Improving risk stratification after acute myocardial infarction : focus on emerging applications of echocardiography

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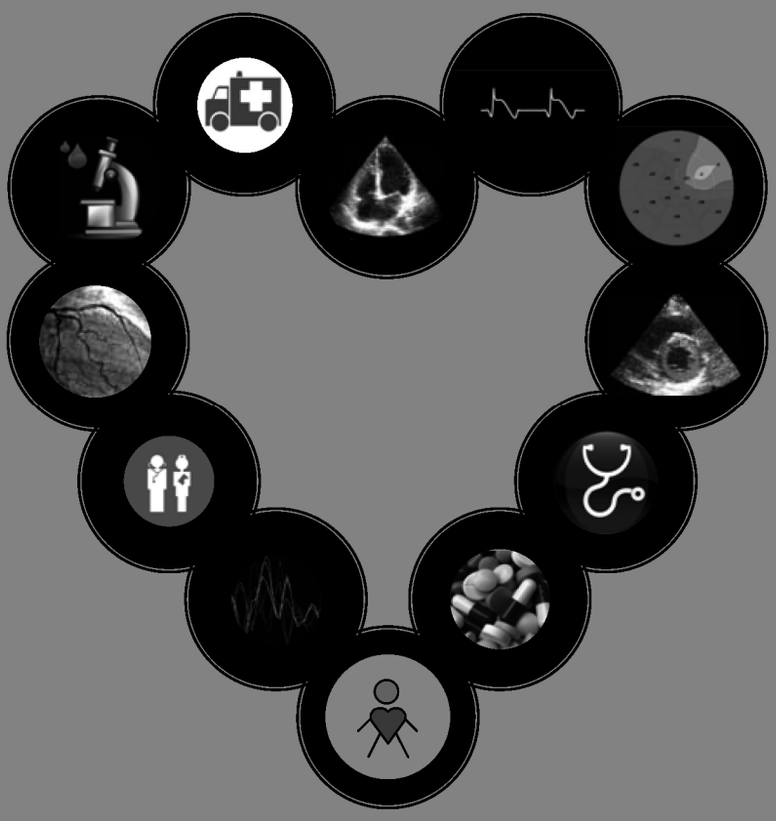

Chapter 13

Left Atrial Strain is Related to Adverse Events in Patients after Acute Myocardial Infarction Treated with Primary Percutaneous Coronary Intervention

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Abstract

Objectives

Left atrial (LA) maximal volume provides important prognostic value in patients after acute myocardial infarction (AMI). Recently, LA mechanical function and LA strain have been introduced as alternative methods to assess LA performance more accurately. The purpose of the current study was to evaluate the relation between LA volumes, mechanical function and strain, and adverse events in patients after AMI.

Methods and results

Patients admitted with AMI underwent 2D-echocardiography within 48 hours of admission. LA volumes and LA performance (mechanical function and systolic strain) were quantified. The endpoint was a composite of all-cause mortality, reinfarction and hospitalization for heart failure. A total of 320 patients (mean age: 60±12 years, 78% men) were followed for 27 ± 14 months. During follow-up, 48 patients (15%) reached the composite endpoint. After adjustment for clinical and echocardiographic parameters, LA maximal volume (HR 1.05, CI 1.00–1.10, p = 0.04) and LA strain (HR 0.94, CI 0.89–0.99, p = 0.02) were independently associated with adverse outcome. In addition, LA strain provided incremental value to LA maximal volume (p = 0.03) for the prediction of adverse outcome.

Conclusions

In patients after AMI treated with primary percutaneous coronary intervention, LA strain provides additional prognostic value beyond LA maximal volume.

Introduction

Left atrial (LA) maximal volume has been recognized as a powerful predictor of mortality and hospitalization for heart failure in patients after acute myocardial infarction(AMI).[1-3] Normal LA volumes are associated with good outcome, even in patients with depressed left ventricular(LV) function.[1 4] On the other hand, larger LA volumes are associated with chronic increased LV filling pressures and adverse outcome post-AMI.[2] Besides LA volume, recent studies have shown the value of several LA functional parameters.[5] For example, LA ejection force is a measure of LA mechanical function that is strongly related to LV diastolic function. In the Strong Heart Study, LA ejection force was an independent predictor of cardiovascular events.[5 6] Therefore, besides quantification of LA size, assessment of LA mechanical function may have additional prognostic value in post-AMI patients. However, the assessment of these parameters involves numerous geometrical assumptions and often results in underestimation of the atrial size.[7]

Direct evaluation of atrial myocardial function is currently feasible with speckle-tracking imaging. This novel technique permits assessment of active myocardial deformation which may provide additive value concerning LA function when compared to conventional echocardiographic measurements.[8 9] Accordingly, the purpose of the current evaluation was to investigate the association between LA performance expressed in LA volumes, mechanical function and strain, and adverse events in post-AMI patients.

Methods

Patient population and data collection

Consecutive patients admitted with ST-segment elevation AMI treated with primary percutaneous coronary intervention were evaluated. Diagnosis of ST-segment elevation AMI was made based on typical electrocardiographic changes with clinical symptoms associated with elevation of cardiac biomarkers.[10] Clinical and echocardiographic data were prospectively entered into the departmental Cardiology Information System (EPD-Vision®, Leiden University Medical Center) and the echocardiography database, respectively, and retrospectively analyzed.[11 12] All patients were treated according to the institutional AMI protocol(MISSION!).[11] This protocol, designed to improve care around AMI, includes structurized medical therapy, 2D-echocardiography performed <48 hours of admission and

standardized follow-up, as described previously.[11] The baseline echocardiogram was used to assess LA and LV function. Specifically, LA function was assessed with phasic volumes by conventional echocardiography and with LA strain and strain rate by speckle-tracking imaging. Of note, patients with atrial fibrillation were excluded.

In addition, 35 normal controls selected from an echocardiographic database were included to provide the normal reference values of LA phasic volumes, strain and strain rate.[13] The group of controls comprised individuals matched for age and gender who were referred for echocardiography with atypical chest pain, palpitations, or syncope without murmur and did not show structural heart disease. Those individuals who showed LV dilatation, had known hypertension, or were referred for echocardiographic evaluation of known valvular disease, murmur, or heart failure were excluded.

Echocardiography

All patients were imaged in the left lateral decubitus position using a commercially available system (Vivid 7, General Electric-Medical Systems, Horton, Norway). Images were obtained with a simultaneous ECG-signal, using a 3.5-MHz transducer at a depth of 16cm in the parasternal and apical views. Standard M-mode and 2D-images were acquired during breath hold and saved in cine-loop format. Analysis of echocardiographic images was performed offline by 2 independent observers using dedicated software (EchoPac version 108.1.5, General Electric-Vingmed).

LV end-systolic volume, end-diastolic volume and ejection fraction were assessed using the biplane Simpson's method in the apical 4-and 2-chamber views.[14]

In addition, the LV was divided into 16 segments and each segment was analyzed individually and scored based on its motion and systolic thickening (1=normokinesis, 2=hypokinesis, 3=akinesis, 4=dyskinesis). Wall motion score index was calculated as the sum of the segment scores divided by the number of segments scored.[14]

Severity of mitral regurgitation was graded semiquantitatively from the jet area of color-flow Doppler data and by measuring the width of the vena contracta. Mitral regurgitation was characterized as: mild=jet area/LA area<20% and vena contracta width<0.30 cm, moderate=jet area/LA area 20%–40% and vena contracta width 0.30–0.69 cm, and severe=jet area/LA area >40% and vena contracta width ≥0.70 cm.[15]

To assess diastolic function, pulsed-wave Doppler of the mitral valve inflow was obtained by placing the Doppler sample volume between the tips of the mitral leaflets. The early (E) and late(A) peak diastolic velocities and E-wave deceleration time were measured. E/E'-ratio was obtained by dividing E by E', which was measured using color-coded tissue Doppler imaging at the septal side of the mitral annulus in the apical 4-chamber view.[16]

Analysis of left atrial function

LA function consists of the reservoir period (inflow during ventricular systole), conduit period (passive emptying during ventricular relaxation and diastasis) and contractile period (active emptying). To analyze all components of LA function, LA volumes were calculated according to the biplane Simpson's method at 3 time points: (1) maximal volume (LAmax) at end-systole, just before mitral valve opening; (2) minimal volume (LAmin) at end-diastole, just before mitral valve closure; and (3) volume before atrial active contraction (LApreA) obtained from the last frame before mitral valve reopening or at time of the P wave on the surface electrocardiogram. All LA volumes were indexed to the body surface area.[14]

LA mechanical function was derived from the LA volumes and expressed with the following formulas: (1) total atrial emptying fraction: LA total ejection fraction = [(LAmax–LAmin)/LAmax]*100; (2) active atrial emptying fraction: LA active ejection fraction = [(LApreA–LAmin)/LApreA]*100, which is considered an index of LA active contraction; (3) passive atrial emptying fraction: LA passive ejection fraction = [(LAmax–LApreA)/LAmax]*100, which is considered an index of LA conduit function; and (4) atrial expansion index: LA expansion index = [(LAmax–LAmin)/LAmin]*100, which is considered an index of LA reservoir function.[17]

Longitudinal LA wall deformation was assessed in the apical views using speckle-tracking analysis.[18] This novel software analyzes motion by tracking frame-to-frame movement of natural acoustic markers in 2 dimensions. All images were recorded with a frame rate of >40fps (range 40–100fps) for reliable analysis. The LA endocardial border was manually traced and the automatically created region of interest was adjusted to the thickness of the myocardium. The extent of LA wall stretching during the reservoir period may be important for maintaining adequate LV filling.[19] Therefore, LA peak systolic longitudinal

strain and strain rate were assessed at each mid-LA segment (septal, lateral, anterior, inferior and posterior) in the apical views and averaged as a measure of LA compliance.[8 20] Segments were discarded if tracking was of poor quality. Strain and strain rate analysis was feasible in 79% of segments.

Follow-up and endpoint definitions

All patients were followed according to the protocol and the occurrence of adverse events was noted. Patients, of whom more than 6 months follow-up data were lacking, were considered as lost to follow-up, and excluded from further analysis. The endpoint was defined as a composite of all-cause mortality, non-fatal reinfarction and hospitalization for heart failure. Nonfatal reinfarction was defined based on criteria of typical chest pain, elevated cardiac enzyme levels, and typical changes on the electrocardiogram[10] Hospitalization for heart failure was defined as hospitalization for new-onset or worsening of heart failure.

Statistical analysis

Continuous data are presented as mean±standard deviation (SD) and categorical data are presented as frequencies and percentages. Differences in characteristics between patient groups were evaluated using the unpaired Student's *t*-test and chi-square test.
The primary aim was to assess the association between LA performance and adverse events after adjusting for clinical and echocardiographic covariates. Separate multivariable models were constructed for LA volumes, mechanical function, strain and strain rate using Cox proportional hazards analysis to evaluate the individual prognostic importance of the different LA measurements. Because of the relative low number of events, the number of covariables had to be limited. Accordingly, based on both clinical judgment and univariable statistical significance, age, Killip class, multivessel disease, peak cardiac troponin T level, LV ejection fraction, E/E'-ratio and mitral regurgitation were introduced in the model. In addition, the potential relationship between renal function and LA volumes, phasic and mechanical function, strain and strain rate was assessed with ANOVA tests. For this purpose, estimated glomerular filtration rate (eGFR) was calculated using the standard formula by Cockcroft and Gault and expressed in ml/min/1.73 m^2.[21] Patients were divided

into 3 subgroups according to the cutoff values proposed by the National Kidney Foundation practice guidelines: an eGF ≥90 ml/min/1.73 m^2 for normal kidney function, eGFR 60-90 ml/min/1.73 m^2 for mildly decreased, and eGFR <60 ml/min/1.73 m^2 for moderately to severely decreased kidney function.[22] To further investigate the clinical relevance of LA performance, the population was stratified into 2 groups according to LA dysfunction. The cut-off value for LA maximal volume was chosen at 32mL/m^2 which corresponds to 2SDs from the normal LA size and has been previously validated in relation to clinically relevant endpoints.[1 2 4] The normal value of LA strain in the group of 35 included normal controls was 39 ± 10%. Patients were therefore divided according to the mean value minus 2SDs, which corresponds to the lower limit of normal LA strain (19%). Event rates were plotted in Kaplan-Meier curves for the composite endpoint and the study population divided by the previously mentioned cut-off values, and groups were compared using the log-rank test. The incremental value of LA performance to known risk factors for adverse outcome (age, Killip class, multivessel disease, peak cardiac troponin T level, LV ejection fraction, E/E'-ratio and mitral regurgitation) was established. For this purpose, those characteristics were entered in the Cox proportional hazard model in a stepwise fashion. Subsequently, LA maximal volume and LA strain were entered individually, to test further incremental value. Global chi-square values including significance levels were calculated.

Finally, 15 patients were randomly selected to test the intra- and interobserver reproducibility of LA measurements. Bland–Altman analyses were performed.

All statistical tests were two-sided, and a *P* value<0.05 was considered statistically significant.

Results

Patient characteristics

A total of 368 consecutive AMI patients treated with primary percutaneous coronary intervention were evaluated. Three (0.8%) patients died before echocardiographic examination could be performed and in 8(2.2%) patients echocardiographic assessment was not available <48 hours of admission due to logistic reasons. Another 19 (5.7%) patients were excluded from further analysis because image quality was not sufficient for analysis

and 18 (4.9%) patients were lost to follow-up. The study population therefore comprised 320 patients. Tables 1 and 2 summarize the clinical and echocardiographic characteristics. Mean age of the patients was 60 ± 12 years and 78% were male. Baseline echocardiography revealed a LV ejection fraction of 46 ± 8% and maximal LA volume was 25 ± 7 ml/m^2. Mean LA strain and strain rate were 33 ± 11% and 2.3 ± 0.7s^{-1}, respectively.
Echocardiographic data obtained in AMI patients were compared with the group of 35 normal controls. Post-AMI patients had significantly larger LA maximal volume (25 ± 7 vs. 22 ± 6 ml/$m^{2,}$ p = 0.04) and lower LA total ejection fraction (56 ± 11 vs. 61 ± 5%, p = 0.002) compared with the group of normal controls. Interestingly, passive emptying fraction was significantly reduced in the post-AMI patients (28 ± 10 vs. 39 ± 13%, p <0.001), which was compensated by an increased active emptying fraction (38 ± 11 vs. 34 ± 14%, p = 0.04). LA reservoir function was significantly reduced (140 ± 65 vs. 164 ± 41%, p = 0.03), also reflected by the lower LA strain (39 ± 10 vs. 33 ± 11%, p = 0.002) in comparison with the normal controls (Table 2).
In addition, the relationship between renal function LA volumes and LA mechanical function was evaluated. There were no significant differences in LA volumes and phasic function among the 3 categories of eGFR. However, decreasing eGFR was associated with significantly lower LA strain and strain rate (from 35 ± 11 to 32 ± 11 and 25 ± 11%, p <0.001 for LA strain and from 2.4 ± 0.7 to 2.2 ± 0.7 and 1.9 ± 0.7s^{-1}, p = 0.005 for LA strain rate).
Fifteen patients were randomly identified for inter- and intraobserver agreement. According to the Bland-Altman analysis, intraobserver variability was good with mean differences of 1.6 ± 2.4 ml/m^2 for LA maximal volume,-1.2 ± 1.6 ml/m^2 for LA minimal volume, 1.0 ± 1.4 ml/m^2 for LA preA volume, 0.5 ± 3.0% for LA strain and 0.06 ± 0.22 s^{-1} for LA strain rate. Interobserver reproducibility was also good with mean differences for LA maximal volume, LA minimal volume, LA preA volume, LA strain and LA strain rate of 2.1 ± 4.2 ml/m^2, -1.8 ± 2.1 ml/m^2, 1.6 ± 2.6 ml/m^2, 1.0 ± 4.4% and -0.12 ± 0.24s^{-1}, respectively.

Follow-up

During a mean follow-up of 27 ± 14 months, 48 patients (15%) reached the composite endpoint: 29 patients died (9%), 11 patients (3%) had a nonfatal reinfarction and 14 patients

(4%) were hospitalized for heart failure. Differences in clinical and echocardiographic characteristics between patients who reached the composite endpoint and patients who remained event-free are shown in Tables 1 and 2.

Table 1. Baseline Clinical Characteristics

	Controls (N = 35)	***All Patients (N = 320)***	***Event (N = 48)***	***Event-free (N = 272)***	***P***
Clinical information					
Age(years)	58 ± 12	60 ± 12	64 ± 14	59 ± 11	0.03
Male gender	24 (69%)	249 (78%)	37 (77%)	212 (78%)	0.90
Killip class≥2		34 (11%)	13 (27%)	21 (8%)	<0.001
Current smoking		169 (53%)	24 (51%)	145 (53%)	0.78
Diabetes		26 (8%)	7 (15%)	19 (7%)	0.08
Hyperlipidemia		65 (20%)	14 (29%)	51 (19%)	0.10
Hypertension		97 (30%)	19 (40%)	78 (29%)	0.13
Prior MI		14 (4%)	3 (6%)	11 (4%)	0.49
eGFR (ml/min/1.73 m^2)		95 ± 30	84 ± 33	97 ± 30	0.01
Infarct characteristics					
LAD culprit vessel		164 (51%)	27 (56%)	137 (50%)	0.45
Multivessel disease		164 (51%)	34 (71%)	130 (48%)	0.003
TIMI flow		3.0 ± 0.3	2.9 ± 0.5	3.0 ± 0.3	0.38
Peak CPK level(U/l)		2629 ± 2087	3824 ± 2780	2418 ± 1868	0.001
Peak cTnT level(μg/l)		8 ± 7	12 ± 10	7 ± 6	0.002
Medication at 6-months follow-up					
ACE inhibitor/ARB		302 (99%)	34 (100%)	268 (99%)	0.48
Antiplatelets		306 (100%)	34 (100%)	272 (100%)	1.00
Beta-blocker		276 (90%)	33 (97%)	243 (89%)	0.15
Statin		301 (98%)	34 (100%)	267 (98%)	0.43

ACE: angiotensin-converting enzyme; ARB: angiotensin receptor blocker; CAD: coronary artery disease; CPK: creatine phosphokinase; cTnT: cardiac troponin T; eGFR: estimated glomerular filtration rate; LAD: left anterior descending coronary artery; MI: myocardial infarction; TIMI: thrombolysis in myocardial infarction.

Table 2. Baseline echocardiographic characteristics

	Controls (N = 35)	*All Patients (N = 320)*	*Event (N = 48)*	*Event-free (N =272)*	*P*
LV end-systolic volume(ml)	39 ± 13	62 ± 21	65 ± 23	62 ± 20	0.29
LV end-diastolic volume(ml)	95 ± 25	115 ± 34	114 ± 36	115 ± 33	0.89
LV ejection fraction(%)	60 ± 6	46 ± 8	43 ± 9	46 ± 8	0.01
Wall motion score index	1.0 ± 0.0	1.5 ± 0.3	1.6 ± 0.3	1.5 ± 0.3	<0.001
E/A-ratio	1.1 ± 0.4	0.9 ± 0.3	1.0 ± 0.4	0.9 ± 0.3	0.13
Deceleration time(ms)	166 ± 59	214 ± 69	201 ± 64	216 ± 69	0.14
E/E'-ratio	11 ± 3	13 ± 5	15 ± 6	13 ± 5	0.14
Moderate or severe MR	0 (%)	25 (8%)	8 (17%)	17 (6%)	0.01
LA max(ml/m^2)	22 ± 6	25 ± 7	27 ± 10	24 ± 7	0.03
LA total ejection fraction(%)	61 ± 5	56 ± 11	51 ± 11	56 ± 11	0.005
LA passive emptying fraction(%)	39 ± 13	28 ± 10	26 ± 9	28 ± 10	0.11
LA active emptying fraction(%)	34 ± 14	38 ± 11	35 ± 11	39 ± 11	0.01
LA reservoir function(%)	164 ± 41	140 ± 65	117 ± 56	144 ± 65	0.008
LA strain(%)	39 ± 10	33 ± 11	26 ± 11	34 ± 11	<0.001
LA strain rate(s^{-1})	2.1 ± 0.5	2.3 ± 0.7	2.0 ± 0.8	2.3 ± 0.7	0.009

E/A: mitral inflow peak early velocity (E) / mitral inflow peak late velocity (A); E/E': mitral inflow peak early velocity (E) / mitral annular peak early velocity (E'); LA: left atrium; LV: left ventricular; MR: mitral regurgitation.

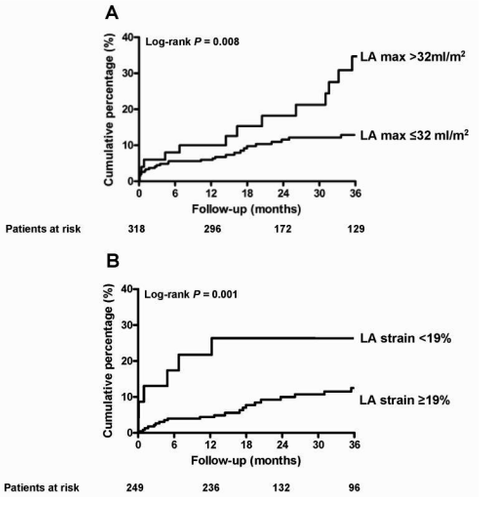

Figure 1.

Cumulative incidence of adverse events in patients stratified by LA maximal volume (panel A) and LA strain (panel B).

Relation between left atrial performance and outcome

Table 3 shows the significant univariable predictors of the composite endpoint. In addition to clinical characteristics and LV function measurements, LA maximal volume, ejection fraction, strain and strain rate were univariable predictors of the composite endpoint. After adjusting LA maximal volume, ejection fraction, strain and strain rate for other variables that predicted adverse outcome, LA maximal volume and LA strain independently predicted the occurrence of the composite endpoint (HR 1.05, 95%CI 1.00–1.10, p = 0.04 and HR 0.94, 95%CI 0.89–0.99, p = 0.02, respectively). However, LA ejection fraction and LA strain rate did not remain significant in the multiple variable analysis (HR 0.99, 95%CI 0.96–1.03, p = 0.63 and HR 0.55, 95%CI 0.28–1.06, p = 0.07, respectively). To further investigate the prognostic value of LA function, LA maximal volume and LA strain were dichotomized according to normal and abnormal LA function with the above described cut-off values. Kaplan-Meier curves for LA maximal volume divided in >32ml/m^2 and ≤32 ml/m^2 and LA strain divided in <19% and ≥19% are shown in Figure 1. The 3-year event rate in patients with LA maximal volume >32ml/m^2 (n = 50) was 35% compared to 14% in patients with LA maximal volume ≤32ml/m^2 (n = 270, p = 0.008). The incidence of adverse events at 3 years was 26% in patients with LA strain <19% (n = 23) and 12% in patients with LA strain ≥19% (n = 228, p = 0.001).

Incremental value of left atrial strain to traditional risk factors and left atrial volume

Global chi-square values were calculated to assess the incremental value of LA function. LA maximal volume provided incremental value to traditional risk factors (age, Killip class≥ 2, multivessel disease, peak cardiac troponin T level, LV ejection fraction, E/E'-ratio and moderate or severe mitral regurgitation) by increasing the global chi square value from 35.5 to 40.2 (p = 0.046). In addition, when LA strain was added to the previous model with LA maximal volume, the predictive power of the model increased even further reflected by the increased in the global chi square from 40.2 to 43.1 (p = 0.03).

Table 3. Cox univariable predictors for the composite endpoint

	Hazard Ratio	*95%CI*	*P*
Age(years)	1.04	1.01–1.07	0.006
Killip≥2	3.64	1.92–6.88	<0.001
Multivessel disease	2.69	1.44–5.02	0.002
Peak creatine phosphokinase level(per 100U/l)	1.02	1.01–1.03	<0.001
Peak cardiac troponin T level(μg/l)	1.07	1.04–1.09	<0.001
LV ejection fraction(%)	0.95	0.92–0.99	0.01
Wall motion score index	8.9	3.1–25.8	<0.001
E/A-ratio	3.1	1.4–7.0	0.005
E/E'-ratio	1.07	1.01–1.14	0.03
Moderate or severe mitral regurgitation	3.3	1.5–7.1	0.002
LA max(ml/m^2)	1.05	1.02–1.08	0.004
LA total ejection fraction(%)	0.96	0.93–0.98	0.001
LA active emptying fraction(%)	0.96	0.94–0.99	0.005
LA reservoir function(%)	0.99	0.99–0.99	0.004
LA strain(%)	0.93	0.89–0.97	<0.001
LA strain rate(s^{-1})	0.42	0.23–0.79	0.006

E/A: mitral inflow peak early velocity (E) / mitral inflow peak late velocity (A); E/E': mitral inflow peak early velocity (E) / mitral annular peak early velocity (E'); LA: left atrium; LV: left ventricular.

Discussion

The main findings of the present retrospective evaluation can be summarized as follows: (1) LA reservoir function assessed with LA strain provides useful information in patients with AMI treated with primary percutaneous coronary intervention. (2) LA strain is a promising novel technique to quantify LA function and provides additional value to baseline risk factors and LA maximal volume for the prediction of adverse events after AMI.

Assessment of left atrial function and outcome

In the current evaluation, LA function was assessed using LA volumes, mechanical function and strain. Currently, guidelines recommend measuring LA volume with the ellipsoid model or Simpson's method.[14] Indeed, LA volume has been found to be strongly

related with cardiovascular disease.[23] Several studies have demonstrated that LA volume, measured early after AMI provides prognostic value incremental to known risk factors.[1 2 4] Recently, Meris et al. demonstrated the strong relationship between LA volume and outcome in patients with LV dysfunction or heart failure after AMI.[1] LA indexed volume $\geq 32 ml/m^2$ was independently associated with death or heart failure (HR 2.35, 95%CI 1.28–4.31, p = 0.006).[1] These results were extended in the present evaluation including patients with AMI treated with primary percutaneous coronary intervention and relatively preserved LV function.

Beyond LA size, LA mechanical function may improve the risk stratification. LA function consists of the reservoir period (inflow during ventricular systole), conduit period (passive emptying during ventricular relaxation and diastasis) and contractile period (active emptying). In post-AMI patients, LV remodeling occurs with concomitant effects on the LA. In the present evaluation, comparisons of LA function with matched normal controls demonstrated that besides LA dilatation, also LA total ejection fraction deteriorated post-AMI. Interestingly, assessment of phasic changes of LA volumes demonstrated that LA passive ejection fraction is significantly diminished which is compensated by an increase in active contractile function of the LA. As a result, LV stroke volume can be maintained despite LV dysfunction.[24 25] This phenomenon has been reported previously by Bozkurt et al, who performed serial echocardiography in 73 patients with an anterior AMI at 4 time points (at admission, after 1 week, 1 month and 3 months).[26] The authors demonstrated that remodeling of the LA starts during the first week post-AMI and continues gradually up to 3 months.

Besides active contraction, LA relaxation reflected by the reservoir function is particularly important during acute ischemia.[24] Due to increased LV chamber stiffness and LV filling pressures, LA pressure may be increased.[27] To maintain adequate LV filling, a preserved LA reservoir function is crucial which can withstand the impact of the increased LA pressure. In contrast, in patients with non-compliant LA and reduced reservoir function, LV filling may be significantly impaired increasing the risk of heart failure and death. However, evaluation of LA reservoir function relies on LA volume measurements and is therefore challenging. Measurements may be inaccurate as they depend on geometrical assumptions and are load dependent.[28] In contrast, speckle-tracking is a comprehensive

imaging tool that permits LA reservoir function assessment by direct evaluation of the atrial myocardium and may better reflect intrinsic LA function properties.[8 19 29]

Left atrial strain

Speckle-tracking derived strain has been used extensively to detect subtle LV dysfunction and associated with outcome in different patient populations.[30-32] Recently, several studies have demonstrated that strain measurements are feasible and useful for the detection of changes in LA performance.[20 33 34] Particularly, the assessment of LA reservoir function post-AMI by direct evaluation of LA myocardium deformation may provide clinically relevant information. Peak positive longitudinal strain of the LA reflects the stretch of the wall during the reservoir period. During acute LV ischemia, atrial contraction is initially increased and compensates LV dysfunction. However, with further progression of LV dysfunction and increased LV filling pressures, the LA distensibility becomes more important. Previous studies have demonstrated that the LA reservoir function is determined by the preceding LA contraction, LV contraction through the descent of the base during systole and influenced by the LA chamber stiffness.[27] LA strain reflects all those components by directly evaluating the amount of deformation of the myocardium as reported by previous studies.[9 33 34]

Recently, in 36 patients with systolic heart failure, Cameli et al. reported that LA strain correlated better with pulmonary capillary wedge pressure than the traditional E/E'-ratio.[35] In addition, excellent sensitivity and specificity of 100% and 93% were observed for LA strain <15.1% to predict elevated filling pressures. The strong correlation between LA strain and LV diastolic dysfunction may explain the strong relation observed with adverse outcome in the current study. Although this is the first study to evaluate the prognostic value of LA strain in patients after AMI, several studies have related LA strain to outcome in other patient populations. For example, in patients with atrial fibrillation studies have demonstrated the predictive value of LA strain for maintenance of sinus rhythm after catheter ablation.[20]

Limitations

The cut-off value for LA strain was chosen at 2SDs from the normal LA strain in a group of 35 normal controls, corresponding with 19%. These results may not apply to larger populations. The addition of LA strain to the model including LA volume yielded a significant but modest increase in the global chi-square value. Therefore, the clinical relevance of these measurements needs to be further investigated. In addition, measurement of LA strain may be challenging as demonstrated by the reported feasibility. However, the semi-automated assessment of LA strain is promising and provides a comprehensive assessment of LA function. In addition, improvements of the software may improve the feasibility of the application in clinical practice. Finally, mitral annular velocities were assessed with color-coded tissue Doppler imaging.

Conclusions

The current retrospective evaluation demonstrates that LA strain provides additional prognostic value beyond LA maximal volume in patients with AMI treated with primary percutaneous intervention.

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