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Association of a 4-Tiered Classification of LV Hypertrophy With Adverse CV Outcomes in the General Population

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ABSTRACT

OBJECTIVES This study was performed to determine whether a 4-tiered classification of left ventricular hypertrophy (LVH) defines subgroups in the general population that are at variable risks of adverse cardiovascular (CV) outcomes.

BACKGROUND We recently proposed a 4-tiered classification of LVH where eccentric LVH is subdivided into "indeterminate hypertrophy" and "dilated hypertrophy" and concentric LVH into "thick hypertrophy" and "both thick and dilated hypertrophy," based on the presence of increased left ventricular (LV) end-diastolic volume.

METHODS Participants from the Dallas Heart study who underwent cardiac magnetic resonance and did not have LV dysfunction or a history of heart failure (HF) (n = 2,458) were followed for a median of 9 years for the primary outcome of HF or CV death. Multivariable Cox proportional hazards models were used to adjust for age, sex, African-American race, hypertension, diabetes, and history of CV disease.

RESULTS In the cohort, 70% had no LVH, 404 (16%) had indeterminate hypertrophy, 30 (1%) had dilated hypertrophy, 289 (12%) had thick hypertrophy, and 7 (0.2%) had both thick and dilated hypertrophy. The cumulative incidence of HF or CV death was 2% with no LVH, 1.7% with indeterminate, 16.7% with dilated, 11.1% with thick, and 42.9% with both thick and dilated hypertrophy (log-rank $p < 0.0001$). Compared with participants without LVH, those with dilated (hazard ratio [HR]: 7.3; 95% confidence interval [CI]: 2.8 to 18.8), thick (HR: 2.4; 95% CI: 1.4 to 4.0), and both thick and dilated (HR: 5.8; 95% CI: 1.7 to 19.5) hypertrophy remained at increased risk for HF or CV death after multivariable adjustment, whereas the group with indeterminate hypertrophy was not (HR: 0.9; 95% CI: 0.4 to 2.2).

CONCLUSIONS In the general population, the 4-tiered classification system for LVH stratified LVH into subgroups with differential risk of adverse CV outcomes. (J Am Coll Cardiol Img 2015;8:1034–41) © 2015 by the American College of Cardiology Foundation.

Left ventricular hypertrophy (LVH), as defined by increased left ventricular (LV) mass, is associated with significant cardiovascular (CV) morbidity and mortality (1–4). LVH assessed by echocardiography is often categorized into 2 patterns based on the relative wall thickness, a ratio derived from LV wall thickness and LV chamber dimension. LVH with increased relative wall thickness is classified as concentric; when the relative wall thickness is not increased, LVH is classified as eccentric (5). Though widely used, this classification system has important limitations, including relying on a ratio of

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linear dimensions for wall thickness and chamber size and not accounting for LV dilation in isolation, an important aspect of geometric remodeling.

We have previously proposed a 4-tiered classification of LVH based on LV end-diastolic volume (EDV) and concentricity (a marker of wall thickness) as assessed by cardiac magnetic resonance (CMR) (6). In this classification, eccentric hypertrophy was further divided into dilated hypertrophy and indeterminate hypertrophy based on whether the LV volume was increased. Similarly, concentric hypertrophy was divided into thick hypertrophy and both thick and dilated hypertrophy (**Figure 1**) (6).

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In our initial cross-sectional description of this classification system, we demonstrated clear phenotypic differences between the 2 newly proposed subcategories of both eccentric and concentric hypertrophy. Those with dilated hypertrophy were more likely to have elevated levels of natriuretic peptides and lower LV ejection fraction (EF) than those with indeterminate hypertrophy. Similarly, those with both thick and dilated hypertrophy had a higher prevalence of reduced LVEF than those with isolated thick hypertrophy. Subsequent studies have related the 4-tiered classification system to clinical outcomes in patients with hypertension and coronary artery disease (7,8). However, these studies used echocardiography, which has known limitations in assessing LV mass and LV volume when compared with CMR (5,9). Moreover, this classification system has not yet been related to clinical outcomes in an unselected general population sample.

Therefore, we classified participants from the Dallas Heart Study with the 4-tiered classification system for LVH using CMR and determined associations of the 4-tiered subgroups with incident heart failure (HF) and CV death. In addition, we compared levels of high-sensitivity cardiac troponin T (hs-cTnT), a marker of cardiac injury shown to be associated with HF and death (10–12), across the subgroups.

METHODS

STUDY POPULATION. The Dallas Heart Study is a multiethnic, population-based, cohort study of Dallas County adults in which deliberate oversampling of African Americans was performed. The design and detailed methods of the Dallas Heart Study have been previously described (13). In brief, the study was conducted in 3 visits. Visit 1 was an initial in-home visit (n = 6,101) in which demographics, medical history, and blood pressure were obtained. This was followed by collection of fasting blood and urine samples at a second in-home visit (n = 3,557). Visit 3 was conducted on the campus of University of Texas Southwestern Medical Center, during which detailed imaging studies including CMR were performed (n = 2,803). Participants were subsequently followed for pre-defined clinical events and death. For this study, we excluded participants with a LVEF <40% and those with a clinical history of HF at baseline, resulting in a final cohort of 2,458 participants. Written informed consent was provided by all participants, and the University of Texas Southwestern Institutional Review Board approved the study.

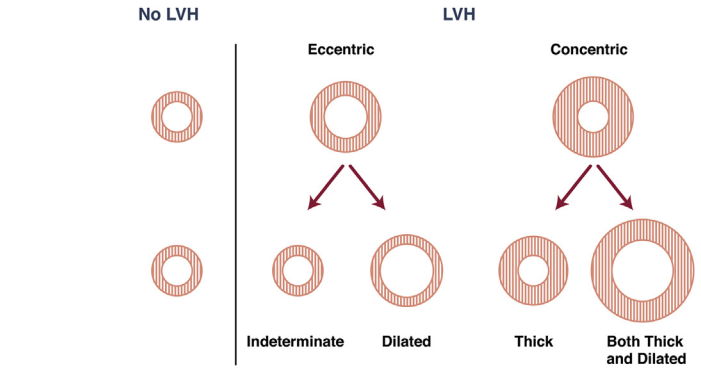
CMR. CMR was performed using 2 comparable 1.5-T systems (Phillips Medical Systems, Best, the Netherlands). As previously described, mass and volume measurements were calculated from short-axis breath-hold electrocardiography-gated cine CMR and MASS software (Medis Medical Imaging Systems, Leiden, the Netherlands) was used to analyze data (14). The papillary muscles were included in the mass of the left ventricle. The mean wall thickness of the left ventricle was determined by averaging the wall thickness of each slice, excluding the apical slice. Further details of the CMR protocol have been previously published including intraobserver, interobserver, and interscan variability (14,15).

DEFINITIONS. For the primary analysis, LVH was classified as increased LV mass when indexed to height$^{2.7}$ using thresholds of ≥48 g/m$^{2.7}$ for men and ≥39 g/m$^{2.7}$ for women (14). A sensitivity analysis was performed indexing LV mass to body surface area (BSA) using thresholds of ≥112 g/m^2 for men and ≥89 g/m^2 for women. LV concentricity$^{0.67}$ was defined as LV mass/LV end-diastolic volume (EDV)$^{0.67}$, as previously described (6). Previously defined thresholds for elevated LVEDV indexed to BSA (≥74 ml/m^2 for men and ≥68 ml/m^2 for women) and LV concentricity$^{0.67}$ (≥9.1 g/ml$^{0.67}$ for men and ≥8.1 g/ml$^{0.67}$ for women) were used (6). The 4 categories of LVH were: 1) indeterminate hypertrophy (neither increased concentricity$^{0.67}$ nor increased LVEDV/BSA); 2) dilated hypertrophy (increased LVEDV/BSA without increased concentricity$^{0.67}$); 3) thick hypertrophy (increased concentricity$^{0.67}$ without increased LVEDV/BSA); and 4) both thick and dilated hypertrophy (increased concentricity$^{0.67}$ and LVEDV/BSA). To contrast the 4-tiered classification with the prior 2-tiered classification, we defined LVH in the 2-tiered classification as concentric when concentricity$^{0.67}$ was increased and eccentric when concentricity$^{0.67}$ was not increased (6).

ABBREVIATIONS AND ACRONYMS

BSA = body surface area
CI = confidence interval
CMR = cardiac magnetic resonance
CV = cardiovascular
EDV = end-diastolic volume
HF = heart failure
HR = hazard ratio
hs-cTnT = high-sensitivity cardiac troponin T
LV = left ventricular
LVH = left ventricular hypertrophy

FIGURE 1 Schematic of Standard 2-Tier and 4-Tier Classification for LVH



Increased LV Mass/Height$^{2.7}$	–	+	+	+	+
Increased Concentricity$^{0.67}$	NA	–	–	+	+
Increased LV EDV/BSA	NA	–	+	–	+

Both of the standard 2-tiered subgroups are subclassified based on the presence of increased indexed LV volume. Reprinted with permission from Khouri et al. (6). BSA = body surface area; EDV = end-diastolic volume; LV = left ventricular; LVH = left ventricular hypertrophy.

Hs-cTnT (Elecys-2010 Troponin T hs STAT, Roche Diagnostics, Indianapolis, Indiana) was measured from baseline samples as previously described (10). Elevated hs-cTnT was defined as equal to or greater than the limit of blank of the assay (>3 ng/l).

OUTCOMES. The primary outcome was the composite of incident HF or CV death. Incident HF was defined as first hospitalization for HF with reduced or preserved EF. A blinded endpoint committee adjudicated nonfatal CV events (including HF). The secondary endpoints were CV death alone and incident HF alone. Death events were determined through December 31, 2010, from the National Death Index and classified as cardiovascular based on the International Classification of Diseases-10 codes I00 to I99 (16).

STATISTICAL ANALYSIS. Statistical comparisons of variables among the 4 groups—indeterminate, dilated, thick, and both thick and dilated—were done using the chi-square test for dichotomous variables and Wilcoxon rank sum test for continuous variables. No adjustments were made for multiple comparisons. The incidence of the primary outcome among each group was estimated using Kaplan-Meier analysis. Multivariable Cox proportional hazards models were used to adjust for age, sex, African-American race, hypertension, diabetes, and history of CV disease. Because of the limited number of events, secondary endpoints of CV death alone and incident HF alone were not adjusted. Four sensitivity analyses were performed, including repeating the primary analyses using LVH defined by LV mass indexed to BSA; using the continuous parameter of systolic blood pressure instead of hypertension as a covariate; including body mass index as a covariate; and repeating the primary analysis after further excluding all patients with a baseline history of CV disease. All statistical analyses were performed with SAS version 9.1 (SAS Institute, Inc., Cary, North Carolina) statistical software and all p values are 2-sided with an alpha of 0.05.

RESULTS

The baseline characteristics of the study cohort are shown in Table 1, stratified by the 4-tiered classification system for LVH. Among the 2,458 participants meeting study criteria (mean age 44 years; 56% women, 48% African American), 730 (30%) had LVH, of whom 404 were classified as indeterminate, 30 as dilated hypertrophy, 289 as isolated thick hypertrophy, and 7 as having both thick and dilated hypertrophy. In the study group, 773 (31%) of the participants had hypertension and 245 (10%) had diabetes, with a higher proportion of hypertension and diabetes seen in the participants with isolated thick and both thick and dilated LVH.

When using the standard 2-tiered LVH classification, the prevalence of detectable hs-cTnT in participants with eccentric LVH was lower compared with those without LVH (18% vs. 24%, p = 0.005) (Figure 2A). In contrast, when eccentric hypertrophy was subdivided in the 4-tiered classification (Figure 2B), the dilated subgroup had a higher prevalence of detectable hs-cTnT (43%) as compared with those without LVH (24%, p = 0.02) or those with indeterminate hypertrophy (16%, p = 0.0004). Similarly, subjects with both thick and dilated hypertrophy were more likely to have elevated hs-cTnT than those with isolated thick hypertrophy (100% vs. 42%, p = 0.002).

Over a median follow up period of 9.1 (interquartile range: 8.6 to 9.6) years, the primary composite outcome of incident HF or CV death occurred in 81 (3.3%; 95% confidence interval [CI]: 2.6 to 4.1) participants, including 35 HF events and 47 CV deaths. In the 2-tiered classification system, the cumulative incidence of HF or CV death in participants with concentric LVH was 11.8% (95% CI: 8.1 to 15.6) compared with 2.8% (95% CI: 1.2 to 4.3) in the eccentric LVH group and 2.0% (95% CI: 1.3 to 2.7) in the group with no LVH (log-rank p < 0.0001).

TABLE 1 Baseline Characteristics of the Study Population

		LVH			
		Eccentric		Concentric	
	No LVH (n = 1,728)	Indeterminate (n = 404)	Dilated (n = 30)	Thick (n = 289)	Both (n = 7)
Age, yrs	43 (36, 52)	43 (36, 51)	43 (35, 46)	49 (42, 56)*	53 (46, 58)
Men	52	14*	43	41*	86
Black	41	55*	73*	79*	71
BMI, kg/m^2	28 (24, 31)	35 (31, 40)*	27 (24, 35)	34 (30, 40)*	33 (29, 45)†
Hypertension	23	37	37	74*	86*
SBP, mm Hg	122 (112, 131)	125 (115, 138)*	131 (122, 141)†	141 (128, 155)*	145 (128, 159)†
DBP, mm Hg	76 (71, 82)	79 (73, 85)*	76 (74, 83)	85 (78, 92)*	86 (76, 89)
Diabetes	7	11	7	24*	57*
CVD	3	3	3	10*	14
eGFR, ml/min/1.73 m^2	96 (84, 109)	103 (91, 116)*	111 (95, 136)*	95 (82, 110)	97 (81, 111)
LVEF, %	73 (68, 77)	75 (71, 80)*	72 (59, 76)	72 (67, 78)	62 (45, 76)
LV mass/height$^{2.7}$	36 (32, 40)	44 (41, 49)*	53 (48, 60)*	52 (47, 57)*	66 (64, 69)*
LV mass/BSA, g/m^2	77 (67, 89)	78 (73, 86)*	109 (93, 126)*	100 (86, 116)*	144 (125, 146)*
LVEDV/BSA, ml/m^2	51 (45, 57)	53 (48, 58)*	81 (71, 92)*	48 (41, 56)*	80 (74, 88)*
Concentricity$^{0.67}$, g/ml$^{0.67}$	7 (6, 8)	7 (7, 8)	7 (6, 8)	10 (9, 11)*	10 (9, 10)*
LV wall thickness, mm	11 (10, 12)	11 (11, 12)*	12 (11, 13)†	14 (13, 15)*	15 (15, 16)*

Values are median (25%, 75% percentile) or %. *p < 0.001 versus no LVH group. †p < 0.01 versus no LVH group.

BMI = body mass index; BSA = body surface area; CVD = cardiovascular disease; DBP = diastolic blood pressure; eGFR = estimated glomerular filtration rate; LV = left ventricular; LVEDV = left ventricular end-diastolic volume; LVEF = left ventricular ejection fraction; LVH = left ventricular hypertrophy; SBP = systolic blood pressure.

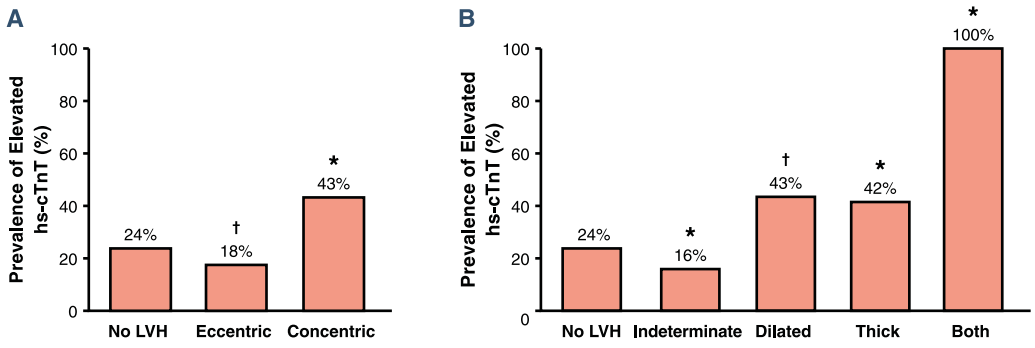
No significant difference in the primary endpoint was seen between the eccentric LVH and no LVH groups (p = 0.31) (Figure 3A). In the 4-tiered classification system, the cumulative incidence of HF or CV death was 2.0% (95% CI: 1.3 to 2.7) with no hypertrophy, 1.7% (95% CI: 0.47 to 3.0) with indeterminate hypertrophy, 16.7% (95% CI: 3.0 to 30.4) with dilated hypertrophy, 11.1% (95% CI: 7.4 to 14.8) with isolated thick hypertrophy, and 42.9% (95% CI: 6.2 to 79.5) in those with both thick and dilated hypertrophy (log-rank p < 0.0001) (Figure 3B). There was no significant difference in the risk of HF or CV death between those with indeterminate hypertrophy and those without LVH (p = 0.74).

In multivariable analyses, using the 2-tiered classification, those with concentric LVH but not eccentric were at increased risk of HF or CV death (Table 2). When applying the 4-tiered classification of LVH, eccentric LVH was stratified into lower risk (indeterminate hypertrophy) and higher risk (dilated hypertrophy) subgroups. Specifically, as compared with those without LVH, participants with indeterminate hypertrophy were not at increased risk, whereas those with dilated hypertrophy were at significantly increased risk of HF or CV death (Table 2). Similarly, compared with participants with no LVH, isolated thick hypertrophy and both thick and dilated hypertrophy remained associated with increased HF and CV death (Table 2). Dilated hypertrophy was associated with increased risk compared with isolated thick hypertrophy (hazard ratio [HR]: 3.1; 95% CI: 1.2 to 8.0). Dilated, thick, and both thick and dilated hypertrophy remained independently associated with adverse CV outcomes when hs-cTnT was added to the multivariable adjustment (Table 2). Finally, the continuous parameters of LVEDV (HR: 1.5; 95% CI: 1.2 to 1.9), LV wall thickness (HR: 1.4; 95% CI, 1.2 to 1.7), and hs-cTnT (HR: 1.2; 95% CI: 1.02 to 1.5) were independently associated with increased risk of HF or CV death in multivariable analysis adjusted for age, sex, African-American race, hypertension, diabetes, and history of CV disease.

Secondary endpoints of incident HF alone and CV death alone are shown in Table 3. In unadjusted analysis, indeterminate hypertrophy was not associated with increased risk of either HF alone or CV death alone when compared with no LVH. Thick, dilated, and both thick and dilated hypertrophy were associated with increased risk of incident HF compared with no LVH. Thick hypertrophy and both thick and dilated hypertrophy were associated with increased risk of CV death alone.

In a sensitivity analysis in which LVH was defined based on LV mass indexed to BSA (Online Table 1), there were no events among the 35 subjects with indeterminate LVH, whereas dilated LVH remained associated with the outcome in both unadjusted (HR: 11.9; 95% CI: 4.7 to 30.1) and adjusted models

FIGURE 2 Prevalence of Elevated hs-cTnT



Prevalence of elevated hs-cTnT in the **(A)** 2-tiered and **(B)** 4-tiered classification of LVH. *p < 0.001 versus no LVH group. †p < 0.02 versus no LVH group. hs-cTnT = high-sensitivity cardiac troponin T; LVH = left ventricular hypertrophy.

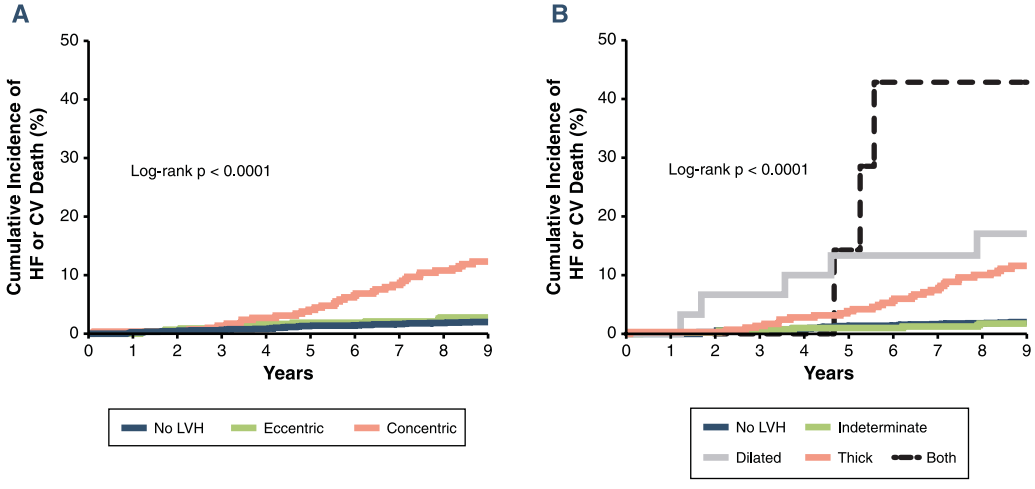
(HR: 8.3; 95% CI: 3.2 to 21.0). Similarly, thick and both thick and dilated hypertrophy remained associated with adverse CV outcomes (Online Table 1). Additional sensitivity analyses excluding participants with a history of CV disease, using systolic blood pressure as a covariate in place of hypertension, and adjusting for body mass index revealed similar associations (data not shown).

DISCUSSION

The present study demonstrates that individuals from the general population with concentric or eccentric LVH, as defined by the standard 2-tiered classification system, can be subclassified based on the presence or absence of LV dilation into 2 further subgroups with distinct longitudinal trajectories. Participants with eccentric hypertrophy can be subclassified into a low-risk (indeterminate hypertrophy) and a high-risk group (dilated hypertrophy). Similarly, concentric hypertrophy can be divided into 2 at risk groups: thick hypertrophy and both thick and dilated hypertrophy.

In our initial description of the 4-tiered classification system, we demonstrated that participants with indeterminate hypertrophy did not have a reduced LVEF, elevated natriuretic peptides, or higher frequency of detectable cTnT by conventional assay

FIGURE 3 Kaplan-Meier Curves for Incident HF or CV Death



Unadjusted Kaplan-Meier curves for incident HF or CV death stratified by **(A)** 2-tiered and **(B)** 4-tiered classification of LVH. CV = cardiovascular; HF = heart failure; LVH = left ventricular hypertrophy.

TABLE 2 Unadjusted and Multivariable Adjusted Associations of 2- and 4-Tiered Classification of LVH with Incident HF or CV Death

LVH Classification	n	E	Unadjusted HR (95% CI)	Adjusted HR (95% CI)	Adjusted HR† (95% CI)
2-Tiered					
No LVH*	1,728	34	1.0	1.0	1.0
Eccentric LVH	434	12	1.4 (0.7–2.7)	1.5 (0.8–3.1)	1.4 (0.7–2.9)
Concentric LVH	296	35	6.3 (3.9–10.1)	2.5 (1.5–4.2)	2.2 (1.3–3.8)
4-Tiered					
No LVH*	1,728	34	1.0	1.0	1.0
Indeterminate	404	7	0.9 (0.4–2.0)	0.9 (0.4–2.2)	0.9 (0.4–2.1)
Dilated	30	5	9.5 (3.7–24.2)	7.3 (2.8–18.8)	5.5 (2.0–14.9)
Thick	289	32	5.9 (3.6–9.5)	2.4 (1.4–4.0)	2.2 (1.3–3.7)
Both	7	3	26.8 (8.2–87.3)	5.8 (1.7–19.5)	4.6 (1.4–15.7)

LVH is indexed to height$^{2.7}$. *Referent group. Adjusted hazard ratio includes adjustments for age, sex, race, diabetes, hypertension, and CVD. †Adjusted hazard ratio (HR) also includes added adjustment for high-sensitivity cardiac troponin T.

CI = confidence interval; E = number of events; n = number of participants; other abbreviations as in Table 1.

when compared with those without LVH (6). The hs-cTnT assay is approximately 10-fold more sensitive than the conventional assay in the assessment of cardiac injury and has been strongly associated with abnormalities in cardiac structure and function and subsequent mortality (10,11). In our present study, we found that participants with indeterminate hypertrophy had a lower prevalence of detectable hs-cTnT and no increased risk of HF or CV death compared with participants without hypertrophy.

TABLE 3 Unadjusted Associations of 2- and 4-Tiered Classification of LVH With Incident HF and CV Death

	Unadjusted Incident HF		
LVH Classification	**n**	**E**	**HR (95% CI)**
2-Tiered			
No LVH*	1,728	7	1.0
Eccentric LVH	434	7	4.0 (1.4–11.3)
Concentric LVH	296	21	18.6 (7.9–43.7)
4-Tiered			
No LVH*	1,728	7	1.0
Indeterminate	404	3	1.8 (0.5–7.0)
Dilated	30	4	37.2 (10.9–127.0)
Thick	289	18	16.2 (6.8–38.9)
Both	7	3	135.4 (34.9–524)
	Unadjusted CV Death		
LVH Classification	**n**	**E**	**HR (95% CI)**
2-Tiered			
No LVH*	1,728	27	1.0
Eccentric LVH	434	5	0.7 (0.3–1.9)
Concentric LVH	296	15	3.3 (1.8–6.2)
4-Tiered			
No LVH*	1,728	27	1.0
Indeterminate	404	4	0.6 (0.2–1.8)
Dilated	30	1	2.2 (0.3–16.1)
Thick	289	14	3.2 (1.7–6.0)
Both	7	1	9.6 (1.3–70.3)

LVH is indexed to height$^{2.7}$. *Referent group.

Abbreviations as in Tables 1 and 2.

Prior studies by others looking at the 4-tiered classification system assessed by echocardiography have also shown that indeterminate hypertrophy was not associated with adverse outcomes in individuals with pre-existing hypertension or coronary artery disease (7,8). CMR has improved accuracy and inter-study reproducibility in the assessment of LV mass (17,18). As such, our results provide further evidence suggesting that indeterminate hypertrophy is a benign phenotype, a finding that has important implications given the strong association of indeterminate hypertrophy with obesity, which is increasingly prevalent in the population.

Recently, we showed that low circulating concentrations of hs-cTnT identify a malignant phenotype of LVH (19). Here, we show that although dilated hypertrophy, thick hypertrophy, and both thick and dilated hypertrophy are each associated with detectable hs-cTnT, the association of these LV geometric subtypes with adverse CV outcome persists despite adjustment for hs-cTnT. This finding suggests that both LV geometry and chronic subclinical myocardial injury are important contributors to HF risk in the population.

The risk of HF or CV death was increased in participants with either dilated hypertrophy or both thick and dilated hypertrophy. In addition, the continuous parameter of LVEDV was independently associated with increased risk of adverse CV outcomes in multivariable analysis. These data, along with previous reports of LV dilation alone being associated with incident HF (20), confirm the value of refining the phenotypic characterization of subjects

with increased LV mass based on the presence or absence of LV dilation. Further, the presence of LV dilation may identify a subpopulation of patients with LVH that may benefit from aggressive prevention and treatment to improve CV outcomes.

STUDY LIMITATIONS. The number of HF and CV death events is relatively few due to the low-risk general population cohort that was studied. The associations of increased risk seen in the participants with both thick and dilated hypertrophy should be considered preliminary given the low number of participants and events in this subgroup. The National Death Index was used to ascertain CV death, which may be inaccurate (21). Finally, our study was limited to assessment of the role of LV dilation among subgroups with LVH and does not consider the role of LV dilation in the absence of LVH.

CONCLUSIONS

Compared with the standard 2-tiered classification system for LVH, the 4-tiered classification system for LVH identifies subphenotypes of LVH in the general population that are at variable risk of HF and CV death. In particular, eccentric LVH can be stratified based on the absence or presence of ventricular dilation into a group at low risk (indeterminate hypertrophy) or high risk (dilated hypertrophy) for these outcomes.

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PERSPECTIVES

COMPETENCY IN MEDICAL KNOWLEDGE: A 4-tiered classification for LVH, which accounts for increased LV wall thickness and EDV, can identify subgroups of patients at differential risk of adverse CV outcomes.

TRANSLATIONAL OUTLOOK: Additional studies are needed to determine why individuals develop different patterns of LV remodeling (i.e., thick hypertrophy, dilated hypertrophy, or both thick and dilated hypertrophy). Further work is also needed to determine the pathophysiological links between subclinical myocardial injury, LV geometry, and subsequent adverse clinical events including the development of HF.

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KEY WORDS cardiac magnetic resonance, heart failure, hypertrophy, left ventricular geometry, troponin

APPENDIX For a supplemental table, please see the online version of this article.