Abstract: **P3400**

# Mixed phenotype of cardiohepatic syndrome is associated with negative prognosis in decompensated heart failure

**Authors:**
A. Soloveva[1], S. Villevalde[1], Z. Kobalava[1], [1]RUDN University - Moscow - Russian Federation,

**Topic(s):**
Heart failure, other

**Citation:**
European Heart Journal ( 2017 ) 38 ( Supplement ), 707

**Objective:** Abnormal liver function tests (LFT) are associated with specific clinical, biological and prognostic features in decompensated heart failure (DHF) and known as cardiohepatic syndrome (CHS). The aim of this study was to assess the prevalence, phenotypes and prognosis CHS in DHF.

**Methods:** In 322 patients with ADHF (190 male, 69.5±10.6 years (M±SD), arterial hypertension 87%, myocardial infarction 57%, atrial fibrillation 65%, diabetes mellitus 42%, chronic kidney disease 39%, chronic anemia 29%, left ventricular 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) were measured on admission. CHS was considered when at least one LFT level exceeded upper normal limit. Only ALT and/or AST increase was considered as hepatocellular CHS, isolated increase of GGT, AP, DB and TB (with DB increase) – as cholestatic CHS, the simultaneous increase of markers of cytolysis and cholestasis – as mixed CHS. Multivariate logistic regression analysis was performed.P<0.05 was considered significant.

**Results:** CHS occurred in 274 (85.1%) of patients with DHF. Increase of ALT was detected in 50 (15.5%), AST in 46 (14.3%), DB in 262 (81.4%), TB in 192 (59.6%), AP in 90 (27.9%) and GGT in 102 (31.7%) of patients. Most of LFT elevations were moderate (≤3UNL): ALT in 38 (76%), AST in 42 (91.3%), DB in 150 (57.3%), TB in 186 (96.9%), AP in 86 (96.7%), GGT in 74 (72.5%) of alterations cases.

Hepatocellular, cholestatic and mixed CHS were detected in 0.4, 32.8 and 66.8% of patients with DHF and CHS. Patients with mixed vs cholestatic CHS had higher levels of AST (median 32 (interquartile range 23; 49) vs 21 (18; 27)U/l), ALT (30 (15; 53) vs 17 (12; 25)U/l), DB (12 (7; 17) vs 6 (4; 9)µmol/l,) and TB (33 (25; 41) vs 19 (15; 22)µmol/l), p<0.001 for all comparisons.

Patients with mixed vs cholestatic CHS had higher cholestatic markers increase incidence (DB (97 vs 93%, p<0.05), TB (90 vs 31%, p<0.001), GGT (44 vs 24%, p<0.01), AP (39 vs 20%, p<0.01)) and severity: incidence of increase >2UNL of DB (82 vs 40.5%, p<0.01), TB (24.4 vs 0%, p<0.01), GGT (60 vs 54.6%, p>0.05) and AP (18 vs 11%, p<0.01).

Patients with mixed vs cholestatic CHS had higher NT-proBNP level (6645 (3608; 12000) vs 3793 (3100; 9000) pg/ml, p<0.05), incidence of EF<35% (47 vs 36%, p<0.05), severe mitral regurgitation (51 vs 31%, p<0.01), vasopressor therapy (11 vs 4%, p<0.05), lower systolic blood pressure (SBP) (132±17 vs 144±21 mmHg, p<0.001) and pulse BP (51±14 vs 60±15 mmHg, p<0.001) on admission.No significant differences in signs of congestion were observed between groups.

Mixed CHS was associated with higher all-cause death in 6 months (30 vs 23%, p<0.05).

**Conclusions:** Patients with mixed vs cholestatic CHS had higher LFT and NT-proBNP levels, incidence of LFT increase, severe mitral regurgitation, EF<35%, vasopressor therapy, lower SBP and pulse BP on admission and had worse prognosis.