultimate outcome, but the more important meaning of this criterion is in relation to outcome in survivors. In patients with complete recovery sCr levels were lower, with higher pH and urine output significantly higher compared to patients who have been with some degree of RF when leaving the hospital. Urine output was with significantly higher values in survived pts. Defenitly APACHE II score is good at differentiating these patients compared to the ultimate outcome. The inclusion of new biomarkers of AKI is needed in the next new staging system

SAP166 CIRCULATING ANGIOPOIETIN-2 AS A MARKER OF A COMPLICATED COURSE OF E.COLI 0104:H4 INDUCED HEMOLYTIC UREMIC SYNDROME IN ADULT PATIENTS

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Introduction and Aims: From May until July 2011 Northern Germany observed the largest reported outbreak of Shiga toxin-induced hemolytic uremic syndrome (HUS) caused presumably by consumption of sprouts contaminated with enterohaemorrhagic E. coli (EHEC) O104:H4. It is widely believed that Shiga toxin induced vascular damage is the culprit of this devastating disease. We analyzed vascular function and circulating Angiopoietin levels in this patient population.
Methods: We characterized and enrolled 47 adult patients with confirmed HUS in the Medical School Hannover. Blood samples were collected prospectively at different time points. In 30 patients we measured flow mediated dilatation (FMD) using the EndoPad device. Circulating Angiopoietin-1 and -2 levels were measured using an in-house immunoluminometric assay.
Results: An inverse correlation between the disease severity of HUS and the measured FMD was noted. Circulating Angiopoietin-2 levels were significantly higher in all HUS patients compared to healthy controls. Angiopoietin-1 and-2 levels correlate with established markers of HUS like platelets, LDH etc. Moreover circulating Angiopoietin-2 on admission correlates significantly with a complicated course of HUS defined by need of renal replacement therapy, need for ICU and mechanical ventilation or occurrence of neurological complications like seizure. Early Angiopioetin-2 levels also predict the renal function on day 42 after admission measured by the eGFR.
Conclusions: Our results show that there is an endothelial dysfunction in terms of a disruption of the microcirculation in HUS. Circulating Angiopoietin-2 correlates with severity of HUS. Furthermore our data suggest that circulating Angiopoietin-2 on admission might be a useful tool to identify patients at risk of developing a complicated course of HUS.

SAP167 FIBRATE-INDUCED ACUTE KIDNEY INJURY: A COMMON AND OCCASIONALLY IRREVERSIBLE COMPLICATION

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Introduction and Aims: Fibrates are increasingly prescribed for the treatment of hypertriglyceridemia. Several case reports have described acute kidney injury (AKI) in patients treated with fibrates, although most of them were due to rhabdomyolysis.
Methods: Single-center analysis of patients who showed fibrate-induced AKI (FB-AKI) in the period 2007-2010. Definition of FB-AKI included a >20% serum creatinine (sCr) increase in at least two consecutive measurements after the onset of fibrate therapy, absence of other possible causes of AKI and improvement of renal function after fibrate withdrawal.
Results: We collected 60 cases of FB-AKI (37/23 M/F, aged 69±11 yr). Forty three (71%) had stage 3 CKD at the onset of fibrate treatment, 52 patients (87%)

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	Complete recovery (51)	Partial recover (9)	p
Age (yr)	69±11	66±13	0,6
Baseline sCr (mg/dl)	1,2±0,6	1±0,7	0,07
Peak sCr (mg/dl)	1,94±0,26	2,2±0,66	0,43
Final sCr (mg/dl)	1,23±0,29	1,4±0,2	0,04
% increase of sCr (range)	51.6 (23-141)	86.4 (43.6-286)	0,05
Duration of fibrate treatment (months)	13.5 (2-132)	29 (7-45)	0,27

hypertension, 37 patients (62%) type2 diabetes, 39 patients (66%) hyperuricemia. Clinical or analytical signs of rhabdomyolysis were not detected in any patient. Median sCr increase from baseline values was 53.4%(r 23-286). Baseline sCr was 1.2 ±0.25 mg/dl, peak sCr 2±0.45 mg/dl and final sCr after fibrate discontinuation 1.26 ±0.28 mg/dl. Renal function improved in all the patients after fibrate withdrawal but 9 (15%) did not recover baseline values of renal function. As shown in the table, these patients had a better renal function at baseline, had shown a greater sCr peak and had tended to receive fibrates for a longer time. Characteristics of patients with complete or incomplete recovery of baseline renal function after fibrate withdrawal
Conclusions: FB-AKI in the absence of rhabdomyolysis is not an uncommon complication. Most of the cases were due to fenofibrate. Although renal function improved after fibrate withdrawal 15% of patients exhibited a partial recovery of baseline renal function values.

SAP168 A PROSPECTIVE OBSERVATIONAL BIOELECTRICAL IMPEDANCE STUDY: PROGNOSIS IN ACUTE KIDNEY INJURY

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Background and Aim: Bioelectrical impedance analysis (BIA) is a simple, cheap and non-invasive tool for monitoring nutritional and hydration status in several diseases, including chronic kidney disease. The aim of this prospective study was to evaluate BIA in the prognosis of Acute Kidney Injury (AKI).
Material and Methods: In a cohort of 96 patients (mean age: 64 years-old, SD: 1.57, 76 males) with a diagnosis of AKI, a bioimpedance analysis was made. Bioelectrical parameters: Phase angle (PA), Total Body Water, Extracellular/Intracellular water ratio (EC/IC), Fat free/Muscle mass ratio (F/M), Na/K exchange rate (Na/K); acute clinical index: individual severity (ISI), multiorgan failure index (MOFI); chronic clinical index: Charlson and Karnofsky; biochemical parameters: C-reactive protein, prealbumin, albumin and peak creatinine; and mortality were evaluated. The mortality rate was 14%.
Results: In Table 1 correlation results are shown among different variables. According to mortality, the PA provided the highest prognostic information with an Area Under the curve (AUC) of 0.78 (95%CI: 0.65-0.91) and the cutoff was 3.2° (Sensitivity: 78%, Specificity: 67%). Respect to survival, the Na/K exchange showed an AUC of 0.81 (IC95%CI: 0.61-1.00), the cutoff was 1.39 (Sensitivity: 83%, Specificity: 67%). Using a stepwise multivariate logistic regression analysis including all the bioelectrical parameters, an independent relationship was found between Na/K exchange and mortality (OR 15.38; 95% CI: 1.61-146.4).
Conclusions: Bioelectrical impedance analysis can be used as a parameter in the prognosis of AKI. High Extracellular water volume, low fat free mass and moreover decreased phase angle and elevated Na/K are related to a worst prognosis in the patient with acute renal failure.

SAP169 METFORMIN-ASSOCIATED LACTIC ACIDOSIS (MALA): A SINGLE CENTRE EXPERIENCE ON 14 PATIENTS

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Introduction and Aims: Metformin-associated lactic acidosis is a well-known side effect of Biguanides. Over the last 10 years, we have observed an increase in hospitalisations for MALA. Our aim was to investigate the underlying and precipitating causes of the phenomenon, the risk categories, and the short, medium, and long term survival.
Methods: We conducted a retrospective analysis on 14 patients affected with MALA and acute renal failure (M:F 1:1; mean age 67 ± 9 yrs) who were admitted to our

SAP168 Table 1 Correlations among different studied variables

	Phase angle (°)		Total Body Water (L)		EC/IC		Na/K exchange rate		F/M	
	r	p	r	p	r	p	r	p	r	p
ISI	-0.22	0.035		ns		0.23	0.033	ns		ns
MOFI	-0.25	0.035		ns	-0.32	0.002	0.34	0.014		ns
C-reactive protein (mg/dL)	-0.42	0.005		ns	0.40	0.001		ns	-0.42	0.005
Prealbumin (mg/dL)	0.38	0.025	-0.364	0.034	-0.48	0.004	-0.43	0.049	0.51	0.016
Albumin (mg/dL)	0.32	0.011		ns	0.39	0.002		ns		ns
Karnosfky	0.44	0.005		ns	-0.54	0.005	-0.32	0.025		ns
Charlson		ns	0.355	0.015		ns		ns	-0.33	0.023
Peak creatinine (mg/dL)		ns		ns		ns		ns	0.38	0.004

ISI: individual severity index, MOFI: multi organ failure index
EC/IC: Extracellular/Intracellular water ratio, F/M: Fat free/Muscle mass ratio
ns: no-significance

division between January 2001 and January 2011 (fig.1). Inclusion criteria were: type II diabetes diagnosed at least one year prior to entry, metformin administration as monotherapy before the development of lactic acidosis, and lactic acidosis (lactate: >5 mmol/L). The treatment of choice was bicarbonate haemodialysis, the duration of which was based on the persistence of metabolic acidosis or hyperlactacidemia. We collected data concerning co-morbidities, vital signs and hydro-electrolytes at admission, at 24h and at discharge (fig.2). In order to evaluate the role of comorbid factors and precipitating causes of metformin and lactate accumulation, we first divided the acute precipitating factors (events preceding the genesis of lactic acidosis <72 hours) from the chronic diseases (chronic renal failure; COPD; etc.). A MALA risk score (fig.3) was then calculated to investigate the clinical course and to find relationships with survival curves (Kaplan Meier, fig.4). After discharge, we followed-up our patients with phone interviews or as outpatients for a mean of 42 months, and monitored them for other manifestations of lactic acidosis after metformin discontinuation.

Results: All patients presented with acute renal failure, and 57% presented with acute on chronic renal failure. The precipitating causes were prevalently based on acute dehydration (64%), sepsis (29%) and infusion of iodinated contrast medium (7%). Hypertension (57%), neoplasms (21%), anaemia and respiratory failure (14%), and chronic heart failure (7%) were concomitant. 28.6% of our patients died within 24 hours following the first haemodialysis treatment, half of whom were at low risk of MALA. Death was not related to the severity of the score. None of our patients were informed about the side effects of the drug or about when to discontinue it. Following Metformin discontinuation, no other lactic acidosis-related deaths were reported at follow-up and no new episodes of lactic acidosis were observed. Most events occurred during the winter months (January-March), which is consistent with the epidemiological spikes of influenza-like illnesses and Influenza virus A in Southern Sardinia (fig.5). The lack of adherence to guidelines and a concomitant lack of information were responsible for most cases of MALA. The current increase in the number of cases of MALA can be accounted for by the renewed confidence in the drug, which is safe when properly prescribed.

Conclusions: Precipitating factors of MALA must be evaluated very carefully before prescribing Metformin to patients with CKD stages 1-2, and especially stage 3, as well as to other patients, due to its high mortality rate even in low risk patients. All patients who are administered the drug must be informed about its side effects and about how to manage it in high risk conditions.

Variable	
N° patients	14
Age - mean (range)	67 ± 9 yrs (45-76)
Age > 65 yrs	57 %
M:F	1:1
Type II Diabetes	100 %
Metformin - daily intake (posology)	1000 mg/day
eGFR <60ml/min/1.73 m² (before the admission)	57 %
Acute Heart Failure	7 %
Respiratory Failure	14 %
Liver failure	0 %

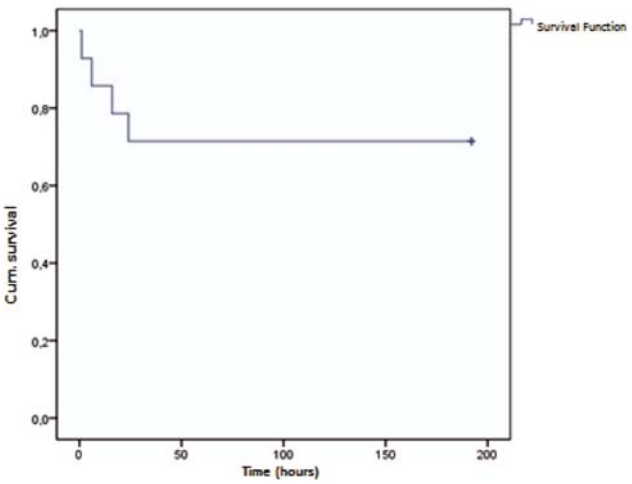
SAP169 Figure 1: Descriptive statistics of selected patients

Variable	n (range) - mean
Serum Creatinine	8.4 (3.1 - 14.8) mg/dl
BUN	118.83 (52 – 264) mg/dl
K+ (serum)	6.0 (4.3 – 9.6) mEq/L
pH	7.14 (6.81 – 7.26)
Venous pCO_2	27.4 (14.6 - 39.6) mmHg
Venous pO_2	71.6 (35 – 45) mmHg
Lactate	9.7 (6.5 - >20) mmol/L
EB	-16.2 (-7.5 - -30.8) mmol/L
Hemoglobin	11.6 (12.8 – 8.20) g/dl

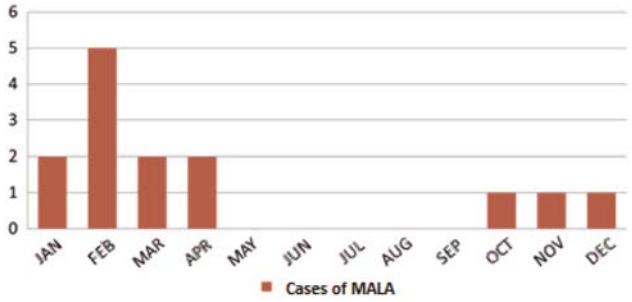
SAP169 Figure 2: Lab exams: renal function, haemoglobin and blood gases analysis

Case	CKD	Chronic Liver Disease	COPD	Benign and malign neoplasms	Chronic Heart Failure	Anaemia	Alchool Abuse	Acute Respiratory Failure	SIRS and Infections	Iodinated Contrast	SCORE
1											1
2											1
3											1
4	X										1
5	X			X							1
6	X								X		2
7	X								X		2
8						X					2
9	X								X		2
10	X		X					X			2
11	X				X	X					3
12				X				X	X		3
13	X					X			X		3
14				X		X			X	X	4
%	57	0	7	21	7	29	0	14	43	7	

SAP169 Figure 3: The MALA reisk score: +1 score points for each comorbidity, chronic and acute, in which exacerbations of chronic diseases were considered as a single score (+1). Each comorbidity was defined according to updated diagnostic criteria set by the litetature. Benign and malign neoplasm are not included in the sum because of their limited extension. CKD: Chronic Kidney Disease; COPD: Chronic Obstructive Pulmonary Disease; SIRS: Systematic Inflammatory Response Syndrome.



SAP169 Figure 4: Kalpan-Meier survival curve of admitted patients affected with MALA (admisson - 192h)



SAP169 Figure 5: Seasonal distribution of MALA

SAP170 PHARMACOKINETICS OF MEROPENEM WITH 3-HOUR INFUSION REGIMEN IN CRITICALLY ILL PATIENTS ON CONTINUOUS RENAL REPLACEMENT THERAPY

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Introduction and Aims: Continuous renal replacement therapy (CRRT) is widely used in critically ill acute kidney injury (AKI) patients. The use of antibiotics in this population is problematic, because despite low renal excretion in AKI, increased clearance is being achieved with more effective therapies, bringing the risk of low plasma levels. Meropenem (Mero), a commonly used carbapenemic, has a time-dependent antibacterial activity, requiring levels continuously above the minimal inhibitory concentration, and a 3-hour infusion regimen per dose has been used to achieve this goal. Since pharmacokinetic data so far were determined with bolus or continuous administration, we aimed to study pharmacokinetics of Mero in AKI patients submitted do CRRT using 3-hour infusion regimen in order to show the adequacy of plasma levels.
Methods: We studied plasma and dialysate Mero concentrations in five oligo-anuric septic patients submitted to continuous veno-venous hemodiafiltration for AKI, using Diapact (Braun*), equipment, AN-69 membrane hemofilters, blood flow of 120 ml/min and effluent flow of 30 ml/Kg. Samples were collected at times 0, 30 min, and 1, 2, 4, 6 and 8 hours after the beginning of a 3-hour infusion of 1 g of Mero, that was being used three times daily for more than 24 hours. Mero determinations were done by high performance liquid chromatography. Plasma and dialysate curves were obtained for each patient, and other parameters were derived: area under the curve(AUC), total clearance(CLt), half-life(t½), distribution volume(Vd), peak (Cmax) and trough (Cmin) concentrations, apparent terminal elimination rate constant(Kel), sieving coefficient (Sc) and the percentage of time above MIC (minimal inhibitory concentration). Values are reported as median and interquartile range.
Results: Four male and one female patients, age 53.0±19.7 (23 to 80 years), 62.1 ±10.6 Kg, were studied. Median pharmacokinetic parameters were: plasma concentrations, 34.86mg/L (10.08- 139.27); t½, 1.8 h (1.4-3.0); Vd, 8.29 L (5.8-15.3); CLt 3.98 L/h(2.51-4.35); Cmax (at the end of Mero infusion-time 3 h), 48.5 mg/L (37.0-105.8); Cmin 20.1 mg/L (14.0-16.6); Kel, 0.38 h- 1 (0.34-0.43); AUC (0 to 8 h), 251.1 mg/L.h(229.7-398.4); AUC (8), 275.1 mg/L.h (263.8-453.6). All plasma samples were above a MIC of 8mg/mL. Dialysate minimum and maximum concentrations at Cmax were 24.35 and 74.81 mg/L, and at Cmin 6.82 and 33.03 mg/L. CRRT clearance varied from 8.46mL/min to 18.33mL/min (median 15.96 mL/min), representing a median proportion of CLt of 35.9% (23.6-70.0). Sieving coefficient was 0.69 (0.65-0.72). Median Mero removal was 175 mg/L.h (151-290).
Conclusions: Elimination of Mero by CRRT with doses of 30 mL/Kg is similar to what is reported by the normal kidney, when a 3-hour infusion every 8 h regimen is used. Plasma levels were always above the usual necessary MICs, and no drug accumulation was noticed. No dose reduction or increment is required in this context.

SAP171 ACUTE MYOGLOBINURIC RENAL FAILURE IN THE INTENSIVE CARE PATIENTS

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Introduction and Aims: Myoglobinuric acute renal failure (Mb-ARF) demanding hemodialysis (HD) contributes considerably to the already high patient morbidity and mortality in the intensive care units (ICU). Besides serum creatinine (Cr) and creatinine kinase (CK) levels also serum myoglobin (Mb) could be used to predict the need for HD.

SAP171 Table 1 Biochemistry and the need for hemodialysis in 484 patients

Group (No.)	A (313)	B (87)	C (27)	D (19)	E (38)
Peak Cr (Avg.)	157	213	261	305	340
Cr, range	71-435	72-673	51-484	79-643	84-888
Peak CK (Avg.)	33.7	55.2	107.3	166.9	565.5
CK, range	0.5-526.3	11.6-244.3	25.5-578.0	6.2-2004.8	27.8-6894.6
Cr>200 (%)	24.6	35.6	48.1	57.9	68.4
Need for HD	21	12	2	2	14

Methods: A one-year analysis of Mb-ARF was performed in the ICU patients with suspected rhabdomyolysis. Serum Mb exceeded 1 mg/l (=15-20 times normal) in 484 patients. Patients were grouped into 5 categories according to the highest Mb levels attained (A :1-5 mg/l, B :5- 10 mg/l, C :10-15 mg/l, D :15-20 mg/l, E : >20 mg/l). The highest serum Cr and CK levels for the individual patients were averaged within the groups and statistical analysis performed. The incidence of significant Mb-ARF (arbitrarily defined as a peak Cr >200 mmol/l) and the need for HD (initiated according to the usual criteria) were compared across the groups.
Results: Peak Cr and CK were significantly lower in groups A and B compared to the groups C, D, and E ($p< 0.01$ for all). Cr and CK were not significantly different between the groups C, D, and E (Table 1). Peak Cr correlated better with Mb ($r = 0.33$, $p< 0.001$) than with CK ($r = 0.15$, $p = 0.01$). Mb correlated with CK ($r = 0.26$, $p< 0.001$). Significant ARF was present in 158 patients: 77 (24.6%); 31 (35.6%); 13 (48.1%); 11 (57.9%); and 26 (68.4%); in the groups A, B, C, D, and E, respectively. Forty-seven patients (10.5%) had one or more HD: 21 (6.7%); 12 (13.8%); 2 (7.4%); 2 (10.5%); and 14 (36.8%) in the groups A, B, C, D, and E, respectively. The incidence of significant ARF was 64.9 % with Mb >15 mg/l ($p = 0.001$ vs. 5 mg/l< Mb <15 mg/l), being 59.5% with Mb >10 mg/l ($p< 0.002$ vs. 5 mg/l< Mb <10 mg/l). The incidence of HD-dependent Mb-ARF was 28.1% with Mb >15 mg/l ($p = 0.01$ vs. 5 mg/l< Mb <15 mg/l), being 21.4% with Mb >10 mg/l (NS vs. 5 mg/l< Mb <10 mg/l).
Conclusions: Rhabdomyolysis was often complicated by acute renal failure in our patient cohort. In parallel with increasing myoglobin levels, serum creatinine and creatinine kinase peaked significantly higher, and a significant renal impairment as well as the need for hemodialysis were present in significantly higher proportion of patients. More than a fifth of the patients with peak serum myoglobin exceeding 10 mg/l, and nearly two fifths with myoglobin levels above 20 mg/l, needed hemodialysis. Serum myoglobin higher than 10 mg/l was significantly associated with significant renal failure, and higher than 15 mg/l with the need for hemodialysis.

SAP172 CLINICAL SIGNIFICANCE OF HIGH URINARY ALPHA-1-MICROGLOBULINE - PROMISING MARKER OF ACUTE KIDNEY INJURY?

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Introduction and Aims: Alpha-1 microglobulin (A1m) is a low molecular weight protein, which passes freely through the glomerular filter and is reabsorbed in the renal proximal tubule. Abnormal tubular function results in higher urinary levels of A1m due to incomplete reabsorption. Urinary A1m is therefore used as a marker of renal tubulointerstitial damage; values are commonly expressed as A1m/creatinine (A1m/creat). In our previous study in patients with glomerular disease, we showed that urinary A1m/albumin (A1m/Alb) correlated with the grade of renal tubulointerstitial damage. We observed extremely high levels of this index in patients with acute tubulointerstitial nephritis. We hypothesised that very high A1m/Alb could identify patients with renal tubulointerstitial damage and that patients with acute and chronic lesions could be differentiated by urinary protein analysis.
Methods: We analysed retrospectively all patients from 2 hospitals (University Hospital Vinohrady, General University Hospital, Prague) whose urine samples were tested for PCR (protein/creatinine ratio), A1m, albumin (Alb) and creatinine (creat) in the years 2007-2011. All patients with proteinuria (PCR > 15 mg/mmol) and A1m/Alb = 0,5 were included in the study. We analysed independently clinical data of all patients and classified them accordingly in 4 groups: 1) patients with clinically evident severe acute kidney injury (due to severe infections, medications, hypersensitivity, monoclonal gammopathy, severe heart failure, or other reasons), 2) patients with chronic tubulointerstitial nephritis, 3) pacients with chronickidney disease of unknown etiology, 4) missing clinical data. We used Mann-Whitney test

and ROC analysis for statistical evaluation.
Results: 291 patients were included in the analysis. 30 patients fulfilled the study criteria: 14 males and 16 females, age 15-81 years. 20 patients had acute kidney injury (Group 1), 6 patients had chronic tubulointerstitial nephritis (Group 2), 2 patients had unknown renal diagnosis (Group 3) and in 2 patients data were missing (Group 4). 1) Clinical significance of A1m/Alb = 0,5: All patients, whose diagnosis was available (N=26, Group 1+2), had either acute kidney injury or chronic tubulointerstitial disease. In 4 patients, the diagnosis was not available. 2) Discrimination of acute versus chronic disease: In comparison of patients with acute kidney injury (Group 1) versus chronic tubulointerstitial nephritis (Group 2), A1m/Alb was not different (p=0,503), while A1m/creat differed significantly (p=0,018). The optimal cut-point for A1m/Alb was 5,132 (AUC 0,592 - 95% CI 0,383-0,778, sens/spec 90/50). The optimal cut-point of A1m/creat was 166,5 mg/g (18,8 mg/mmol, AUC 0,825 - 95% CI 0,626-0,944, sens/spec 65/100).
Conclusions: 1) Urinary A1m/Alb = 0,5 and PCR > 15 mg/mmol identified patients with tubulointerstitial damage due to acute kidney injury or chronic tubulointerstitial nephritis. 2) Urinary A1m/creat can differentiate patients with acute kidney injury from those with chronic tubulointerstitial nephritis. 3) Urinary A1m/Alb and A1m/creat seem to be promising markers of acute kidney injury.

SAP173 'DOES THE CAUSE OF 'PRE-RENAL' AKI IN A GENERAL HOSPITAL SETTING MAKE A DIFFERENCE TO OUTCOMES?'

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Introduction and Aims: AKI is a complex heterogeneous condition with a multitude of causes. Pre-renal AKI makes up the majority of cases of AKI but has many often overlapping inciting factors. The question remains whether or not the outcomes from these factors differ enough to influence the interpretation and design of AKI cohort studies.
Methods: We have conducted a prospective single centre observational study with the aim of exploring the natural history of AKI in the general hospital population. Subjects were recruited over 17 months from Nov. 2009 to April 2011 from unselected admissions to a non-tertiary acute hospital with a catchment population of 600,000. AKI was defined according to the AKIN criteria. Two groups were recruited – AKI Group 1: with previously normal kidney function defined as an eGFR > 60 mls/min and AKI on CKD Group 2: with a background of CKD defined as an eGFR < 60 mls/min. Extensive baseline data were recorded on all patients at the time of the AKI and all measurements of renal function were carried out within the same laboratory. Patients were followed up after 6 months to assess outcomes. Pre-renal causes of AKI were stratified into three categories: Septic, where infection was the dominant inciting factor, Perfusional, where the primary insult caused a haemodynamic disturbance, and Complex, a combination of a septic or perfusional cause with at least one other insult which included causes such as contrast and obstruction.
Results: We recruited 375 patients for follow up (AKI Group 1 n=190, AKI on CKD Group 2 n = 185). Mean age was 72 years (range 18-97 , 53% male, 47% female). Patients in the CKD group were older and had a greater co- morbidity burden. A pre-renal cause was found in 96.8% of Group 1 and 100% of Group 2 all of which fell within the categories described. AKI Group 1 – Septic 22.6% , Perfusional 45.8%, Complex 28.4%. AKI on CKD Group 2 – Septic 31.4%, Perfusional 42.2%, Complex 25.9%. Baseline characteristics were similar across all categories within groups but with a trend toward a greater co- morbidity load in those with septic AKI. In the AKI group there was a trend toward more severe AKIN stages 2 and 3 in the Septic and Complex categories while in the AKI on CKD group less severe AKIN stage 1 was more common in all groups. Inhospital mortality was higher in the AKI on CKD group than AKI Group (8.6% v 3.8% p= .045) which increased after 6 months follow up (24.3% v 12.6% p=.003). In the AKI group mortality outcomes were similar across all categories. In the AKI on CKD group there was a trend toward worse mortality in the septic and complex categories both in hospital and after six months follow up. In terms of functional recovery, defined as returning to within 5mls/min of baseline eGFR, outcomes were again similar across all categories in both groups.
Conclusions: The outcomes of mortality and functional recovery were broadly similar across all categories of pre-renal AKI but care should be exercised in interpreting mortality outcomes in those with previous CKD.These findings will aid in the interpretation and future design of AKI research. Further multivariate analysis is underway to explore these findings.

SAP174 IMPROVEMENT IN OUTCOMES OVER TIME ARE BIASED BY ACUTE KIDNEY INJURY PATIENTS WHO DO NOT BENEFIT FROM RENAL REPLACEMENT THERAPY

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Introduction and Aims: Acute kidney injury (AKI) in critically ill patients that need renal replacement therapy (RRT) has high morbidity and mortality and there is some controversy if prognosis is improving as new knowledge is gained. Early mortality of these patients often reflects the severity of the underlying clinical condition, and the benefit of RRT occurs when the patient survives the initial days of illness. Our objective is to analyze clinical differences between AKI stage 3 patients that survived the first two days of RRT and those who didn't, and determine if the former group is having more benefit from treatment over the years in our institution.
Methods: Every patient that needed RRT for AKI 3 in our intensive care unit (ICU) from may 2006 to may 2011 was followed, excluding patients with renal transplant and stage 5 chronic kidney disease. Hemodinamically stable patients received intermittent hemodialysis (IHD) (Fresenius® equipments and filters) and unstable patients received continuous veno-venous hemodiafiltration , mostly with citrate regional anticoagulation (CRRT)(Diapact-B.Braun®and Prisma/PrismaFlex-Gambro®equipment, AN-69 membrane filters). Several clinical variables were recorded prospectively, including age, gender, race, type and cause of AKI, APACHE II score, co-morbidities, presence of sepsis, basal creatinine. Several variables related to the treatment, including type and duration of RRT, length of stay in the ICU and in the hospital and outcomes (case-fatality and dependence of dialysis) were recorded. Student's t test or chi-square tests were used for comparisons, and logistic regression was used for multivariate comparisons. SPSS version 19.0 software was used for statistics.
Results: 11 % of all patients admitted to the ICU, - 1,107 patients - required RRT because of AKI 3: age 57.3±16,8 years, 55% male, 87% caucasians. Basal creatinine above 1.5 mg/dl was present in 24 %, 64 % had clinical AKI, 83 % were septic, 91% needed mechanical ventilation and 86 % vasopressors. APACHE II score was 27.0 ±8.5. IHD was used in 62% and CRRT in 85%. Median number of days on RRT was 5 (2-13), and length of stay in ICU and in the hospital were 10 (4-19) and 15 (5-31) respectively. Case-fatality rate was 67.1% in the ICU and 73.6% in the hospital. Sepsis, age, APACHE II score and liver failure were related to mortality in multivariate analysis. Patients that died after receiving one or two days of RRT (n=285) were older (60.6 ±16.5 years), had higher APACHE II score (31.6±13.4) and needed more mechanical ventilation and vasopressors than the others (n=815). Analyzing the characteristics and outcomes of both groups according to the year of treatment (2006 to 2011), patients with early deaths didn't have significant differences, but patients receiving RRT for more than 2 days had progressive reduction in case-fatality rate: 61% to 52% in ICU, and 66% to 62% in the hospital (p=0,046). There was also a progressive reduction in ICU (19.2 to 14.4 days) and hospital (29.7 to 19.8 days) length of stay. Analyzing both groups together, there were no differences in outcomes in the six years of our experience.
Conclusions: The outcomes of critically ill patients with severe AKI, treated by RRT, may be improving over time, but these advances are overshadowed by severely ill patients that do not benefit from RRT. We suggest that future studies take these prognostic differences in consideration.

SAP175 MAIN PAPER SHIGA-TOXIN INDUCED HUS

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Introduction and Aims: From May until July 2011 Northern Germany observed the largest reported outbreak of Shiga toxin-induced hemolytic uremic syndrome (HUS) caused by consumption of fenugreek sprouts contaminated with enterohaemorrhagic E. coli (EHEC) O104:H4. We present the clinical course and outcome with different treatment algorithms.
Methods: We analyzed data of 298 adult patients with HUS. Differences in the treatment strategies between hospitals allowed us to perform an exploratory data analysis including a multivariate statistical approach to evaluate the effect of treatment modalities on clinical outcomes.
Results: 160 (53.7%) HUS patients temporarily required dialysis, but nearly all regained renal function. Neurological signs and symptoms (143, 48.0%) including seizures (37, 12.4%) occurred frequently in our cohort. 54 (18.1%) required mechanical ventilation. 12 patients (4.0%) died. Multivariate statistical analysis showed no significant benefit of therapeutic plasma exchange (TPE) and TPE related glucocorticoid therapy on clinical outcome parameters. 67 patients with a severe course of the disease were treated with eculizumab. No short term benefit was detected that could be attributed to this treatment in comparison to a group of 65 matched patients with a similar severe disease course that were not treated with eculizumab. 52 patients in a center administering an aggressive antibiotic combination strategy had fewer seizures (1.9% vs. 14.6%, p=0.03) and deaths (0% vs. 5.1%, p=0.029) and a shorter duration of EHEC shedding.
Conclusions: EHEC O104:H4 induced HUS is a very severe self-limited acute illness. Retrospective analysis does question a therapeutic benefit of TPE or associated glucocorticoid therapy. Antibiotic therapy in patients with established HUS appears beneficial and should be investigated in future EHEC-HUS outbreaks. The long-term effect of eculizumab requires further investigation.

SAP176 SYMPTOMS IN SHIGA-TOXIN INDUCED HUS PATIENTS

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Introduction and Aims: Consumption of sprouts contaminated with the Shiga-toxin 2 producing E.coli (STEC) O104:H4 lead to the largest reported outbreak of hemolytic-uremic syndrome (HUS). Here, we give an in debt presentation of the clinical course of this rare disease.
Methods: We have collected the laboratory and clinical data of more than 400 EHEC-HUS patients from 28 centers. The Medical Symptoms Questionnaire (MSQ) with a total of 70 clinical items was evaluated regarding the severity (0: never or seldom to 4: frequently, effect is severe) the beginning and ending. The questionnaire was answered by 128 patients from 13 different clinics. Most of the patients and the relatives were interviewed during the hospital and after discharge. In three university hospitals 101 EHEC-HUS patients were assessed by neurologists regularly until their symptoms subsided.
Results: 87.7% of patients in whom sprout consumption was assessed recalled having eaten sprouts 7.4±3.3 days before onset of diarrhea. We defined the onset of diarrhea as day 0. Diarrhea (usually bloody within one day) was present in nearly 100% of patients and the average severity score was 3.9. The diarrhea lasted for 9.1±6.6 days. During the early phase of the disease nausea and vomiting and a few days later a bloated feeling was also very prominent GI symptoms. Between day 5-15 50-60% of patients developed peripheral edema (often marked) and >50% also pleural effusion and/or ascites probably due to acute renal failure (60% required dialysis) and vascular leakage. 20% of patients developed shortness of breath and 5-10% complained about angina pectoris like symptoms. Importantly most patients developed neurological symptoms at day 10-14 well behind the LDH peak and platelet count nadir (around day 7-8). The following symptoms were described most commonly by the patients: 65% headache (intensity 2.9), 58.6% poor concentration (intensity 2.75), 54% tremor (intensity 2.9) and 52% insomnia (intensity 3.2). At neurological assessment more than 80% of patients had neurological symptoms. These included dysphasia, hyperreflexia, blurred or double vision, alterations of working memory and short term memory, disorientation, apraxia, agraphia, ataxia, para- or tetraparesis, tremor, panic attacks, myoclonia, seizures, and alterations of consciousness. Magnetic resonance imaging showed lesions in the brainstem, mid-brain, thalamus, corpus callosum and white matter in half of the patients, predominantly in diffusion weighted images. The electroencephalograms showed generalized slowing in part of the patients, while there were no focal alterations. Of note, neither magnetic resonance findings nor the electroencephalograms correlated with the clinical symptoms. After discharge night mares and anxiety persisted for many weeks/months in a substantial amount of patients. Fatigue and weakness (intensity 3.7) that started after day 5 were very disturbing and persisted in most patients for several months and might be partially due to the prolonged anemia.
Conclusions: Shiga-toxin HUS is a very severe disease and most patients felt miserable and were affected by a wide amount of symptoms.

SAP177 WOMEN ARE PRONE TO DEVELOP A MORE SEVERE COURSE OF SHIGA-TOXIN MEDIATED HUS

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Introduction and Aims: In the 2011 German EHEC O104:H4 outbreak, the largest EHEC-HUS outbreak (855 cases) ever reported worldwide, a high number of EHEC-HUS patients (68%) were woman despite that they only represented 58% of all EHEC cases. This observation suggested that woman were more prone to develop HUS and we analyzed if sex had also an impact on the severity of HUS.
Methods: 299 adult EHEC-HUS patients treated during this outbreak at 5 university and 14 district hospitals are presented. Patients suffering from EHEC-associated enterocolitis without HUS were not included. We prospectively and retrospectively collected demographic, clinical, laboratory, and imaging data, as well as information on the hospital course and medication on a standardized case-history form.
Results: 72.2% of patients were female. At time of formal diagnosis of HUS approximately 6 days after the onset of diarrhea no significant difference was observed between both sex regards all major clinical and laboratory parameters except Hb, which was by 1g/dl lower in women than in men. Of interest was the fact that overall the renal function worsened more dramatically in women and hence they required significantly more often renal replacement therapy 66% versus 52% in men (p= 0.033). They also had a significantly higher incidence of pulmonary edema, pleural effusions and severe colitis. Differences in ventilation rate (24.7% vs. 16.9%), neurological symptoms (62.4% vs. 53.8%), seizures (19.3% vs. 15.4%) and overall mortality (4.2% vs. 1.2%) missed significant levels but support the notion that female patients developed a more severe form of HUS. Hospitalization was also longer in women (24.7 vs. 21.4 days, p = 0.06). No obvious gender specific advantages between treatment strategies were identified.
Conclusions: Women are susceptible to develop a more severe clinical course of HUS. Interestingly, also in previous EHEC epidemics women were more prone to develop HUS. The cause for these observations is unclear, but might have to do with a higher bacterial load at presentation or a higher gender specific susceptibility. Further investigations are required.

SAP178 PREDICTIVE PARAMETERS FOR PATIENTS WITH SHIGA-TOXIN INDUCED HUS

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Introduction and Aims: From May to July 2011 a large outbreak of Shiga-toxin producing enterohemorrhagic E. coli (EHEC/STEC) O104:H4 struck northern Germany, affecting thousands of patients and inducing hemolytic-uremic syndrome (HUS) in 852. As huge differences in the clinical courses reached from complete recovery after few days to fatality of previously healthy individuals an early indicator of the outcome in the clinical course became subject of our interest.
Methods: The clinical records of more than 400 patients treated for HUS in 28 hospitals from Germany and Sweden were analysed, data about treatment, clinical course and outcome was surveyed and imported into a database, also including all laboratory findings and follow up data up to 6 months. Univariate and multivariate analysis were performed to identify the parameters with the best predictive power.
Results: Our results show significant predictive parameters, both recently known and new ones. Investigated outcome parameters were the need for dialysis treatment, ventilation, death and the occurrence of neurological symptoms in general and seizures in particular. The best predictors for a poor outcome were the following parameters: patient's age and LDH, CRP as well as leukocyte count (each at the date of HUS diagnosis). Several other laboratory parameters were also predictor for a poor outcome in at least one of the aforementioned outcome parameters. Moreover, patients with the co-morbidities hypertension and diabetes mellitus had more often suffered more severe courses.
Conclusions: The expected severity of the clinical course of a patient diagnosed with HUS can be evaluated by multiple parameters, including but not limited to age, LDH, CRP and leukocyte count, each specific for some or many complications and conditions.

SAP179 BEST SUPPORTIVE CARE AND THERAPEUTIC PLASMA EXCHANGE

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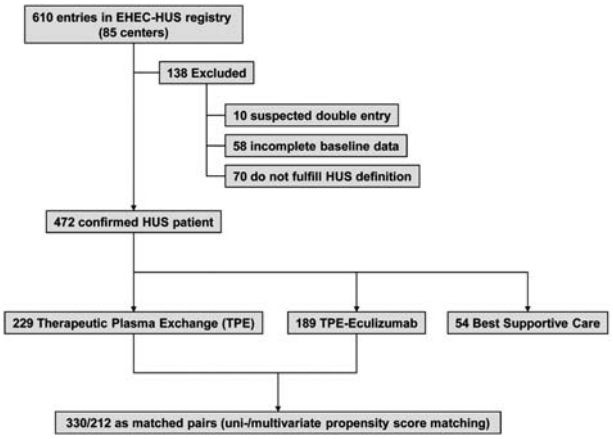
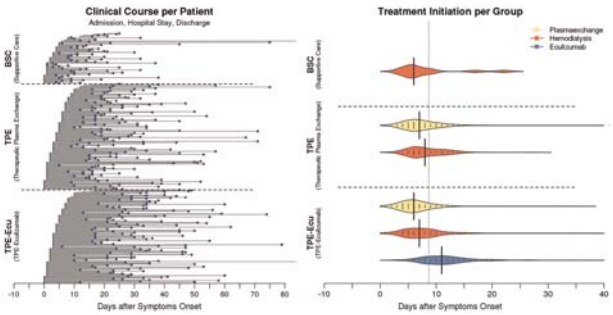
Introduction and Aims: May 22nd marks the beginning of a Shiga-toxin-producing E. coli (STEC) O104:H4 outbreak in Northern Germany. By its end on July 27th, it had claimed 52 deaths among 3,052 STEC and 733 confirmed HUS cases. Here we provide the first analysis of data of an online registry which had been created for



SAP179 Figure 1: Flowchart of the 610 patients enrolled in the STEC-HUS registry.



SAP179 Figure 2: Illustration of the clinical course and corresponding treatment initiation.

clinical outbreak surveillance by the German Society of Nephrology (DGfN). In this analysis we aim to describe short term effectiveness of: 1) best supportive care (BSC), 2) therapeutic plasma exchange (TPE), and 3) TPE with eculizumab (TPE-Ecu) in HUS.

Methods: On May 27th, five days after the outbreak had been officially reported, the DGfN set up a web-based STEC-HUS registry, which was continuously updated in the following weeks. This registry presents the largest population of HUS patients and provides analysis of essential epidemiologic and laboratory data, as well as clinical information from hospital admission to discharge. We compared the effectiveness of best supportive care (BSC), therapeutic plasma exchange (TPE) and TPE combined with Eculizumab (TPE-Ecu) with regard on renal, neurological, and survival outcome. To adjust imbalances in disease severity of treatment groups statistical models using a propensity score based method were applied.

Results: Of 610 patients treated in 83 hospitals in Germany, Sweden, and the Netherlands, 472 fulfilled the definition of HUS (median age 46 years; 72% females. Median (interquartile range) hospital stay was 22 (14-31) days. 270 (57%) patients underwent dialysis, 108 (23%) mechanical ventilation. 54 patients received BSC, 229 TPE and 189 TPE-Ecu. Disease severity (laboratory signs of hemolysis, need for dialysis, frequency of seizures) were lower in BSC than in TPE and TPE-Ecu patients. At hospital discharge, median creatinine was lower (1.1 mg/dL [0.9-1.3], $p<0.05$) in BSC than in TPE (1.2 mg/dL [1.0-1.5]) and TPE-Ecu (1.4 mg/dl [1.0-2.1]), while need for dialysis was not different between BSC (1,9%, n=1) , TPE (3.5 %; n=8) and TPE-Ecu (4.2%, n=8). At discharge, seizures were absent in BSC and rare (0.4 %; n=1) in TPE and TPE-Ecu (2.4 %; n=2) patients. Total hospital mortality was 4.2% (n=20) and did not differ significantly between TPE and TPE-Ecu-patients.

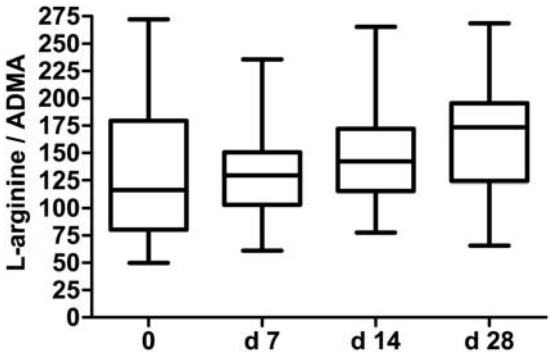
Conclusions: Despite frequent and severe renal impairment, neurological disorders and respiratory failure during STEC-HUS, short term outcome was benign. Within the limitations of a retrospective registry analysis, our data do not prove marked differences in treatment efficiency between the three groups. A randomized trial comparing BSC, TPE and Ecu seems to be prudent and necessary prior to establishing new treatment guidelines for STEC-HUS.

SAP180 CAMPYLOBACTER INFECTION PRESENTING WITH BLOODY DIARRHEA: SONOGRAPHIC DIFFERENTIATION FROM EHEC/STEC INFECTION DURING A LARGE OUTBREAK IN GERMANY

Ansgar Reising[1], Carsten Hafer[2], Jan Kielstein[3], Bernhard Schmidt[4], Franz-Christoph Bange[5] and Marcus Hiss[6]

SAP181 Table 1 Baseline characteristics of patients with STEC-HUS

	n= 50
Female, n (%)	36 (72)
Age (yrs)	44.9±1.,5
Body Mass Index (kg/m²)	25.6±3.8
Heart rate (bpm)	83.7±14.3
Temp. (°C)	37±0.5
Duration of diarrhea (days)	6.1±3.5
Haemolysis	
LDH (U/l)	1030±631
platelets (10^3/µl)	71±47
Hemoglobin (g/dl)	12.2±1.8
Renal parameters	
creatinine (µmol/l)	187.1±131.1
urea (mmol/l)	9.2±6.1
Additional values	
Leukocytes (Tsd/µl)	12.7±5.4
CRP (mg/dl)	66.4±72.6
AST (U/l)	75.7±73.1
ALT (U/l)	37.7±50.7
AP (U/l)	62.4±21.1



SAP181 Figure 1: Box and whisker plots show the L-arginine / ADMA ratio – a surrogate for NO production capacity from the day of hospital admission until day 28. Horizontal bars indicate median values, whiskers indicate 1.5 times the interquartile distance; one way ANOVA $p<0.0095$.

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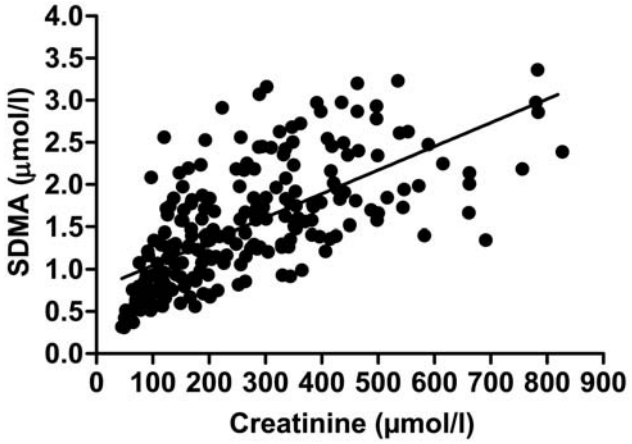
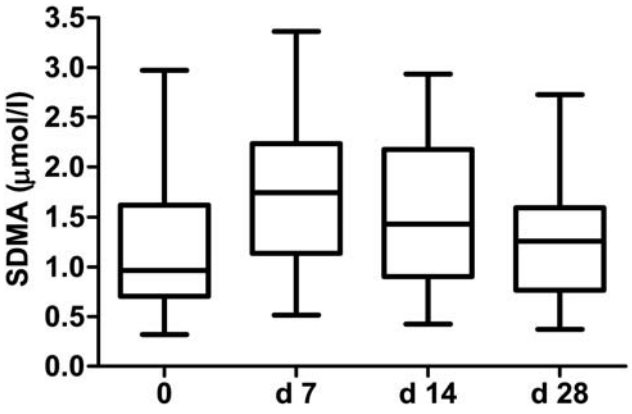
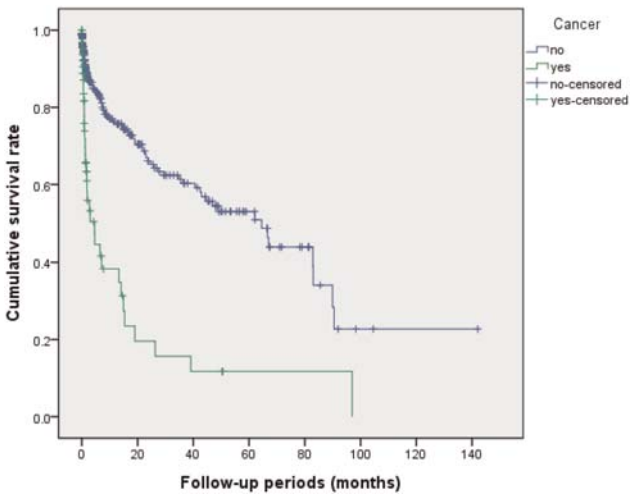
Introduction and Aims: Bloody diarrhea is a symptom often prompting hospital admission in intestinal infections caused by various pathogens such as Yersinia, Shigella, Campylobcater, and EHEC/STEC. During a large outbreak with EHEC/STEC in Germany in May 2011, patients presenting with bloody diarrhea were almost exlcusively considered to be infected with EHEC/STEC. These patients were were isolated, and antibiotic treatment was withheld until microbiological test results were available to avoid overwhelming shiga toxin release. As part of a routine work-up abdominal ultrasound,was performed to screen for sonographic signs of



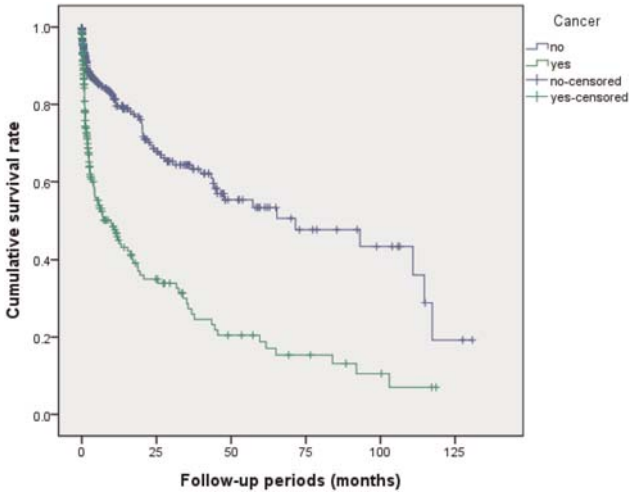


SAP181 Figure 3: Box and whisker plots show SDMA from the day of hospital admission until day 28. Horizontal bars indicate median values, whiskers indicate 1.5 times the interquartile distance; one way ANOVA p<0.0019.



SAP181bis Figure 1: Survival curve in cancer and non-cancer patients with DM

kidney, gut or other organ involvement.
Methods: During the ongoing outbreak we performed intestinal sonography in 29 patients with confirmed EHEC/STEC and a history of bloody diarrhea.



SAP181bis Figure 2: Survival curve in cancer and non-cancer patients without DM

SAP181bis Table 1 Main patient characteristics and outcomes

Variables	All patients	Non-cancer patients	Cancer patients	P-value
	(n = 2211)	(n = 1360, 61.5%)	(n = 851, 38.5%)	
Age (years)	61.1 ± 14.1	61.4 ± 15.3	60.7 ± 12.1	0.221
Male gender	1356 (61.3%)	789 (58%)	567 (66.6%)	0.001
Hospital mortality	671 (30.3%)	306 (22.5%)	365 (42.8%)	0.001
Conventional hemodialysis	241 (10.9%)	165 (12.1%)	76 (8.9%)	0.011
DM	833 (37.7%)	687 (50.5%)	146 (17.1%)	0.001
Sepsis	687 (31.1%)	442 (32.5%)	245 (28.7%)	0.001
Type of cancer				
Locoregional solid tumor	189 (8.5%)		189 (22.2%)	NA
Metastatic solid tumor	604 (27.4%)		604 (71.0%)	
Hematological malignancy	58 (2.6%)		58 (6.8%)	
Hospital mortality	671 (30.3%)	306 (22.5%)	365 (42.8%)	0.001
ICU admission	523 (23.7%)	385 (28.3%)	138 (16.2%)	0.001
ICU mortality	340 (65.0%)	230 (59.7%)	110 (79.9%)	0.001
Inotropics	607 (27.4%)	441 (30.2%)	166 (19.5%)	0.001
Mechanical ventilator	480 (21.7%)	385 (28.3%)	95 (11.2%)	0.001
Chronic hepatitis B	211 (9.5%)	93 (6.8%)	118 (13.8%)	0.001
Chronic hepatitis C	185 (8.4%)	114 (8.4%)	71 (8.3%)	0.512
Cause of death				
cancer	234 (34.9%)	NA	225 (61.7%)	
cerebrovascular disease	58 (8.6%)	52 (17.0%)	7 (2.0%)	0.001
heart disease	49 (7.3%)	50 (16.3%)	4 (1.1%)	0.001
DM	1 (0.2%)	1 (0.4%)	0	0.001
infection	273 (40.8%)	160 (52.0%)	118 (32.0%)	0.001
liver diease	93 (7.7%)	40 (13.2%)	11 (3.2%)	0.001
hypertensive disease	3 (0.5%)	3 (1.1%)	0	0.001
Cancer status				
controlled	143 (6.4%)	NA	143 (16.8%)	
uncontrolled/newly diagnosed	81 (3.6%)	NA	81 (9.5%)	
uncontrolled/recurrence,progression	627 (28.3%)	NA	627 (73.8%)	

SAP181bis Table 2 Clinical and laboratory data related to kidney function

Variables	All patients	Non-cancer patients	Cancer patients	P-value
	(n = 2211)	(n = 1360, 61.5%)	(n = 851, 38.5%)	
Chronic kidney disease	445 (20.1%)	340 (25%)	88 (10.4%)	0.001
DM as cause of CKD	232 (52.2%)	197 (58.1%)	35 (39.7%)	0.001
Etiology of AKI				
Sepsis	687 (31.1%)	408 (30.0%)	282 (33.2%)	0.001
Ischemia or shock	1165 (52.7%)	816 (60.0%)	324 (38.1%)	0.001
radiocontrast or nephrotoxin	179 (8.1%)	73 (5.4%)	114 (13.4%)	0.001
hemolysis or rhabdomyolysis	51 (2.3%)	41 (3%)	9 (1.0%)	0.001
urinary tract obstruction	113 (5.1%)	19 (1.4%)	105 (12.4%)	0.001
multiple meyloma	7 (0.3%)	3 (0.2%)	5 (0.6%)	0.001
tumor lysis syndrome	9 (0.4%)	0 (0%)	12 (1.3%)	0.001
Laboratory data at diagnosis of AKI				
Creatinine (mg/dL)	1.4 ± 1.3	1.6 ± 1.0	1.3 ± 0.9	0.001
Urea (mg/dL)	22 ± 16	23 ± 18	20 ± 13	0.001
Sodium (mEq/L)	138.0 ± 4.8	139.0 ± 4.7	136.9 ± 4.8	0.001
Potassium (mEq/L)	4.3 ± 0.6	4.3 ± 0.7	4.3 ± 0.6	0.700
Phosphorus (mg/dL)	3.7 ± 2.3	3.8 ± 3.2	3.6 ± 0.9	0.184
pH	7.42 ± 0.05	7.42 ± 0.06	7.43 ± 0.05	0.080
Bicarbonate (mEq/L)	22.4 ± 4.5	22.4 ± 4.9	22.3 ± 4.1	0.689

SAP181bis Table 5 Multivariate Cox proportional hazards analysis of mortality in all patients (n = 2211)

Variables	Unit increase	Hazard ratio (95% CI)	P-value
CKD	vs. Absence	0.749 (0.547-1.024)	0.070
DM	vs. Absence	1.463 (1.194-1.604)	0.047
Conventional HD	vs. Absence	0.901 (0.638-1.272)	0.554
Mechanical ventilation	vs. Absence	0.952 (0.725-1.250)	0.724
Cancer	vs. Absence	3.010 (2.340-3.873)	0.001
Sex	vs. Female	1.129 (0.895-1.423)	0.303
Sepsis	vs. Absence	1.000 (0.787-1.270)	0.998
Age	vs. ≤ 64 yrears	1.038 (0.829-1.298)	0.998

SAP181bis Table 3 Cancer site Site

A

Cancer	N
Acute leukemia	12 (1.4%)
lymphoma	23 (2.7%)
Myeloma	23 (2.7%)
Solid tumors	788 (92.6%)
miscellaneous malignancies	5 (0.6%)
Total	851 (100%)

B

Site	N
prostate	9 (1.1%)
urinary bladder	20 (2.4%)
kidney	18 (2.1%)
ovary	27 (3.2%)
corpus uteri	8 (1.0%)
cervix uteri	53 (6.3%)
lymphoma	23 (2.7%)
leukemia	12 (1.4%)
multiple myeloma	23 (2.7%)
colon	30 (3.5%)
rectum	34 (4.0%)
stomach	153 (18.0%)
esophagus	3 (0.4%)
liver	167 (19.7%)
gall bladder/biliary tract	99 (11.5%)
pancreas	30 (3.6%)
breast	6 (0.7%)
lung/bronchus/trachea	97 (11.2%)
brain/CNS	3 (0.3%)
malignant melanoma	2 (0.2%)
other sites	34 (4.0%)
Total	851 (100%)

Results: In 16 out of 29 patients we found sonographic signs of colitis, as mild to pronounced thickening of the colonic wall. In those with severe involvement of the gut - complicated due to additional hemolytic uremic syndrome – loss of layering or reduced echo-genicity of colonic wall and mesenteric edema was seen to some extent. None of of the patients showed mesenterial lymphadenopathy. Here we present a case of a 24 year old female with bloody diarrhea, fever, and a travel history to Spain, who was suspected to suffer from EHEC/STEC. While awaiting microbiological results, abdominal ultrasound was performed. The examination was showed a slightly thickened wall of colon ascendens with an expanded highly echogenic submucosa, preserved layering and regular motility. Furthermore we found a mesenterial lymphadenopathy in the ileocecal region. This was in striking contrast to the findings in the EHEC/STEC cohort, which lead us to conclude that this patient does not have EHEC/STEC infection. Due to signs of a systemic infection we immediately started antibiotic treatment with a carbapenem (ertapenem). The patient improved rapidly and 48 hours later Campylobacter was isolated from stool cultures, that were on the day of admission.

Conclusions: Thus the ultrasound findings with mesenteric lymphadenopathy accompanying a thickened colonic wall with preserved layering and an expanded highly echogenic submucosa, together with an accurate medical history allowed us to rule out EHEC/STEC infection rapidly.

SAP181bis Table 6 Multivariate Cox proportional hazards analysis of mortality in cancer (n =851)

Variables	Unit increase	Hazard ratio (95% CI)	P-value
CKD	vs. Absence	0.934 (0.503-1.736)	0.830
DM	vs. Absence	1.497 (1.008-2.221)	0.045
Conventional HD	vs. Absence	0.790 (0.462-1.351)	0.390
Mechanical ventilation	vs. Absence	1.179 (0.740-1.878)	0.486
Sex	vs. Female	1.088 (0.754-1.570)	0.652
Sepsis	vs. Absence	0.890 (0.630-1.256)	0.507
Age	vs. ≤ 64 yrears	0.951 (0.675-1.339)	0.773

SAP181bis Table 7 Multivariate Cox proportional hazards analysis of mortality in non-cancer (n =1360)

Variables	Unit increase	Hazard ratio (95% CI)	P-value
CKD	vs. Absence	0.676 (0.470-1.272)	0.075
DM	vs. Absence	1.149 (1.154-1.546)	0.032
Conventional HD	vs. Absence	0.963 (0.612-1.514)	0.870
Mechanical ventilation	vs. Absence	0.839 (0.599-1.176)	0.309
Sex	vs. Female	1.201 (0.882-1.635)	0.244
Sepsis	vs. Absence	1.123 (0.804-1.569)	0.495
Age	vs. ≤ 64 years	1.109 (0.816-1.508)	0.507

SAP135 Table.1 The levels of biologic markers in BALF

Variables	non-AKI (n=28)		AKI (n=24)		p value
Activated Inflammatory cells					
Neutrophil, $*10^4/mm^3$	2.01	(0.27-8.45)	5.48	(1.51-18.63)	0.0017
macrophage , $*10^4/mm^3$	8.50	(3.93-16.99)	11.62	(7.15-17.16)	0.2120
Inflammatory Mediators					
TNF-α, pg/ml	2.03	(1.02-4.36)	4.79	(3.06-16.38)	0.0107
IL-1β, pg/ml	1.72	(0.80-2.01)	3.16	(0.80-7.66)	0.0311
IL-6, pg/ml	501.6	(80.0-3098.8)	4132.0	(1034.2-8878.0)	0.0012
IL-8, pg/ml	230.1	(121.1-685.0)	782.9	(372.5-2481.8)	0.0020
IL-18, pg/ml	156.6	(84.2-413.3)	280.4	(203.5-474.2)	0.0207
INF-γ, pg/ml	3.97	(2.01-13.75)	9.65	(6.37-15.19)	0.0107
MCP-1, pg/ml	827.7	(418.7-1805.8)	3196.0	(1171.6-8154.8)	0.0009
Inhibitors of Inflammatory Mediators					
IL-10, pg/ml	0.44	(0.44-0.44)	0.44	(0.44-1.34)	0.0236
Expression of activated adhesion molecules					
sICAM-1, ng/ml	76.0	(51.1-171.0)	141.5	(89.5-288.3)	0.0495
Endothelial injury					
TM, U/ml	3.83	(1.05-5.73)	4.36	(2.68-9.03)	0.0069
Dysregulated coagulation					
PAI-1, ng/ml	5.00	(5.00-17.75)	25.50	(7.00-41.25)	0.0052
Epithelial injury					
SP-D, ng/ml	617.5	(293.3-1585.0)	868.0	(493.8-1900.0)	0.2829
Increased capillary permeability					
Alb, mg/dl	41.2	(13.2-142.0)	67.0	(21.6-152.8)	0.0611

SAP181 **MARKED AND PROLONGED DECREASE IN THE NO PRODUCTION CAPACITY IN PATIENTS WITH STEC-HUS INDICATED BY A LOW L-ARGININE/ADMA RATIO**

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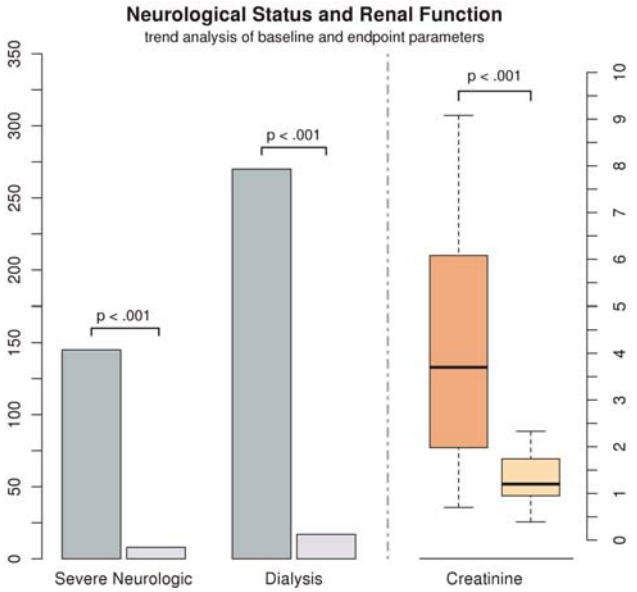

SAP179 Figure 3: Comparison of patients affected by severe neurological symptoms (seizure and come) during hospital stay and at the time of discharge. Cumulative frequency of patients requiring dialysis as well as maximum creatinine during hospital stay compared to discharge.

Introduction and Aims: The endogenous nitric oxide synthase (NOS) inhibitor asymmetric dimethylarginine ADMA) is a strong inhibitor of NOS. Elevated ADMA levels have been shown to lead to the reduction in blood flow in the kidney and brain. Acute hemolysis increases ADMA and lowers the substrate for NOS, i.e. L-arginine. Hence lowering of the L-arginine/ADMA ratio could contribute to the thrombotic microangiopathy in STEC-HUS. Aim of our study was to investigate the role ADMA and L-arginine levels in patients with STEC-HUS at different time points during the course of the disease. Moreover we aimed to investigate the structural isomer symmetrical dimethylarginine (SDMA), has been shown to be an excellent marker of renal function.
Methods: Fifty patients with STEC-HUS whose baseline characteristics is presented in Table underwent blood draw at the time of hospital admission as well 7, 14 and 28 days thereafter. Serum ADMA, L-arginine, and SDMA were measured by liquid chromatography–mass spectrometry method.
Results: During the active phase of HUS the L-Arginine/ADMA ratio, a surrogate of NO production capacity was markedly reduced but improved in parallel to the clinical recovery of the patients (Figure 1).The L-arginine / ADMA ratio correlated inversely with LDH (Spearman r -0.35 , p<0.001). The structural isomer of ADMA, i. e. SDMA correlated well (Spearman r 0.7267, p<0.001) with serum creatinine (Figure 2) and changes were related to the change in renal function during the hospital course (Figure 3).
Conclusions: The prolonged and marked reduction in the L-arginine/ADMA ratio in STEC-HUS might play an important role in the pathogenesis of thrombotic microangiopathy. As in other diseases, SDMA in STEC-HUS is associated with renal function.

SAP181bis OUTCOMES OF ACUTE KIDNEY INJURY PATIENTS WITH AND WITHOUT CANCER

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Introduction and Aims: Few studies have examined the characteristics and outcomes of acute kidney injury (AKI) patients with and without cancer.
Methods: We conducted a retrospective cohort study in a South Korean tertiary care hospital. A total of 2211 consecutive patients (without cancer 61.5%; with cancer 38.5%) were included over a 140-month period. Predictors of all-cause death were examined using the Kaplan-Meier method and the Cox proportional hazards model.
Results: The main contributing factors of AKI were sepsis (31.1%) and ischemia (52.7%). AKI was multifactorial in 78% of patients with cancer and in 71% of patients without cancer. Hospital mortality rates were higher in patients with cancer (42.8%) than in patients without cancer (22.5%) (P = 0.014). In multivariate analyses, diabetes mellitus (DM) and cancer diagnosis were associated with hospital mortality. Cancer diagnosis was independently associated with mortality [odds ratio = 3.010 (95% confidence interval, 2.340-3.873), P = 0.001]. Kaplan-Meier analysis revealed that subjects with DM and cancer (n = 146) had lower survival rates than subjects with DM and without cancer (n = 687) (log rank test, P = 0.001).
Conclusions: The presence of DM and cancer were independently associated with mortality in patients both with and without cancer.