081 Results of the survey Ithaque: monitoring and therapies for systolic heart failure
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Electrical therapies by defibrillators (ICD) and resynchronization devices (CRT-D – CRT-P), have demonstrated their benefits on symptoms, morbidity and mortality in selected heart failure patients (HF). The ITHAQUE survey aims to describe the different supports of patients in HF patients with impaired left ventricular (LV) function (LV ejection fraction < 45%)

Methods: 927 patients treated for HF were seen in outpatient visit with a general cardiologist (75%) or in a tertiary center (25%). A survey of treatments and follow-ups was completed by the physician. 3 groups under optimal treatment, defined by the French Society of Cardiology (SFC) guidelines, have been analyzed:
- Gr1 – Class 1B (patients with ischemic cardiomyopathy and coronary revascularization, NYHA II and III, LVEF <30%) – Class 2Aa (30%)
- Gr2 – Class 2aB (dilated cardiomyopathy, NYHA II and III, LVEF <30%) and class 2BC (30%)
- Gr3 – Class 1B (HF Patients, NYHA III and IV, LVEF <35% and QRS >120 ms)

Results: Of the total patients, 79 received an ICD (8.5%), 22 a CRT-D (2.4%) and 76 a CRT-P (8.2%). On the other side, 279 patients eligible according to the recommendations of the SFC did not receive additional treatment with ICD or CRT (30%). Pharmacological treatment associated is mainly composed of IEC (76.5%), Beta-blockers (76%), Diuretics (90%), anticoagulants (80%) and ACE inhibitors (70%).

Rules lifestyle modifications (77.8%) and practice of physical activity (31.2%) are also prescribed, associated or not.

Conclusion: Although the specific conditions of therapeutic patient must be taken into account, the ITHAQUE survey did show that the electrical therapies recommended by the SFC for heart failure patients with systolic dysfunction are underused.

082 A retrospective study of systematic research of chronic obstructive pulmonary disease in a systolic heart failure population
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Background: Chronic obstructive pulmonary disease (COPD) is an independent risk factor for cardiovascular disease (CVD) including heart failure (HF) and coronary arteries diseases, which are one of the leading causes of morbidity and mortality in COPD patients. Thus, COPD could deteriorate HF and explain the worst prognostic of the association of the two diseases. Actually, there is no information about the prevalence of COPD assessed by systematic pulmonary function test in HF patients.

Purpose: Describe the prevalence of COPD in a systolic HF population.

Methods: COPD was systematically researched by pulmonary function test in 274 patients (216 men) followed for systolic HF in the University’s Hospital of Rangueil, Toulouse, France between April 2002 and April 2009. Degrees of COPD were defined according to the GOLD classification.

Results: In the 274 systolic HF patients mean age was 61±11 years and mean ejection fraction (EF) was 32±15 % with 138 (47 %) patients with ischemic systolic HF. There was 112 (41 %) of COPD with 57 (20.8 %), 44 (16.1 %), 7 (2.6 %) and 4 (1.5 %) GOLD 1, 2, 3 and 4 respectively. There was no difference between no-COPD and COPD groups in sex (77.7 vs 75.9 %; p=0.1) and NYHA stage (mean 2.2±0.7 vs 2.3±0.8; p=0.3) but patients with COPD were older (66±14 vs 57±13.7 years; p<0.001) and had better EF (33±5 vs 40 vs 30±2±12.9 %; p=0.016). There was 43.8 % ischemic heart failure in the COPD group versus 51.8 % in the no-COPD group (p=0.13) and no-COPD group had a tendency to have more Î±-blocker treatment (74.1 % vs 62.5 %; p=0.05).

Conclusion: COPD has a high prevalence in systolic HF population but clinical diagnostic is difficult because of the lack of specificity of dyspnoea assessed by NYHA stage. HF patients with COPD are older and have a better EF suggesting that dyspnoea from pulmonary disease is interpreted as a symptom of HF.

083 Heart failure with preserved ejection fraction.

Age of atrial fibrillation: two main characteristics of patients enrolled in the KaRen project
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Background: We describe the baseline characteristics of subjects prospectively recruited in the KaRen morbidity-mortality French and Swedish registry. KaRen’s purpose is to study the impact of dyssynchroon (electrical and/or mechanical) on the prognosis of heart failure patients with preserved ejection fraction.

Methods and results: The Karen study is an ongoing study with a minimum follow-up of 18-month for every patient. Here, we are reporting the initial characteristics of the 268 first patients included in the registry. These heart failure patients with preserved ejection fraction recruited in university hospitals are old. The mean age is 76.5 years old. 43% are between 80 and 89 years old. 59% were women. 10% of them have a bundle branch block. The prevalence of hypertension is 75%, coronary artery disease 31%, valvular heart disease 20%, atrial fibrillation 85%, pulmonary primitive disease 24%, peripheral vascular disease 15%, renal insufficiency 62% (creatinine >120µmol/l). The percentage of death at 4-8 weeks follow-up after the inclusion is 4% (9% in Sweden and 2 in France despite the great similarity between the other characteristics of the population).

Conclusions: Patients in KaRen are “real world” patients with heart failure and preserved ejection fraction. Age and atrial fibrillation are the two most determinative characteristics of our population.

084 Diabetes prevents compensatory hypertrophy after myocardial infarction and impairs cardiac function: possible implication of stretched induced kinase growth signaling
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Aim: The present study investigated possible mechanisms underlying postischemic remodeling in diabetic hearts. Diabetes (DM) accelerates postischemic cardiac remodeling and increases mortality after myocardial infarction.

Methods: Acute myocardial infarction (AMI) was induced in rats with type 1 diabetes (DM) and non diabetic rats (NDM-AMI) while sham operated animals served as controls (SHAM). All groups were subjected to echocardiographic analysis 2 weeks after infarction.

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Thyroid hormone receptor alpha is down regulated during the progression to congestive heart failure after myocardial infarction
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Aim: Thyroid hormone (TH) signaling is altered in response to various stresses including myocardial ischemia. The present study investigated potential implication of TH in the pathophysiology of postischaemic remodeling.

Methods: Acute myocardial infarction was induced in rats by coronary artery ligation (AMI). After 34 wk, n=6 animals were on congestive heart failure (CHF) as indicated by measurements in lung and right ventricular weight. N=7 animals were in compensated state (non-CHF) and n=8 SHAM operated animals served as controls (SHAM).

Results: Progression to congestive heart failure was characterized by marked decrease in EF% and all other functional echocardiographic parameters. Furthermore, β-MHC expression was significantly increased in CHF. A distinct pattern of TR expression was observed in the course of postischaemic remodeling: TRα1 was up-regulated and TRβ1 was down-regulated in non-CHF and TRβ1 expression was markedly decreased during the transition from non-CHF to CHF resulting in tissue hypothyroidism. Circulating T3 and T4 remained unchanged. This response was associated with marked decrease in ERK and mTOR activation. A potential link between ERK, mTOR and TRα1 expression was shown in a neonatal cardiomyocytes model of PE (phenytoin). A potential link between ERK, mTOR and TRα1 expression was abrogated after inhibition of mTOR or ERK.

Conclusion: Thyroid hormone signaling is altered in response to various stresses including myocardial ischemia. The present study investigated potential implication of TH in the pathophysiology of postischaemic remodeling.

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Lipoprotein-associated phospholipase a2 (Lp-PLA2) and markers of oxidative stress in patients with acute heart failure
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Background: Oxidative stress (OS) in heart failure (HF) is increased and contribute to the pathogenesis of myocardial remodeling. Vascular inflammation is involved in deterioration of HF patients, with production of cytokines, complement and vascular cellular adhesion molecules that perpetuate the inflammatory state. Lipoprotein-associated phospholipase A2 (Lp-PLA2), a calcium-dependent protein that hydrolyze phospholipids and may be involved in inflammation and atherosclerosis.

Aims: We studied association between Lp-PLA2 and cardiogenic shock and the relation between Lp-PLA2 and OS markers.

Methods: Inclusion criteria: CS defined by systolic BP<90 mmHg with peripheral hyperperfusion. Exclusion criteria: pace makers or other shock etiologies. Cardiac (echocardiography, coronarography) and serial biological assays were performed upon admission and at 1 month. Lp-PLA2 was measured with turbidimetric immunoassay. To determine the OS, we measured marker of lipid peroxidation, protein oxidation, total anti-oxidative status and antioxidant enzymes.

Results: 22 consecutive patients with CS (90% men, 57.5±10.7y, LVEF 25.3±8.5%, proBNP 8540 ng/L) were included; 7 ischemic CM, 15 dilated CM [12 idiopathic, 2 toxic, and 1 myocardiat]. Cardiovascular risks: diabetes (n=11), HBP (n=9), tabacco (n=9), dyslipidemia (n=8). Lp-PLA2 were significantly elevated in population with CS (226.8±73 μg/L), when referred to controls (p=0.005) whatever the cardiomyopathies. Lp-PLA2 was positively correlated with glutathione reductase but not significantly correlated with plasma and erythrocyte malondialdehyde, plasma carbonylated proteins and oxidized LDL; erythrocyte superoxide dismutase, glutathione peroxidase and catalase activities; plasma α-tocopherol and vitamin A, erythrocyte reduced glutathione (GSH) and GSSG/GSH ratio.

Conclusion: This study showed that Lp-PLA2 does not affect OS in patients particularly ox-LDL. It is noteworthy that the plasma OS is not the exact reflects of inflammation in vascular wall because the main role of Lp-PLA2 in atherogenesis is the hydrolysis of ox-LDL, which is generated when LDL becomes oxidized in the milieu of the artery wall.