ORIGINAL RESEARCH

Left Ventricular Geometry and Risk of Sudden Cardiac Arrest in Patients With Severely Reduced Ejection Fraction

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***Background*—**Recent reports indicate that specific left ventricular (LV) geometric patterns predict recurrent ventricular arrhythmias in patients with implantable cardioverter-defibrillators and reduced left ventricular ejection fraction (LVEF). However, this relationship has not been evaluated among patients at risk of sudden cardiac arrest (SCA) in the general population.

***Methods and Results*—**Adult SCA cases from the Oregon Sudden Unexpected Death Study were compared with geographic controls with no prior history of SCA. Archived echocardiograms performed closest and prior to the SCA event were reviewed. LV geometry was defined as normal (normal LV mass index [LVMI] and relative wall thickness [RWT]), concentric remodeling (normal LVMI and increased RWT), concentric hypertrophy (increased LVMI and RWT), or eccentric hypertrophy (increased LVMI and normal RWT). Analysis was restricted to those with LVEF ≤40%. A total of 246 subjects were included in the analysis. SCA cases (n=172, 68.6±13.3 years, 78% male), compared to controls (n=74, 66.8±12.1 years, 73% male), had lower LVEF (29.4±7.9% vs 30.8±6.3%, *P*=0.021). Fewer cases presented with normal LV geometry (30.2% vs 43.2%, *P*=0.048) and more with eccentric hypertrophy (40.7% vs 25.7%, *P*=0.025). In a multivariate model, eccentric hypertrophy was independently predictive of SCA (OR 2.15, 95% CI 1.08–4.29, *P*=0.03).

***Conclusions*—**Eccentric LV hypertrophy was independently associated with increased risk of SCA in subjects with EF ≤40%. These findings, now consistent between device-implanted and non-implanted populations, indicate the potential of improving SCA risk stratification from the same noninvasive echocardiogram at no additional cost. **(*J Am Heart Assoc*. 2016;5:e003715 doi: 10.1161/JAHA.116.003715)**

Key Words: eccentric hypertrophy • left ventricular geometry • sudden cardiac arrest

The annual incidence of sudden cardiac arrest (SCA) in the United States is estimated to be over 300 000, with low survival in the range of 5% to 7%.[1,2] The implantable cardioverter-defibrillator (ICD) has been shown to reduce mortality rates from SCA in large randomized clinical trials.[3,4] Based on the current guidelines, the decision for primary prevention ICD placement is largely reliant on measurement of the left ventricular ejection fraction (LVEF).[5] However, there is increasing evidence that LVEF may be inadequate as the sole SCA risk stratifier, and only a minority of patients meeting criteria for ICD implantation receive lifesaving therapies from the device.[6] The process of clinical risk stratification clearly requires further improvement.[7,8] Furthermore, a recent analysis from the Oregon Sudden Unexpected Death Study (Oregon SUDS) found that only a small percentage of those who do meet these criteria receive ICD implantation prior to the SCA event.[9] This emphasizes the need for additional parameters to improve SCA risk stratification, including those who already meet criteria based on LVEF, in the general population.

Left ventricular hypertrophy (LVH) and increased left ventricular (LV) mass are known to be associated with increased risk of cardiovascular disease, death from cardiovascular disease, all-cause mortality, supraventricular and ventricular arrhythmias, and SCA.[10–13] LV mass index (LVMI) along with relative wall thickness (ratio of wall thickness to LV diameter) (RWT) have been employed for classification of 4 LV

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An accompanying Table S1 is available at http://jaha.ahajournals.org/content/5/8/e003715/DC1/embed/inline-supplementary-material-1.pdf

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geometric patterns: normal geometry (normal LVMI and normal RWT), concentric remodeling (normal LVMI and increased RWT), eccentric hypertrophy (increased LVMI and normal RWT), and concentric hypertrophy (increased LVMI and increased RWT).[14] The 4 LV geometry patterns have been shown to confer unique risks for cardiovascular morbidity and all-cause mortality, with concentric hypertrophy typically conferring the highest risk, followed by eccentric hypertrophy, and then concentric remodeling.[15-17] A recent study in subjects with ICDs and reduced LVEF found the magnitude of eccentric geometry, as determined by low versus high RWT, to be a significant predictor of recurrent ventricular arrhythmias.[18] However, to the best of our knowledge, the risk associated with the pattern of LV geometry, specifically eccentric hypertrophy, has not yet been evaluated in subjects with reduced LV function at risk of SCA in the general population. We therefore sought to determine whether different LV geometry patterns are associated with higher SCA risk in patients with reduced LVEF.

Methods

Study Population

SCA cases occurring between February 1, 2002 and January 31, 2015 as well as geographic controls were obtained from subjects in the Oregon Sudden Unexpected Death Study (Oregon SUDS), a prospective study of out-of-hospital cardiac arrest in Portland, Oregon (population ~1 million) ongoing since 2002. Methods for this study have been published in detail previously.[19] Briefly, cases of out-of-hospital cardiac arrest were identified through multiple sources including fire department, ambulance services, local hospital emergency rooms, and the county medical examiner's office. SCA was defined as a sudden, unexpected, pulseless condition of likely cardiac etiology if witnessed, and within 24 hours of last having been seen in usual state of health if unwitnessed. Noncardiac causes of death such as trauma, drug overdose, pulmonary embolism, cerebrovascular accident, or chronic terminal illness were excluded. Both survivors and nonsurvivors of the cardiac arrest event were included in the cases. A 3-physician review of available medical records/autopsy reports was performed for adjudication of SCA. Unmatched geographic controls were used as the comparison group. Because previous community-based studies have shown ≥80% of SCA patients to have associated coronary artery disease (CAD),[20] around 80% of controls included in our analysis had CAD. These subjects were required to have had no history of prior ventricular arrhythmia or cardiac arrest. CAD was defined as having ≥50% stenosis of a major coronary artery, history of myocardial infarction, or history of coronary artery bypass grafting or percutaneous coronary intervention. Hypertension was defined as clinical history of hypertension documented in the medical records. Diabetes mellitus was defined as documented history of diabetes mellitus in the medical records or by the use of insulin or other hypoglycemic agent. Control subjects were ascertained from multiple sources: chest pain patients attended by emergency medical services, outpatient clinics, patients undergoing angiography, and patients from a large health maintenance organization in the Portland metro area.

All subjects aged ≥18 years with echocardiograms available and LVEF ≤40% were included in the analysis. Medical records were reviewed for demographic data and clinical history (age, sex, race, history of diabetes mellitus, chronic kidney disease [CKD], obesity [body mass index ≥30 kg/m^2], and hypertension). All archived reports for echocardiograms performed closest and prior to the SCA event were used for analysis. This study was approved by the Institutional Review Boards of Cedars-Sinai Medical Center, Oregon Health and Science University, and all participating hospitals and health systems. All survivors of sudden cardiac arrest provided informed consent; for nonsurvivors this requirement was waived.

Echocardiogram Analysis

LVEF, LV end-diastolic diameter (LVEDD), interventricular septal thickness at end diastole (IVSd), LV posterior wall thickness at end diastole (PWd), and presence of valvular disease were obtained from echocardiograms. From these values, LV mass was calculated via the linear formula as recommended by the American Society of Echocardiography, $0.8 \times \{1.04([\text{LVEDD}+\text{PWd}+\text{IVSd}]^3-[\text{LVEDD}]^3)\}+0.6$ g,[14] and LVMI was calculated by dividing the LV mass by the body surface area (g/m^2). RWT was calculated by multiplying 2 times PWd divided by LVEDD. A cutoff of 134 g/m^2 for males and 110 g/m^2 for females was used to define an increased LVMI.[21] RWT was defined as increased if ≥0.45.[15] Classification of LV geometry, based on LVMI and RWT, was into normal (normal LVMI and normal RWT), concentric remodeling (normal LVMI and increased RWT), concentric hypertrophy (increased LVMI and RWT), and eccentric hypertrophy (increased LVMI and normal RWT).[14] Subjects with severe aortic stenosis, hypertrophic cardiomyopathy, and LVEF >40% were excluded.

Statistical Analysis

Cases and controls were compared using independent-samples t-tests and chi-squared tests for continuous and categorical variables, respectively. The different LV geometry types in SCA cases were compared using ANOVA and chi-squared test for continuous and categorical variables, respectively. Equality of variances was tested using the Levene test, and for those parameters that did not have equal

variances the nonparametric Kruskal-Wallis test was used instead of ANOVA. A 2-tailed *P* value of ≤0.05 was considered statistically significant. Multivariable logistic regression was used to determine the odds ratio (OR) for independent association between LV geometry and SCA, using normal LV geometry as the reference. Independent models were developed that adjusted for demographic parameters. To determine the independent effect of RWT on SCA risk, we performed a similar analysis for RWT as a continuous variable and a categorical variable using the lowest quartile as the cutoff (<0.31). All analyses were performed using IBM SPSS Statistics for Windows, version 21.0 (IBM Corp, Armonk, NY).

Results

Demographic and Clinical Characteristics

Echocardiographic data on LV geometry were available from 535 cases and 414 controls. After exclusion of subjects with preserved LVEF >40%, a total of 246 subjects (172 cases, 74 controls) were included in the analysis (Table 1). Cases were more likely than controls to have hypertension (77.9% versus 64.9%, *P*=0.03) and CKD (44.2% versus 20.3%, *P*<0.001). There were no significant differences in other demographic and medical history parameters evaluated.

Characteristics of SCA Cases With Different LV Geometry

We compared several parameters across LV geometry types in SCA cases (Table 2). History of prior myocardial infarction was highest in concentric hypertrophy. Otherwise, there were no significant differences in demographics or medical history. LVMI and LVEDD were highest and LVEF lowest in eccentric hypertrophy. IVSd and PWd were highest in concentric hypertrophy. RWT was lowest in normal and eccentric hypertrophy. A similar comparison was made in the unmatched geographic controls (Table S1). Findings were similar, except hypertension prevalence was highest in concentric hypertrophy, and there were no significant differences in history of myocardial infarction or LVEF between groups.

Table 1. Baseline Demographic and Clinical Characteristics of SCA Cases Versus Controls With EF ≤40%

Total (n=246)	Case (n=172)	Control (n=74)	*P* Value
Age, y	68.6±13.3	66.8±12.1	0.32
Male sex	134 (77.9%)	54 (73.0%)	0.40
Race*			0.34
White	141 (82.5%)	62 (87.3%)	
Black	23 (13.5%)	5 (7.0%)	
Other	7 (4.1%)	4 (5.6%)	
Hypertension	134 (77.9%)	48 (64.9%)	0.03
Diabetes mellitus	84 (48.8%)	32 (46.0%)	0.68
CKD	76 (44.2%)	15 (20.3%)	<0.001
Obesity (BMI ≥30 kg/m^2)	67 (39.0%)	32 (43.2%)	0.53

CKD indicates chronic kidney disease; EF, ejection fraction; SCA, sudden cardiac arrest. Data are presented as mean±SD or n (%). BMI indicates body mass index.
*Race data available for 171 cases and 71 controls.

Echocardiographic Characteristics of Cases and Controls

With regard to echocardiographic parameters (Table 3), cases were significantly more likely than controls to have a higher mean LVMI (142.4±43.3 vs 123.2±38.2 g/m^2, *P*<0.001), increased LVMI (>134 g/m^2 for males, >110 g/m^2 for females) (55.8% vs 39.2%, *P*=0.017), higher mean LVEDD (60.1±9.5 vs 56.7±9.0 mm, *P*=0.003), and lower mean LVEF (29.4±7.9% vs 30.8±6.3%, *P*=0.021). There were no significant differences in IVSd, PWd, or RWT between groups.

Risk of SCA Associated With Abnormal LV Geometry

The LV geometry pattern differed significantly in SCA cases compared to controls. Normal LV geometry was significantly less prevalent (30.2% vs 43.2%, *P*=0.048), and eccentric hypertrophy was more prevalent (40.7% vs 25.7%, *P*=0.025) (Figure), in cases compared to controls. There were no significant differences in occurrence of concentric remodeling and concentric hypertrophy between groups.

In multivariable analysis adjusting for age, sex, and race (Table 4), eccentric hypertrophy was independently predictive of SCA, increasing the odds by over 2-fold compared to normal LV geometry (OR 2.15, 95% CI 1.08-4.29, *P*=0.03). Concentric remodeling and concentric hypertrophy were not statistically significant predictors of SCA. When RWT was examined as an independent predictor of SCA, both as a continuous variable and employing a cutoff of <0.31 (lowest quartile in our sample), RWT was not associated with SCA.

Discussion

To the best of our knowledge, there are no existing community-based data on the risk of SCA associated with different LV geometry patterns in patients with reduced LVEF. This is likely to be the first study to report that eccentric hypertrophy is predictive of SCA in subjects with reduced LV function in the general population. The other LV geometry

Table 2. Characteristics by LV Geometry in SCA Cases With EF ≤40%

Total (n=172)	Normal (n=52)	Concentric Remodeling (n=24)	Concentric Hypertrophy (n=26)	Eccentric Hypertrophy (n=70)	*P* Value
Age, y	65.0±12.1	72.0±10.6	72.5±14.0	68.6±14.2	0.056
Male sex	42 (80.8%)	19 (79.2%)	18 (69.2%)	55 (78.6%)	0.700
Race*					0.201
White	42 (80.8%)	23 (100%)	21 (80.8%)	55 (78.6%)	
Black	8 (15.4%)	0 (0%)	8 (15.4%)	10 (14.3%)	
Other	2 (3.9%)	0 (0%)	0 (0%)	5 (7.1%)	
CAD	40 (76.9%)	19 (79.2%)	23 (88.5%)	52 (74.3%)	0.519
Hypertension	40 (76.9%)	19 (79.2%)	19 (73.1%)	56 (80.0%)	0.901
Diabetes mellitus	24 (46.2%)	15 (62.5%)	15 (57.7%)	30 (42.9%)	0.289
CKD	17 (32.7%)	11 (45.8%)	15 (57.7%)	33 (47.1%)	0.173
Obesity (BMI ≥30 kg/m^2)	20 (38.5%)	11 (45.8%)	14 (53.9%)	22 (31.4%)	0.206
History of MI	35 (67.3%)	15 (62.5%)	22 (84.6%)	38 (54.3%)	0.048
Echocardiographic parameters					
LV mass index, g/m^2	104.0±16.6	107.7±21.2	167.2±36.7	173.7±32.5	<0.001
LVEDD, mm	58.2±7.1	48.9±6.7	55.7±5.7	67.1±7.5	<0.001
LVEF, %	29.9±7.7	32.4±6.4	31.5±6.4	27.1±8.6	0.010
IVSd, mm	9.4±1.6	11.8±1.7	13.8±2.8	11.7±2.6	<0.001
PWd, mm	9.2±1.4	11.6±1.1	13.7±1.4	10.7±1.6	<0.001
RWT	0.32±0.06	0.48±0.07	0.50±0.08	0.32±0.06	<0.001

Data are presented as mean±SD or n (%). BMI indicates body mass index; CAD, coronary artery disease; CKD, chronic kidney disease; EF, ejection fraction; IVSd, interventricular septum in diastole; LV, left ventricular; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; LVH, left ventricular hypertrophy; MI, myocardial infarction; PWd, posterior wall in diastole; RWT, relative wall thickness (calculated as: [2×PWd]/LVEDD); SCA, sudden cardiac arrest.
*Race data available for 52 normal, 23 concentric remodeling, 26 concentric hypertrophy, 70 eccentric hypertrophy.

patterns, concentric remodeling and concentric hypertrophy, were not found to be significantly associated with SCA. These findings indicate that LV eccentric hypertrophy confers increased risk of SCA independent of reduced LVEF, and both can be measured from the same noninvasive echocardiogram, potentially providing enhanced clinical utility at no additional cost. Following on the recently published similar findings in a primary prevention device population, the Multicenter Automatic Defibrillator Implantation Trial with Cardiac Resynchronization Therapy (MADIT-CRT),[18] these data from nonimplanted patients who suffered SCA indicate the significant potential of this marker to improve clinical risk stratification.

Abnormal LV geometry has also been studied in the context of overall cardiovascular morbidity and mortality and shown to be associated with worse prognosis and outcomes. Analysis from 3216 subjects in the Framingham Heart Study found that event rates of cardiovascular disease or death were highest in concentric hypertrophy, followed by eccentric hypertrophy, then concentric remodeling, and normal geometry.[16] Another analysis from 5098 subjects in the Multi-Ethnic-Study of Atherosclerosis (MESA) showed LV geometry based on cardiac MRI was a better predictor of stroke and coronary heart disease compared to LV mass alone.[22] Similar associations were reported in several other disease populations, such as those with CAD,[17] post–myocardial infarction,[23] atrial fibrillation,[24] hypertension,[15,25-28] CKD,[27] preserved LV function,[28-30] and advanced age.[31] However, most recently an analysis among patients with LVEF ≤30% enrolled in the MADIT-CRT study found the magnitude of eccentric remodeling to be predictive of risk of recurrent ventricular arrhythmias.[18] Now, our findings provide additional insight into the relationship between LV geometry and SCA among subjects with reduced LV function in the general population.

Among the SCA cases in our study, 30% had normal geometry, and 41% had eccentric hypertrophy on echocardiograms prior to the event. In contrast, controls had significantly more subjects with normal geometry (43%) and fewer with eccentric hypertrophy (26%). There were no significant differences in concentric remodeling and hypertrophy prevalence compared between groups. On comparison between LV geometry patterns, the eccentric hypertrophy subgroup was observed to have the lowest value of LVEF.

Table 3. Comparison of Echocardiographic Characteristics of SCA Cases and Controls With EF ≤40%

	Case (n=172)	Control (n=74)	*P* Value
LV mass index, g/m^2	142.4±43.3	123.2±38.2	<0.001
Increased LV mass index	96 (55.8%)	29 (39.2%)	0.017
LVEDD, mm	60.1±9.5	56.7±9.0	0.003
LVEF, %	29.4±7.9	30.8±6.3	0.021
IVSd, mm	11.3±2.6	10.9±2.7	0.143
PWd, mm	10.8±2.1	10.3±2.3	0.061
RWT	0.37±0.10	0.37±0.10	0.883
RWT ≥0.45	50 (29.1%)	23 (31.1%)	0.752
LV geometry			
Normal	52 (30.2%)	32 (43.2%)	0.048
Concentric remodeling	24 (14.0%)	13 (17.6%)	0.467
Concentric hypertrophy	26 (15.1%)	10 (13.5%)	0.744
Eccentric hypertrophy	70 (40.7%)	19 (25.7%)	0.025

Data are presented as mean±SD or n (%). EF indicates ejection fraction; IVSd, interventricular septum in diastole; LV, left ventricular; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; LVH, left ventricular hypertrophy; PWd, posterior wall in diastole; RWT, relative wall thickness; SCA, sudden cardiac arrest.

Table 4. Odds Ratios of SCA Associated With Eccentric Hypertrophy

	OR	95% CI	*P* Value
Unadjusted (n=246)			
Normal	1	1	—
Concentric remodeling	1.14	0.51 to 2.54	0.756
Concentric hypertrophy	1.60	0.68 to 3.75	0.280
Eccentric hypertrophy	2.27	1.16 to 4.44	0.017
Model 1 (n=246)			
Normal	1	1	—
Concentric remodeling	1.06	0.47 to 2.39	0.890
Concentric hypertrophy	1.62	0.68 to 3.84	0.278
Eccentric hypertrophy	2.26	1.15 to 4.45	0.018
Model 2 (n=242)			
Normal	1	1	—
Concentric remodeling	0.98	0.43 to 2.25	0.958
Concentric hypertrophy	1.56	0.63 to 3.86	0.337
Eccentric hypertrophy	2.15	1.08 to 4.29	0.030

Model 1: adjusted for age and sex. Model 2: adjusted for age, sex, and race. CI indicates confidence interval; OR, odds ratio; SCA, sudden cardiac arrest.

Eccentric hypertrophy was found to increase risk of SCA by over 2-fold, even when adjusted for demographic parameters. These findings suggest that abnormal LV geometry, specifically eccentric hypertrophy, increases risk for SCA, which may aid in further SCA risk stratification in subjects with reduced LV function in the general population.

A potential explanation for eccentric hypertrophy increasing risk for SCA compared to the other LV geometry patterns may be related to the increased arrhythmogenic risk associated specifically with eccentric hypertrophy.[32] This has been supported in the literature by a recent study from Draper et al that looked at ventricular tachycardia or fibrillation (VT/VF) occurrence in subjects with reduced LV function in the general population.[33] They found among the 127 patients studied, occurrence of ventricular arrhythmias was highest in those with eccentric hypertrophy, compared to concentric remodeling/hypertrophy and normal geometry. The MADIT-CRT study found decreased RWT in patients with eccentric hypertrophy to be associated with higher risk of ventricular arrhythmias compared to those with higher RWT.[18] Enlargement in LV size could potentially be the driving force behind increased arrhythmogenesis in eccentric hypertrophy as well as decreased RWT. LV diameter has been shown to be independently predictive of SCA and to have an additive effect with LVEF on predicting SCA risk.[34,35] Two studies, 1 in patients post–myocardial infarction[36] and the other in patients with severe LV dysfunction,[37] found that increased LV size was predictive of ventricular arrhythmias and frequent premature ventricular contractions (PVC). This association was further supported in a study that found reduction of LV end-systolic size in patients undergoing cardiac resynchronization therapy reduced occurrence of PVCs and VT/VF events.[38] With eccentric hypertrophy commonly occurring secondary to an increase in preload volume and resulting in enlargement of the LV, the increased ventricular arrhythmic

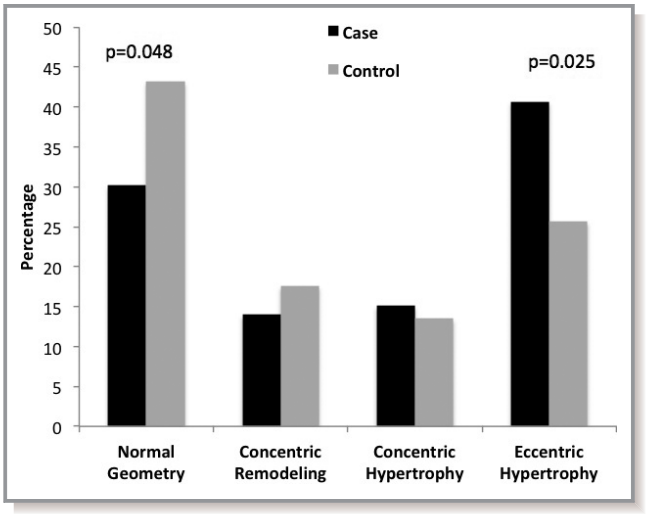

Figure. Distribution of LV geometry patterns in sudden cardiac arrest case versus control subjects. Cases were significantly more likely to have eccentric LV hypertrophy. *P* values were obtained using chi-squared test for each LV geometry type, with a value of ≤0.05 indicating a statistically significant difference.

risk associated with this LV geometric pattern is a potential explanation linking the association we found between eccentric hypertrophy and SCA.

It is possible that adverse myocardial interstitial remodeling could have a role in increasing arrhythmic risk in eccentric hypertrophy. Increased interstitial collagen has been found in diseased hearts, such as in noninfarcted tissue in myocardial infarction or hypertensive hypertrophy.[39] Presence of fibrosis can create conditions that promote reentry and ventricular arrhythmogenesis. This has been supported in a study looking at patients with nonischemic dilated cardiomyopathy that found presence of fibrosis on cardiac magnetic resonance increases risk of SCA, ICD shocks, nonfatal VF, and sustained VT.[40] However, concentric hypertrophy and eccentric hypertrophy have different collagen remodeling patterns.[41] These different remodeling patterns may carry unique arrhythmic risks. Abnormal myocardial fibrosis, common to all forms of LVH, is the leading substrate for ventricular arrhythmogenesis. However, due to ventricular wall thinning and dilatation, increased wall stress may further increase arrhythmic risk in patients with eccentric hypertrophy, even at a relatively late stage of LV remodeling. Further studies would be needed to explore this hypothesis.

Limitations

Given the relatively infrequent occurrence of SCA compared to the size of the general population, a case-control design was employed that has some inherent limitations. Analysis was restricted to subjects with appropriate echocardiograms available, and since SCA may occur as the first presentation or evidence for CAD, sampling of subjects may be biased. Furthermore, echocardiograms used in this study were obtained based on routine clinical practice, and thus, reproducibility of the measurements could not be assessed. Also, as expected, the control subjects had lower rates of echocardiography as well as a lower proportion of severe LV systolic dysfunction. Multivariable models were developed; however, for any observational study, unknown confounders cannot be excluded with certainty. The strength of this study is the community-based prospective ascertainment of SCA cases for sampling of this adverse event in the general population.

Conclusion

Eccentric hypertrophy is independently predictive of SCA in subjects with EF ≤40% in the general population. Given the well-recognized limitations of using LVEF as the sole risk stratifier and the recent similar observations made from the MADIT-CRT population, our findings suggest that evaluation of LV geometry may supplement and enhance LVEF-based SCA risk stratification.

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Disclosures

None.

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SUPPLEMENTAL MATERIAL

Table S1. Characteristics by LV geometry in controls with EF ≤40%

Total (n=74)	Normal (n= 32)	Concentric Remodeling (n= 13)	Concentric Hypertrophy (n= 10)	Eccentric Hypertrophy (n= 19)	P Value
Age (years)	68.3±9.4	67.1±13.1	66.0±13.2	64.6±15.3	0.764
Male Sex	24 (75.0%)	12 (92.3%)	5 (50.0%)	13 (68.4%)	0.144
Race*					0.473
Caucasian	28 (93.3%)	11 (84.6%)	7 (77.8%)	16 (84.2%)	
African American	1 (3.3%)	1 (7.7%)	2 (22.2%)	1 (5.3%)	
Other	1 (3.3%)	1 (7.7%)	0 (0.0%)	2 (10.5%)	
Coronary Artery Disease	24 (75%)	10 (76.9%)	6 (60.0%)	18 (94.7%)	0.156
Hypertension	19 (59.4%)	6 (46.2%)	10 (100%)	13 (68.4%)	0.047
Diabetes	14 (43.8%)	5 (38.5%)	6 (60.0%)	9 (47.4%)	0.761
Chronic Kidney Disease	7 (21.9%)	5 (38.5%)	0 (0.0%)	3 (15.8%)	0.139
Obesity (BMI ≥30 kg/m^2)	13 (40.6%)	7 (53.9%)	60 (60.0%)	6 (31.6%)	0.410
History of MI	20 (62.5%)	7 (53.9%)	6 (60.0%)	14 (73.7%)	0.696
Echocardiographic parameters					
LV Mass Index (g/m^2)	99.5±18.3	99.3±17.2	157.3±25.2	161.6±34.8	<0.001
LVEDD (mm)	57.0±9.4	47.9±4.9	55.0±4.6	63.2±6.8	<0.001
LVEF (%)	31.6±7.2	30.8±4.9	31.7±5.2	29.2±5.9	0.581
IVSd (mm)	9.5±1.8	11.3±1.9	13.5±2.1	11.6±3.5	<0.001
PWd (mm)	8.6±1.6	11.7±1.2	13.5±1.6	10.7±1.4	<0.001
RWT	0.31±0.08	0.49±0.06	0.49±0.08	0.34±0.04	<0.001

Data are presented as mean ± SD or n (%). BMI-body mass index; LV-left ventricular; LVEDD-left ventricular end-diastolic diameter; LVEF- left ventricular ejection fraction; MI-myocardial infarction; IVSd-interventricular septum in diastole; PWd-posterior wall in diastole; RWT-relative wall thickness (calculated as: [2 x PWd]/LVEDD); LVH-left ventricular hypertrophy

[*]Race data available for 30 normal, 13 concentric remodeling, 9 concentric hypertrophy, 19 eccentric hypertrophy