

day before and on the day of CM administration; 2- Carnitine group (n = 10), where the patients were infused with 20 mg/kg carnitine over 10 minutes 2 h prior to the CM administration and 24 hours post CT; 3- Phosphodiesterase type 5 inhibitor group (n = 12), where patients were given orally 20 mg tablets of PDE5 inhibitor-Tadalafil 2 h prior to the administration of the CM and in the subsequent day. Urine and blood samples were collected before and at the following time sequence: 2, 6, 12, 24, 48, 120 hours after the contrast administration, for creatinine and NGAL determination. **Results:** Administration of CM to CKD patients who were pretreated with Acetyl cysteine caused a significant increase in urinary NGAL, but not of plasma NGAL and SCr. In contrast, pretreatment with carnitine prior to CM prevented the increase in urinary NGAL throughout the follow up period and reduced SCr below basal levels. Similarly, tadalafil administration attenuated the elevation in CM-induced urinary NGAL, but did not affect neither plasma NGAL nor Scr. **Conclusions:** These results suggest that carnitine and PDE-5 inhibition may comprise novel nephroprotective approaches against CIN.

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### COMMUNITY-ACQUIRED ACUTE KIDNEY INJURY IS ASSOCIATED WITH CARDIOHEPATIC SYNDROME IN DECOMPENSATED HEART FAILURE

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**Introduction and Aims:** Liver and kidney dysfunction is frequently encountered in heart failure and related to worse prognosis. Over the last several years interdependent feedback mechanisms involving the heart, kidney and liver have been discussed. The aim of the study was to assess the prevalence of acute kidney injury (AKI) and abnormal liver function tests (LFTs) and their interrelations in acute decompensated heart failure (ADHF).

**Methods:** In 322 patients with ADHF (190 male, 69.5±10.7 years (M±SD), arterial hypertension 87%, myocardial infarction 56.5%, atrial fibrillation 65.5%, diabetes mellitus 41.6%, known chronic kidney disease 39.1%, chronic anemia 29.2%, chronic obstructive lung disease 32.2%, ejection fraction (EF) 37.6±12.6%, EF <35% 39.1%) alanine transaminase (ALT), aspartate transaminase (AST), direct and total bilirubin (DB and TB), alkaline phosphatase (AP), gamma-glutamyl transpeptidase (GGT) and international normalized ratio (INR) were measured on admission. LFTs were considered abnormal when levels exceeded local upper normal limit. Patients on warfarin were excluded from INR analysis. AKI was diagnosed based on KDIGO 2012 Guidelines. Mann-Whitney test and multivariate logistic regression analysis were performed, p<0.05 was considered statistically significant.

**Results:** Abnormal LFTs occurred in 274 (85.1%) patients. Increase of transaminases were detected in 68 (21.1%) patients (alone ALT/ alone AST/ both TA in 35.3, 26.5, 38.2% respectively), DB and/or TB in 264 (82%) patients (alone DB/ alone TB/ DB and TB - in 28, 0.8, 71.2% respectively), AP in 90 (27.9%) and GGT in 102 (31.7%) patients. Mean value ALT was 34.9±46.5 U/L, AST 34.6±25.7 U/L, DB 9.6±7.1 μmol/L, TB 26.3 ±14.5 μmol/L, GGT 115.6±85.9 U/L, AP 113.9±85.4 U/L, INR 1.33±0.29. Community-acquired AKI (CA-AKI) was diagnosed in 60 (18.6%) patients. Patients with versus without CA-AKI had higher levels of ALT (60±88 vs 29±26 U/L, p<0.05), AST (52±45 vs 31±16 U/L, p<0.001), TB (29±13 vs 25±15 μmol/L, p<0.01), DB (12±7 vs 9±7 μmol/L, p<0.001), GGT (157±117 vs 102±68 U/L, p<0.001), AP (124±74 vs 112±88 U/L, p<0.05), INR (1.49±0.42 vs 1.29±0.23, p<0.01). Patients with vs without AKI had higher prevalence of increase of ALT (30 vs 12.3%, p<0.001), AST (33.3 vs 9.2%, p<0.001), TB (73.3 vs 56.2%, p<0.05) and INR (60.8% vs 43.8%, p<0.05). AKI was predictor for increase of ALT (odds ratio (OR) 3.1, 95% confidence interval (CI) 1.6-5.9), AST (OR 4.9, CI 2.5-9.7), TB (OR 2.1, CI 1.2-4.0) and INR (OR 2.0, CI 1.0-3.9).

**Conclusions:** CA-AKI occurred in 18.6%, abnormal LFTs - in 85.1% of patients admitted with ADHF. Patients with versus without AKI had higher prevalence of abnormal LFTs. In patients with ADHF increase of transaminases, total bilirubin and INR can directly contribute to AKI and vice versa.

MP200

### THE IMPACT OF ACUTE KIDNEY INJURY ON IN-HOSPITAL MORTALITY IN ACUTE ISCHEMIC STROKE PATIENTS UNDERGOING INTRAVENOUS THROMBOLYSIS

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**Introduction and Aims:** Intravenous thrombolytic therapy (iv. rt-PA) seems to be the most effective treatment for the acute ischemic stroke (AIS). In the same time, acute kidney injury (AKI) continues to contribute significantly to morbidity and mortality in a variety of critical care settings. Nevertheless, the impact of AKI on iv. rt-PA for AIS has not been evaluated. This study aimed to investigate whether AKI impacts on in-hospital mortality in AIS patients undergoing iv. rt-PA.

**Methods:** This prospective observational study included 45 patients (median age = 64 years; 29 (64.44%) males) who consecutively underwent iv. rt-PA for AIS in a tertiary academic hospital from January 2013 to January 2015. Control group consisted of 59 AIS patients (median age = 64 years, 29 (49.15% males) which were not eligible for iv. rt-PA because onset-to-door time was longer (>4.5h) than the therapeutic window-time. Subjects have been followed-up until hospital release or death (median follow up time = 12 days). AKI was defined according to KDIGO AKI Guidelines. We assessed the incidence, predictors and effect of AKI on in hospital mortality. Intravenous thrombolytic therapy was used according to the current guidelines. The study was approved by the hospital ethics committee. All patients have been divided into two groups: (1) those with AKI and (2) those without AKI.

**Results:** The prevalence of AKI in the group treated with iv. rt-PA was 35.5% (16 cases) being similar to that observed in the not thrombolysis group - 33.89% (20 cases) (p = 0.86). The development of AKI was associated with increased baseline average serum creatinine (1.2 vs. 0.8 mg/dL; p = 0.028) and uric acid (6.7 vs. 5.0 mg/dl; p < 0.001) respectively, decreased baseline average eGFR (69.6 vs. 90.0 mL/min; p = 0.007). The occurrence of AKI was associated with an increased incidence of in-hospital mortality, both in the group treated with iv. rt-PA (50.0% vs. 3.4%; p < 0.001), and in the group who did not receive iv. rt-PA (45% vs. 25.6%). In the AKI patients, in-hospital mortality risk did not differ between the two groups (thrombolysis vs. not thrombolysis patients) - OR = 1.22, 95% CI [0.32 to 4.56], p = 0.76. In patients without AKI, the risk for in-hospital mortality was significantly lower in the iv. rt-PA group - OR = 0.1, 95% CI [0.01 to 0.86], p = 0.01. After multivariable adjustment, AKI still remained an independent predictor of in-hospital mortality in AIS patients treated with iv. rt-PA (HR=15.2; p = 0.011). In the logistic regression analysis, independent predictors for AKI developed were eGFR at admission, body mass index and hematocrit (model explained 59.9% of the development of AKI, Nagelkerke R<sup>2</sup>=0.599).

**Conclusions:** Our study indicates that AKI is a common finding and an independent predictor of in-hospital mortality in AIS treated with iv. rt-PA. In addition, this study demonstrates that iv. rt-PA therapy is associated with a better in-hospital survival rate only in patients without AKI.

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### URINARY TISSUE INHIBITOR METALLOPROTEINASE-2 (TIMP-2) AND IGF-BINDING PROTEIN-7 (IGFBP7) LEVELS ARE ASSOCIATED WITH PERSISTENT OLIGURIA IN ACUTE KIDNEY INJURY: PRELIMINARY RESULTS

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**Introduction and Aims:** Acute kidney Injury (AKI) occurs in more than half of Intensive care Unit (ICU) patients and is associated with increased mortality. Nephrocheck® is a new diagnostic test used to detect early kidney injury by fluorescent immunoassay of two biomarkers associated with kidney injury (TIMP-2 and IGFBP-7) involved in cell cycle arrest to be measured within 20 minutes. Higher scores allow to recognize patients at greater risk for developing AKI in the next 12-24 hours. Recent studies define minimum risk points (NDT 2014; 29:2054-61) and they are useful in cardiac surgery and sepsis. Aim: Analyse the potential clinical usefulness of a single assessment by Nephrocheck in ICU patients with AKI.

**Methods:** Forty - seven Acute kidney Injury patients (62 years old SD: 14.3)) were included according to AKI (Acute Kidney Injury) criteria in Nephrocheck investigation test. Data compiled: Diabetes, types of Acute kidney Injury, AKI stage, urine output, use of diuretics, requirement of continuous renal replacement therapy (CRRT), serum creatinine at baseline, at the moment of AKI and at discharged, length of hospital stay and death. According to the Nephrocheck investigation test, results were divided in three stages: Low risk : less than 0.3, Intermediate risk: 0.3 -1.5 and High risk : more than 1.5. (ns: non significant).

**Results:** Table 1 shows comparative results of analyzed variables by Nephrocheck investigation test ranges (Data expressed as average (standard deviation)). The high risk patients as opposed to the low risk ones had more baseline oliguria in the next 48h and diuresis took more time to recover (p <0.05). The groups were comparable in basal characteristics and there were no differences between them in the rest of the variables analyzed (renal function, length of hospital stay and death).

**Conclusions:** The Nephrocheck assessment not only predicts the level of risk but also informs about the intensity and persistence of oliguria. By confirming this result in more patients guaranteed the cut - off point, which could facilitate taking clinical