Heart failure: pathophysiology and prognosis / Heart failure: mechanisms for progression

P4226 | BEDSIDE
Epicardial fat volume is inversely correlated with the degree of diastolic dysfunction and outcome in patients with heart failure with preserved ejection fraction

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Introduction: Epicardial adipose tissue has been linked to cardiovascular metabolism and inflammation and has been shown to predict prevalence and progression of coronary artery disease

Methods: HPPEF was defined as serum NT-proBNP levels >220 pg/ml, e' by echocardiography >8, signs or symptoms of heart failure and preserved left ventricular ejection fraction (EF>50%)

98 HFP EF patients and 34 controls were prospectively evaluated. All patients underwent right heart catheterization. CMR studies included the assessment of cardiac function and dimensions by standard cine sequences. Epicardial fat volume was quantified offline, using dedicated software.

Results: Epicardial fat volume ranged from 23 to 89 ml (mean 49.3±16.2 ml); patients- 43.8±13.4 ml, controls- 58.6±16.6 ml; p<0.001. Epicardial fat volume was significantly correlated with e'(R=0.37, p<0.001), NT-proBNP (R=0.27, p=0.012), right ventricular size and function (R= -0.32; p=0.002 and R=0.40; p<0.001), left ventricular ejection fraction (R=0.36, p=0.001), left and right atrial size (R= -0.27, p=0.011; R= -0.34; p=0.001), mean pulmonary arterial pressure (R= -0.36, p=0.005), pulmonary capillary wedge pressure (R= -0.33; p=0.013), and pulmonary vascular resistance (R= -0.34; p=0.011). Epicardial fat volume was not correlated with gender, age, renal function, or body mass index

Conclusion: Epicardial fat volume was inversely correlated with diastolic dysfunction, serum NT-proBNP, invasive measures of pulmonary hypertension, but not total body fat. Decreasing epicardial fat volume predicts adverse outcome in HFP EF patients. The mechanism causing decreasing epicardial fat volume in advanced disease remains to be determined.

HEART FAILURE: MECHANISMS FOR PROGRESSION

P4228 | BEDSIDE
Clinical significance of tissue fibrosis and conduit abnormality in long-term prognosis in hypertrophic cardiomyopathy

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Purpose: This study evaluates the level of tissue fibrosis by RV biopsy in hypertrophic cardiomyopathy (HCM) patients and assessed its relevance to the long-term prognosis

Methods: Of 494 consecutive HCM patients, 194 patients histologically diagnosed were enrolled (58±13 years old). The amount of fibrosis (%-area) in tissue samples from RV biopsy were quantified and classified into mild (<6%), moderate (6–17%) and severe (>17%). Hemodynamic, echocardiographic and electrophysiological parameters were also evaluated. Patients were followed and primary endpoint was adverse cardiac events; heart failure or lethal ventricular arrhythmia, and secondary endpoint was death from any cause.

Results: Degree of tissue fibrosis was associated with cellular diameter, LVEDP but not LVEF and BNP level. Patients with severe fibrosis had longer filtered QRS (QRSF) duration. Positive late potential by SAECG and positive fragmented QRS, resulting in a higher incidence of VT/VF. During 5.4±5 years follow-up, 92 (47%) patients had adverse cardiac events. The higher degree of fibrosis had a greater risk of following adverse events (Fig.1). Multivariate Cox analysis revealed that tissue fibrosis (>6%) and longer QRSF duration (0–2) was useful for risk stratification of adverse cardiac events in HCM (Fig.2). On the other hand, lower LVEF (p<0.008) and history of VT/VF (p=0.02) were the independent predictor for any cause of death.

Cardiac events and tissue fibrosis

Conclusion: Higher LVEDP-related fibrotic change may contribute to the abnormal conduction delay as well as spontaneous VT/VF, leading to poor prognosis in HCM patients.

P4229 | BEDSIDE
Factors influencing transition to symptomatic heart failure in Stage-B asymptomatic patients - A report from the CHART-2 study

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Background: Increasing prevalence of heart failure (HF) is an urgent public health issue worldwide. Although the ACC/AHA Guidelines emphasize the importance to prevent the development of HF in asymptomatic Stage-B patients, little is known about the etiology and predictors for transition to symptomatic HF in Stage-B patients.

Method: Among the 10,219 subjects registered in our cohort, named as the Chronic Heart Failure Analysis and Registry District in the Tohoku District 2 (CHART-2) study, we enrolled 4,463 Stage-B patients in the present study. We analyzed the predictors of de novo HF in asymptomatic Stage-B patients by Akaike’s information criterion (AIC).

Results: Mean age was 67±12.4 years old, and male patients accounted for 71%. Regarding etiologies for asymptomatic cardiac structure abnormalities, the prevalence of ischemic heart disease, valvular heart disease, and cardiomyopathy was 51, 19, and 10%, respectively. During the median follow-up period of 3.0 years, 280 deaths (6.0%), 103 cardiovascular deaths (2.2%) and 165 de novo HF requiring hospitalization (3.5%) were noted. A stepwise Cox regression analysis with AIC revealed that development of de novo HF in Stage-B patients could be predicted by age (Hazard ratio (HR) 1.02, p=0.013), diastolic blood pressure (DBP) (HR 0.97, p=0.001), paroxysmal atrial fibrillation (PAF) (HR 2.50, p=0.001), chronic atrial fibillation (CAF) (HR 2.20, p=0.001), left ventricular (LV) dilatation (HR 1.62, p=0.017), LV ejection fraction(<50%) (HR 2.15, p=0.001), anemia (HR 2.02, p=0.001), chronic kidney disease (CKD) (HR 1.79, p=0.002), and systolic blood pressure (SBP) (HR 0.67, p=0.038). A sub-analysis showed that statin use was significantly associated with lower event rate of de novo HF in female patients (HR 0.41, P=0.009), older age (>70 yrs) (HR 0.56, P=0.001) and CKD (HR 0.60, P=0.041). Event rates of de novo HF in patients with PAF and CAF were comparable (2.2 vs. 2.3 events/100 person-years). In groups categorized by systolic blood pressure (SBP) and DBP, DBP lower than 70mmHg was a risk for de novo HF (HR 2.38, P=0.001), especially for patients with SBP >150mmHg (HR 9.39, P<0.001).

Conclusions: These results indicate that several factors could influence the progression from asymptomatic to symptomatic HF in Stage-B patients, including higher age (>70 yrs), low DBP (<70mmHg), PAF, CAF, LV dysfunction (EF<50%), anemia, renal dysfunction and no statin use, suggesting that management of these factors could prevent the development of de novo HF in Stage-B patients.

P4230 | BEDSIDE
Soluble vascular endothelial growth factor receptor-2 serves as a predictor of cardiovascular events in patients with chronic heart failure

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Background: Vascular endothelial growth factor (VEGF) is a key regulator of cardiovascular angiogenesis and is required for preventing decompensated heart failure. Soluble VEGF receptor-2 (sVEGF-2) acts as an endogenous inhibitor of VEGF. Recently, we demonstrated that serum sVEGF-2 levels are increased in patients with chronic heart failure.

Methods and results: We performed a prospective cohort study involving 117 symptomatic patients with CHF. Patients were followed up over 2 years. The pri-