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Subclinical and Clinical Correlates of Left Ventricular Wall Motion Abnormalities in the Community

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Abstract

The prevalence and clinical correlates of left ventricular (LV) wall motion abnormalities (WMAs), associated with morbidity and mortality, are not well-characterized in the population. Framingham Heart Study Offspring Cohort participants (n=1794, 844M, 65±9 years) underwent cine cardiovascular magnetic resonance (CMR) for evaluation of LV function, and a subset (n=1009, 460M) underwent cardiac multidetector computed tomography (MDCT) for analysis of coronary artery calcium (CAC). Presence of coronary heart disease and heart failure (CHD-HF) were assessed in relation to WMAs. WMAs were present in 117 participants (6.5%) and were associated with male sex, elevated hemoglobin A1c, LV mass, LV end-diastolic volume (LVEDV), and lower LV ejection fraction. Among 1637 participants without CHD-HF, 68 (4.2%) had WMAs. In this group, WMAs were associated with obesity, hypertension, and Framingham coronary heart disease risk score in age- and sex-adjusted analyses, and associated with male sex and hypertension in multivariable analysis. Most individuals with WMAs fell in the highest CAC groups. The presence of CAC >75th percentile and Agatston score >100 were associated with a >2-fold risk of WMAs in age- and sex-adjusted analysis, were no longer significant when additionally adjusted for CHD-HF. Past Q wave myocardial infarction was present in 29% of the

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117 participants with WMAs. In conclusion, in this longitudinally followed free-living population, 4.2% of participants without CHD-HF had WMAs. WMAs were associated with clinical parameters associated with cardiovascular disease risk. Aggressive risk factor modification may be prudent in individuals with WMAs, particularly in those free of clinical CHD-HF.

Keywords

Left ventricular wall motion abnormalities; cardiovascular magnetic resonance; risk factors; coronary calcium score

Echocardiographic data suggests increased morbidity and mortality associated with LV dysfunction in individuals without known cardiovascular disease (CVD).¹ However, analysis of WMAs on surface echocardiography may be limited by suboptimal image quality in those with CVD risk factors.^{2,3} Thus, the prevalence and clinical correlates of LV WMAs in the general population are not well-established. Cardiovascular magnetic resonance (CMR) is well-suited for analysis of LV WMAs as it provides excellent endocardial border definition without acoustic window constraints.⁴ We hypothesized that CMR would detect LV WMAs in both those with and without a history of coronary heart disease (CHD) and heart failure (HF), and that these WMAs would be associated with CHD risk factors, subclinical disease of the ventricle and coronary arteries, and clinically apparent CHD or HF. We thus applied CMR methods to determine the prevalence and clinical correlates of LV WMAs in a community, free-living population.

METHODS

A subset (n=1794) of 3539 FHS Offspring Cohort participants who attended the 7th examination cycle (1998-2001), were in sinus rhythm, had no contraindication to CMR, and who lived in a state contiguous with Massachusetts were recruited.^{5,6} At each examination every 3-4 years, participants underwent routine medical history and physical, anthropometry, and assessment of cardiovascular risk factors. CMR scanning (2002-2006) was unable to be completed for claustrophobia (n=13), scanner dysfunction (n=7), metallic devices (n=10), or miscellaneous reasons (n=2). A subset of this Cohort (n=1009) also underwent cardiac multidetector coronary computed tomography (MDCT) for analysis of coronary arterial calcification (CAC). The study was approved by the institutional review boards of Boston University Medical Center and Beth Israel Deaconess Medical Center. All participants provided written informed consent.

Supine CMR imaging was performed using a 1.5T CMR scanner (Gyroscan NT, Philips Medical Systems, Best, The Netherlands) with a 5-element commercial cardiac array coil for radiofrequency signal reception. Following localizing scans to determine the position and orientation of the heart within the thorax, end-expiratory breath-hold, ECG-gated cine steady state free precession images were acquired in 2-chamber, 4-chamber, and contiguous short axis orientations (temporal resolution 39 ms, repetition time = R-R interval, echo time 9 ms, flip angle 60 degrees, field of view 400 mm, matrix size 208×256, slice thickness 10 mm, gap=0).

Quantitative CMR data analysis was performed using dedicated software (EasyVision 5.1, Philips Medical Systems, Best, The Netherlands) by an observer blinded to clinical data. LV wall motion was analyzed according to a 17-segment model.⁷ Global and regional wall motion score were computed using a 5-point scale (1=normal, 2=hypokinetic, 3=akinetic, 4=dyskinetic, 5=aneurysm), with a normal LV segment summation of 17. Wall motion score index (WMSI) was calculated as the total wall motion score divided by number of segments,

with a WMSI $\geq 19/17$ (≥ 1.12) considered abnormal (i.e., ≥ 2 contiguous segments hypokinetic, and/or one segment akinetic or dyskinetic).¹ WMAs noted by the reviewer were confirmed by two additional reviewers. Quantitative measures of LVEF and mass (LVM) were obtained by manually tracing epicardial and endocardial LV borders as previously described.⁶ LV end-diastolic volume (EDV) and end-systolic volume (ESV) were computed using the summation of discs method. LV ejection fraction (LVEF) was computed by $(EDV-ESV)/EDV$. LVM was determined by summing myocardial wall volume and multiplying by myocardial density (1.05g/ml). LVM was indexed to body surface area (BSA). LVM index (LVMI), relative wall thickness (RWT, $2 \times$ diastolic inferolateral wall thickness/LV end-diastolic dimension), the ratio of LVM to LVEDV (LVM/LVEDV), and LVEF were tabulated.

MDCT scanning was conducted in the FHS Offspring Cohort contemporaneous to the CMR, as described.⁸ An 8-slice MDCT scanner (LightSpeed Ultra, GE, Milwaukee, Wisconsin) was used for image acquisition using prospective electrocardiographic (ECG) triggering (initiated at 50% of the R-R interval) during a single breath hold. Contiguous 2.5 mm thick slices were acquired. A field of view of 35 mm was used for image reconstruction. The presence and amount of CAC was analyzed by an experienced reader on a dedicated workstation (Aquarius, Terarecon, San Mateo, California). A calcified lesion was defined as an area ≥ 3 connected pixels with attenuation >130 Hounsfield units. A modified Agatston score (AS) was calculated by multiplying the area of each lesion with a weighted attenuation score dependent on the maximal attenuation in the lesion, and sex-specific $>75^{\text{th}}$ percentile values were used as previously established.^{9,10}

Participants underwent a routine physical examination, anthropometry, and laboratory assessment of CVD risk factors at Examination 7 as previously described.¹¹ Hypertension was defined as systolic pressure ≥ 140 mm Hg or diastolic pressure ≥ 90 mm Hg or use of antihypertensive medications. Plasma glucose, total and high-density lipoprotein (HDL) cholesterol, and hemoglobin A1c (HbA1c) were measured on morning samples after an 8-hour fast. Diabetes mellitus was defined as fasting glucose ≥ 126 mg/dl or the use of oral hypoglycemic medications or insulin. To account for a history of elevated cholesterol and/or statin therapy, we used total cholesterol and ratio of total/HDL cholesterol, averaged over Examinations 1-7. Dyslipidemia was defined by total cholesterol ≥ 200 mg/dL or the use of lipid-lowering therapy. Framingham risk score (FRS) was computed using sex-specific equations that incorporated age, sex, history of smoking, presence of diabetes, systolic and diastolic blood pressure, fasting serum total cholesterol, and HDL cholesterol.

Pathologic Q waves in a coronary artery distribution (QWMI) were adjudicated by a physician committee who reviewed 12-lead ECGs performed at each Examination. Prior CVD events were confirmed by review of all available data including hospital records by a three-physician endpoint committee, as previously described.⁵ A history of CHD-HF was defined by the occurrence of any one of the following events: recognized or unrecognized MI, angina, coronary insufficiency, or congestive heart failure.

Participants were categorized by the presence or the absence of each CHD-HF and WMAs. Descriptive statistics for all covariates are presented as either percentages or means $\pm$ SD. Group differences were evaluated using two-sample t tests and analysis of covariance (ANCOVA) for continuous measures and using Chi-squared test and logistic regression for categorical variables. Age- and sex-adjusted and multivariable adjusted logistic regression models (incorporating age, sex, obesity, history of hypertension, diabetes, dyslipidemia, smoking history, and FRS) were constructed to assess the association of covariates to the presence or absence of a WMA. Furthermore, to assess sex-dependent cardiac anatomic differences, sex-specific multivariable ANCOVA models were constructed to evaluate

differences in CMR measures with WMAs, and also for groups with and without CHD-HF. All analyses were performed with SAS 8.0 (SAS Institute, Cary, NC). A p-value <0.05 was considered statistically significant.

The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

RESULTS

Of the 1794 participants (844M, 65±9 yrs) who completed CMR scanning with analyzable data, WMAs were present in 117 (6.5%) participants including 68 (58% of the 117) free of CHD-HF (Figure 1). WMAs were predominantly focal in both those with and without CHD-HF. Participants with CHD-HF had a higher prevalence of WMAs in all regions (p<0.001 vs. without CHD-HF). The characteristics of WMAs are presented in Figure 2. The regional distribution of WMAs was similar between those with and without CHD-HF, with the exception of greater prevalence of lateral WMAs in participants with CHD-HF (82% vs. 59%, p=0.009). Participants without CHD-HF had a higher prevalence of hypokinesis, while those with CHD-HF had a higher prevalence of akinesis, dyskinesis, or aneurysm (p<0.001; Figure 3).

The clinical characteristics of participants without CHD-HF are presented in Table 1. Of these participants, those with WMAs were older, and more likely to be male, obese (body mass index >30), have a history of hypertension and diabetes, and have a higher HbA1c, total cholesterol/HDL ratio, and FRS (all p<0.02). The proportion of participants with WMAs significantly increased across increasing tertiles of 10-year FRS risk groups (p<0.001, Figure 4). In age- and sex- adjusted models, WMAs were associated with obesity, hypertension, and greater FRS (Table 2). After adjusting for age, sex, obesity, hypertension, diabetes, dyslipidemia, and history of smoking, hypertension and male sex were associated with a two-fold and nearly four-fold increased odds for a WMA, respectively (p=0.011 and p<0.001, respectively) (Table 2). In further analysis (not shown in the Tables) adjusted for age, sex, and antihypertensive treatment, a history of hypertension at Examination 1 (1971-1975) was not associated with WMAs (OR 0.92, 95% CI 0.40-2.13, p=0.844), but any history of hypertension was associated with a two-fold risk for WMAs (OR 2.26, 95% CI 1.08-4.71, p=0.030).

CMR correlates of WMAs among participants without CHD-HF included increased LVM (raw and indexation for BSA, height, and height^{2.7}), increased LVEDV, and lower LVEF, in age-adjusted sex-specific analyses (all p<0.001) (Table 3). LVM/LVEDV and RWT were not correlated with WMAs in those without CHD-HF.

The clinical characteristics of the CHD-HF group are presented in Table 4. Participants with CHD-HF had a higher WMSI than those without CHD-HF (1.67±0.44 vs. 1.47±0.39, p<0.001). WMAs were associated with male sex and elevated HbA1c in univariable analysis. In multivariable-adjusted analysis, male sex and history of diabetes each conferred a three-fold increased risk of WMAs (OR 3.07, 95% CI 1.16-8.13, p=0.024, and OR 2.87, 95% CI 1.03-7.96, p=0.043, respectively). However, obesity, hypertension, and FRS were not associated with WMAs. WMAs were also associated with elevated LVM, LVMI, LVM/ht^{2.7}, and LVEDV, and with decreased RWT, LVM/LVEDV, and LVEF (Table 4). In age-adjusted linear regression analyses, in men these relationships persisted (Table 5). However, in women, WMAs were only associated with elevated LVEDV and decreased LVEF. Among all participants, there were no significant differences between the groups with and without WMAs with respect to dyslipidemia, tobacco use, or history of other CVD, including cerebrovascular accident, transient ischemic attack, or claudication.

Among the 1009 participants who also underwent MDCT, 68% had CAC, 48% had CAC >75th percentile for age, and 37% had AS >100. Among those with highest CAC (CAC >75th percentile and AS >100), 39% and 42% (respectively) of those with WMAs had CHD-HF, as compared to 8% and 10%, respectively, of those without WMAs. While the presence of CAC >75th percentile and AS >100 were associated with a >2-fold risk of WMAs in age- and sex-adjusted analysis, these associations were attenuated when additionally adjusted for a history of CHD-HF (Table 6). The greater prevalence of any CAC in those with WMAs compared to those without WMAs was not significant in age- and sex-adjusted analyses. When participants with CHD-HF were excluded from analysis, CAC >75th percentile and AS >100 were more common in those with WMAs than in those without WMAs (63% vs. 46%, $p=0.06$, and 56% vs. 34%, $p=0.02$, respectively). However, these associations were not significant after age- and sex-adjustment.

A QWMI was present in 52 (2.9%) of the 1794 participants, including 18 (1%) of 1677 participants with normal wall motion, and in 34 (29%) of the 117 participants with WMAs. A WMA was associated with a 21-fold increased odds of past QWMI (OR 20.5, 95% CI 10.7-39.3, $p<0.001$). WMAs were present in 65% of the group with QWMI, whereas WMAs were present in only 5% of those without QWMI. WMSI was likewise greater in those with QWMI than without pathologic Q waves (1.46 ± 0.51 vs. 1.02 ± 0.14 , respectively, $p<0.001$).

DISCUSSION

In this population of free-living individuals, we identified 6.5% with WMAs, of whom the majority (58%) did not have clinical CHD-HF. In participants without prior CHD-HF, male sex and hypertension were the strongest predictors of a WMA. Our findings of the relationship between WMAs and CMR morphometric measures are consistent with the association of CHD and CVD risk with increased LVM and LVEDV,^{12,13} and suggest an increased risk of WMAs in both men and women with increasing LVM and LVEDV, even without known CHD.

Participants with CHD-HF had both a higher prevalence of WMAs and more severe WMAs. In this population, WMAs were associated with male sex and diabetes, consistent with established data of their increased risk for cardiovascular disease.^{14,15} The small number of women in this group preclude definitive conclusions regarding gender differences of CMR measures among participants with CHD-HF. WMAs were associated with decreased RWT and LVM/LVEDV. This relative chamber dilation, not evident in the group without CHD-HF, is consistent with more severe and prevalent WMAs in this group, likely resulting from prior myocardial ischemia and infarction.

A significant amount of CAC, but not the presence of CAC alone, was associated with an increased odds for WMAs. The associations between increased CAC burden and WMAs were attenuated after adjustment for CHD-HF, suggesting the association is largely mediated through clinically evident cardiovascular events. Our results are consistent with the association of WMAs with CAC in those with CVD risk factors¹⁶ and the totality of data for a strong relationship between CAC and cardiovascular events.^{17,18}

While WMAs were present in most participants with prior QWMI, most WMAs were found in participants without a history of QWMI or CHD-HF. It is possible that a large number of those with WMAs in the absence of QWMI sustained non-QWMI. Indeed, absence of Q waves are seen in a quarter of patients with CMR evidence of delayed enhancement representing past MI.^{19,20} Our findings are also consistent with data that ventricular function may be preserved or recover post MI,²¹ that some MIs are clinically silent,¹⁹ and that Q waves are an imperfect measure of past ischemic insults.²²

The possible etiologies of these WMAs in our population-based study include ischemic insults such as hibernating and stunned myocardium, myocardial infarction and infarct-related cardiomyopathy. Rarer possibilities include myocarditis or other nonischemic cardiomyopathies leading to focal and/or global LV dysfunction. Ischemic etiologies are supported by our findings that most WMAs were focal, increasing prevalence of WMAs with greater 10-year FRS tertile, and association of WMAs with other CVD risk factors and CAC. Consistent with our data, echocardiographic resting WMAs in those free of known CVD are independently associated with a higher risk for CVD morbidity and mortality in follow-up.¹

There are few population studies that describe the presence of WMAs in those free of CHD-HF, and none to our knowledge using CMR for identification of resting WMAs. Two population studies in individuals of similar age have been conducted using transthoracic echocardiography, also supporting associations of WMAs with male sex, hypertension, diabetes, and increased LVM and decreased LVEF.^{1,2} The Strong Heart Study reported a prevalence of 4.9% segmental and 1.5% global WMAs by surface echocardiography in participants without overt CHD, stroke, or CHF.¹ In contrast, the Cardiovascular Health Study reported only a 1.9% prevalence of echocardiographic WMAs in participants without clinical CHD or hypertension.² Our data are intermediate between these two studies, a finding which may be related to differences in population and methodology. The greater prevalence of WMAs in the Strong Heart Study may have been due to their higher prevalence of diabetes and tobacco use.^{23,24} The lower prevalence in the Cardiovascular Health Study may reflect exclusion of participants with hypertension. Exclusion of hypertensives in the Strong Heart Study resulted in a 1.4% decrease in the prevalence of WMAs. Although we included participants with a history of vascular disease, we did not find an association between claudication, stroke, or transient ischemic attack with WMAs in either those with or without CHD-HF.

Prior population studies examining the prevalence of asymptomatic WMAs by surface echocardiography excluded 3-7% of participants due to inability to assess wall motion due to suboptimal acoustic windows associated with male sex, a history of CHD, advanced age, and obesity.^{1,2} In our CMR study, we were able to assess wall motion in 99% of all participants.

Limitations of our study include that assessment of regional WMAs is observer-dependent, though CMR allows superior endocardial border definition and thus excellent interobserver agreement.²⁵ Late gadolinium enhancement CMR was not performed. Such imaging would have enabled correlation between evidence of myocardial scarring and WMAs. Finally, the majority of our study population was Caucasian. Our results may not be generalizable to other races or ethnicities. While WMAs correlated with clinical markers of CVD risk, future outcomes data will add additional prognostic information.

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Figure 1. Distribution of wall motion abnormalities (WMAs) in the Framingham Offspring Cohort. CHD-HF = Myocardial infarction, angina pectoris, coronary insufficiency, or congestive heart failure.

Figure 2.

Distribution of WMAs among all participants and those with and without CHD-HF. Among both groups, WMAs were most commonly focal. The greater prevalence of focal WMAs among those with CHD-HF than those without CHD-HF was not significant ($p=0.154$).

Figure 3. Relationship of WMA severity to history of CHD-HF. Participants without CHD-HF had a higher prevalence of hypokinesis than those with CHD-HF, whereas those with CHD-HF had a greater prevalence of akinesis (AK), dyskinesia (DK), or aneurysm (both $p<0.001$).

Figure 4. Association of WMAs with Tertile of Framingham 10-year Risk for Coronary Heart Disease (CHD, myocardial infarction or coronary death). Low = <10% 10-year risk for CHD. Intermediate=10-20% risk for CHD. High= >20% risk for CHD.

Table 1

Baseline Characteristics of Cohort without Coronary Heart Disease or Heart Failure (n=1637)

Characteristic	Wall Motion Abnormality		
	No (n=1569)	Yes (n=68)	P value
Age (years)	64±9	67±10	0.006
Men	44%	77%	<0.001
Obesity	31%	50%	0.001
Body Mass Index (kg/m ²)	27.7±4.9	29.8±5.9	0.006
Waist/hip ratio	0.9±0.1	1.0±0.1	<0.001
Hypertension	49%	77%	<0.001
Systolic Blood Pressure (mm Hg)	124±18	130±17	0.011
Diastolic Blood Pressure (mm Hg)	74±9	77±11	0.052
Diabetes Mellitus	8%	16%	0.013
Hemoglobin A1c (%)	5.5±0.8	5.8±1.0	0.013
Dyslipidemia	79%	82%	0.509
Average total cholesterol/HDL	4.2±1.2	4.5±1.3	0.018
Other cardiovascular disease	3%	4%	0.600
Any history of tobacco use	60%	68%	0.195
Framingham Risk Score	7.4±4.0	9.1±4.3	0.001
Left ventricular mass (g)	103±28	140±37	<0.001
Left ventricular mass index (g/m ²)	54±11	68±14	<0.001
Left ventricular mass/height ^{2.7} (g/m ^{2.7})	25±5	32±7	<0.001
Left ventricular end-diastolic volume (ml)	123±29	161±38	<0.001
Relative wall thickness	0.27±0.05	0.27±0.05	0.666
Left ventricular mass/left ventricular end-diastolic volume (g/ml)	0.84±0.16	0.88±0.18	0.055
Left ventricular ejection fraction (%)	68.0±5.7	54.7±8.8	<0.001

Data presented as Mean±SD.

Average total cholesterol/HDL = averaged over Examinations 1-7. Dyslipidemia= total cholesterol ≥200 or on lipid-lowering therapy. Obesity = body mass index >30 kg/m². Other cardiovascular disease = claudication, cerebrovascular accident, or transient ischemic attack.

Table 2

Odds of Cardiovascular Risk Factors with Wall Motion Abnormalities in Participants without Coronary Heart Disease or Heart Failure (n=68)

Age-and Sex-adjusted model			
	OR	95% CI	P Value
Obesity	1.98	1.21-3.25	0.007
Hypertension	2.59	1.42-4.73	0.002
Diabetes Mellitus	1.82	0.91-3.61	0.089
Dyslipidemia	1.10	0.57-2.12	0.772
Smoker- ever	1.21	0.72-2.05	0.475
Framingham Risk Score	1.10	1.01-1.20	0.027
Multivariable model			
	OR	95% CI	P Value
Age	1.02	0.99-1.06	0.122
Male sex	3.69	2.07-6.58	<.0001
Obesity	1.64	0.98-2.74	0.058
Hypertension	2.23	1.20-4.14	0.011
Diabetes Mellitus	1.36	0.68-2.74	0.387
Dyslipidemia	0.96	0.50-1.87	0.913
Smoker- ever	1.14	0.67-1.94	0.628

Table 3
Gender-Specific Cardiovascular Magnetic Resonance Characteristics of Cohort without Coronary Heart Disease or Heart Failure

Characteristic	Men			Women		
	No WMA (n=682)	WMA (n=52)	P value	No WMA (n=887)	WMA (n=16)	P value
Left ventricular mass (g)	126±24	150±33	<0.001	85±16	108±29	<0.001
Left ventricular Mass index (g/m ²)	61±10	70±12	<0.001	48±7	60±15	<0.001
Left ventricular mass/ height ^{2.7} (g/m)	28±5	33±7	<0.001	23±4	30±7	<0.001
Left ventricular end-diastolic volume (ml)	143±27	166±38	<0.001	107±19	142±33	<0.001
Relative wall thickness	0.29±0.05	0.28±0.05	0.296	0.26±0.05	0.25±0.05	0.603
Left ventricular mass/left ventricular end-diastolic volume (g/ml)	0.90±0.17	0.92±0.18	0.618	0.80±0.13	0.76±0.13	0.070
Left ventricular ejection fraction (%)	67±6	55±9	<0.001	69±6	55±7	<0.001

Data presented as mean±SD. *All age-adjusted analyses.

WMA = Wall motion abnormality.

Table 4

Baseline Characteristics of Cohort with Coronary Heart Disease or Heart Failure (n=157)

Characteristic	Wall Motion Abnormalities		
	No (n=108)	Yes (n=49)	P value
Age (years)	70±8	69±8	0.643
Men	63%	86%	0.004
History of coronary heart disease	94%	90%	0.520
History of heart failure	11%	18%	0.225
History of Q wave Myocardial infarction	17%	69%	<0.001
Obesity	53%	45%	0.360
Body Mass Index (kg/m ²)	29.3±4.5	29.1±5.0	0.774
Waist/hip ratio	1.0±0.1	1.0±0.0	0.227
Hypertension	79%	84%	0.469
Systolic Blood Pressure (mm Hg)	131±19	126±19	0.111
Diastolic Blood Pressure (mm Hg)	72±9	73±11	0.565
Diabetes Mellitus	23%	35%	0.130
Hemoglobin A1c (%)	5.9±1.0	6.7±1.9	0.028
Dyslipidemia	89%	96%	0.152
Average total cholesterol/HDL	5.0±1.5	5.2±1.1	0.245
Other cardiovascular disease	17%	16%	0.958
Any history of tobacco use	65%	78%	0.111
Framingham Risk Score	11.0±3.1	11.2±3.0	0.720
Left ventricular mass (g)	118±32	138±28	<0.001
Left ventricular mass index (g/m ²)	60±14	70±15	<0.001
Left ventricular mass/height ^{2.7} (g/m ^{2.7})	29±7	33±7	0.002
Left ventricular end-diastolic volume (ml)	132±32	169±34	<0.001
Relative wall thickness	0.29±0.06	0.25±0.05	<0.001
Left ventricular mass/left ventricular end-diastolic volume (g/ml)	0.90±0.15	0.83±0.18	0.016
Left ventricular ejection fraction (%)	68.9±5.7	52.0±9.5	<0.001

Data presented as Mean±SD.

Average total cholesterol/HDL = averaged over Examinations 1-7. Dyslipidemia= total cholesterol ≥200 or on lipid-lowering therapy. Obesity = body mass index >30 kg/m². Other cardiovascular disease = claudication, cerebrovascular accident, or transient ischemic attack.

Table 5
Gender-Specific Cardiovascular Magnetic Resonance Characteristics of Cohort with Coronary Heart Disease or Heart Failure

Characteristic	Men			Women		
	No WMA (n=68)	WMA (n=42)	P value	No WMA (n=40)	WMA (n=7)	P value
Left ventricular mass (g)	132±30	143±26	0.059	94±20	107±16	0.116
Left ventricular Mass index (g/m ²)	64±13	71±14	0.012	52±11	60±14	0.132
Left ventricular mass/ height ^{2.7} (g/m)	30±7	33±7	0.044	27±6	30±9	0.181
Left ventricular end-diastolic volume (ml)	143±30	174±30	<0.001	113±26	142±41	0.020
Relative wall thickness	0.30±0.06	0.25±0.05	<0.001	0.27±0.05	0.25±0.04	0.286
Left ventricular mass/left ventricular end-diastolic volume (g/ml)	0.93±0.14	0.84±0.18	0.003	0.84±0.16	0.79±0.18	0.406
Left ventricular ejection fraction (%)	68±5	52±8	<0.001	70±6	51±15	<0.001

Data presented as mean±SD. *All age-adjusted analyses.

WMA = Wall motion abnormality.

Table 6

Association of Wall Motion Abnormalities with Coronary Artery Calcification

	No WMA (n=960)	WMA (n=49)	Age- and sex-adjusted OR (95% Confidence Interval)	Age, Sex-adjusted p value	Age, sex, and CHD-HF adjusted p value
Any coronary artery calcium	67%	86%	2.009 (0.83-4.87)	0.123	0.241
Coronary artery calcium >75 th percentile	47%	67%	2.208 (1.11-4.38)	0.024	0.156
Agatston score 0	33%	14%	Referent		
Agatston score 0-100	32%	23%	1.44 (0.53-3.86)	0.473	0.487
Agatston score >100	36%	63%	2.926 (1.12-7.68)	0.029	0.131