

Assessment of Myocardial Infarction by Cardiac Magnetic Resonance Imaging and Long-Term Mortality

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Abstract

Background: Cardiac magnetic resonance imaging provides detailed anatomical information on infarction. However, few studies have investigated the association of these data with mortality after acute myocardial infarction.

Objective: To study the association between data regarding infarct size and anatomy, as obtained from cardiac magnetic resonance imaging after acute myocardial infarction, and long-term mortality.

Methods: A total of 1959 reports of “infarct size” were identified in 7119 cardiac magnetic resonance imaging studies, of which 420 had clinical and laboratory confirmation of previous myocardial infarction. The variables studied were the classic risk factors – left ventricular ejection fraction, categorized ventricular function, and location of acute myocardial infarction. Infarct size and acute myocardial infarction extent and transmural extent were analyzed alone and together, using the variable named “MET-AMI”. The statistical analysis was carried out using the elastic net regularization, with the Cox model and survival trees.

Results: The mean age was 62.3 ± 12 years, and 77.3% were males. During the mean follow-up of 6.4 ± 2.9 years, there were 76 deaths (18.1%). Serum creatinine, diabetes mellitus and previous myocardial infarction were independently associated with mortality. Age was the main explanatory factor. The cardiac magnetic resonance imaging variables independently associated with mortality were transmural extent of acute myocardial infarction ($p = 0.047$), ventricular dysfunction ($p = 0.0005$) and infarcted size ($p = 0.0005$); the latter was the main explanatory variable for ischemic heart disease death. The MET-AMI variable was the most strongly associated with risk of ischemic heart disease death (HR: 16.04; 95%CI: 2.64-97.5; $p = 0.003$).

Conclusion: The anatomical data of infarction, obtained from cardiac magnetic resonance imaging after acute myocardial infarction, were independently associated with long-term mortality, especially for ischemic heart disease death. (Arq Bras Cardiol. 2015; 104(2):159-168)

Keywords: Myocardial Infarction/physiology; Magnetic Resonance Imaging; Diagnostic Imaging; Mortality; Risk Factor.

Introduction

The size and morphology of myocardial fibrosis or necrosis area are potentially associated with the occurrence of ventricular dysfunction and ventricular arrhythmias, which are themselves associated with mortality in patients after acute myocardial infarction (AMI)^{1,2}. The introduction of cardiac magnetic resonance imaging (CMRI) in the assessment of AMI³ was a major advance. It permits thorough documentation of myocardial infarct size and anatomy, improving both the diagnostic ability and the perspective of post-AMI risk

assessment. However, some knowledge gaps regarding the association of AMI anatomical data provided by CMRI with long-term mortality still exist, as well as regarding its ability to add relevant prognostic information. The number of studies is limited, and most of them had short follow-up periods and involved North American and European populations⁴⁻¹².

Objective

To verify the association of data regarding infarct size and anatomy with overall mortality, circulatory system disease mortality, and ischemic heart disease (IHD) mortality in patients undergoing CMRI after AMI.

Methods

Study design and population

Between June 2001 and December 2010, 7119 CMRI records and 1959 reports with the term “infarct size” (IS)

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were found in a tertiary health care network of imaging laboratories and hospitals. There were 489 patients undergoing CMRI after hospitalization, with documented elevation of troponin levels, of whom 420 were selected for diagnostic confirmation of AMI from their medical records; patients with non-ischemic myocardial injury were excluded. This study was approved by the Research Ethics Committee of University Hospital Clementino Fraga Filho (HUCFF), Federal University of Rio de Janeiro (UFRJ).

Variables analyzed

The demographic and clinical variables analyzed were: age; gender; previous AMI; diabetes mellitus; systemic hypertension; smoking habit; dyslipidemia; angiographic classification of obstructive coronary artery disease; treatment with CABG; serum creatinine levels; and peak serum troponin I level. Mortality and the basic cause of death were obtained from probabilistic relationship of identification data of the study population, with 1,485,735 records of the Mortality Information System (*Sistema de Informação de Mortalidade -SIM*) between 2001 and 2012.

Cardiac magnetic resonance imaging findings

CMRI findings were obtained from the medical records of each patient. The resonance techniques routinely used for the assessment of patients with AMI were cine-magnetic resonance, for left ventricular (LV) functional assessment, and late enhancement technique¹³, for the assessment of myocardial necrosis or fibrosis.

LV function was obtained from LV ejection fraction (EF) (Simpson's method) and from the LV function category by subjective visual analysis, and was classified as normal or mild, moderate or severe dysfunction.

AMI location was based on the 17-segment model used for myocardial segmentation nomenclature¹⁴. The presence of transmural AMI was defined by the involvement of > 50% of the segment^{15,16}. The AMI type, regarding transmural, was thus classified into three categories: transmural (TM), non-transmural (NTM), and mixed (when both AMI types were present). The percentage of infarcted LV mass was calculated using the semi-quantitative visual score method, as described elsewhere¹⁷. The AMI transmural index (TI) was obtained by calculating the ratio between the number of segments with transmural infarction (SegTM AMI) and the total number of segments with AMI (NSegAMI).

The MET-AMI variable was elaborated to express AMI size and complexity, combining three categorized primary variables: AMI transmural, IS, and NSegAMI.

The definitions of abnormality (negative vs positive) for the variables comprising MET-AMI were: (1) transmural, if mixed AMI present; (2) categorization of components of the MET-AMI composite variable was obtained from the most accurate cut-off value found in the ROC (Receiver Operating Characteristic) curve, i.e., IS \geq 14% and NSegAMI \geq 4.

The MET-AMI variable was graded according to the number of abnormal components: grade-1 MET-AMI for no abnormality; grade-2 MET-AMI for one abnormal variable;

grade-3 MET-AMI for two abnormal variables; grade-4 MET-AMI for three abnormal variables.

The presence of microvascular obstruction (no reflow) was documented whenever described in the test report.

Statistical analysis

Data analysis was carried out to evaluate the three endpoints: overall mortality, circulatory system disease death, and IHD death, as codified in the Declaration of Death (DD) by the 10th Revision of the International Classification of Diseases (ICD-10). Mann-Whitney, chi-square or Fisher's exact test were used for the univariate analysis; the log rank test was used for the analysis of Kaplan-Meier curves. Multivariate analysis was carried out sequentially by means of an initial selection of variables using elastic net regularization (EM), which were analyzed by the Cox model. The survival tree was used for the identification of the main explanatory variables for each endpoint. The significance level was set at 5%. The Pearson method was used for the analysis of correlations between numeric variables.

Results

The mean follow-up period from the day CMRI was performed until the occurrence of death or the end of the observation period was 6.4 ± 2.9 years. The median time between admission for AMI and performance of CMRI was 13 days, and 278 patients (66.1%) underwent the imaging test within the first 30 days after AMI. There were 76 deaths (18.09%), of which 34 (44.7%) were for circulatory system diseases and 22 (29%), for IHD.

Demographic and clinical data

Demographic and clinical characteristics of the study population, by subgroup of survivors and of all-cause deaths, are shown in Table 1.

The subgroup of deaths compared to that of survivors showed: higher mean age; higher prevalences of diabetes mellitus, of previous AMI and two-vessel coronary artery disease; higher median peak troponin levels; and higher percentage of cases with serum creatinine levels > 2 mg/dL. A greater number of myocardial revascularization procedures was observed in the subgroup of survivors (73.3% vs. 47.4%). The angiographic profile of single-vessel disease was more frequent among survivors, and only 19.8% did not undergo cardiac catheterization.

CMRI data in the study population, in the subgroup of survivors and in the all-cause death group are shown in Table 2.

Comparing the subgroup of deaths with that of survivors, a higher prevalence of mixed AMI was observed in the group of deaths. Number of segments with TM AMI, total number of segments with AMI, and IS were significantly higher in the subgroup of deaths.

The Pearson test showed positive linear correlations between IS and SegTM AMI ($r = 0.83$) and between IS and NSegAMI ($r = 0.78$). LVEF showed a negative linear association with IS ($r = -0.57$).

Table 1 – Demographic and clinical characteristics of the study population and of the subgroups of survivors and all-cause deaths

Variables	Total population (n = 420)	Survivors (n = 344)	Deaths (n = 76)	p value
Age, mean ± SD (years)	62.3 ± 12.0	60.4 ± 12.4	72.6 ± 12.1	< 0.001
Male gender	325 (77.3)	270 (78.4)	55 (72.3)	0.248
SH	312 (74.2)	254 (73.8)	58 (76.3)	0.655
Diabetes, n (%)	127 (30.2)	91 (26.4)	36 (47.3)	< 0.001
Cigarette smoking, n (%)	136 (32.3)	119 (34.6)	17 (22.3)	0.039
Dyslipidemia, n (%)	212 (50.4)	179 (52.0)	33 (43.4)	0.174
Previous AMI, n (%)	207 (49.2)	153 (44.4)	54 (71.0)	< 0.001
Angiographic profile of CAD, n (%)				0.566
Single-vessel	176 (41.9)	147 (42.7)	29 (38.2)	
Two-vessel	72 (17.1)	56 (16.3)	16 (21.0)	
Three-vessel or multivessel	89 (21.2)	73 (21.2)	16 (21.0)	
CAT not performed	83 (19.8)	68 (19.7)	15 (19.8)	
Treatment during hospitalization, n (%)				< 0.001
Medical only	132 (31.4)	92 (26.8)	40 (52.6)	
Revascularization by PCI	226 (53.8)	197 (57.3)	29 (38.2)	
Surgical revascularization	62 (14.7)	55 (15.9)	7 (9.2)	
Laboratory data				
Peak troponin I level, median (ng/mL)	12.9	12.2	19.1	0.614
Serum creatinine, mean ± SD (mg/dL)	1.06 ± 0.45	1.0 ± 0.4	1.1 ± 0.5	0.085
Serum creatinine (> 2 mg/dL), n (%)	11 (2.61)	7 (2.03)	4 (5.26)	0.037

SD: standard deviation; SH: systemic hypertension; AMI: acute myocardial infarction; CAD: coronary artery disease; CAT: catheterization; PCI: percutaneous coronary intervention.

Associations of variables with the endpoints studied

Endpoint all-cause mortality

The multivariate Cox model was used for the all-cause death endpoint (time elapsed between performance of CMRI and occurrence of death), with the variables selected by EN (Table 3).

Among the CMRI data, the model identified the following independent variables: moderate or severe ventricular dysfunction, IS and NTM AMI. The model also identified the following clinical or demographic variables as independent variables: age, diabetes mellitus and serum creatinine level. An inverse association was also found between treatment with CABG and mortality.

The MET-AMI variable was tested using the multivariate Cox model again. However, this was proven to be a significant variable for overall mortality ($p = 0.25$).

The survival tree identified age at CMRI, with a cut-off point at 69.7 years, as the most relevant explanatory variable for overall mortality.

Endpoint mortality for circulatory system diseases

The variables selected by EN for the endpoint mortality for circulatory system diseases were: age, diabetes mellitus,

type of treatment, serum creatinine level, IS (%), AMI of anterior location, moderate or severe LV dysfunction, AMI transmural, previous AMI, AMI-CMDRI time (days), male gender, SegNTM AMI and no reflow.

The Cox model identified, among the demographic or clinical variables, age ($p < 0.0001$) and serum creatinine level ($p = 0.001$) as independent variables. Among CMRI variables, the independent variables identified were ventricular dysfunction (hazard ratio – HR: 3.34; 95% confidence interval – 95% CI: 1.35-8.24; $p = 0.008$), IS (HR: 1.10; 95%CI: 1.05-1.15; $p = 0.0001$) and previous AMI (HR: 2.87; 95%CI: 1.04-7.89; $p = 0.041$). The presence of mixed AMI was also a factor identified with significance $< 10\%$ ($p = 0.055$).

The MET-AMI variable was tested again using the Cox multivariate model. Table 4 shows the independent variables identified in the new model. Moderate/severe LV dysfunction, age, serum creatinine level, and grades 3 and 4 MET-AMI were independent variables related to the endpoint circulatory system disease death.

Survival tree for the endpoint circulatory system disease mortality

The survival tree (Figure 1) identified ventricular function, categorized as moderate or severe dysfunction vs mild

Table 2 – Values of variables obtained from cardiac magnetic resonance imaging (CMRI) in the study population, and in the subgroups of survival and all-cause deaths

CRMI variables	Total population (n = 420)	Survivors (n = 344)	Deaths (n = 76)	p value
Anterior AMI, n (%)	218 (51.9)	175 (50.8)	43 (56.5)	0.37
LV IS, median (%)	11.0	10.5	15.0	0.006
LV IS, mean ± SD (%)	14.3 ± 11.0	13.5 ± 10.6	17.7 ± 12.1	0.003
AMI type				0.07
Transmural, n (%)	99 (23.6)	84 (24.4)	15 (19.7)	
Non-transmural, n (%)	165 (39.2)	141 (40.9)	24 (31.5)	
Mixed transmural, n (%)	156 (37.2)	119 (34.6)	37 (48.6)	
NSegTM AMI, mean ± SD	2.3 ± 2.6	2.1 ± 2.5	3.0 ± 2.9	0.013
NSegNTM AMI, mean ± SD	1.8 ± 1.6	1.7 ± 1.6	1.9 ± 1.6	0.411
NSegAMI, mean ± SD	4.1 ± 2.7	3.9 ± 2.5	4.9 ± 3.0	0.003
Transmurality index	0.44 ± 0.41	0.43 ± 0.41	0.49 ± 0.3	0.29
Presence of no reflow, n (%)	28 (6.7)	22 (6.4)	6 (8.6)	0.82
LV systolic function, n (%)				< 0.0001
Normal	221 (52.6)	199 (57.8)	22 (28.9)	
Mild dysfunction	75 (17.8)	64 (18.6)	11 (14.4)	
Moderate dysfunction	47 (11.2)	38 (11.0)	9 (11.8)	
Severe dysfunction	77 (18.3)	43 (12.5)	34 (44.7)	
LV ejection fraction, mean ± SD (%)	51.0 ± 17.0	53.7 ± 15.6	39.8 ± 18.3	< 0.0001

AMI: acute myocardial infarction; IS: infarct size; LV: left ventricle; SD: standard deviation; NSegTM AMI: number of segments with transmural myocardial infarction; NSegNTM AMI: number of segments with non-transmural acute myocardial infarction; NSegAMI: total number of segments with acute myocardial infarction.

Table 3 – Cox model for the all-cause mortality endpoint

Variables	Coefficient	SE	p value	HR	95%CI
Clinical and demographic data					
Age (years)	0.082	0.012	< 0.0001	1.08	1.06-1.11
Diabetes mellitus	0.516	0.246	0.036	1.67	1.03-2.71
Treatment					
Medical only	Reference				
PCI	-0.368	0.291	0.21	0.69	0.39-1.22
CABG	-0.883	0.429	0.039	0.41	0.18-0.96
Serum creatinine (mg/dL)	0.510	0.223	0.022	1.67	1.08-2.58
Previous AMI	0.486	0.296	0.10	1.63	0.91-2.90
CMRI data					
Infarct size (%)	0.048	0.014	0.0008	1.05	1.02-1.08
Anterior AMI	0.486	0.277	0.079	1.63	0.94-2.80
Moderate / severe LV dysfunction	1.054	0.304	0.0005	2.87	1.58-5.20
Type of AMI					
Transmural	Reference				
Non-transmural	0.867	0.436	0.047	2.38	1.01-5.60
Mixed	0.495	0.318	0.11	1.64	0.88-3.06

SE: stand error of the coefficient; HR: hazard ratio; 95%CI: 95% confidence interval; PCI: percutaneous coronary intervention; CABG: coronary artery by-pass grafting; AMI: acute myocardial infarction; CMRI: cardiac magnetic resonance imaging; LV: left ventricle.

Table 4 – Cox model for the endpoint circulatory disease mortality, with the MET-AMI variable included

Variables	Coefficient	SE	p value	HR	95%CI
Age (years)	0.077	0.019	< 0.0001	1.08	1.04-1.12
Diabetes mellitus	0.441	0.390	0.26	1.55	0.72-3.34
Treatment					
Medical	Reference		0.13		
PCI	-0.830	0.479	0.083	0.44	0.17-1.12
CABG	-0.885	0.599	0.13	0.41	0.13-1.33
Serum creatinine level (mg/dL)	0.899	0.303	0.003	2.46	1.36-4.44
Anterior AMI	0.347	0.423	0.41	1.41	0.62-3.24
Moderate/severe LV dysfunction	1.152	0.420	0.006	3.16	1.39-7.20
Previous AMI	0.949	0.516	0.066	2.58	0.94-7.11
AMI-CMRI time (days)	-0.0016	0.001	0.10	1.00	1.00-1.00
Male gender	-0.533	0.448	0.23	0.59	0.24-1.41
No reflow	0.513	0.701	0.46	1.67	0.42-6.59
Variable MET-AMI					
1	Reference				
2	0.028	0.733	0.97	1.03	0.24-4.33
3	1.399	0.612	0.022	4.05	1.22-13.44
4	2.152	0.618	0.0005	8.60	2.56-28.90

SE: standard error of the coefficient; HR: hazard ratio; 95%CI: 95% confidence interval; PCI: percutaneous coronary intervention; CABG: coronary artery by-pass grafting; LV: left ventricle; AMI: acute myocardial infarction; CMRI: cardiac magnetic resonance imaging.

dysfunction or normal, as the most relevant explanatory variable for the classification of survivors and of circulatory system disease deaths. The age variable was also relevant in the model.

Joint assessment of MET-AMI and ventricular function variables

Figure 2 shows the Kaplan-Meier survival curves for the endpoint circulatory system disease mortality, classified in four groups according to relevant changes of the MET-AMI composite variable (defined by cohort ≥ 3) and of left ventricular function (defined by the presence of moderate or severe ventricular dysfunction).

A significant reduction in survival was observed in the group with relevant changes in both variables in comparison to the subgroup with no changes. The subgroups with only one abnormal variable had intermediate survival, however significantly shorter than that of the subgroup with no abnormalities.

Endpoint ischemic heart disease mortality

The variables selected by EN for the endpoint IHD mortality were age, diabetes mellitus, serum creatinine level, IS (%), moderate or severe LV dysfunction, AMI transmural, previous AMI, and SegNTM AMI.

The Cox model applied to these variables showed that, among CMRI variables, IS was the one that demonstrated independent value (HR: 1.09; 95%CI: 1.03-1.15; $p = 0.0015$). Among the clinical and demographic variables, independent value was identified for age (HR: 1.08; 95%CI: 1.03-1.13; $p = 0.001$), serum creatinine level (HR: 2.62; 95%CI: 1.35-5.03; $p = 0.004$) and previous AMI (HR: 2.87; 95%CI: 1.04-7.89; $p = 0.041$).

Table 5 shows the results of the Cox regression model applied to the MET-AMI variable. The MET-AMI variable showed an increasing risk ratio, according to its grade, showing independent value for grades 3 or 4, and was the variable with the highest risk ratio in the model.

Survival tree

The survival tree (Figure 3) identified IS as a relevant explanatory variable, with a cut-off point at 21.0% for the endpoint IHD mortality.

Discussion

Most of the studies assessing data obtained from CMRI after AMI adopted composite endpoints involving the occurrence of decompensated heart failure, reinfarction, ventricular arrhythmias, and death. Their mean follow-up period was 2 years^{7,18}.

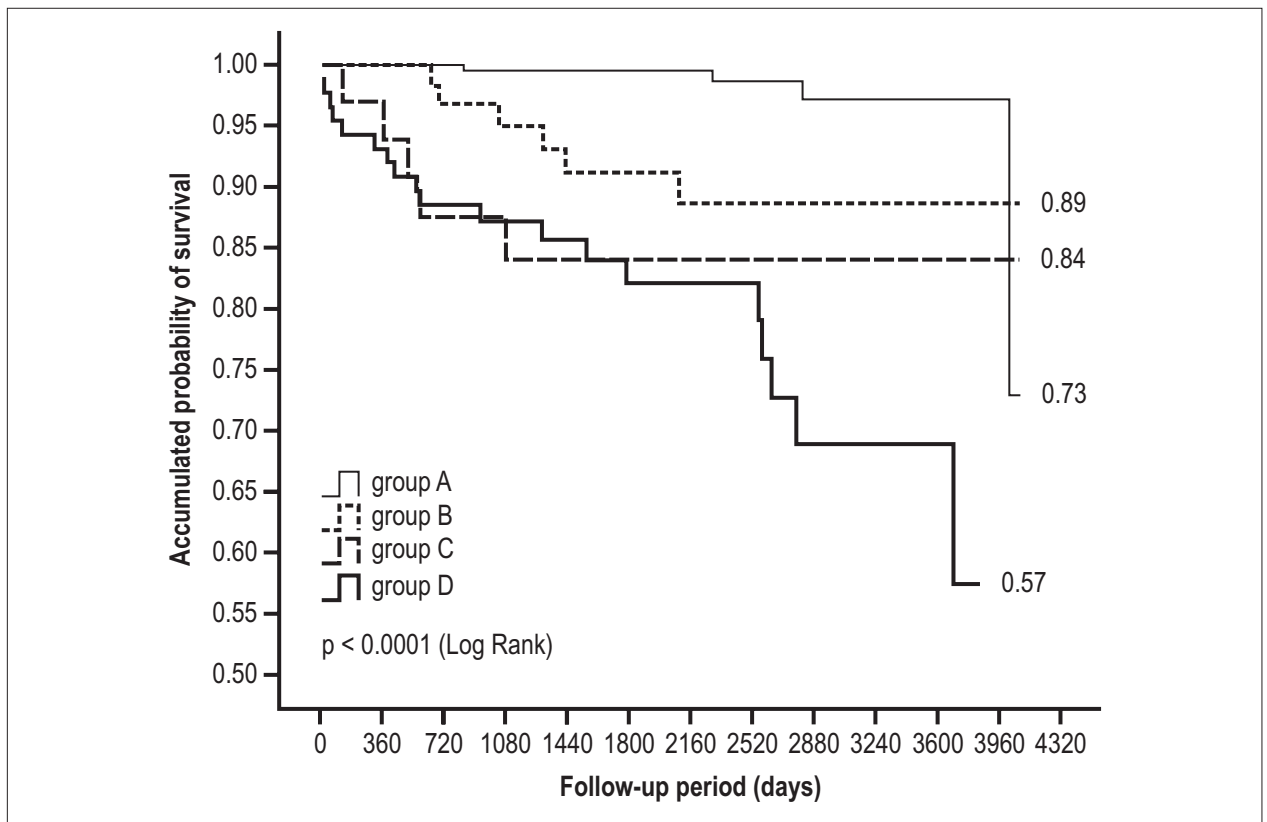


Figure 2 – Kaplan-Meier survival curves for the endpoint circulatory system disease mortality, according to the MET-AMI and left ventricular function variables. Group A: dysfunction absent and grades 1 or 2 MET-AMI; Group B: dysfunction absent and grades 3 or 4 MET-AMI; Group C: moderate or severe dysfunction and grades 1 or 2 MET-AMI; Group D: moderate or severe dysfunction and grades 3 or 4 MET-AMI.

Kwong et al¹⁹ reported a relevant study on the value of late enhancement documented by CMRI in a population with suspected coronary artery disease, and verified that the presence of subclinical AMI, as detected in CMRI, was the factor more significantly associated with the risk of events and mortality. Differently, the focus of the present study was to evaluate the role of CMRI after clinical documentation of AMI.

Other studies have evaluated patients with ischemic heart disease in more advanced stages and with longer follow-up periods^{4,8}, however shorter than that of the present study.

Another differential aspect of this study was the assessment of the basic death cause using DD data. Cheong et al⁴ similarly obtained mortality data using the social security computed system, however without the definition of death cause.

The study population showed demographic and clinical characteristics consistent with those found in national studies and registries²⁰⁻²², as well as in international data²³. The mean age of 62 years is similar to that found for patients with AMI of the ACCEPT registry²¹.

Bello et al⁸ investigated the importance of late enhancement provided by CMRI in 100 patients with coronary artery disease in a stable stage and, in corroboration with our findings, observed an independent association of the ventricular function, IS, and diabetes mellitus variables

with mortality; also, cigarette smoking was more prevalent in the subgroup of survivors. These findings may be potentially attributed to the high prevalence of more severely ill patients with previous coronary artery disease (49.2% with previous AMI in this study), who had probably quit smoking before the index AMI episode.

Four clinical variables seemed always significant to predict post-AMI death, whether in ours or in other studies^{24,25}, namely: age, renal dysfunction (as expressed by serum creatinine levels), history of previous AMI, left ventricular dysfunction, and diabetes mellitus.

The association between the type of AMI and mortality was verified for NTM or mixed AMI, reinforcing the potential anatomical risk for arrhythmic or ischemic phenomena reported for NTM infarction¹¹ or heterogeneous scar^{26,27}.

IS remained a variable of AMI magnitude, with an independent value in the present study, in corroboration with most of the studies^{5,6,8,10,11,18}. The independent value for ventricular dysfunction was more frequently found in studies that also adopted the endpoint all-cause mortality^{4,8}; however, it was also significant to predict short-term composite cardiovascular events after AMI^{7,12}. This was also verified in the present study, in which age and ventricular function were identified as explanatory variables for the endpoint all-cause mortality.

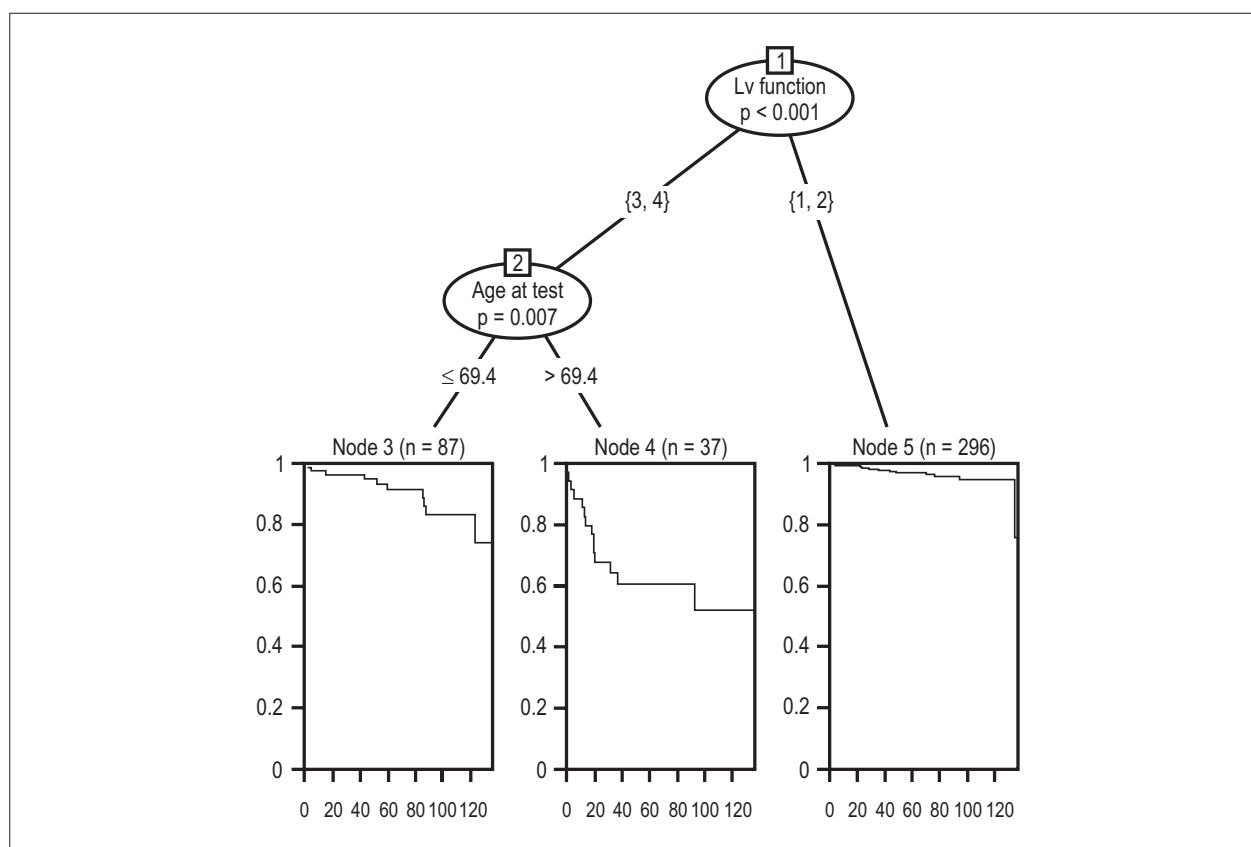


Figure 1 – Survival tree for the endpoint circulatory system disease deaths. (1) Normal LV function; (2) Mild LV dysfunction; (3) Moderate LV dysfunction; (4) Severe LV dysfunction. LV: left ventricle.

Table 5 – Cox model for the endpoint ischemic heart disease mortality, with inclusion of the MET-AMI variable

Variables	Coefficient	SE	p value	HR	95%CI
Age (years)	0.064	0.021	0.003	1.07	1.02-1.11
Diabetes mellitus	0.690	0.463	0.14	1.99	0.80-4.95
Serum creatinine level (mg/dL)	0.797	0.342	0.019	2.22	1.13-4.34
Moderate/severe LV dysfunction	0.623	0.485	0.20	1.86	0.72-4.82
Previous AMI	1.310	0.574	0.022	3.71	1.20-11.4
MET-AMI variable					
Grade 1	Reference				
Grade 2	1.222	0.973	0.21	3.39	0.50-22.8
Grade 3	2.056	0.906	0.023	7.82	1.32-46.2
Grade 4	2.775	0.921	0.003	16.04	2.64-97.5

SE: standard error of the coefficient; HR: hazard ratio; 95%CI: 95% confidence interval; AMI: acute myocardial infarction; LV: left ventricle.

The DETERMINE multicenter randomized clinical trial²⁸ evaluated the prophylactic use of implantable cardiac defibrillator in patients after AMI guided by the IS value found on CMRI, adopting a cut-off value of 10% for CMRI in the late phase or 15% for CMRI in the acute phase, based on previous findings²⁹ of the arrhythmogenic risk of an AMI scar.

In the present study, we observed a 14% cut-off value for IS for the endpoint overall mortality, by the ROC curve; however, for the endpoint IHD mortality, the survival tree identified IS as a relevant variable for a higher cut-off value (21.0%).

Isquierdo et al¹² studied 440 patients in the acute phase of AMI in a 2-year follow-up. The occurrence of composite

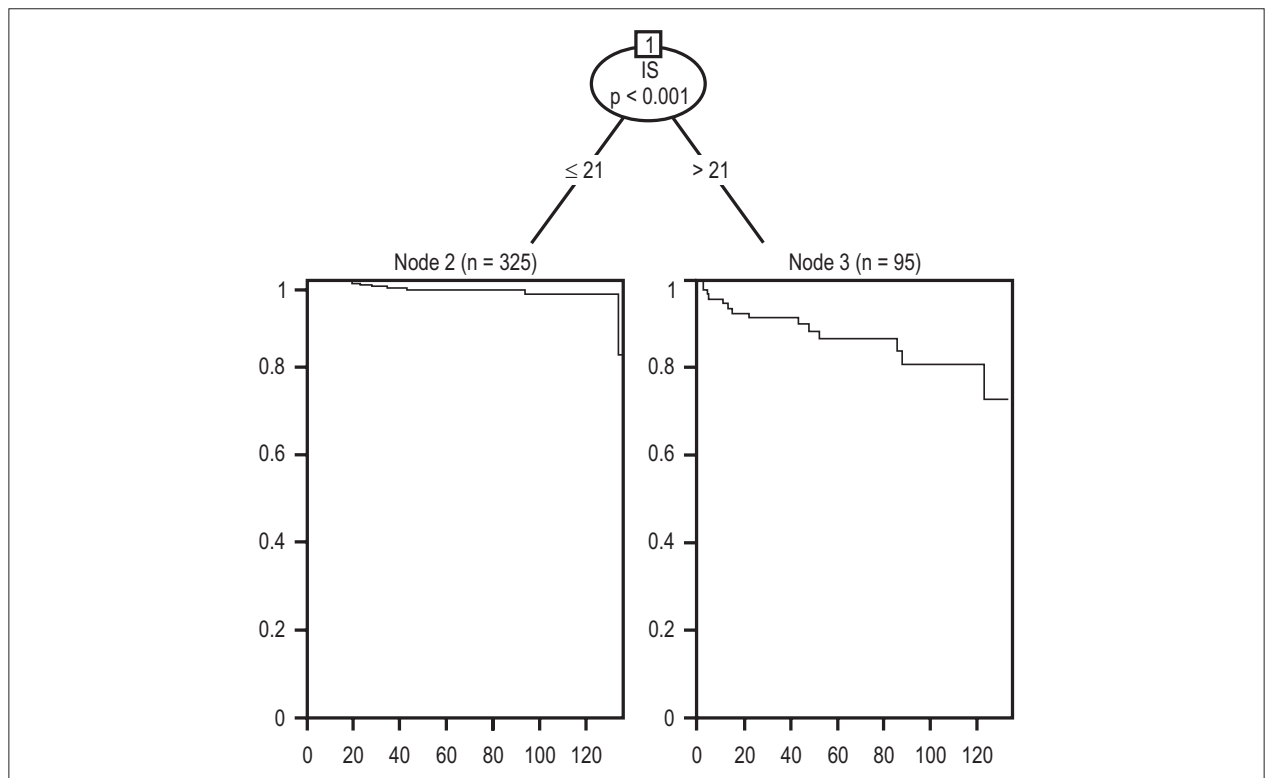


Figure 3 – Survival tree for the endpoint circulatory system disease mortality. IS: infarct size.

endpoints (deaths or severe ventricular arrhythmias) was independently associated with IS; however they occurred predominantly in the presence of ventricular dysfunction. Differently, in the present study, in the subgroup without significant ventricular dysfunction, the presence of \geq grade-3 MET-AMI was associated with shorter survival. The longer follow-up period and the high risk ratio observed for this variable may justify this finding.

limitations

Retrospective study that assessed cases from the partial case series of patients hospitalized for AMI, with a subgroup selected as having a better prognosis, for which CMRI was indicated. A population at different stages after AMI was studied, since CMRI was adopted as the reference.

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Conclusions

The general findings of this study reinforce the ability of clinical and left ventricular function data to predict the risk after acute myocardial infarction. However, the anatomical data provided by cardiac magnetic resonance imaging regarding acute myocardial infarction showed an independent association with long-term mortality, especially for ischemic heart disease death, and thus can contribute for risk identification in selected cases.

Author contributions

Conception and design of the research: Petriz JLF, Souza e Silva NA; Acquisition of data: Petriz JLF, Gomes BFO, Rua BS, Azevedo CF, Hadlich MS, Mussi HTP, Taets GC; Analysis and interpretation of the data: Petriz JLF, Souza e Silva NA; Statistical analysis: Petriz JLF, Azevedo CF, Nascimento EM, Pereira BB; Obtaining Financing: Petriz JLF; Writing of the manuscript: Petriz JLF, Souza e Silva NA; Critical revision of the manuscript for intellectual content: Petriz JLF, Souza e Silva NA.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

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