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Left Atrial Function and Mortality in Patients With NSTEMI

An MDCT Study

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OBJECTIVES We sought to test the hypothesis that measures of left atrial (LA) function are independent predictors of mortality in patients with acute myocardial infarction.

BACKGROUND Left atrial maximal volume (LAmax) is known to predict mortality in patients with acute myocardial infarction. In a previous pilot study, however, we found that LA function in terms of fractional change and left atrial ejection fraction (LAEF) assessed by multidetector computed tomography (MDCT) is more closely related to clinical heart failure than LAmax.

METHODS We prospectively included 384 patients presenting with non–ST-segment elevation myocardial infarction (NSTEMI) who underwent retrospectively gated, 64-slice MDCT coronary angiography and subsequent measurements of LA size and function. All patients were treated according to the current guidelines based on invasive coronary angiography. Patients were followed for a minimum of 2 years. The study endpoint was all-cause mortality.

RESULTS The median follow-up time was 36 months (range 10 to 1,551 days). During follow-up, 35 (9%) patients died. Overall, 1- and 2-year survival in the study cohort was 97% and 94%. LA size and mechanical function was obtained in all patients: mean LAmax was 55 ± 11 ml/m^2, LA minimal volume 31 ± 11 ml/m^2, fractional change 45 ± 9%, and LAEF 32 ± 9%. Using a Cox proportional hazards model with adjustments for age, number of diseased coronary vessels, left ventricular ejection fraction (LVEF), and Killip class, both fractional change (hazard ratio [HR]: 0.65; 95% confidence interval [CI]: 0.45 to 0.94) and LAEF (HR: 0.63; 95% CI: 0.44 to 0.91) remained independent predictors of mortality. In contrast to this, LAmax was not significantly associated with an increased risk of mortality in this population.

CONCLUSIONS In a low-risk group of patients with NSTEMI, reduced LA function is an independent predictor of mortality and provides prognostic value incremental to that of LAmax. (J Am Coll Cardiol Img 2011;4:1080–7) © 2011 by the American College of Cardiology Foundation

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The left atrium (LA) acts as a reservoir and conveys blood from the pulmonary vascular bed to the left ventricle. The left atrial maximal volume (LAmax) is determined by multiple factors, including the pressure gradient across the mitral valve in early diastole, left ventricular (LV) chamber compliance, LA active contraction, and intrinsic LA wall factors. It is believed that the LA will dilate in response to either volume or pressure overload (1–3). In agreement with this, several studies have demonstrated that LA dilation is associated with increased morbidity and mortality in patients with acute myocardial infarction and cardiomyopathy (4–6). However, the LA modulates blood flow to the left ventricle in a complex manner, and measurements of LAmax may only partially reflect the prognostic implications of volume or pressure overload of the chamber. Multidetector computed tomography (MDCT) with high spatial and temporal resolution has been shown to be suitable for a more elaborate description of LA function (7,8).

We have recently reported that LA fractional change (difference between maximal and minimal LA volume) and left atrial ejection fraction (LAEF) were more closely related to clinical signs of heart failure than LAmax measured with MDCT in a group of patients with coronary artery disease (8). Based on this observation, we hypothesized that decreased LA function assessed with MDCT is an independent predictor of adverse outcome in patients with acute coronary artery disease, and is incremental to LAmax.

In the current study, we therefore tested the hypothesis that MDCT measures of LA function predict outcome in patients with a recent non–ST-segment elevation myocardial infarction (NSTEMI).

METHODS

Between December 2006 and January 2009, consecutive patients (n = 1,409) with NSTEMI, referred for invasive coronary angiography at Copenhagen University Hospital Rigshospitalet, were screened for participation in the study. NSTEMI was defined according to guidelines (9) as symptoms with acute chest pain and/or electrocardiographic changes without persistent ST-segment elevation and with a characteristic rise and fall in serum troponin T. Patients were scanned with MDCT prior to the invasive procedure as part of a research project. Patients with contraindication to MDCT, history of chronic renal disease or elevated plasma creatinine (>125 μmol/l, n = 157), history of atrial arrhythmias or arrhythmia during MDCT (n = 59), known allergy to iodine contrast (n = 5), hemodynamic instability (n = 18), mitral insufficiency (n = 7), and refusal to participate (n = 76), were not enrolled in the study. Due to logistical reasons, 699 patients were not enrolled, primarily due to absence of scanner availability or coincidence with the invasive procedure. After enrolment of 388 patients, 4 had to be excluded because they could not be in follow-up due to nonresidency. Accordingly, the final study population consisted of 384 patients (Fig. 1). The study was approved by the local ethics committee, and informed consent was obtained from all patients.

MDCT and invasive coronary angiography were performed in all included patients. Treatment strategy was decided by the interventional cardiologist according to international guidelines, blinded to MDCT findings (9).

Previous medical history and cardiovascular risk profile of the patients was recorded from hospital charts. Clinical signs of heart failure within 5 days prior to MDCT were recorded according to the Killip class.

The endpoint of the study was death from all causes. Vital status of included patients was recorded for a minimum of 2 years after inclusion from electronic databases containing vital status in Denmark (Green System, CSC Scandihealth) and the Faroe Islands (Cambio COSMIC registry).

Multidetector computed tomography. All patients were scanned using a 64-slice MDCT scanner (Toshiba Aquillion, Toshiba Medical Systems Corporation, Otawara, Japan) prior to angiography and/or intervention with the following parameter settings: tube voltage 120 to 135 kV depending on body mass index, detector collimation 64 × 0.5 mm, and rotation time between 350 ms and 500 ms depending on the heart rate. Depending on expected scan time, 70 to 100 ml of contrast agent (Visipaque 320, GE Healthcare, Chalfont St. Giles, United Kingdom) was infused with a rate of 5 ml/s, followed by 30 ml of 70:30 (%) contrast/saline mix, and then 30 ml of pure saline chaser. Image acquisition was initiated by automatic bolus triggering. The estimated radiation dose was 14 to 20 mSv with use of retrospective gating and dose modulation. Raw data were reconstructed in 5% intervals throughout the cardiac cycle, with a slice thickness and increment of 2.0/2.0 mm. No additional beta-blocker or other medication was administered prior to MDCT examination. All image data were transferred to an

ABBREVIATIONS AND ACRONYMS

AUC = area under the receiver-operator characteristic curve
CC = cyclic change
LA = left atrium
LAEF = left atrial ejection fraction
LAmax = left atrial maximal volume
LAmin = left atrial minimal volume
LASV = left atrial stroke volume
LV = left ventricular
MDCT = multidetector computed tomography
NSTEMI = non–ST-segment elevation myocardial infarction

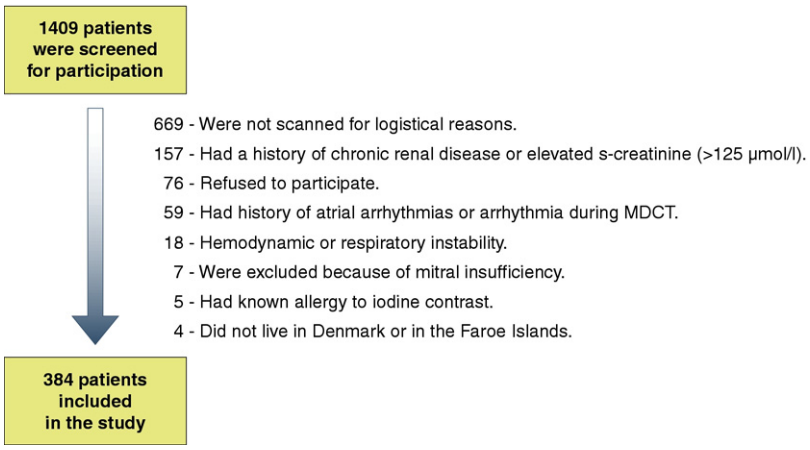

Figure 1. Flow Chart of Patient Inclusion

Flow chart of patient inclusion, with reasons for exclusion and total number of patients in the study population. MDCT = multidetector computed tomography.

external workstation (Vitrea 2, version 4.0, Vital Images Inc, Minnetonka, Minnesota) for image analysis blinded to clinical information.

Image analysis. LA volumes were measured as previously described (8), by tracing the endocardial border on 15 to 20 tomographic slices—depending on size and shape of the chamber—using axial view images. The pulmonary veins were carefully excluded, whereas the LA appendage was included in the LA cavity. The software interpolated the endocardial segmentation to calculate the volume. Volumes were measured in 5% intervals during the RR cycle, and a time–volume plot was generated for each patient.

LA reservoir function was assessed as cyclic change (CC) (calculated as the difference between LAmax and left atrial minimal volume [LAmin]) and fractional change (calculated as CC/LAmax).

LA passive emptying was assessed as reservoir volume (calculated as the difference between LAmax and the minimal mid-diastolic volume; defined as the lowest point between LAmax and "the LA volume immediately before atrial systole") and reservoir fraction (reservoir volume/LAmax). LA active emptying was assessed as left atrial stroke volume (LASV) (defined as the difference between "LA volume immediately before atrial systole" and LAmin) and LAEF (LAEF = LASV/"LA volume just before atrial systole").

Interobserver variability was assessed in 50 patients. Mean percentage error of LAmin was 1 ± 11%, of LAmax 3 ± 9%, of fractional change 5 ± 11%, and of LAEF 2 ± 19%. At our center, agreement between assessment of LA size and function between cardiac magnetic resonance imaging and MDCT has been assessed in 50 patients with ischemic heart disease. Mean percentage error was: LAmin 4 ± 14%, LAmax 9 ± 12%, fractional change 2 ± 13%, and LAEF −3 ± 20% (10).

LV end-diastolic volume, LV end-systolic volume, and left ventricular ejection fraction (LVEF) were measured as previously described (11).

Statistics. Statistical analysis was performed using SAS version 9.13 (SAS Institute, Cary, North Carolina). Continuous variables are presented as mean ± SD and categorical variables as frequencies and percentages. For statistical comparisons, a 2-tailed *t* test for independent samples was used for continuous values and chi-square test was used for categorical variables. Continuous variables that were not normally distributed were compared with Mann-Whitney *U* test. Chi-square test for trend was used to test differences between ordered groups.

The relation between mortality and tertiles of LAmax, LAmin, fractional change, and LAEF was plotted according to the Kaplan-Meier method, and death rates were compared by the log-rank test. The relationship between MDCT variables, clinical variables, and all-cause mortality was assessed using Cox proportional hazards regression. First, a univariable analysis was performed for potential LA predictors of clinical events: LAmax, LAmin, fractional change, CC, reservoir volume, reservoir fraction, LAEF, LASV, and in addition, for age, sex, hypertension, LVEF, number of diseased coronary vessels, Killip class, diabetes, and previous myocardial infarction. Second, multivariable regression analysis with forced entry of LAmax, LAmin, fractional change, CC, LAEF, and

LASV was performed separately with adjustment for established clinical predictors (age, LVEF, number of diseased vessels, and Killip class ≥2). Finally, the overall Wald chi-square of a model including LAmax, age, LVEF, number of diseased vessels, and Killip class ≥2 was compared with a model with the aforementioned variables and either fractional change or LAEF to assess the incremental information of LA function. The proportional hazard assumption was checked through the method of cumulative residuals. The discriminative power of LA size and function after 2 years of follow-up was assessed by calculating the mean of the area under the receiver-operator characteristic curve (AUC). A p value <0.05 was considered statistically significant.

RESULTS

Demographic information of patients enrolled in the study is given in Table 1. Patients screened for participation in the study but not enrolled (n = 1,025) were older (67 ± 12 years vs. 61 ± 12 years, p < 0.001), had lower estimated glomerular filtration rate (76 ± 38 ml/min vs. 87 ± 20 ml/min, p < 0.001), and were more likely to have diabetes (24% vs. 16%, p = 0.001) and 3-vessel disease (26% vs. 21%, p = 0.03) than patients who were enrolled, but did not differ with regard to other risk factors or treatment strategy.

Table 1. Demographics (n = 384)

Age, yrs	61 ± 12
Female	92 (24)
Body mass index, kg/m^2	28 ± 5
Hypertension	191 (50)
Diabetes	61 (16)
eGFR	87 ± 20
Smoker (current or former)	285 (74)
Previous myocardial infarction	85 (23)
Coronary artery disease	
No significant stenosis	67 (17)
1-vessel disease	138 (36)
2-vessel disease	98 (26)
3-vessel disease	79 (21)
Left main disease	21 (5)
Left ventricular ejection fraction, %	58 ± 12
Troponin T maximum, ng/ml	0.84 ± 1.30
Killip class ≥2	45 (12)
Medication at discharge	
Beta-blocker	319 (83)
ACE-inhibitor	107 (28)
AIIA	34 (9)
Aspirin	370 (96)
Clopidogrel	363 (95)
Statin	371 (97)

Values are mean ± SD or n (%).
ACE = angiotensin-converting enzyme; AIIA = angiotensin II receptor antagonist; eGFR = estimated glomerular filtration rate (calculated using the Modification of Diet in Renal Disease formula).

In all enrolled patients, LA size and function could be assessed. Mean LAmax was 55 ± 11 ml/m^2, LAmin 31 ± 11 ml/m^2, fractional change 45 ± 9%, and LAEF 32 ± 9%.

In univariate regression analysis, LA volumes were correlated with EDV index (LAmax: β = 0.26, p < 0.001, and LAmin: β = 0.22, p < 0.001) and inversely correlated with LA function (fractional change: β = −0.08, p < 0.001, LAEF: β = −0.09, p < 0.001).

All-cause mortality. The median follow-up time was 36 months (range 10 to 1,551 days). During follow-up, 35 (9%) patients died. Overall, 1- and 2-year survival in the study cohort was 97% and 94%, respectively, which was significantly higher than the 1- and 2-year survival among patients excluded from the study (89% and 84%, respectively, p < 0.001).

In the study cohort, surviving patients were characterized by lower age, lower prevalence of 3-vessel or left main disease, higher LVEF, and lower Killip class (Table 2). Mortality according to tertiles of LA size and function is presented in Figure 2. High LAmin was associated with poor survival, whereas there was no significant difference between LAmax tertiles using log-rank statistics. Poor LA function (reduced fractional change and LAEF) was associated with adverse outcome.

In a univariate Cox regression analysis, the significant LA predictors of all-cause mortality were LAmin, fractional change, CC, LAEF, and LASV (Table 2). LAmax, LAmin, fractional change, CC, LAEF, and LASV were separately included in separate multivariate Cox regression models with forced entry of a priori known predictors of mortality including: age, LVEF, number of diseased vessels, and Killip class ≥2. In those models, fractional change, CC, LAEF, and LASV remained independent predictors of all-cause mortality, whereas LA size was no longer significant (Table 3).

The overall Wald chi-square of a model including LAmax, age, Killip class, LVEF, and number of diseased vessels was 38.00. If fractional change was added to this model, the overall Wald chi-square was 43.20, and if LAEF was added to the model, the overall Wald chi-square was 44.61, p < 0.05 for difference.

Receiver-operator characteristic curves are displayed in Figure 3. The AUC was 0.57 for LAmax, 0.74 for fractional change, and 0.66 for LAEF. Only

Table 2. Unadjusted Predictors of All-Cause Mortality

	Survivor (n = 349)	All-Cause Mortality (n = 35)	Wald	HR	95% CI	p Value
Age, yrs	60 ± 12	71 ± 7	24	1.09	1.05–1.13	<0.001
Female	86 (25)	6 (17)	1	1.50	0.63–3.60	0.33
Hypertension	171 (49)	20 (57)	1	1.43	0.73–2.80	0.29
Diabetes	52 (15)	9 (26)	3	1.86	0.87–3.97	0.11
Previous myocardial infarction	74 (22)	11 (32)	2	1.69	0.79–3.30	0.19
Vessel disease (0, 1, 2, 3, or LM)	76 (22)*	16 (46)*	12	1.80	1.29–2.64	<0.001
Left ventricular ejection fraction, %	59 ± 12	52 ± 14	10	0.96	0.94–0.99	0.003
Troponin T maximum, ng/ml	0.8 ± 1.2	1.2 ± 2.3	2	1.15	0.96–1.37	0.14
Killip class ≥2	33 (9)	12 (34)	19	4.40	2.17–8.77	<0.001
LA maximal volume index, ml/m^2	55 ± 11	58 ± 14	2	1.02	0.99–1.05	0.12
LA minimal volume index, ml/m^2	30 ± 10	37 ± 15	16	1.05	1.02–1.07	<0.001
Fractional change, %	46 ± 9	37 ± 11	29	0.92	0.90–0.95	<0.001
Cyclic change, ml/m^2	25 ± 5	21 ± 5	18	0.90	0.85–0.95	0.001
Reservoir volume, ml/m^2	11 ± 4	10 ± 3	1	0.95	0.95–1.04	0.28
Reservoir fraction, %	20 ± 7	18 ± 6	3	0.11	0.01–1.84	0.10
LA ejection fraction, %	33 ± 8	26 ± 10	22	0.92	0.89–0.95	<0.001
LA stroke volume, ml/m^2	14 ± 4	12 ± 4	11	0.85	0.78–0.93	0.001

Values are mean ± SD or n (%). *3-vessel or left main stem (LM) disease.
CI = confidence interval; HR = hazard ratio; LA = left atrial.

AUC for fractional change was significantly higher than AUC for LAmax (p = 0.018).

To determine whether the location of the infarct-related region had any influence on the LA function, we made a subgroup analysis of 138 patients with 1-vessel disease, and tested whether the culprit location in the left anterior descending coronary artery, left circumflex coronary artery, or right

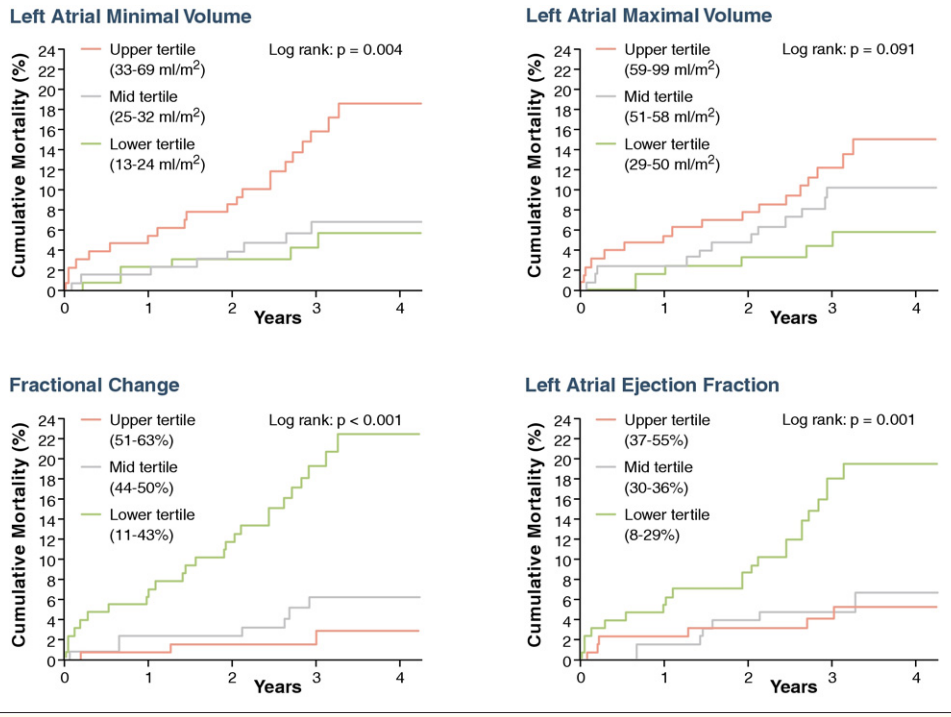

Figure 2. Kaplan-Meier Plots

Mortality in the study population stratified in tertiles of left atrial size and function.

Table 3. Univariate and Adjusted LA Predictors of All-Cause Mortality According to Standard Deviations of the LA Variables

	SD	Univariate HR	95% CI	p Value	Adjusted HR	95% CI	p Value
LA maximal volume, ml/m^2	11	1.27	0.94–1.71	0.120	1.06	0.76–1.47	0.53
LA minimal volume, ml/m^2	11	1.61	1.27–2.04	<0.001	1.31	0.96–1.79	0.08
Fractional change, %	10	0.47	0.36–0.62	<0.001	0.65	0.45–0.94	0.02
Cyclic change, ml/m^2	6	0.46	0.36–0.65	0.001	0.68	0.47–0.98	0.04
LA ejection fraction, %	9	0.47	0.35–0.63	<0.001	0.63	0.44–0.91	0.01
LA stroke volume, ml/m^2	4	0.55	0.38–0.80	0.001	0.64	0.44–0.92	0.02

Adjustments were made for age, left ventricular ejection fraction, Killip class, and number of diseased coronary vessels.
Abbreviations as in Table 2.

coronary artery was associated with different values for fractional change and LAEF; no difference between the groups was noted (fractional change p = 0.47, LAEF p = 0.17). We also made an analysis of the left anterior descending coronary artery versus non–left anterior descending coronary artery and found no difference between groups (fractional change p = 0.43, LAEF p = 0.22).

DISCUSSION

To the best of our knowledge, this is the first study that has investigated the prognostic information of LA size and function assessed with MDCT in acute coronary syndrome. The study suggests in a population with NSTEMI that LA functional measures are superior to LA size to predict outcome. This association remained after adjustment for known risk factors of outcome including age, LVEF, and Killip class.

LA size and function. During ventricular systole, the endocardium undergoes significant radial displacement, and as a result of contraction of longitudinally oriented myocardial fibers, the atrioventricular plane is pulled towards the apex of the heart. This will cause stretching of the LA, augmenting filling of the atrium. LV active relaxation starts in late systole, which together with elastic recoil results in rapid early ventricular filling and return of the atrioventricular plane to the resting level. Consequently, the LA will rapidly return to a smaller volume during early LV filling. With subsequent diastasis, there is no wall motion, and the atrium and the ventricle form a single chamber with equalization of LA and LV diastolic pressures. During this phase, almost no changes in LA volume will occur. Only during the final portion of ventricular diastole, atrial systole generates energy with contraction and subsequent additional LV filling. On the basis of this, LAEF will reflect the difference in LA volume from the volume at the end of diastasis and at the end of atrial contraction relative to diastasis volume. The diastasis volume is, as discussed, dependent on LV diastolic pressure and LV function, whereas LAmin is determined by a combination of intrinsic LA contractility and the load the LA faces (LV effective chamber compliance and retrograde flow into the pulmonary veins determined by pulmonary venous capacitance). Finally, fractional change, which describes the maximal change in LA volumes relative to LAmax, can be regarded as the sum of the numerous intrinsic ventricular and atrial properties discussed.

Despite this complexity, the clinical relevance of the LA imaging has been derived almost exclusively from measurements of LAmax or merely maximal diameter assessed by 2-dimensional or M-mode echocardiography. Clearly, this may not encompass the full potential of prognostic information of the LA.

In agreement, the present study demonstrates that impaired LA function with reduced fractional change and LAEF were independent predictors of mortality in low-risk patients with NSTEMI after adjustment for known risk factors. Moreover, the

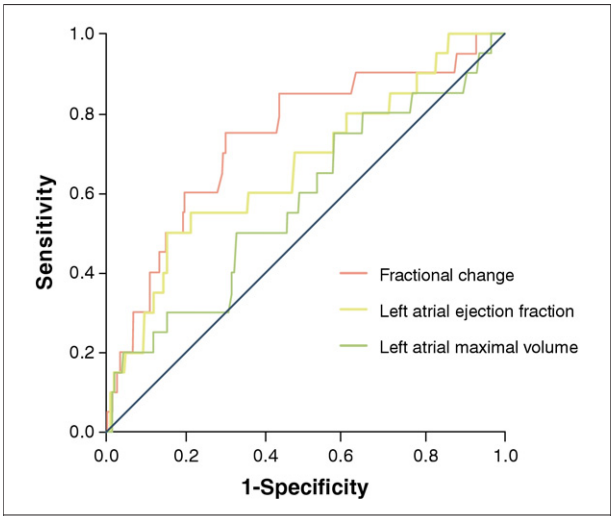

Figure 3. Receiver-Operator Characteristic Curves

Receiver-operator characteristic curves for fractional change, left atrial ejection fraction, and left atrial maximal volume, demonstrating the ability to predict all-cause mortality after 2 years follow-up.

prognostic significance of fractional change and LAEF contained incremental value to LAmax.

The pathophysiological link between reduced LA function and adverse clinical outcome cannot be directly elucidated from the present study. However, in a previous study, Torabi et al. (12) found in a consecutive population of 896 patients that 84% of deaths occurring during follow-up were preceded by heart failure at some point. The development of symptomatic heart failure (shortness of breath on exertion) in patients with NSTEMI requires LV filling pressures to be elevated; the cause of this may be a complex interplay of multiple factors including myocardial ischemia, neurohormonal activation, renal failure, left ventricular remodeling, hypertrophy, and pre-existing decrease of effective chamber compliance. All of these factors would be anticipated to affect atrial size and contraction. The present results and the results of a previous hypothesis-generating study (8), where we found that clinical signs of heart failure are associated with impaired LA mechanical function (fractional change and LAEF) to a greater extent than LAmax, concur with this theory. Impaired LA function could perhaps be interpreted as an early morphological manifestation for increased risk of developing heart failure and, eventually, death. Thus, although it is speculative, we believe that impaired LA function is a risk factor for the development of heart failure and that this could provide the link to poor prognosis.

Regardless of the mechanism responsible for the predictive value of LA functional imaging, an improved prognostic evaluation could be clinically useful. Further studies are needed to explore the potential clinical benefits of LA functional imaging.

In the present study, LAmax was only a weak predictor of outcome as opposed to previous studies where LAmax on echocardiography has been reported to be an important predictor of outcome. Although the present study provides no direct insight into this discrepancy, it could likely be caused by different risk profiles in study populations. Patients with increased risk due to renal dysfunction, hemodynamic instability, and so on were excluded from the present study, leaving a population with a considerably lower risk profile and an a priori lower likelihood of presenting with an enlarged LA than previous echocardiographic studies. Possibly this explains that LAmax is less potent in the present study in predicting outcome. In addition, differences in defining LA volume on echocardiography and MDCT may have influenced the results.

Study limitations. In the present study, patients at the highest risk were not enrolled, thus the study describes a low-risk group without cardiac arrhythmias or renal dysfunction suitable for MDCT scanning. Accordingly, the present results may not be generalized to the average NSTEMI population. However, this may emphasize 2 important points. First, it may explain that we do not find a significant relation between LAmax and survival. LAmax has previously been shown to be a powerful predictor in myocardial infarction patients with more comorbidity and higher mortality rates (4,5). Second, this seems to emphasize that LA functional values are sensitive predictors of mortality.

To measure LA size and function, we used retrospectively gated MDCT scanning. MDCT has high spatial resolution, but lower temporal resolution than magnetic resonance imaging and echocardiography, which could affect the precision of MDCT. However, several studies have consistently demonstrated very good agreement and correlation between MDCT and magnetic resonance imaging, and good correlation between MDCT and echocardiography when measuring LA size and function (13–17).

In contemporary clinical cardiac MDCT imaging implementing prospective data acquisition, the use of retrospective gating is limited because of the relatively high radiation exposure. Accordingly, functional information may not be routinely available in future MDCT examinations of low-risk patients. LA size and functional values, however, may be assessed accurately with both magnetic resonance imaging (18) and promisingly also with 2- and 3-dimensional echocardiography (19–21).

Echocardiography was performed at referring hospitals, and not according to a specific protocol, on multiple different ultrasound systems with focus on assessment of LV systolic function and detection of left-sided valve disease. However, unfortunately, no systematic assessments of LV diastolic function or LAmax were available.

We had four obvious explanatory variables—a priori known predictors of death, which were significant in univariate analysis—for a multivariable regression model: age, Killip class, LVEF, and number of diseased vessels. This number of variables is in the higher end of what is statistically reasonable with 35 events, and the results of the multivariable analysis should be interpreted with appropriate caution. The low number of events prevented us from including other interesting variables in the models, which may have decreased the prognostic utility of the LA variables.

CONCLUSIONS

The present study demonstrates that impaired LA function in terms of reduced LA fractional change and LAEF predict a poor prognosis in low-risk patients with NSTEMI, even after adjustment for conventional risk factors. Furthermore, the prognostic significance of fractional change and LAEF contained incremental value to LAmax. Measurement of LA function could serve as a sensitive tool for early risk stratification in apparently low-risk patients with NSTEMI. Further studies should reproduce these results in other patient categories with coronary artery disease to explore the extent of patients that we may be able to risk-stratify with this method, preferably also using echocardiography or magnetic resonance imaging.

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