Abstract: **P4231**

Cardiorenohepatic syndrome reflects severity of decompensated heart failure and is related with worse prognosis

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Objective: Similar factors such as venous congestion and hypoperfusion are thought to underlie both renal and liver injuries in decompensated heart failure (DHF), known as cardiorenal syndrome (CRS) and cardiohepatic syndrome (CHS). Traditionally CRS and CHS have been investigated in isolation, but several organs damage can be a marker of a more severe course of AHF. The aim of this study was to assess the prevalence and predictors of combination of CRS and CHS in DHF.

Methods: Kidney and liver function was assessed in 322 patients with DHF (190 male, 69.5±10.6 years (M±SD), arterial hypertension 87%, myocardial infarction 57%, atrial fibrillation 65%, diabetes mellitus 42%, known chronic kidney disease 39%, chronic anemia 29%, left ventricular (LV) ejection fraction (EF) 37.6±12.6%, EF<35% 39.1%). CRS was diagnosed if serum creatinine decreased ≥26.5 μmol/l in first 48 hours of hospitalization. CHS was considered when at least one of liver function tests (LFT) level exceeded upper normal limit on admission. Simultaneous CHS and CRS were considered as cardiorenohepatic syndrome (CRHS). Mann-Whitney test and multivariate logistic regression analysis were performed. P<0.05 was considered statistically significant.

Results: CRS occurred in 60 (18,6%) patients. CHS was diagnosed in 274 (85.1%) patients. Isolated CHS, isolated CRS and CRHS occurred in 78.4, 1.5 and 20.1% patients with hepatic or kidney injury.

Patients with versus without CRHS had lower systolic blood pressure (SBP) (130±18 vs 138±19 mmHg, p<0.01), EF (32±10 vs 38±13%, p<0.01), pulse BP (49±16 vs 56±15 mmHg, p<0.01), higher LV mass index (200±50 vs 178±52 g/m^2, p<0.01), LV end diastolic volume (62±6 vs 56±9 mm, p<0.001), higher incidence of severe mitral regurgitation (64.3 vs 39.6%, p<0.001), signs of congestion – jugular venous distension (57.1 vs 39.6%, p<0.05), hepatomegaly (85.7 vs 70.3%, p<0.05), echo-hydropericardium (46.4 vs 22.5%, p<0.001), vasopressor therapy (17.9 and 6.3%, p<0,01).

The independent predictors of CRHS were baseline GFR<45 ml/min/1.73 m^2 (odds ratio (OR) 3.95, 95% confidential interval (CI) 2.15–7.21, p<0.01), SBP<110 mmHg on admission (OR 3.51, CI 1.55–7.94, p<0.05), vasopressor therapy (OR 3.23, CI 1.35–7.73, p<0.05), echo-hydropericardium (OR 2.98, CI 1.62–5.50, p<0.01) and EF<35% (OR 2.96, CI 1.61–5.44, p<0.05).

Patient with vs without CHRS had worse prognosis: longer length of stay (15.7±6.5 vs 13.5±4.8 days, p<0.05) and trend of increased all-cause death rate in 6 months (31.8 vs 25%, p=0.07).

Conclusions: CRHS occurred in 20.1% of patients with DHF and hepatic or kidney injury and is related with negative prognosis. The independent predictors of CRHS were baseline GFR<45 ml/min/1.73 m^2, SBP<110 mm Hg on admission, echo-hydropericardium and EF<35%.