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Non Invasive Assessment of Left Ventricular Filling Pressure and Remodeling after Acute Myocardial Infarction

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Abstract

Background: Left ventricular (LV) dilation after acute myocardial infarction (AMI) is an important determinant of prognosis. The ratio of early mitral inflow velocity (E) to peak early diastolic annular velocity (e') provides the best single index for noninvasive detection of acute elevation of LV filling pressure.

Objective: To assess whether E/e' ratio predicts LV remodeling after properly treated AMI compared with traditional clinical, laboratory and echocardiographic data.

Methods: Comprehensive echocardiograms were performed in a series of consecutive patients with first AMI successfully treated with primary percutaneous transluminal angioplasty (PTCA), both 48 hours after intervention and 60 days later. Mean E/e' was determined from four sites of the mitral annulus. LV remodeling was defined as more than 15% increase in end-systolic volume estimated by Simpson method. Statistical analysis included Student's t test, receiver-operator curves (ROC) and multivariate logistic regression (all significant with $p < 0.05$).

Results: Fifty-five patients were included, with mean age 58 ± 11 years, 43 men. The group of patients who underwent LV remodeling (n = 13) had higher baseline E/e' than those without (13 ± 4 versus 8.5 ± 2, $p < 0.001$). The ROC curve showed E/e' > 15 as a predictor of remodeling (AUC = 0.81, $p = 0.001$). In addition, regression analysis (comprising clinical, laboratory and echocardiographic variables along with AMI site) confirmed the independent value of E/e' in the prediction of LV remodeling (odds ratio 1.42, $p = 0.01$).

Conclusion: The E/e' ratio is an useful predictor of LV remodeling after AMI, indicating patients with increased cardiovascular risk (Arq Bras Cardiol. 2013;100(6):531-537).

Keywords: Ventricular Remodeling; Myocardial Infarction; Echocardiography / utilization; Stroke Volume / physiopathology.

Introduction

Dilation of the left ventricle (LV) after acute myocardial infarction (AMI) may occur even after successful transluminal coronary angioplasty (TCA). Previous studies have observed significant LV dilation in approximately one third of patients, despite the patency of the artery affected by the AMI[1,2]. The increase in the left ventricular cavity in response to injury is often accompanied by alterations in geometry and decrease in overall chamber performance, which is conventionally called remodeling. Such LV dilation after AMI is an important determinant of prognosis, increasing the risk of heart failure and sudden death[3].

Several indices derived from Doppler echocardiography have been used to predict outcomes in patients after AMI. The presence of restrictive mitral flow, characterized by shortened deceleration time (DT) of the mitral flow E-wave secondary to increased end diastolic pressure, was associated with increased risk of LV dilation after AMI[4,5]. Elevated filling pressures, as suggested by DT < 130-140 ms, usually indicates larger infarctions, with more pronounced systolic dysfunction and particular predisposition to remodeling[6].

However, the velocities of mitral flow and DT are of limited value in patients with preserved LV systolic function[7]. Fortunately, the desirable early reperfusion obtained by recent advances in TCA techniques has resulted in better clinical outcomes and better recovery of left ventricular function[8]. On the other hand, the ratio between the early diastolic mitral flow velocity at conventional pulsed Doppler (E) and early diastolic mitral annular velocity at tissue Doppler (e'), known as the E/e', showed a strong correlation with invasively determined LV filling pressure[7], even in subjects with preserved ejection fraction (EF).

Our hypothesis was that an increased E/e' ratio (noninvasive marker of elevated LV filling pressure) is associated with higher incidence of LV dilation in patients with successful reperfusion after AMI. Therefore, the aim

Barberato et al.
Left ventricular remodeling after myocardial infarction

Original Article

of this study was to determine whether the E/e' ratio predicts infarcted LV remodeling and adds information to the traditional clinical, laboratory and echocardiographic approach.

Methods

Study population

Consecutive patients admitted to the coronary care unit of our hospital with a diagnosis of first AMI were evaluated for study enrollment. The diagnosis of AMI was defined by the recommendations of the European American College of Cardiology/ American Heart Association[9]. Inclusion criteria were: 1) coronary angiography after TCA showing patent artery related to the AMI, with TIMI flow grade III[10]; 2) echocardiogram showing wall akinesia related to compromised arterial site. Exclusion criteria were: 1) need for hemodynamic support with intra-aortic balloon, 2) sustained arrhythmia, precluding measurement of Doppler echocardiographic indices, 3) inadequate thoracic acoustic window; 4) refusal to participate in the study.

The study was approved by the ethics committee and patients were included in the study after signing the free and informed consent form.

Echocardiography

Complete echocardiographic assessment was carried out in a consecutive series of patients with first AMI at two times: 48 hours after TCA with successful recanalization of the culprit vessel and approximately 60 days after AMI. At our institution, all patients are directly treated with primary TCA and not thrombolysis. Therefore, the value of E/e' for the studied outcome was tested under these circumstances for all study subjects. Echocardiograms were performed by the same examiner using a Philips IE33 echocardiograph (Philips Medical Systems, USA) equipped with a 2.5-4 MHz transducer. The usual slices were made to allow a detailed study by M-mode, two-dimensional and Doppler techniques (pulsed, continuous, color and tissue).

Consistent with the recommendations of the American Society of Echocardiography (ASE)[11], the following parameters were determined: LV interventricular septal thickness and inferolateral wall in diastole, LV systolic and diastolic dimensions. The score of parietal motility (SPM) was calculated taking into account the standard model of 16 segments and graduated in a four-point scale: 1 = normal, 2 = hypokinetic, 3 = akinetic, 4 = dyskinetic. LV mass was calculated using the formula of the ASE and indexed by body surface area (DuBois and DuBois method). The volumes and ejection fraction (EF) were calculated using the biplane Simpson method. LV remodeling was defined prospectively as an increase $\geq 15\%$ in end-systolic volume[12].

The mitral flow velocities were measured by pulsed Doppler in the apical four-chamber view, with the sample volume positioned between the tips of the mitral valve leaflets, and patients were instructed to hold their breath when possible. We determined early diastolic (E) and atrial contraction (A) velocities as well as the E / A ratio and the DT.

The early diastolic annular (e') and atrial (a') velocities were recorded by tissue Doppler in the apical four and two-chamber view, with a sample volume of 1-2 mm placed at the junction of the LV wall with four sites of the mitral annulus (septal, lateral, anterior and inferior). The mean of the velocities at the four sites represented the e' and a' waves used for analysis in this study, as well as the E/e' ratio. All measurements represent the mean of three cardiac cycles.

LV diastolic function was graded according to the combined interpretation of the indices derived from conventional pulsed Doppler of mitral flow and tissue Doppler. Abnormal relaxation was diagnosed with the presence of E / A ratio < 0.9 and e' <10 cm /s; restrictive flow was determined with the E / A > 2, DT < 140 ms and e' < 8 cm/s[13]. When differentiating between normal and pseudo-normal pattern, the presence of e' < 8 cm and E/e' $\geq$ 15 was used[13]. The left atrial volume index (LAVi) was measured using the biplane Simpson's technique followed by body surface indexation.

Basal clinical data and follow-up

Demographic and clinical data were obtained by detailed chart review. Data such as age, gender, weight, height, body mass index (BMI), history of diabetes, hypertension, dyslipidemia, smoking, drug use and prior TCA were recorded. The time from symptom onset to recanalization of the culprit vessel, location (compromised LV wall), Killip classification, blood pressure on admission, peak creatine kinase MB isoenzyme (CK-MB) and troponin levels were also recorded. During and after completion of the TCA, all patients were treated with the recommended doses of aspirin, clopidogrel, unfractionated heparin and abciximab[9]. Drug treatment after the event was conducted by the patient's physician, recommending the administration of angiotensin-converting enzyme inhibitors (or angiotensin receptor blockers II), beta-blockers and diuretics, according to current guidelines[9]. The primary endpoint was LV remodeling 60 days after the AMI.

Statistical Analysis

The estimated sample size was calculated as 51 patients, considering an incidence of remodeling of approximately 30%[1], statistical power of 90% and a significance level of 5%. Data are shown as mean and standard deviation (continuous variables) and percentages (categorical variables). The differences between the groups with and without remodeling were determined by Student's *t* test (continuous variables with normal distribution), Mann-Whitney test (continuous variables with non-normal distribution) and Chi-square (categorical variables).

The correlations between the Doppler echocardiographic indices and alterations in end-systolic volume were analyzed using Pearson's coefficient. Receiver-operator curve (ROC) was constructed to determine E/e' as a predictor of remodeling, as well as sensitivity and specificity. Finally, multivariate logistic regression analysis was used to identify the independent value of E/e' regarding several traditional variables. Statistical significance was defined as p < 0.05. Analyses were processed using the statistical program SPSS 13.0 for Windows (SPSS Inc., Chicago, Illinois).

Original Article

Results

The study included 55 patients aged 58 ± 11 years, 43 men, with remodeling being detected (group I) in 13 individuals (24%), which were compared with the group without remodeling (Group II), consisting of 42 individuals.

The demographic, clinical and laboratory characteristics of the study population (and its subgroups) are shown in Table 1. The group I had the highest percentage of prior history of hypertension (60% *versus* 38%, p = 0.02) and higher peak troponin level (20 ± 12 *vs.* 7 ± 8 ng / mL, p = 0.005) compared to group II. There was also a tendency to higher CK-MB peak (p = 0.06) and time between symptom and vessel recanalization (p = 0.08) in group I. There were no statistically significant differences between groups regarding age, gender, BMI, diabetes, dyslipidemia, smoking, prior use of angiotensin-converting enzyme inhibitors/angiotensin receptor blockers II, prior TCA, infarction located in the anterior wall, Killip II on admission, blood pressure on admission and serum levels of hemoglobin, glucose and creatinine (all with p > 0.05).

Table 2 shows the major Doppler echocardiographic characteristics of the study sample studied. Group I had higher LV systolic dimension (p = 0.02), lower EF (p = 0.01), higher

SPM (p < 0.001) and lower e' (p = 0.02) and a' (p = 0.03), as well as higher E / e' (p < 0.001). There were no differences in LV diastolic dimension, LVMI, E/ A ratio, DT and LAVi (all with p > 0.05) between the groups.

There was a correlation between the variation in end-systolic volume after 60 days (r = 0.26, p = 0.03) and E/e', but not with the other variables. The ROC curve (Figure 1) indicated E/e' as a predictor of remodeling (area under the curve = 0.81, 95% confidence interval: 0.68 - 0.94, p = 0.001). The cutoff value for E/ e' > 15 showed a sensitivity of 70% and specificity of 98% for an increase of 15% in the LV end-systolic volume.

Multivariate regression analyses containing clinical (history of hypertension, duration of symptoms), laboratory (troponin and CK-MB levels) and Doppler echocardiographic variables (systolic dimension of LV, EF, SPM, a', E/e' ratio and location of AMI) confirmed E/e' as the sole independent predictor of remodeling (odds ratio: 1.42; 95% confidence interval: 1.1 - 1.9, p = 0.01), above and beyond all other parameters. Speculative analyses, forcing the input of other traditionally used variables into the model, such as age, gender, DT and location of the AMI in the anterior wall, did not change the independent predictive value of E/e' ratio.

Table 1 – Main demographic, clinical and laboratory data of the study population and its subgroups: group I, with LV remodeling and group II with no LV remodeling

Variable	Total (n = 55)	Group I (n = 13)	Group II (n = 42)	p
Age (years)	58 ± 11	59 ± 13	58± 11	0.8
Male gender (%)	78 (43)	77	78	0.9
BMI (kg/m²)	27 ± 4	28 ± 4	27 ± 4	0.6
SAH (%)	47 (26)	69	38	0.02
DM (%)	20	15	21	0.7
DL (%)	49	61	45	0.2
Smoking (%)	36	38	36	0.7
ACEI/ARB (%)	84	100	72	0.8
Previous PTA (%)	7	15	5	0.2
AMI anterior (%)	53	69	48	0.2
Time (hrs)	5 ± 6	7 ± 8	4.5 ± 6	0.08*
Killip II (%)	13	30	9.5	0.2
SBP (mmHg)	126 ± 21	131 ± 25	125 ± 20	0.5
DBP (mmHg)	75 ± 14	76 ± 19	75 ± 12	1.0
HR (bat./min)	74 ± 13	70 ± 18	75 ± 12	0.5
CK-MB (U/L)	128±118	194 ± 136	99 ± 103	0.06*
Troponin (ng/ml)	10 ± 11	20 ± 12	7 ± 8	0.005*
Hemoglobin (g/dl)	13 ± 2	13 ± 2	13 ± 2	0.8
Glucose (mg/dl)	126 ± 50	129 ± 29	126 ± 55	0.9
Creatinine (mg/dl)	1.07 ± 0.2	1.06 ± 0.2	1.07 ± 0.2	0.9

*BMI: body mass index, SAH: systemic arterial hypertension, DM: diabetes mellitus, DL: dyslipidemia; ACEI /ARB: angiotensin converting enzyme inhibitors / angiotensin receptor blockers; PTA: percutaneous transluminal angioplasty; AMI: acute myocardial infarction; SBP: systolic blood pressure, DBP: diastolic blood pressure, HR: heart rate, CK-MB: creatine kinase isoenzyme, myocardial fraction. * Mann-Whitney test was used. Other continuous variables were compared by Student's t test.*

Table 2 – Main Doppler-echocardiographic characteristics of the study population and its subgroups: group I, with LV remodeling and group II with no LV remodeling

Variable	Total (n = 55)	Group I (n = 13)	Group II (n = 42)	p
LVEDD (mm)	50 ± 4	51 ± 5	50 ± 4	0.2
LVESD (mm)	34 ± 7	38 ± 6	32 ± 7	0.02
LVEDV (mm)	90 ± 29	84 ± 19	91 ± 31	0.4
LVESV (mm)	41 ± 17	44 ± 16	40 ± 18	0.5
Percentage of EF	55 ± 12	47 ± 11	56 ± 12	0.01
SPM	1.3 ± 0.2	1.51 ± 0.2	1.27 ± 0.2	< 0.001
LVMi (g/m²)	101 ± 17	108 ± 18	99 ± 17	0.08
LAVi (ml/m²)	25 ± 5	26 ± 5	24 ± 5	0.5
E (cm/s)	77 ± 19	84 ± 19	75 ± 18	0.1
A (cm/s)	70 ± 24	69 ± 27	70 ± 23	0.9
E/A	1.2 ± 0.5	1.4 ± 0.6	1.2 ± 0.4	0.1
TD (ms)	186 ± 31	172 ± 21	190 ± 33	0.2
e' (cm/s)	8.1 ± 2	6.9 ± 2	8.5 ± 2	0.02
a' (cm/s)	8.9 ± 2	7.7 ± 3	9.4 ± 2	0.03
E/e'	10 ± 3	13 ± 4	8.5 ± 2	< 0.001

LVEDD: left ventricular end-diastolic dimension; LVESD: left ventricular end-systolic dimension; LVEDV: LV end-diastolic volume; LVESV: LV end-systolic volume; EF: ejection fraction; SPM: score of parietal motility; LVMi: left ventricular mass index, LAVi = left atrial volume index; E: early diastolic velocity of mitral flow; A: atrial mitral flow velocity; DT: deceleration time, e ': early diastolic annular velocity, a ': atrial contraction annular velocity.
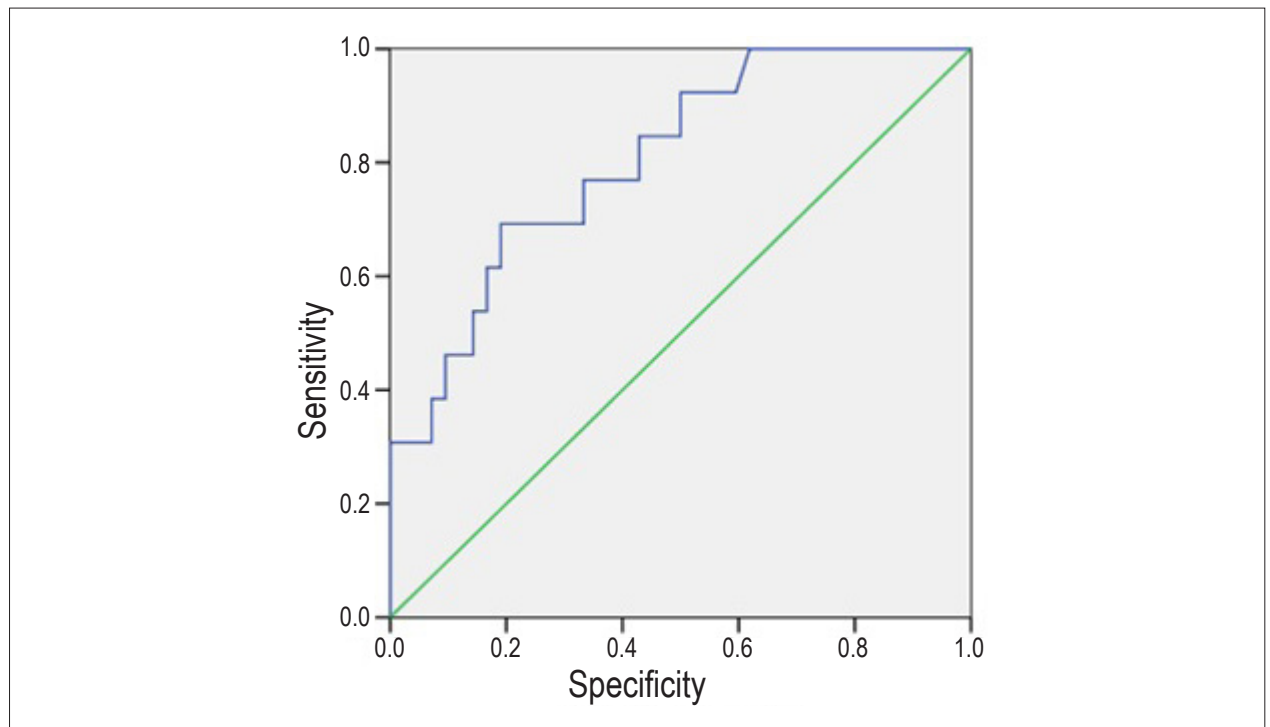

Figure 1 – *Receiver-operator curve of the E/e' ratio in the prediction of left ventricular remodeling after treated acute myocardial infarction.*

Original Article

Discussion

The main finding of this study was that the E/e' ratio seems to be an independent predictor of left ventricular remodeling after AMI successfully treated with reperfusion by TCA. In particular, echocardiographic evidence of elevated LV filling pressures, provided by E/e' > 15, was strongly associated with LV dilation, with a higher value than the previously established clinical, laboratory and echocardiographic parameters. Considerable body of evidence demonstrated that the presence of restrictive mitral flow (shortened DT) is a strong predictor of prognosis after myocardial infarction, as it is generally associated with greater area of LV wall akinesia and increased end diastolic pressure[4-6].

However, the DT has known limitations in patients with preserved systolic function[7], a situation increasingly common after the advances obtained with prompt percutaneous intervention therapy in acute myocardial infarction. In parallel, it was demonstrated that the E/e' ratio was the best indicator of the presence of elevated end-diastolic pressure in the comparison between multiple echocardiographic parameters and the pressure measured by hemodynamic catheterization[7,14].

The present study indicates that the E/e' ratio can be better than the DT to predict remodeling when evaluating a patient population with relatively preserved EF (overall sample with a mean percentage of EF = 55 ± 12). Another interesting finding was that the E/e' ratio, representing the acute effects of elevated LV filling pressures, showed a batter performance when predicting remodeling after AMI than the LAVi, an index traditionally associated with chronic elevation of filling pressures. Although LAVi is a clear predictor of mortality after AMI[15], its impact on remodeling was not observed in our study.

It is possible that patients with chronic LAVi increase have suffered, prior to the AMI, some degree of left ventricular remodeling, thus mitigating the effects generated by the latest ischemic injury. Therefore, the impact of increased LAVi on LV remodeling would not be apparent in a relatively small cohort with short follow-up, such as ours. Alternatively, one may speculate that the association between LV remodeling and the acute increase in filling pressures is more important than between remodeling and the chronic increase.

In the context of myocardial ischemia and infarction, tissue Doppler (from which the e' velocity derives and, ultimately, the E/e' ratio) shows low systolic and diastolic velocities in the mitral annulus[16]. These velocities decrease with regional hypoperfusion, recover with reperfusion and differentiate between transmural and non-transmural infarction[17]. After the AMI, the acute effects of ischemia and necrosis cause loss of myocyte integrity and LV geometry disarray, leading to a decrease in the overall performance and increase in LV end-diastolic pressure. LV dilation/remodeling emerge from this process as a compensatory mechanism in an attempt to restore the filling pressure to the normal level (or near normal). This might explain why a surrogate marker of the acute measure of LV filling pressure works best when predicting remodeling.

In addition to estimating cardiac function and filling pressures, some authors investigated the prognostic value of E/e' in AMI. In 250 patients followed for a median period of 13 months after AMI, Hillis et al[18] showed that E/e ' > 15 was a strong predictor of poor survival, with an incremental value to the traditional clinical parameters and echocardiographic indices of systolic and diastolic function. LV remodeling certainly plays an important role in the physiopathology of the phenomena involved in the decreased survival in this group. These researchers published a study, following the same investigation line as ours, showing that E/e' > 15 identified individuals with increased risk of developing LV dilation after AMI[19].

However, there are several methodological differences between this report and the present study that should be highlighted. First, the population studied by Hillis et al[18] consisted of 47 individuals, of whom only 22 patients (47%) were treated with primary TCA, whereas 25 (53%) were treated with thrombolysis[19]. In contrast, all patients in our group underwent percutaneous intervention, which is known as having better outcomes[8].

Furthermore, the criteria to define LV remodeling were dissimilar: ≥ 15% increase in LV end-systolic volume in our study, in contrast with ≥ 15% increase in end-diastolic volume in the aforementioned article. There is no consensus in the literature whether the variation in LV systolic volume or end-diastolic volume should be used in the diagnosis of remodeling and other authors have also used our choice as reference[12].

The choice of employing the variation in end-systolic volume was due to prior evidence that this parameter is superior to the end-diastolic volume and even to ejection fraction itself as the primary predictor of prognosis after AMI[12]. Finally, it is important to emphasize that, in the study by Hillis et al[18], the E/e' ratio was obtained by measuring the e' velocity exclusively on the septal mitral annulus, while our study used the mean of the four sides of the mitral annulus (septal, lateral, anterior and inferior). This conduct is vital to reduce discrepancies in the presence of regional myocardial dysfunction[13].

Our approach has some limitations. It is known that the tissue Doppler velocities are affected by translation, traction and tethering of adjacent myocardial segments[20] and, therefore, it is highly dependent on the angle. New imaging methods, such as speckle-tracking echocardiography[21] and magnetic resonance imaging[22], which do not have this limitation, have shown to be extraordinary significant to predict remodeling after AMI. However, its clinical applicability is still limited for several reasons, including availability, which makes the E/e' ratio the best choice in terms of cost-benefit at the moment. Another limitation was the fact that all assessments were performed by a single examiner, precluding the measurement of interobserver variability.

Thus, the use of the E/e' ratio, a noninvasive marker of elevated LV filling pressures, may be a useful predictor

of LV dilation/ remodeling scenario after AMI, indicating patients with increased cardiovascular risk and allowing early intervention.

Author contributions

Conception and design of the research, Analysis and interpretation of the data, Statistical analysis, Writing of the manuscript, Critical revision of the manuscript for intellectual content: Barberato SH; Acquisition of data: Barberato SH, Souza AM; Obtaining funding: Costantini CO, Costantini CR.

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