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Left ventricular hypertrophy by ECG versus cardiac MRI as a predictor for heart failure

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Abstract

Objective—To determine if there is a significant difference in the predictive abilities of left ventricular hypertrophy (LVH) detected by ECG-LVH versus LVH ascertained by cardiac MRI-LVH in a model similar to the Framingham Heart Failure Risk Score (FHFRS).

Methods—This study included 4745 (mean age 61 ±10 years, 53.5% women, 61.7% non-whites) participants in the Multi-Ethnic Study of Atherosclerosis. ECG-LVH was defined using Cornell voltage product while MRI-LVH was derived from left ventricular mass. Cox proportional hazard regression was used to examine the association between ECG-LVH and MRI-LVH with incident heart failure (HF). Harrells concordance C-index ‘ was used to estimate the predictive ability of the model when either ECG-LVH or MRI-LVH was included as one of its components.

Results—ECG-LVH was present in 291 (6.1%), while MRI-LVH was present in 499 (10.5%) of the participants. Both ECG-LVH (HR 2.25, 95% CI 1.38 to 3.69) and MRI-LVH (HR 3.80, 95% CI 1.56 to 5.63) were predictive of HF. The absolute risk of developing HF was 8.81% for MRI-LVH versus 2.26% for absence of MRI-LVH with a relative risk of 3.9. With ECG-LVH, the absolute risk of developing HF 6.87% compared with 2.69% for absence of ECG-LVH with a

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relative risk of 2.55. The ability of the model to predict HF was better with MRI-LVH (C-index 0.871, 95% CI 0.842 to 0.899) than with ECG-LVH (C-index 0.860, 95% CI 0.833 to 0.888) ($p<0.0001$).

Conclusions—ECG-LVH and MRI-LVH are predictive of HF. Substituting MRI-LVH for ECG-LVH improves the predictive ability of a model similar to the FHFRS.

INTRODUCTION

Heart failure (HF) is estimated to affect almost seven million Americans.[1] The prevalence and the medical costs related to the management of patients with HF are expected to increase significantly over the next 15 years.[12] Although there is not yet a proven therapy to prevent the most common forms of HF in asymptomatic individuals, the subject remains an area of investigation. With expectation that such preventive therapy could be developed in the near future, it is important to identify individuals at risk for HF.

Left ventricular hypertrophy (LVH) is an established risk factor for HF and a component of the Framingham Heart Failure Risk Score (FHFRS).[3] The FHFRS was developed to identify individuals at risk for HF. It has previously been shown that LVH ascertained by ECG-LVH is a strong predictor of HF, despite the known low sensitivity of ECG to detect increase in left ventricular mass (LVM).[4–6] A recent report from the Cardiovascular Health Study has shown that ECG-LVH and echocardiography LVH are equally predictive of HF in the elderly.[7] However, it is yet to be determined if there is a difference in the predictive ability of a model similar to the FHFRS using ECG-LVH as a component versus a model where LVH ascertained by cardiac MRI-LVH, the current gold standard for estimating LVM.[8]

Thus, the aim of this study was to compare the associations of ECG-LVH and MRI-LVH with incident HF, and to determine if there is a significant difference in the predictive abilities of ECG-LVH versus MRI-LVH when used as a component of a model similar to the FHFRS.

METHODS

Study population

Data from the Multi-Ethnic Study of Atherosclerosis (MESA) were used for this analysis. Details of the MESA study have been previously described.[9] In summary, MESA is a prospective longitudinal study that was initiated in 2000 to study the prevalence, correlates and progression of subclinical cardiovascular disease (CVD) in a population-based sample. From 2000 to 2002, a total of 6814 men and women aged 45–84 years and free of clinical CVD (myocardial infarction, angina, stroke, transient ischaemic attack, HF, atrial fibrillation, revascularisation, valve replacement, pacemaker or defibrillator implantation, or taking nitroglycerine) were recruited from six US communities: Baltimore City and Baltimore County, Maryland; Chicago, Illinois; Forsyth County, North Carolina; Los Angeles County, California; Northern Manhattan and the Bronx, New York; and St. Paul, Minnesota. The ethnic origins of the cohort are as follows: 38% white, 28%, black/African-American, 23% Hispanic and 11% Asians of Chinese descent. Institutional review board

approval was obtained at each site, and a written informed consent was obtained from each participant during enrolment.

For the purpose of this analysis, only MESA participants with baseline cardiac MRI and ECG data were included in the analysis. Participants with major ventricular conduction defects (ie, QRS≥ 120 ms) including left bundle branch block were excluded. After all exclusions, a total of 4745 participants remained and were included in this analysis.

ECG-LVH

Standard 12-lead ECGs were digitally acquired using a Marquette MAC-PC ECG (Marquette Electronics, Milwaukee, Wisconsin) at 10 mm/mV calibration and speed of 25 mm/s. The same equipment was used at all sites, and all the ECGs were centrally read at the Epidemiological Cardiology Research Center located at Wake Forest School of Medicine, Winston Salem, North Carolina, USA. All ECGs were visually inspected for adequate quality and technical errors. The Cornell voltage product was used to determine ECG-LVH. This was defined by Cornell criteria [R-wave amplitude in aVL + S wave amplitude in V3 using the conventional gender specific cut offs (≥28 mm for males and ≥20 mm in females) multiplied by the QRS duration.[10]

MRI-LVH

The cardiac MRI protocol employed in MESA has been previously reported.[11] Briefly, LVM was measured as the sum of the myocardial area (the difference between endocardial and epicardial contours) times slice thickness plus image gap in the end-diastolic phase multiplied by the specific gravity of the myocardium (1.05 g/mL).[11] Observed LV mass (oLVM) was determined from MRI. Individual LVM was predicted using the following allometric height and weight indexation equations previously derived from a separate reference MESA subpopulation of 822 men and women (47% Caucasians, 22% Chinese, 18% African-American, 13% Hispanics) without LVH risk factors.[41112]

$$\begin{aligned}\text{Predicted LV mass (pLVM)} &= 8.17 \times \text{height (m)}^{0.561} \times \text{weight (kg)}^{0.608} \text{ for men} \\ &= 6.82 \times \text{height (m)}^{0.561} \times \text{weight (kg)}^{0.608} \text{ for women}\end{aligned}$$

The 95th percentile cut-off value of (oLVM/pLVM) was calculated as 1.31. This indicates that all subjects with oLVM more than 1.31 times of that predicted on the basis of height, weight and gender had LVM greater than 95% of the reference population and constituted LVH for the purposes of this study.[411]

Incident HF

Incident HF was ascertained by the MESA adjudication committee. The methods used by the adjudication committee have been previously reported.[12–14] Briefly, at the conclusion of the baseline examination, participants were re-examined every 2 years. In addition to the study examinations, a trained telephone interviewer followed up with the participants to determine if there was any hospital admission, CVD outpatient diagnosis or death. All events recorded were confirmed with medical records and death certificates. Medical records

were successfully obtained in >95% of all cases. For death occurring out of the hospital, next of kin interviews were obtained. Two physicians from the MESA events committee reviewed all medical records for incidence dates and endpoint classification. HF was classified into definite and probable. Probable HF was defined as participants that had signs and symptoms of HF such as shortness of breath and oedema. Definite HF was defined as meeting the criteria for probable HF as well as having objective evidence of pulmonary oedema/congestion on chest X-ray and evidence of depressed LV function, LV diastolic dysfunction or dilated ventricle on echo-cardiography or ventriculography. Definite or probable HF was considered as HF event in this analysis.

Covariates

The characteristics of participants were collected at the baseline MESA examination. Standard questionnaires were used to collect data on age, gender, race/ethnicity and smoking status. Body mass index (BMI) was calculated as weight in kilograms divided by the square of the height in metres (kg/m^2). The diagnosis of diabetes was determined by fasting blood glucose of ≥126 mg/dL or the use of insulin or oral hypoglycaemic agents. Resting blood pressure was obtained three times in a seated position after 5 min of rest and the average of the last two systolic pressures was used. Hypertension was defined as a systolic blood pressure (BP)≥140 mm Hg, diastolic BP≥90 mm Hg, reported use of antihypertensive or self-reported history of untreated hypertension. Resting heart rate was obtained from the ECG. Total cholesterol and high-density lipoprotein (HDL) cholesterol were measured after a 12-hour fast. The use of anti-hypertensive and cholesterol lowering medication was self-reported. Coronary heart disease (CHD) was determined by the MESA events committee using CHD endpoints including myocardial infarction, probable angina if followed by revascularisation, resuscitated cardiac arrest and CHD death. Hard CHD included myocardial infarction and fatal CHD.

Statistical analysis

Categorical variables were presented as frequency and percentages, while continuous variables were represented as median, Q1 and Q3. Statistical significance for categorical variables was tested using the χ^2 method, while the Wilcoxon rank-sum procedure was used for continuous variables. Comparisons were examined between participants with and without ECG-LVH and MRI-LVH, separately. The association between ECG-LVH and MRI-LVH at baseline with incident HF was examined. The follow-up time was from baseline examination to the development of HF, death or loss to follow-up.

Kaplan-Meier curves and the log-rank procedure[15] were used to compare the cumulative incidence of HF by LVH status using ECG or MRI using the log-rank test. Cox proportional hazard regression was used to compute HR and 95% CI for the association between ECG-LVH and MRI-LVH, separately, with incident HF. Multivariable models were constructed as follows: model 1 (demographic model) adjusted for age, sex, education and income; and model 2 (fully adjusted model) further adjusted for smoking, systolic blood pressure, heart rate, blood pressure lowering medications, diabetes, BMI, total cholesterol, HDL cholesterol and lipid lowering medication. These are the FHFRS predictors with the addition of race, sex, total cholesterol, HDL and cholesterol medication use due to the relevance of these in

CVD. We conducted two sensitivity analyses: (1) we used definite HF only as an outcome and (2) we examined the association between HF and other ECG-LVH criteria namely Cornell voltage and Sokolow-Lyon. The proportional hazard assumption was examined by visually using the log–log plots and was not violated in any of our analyses.

Harrell's concordance index (C-index) was computed to examine the predictive ability of the model similar to FHFRS using ECG-LVH versus MRI-LVH as the LVH component. The covariates used were the original FHFRS except for valvular disease by echocardiogram (which is not available in the MESA study). To explore the potential interchangeability of ECG-LVH and MRI-LVH in this model, we compared the C-indices for models containing ECG-LVH versus MRI-LVH using the likelihood ratio test.

The added predictive values of ECG-LVH and MRI-LVH to the components of FHFRS (age, heart rate, systolic blood pressure, BMI, diabetes, CHD) were also investigated using category-free net reclassification improvement (NRI) with and without the addition of LVH variables using previously developed methodology for survival analyses.[16] CIs for NRI were computed using bootstrapping with 1000 replicates.[17] Statistical significance was defined as $p<0.05$. SAS V.9.3 (Cary, North Carolina, USA) was used for all analyses.

RESULTS

A total of 4745 participants (mean age 61.3±10.0 years, 53.5% women, 38.3% whites, 25.8% African-Americans, 13.4% American Chinese and 22.6% Hispanics) were included in this analysis. ECG-LVH was present in 291 (6.1%) participants, while MRI-LVH was present in 499 (10.5%) participants. A total of 687 participants (14.5%) had either ECG-LVH or MRI-LVH. Table 1 shows the baseline characteristics of the cohort stratified by ECG-LVH and MRI-LVH status. With either ECG or MRI, participants with LVH were more likely to be older, smokers, diabetics, with higher levels of blood pressure, heart rate and BMI. On the other hand, participants with LVH were not different from those without LVH in terms of lipid profile, use of lipid lowering medications. Overall, African-Americans had the highest proportion of LVH by either modality (32.3% for ECG and 38.3% for MRI) while Asians had the lowest proportion of LVH (16.1% for ECG and 6.0% for MRI). Males and current smokers were more likely to have MRI-LVH (40% for males and 21% for smokers) when compared with ECG-LVH (33% for males and 10% for smokers).

Over a median follow-up of 10.4 years, 140 participants developed HF (incidence rate=7.7 per 1000 person years). The incidence rate of HF was two to three times greater in participants with ECG-LVH or MRI-LVH when compared with participants without LVH by either modality ($p<0.0001$; table 2). Overall, the absolute risk of developing HF was 8.81% for MRI-LVH versus 2.26% for absence of MRI-LVH with a relative risk of 3.9. In the presence of ECG-LVH, the absolute risk of developing HF was 6.87%, while it was 2.69% for the absence of ECG-LVH with a relative risk of 2.55. Figures 1 and 2 show the unadjusted cumulative incidence of HF by MRI-LVH and ECG-LVH status (log rank $p<0.001$ for both), respectively.

Both ECG-LVH and MRI-LVH were associated with an increased risk of HF in the demographic adjusted model and the fully adjusted model, but the risk was higher with MRI-LVH; table 2. Similar results were observed when other ECG-LVH criteria were used: Sokolow-Lyon and Cornell voltage (see online supplementary table S1). Also, in a sensitivity analysis limited to definite HF events only (n=98), the results were on the same direction (see online supplementary table S2).

The ability of a model similar to the FHFRS to predict HF was better with MRI-LVH (C-index 0.871, 95% CI 0.842 to 0.899) when compared with ECG-LVH (C-index 0.860, 95% CI 0.833 to 0.888) (−2LL χ^2=33, p<0.0001). To assess the potential clinical utility of using LVH-MRI instead of ECG-LVH in the model, we calculated several measures of reclassification (table 3). Results were consistent with the statistically significantly improved C-statistic with MRI-LVH. Reclassification index showed that MRI-LVH reclassified a higher proportion of participants into higher risk groups (see online supplementary table S3).

DISCUSSION

The key finding of this analysis is that, while ECG-LVH was predictive of the risk of incident HF, substituting MRI-LVH for ECG-LVH improved the predictive ability of a model similar to the FHFRS.

Our finding that using imaging for LVH assessment can improve the predictive ability of HF prediction models contradicts the findings of a recently published study showing that ECG-LVH can be used interchangeably with echocardiographic LVH (echo-LVH) for prediction of HF.[7] The aforementioned study was conducted on a predominantly white elderly population, which raises concerns about the generalisability in a younger multiethnic population. Also, cardiac MRI, although expensive, offers significant advantages over echocardiographic estimation as it provides a precise measurement with a high degree of reproducibility.[1819] More importantly, LVM by cardiac MRI is not based on geometric assumptions of LV shape and there is no acoustic window limitation as in echocardiography.[20] These advantages over echocardiography could possibly explain the difference in the predictive ability of the model observed in our analysis when compared with the previously published paper that used echo-LVH.

The financial impact of the treatment and hospitalisations associated with HF reached $39.2 billion in 2010.[21] With the expected increase in the prevalence of HF,[1] the ability to correctly identify individuals at risk of HF is the first step to prevent or delay the development of HF. The Heart outcome Prevention Evaluation (HOPE) trial demonstrated that the onset of HF can be delayed or prevented with the use of ACE inhibitors like ramipril in individuals with CVD.[22] Thiazide diuretics may also be used when targeting high-risk individuals as a recent meta-analysis showed a risk reduction in all-cause mortality, CHD and stroke with thiazide-type diuretics when compared with other antihypertensives.[23] This risk reduction in cardiovascular events was independent of blood pressure reduction. It is anticipated that additional trials of therapies designed to prevent HF in asymptomatic CVD free individuals will developed in the near future. Therefore, the ability to correctly identify asymptomatic individuals at highest risk for developing HF would be useful.

Our analysis has also shown that the prediction model using MRI-LVH is useful in an ethnically heterogeneous population. This is especially important as African-Americans, Hispanics and Native Americans have higher incidence and prevalence of HF when compared with other ethnicities.[24]

The results of our analysis are not without limitations. Although there are several criteria for detection of LVH by ECG, we only used Cornell voltage product, one of the most commonly used ECG criteria in clinical practice,[25] and confirmed the results using another couple of criteria. There have been some controversies regarding whether to include the papillary muscles in quantifying the myocardial mass when using cardiac MRI. However, excluding the papillary muscles has been shown to have better reproducibility.[2627] Despite these limitations, our study is the first to compare the predictive ability of ECG-LVH with MRI-LVH (the gold standard for estimating LVM), using a model similar to a validated predictive model, in an ethnically diverse population of middle age and old adults.

In conclusion, both ECG-LVH and MRI-LVH are predictive of HF. However, MRI-LVH improves the predictive ability of a model similar to the FHFRS and possibly other HF risk prediction models than ECG-LVH. These results are applicable to multiethnic populations that include ethnic minorities. This provides evidence that imaging modalities like MRI can be used to improve existing HF prediction models.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Key messages

What is already known on this subject?

- Left ventricular hypertrophy (LVH) determined by ECG is used in most heart failure (HF) prediction risk scores.
- Cardiac MRI is the gold standard of determining left ventricular mass and may be more predictive of cardiovascular events.

What might this study add?

- This study provides evidence that LVH determined by cardiac MRI improves the prediction of HF in a model similar to a well-validated risk score for predicting HF—the Framingham Heart Failure Risk Score.

How might this impact on clinical practice?

- When available, cardiac MRI determined LVH should be used in HF risk scores to achieve improved risk stratification especially in asymptomatic individuals. However, the cost-effectiveness of using MRI-LVH instead of ECG-LVH needs to be examined.

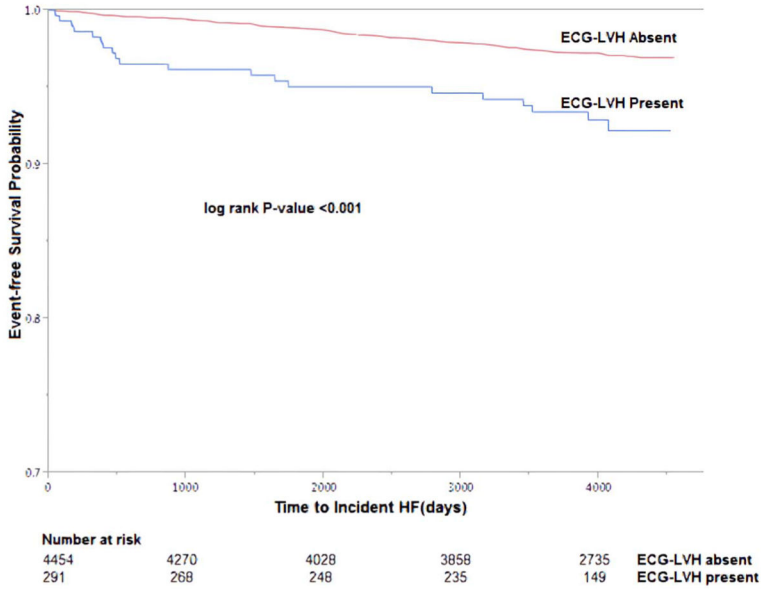

Figure 1.

Kaplan-Meier curves for incident heart failure (HF) by left ventricular hypertrophy by cardiac MRI (MRI-LVH) status.

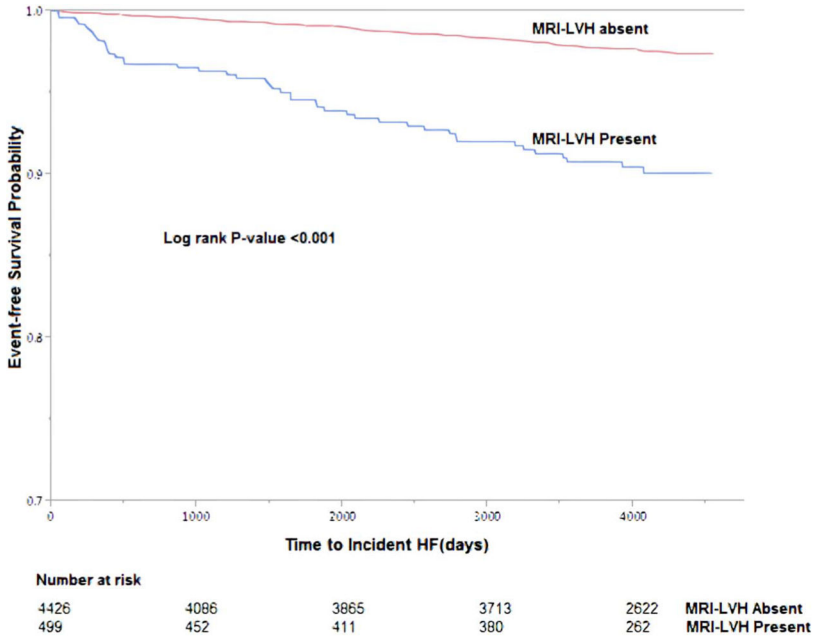

Figure 2.

Kaplan-Meier curves for incident heart failure (HF) by ECG left ventricular hypertrophy (ECG-LVH) status.

Table 1

Baseline characteristics

Characteristic N (%) or median (Q1, Q3)	ECG-LVH (N=291)	No ECG-LVH (N=4454)	p Value	MRI-LVH (N=499)	No MRI-LVH (N=4246)	p Value
Age (years)	65 (57, 71)	61 (53, 60)	<0.001	63 (54, 71)	61 (53, 69)	0.001
Male	95 (33)	2111 (47.4)	<0.001	198 (40)	2008 (47)	0.001
Race/ethnicity			<0.001			<0.001
White	71 (24.4)	1746 (39.2)		135 (27.0)	1682 (39.6)	
Asian	47 (16.1)	588 (13.2)		30 (6.0)	605 (14.2)	
Black	94 (32.3)	1128 (25.3)		191 (38.3)	1031 (24.3)	
Hispanic	79 (27.2)	992 (22.3)		143 (28.7)	928 (21.9)	
BMI (kg/m^2)	29 (25.4, 33.3)	27.1 (24.2, 30.4)	<0.001	28.6 (25.3, 32.6)	27.1 (24.2, 30.3)	<0.001
Smoking status			0.02			<0.001
Current	29 (10)	572 (13)		103 (21)	498 (12)	
Past	88 (30)	1595 (36)		162 (33)	1521 (36)	
Diabetes mellitus	43 (15)	369 (8)	<0.001	75 (15)	337 (9)	<0.001
Hypertension	195 (67)	1797 (40)	<0.001	354 (71)	1638 (39)	<0.001
Hypertension medications	154 (53)	1496 (34)	<0.001	263 (53)	1387 (32)	<0.001
Systolic BP (mm Hg)	136 (120.5, 153.5)	121 (110, 137.5)	<0.001	140 (123, 156)	120.5 (109, 136)	<0.001
Heart rate (bpm)	62 (57, 69)	62 (56,69)	0.69	61 (55, 69)	62 (57, 69)	0.007
Total cholesterol (mg/dL)	196 (173, 223)	192 (171, 214)	0.15	194 (170, 218)	192 (171, 215)	0.90
HDL cholesterol (mg/dL)	49.0 (40.8, 60.0)	49 (41, 59)	0.60	48 (40, 61)	49 (41, 59)	0.65
BP lowering medications	154 (53)	1496 (34)	<0.001	263 (53)	1387 (33)	<0.001
Lipid lowering medications	59 (20)	680 (15)	0.03	73 (15)	666 (16)	0.60

Statistical significance for continuous data was tested using Wilcoxon rank-sum procedure and categorical data was tested using χ^2.

BMI, body mass index; ECG-LVH, ECG left ventricular hypertrophy; echo-LVH, echocardiographic left ventricular hypertrophy; HDL, high-density lipoprotein; LVH, left ventricular hypertrophy; MRI-LVH, left ventricular hypertrophy by cardiac MRI.

Table 2

ECG-LVH and MRI-LVH and risk of incident heart failure

			HR (95% CI)			
LVH status	**N events/N at risk**	**Incidence rate/1000 person years**	**Demographically adjusted model***	**p Value**	**Fully adjusted model†**	**p Value**
ECG-LVH absent	120/4454	2.6	Ref		Ref	
ECG-LVH present	20/291	7.1	2.62 (1.62 to 4.24)	<0.0001	2.25 (1.38 to 3.69)	<0.0001
MRI-LVH absent	96/4246	2.2	Ref		Ref	
MRI-LVH present	44/499	9.3	4.56 (3.18 to 6.55)	<0.0001	3.80 (2.56 to 5.63)	<0.0001

*Adjusted for age, sex, race, education and income.

†Demographically adjusted plus smoking status, systolic blood pressure, diabetes, body mass index, heart rate, total cholesterol, high-density lipoprotein cholesterol, lipid lowering medication use, antihypertensive medications use and coronary artery disease.

ECG-LVH, ECG left ventricular hypertrophy; MRI-LVH, left ventricular hypertrophy by cardiac MRI.

Table 3

Performance of a model similar to the Framingham Heart Failure Risk Score using ECG-LVH and MRI-LVH*

LVH detection method	Harrell's C—index (95% CI)	−2 Log likelihood	Continuous NRI (95% CI)
ECG-LVH	0.860 (0.833 to 0.888)	2152 (p<0.0001)	0.12 (0.02 to 0.24)
MRI-LVH	0.871 (0.842 to 0.899)	2119 (p<0.0001) χ^2 test p<0.0001	0.36 (0.22 to 0.54)

*Adjusted for the components of the Framingham Heart Failure Risk Score (age, heart rate, systolic blood pressure, body mass index, diabetes and coronary heart disease). ECG-LVH, ECG left ventricular hypertrophy; MRI-LVH, left ventricular hypertrophy by cardiac MRI; NRI, net reclassification improvement.