INCREMENTAL VALUE OF CYSTATIN C BEYOND CONVENTIONAL MARKERS OF RENAL FUNCTION FOR PREDICTING CLINICAL RESPONSE IN PATIENTS RECEIVING CARDIAC RESYNCHRONIZATION THERAPY: THE BIOCRT STUDY

Poster Contributions
Hall C
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Session Title: Approaches to Advanced Heart Failure: From VAD, Transplant, Palliative Care to New Percutaneous Therapies
Abstract Category: 12. Heart Failure and Cardiomyopathies: Clinical
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Background: Cystatin C, an emerging marker of renal function, has been shown to be predictive of clinical outcome in patients with heart failure (HF). This study compares cystatin C to conventional measures of renal function (serum creatinine [Cr] and estimated glomerular filtration rate [eGFR]) in its ability to predict clinical response and major adverse cardiac events (MACE) in patients undergoing cardiac resynchronization therapy (CRT).

Methods: In 132 patients undergoing CRT (age 65±14; 77% male; LVEF 26±7%; 45% ischemic cardiomyopathy; 95% NYHA Class II-III) pre-implant serum cystatin C levels (Siemens, Dimension Vista) were measured from the peripheral vein and compared to serum creatinine and eGFR. A positive CRT response was adjudicated as an improvement in the HF Clinical Composite Score at 6-month follow-up. MACE was defined as the composite endpoint of death, cardiac transplant, left ventricular assist device, and HF hospitalization at 2 years. We also assessed functional capacity by 6 minute walk distance (6MWD) and Minnesota Quality of Life (MQOL) score at baseline and 6 months.

Results: Median cystatin C levels were 0.94 [0.76, 1.29] mg/L, Cr 1.23 [0.99, 1.51] mg/dL, and eGFR 60.3 [46.7, 78.3] mL/min/1.73 m2. There were 53 (40%) CRT non-responders and 31 (23%) patients with MACE. After adjustment for age, sex, ischemic cardiomyopathy, and left bundle branch block, all 3 renal metrics remained predictive of MACE (all p≤0.01). In contrast, only cystatin C was associated with CRT non-response with over 2-fold increase risk in odds per 1 mg/L increase (adjusted odds ratio 2.6, 95% CI 1.1-6.2, p=0.03), whereas Cr and eGFR were not (both p ≥0.11). Using an optimal cutpoint for Cystatin C of 1 mg/L for CRT response, we found that patients with cystatin C > 1mg/L had significantly reduced functional capacity (6MWD: 930±370 vs. 1224±399 feet, p<0.001) and a trend to worse MQOL score (MQOL: 37 vs. 28, p=0.06) when compared to patients with cystatin C ≤ 1 mg/L at 6 month follow-up.

Conclusions: In patients undergoing CRT, cystatin C but not other standard measures of renal function (creatinine, eGFR) was independently predictive of CRT non-response.