Abstract No. **62**
Category: **Acute Coronary Syndromes**
Title: **Characterization of the electrical and extracellular matrix remodeling in patients with HF: comparison between HFpEF and HFrEF.**
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Abstract:

Introduction: Individuals with heart failure (HF) with preserved ejection fraction (HFpEF) experience high morbidity and mortality, but contrarily to HF with reduced EF (HFrEF), anti-remodeling therapies have failed to improve mortality. Current methods to detect LV reverse remodeling reveal primarily advanced disease and fail to detect early staged of tissue phenotyping.

Objective: In this study we aim to investigate and compared the myocardial tissue remodeling in HFpEF and HFrEF using a contemporary multimodality approach to assess myocardial electrical and extracellular matrix remodeling.

Methods: Symptomatic HF patients (NYHA II-III) stratified according to LVEF in HFrEF (<50%) and HFpEF (>/=50%) underwent Cardiac MRI (CMRI) including T1-mapping, Echocardiogram (ECHO) with GLS, Cardiopulmonary exercise test (CPET), Cardiac sympathetic imaging with mIBG and biomarkers. All individuals were recruited when stabilized using optimized HF therapy.

Results: Forty-seven individuals (age:54.1±11 years, BMI:30.5±6, 22 females, mean-LVEF: 42.2 ± 15%, 24 HFrEF and 23 HFpEF) were prospectively recruited. As expected LVEF were different among groups (32 ± 8.5 %vs. 58.2 ± 7%, p<0.001) and the adjusted-VO2max were more pronounced reduced in HFrEF (18.3 ± 4.7 vs. 22.8 ± 5.2 ml/min/kg, p=0.01). While ECHO derived GLS were reduced in HFrEF compared to HFpEF (HFrEF:-8.2 ± 3.7 %vs. HFpEF:-15.2 ± 3.7%, p<0.001), both the native-T1 (HFrEF:1101.6 ± 213 vs. HFpEF:1146 ± 58, p=0.4) and extracellular volume fraction (ECV) although abnormal were not different among groups (HFrEF:0.36 ± 0.07 vs. HFpEF:0.33 ± 0.03, p=0.06). The mIBG derived heart-to-mediastinum ratio (HMR) were also reduced in both groups but more evident in the HFrEF (1.44 ± 0.17 vs. 1.62 ± 0.21, p=0.007). Considering the entire cohort, ECV was inversely associated to HMR (r=-0.45, p=0.023) and to adjusted-VO2max (r=-0.41, p=0.02); and positively associated to NT-proBNP (r=0.52, p<0.001), US-Troponin (r=0.6, p= 0.009) and to GLS (r=0.59, p<0.001). Whilst all these associations were maintained in HFrEF, only the association of ECV and GLS remained significant (r=0.7, p< 0.05) in HFpEF subgroup.

Conclusion: This pilot study highlights the considerable myocardial tissue remodeling present in patients with HFpEF. Interestingly, the extracellular matrix remodeling, assessed by the ECV, were similar among HEpEF and HErEF, confirming that the irreversible fibrosis frequently occur in HFpEF, which may at least partially, explain its unfavorable prognosis and limited response to anti-remodeling therapies seem in contemporary clinical trials.