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Welcome address

Dear friends and colleagues,

On behalf of the board of the Heart Failure Association and the organising committee, it is our great pleasure to welcome you to our annual congress Heart Failure 2017 & World Congress on Acute Heart Failure.

"Rendez-vous with the future" is the theme of this year's meeting of the world's leading heart failure association. Our congress is an essential platform for scientific exchange and networking with invaluable contributions of experts from around the world.

The scientific programme will provide the latest updates from the field of heart failure and recommendations for implementation in daily practice. Original scientific research is the core of the congress and we are proud to report the submission of a record-breaking 2 197 abstracts & clinical cases.

In addition, we are honoured that our esteemed colleagues Prof. Michel Komajda and Prof. Faiez Zannad will be awarded HFA Lifetime Achievement Awards and present the Eugene Braunwald and Philip Poole-Wilson Lectures during the Inaugural session.

On Sunday, join us for the HoT Walk organised by the Heart Failure specialist of Tomorrow to raise the public awarness on heart failure.

We look forward to welcoming you to beautiful Paris - the "City of Lights".

Alexandre Mebazaa
Heart Failure 2017 Chairperson

Frank Ruschitzka
HFA President

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determine the prognosis of patients becomes urgent. Purpose. To estimate the prevalence of renal dysfunction and its prognostic value in patients with AHF.
Materials and methods. The study included 141 patients (mean age 57.4 ± 9.86 years, 75.2% men), admitted to the hospital due to the development of symptoms of AHF, left ventricular ejection fraction of 15% to 80% (mean 37.8 ± 14.19%). Indicators of renal function were divided into 6 categories according to the estimated glomerular filtration rate (GFR) using a calculation formula CKD-EPI. Acute kidney injury (AKI) diagnosed according to the KDIGO recommendations (2012). The primary endpoint was defined as rehospitalization due to symptoms of AHF or death from cardiovascular causes. Result. Analyzed the frequency of occurrence of the endpoint, depending on the level of GFR and microalbuminuria (MA). With the decline of GFR levels increased in direct proportion to the frequency of rehospitalization due to AHF, so if GFR greater than 90 ml/min it amounted to 37.5%, 60-89 ml/min – 33.9%, while the GFR 45-59 ml/min – 48.9%, 15-44 ml/min – 78.6%, less than 15 ml/min - 100% (p = 0.027). Reduced GFR of less than 45 ml/min was associated with an increase in death from cardiovascular causes - 43.8%, while in patients with GFR over 45 ml/min death from cardiovascular causes registered with 12,8% (p = 0.005). Increased MA to levels of over 300 mg/l was associated with a poor prognosis, the endpoint and death from cardiovascular causes was reported in 81.8% and 36.4% of patients, and in patients without MA – 39.8% and 12.0% respectively (p = 0.012 and p = 0.036). Analyzed OR of the primary endpoint of the depending on the level of GFR and MA. GFR of less than 60 ml/min increases the risk of an endpoint of 2.5 times (OR 95% 2.541 (1.284-5.028), p = 0.007), and GFR less than 45 ml/min - 10 times (OR 95% 10.157 (2.213-46.622), p = 0.003). GFR of less than 45 ml/min increases the risk of death from cardiovascular causes of 5 times (OR 95% 5.299 (1.732-16.214), p = 0.003). The presence of MA over 300 mg/l increases the risk of the primary endpoint in 6 times (OR 95% 5.946 (1.236-28.611), p = 0.026). Development AKI associated with an increase of the frequency of the primary endpoint as compared to patients without AKI, 85.7% and 41.7% respectively (p = 0.003). The frequency of death in patients with AKI also increased to 57.1%, whereas in patients without AKI – 11.8% (p < 0.001). The AKI increases the risk of the primary endpoint in 8 times (OR 95% 8.377 (1.8-38.996), p = 0.007) and the risk of death from cardiovascular causes of 10 times (OR 95% 9.956 (3.035-32.652), p < 0.001). Conclusion. Reduced GFR and MA is important predictors of cardiovascular morbidity and mortality in patients with AHF.

P412
The interaction between heart failure and renal failure

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Introduction: Heart failure (HF) is an important public health problem whose incidence and prevalence are progressively increasing. Multiple comorbid conditions are common in patients with HF. Renal failure (RF) is one of the important comorbid condition that shares common cardiovascular risk factors with HF. The interaction between HF and RF has been suggested to cause a deterioration of each of these conditions. Moreover, the interaction between these conditions has been suggested to form a vicious cycle, termed cardio-renal syndrome, which increases mortality.
Aim: Our aim was to investigate the prevalence, the clinical characteristics of HF patients with different deg rees of renal dysfunction
Methods: This is a retrospective study of all HF patients registered in the therapeutic Unit of Chronic Heart failure in the cardiology department over a period of 2 years. Renal function was determined from the estimated glomerular filtration rate (GFR), and patients were divided into 4 stages : GFR 60-89(Mildly reduced kidney function), GFR 30-59 (Moderately reduced kidney function), GFR 15-29 (Severely reduced kidney function) and GFR < 15 mL/min per 1.73 m^2 or on dialysis (endstage kidney failure).
Results: The study was conducted in 1280 patients with a mean age of 68.6 years and male predominance (60,1%). RF was found in 62% of the patients. Patients with moderate RF were characterized by the higher prevalence of strokes, myocardial infarction and the lowest ejection fraction. Patients with severe RF had the worst cardiovascular risk profile : older age, higher prevalence of cardiovascular risk factors, diabetes, hypertension, dyslipidemia, the lowest walking parameter of the 6-minute walk test and the most prescription of diuretics. Patients with endstage RF had more anemia and less prescription of angiotensin-converting enzyme (ACE) inhibitors.
Conclusion: RF seem to be highly prevalent in HF patients with a worse risk profile. The presence of cardio-renal syndrome highlights the necessity of early treating these comorbidities.

P413
Cardiohepatic syndrome is driven by congestion rather than hypoperfusion in decompensated heart failure

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Objective: Abnormal liver function tests (LFT) are associated with specific clinical, biological and prognostic features in decompensated heart failure (DHF) and defined as cardiohepatic syndrome (CHS), but their predictors are still unknown. The aim of this study was to assess the prevalence and predictors of 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 (AF) 65%, diabetes mellitus 42%, known 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. Multivariate logistic regression analysis was performed. p < 0.05 was considered statistically 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 with DHF. 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. The independent predictors of CHS were severe tricuspid regurgitation (odds ratio (OR) 32.3, 95% confidential interval (CI) 7.7-135.7, p < 0.001), right ventricular end diastolic volume >31 mm (OR 11.7, CI 5.2-26.2, p < 0.05), heart rate on admission >115 per minute (OR 10.9, CI 2.6-45.9, p < 0.05), EF<31% (OR 6.7, CI 2.4-19.3, p = 0.02), chronic AF (OR 5.7, CI 2.7-11.8, p < 0.05), hydrothorax (OR 4.3, CI 1.9-9.4, p < 0.05) and ascites (OR 2.7, CI 1.3-5.7, p < 0.05).
Conclusions: severe tricuspid regurgitation and right ventricular dilation were more powerful predictors of cardiohepatic syndrome in DHF than heart rate on admission >115 per minute and EF<31%.

P414
Nonalcoholic fatty liver disease and increased risk of 1-year all-cause and cardiac hospital readmissions in elderly patients admitted for acute heart failure

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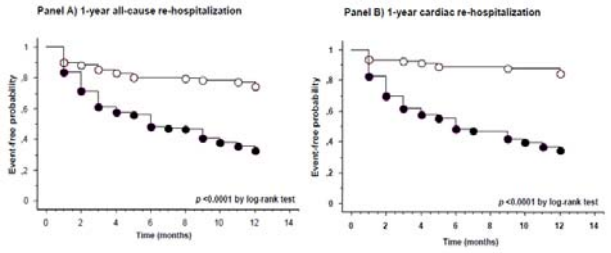
Background: Nonalcoholic fatty liver disease (NAFLD) is an emerging risk factor for heart failure (HF). The rates of hospital readmissions and the related costs continue to rise dramatically. The aim of this study was to investigate whether NAFLD diagnosed at hospital admission was independently associated with 1-year all-cause and cardiac re-hospitalizations in patients admitted for acute HF (AHF).
Methods: We studied 212 elderly patients who were consecutively admitted with AHF to our hospital over a 1-year period. NAFLD was diagnosed by ultrasonography. Patients with known acute myocardial infarction, severe valvular heart diseases, endstage renal disease, cancer, known liver diseases or decompensated cirrhosis were excluded. Cox regression was used to estimate hazard ratios (HR) for the associations between NAFLD and the outcomes of interest.
Results: The cumulative rate of 1-year all-cause re-hospitalizations was 46.7% (n = 99). Patients with NAFLD (n = 109; 51.4%) had remarkably higher 1-year all-cause and cardiac re-hospitalization rates compared with their counterparts without NAFLD. NAFLD was associated with an approximately 5-fold increased risk of 1-year all-cause re-hospitalization (adjusted-HR 5.26, 95% CI 3.03-9.08, p < 0.0001) after adjustment for established risk factors and potential confounders. Similar results were found for 1-year cardiac re-hospitalization (adjusted-HR 8.33, 95% CI 3.98-16.7, p < 0.0001).
Conclusions: NAFLD was independently associated with increased risk of 1-year all-cause and cardiac re-hospitalization in elderly patients admitted with AHF.

Table 1

Cox regression analyses - Association between NAFLD and risk of 1-year all-cause or cardiac re-hospitalization rates in hospitalized patients with AHF at baseline			
CoxHazard Models	Hazard ratio(s)	95% CI	p value
1-year all-cause re-hospitalization: NAFLD (yes vs. no) (n: 212)			
Unadjusted model	3.50	2.23-5.49	< 0.0001
Adjusted model	5.26	3.03-9.09	< 0.0001
1-year cardiac re-hospitalization: NAFLD (yes vs. no) (n: 187)			
Unadjusted model	5.86	3.27-10.4	< 0.0001
Adjusted model	8.33	3.98-16.7	< 0.0001

Data are expressed as hazard ratios ± 95% confidence intervals (CI) as assessed by either univariable (unadjusted) or multivariable Cox hazard models. Covariates included in multivariable regression models, with NAFLD, were: age, sex, hospital ward, pre-existing diabetes, CHD, LV-ejection fraction, eGFR and NT-proBNP, serum sodium and GGT levels.



P415

Mixed phenotype of cardiohepatic syndrome is common and associated with worse prognosis in patients with decompensated heart failure

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Objective: Over the last several years different mechanisms of cardiohepatic syndrome (CHS) in decompensated heart failure (DHF) have been discussed. The purpose of the study was to assess the prevalence and associations of different phenotypes of CHS in patients with DHF.
Methods: In 322 patients with DHF (190 male, 69.5 ± 10.7 years (M ± SD), arterial hypertension 87%, myocardial infarction 57%, atrial fibrillation 66%, diabetes mellitus 42%, known chronic kidney disease 39%, chronic anaemia 29%, ejection fraction (EF) 38 ± 13%, EF<35% 39%) liver function tests (LFT) - alanine transaminase (ALT), aspartate transaminase (AST), direct and total bilirubin (DB and TB), alkaline phosphatase (AP), gamma-glutamyl transpeptidase (GGT) were measured on admission.
LFT were considered abnormal when levels exceeded local upper normal limit (UNL). 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 was considered as mixed CHS. Mann-Whitney test was performed, $p < 0.05$ was considered statistically significant.
Results: Abnormal LFT occurred in 274 (85.1%) of patients. 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%, $p < 0.01$) and 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>.05) and AP (18 vs 11%, $p < 0.01$). Patients with mixed vs cholestatic CHS had higher NT-proBNP level (9200 ± 7985 vs 7122 ± 6572 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 and 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: The prevalence of mixed CHS in patients with DHF was 66.8%. 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.

P416

In-hospital changes of liver stiffness and biomarkers of liver fibrosis in patients with acute decompensated heart failure

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Purpose: To assess the dynamic changes in liver stiffness measurements (LSM) and direct fibrosis markers in patients hospitalized with acute decompensated heart failure (ADHF) receiving standard heart failure therapy according to the international guidelines.
Methods: 35 patients [8 female, median age 58 (IQR 47-68) years, NYHA III class n=16 (46%), NYHA IV class n=19 (54%)] were included. Transient elastometry measurements (FibroScan®) and direct markers of fibrosis were analyzed at baseline and discharge from hospital. The median duration of hospitalization was 14 days. Serum concentration of direct markers of fibrosis [matrix metalloproteinase-9 (MMP-9), N-Terminal Propeptide of Type III Collagen (PIIINP), laminin] was determined by means of immunosorbent assay.
Results: Heart failure therapy resulted in the reduction of signs of congestion [weight loss 5.5 (4.0-7.0) kg]. There was a significant decrease in LSM (26,3 [19-48] vs. 16.6 [11-21.8] kPa; $p < 0.01$) from baseline to end of hospitalization, although they remained elevated (grade 4 METAVIR score). A decline in LSM significantly correlated with weight loss (rho 0.4, < 0.05) and reduction of leg circumference (rho 0.3, < 0.05). On admission direct markers of fibrosis were elevated above reference values [MMP-9 850 (525.0-1390.0) ng/ml, PIIINP 33.4 (24.4-55.2) ng/ml, laminin 218 (167.0-248.5) ng/ml], and no significant changes occurred during hospitalization [ΔMMP-9 50 (-420 - 260) ng/ml, ΔPIIINP 5 (-0.7 - 13.7) ng/ml, Δlaminin 12 (-33 - 50) ng/ml, p>0.05 for all comparisons].
Conclusion: Increased LSM in patients with ADHF is not explained by fibrosis alone, and tends to be overestimated due to the presence of congestion This is confirmed by the absence of dynamic changes in fibrosis markers. Therefore, the application of transient elastometry for assessment of fibrosis in patients with ADHF is limited.

P417

Improvement of heart failure symptoms through novel implantable remede central sleep apnea treatment device: first long-term experience

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Background: Sleep-disordered breathing (SDB) and Cheyne-Stokes respiration (CSR) are associated with shorter survival periods in patients with heart failure (HF). A novel treatment method for this patient group is unilateral phrenic nerve stimulation by the remedē system, a transvenously implantable neurostimulation device, which has recently been studied in a short-term randomized, controlled trial. Previous literature has shown the efficacy and safety of the treatment with this first-generation device, but hardly any data is available on long-term clinical parameters in HF patients.
Methods: We performed remedē® device replacement in the first three consecutive HF patients for battery depletion and documented observations on clinical parameters, longevity, operation procedure, complications and difficulties with this novel device therapy.
Results: All patients were on permanent neurostimulation treatment by phrenic nerve neurostimulation when device replacement became necessary. Apnoea-hypopnoea index (AHI from 45 ± 4/h to 9 ± 4/h), oxygen-desaturation index (ODI from 35 ± 7/h to 7 ± 6/h) and time spent with oxygen saturation of < 90% (T < 90% from 5 ± 7 to 0 ± 0%) were improved and improvements remained constant throughout the four-year follow-up. Mean battery life was 4.2 ± 0.2 years, mean replacement procedure time was 25 ± 5.1 minutes. Using conventional X-ray

determine the effect of the type of COPD on endothelial function.
Results: 97 patients were evaluated (age: 72.16± 9.95 years), of whom, 53.6% were women. In the BS group, women predominated (90.7% vs 24.1%, p < 0.001), they were older (76.1± 9.1 vs 69.0± 9.5, p < 0.001), had a higher prevalence of hepato-jugular reflux (50 % vs. 20.7%, p=0.022), and history of heart failure (30.2% vs. 16.7%, p=0.113), compared to those in the TS group. The overall prevalence of ED was 78.9%. Prevalence rate was higher in those with BS compared to TS (83.7% vs 75 %, p=0.299), although according to multiple linear regression, patients with BS had 0.04 more TAM/TT index than TS subjects (β: 2.5, CI 95 %; 1.2-3.7, p <0.001).
Conclusions: ED is higher in patients with COPD from biomass smoke than in patients from tobacco smoke. Although, the prevalence between the two groups was not statistically different, the patients in the BS group had a higher prevalence of heart failure, especially right heart failure.

P1098
Relationship between endothelial dysfunction and forced expiratory volume in first second (FEV1) in chronic obstructive pulmonary disease patients

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BACKGROUND: The characteristic of Chronic Obstructive Pulmonary Disease (COPD) is the chronic inflammation in the small airways and alveoli, this takes to a limitation of the flux that is not reversible. Cardiovascular disease especially heart failure (HF) represents a high mortality and morbidity, being the cause of a worst prognostic in these patients. Has recently been found that an injury of the endothelium can contribute to emphysema and COPD and indirectly relates that the damaged in the pulmonary vasculature can developed such emphysema and COPD.
Purpose: To evaluate the relationship between endothelial dysfunction and respiratory function in patients with COPD and HF.
Methods: Cross sectional study, with 117 patients. The endothelial function was determined by Photopletysmography evaluating the amplitude, maximum amplitude time (MAT), total time of wave (TT), and the relation between both MAT/TT index, >30 was considerate as endothelial dysfunction.
Results: We evaluated 117 patients (mean age 71.4 + 11.09), the 60.7% of them had endothelial dysfunction (ED), and 56.8% were female. Those patients with ED have higher prevalence of cardiovascular disease (47.9% vs 9%, p=0.005), Pulmonary Arterial Hypertension (PAH) 21.6% vs 3% comparing it with those without ED. No significance difference were found in sex, age, weight, high, arterial hypertension, diabetes, obesity. A linear regression determined that these patients with ED has 11 % less of FEV1 comparing with those without ED. (β-11.251 p=0.05)
Conclusion: The patients with ED had a lower FEV1 compared with those without ED. In patients with EPOC those with ED will have a worst degree of EPOC and in which turn to the hypothesis that the ED is not only local but instead is at capillary, alveoli and systemic.

P1099
Association between heart failure and nonalcoholic fatty liver disease

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In the last decade the role of alcoholic fatty liver disease discuss as a disease, which determines not only the severity of injury, but associated with the progression of cardiovascular disease (CVD) and the development of type 2 diabetes mellitus (DM) and other system injuries [1,2].
Purpose: to determine the relationship between NAFLD and heart failure
Methods: The study involved 112 NAFLD patients with normal, overweight and obese without of type 2 diabetes mellitus and 64 patients with normal, overweight and obese with type 2 diabetes mellitus. Conducted anthropometric survey measured levels of AST, ALT, GGT, the degree of liver fibrosis using elastography (FibroScan), ECG and echocardiography. The stratification of CVrisk was carried by traditional SCORE scale version for countries with high risk. We determined the level of inflammatory mediators (TNF-α, IL-1, IL-6), markers (CRP, fibrinogen), endothelin -1, the activity of the Willebrand factor (vWF), the thickness of the intima-media complex, presence atherosclerotic plaque and stenosis of the carotid arteries, insulin resistance index HOMA-IR for all examined patients.
Results: In both groups was revealed left ventricular diastolic dysfunction and QT prolongation in patients with NAFLD and type 2 diabetes that was associated with the severity of the disease. Most patients with NAFLD by obesity showed a reduction in endothelium-dependent vasodilation, indicating the presence of endothelial dysfunction. The concentration of pro-inflammatory cytokines such as TNF-α and IL-6 in patients with NAFLD was 3-7 times higher than the similar parameters of patients with a similar degree of obesity, but without evidence NAFLD. The concentration of ET-1 in the blood plasma of patients with NAFLD has a strong direct correlation with the degree of cardiovascular risk of surveyed patients. It is found that many inflammatory mediators (TNF-α, IL-1, IL-6) and markers (C-reactive protein, fibrinogen) highly correlate with the degree of obesity, the concentration of ET-1, vWF and markers of insulin resistance, a predictor for cardiovascular risk.
Conclusions: Presents of diastolic dysfunction of left ventricular and heart failure, disturbances of endothelium-dependent vasodilation, the concentration of ET-1, mediators of systemic inflammation, increase the values of intima-media thickness, an increase the frequency of cardiac arrhythmias is highly correlated with the degree of cardiovascular risk. Presence of NAFLD dictates mandatory screening for cardiovascular disease in these patients.

P1100
Liver stiffness assessed by transient elastography is associated with congestion in patients with decompensated heart failure

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Objective: Heart failure (HF) has a negative impact on liver leading to hepatic fibrosis or even cirrhosis. Studies dealing with assessment of liver stiffness (LS) in decompensated heart failure (DHF) are relatively few and have contradictory results. The aim of the study was to assess LS by the transient elastography (TE) on admission and its associations in patients with DHF.
Methods: LS was measured using TE (FibroScan Echosens, France) in 94 patients with DHF on admission (60 male, 72 ± 10 years (M ± SD), arterial hypertension 96%, myocardial infarction 55%, atrial fibrillation (AF) 63%, diabetes mellitus 45%, known chronic kidney disease 29%, chronic anaemia 30%, left ventricular ejection fraction (EF) 40 ± 14%, EF < 40% 28%, NYHA IV 41%). Ten valid measurements were required with success rate of >60%. LS≤5.8 was considered normal and LS ≥5.9, 7.2, 9.5 and 12.5 kPa are considered fibrosis (F1-F3 METAVIR score) and cirrhosis (F4) according to thresholds in studies with chronic liver disease. Mann-Whitney test was performed. p < 0.05 was considered statistically significant.
Results: The median value of LS was 13.1 (interquartile range 7.4;26) kPa. Normal LS was observed in 15.9% of patients. Abnormal LS ≥5.9, 7.2, 9.5 and 12.5 kPa occurred in 8.5, 13.8, 10.6 and 51.1% of patients respectively.
Patient with vs without LS ≥12.5 kPa had higher incidence of ischemic HF (79 vs 65%, p < 0.05), chronic anaemia (42 vs 19%, p < 0.01), glomerular filtration rate <60 ml/min/1.73 m2 (71 vs 55%, p < 0.05), chronic AF (54 vs 36%, p < 0.01), NYHA IV (52 vs 35%, p < 0.05).
LS ≥12.5 kPa was associated with high incidence of signs of congestion: peripheral oedema (98 vs 87%, p < 0.05), rales (94 vs 74%, p < 0.001), jugular venous distension (44 vs 32%, p < 0.05), hepatomegaly (69 vs 35%, p < 0.01), ascites (33 vs 10%, p < 0.001), severe mitral and tricuspid regurgitation (38 vs 19% and 58 vs 10%, p < 0.001 for both) and higher diameters of inferior vena cava (25 ± 6 vs 21 ± 7 mm, p < 0.05), right and left atriums (64 ± 13 vs 59 ± 15 mm, p < 0.05 and 51 ± 11 vs 47 ± 11 mm, p < 0.001), right ventricular end diastolic diameter (33 ± 5 vs 30 ± 5 mm, p < 0.001), pulmonary artery pressure (56 ± 18 vs 45 ± 13 mmHg, p < 0.001), higher levels of NT-proBNP (4655 (3120;7009) vs 4319 (2500;4961) pg/ml, p < 0.001), serum urea (10.1 (7.3;13.5) vs 7.4 (5.9;9.8) mmol/l, p < 0.05) and creatinine (109 (100;129) vs 105 (88;125) μmol/l, p < 0,05).
Patient with LS ≥12.5 kPa had higher rate of vasopressor therapy (10.4 vs 0%, p < 0.01) and longer length of stay (9 (8;15) vs 9 (6;12) days, p < 0.05).
Conclusions: Abnormal LS was occurred in 84.1% of patients with DHF. LS ≥12.5 kPa was associated with signs of congestion and right-sided HF, higher rate of vasopressor therapy and longer length of stay.

P1101
Central venous pressure is the main hemodynamic determinant of liver function in advanced heart failure.

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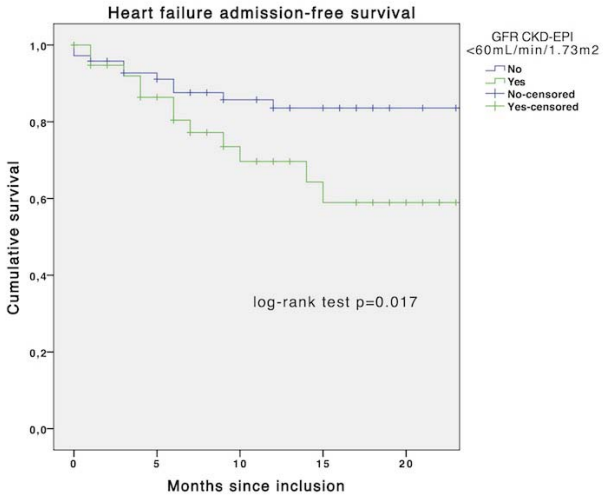
Background: The relationship between liver function (LF) abnormalities and hemodynamics in heart failure (HF) is still incompletely understood as previous studies mainly included patients without contemporary HF medications and did not investigate liver synthetic function.
Methods: We assessed the relationship between LF tests and hemodynamics in 309 consecutive patients with a left ventricular ejection fraction < 45 % treated with contemporary medical HF therapy, and undergoing right heart catheterization using Swann-Ganz catheters. Cardiac output was measured using thermodilution.
Results: Mean age was 50 + 13 years, and 239 (77%) were men. Only 22 (7%)



Heart failure admission-free survival

P1802

Cardiorenohepatic syndrome is common in patients with decompensated heart failure

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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). 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 vs 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, $p < 0.05$).
Conclusions: Isolated CHS, isolated CRS and CRHS occurred in 78.4, 1.5 and 20.1% patients with DHF and hepatic or kidney injury. 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%.

P1803

Low ferritin reflecting depleted iron stores predicts inspiratory muscle weakness independently of skeletal muscle mass in men with heart failure with reduced ejection fraction

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Background: Skeletal and respiratory muscle dysfunction (specific myopathy) constitutes an important pathophysiological feature of heart failure syndrome. We assessed the relationships between respiratory muscle function, skeletal muscle mass, and physical fitness in men with heart failure with reduced ejection fraction (HFrEF), and investigated the hypothesis of whether iron deficiency contributes to respiratory muscle dysfunction in these patients.
Methods: We examined 53 male outpatients with stable HFrEF without asthma or chronic obstructive pulmonary disease (age: 64 ± 10 years; New York Heart Association [NYHA] class I/II/III: 36/51/13%; ischaemic aetiology of HFrEF: 83%; all patients with left ventricular ejection fraction [LVEF] ≤40%) and 10 middle-aged healthy men (control group). We analyzed respiratory muscle strength (maximal inspiratory and expiratory pressure at the mouth [MIP and MEP, respectively]), appendicular lean mass (ALM; reflects appendicular skeletal muscle mass; measured using dual-energy X-ray absorptiometry), physical fitness (components of Functional Fitness Test [FFT] for Older Adults - "Up and go", "Chair stand", and "Arm curl", and 2-minute step test), and parameters of iron status.
Results: MIP, MEP, ALM adjusted for body mass index (ALM/BMI), and MIP adjusted for ALM/BMI were lower in men with HFrEF vs. healthy men (74 ± 23 vs. 106 ± 33 cm H2O, $p < 0.001$; 122 ± 23 vs. 161 ± 28 cm H2O, $p < 0.001$; 81 ± 10 vs. 96 ± 11 100*m^2, $p < 0.001$; and 93 ± 27 vs. 117 ± 40 cm H2O/m^2, $p = 0.04$; respectively). MIP (but not MEP) and MIP adjusted for ALM/BMI (but not ALM/BMI) were lower in men with HFrEF with ID compared with those without ID (67 ± 20 vs. 106 ± 33 cm H2O, $p = 0.02$; and 84 ± 22 vs. 103 ± 28 cm H2O/m^2, $p = 0.02$; respectively). In univariable linear regression analyses MIP was related to (all $p < 0.05$): LVEF (r=0.31), ALM/BMI (r=0.35), N-terminal pro B-type natriuretic peptide (NT-proBNP, r=-0.30), haemoglobin (r=0.35), mean corpuscular haemoglobin (r=0.36), serum ferritin (r=0.41), the presence of ID (r=-0.32), reticulocyte haemoglobin content (r=0.45), and percentage of hypochromic red cells (r=-0.31). In a multivariable linear regression model lower serum ferritin remained an independent predictor of lower MIP when adjusted for ALM/BMI, LVEF, NT-proBNP, and haemoglobin (β=0.36, $p = 0.017$, corrected R^2=34%). Importantly, in multivariable linear regression models, lower MIP was also an independent predictor of worse results in FFT when adjusted for ALM/BMI or relevant clinical variables (NYHA class, estimated glomerular filtration rate, NT-proBNP, and haemoglobin).
Conclusions: In men with HFrEF decreased iron stores (as reflected by low circulating ferritin) predict inspiratory muscle weakness independently of skeletal muscle mass. Importantly, the dysfunction of inspiratory muscles in these patients is associated with worse physical fitness independently of either skeletal muscle mass or disease severity.

P1804

Iron deficiency predicts decreased exercise capacity and physical fitness independently of skeletal muscle mass in men with heart failure with reduced ejection fraction

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BACKGROUND: In patients with heart failure concomitant iron deficiency (ID) is associated with decreased exercise capacity. Another important predictor of reduced functional capacity in these patients is skeletal muscle mass loss. We investigated the hypothesis of whether in men with heart failure with reduced left ventricular ejection fraction (HFrEF) ID predicts decreased exercise capacity and physical fitness independently of indices of skeletal muscle mass loss.
Methods: We examined 49 male outpatients with stable HFrEF (age: 65 ± 11 years; New York Heart Association [NYHA] class I/II/III: 39/45/16%; ischaemic aetiology of HFrEF: 86%; all patients with left ventricular ejection fraction [LVEF] ≤40%). We analyzed exercise capacity (6-minute walking test distance - 6MWD), physical fitness

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