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Heart Failure and Cardiomyopathies

INCREASED PREVALENCE OF LEFT VENTRICULAR NONCOMPACTION IN CERTAIN POPULATIONS MAY CONFER INCREASED RISK OF CATASTROPHIC EVENTS

Poster Contributions
Poster Hall, Hall C
Saturday, March 18, 2017, 9:45 a.m.-10:30 a.m.

Session Title: Advances in HCM, PPCM and Other Cardiomyopathies
Abstract Category: 13. Heart Failure and Cardiomyopathies: Clinical
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Background: Left Ventricular Noncompaction (LVNC) is a known cause of dilated cardiomyopathy (DCM), heart failure (HF) and death. The prevalence of LVNC in the general population is estimated to be between 1/30,000 and 1/5,000. The incidence among patients with LVEF < 45% has been reported to be as high as 3.7% in previous studies. This incidence may be higher in populations that are more genetically homogeneous. Based on clinical observation of a HF population, LVNC as a cause of DCM appears significantly more common than would be predicted for this patient cohort.

Methods: The echocardiographic database at our university-based institution was reviewed. Patients with reduced ejection fraction (<35%) undergoing transthoracic echocardiography from January 2014 through January 2016 were evaluated for the presence of LVNC as defined by American Society of Echocardiography criteria listed: 1) two distinct layers of myocardium 2) prominent deep intratravecular recesses 3) ratio of noncompacted to compacted myocardium >2. If the endocardial definition was questionable, the patients were given IV echocontrast solution. If possible a confirmatory Cardiac MRI was obtained. Patient demographics such as age, gender, race were evaluated for the entire HF population

Results: A total of 312 patients met the criteria for reduced EF HF on echocardiogram, 27 (8.7%) also met phenotypic criteria for LVNC. This rate is 2.3 times higher than that reported in the literature. More interestingly 22/27 (81%) were African American, 4/27 (15%) were Caucasian and 1/27 (4%) were hispanic. Male patients were 24/27 (89%) of the LVNC patients

Conclusions: The prevalence of LVNC in our patient population with reduced EF is 2.3 times greater than reported in other series. The vast majority of these individuals were Aferican American males. In view of a worse prognosis of HF in patients with LVNC, related to increased risk of CVA, life threatening arrhythmia; and reduced likelihood of recovery of ventricular function, this population should be further evaluated for genetic markers. If found these markers could identify at risk individuals prior to phenotypic conversion to DCM HF, or to suffering a cardioembolic cerebrovascular event.