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IMPACT OF LEFT VENTRICULAR DYSSYNCHRONY ON LEFT VENTRICULAR SYSTOLIC FUNCTION IN CHRONIC HEART FAILURE ASSESSED BY 99MTC-SESTAMIBI GATED MYOCARDIAL SCINTIGRAPHY

ACC Poster Contributions

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Background: Left ventricular (LV) dyssynchrony is a phenomenon extensively described and related to reduced LV systolic function and poor diagnosis in patients with chronic heart failure (HF). However, it remains unknown whether LV dyssynchrony is a determinant of reduced LV systolic function independently from the severity of LV myocardial perfusion defect.

Methods: A total of 105 patients with chronic HF (age; 71±13 years old, 71 men) were enrolled in the present study. 99mTc-sestamibi (MIBI) gated myocardial scintigraphy was performed at rest to assess LV myocardial perfusion as evaluated by the total defect score of perfusion SPECT images (TDS-MIBI), LV systolic function as evaluated by LV ejection fraction (LVEF), and LV systolic dyssynchrony as evaluated by the maximal difference of time to end systole (MD-TES), which is the time lag between the earliest and the latest end systole among 17 LV segments analyzed with a novel software program, "cardioGRAF" (FUJIFILM RI Pharma Co, Ltd, Tokyo, Japan). The relationships among LVEF, the TDS-MIBI, and the MD-TES were examined.

Results: The mean±SD and the range of MD-TES were 147.8±117.5 ms and 14.0-458.3 ms, respectively. The MD-TES was significantly higher in patients with LVEF<45% (199.4±117.6 ms) than in those with LVEF ≥45% (60.5±41.2 ms, p<0.001). The MD-TES showed moderate correlation with the TDS-MIBI (r=0.55, p<0.001). In a multiple logistic regression analysis, the MD-TES was independently associated with LVEF<45% (odds ratio; 2.63 [95% CI; 1.46-4.77] per 1-decile increase, p=0.001), even after adjusting for age, sex, history of myocardial infarction, the TDS-MIBI, heart rate, QRS interval on electrocardiogram, and other clinical characteristics.

Conclusions: LV dyssynchrony was a significant determinant of reduced LV systolic function and this relationship was independent of the TDS-MIBI and history of myocardial infarction. These results suggest that therapeutic approaches aiming to recover a more synchronous LV contraction (e.g., with cardiac resynchronization therapy) may improve LV systolic function, regardless of the severity of LV myocardial perfusion defect or the presence of coronary artery disease as a cause of HF.