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Left Atrial Volume Index and Prediction of Events in Acute Coronary Syndrome: Solar Registry

Jose Alves Secundo Junior[1], Marcos Antonio Almeida Santos[1,2], Gustavo Baptista de Almeida Faro[1], Camile Bittencourt Soares[1], Allysson Matos Porto Silva[1], Paulo Fernando Carvalho Secundo[1], Clarissa Karine Cardoso Teixeira[1], Joselina Luzia Menezes Oliveira[1,2], Jose Augusto Soares Barreto Filho[1,2], Antônio Carlos Sobral Sousa[1,2]

Universidade Federal de Sergipe[1]; Centro de Ensino e Pesquisa e Laboratório de Ecocardiografia (ECOLAB) do Hospital e Fundação São Lucas[2], Aracaju, SE - Brazil

Abstract

Background: According to some international studies, patients with acute coronary syndrome (ACS) and increased left atrial volume index (LAVI) have worse long-term prognosis. However, national Brazilian studies confirming this prediction are still lacking.

Objective: To evaluate LAVI as a predictor of major cardiovascular events (MCE) in patients with ACS during a 365-day follow-up.

Methods: Prospective cohort of 171 patients diagnosed with ACS whose LAVI was calculated within 48 hours after hospital admission. According to LAVI, two groups were categorized: normal LAVI ($\leq$ 32 mL/m²) and increased LAVI (> 32 mL/m²). Both groups were compared regarding clinical and echocardiographic characteristics, in- and out-of-hospital outcomes, and occurrence of ECM in up to 365 days.

Results: Increased LAVI was observed in 78 patients (45%), and was associated with older age, higher body mass index, hypertension, history of myocardial infarction and previous angioplasty, and lower creatinine clearance and ejection fraction. During hospitalization, acute pulmonary edema was more frequent in patients with increased LAVI (14.1% vs. 4.3%, p = 0.024). After discharge, the occurrence of combined outcome for MCE was higher (p = 0.001) in the group with increased LAVI (26%) as compared to the normal LAVI group (7%) [RR (95% CI) = 3.46 (1.54-7.73) vs. 0.80 (0.69-0.92)]. After Cox regression, increased LAVI increased the probability of MCE (HR = 3.08, 95% CI = 1.28-7.40, p = 0.012).

Conclusion: Increased LAVI is an important predictor of MCE in a one-year follow-up. (Arq Bras Cardiol. 2014; 103(4):282-291)

Keywords: Acute Coronary Syndrome; Cardiac Volume; Heart Atria.

Introduction

Acute coronary syndrome (ACS) is one of the major causes of cardiovascular mortality in the modern world, left ventricular function being an important prognostic marker for patients with that syndrome[1]. Patients with ACS and clinical signs of congestive heart failure have worse prognosis, and, in general, should be referred to an interventional cardiology service to undergo reperfusion. Asymptomatic individuals with evidence of subclinical dysfunction also have worse prognosis[2,3]. In addition, it is speculated that proper treatment might benefit that subgroup of patients. Thus, the development and identification of early markers of left ventricular dysfunction are required to select ideal candidates for invasive therapy[2,3].

Left ventricular diastolic function assessed by use of Doppler echocardiography, complementary to systolic function, provides important prognostic information, after acute myocardial infarction (AMI). Left atrial volume index (LAVI) is less sensitive to acute variations, reflecting subacute or chronic changes in diastolic function[4-8]. Individuals with increased LAVI, i.e., with significant diastolic dysfunction, are at high risk for complications and could benefit from choosing the most appropriate treatment[9-11].

Several clinical studies have reported the usefulness of left atrial volume (LAV) in the prognosis of several diseases[12-18]. Regarding ACS, almost all studies have assessed the prognostic influence of LAVI only on AMI, without approaching its importance in unstable angina (UA)[19-21]. However, despite those international studies, we have not succeeded in identifying studies on that specific topic in Brazil. It is worth noting that study findings are not always reproducible for different sociodemographic and anthropometric contexts or even for different health care conditions. Nevertheless,

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a study should be reproducible so that its results could be more widely validated.

Therefore, the major objective of this study is to assess the role of LAVI as a predictor of late cardiovascular events in patients with ACS followed up for one year at a referral hospital for cardiology in Brazil.

Methods

Study population

This is a prospective cohort of the "Solar Registry" (Acute Coronary Syndrome Registry of the Hospital São Lucas), carried out at Hospital São Lucas, a referral center for cardiology in the state of Sergipe, Brazil, which meets accreditation standards (level 3) (IQG - *Instituto Qualisa de Gestão*).

From May 2008 to April 2012, individuals of both sexes admitted to the chest pain unit with ACS findings were selected for investigation and treatment at that hospital. All patients originated from the center for supplementary health services.

It was a non-random convenience sample consecutively obtained.

Inclusion and exclusion criteria

In the period studied, 519 patients had UA or AMI, the latter subdivided into ST-segment elevation AMI and non-ST-segment elevation AMI. Those three categories were defined according to clinical history (symptoms consistent with acute ischemia) or gradual increase in heart necrosis markers (in the case of AMI), in addition to at least one of the following tests: electrocardiography; transthoracic Doppler echocardiography; and coronary angiography[22]. Of the initial population, only 171 patients had their LAVI measured by use of echocardiography, being included in the study. The other patients were excluded due to either lack of that measurement, or presence of a malignancy or severe valvar heart disease.

Within the first 48 hours from hospital admission, all patients included in this study underwent transthoracic Doppler echocardiography at rest. The patients were distributed into two groups according to LAVI values, determined by Simpson's method, as follows: 1) normal LAVI ($\leq$ 32 mL/m^2); and 2) increased LAVI ($>$ 32 mL/m^2)[23].

Materials

To determine the clinical and laboratory profile and the in-hospital outcome of patients with ACS, a standardized assessment was performed by the examiner and confirmed with data from the medical records. The groups were compared based on the following parameters: a) baseline clinical characteristics; b) creatinine clearance on admission; c) comorbidities, such as systemic arterial hypertension (SAH), diabetes mellitus, dyslipidemia, previous cardiovascular disease; d) diagnosis; e) echocardiographic data; f) treatment in the acute phase (only clinical treatment, angioplasty or

myocardial revascularization); and g) in-hospital outcome regarding cardiovascular events (acute pulmonary edema, atrial fibrillation, shock, re-infarction, cardiorespiratory arrest, or death).

Patients were classified as smokers, when reporting current smoking habit, or ex-smokers, when reporting quitting smoking for at least one year. Diabetic patients were those with a previous diagnosis of diabetes mellitus and/or who were on oral anti-diabetic agents or whose fasting glycemia was $>$ 126 mg/dL during hospitalization. Systemic arterial hypertension was considered in those who had been diagnosed prior to hospitalization and/or were on antihypertensive agents or who had systolic blood pressure $\geq$ 140 mmHg and/or diastolic blood pressure $\geq$ 90 mmHg. Dyslipidemia was determined by the presence of high serum levels of LDL-C ($>$ 130 mg/dL) and/or low serum levels of HDL-C ($<$ 40 mg/dL) and/or increased serum levels of triglycerides ($>$ 150 mg/dL). Patients with body mass index (BMI) $>$ 25 kg/m^2 were considered overweight[24]. Renal function was estimated based on glomerular filtration rate by using the Cockcroft & Gault formula: (140 - age in years) x body weight (kg) / 72 x creatinine (mg/dL), corrected for the 0.85 factor for women[25-27].

Acute pulmonary edema was defined as the presence of clinical signs of left ventricular failure, dyspnea, and signs of hypoxia and of fluid in the lungs (rales on pulmonary auscultation, and bilateral pulmonary infiltrates on chest radiography consistent with congestion)[28]. Stroke was defined as the fast development of clinical signs of focal or global cerebral function disorder for more than 24 hours, with no other apparent cause than vascular origin[29]. Reinfarction was defined as the presence of ischemic symptoms, new electrocardiographic changes and/or subsequent elevation in heart necrosis markers after a decrease[30]. Cardiogenic shock was defined as the presence of arterial hypotension (systolic blood pressure $<$ 90 mmHg or 30 mmHg below the baseline value) and evidence of tissue hypoperfusion, such as oliguria, cyanosis, cold extremities, changes in the levels of consciousness, pulmonary capillary pressure $>$ 18 mmHg, cardiac index $<$ 1.8 L/min/m^2, systemic vascular resistance index $>$ 2000 dyn.s/cm^5/m^2, and increased arteriovenous oxygen difference $>$ 5.5 mL/dL[31-32].

Follow-up

The patients with ACS assessed in this study were followed up for up to 365 days after hospital admission, by use of medical record investigation or telephone contact with them or a close relative. After hospital discharge, they were assessed regarding the appearance of any major cardiovascular event, such as stroke, AMI or death due to any cause. For the statistical analysis purpose, only the first event occurring after hospital discharge was considered.

Data analysis

The quantitative variables were described as mean and standard deviation. The Kolmogorov-Smirnov test was used to assess the assumption of sample normality. Then, Student *t* test or Mann-Whitney test was used for independent groups,

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according to the sample normality pattern. Regarding the categorical variables, absolute frequency and percentage were used. To compare the characteristics of the categorical variables between the two groups, the chi-square test or Fisher exact test, when more appropriate, was used.

Survival was assessed by use of the Kaplan-Meier curve and the differences between the two groups were quantified with the log-rank test. To assess the influence of in-hospital events, a landmark analysis was performed excluding that period. In addition, the relative risk (RR) was calculated for the combined out-of-hospital outcome with a maximum follow-up of one year.

Cox proportional hazards regression was performed according to the Breslow method with adjustments to the following variables: age; sex; type of ACS; diabetes mellitus; dyslipidemia; SAH; ejection fraction; and LAVI. Hazard ratio (HR) values and respective 95% confidence intervals (95% CI) were provided. The assumptions of hazards proportionality were tested by calculating the Schoenfeld residuals and by analyzing the coefficients.

The Stata software, version 13.0, was used for statistical analysis, and the significance level adopted for all tests was two-tailed $p < 0.05$.

Ethical aspects

A questionnaire was applied to the patients after they were informed about the study's objective and after they provided written informed consent. This study is part of the "Solar Registry", which had already been approved by the local ethics committee.

Results

Baseline characteristics of the study population

During the period assessed, 171 patients (mean age, 66 ± 13 years; 73 women) were included in this study. The LAVI was > 32 mL/m^2 in 78 patients (45%). Comparing with individuals with LAVI ≤ 32 mL/m^2, those patients were older, had higher BMI and higher prevalence of SAH, history of AMI and previous angioplasty. In addition, they had a lower estimated creatinine clearance. No significant difference was observed regarding the following characteristics: sex; and history of smoking, diabetes mellitus, dyslipidemia, previous stroke, previous myocardial revascularization, and type of ACS on hospital admission (Table 1).

Echocardiographic characteristics

LAVI > 32 mL/m^2 is significantly associated with lower left ventricular ejection fraction (0.56 vs. 0.61; $p = 0.021$) and with the restrictive pattern, which is the most severe manifestation of diastolic dysfunction, obtained by use of the mitral flow ($p = 0.004$). However, higher frequency of mild diastolic dysfunction was observed in the group with normal LAVI. The presence of artificial pacemaker accounted for the occurrence of indeterminate pattern in the sample (Table 2).

Treatment and hospital outcome

The groups did not differ statistically regarding the treatment used (only clinical treatment, percutaneous intervention or surgical myocardial revascularization). Regarding the in-hospital complications, acute pulmonary edema was more frequent in patients with LAVI > 32 mL/m^2 [11/78 (14.1%) vs. 4/93 (4.3%); $p = 0.024$]. No difference was observed regarding the occurrence of atrial fibrillation, shock, reinfarction, cardiorespiratory arrest and death (Table 3).

Population follow-up

During the out-of-hospital follow-up (up to 365 days from hospital admission), the occurrence of the first major cardiovascular event was higher in individuals with LAVI > 32 mL/m^2. In that group, stroke, AMI and death were observed in 10, 8 and 2 patients, respectively. Conversely, in the group with normal LAVI, those events were observed in only 1, 5 and 1 patients, respectively. Thus, the comparison between groups regarding combined out-of-hospital outcomes, by using the log-rank test, shows a significantly higher frequency in those with increased LAVI [20/76 (26%) vs. 7/92 (7%), $p = 0.001$], as shown in Figure 1.

Still considering the combined outcome, the Kaplan-Meier curve clearly showed ($p < 0.001$, log-rank test) that the group with increased LAVI underwent unfavorable change 200 days after hospital admission. The higher occurrence of stroke accounted for that finding (Figure 2).

Figure 3 shows the increase in the RR for combined outcome in 365 days in patients with increased LAVI (RR = 3.459; 95% CI = 1.54-7.73) as compared with the group of patients with normal LAVI (RR = 0.798; 95% CI = 0.69-0.92).

Several Cox proportional hazards regression models were used, always with significant "p" values for the variable LAVI. In the complete model comprising eight variables (Table 4), LAVI increased by more than 200% the probability of outcomes (HR = 3.08; 95% CI = 1.28- 7.40; $p = 0.012$).

In addition, similar results were obtained in the landmark analysis performed in association with Cox regression, adjusted for the same predictors, and excluding in-hospital outcomes. The assessment of the assumptions of hazards proportionality for covariables was considered satisfactory, with p values > 0.05 for all items (p value for the global test = 0.70). Similarly, when plotting Schoenfeld residuals, the horizontal curve showed the high properness of the model.

Figure 4 shows superposition of the Kaplan-Meier curves and the adjusted Cox regression curves, confirming the predictive capacity of the LAVI for outcomes.

Discussion

This prospective study confirmed that increased LAVI, obtained in the first 48 hours from admission of patients with ACS, is an important predictor of major cardiovascular events. Patients with LAVI > 32 mL/m^2 had worse prognosis regarding in-hospital outcome. In addition, they had a higher number of major cardiovascular events in the 365-day follow-up, in which the occurrence of stroke was important.

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Table 1 – Clinical characteristics of patients with acute coronary syndrome according to left atrial volume index

	LAVI ≤ 32 mL/m² (n = 93)	LAVI > 32 mL/m² (n = 78)	p
Age, years (mean ± SD)	63 ± 13	70 ± 13	0.001 *
Sex, female	35 (37.6)	38 (48.7)	0.14
BMI (kg/m²)	26.8 ± 4.2	28.5 ± 5.4	< 0.001 †
Creatinine clearance ‡	81 ± 29	68 ± 34	0.004 *
Comorbidities			
Smoking §	44 (47.8)	33 (42.3)	0.31
Diabetes mellitus	34 (36.5)	38 (48.8)	0.15
Dyslipidemia	60 (64.5)	59 (75.6)	0.11
SAH	63 (67.7)	68 (87.2)	0.003
Previous AMI	18 (19.4)	29 (37.2)	0.009
Previous stroke	8 (8.6)	9 (11.8)	0.48
Previous angioplasty	20 (21.5)	34 (43.6)	0.002
Previous MR	12 (12.9)	15 (19.2)	0.25
ACS type			
UA	24 (25.8)	26 (33.3)	
NSTEMI	46 (49.5)	40 (51.3)	0.26
STEMI	23 (24.7)	12 (15.4)	

Data shown as numbers (%) or mean and standard deviation (SD). ACS: acute coronary syndrome; BMI: body mass index; SAH: systemic arterial hypertension; LAVI: left atrial volume index; AMI: acute myocardial infarction; MR: myocardial revascularization; UA: unstable angina; NSTEMI: non-ST elevation acute myocardial infarction; STEMI: ST elevation acute myocardial infarction; (*) Mann-Whitney test; (†) Student t test; (‡) value estimated with the Cockroft & Gault formula and corrected for body mass index (mL/min/1.73m²); (§) Smoker or ex-smoker.

Table 2 – Echocardiographic characteristics of patients with acute coronary syndrome according to left atrial volume index

	LAVI ≤ 32 mL/m² (n = 93)	LAVI > 32 mL/m² (n = 78)	p
LA size	3.6 ± 0.3	4.2 ± 0.5	< 0.001*
EF †	0.61 ± 0.1	0.56 ± 0.1	0.021‡
Mitral flow			
Normal	2 (2.2)	5 (6.4)	
Relaxation deficit	63 (67.7)	37 (47.4)	
Pseudonormal	24 (25.8)	19 (24.4)	0.004
Restrictive	2 (2.2)	11 (14.1)	
Indeterminate	2 (2.2) §	6 (7.6) //	

Data shown as numbers (%) or mean and standard deviation. LA: left atrial; EF: ejection fraction; LAVI: left atrial volume index. (*) Student t test; (‡) Mann-Whitney test; (†) data lost in the group with LAVI >32 mL/m²; (§) one due to impaired analysis and one due to atrial fibrillation; (//) four due to pacemaker and two due to impaired analysis.

Similarly to this study, Beinart et al[15] have prospectively assessed, in a five-year follow-up, 395 patients, correlating increased LAVI with mortality. By comparing clinical characteristics and previous history, those authors have reported that patients with increased LAVI showed the following significant differences: more advanced age and a higher frequency of history of smoking, SAH and stroke. The present study showed a significant difference regarding higher BMI and history of previous AMI and angioplasty, in addition to confirming the more advanced age and higher prevalence of SAH in patients with increased LAVI. However, none of those studies could differentiate between the groups with normal and increased LAVI regarding the presentation form of ACS.

Similarly to this study, Beinart et al[15] have found no significant difference for the hospital treatment between those two groups; however, regarding complications during hospitalization, those authors have shown a higher frequency of congestive heart failure in patients with increased LAVI. Nevertheless, this study did not assess that outcome because of difficulty in standardizing the diagnostic method. It is worth

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Table 3 – In-hospital treatment and outcome of patients with acute coronary syndrome according to left atrial volume index

	LAVI ≤ 32 mL/m² (n = 93)	LAVI > 32 mL/m² (n = 78)	p
In-hospital treatment			
Only clinical treatment	42 (45.2)	40 (51.3)	0.42
Angioplasty	47 (50.5)	36 (46.2)	0.56
MR	4 (4.3)	2 (2.6)	0.68 *
Complications			
APE	4 (4.3)	11 (14.1)	0.024
Atrial fibrillation	2 (2.2)	6 (7.7)	0.14 *
Cardiogenic shock	1 (1.1)	4 (5.1)	0.17 *
Re-infarction	7 (7.5)	11 (14.1)	0.16
CRA	3 (3.2)	1 (1.3)	0.62 *
Death	1 (1.1)	2 (2.6)	0.59 *

Data shown as numbers (%) or mean and standard deviation. MR: myocardial revascularization; APE: acute pulmonary edema; CRA: cardiorespiratory arrest; LAVI: left atrial volume index. (*) Fisher exact test.
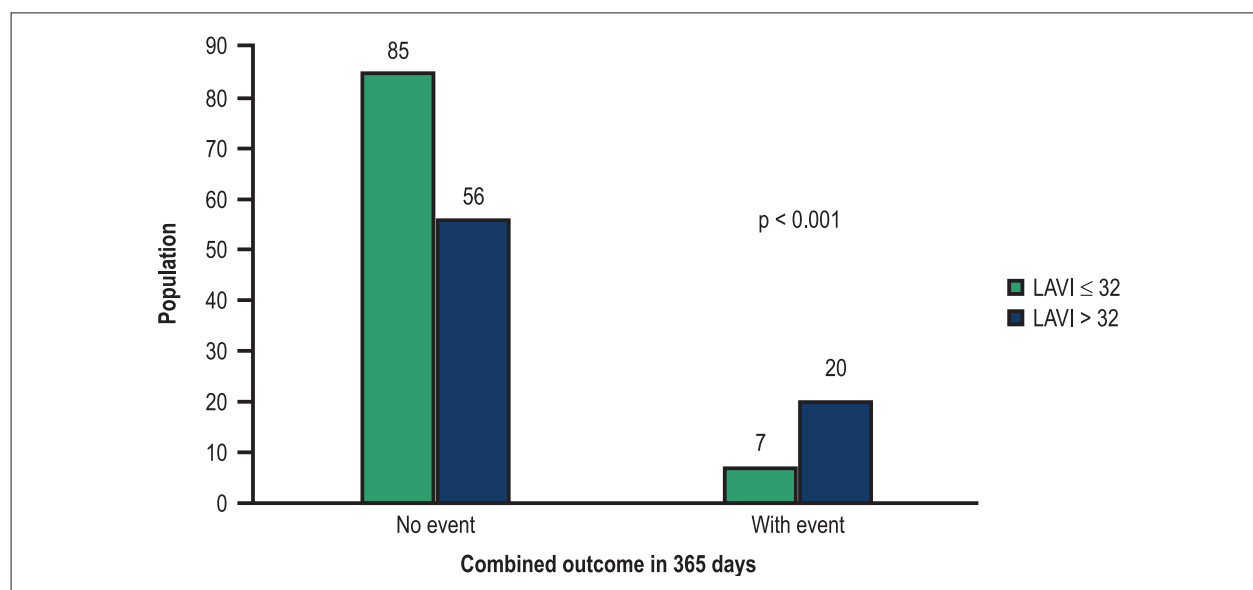

Figure 1 – Frequency of events for out-of-hospital combined outcome in 365 days. Combined outcome: stroke, acute myocardial infarction or death. LAVI: left atrial volume index.

noting that the group with increased LAVI had a significantly higher frequency of acute pulmonary edema, probably reflecting similar pathophysiological processes.

Barnes et al[33] have shown the importance of LAV as an independent predictor of first ischemic stroke and of death in individuals with no prior atrial fibrillation. Regarding out-of-hospital outcome, the present study found that patients with increased LAVI had a higher frequency of stroke, apparently not related to the presence of atrial fibrillation, whose occurrence was equal in both groups. The increase in LAVI, reflecting the lack of a pattern of atrial architecture, certainly contributes to thrombus formation, independently of the presence of atrial fibrillation.

Regarding echocardiographic characteristics, patients with increased LAVI had lower ejection fraction and more severe diastolic dysfunction as compared with those with normal LAVI. Confirming those data, the study by Moller et al[14] has found similar results.

Tsang et al[18] have reported that a 30% increase in LAV was accompanied by a 43% increase in the risk of atrial fibrillation in an elderly population at the beginning of the investigation, with sinus rhythm and no significant heart disease. It is worth noting that, in the present study, although patients with LAVI > 32 mL/m² had higher frequency of atrial fibrillation during hospitalization, no significant difference was observed [6/78 (7%) vs. 2/93 (2%), p = 0.14].
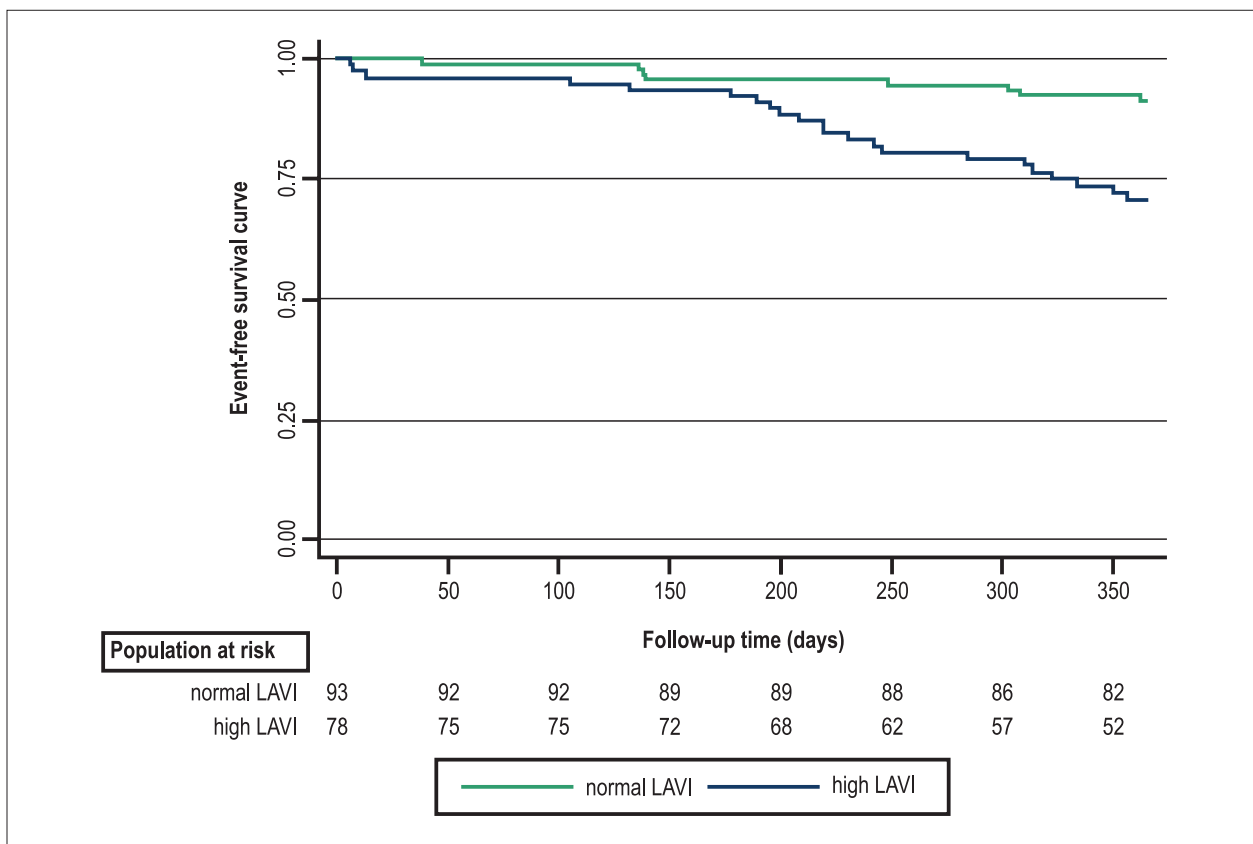
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Figure 2 – *Kaplan-Meier curve for out-of-hospital combined outcome in 365 days between the group with LAVI ≤ 32 mL/m², considered normal, and the group with LAVI > 32 mL/m², considered high. Log-rank test: p = 0.001. LAVI: left atrial volume index. Events: stroke, acute myocardial infarction or death.*
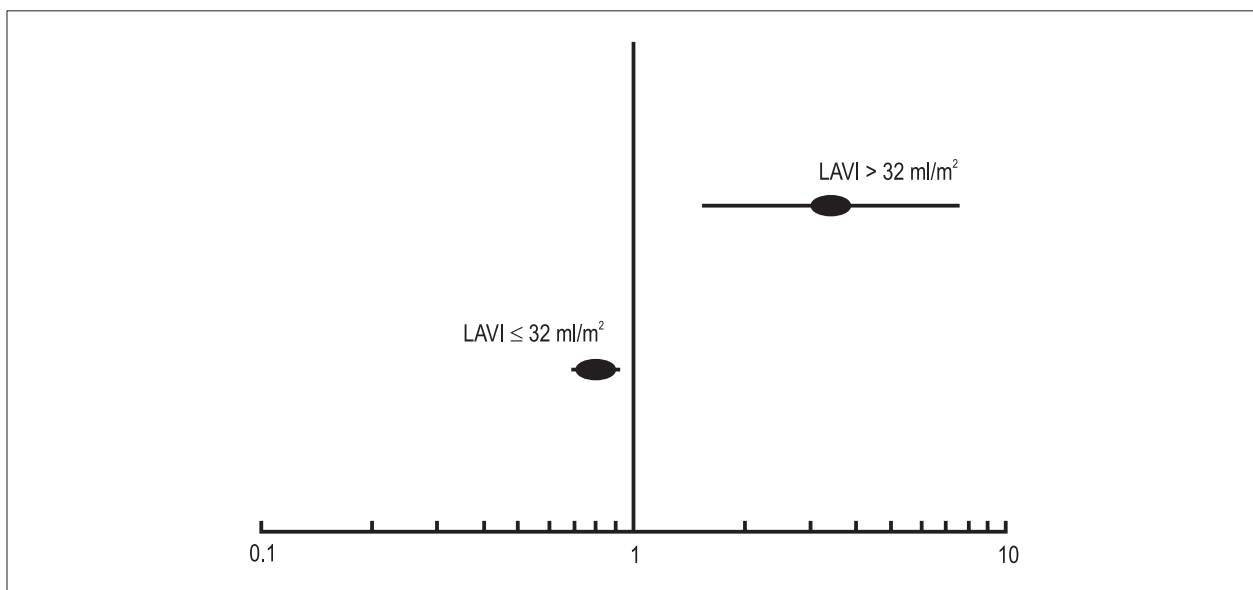

Figure 3 – *Forest plot of relative risk (RR) and 95% confidence intervals (CI) for out-of-hospital combined outcome in 365 days, according to left atrial volume index (LAVI). The estimates found for the groups with high LAVI and normal LAVI were, respectively, RR = 3.5 (CI: 1.54-7.73) and RR = 0.79 (CI: 0.68-0.92)".*

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Table 4 – Cox regression adjusted by use of the Breslow method for left atrial volume index in acute coronary syndrome with combined outcome* in 365 days

	HR	95% CI	p
Age	1.01	0.97 - 1.04	0.52
Sex	0.87	0.38 – 1.99	0.74
ACS type †	1.39	0.79 – 2.47	0.26
DM	1.10	0.73 – 1.66	0.63
Dyslipidemia	0.78	0.32 – 1.89	0.59
SAH	3.07	0.69 – 13.65	0.14
EF	0.74	0.04 – 15.37	0.85
LAVI	3.08	1.28 – 7.40	0.012

HR: hazard ratio; 95% CI: 95% confidence interval; DM: diabetes mellitus; SAH: systemic arterial hypertension; EF: ejection fraction; ACS: acute coronary syndrome; LAVI: left atrial volume index. (*) combined outcome: stroke, acute myocardial infarction or death; (†) subdivided into unstable angina, ST elevation acute myocardial infarction and non-ST elevation acute myocardial infarction.
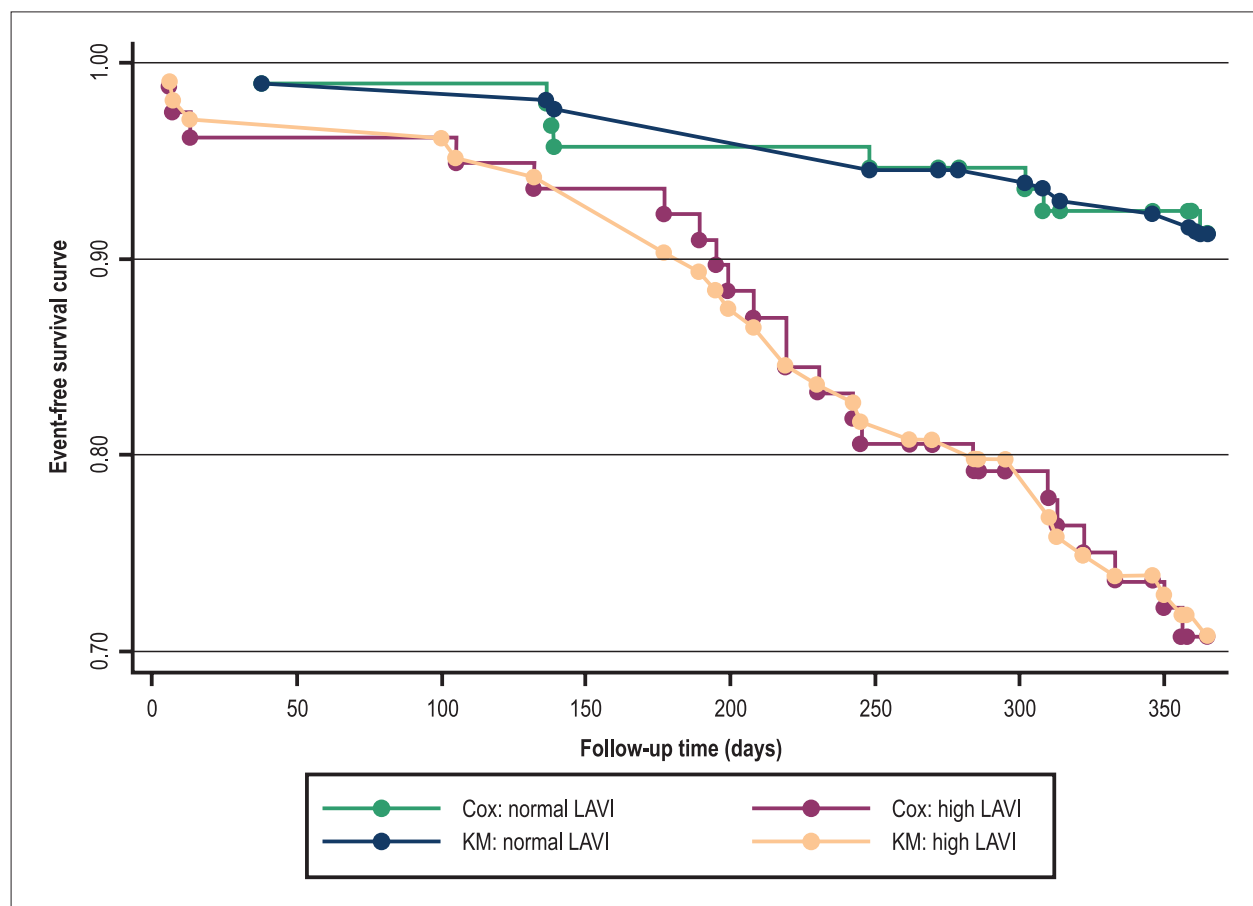

Figure 4 – *Adjusted Cox regression curves compared with Kaplan-Meier curves for out-of-hospital combined outcomes in 365 days between the group with LAVI ≤ 32 mL/m², considered normal, and the group with LAVI > 32 mL/m², considered high. KM - Kaplan-Meier; LAVI: left atrial volume index. Events: stroke, acute myocardial infarction or death.*

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Moller et al[14] have shown for the first time the importance of LAV as a predictor of survival after AMI. In that study, during a 15-month follow-up, LAVI > 32 mL/m² was a powerful independent predictor of all-cause mortality. Similarly, although without significance, mortality was higher among patients with increased LAVI. Maybe because of the relatively younger population sample (mean age, 66 vs. 70 years) and the shorter follow-up, the present study showed no significant difference. Recently, in a cohort of 1886 African Americans, left atrial size was a predictor of all-cause mortality after adjusting for traditional cardiovascular risk factors, left ventricular hypertrophy and low ejection fraction[34].

Regarding out-of-hospital outcome, major adverse cardiovascular events (stroke, AMI or death) were significantly more frequent in patients with LAVI > 32 mL/m². In a recent publication, Gunasekaran et al[13] have also reported that an increased LAVI (cut-off point of 28 mL/m²) is an independent risk factor for major cardiovascular event (except for mortality) six months after ACS.

In this study, the Kaplan-Meier survival curves for combined outcome in 365 days evidenced significant discrepancy between the groups. By using RR, it is evident that belonging to the group with LAVI > 32 mL/m² represents a three-fold increase in the risk of having a cardiovascular event. On the contrary, belonging to the group with normal LAVI is a mild protective factor against those events (Figure 3). Beinart et al[15], assessing LAVI > 32 mL/m² as an independent predictor of mortality in five years in patients with AMI, have found a RR of 2.05 (95% CI: 1.02-4.11; p=0.044).

On univariate analysis to predict mortality, Moller et al[14] have found a 500% increased risk when comparing increased LAVI and normal LAVI (HR = 6.1; 95% CI: 2.8-13.0; p < 0.001). Later, Beinart et al[15] have found a risk increased by almost 190% on univariate analysis for mortality in five years in patients with increased LAVI (HR = 2.78; 95% CI: 1.63-4.72; p < 0.001). Similarly, in our study, Cox regression with eight variables (Table 4) showed that increased LAVI increased by more than 200% the probability of outcomes (HR = 3.67; 95% CI: 1.45-9.26; p = 0,006).

Several resources of analysis of sensitivity were used to assess the properness of the parameters. Similarly, our model proved to have no violations and to be compatible with the assumptions necessary to validate the survival analyses.

Limitations

Our study has some limitations. The patients were selected at a single center. This was a non-probability sample selection from a registry, from where only individuals whose LAVI values were measured were extracted.

In addition, it is worth noting that the number of variables assessed as compared with the sample size might have reduced the power to identify other factors. The loss to follow-up rate, 7 patients (7%) with normal LAVI and 8 patients (10%) with increased LAVI, can also be associated with bias.

Conclusion

In our study, increased LAVI was an important predictor of major cardiovascular events in the one-year follow-up. Because it is easily obtained on echocardiography, and because it provides important information for clinical practice, LAVI measurement should be incorporated as routine to the assessment of patients suspected of having or diagnosed with ACS.

Author contributions

Conception and design of the research: Secundo Junior JA, Santos MAA, Oliveira JLM, Barreto Filho JAS, Sousa ACS; Acquisition of data: Secundo Junior JA, Faro GBA, Soares CB, Silva AMP, Secundo PFC, Teixeira CKC; Analysis and interpretation of the data: Secundo Junior JA, Santos MAA, Barreto Filho JAS, Sousa ACS; Statistical analysis: Santos MAA; Writing of the manuscript: Secundo Junior JA, Santos MAA, Faro GBA, Soares CB, Silva AMP, Secundo PFC, Teixeira CKC, Barreto Filho JAS, Sousa ACS; Critical revision of the manuscript for intellectual content: Secundo Junior JA, Santos MAA, Faro GBA, Soares CB, Silva AMP, Secundo PFC, Teixeira CKC, Oliveira JLM, Barreto Filho JAS, Sousa ACS.

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