Abstract: P267

Arterial stiffness in heart failure patients after an acute episode of acute decompensation: preliminary results

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Background: The role of arterial stiffness in the pathogenesis and clinical outcome in heart failure (HF) patients should be elucidated yet. Preliminary data demonstrated an increased pulse wave velocity (PWV) in HF patients in comparison with healthy subjects and patients with cardiovascular risk factors. Moreover, growth differentiation factor-15 and soluble ST2 (sST2), biomarkers of cardiac stress and validate prognostic tools of HF, seemed to be strictly related to PWV.

Purpose: The aim of this observational experience was to analyze the arterial stiffness in HF patients in comparison with a healthy control population trying to correlate it with functional parameters.

Methods: consecutive patients admitted for acute decompensation underwent clinical examination, echocardiogram, biomarkers dosage and evaluation of arterial stiffness by measuring the PWV (expressed in meter/second from the pressure wave transit and the distance between carotid and femoral artery) and the augmentation index (AIX75) (using the manufacturer’s proprietary software) with a Sphygmocor applanation tonometer system according to the established protocol. The arterial stiffness was also calculated in a control group formed by healthy volunteers.

Results: 59 hospitalized HF patients (40 males; age 73.3±11.5 years) with mean LVEF 38.5±12.2% and NT-proBNP (10765.7±10096.4 pg/ml) entered the study. Twenty-seven (47.5%) had a coronary artery disease (CAD), in 27 (45.7%) the etiology was dependent from a valvular disease and the others are hypertensive of idiopathic. The HF population were compared with 42 subjects [25 healthy controls (11 males, age 54.8±13.6 yrs) and 17 patients (11 males, age 71.9±8 yrs) with cardiovascular risk factors (CVRF) but in absence of heart failure]. The control group was formed by younger subject in comparison to CVRF group and HF patients (p<0.001 for both groups). The analysis of PWV demonstrated a velocity of 10±1.68 m/sec, 12.41±2.18 m/sec and 10.94±2.19 m/sec in controls, CVRF and HF patients, respectively (HF vs CVRF p=0.03; HF vs controls p=0.14; CVRF vs controls p=0.001). Aix75 (corrected for heart frequency) demonstrated to be higher in CVRF group vs HF patients (35.12±9.21 vs 23.28±13.22, p=0.004) so as central systolic pressure resulted higher in CVRF than in others (CVRF 63.23±14.50 mmHg, HF 54.16±14.09 mmHg, controls 50.08±8.94 mmHg: CVRF vs HF p=0.03; HF vs controls p=0.40; CVRF vs controls p=0.005).

Conclusion: CVRF patients seemed to present a worst arterial stiffness in comparison to healthy subjects and HF patients hospitalized. These preliminary results deserve future investigation on a larger population.