




## Sudden cardiac arrest due to ventricular arrhythmia in patients with acute myocardial infarction

Maikel Santos Medina<sup>1,2</sup>✉ , MD, MSc; Erlinda Ricardo Mora<sup>1,2</sup> , MD; Miguel A. Rodríguez Ramos<sup>3,4</sup> , MD; and Santiago Batista Bofill<sup>1,2</sup>, MD

<sup>1</sup> Department of Cardiology, Hospital General Docente Dr. Ernesto Guevara de la Serna. Las Tunas, Cuba.

<sup>2</sup> Universidad de Ciencias Médicas de Las Tunas. Las Tunas, Cuba.

<sup>3</sup> Department of Cardiology, Hospital General Docente Camilo Cienfuegos. Sancti Spíritus, Cuba.

<sup>4</sup> Universidad de Ciencias Médicas Dr. Faustino Pérez Hernández. Sancti Spíritus, Cuba.

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### ARTICLE INFORMATION

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### Competing interests

The authors declare no competing interests.

### Abbreviations

**AMI:** acute myocardial infarction

**ICD:** implantable cardioverter-defibrillator

**LVEF:** left ventricular ejection fraction

**SCA:** sudden cardiac arrest

**SCD:** sudden cardiac death

### ABSTRACT

**Introduction:** Sudden cardiac death has not decreased in the same way as mortality due to coronary heart disease, because of the high incidence of sudden cardiac arrest (SCA).

**Objectives:** To determine predictive factors of SCA due to ventricular arrhythmia in patients with acute myocardial infarction.

**Method:** An analytical study was carried out in the Cardiology Department of Las Tunas between 2011 and 2017. The population of study was 917 patients admitted with acute myocardial infarction. All 90 patients with SCA in ventricular arrhythmia represented the “case group”; the rest were the “control group”. Descriptive statistics were used, as well as a multiple logistic regression model; the odds ratios (OR) was calculated, with 95% reliability to determine the predictive factors.

**Results:** In the patients of the case group predominated: male sex (73%), biventricular infarction (24.7%), left ventricular ejection fraction (LVEF) <35% (18.9%) and deceased (41.1%); all these results compared to the control group. A 10.0% was given beta-blockers on their first medical assistance. Cardiogenic shock (OR 15.3), LVEF <35% (OR 8.51), creatine kinase >1200 IU (OR 2.77), obesity (OR 3.16), smoking (OR 2.28), ST-segment elevation/depression on the electrocardiogram >15 mm (OR 2.23) and anterior wall infarction (OR 2.39) were associated with SCA due to ventricular arrhythmia.

**Conclusions:** Cardiogenic shock, LVEF <35%, creatine kinase > 1200 IU, obesity, smoking, ST-segment elevation/depression on the electrocardiogram >15 mm and anterior wall infarction were predictive factors of SCA due to ventricular arrhythmia.

**Keywords:** Acute myocardial infarction, Sudden cardiac death, Cardiac arrest, Ventricular arrhythmia, Risk factors

### *Parada cardíaca súbita por arritmia ventricular en pacientes con infarto agudo de miocardio*

### RESUMEN

**Introducción:** La muerte súbita cardíaca no ha disminuido de igual manera que la mortalidad por enfermedad coronaria, por la elevada incidencia de las paradas cardíacas súbitas (PCS).

**Objetivo:** Determinar factores predictivos de PCS por arritmia ventricular en pa-

✉ M Santos Medina

Jorge Rodríguez Nápoles N° 50 Altos.  
Buena Vista CP 75100. Las Tunas,  
Cuba.

E-mail address: maik@ltu.sld.cu

**Authors' contribution**

MSM: Research planning and design; data collection, analysis and interpretation; and manuscript writing.

ERM: Research design, raw data collection and analysis, and assistance for manuscript writing.

MARR: Research design, data analysis and interpretation.

SBB: Raw data collection and analysis.

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cientes con infarto agudo de miocardio.

**Método:** Se realizó un estudio analítico en el Servicio de Cardiología de Las Tunas entre 2011 y 2017. La población de estudio estuvo conformada por los 917 pacientes ingresados con infarto agudo de miocardio. Los 90 pacientes con PCS en arritmia ventricular constituyeron el «grupo de casos»; el resto fue el «grupo control». Se utilizó estadística descriptiva y un modelo de regresión logística múltiple, y se calculó el índice de probabilidad (odds ratio [OR]), con un 95% de confiabilidad para determinar los factores predictivos.

**Resultados:** En los pacientes del grupo de casos predominó el sexo masculino (73%), el infarto biventricular (24,7%), la fracción de eyección ventricular izquierda (FEVI) <35% (18,9%) y los fallecidos (41,1%) en relación al grupo control. Al 10,0% se le administró betabloqueadores en la primera asistencia médica. El shock cardiogénico (OR=15,3), la FEVI <35% (OR=8,51), la creatina quinasa >1200 UI (OR=2,77), la obesidad (OR=3,16), el hábito de fumar (OR=2,28), el supra/infradesnivel del ST en el electrocardiograma >15 mm (OR=2,23) y el infarto anterior (OR=2,39) se asociaron a la PCS en arritmia ventricular.

**Conclusiones:** El shock cardiogénico, la FEVI <35%, la creatina quinasa >1200 UI, la obesidad, el hábito de fumar, el supra/infradesnivel del ST en el electrocardiograma >15 mm y el infarto anterior fueron factores predictivos de PCS en arritmia ventricular.

**Palabras clave:** Infarto agudo de miocardio, Muerte súbita cardíaca, Paro cardíaco, Arritmia ventricular, Factores de riesgo

**INTRODUCTION**

Sudden cardiac death (SCD) is a major public health problem owing to its frequency and demographic characteristics. Sudden cardiac death accounts for half of all cardiovascular deaths which unfortunately often occur in the victims' most productive years. Although coronary heart disease mortality has decreased over the last 30 years, SCD has not behaved the same way as the occurrence of sudden cardiac arrest (SCA) has remained stable or has currently increased in relation to total mortality<sup>1,2</sup>.

There have been a wide variety of criteria used in the literature to define SCA and SCD which have gradually evolved over time to achieve an appropriate specific definition. According to Koene *et al*<sup>3</sup> the SCD definition of Myerburg and Castellanos is widely accepted today. According to the authors, SCD is a death due to cardiovascular causes in a patient with or without known preexisting heart disease, in whom the mode and time of death are unexpected. The generally accepted temporal definition is bracketed by a period of up to 1 hour between the onset of an abrupt change in clinical status and loss of consciousness which is considered up to 24 hours when the patient was stable and was found dead with no eye witnesses. It is considered as SCA when recovery maneuvers succeed in restoring blood flow<sup>3</sup>.

The incidence of SCA due to ventricular arrhythmias in the in-hospital phase of acute myocardial infarction (AMI) accounts for up to 6% in the first 48 hours after the onset of symptoms, most often during reperfusion or before. In addition to rapid and complete bypass, non-pharmacological interventions and drug treatment may be necessary to control ventricular arrhythmias in this situation<sup>4</sup>.

The two most involved mechanisms related to the appearance of fatal ventricular arrhythmias in patients with ischemic heart disease are: acute coronary ischemia in patients with plaque rupture and epicardial coronary artery occlusion (both detected in autopsies in 20-80% of cases) which is also associated with primary ventricular fibrillation in more than 60% of cases. Coronary artery disease causes SCA and SCD through a second mechanism consisting on the appearance of scar-related ventricular tachycardia after myocardial infarction, which is a requirement for reentry, causing unidirectional block and slow conduction areas<sup>5,6</sup>.

Although the association between warning symptoms such as precordial pain and syncope was known as early as the time of Hippocrates around 400 BC, true advances in predicting, preventing and treating SCA and SCD did not begin to appear until about 50 years ago<sup>1</sup>.

Predicting sudden cardiac death is the cornerstone of arrhythmology and the many attempts to

provide reliable risk factors or predictors of SCD have driven one of the most dynamic areas of research in arrhythmology over the past decades<sup>4</sup>.

For more than 20 years, researchers around the world have been developing several non-invasive SCD risk markers for patients with myocardial ischemia; among them we could mention: the need for programmed ventricular stimulation, late potentials, heart rate variability, baroreflex sensitivity, QT interval dispersion, microvolt T-wave alternans and heart rate turbulence<sup>7-9</sup>. Accordingly, left ventricular ejection fraction (LVEF) is the only indicator that has consistently shown an association with increased risk of sudden death in an AMI/left ventricular dysfunction scenario. For more than a decade this variable has been used to indicate the use of an implantable cardioverter-defibrillator (ICD) for primary prevention of SCD due to ventricular arrhythmias. However, LVEF is not an exact clinical parameter. A number of studies conclude that LVEF has low sensitivity to prevent sudden death especially during hospital admission due to an acute coronary event, as less than 50% of patients with a first AMI, who suffer from SCA or SCD, show an LVEF below 30%<sup>7,8</sup>.

Despite progress, risk stratification for primary prevention of SCD due to malignant ventricular arrhythmias is still deemed to be insufficient. As there are several factors, in addition to LVEF, that may affect the prognosis of patients (both in the in-hospital phase and during follow-up) a large number of non-invasive variables are being studied in order to be considered risk predictors. Among them are those related to patient history, clinical, genetic, laboratory, electrocardiographic and echocardiographic, so as to identify patients at increased risk of SCA and SCD<sup>9,10</sup>.

Cardiovascular diseases continue to be the main cause of death in Cuba and 64.9 per cent of them are due to ischemic heart diseases, of which 45.3% are caused by an AMI. Therefore, by the end of 2017 some 7982 patients had died from this cause, many of whom presented with out-of-hospital SCD or during admission<sup>11</sup>.

Considering Cuba's aging population and the poor primary and secondary prevention of risk factors, the number of

deaths may well increase in the coming years. It is therefore of special interest to stratify risk in this type of patient –and especially in those who may die suddenly due to SCA– in order to draw up strategies to provide the most effective therapy for the patients most at risk. This work was therefore carried out to determine predictive factors for SCA due to ventricular arrhythmia in patients with AMI.

## METHOD

An analytical study was accomplished at the Department of Cardiology of Las Tunas in the period from January 2011 to December 2017. The study population encompassed a total of 917 patients admitted with AMI within the research period to whom

**Table 1.** Baseline characteristics of patients with acute myocardial infarction with or without sudden cardiac arrest due to ventricular arrhythmia. Servicio de Cardiología, Hospital Ernesto Guevara, 2011-2017.

| Variables                                | With SCA (n=90) | Without SCA (n=827) |
|--|-----------------|---------------------|
| Male                                     | 73.0            | 64.3                |
| Age (years, mean±SD)                     | 65±16           | 66±21               |
| <b>Associated factors</b>                |                 |                     |
| High blood pressure                      | 77.8            | 75.8                |
| Diabetes mellitus                        | 17.8            | 23.9                |
| Smoking                                  | 44.4            | 38.1                |
| Hypercholesterolemia                     | 7.8             | 6.3                 |
| Hypertriglyceridemia                     | 15.6            | 11.5                |
| Obesity                                  | 18.6            | 6.6                 |
| PPH of ischemic heart disease            | 38.9            | 27.8                |
| Late arrival at FMC                      | 35.6            | 32.4                |
| <b>Medication during FMC</b>             |                 |                     |
| Antiplatelet drugs                       | 77.8            | 87.2                |
| Beta-blockers                            | 10.0            | 24.6                |
| <b>Laboratory variables on admission</b> |                 |                     |
| Glycemia (mmol/L)                        | 9.3±6           | 7.3±4               |
| Creatinine (mmol/L)                      | 109±20          | 97±28               |
| Total CK (UI)                            | 1736±22         | 887±32              |

The values are expressed as percentage (%) or mean±standard deviation. FMC, first medical care; PPH personal pathological history; SD, standard deviation.

all variables were applied and who were subsequently divided into two groups: the study group, consisting of all 90 patients who presented SCA on hospital admission and in whom ventricular tachycardia or ventricular fibrillation, or both, were found; and the control group, consisting of all 827 patients who did not present SCA.

The variables used were: sex, age, history of high blood pressure, diabetes mellitus, previous history of myocardial infarction, smoking, hypercholesterolemia, hyperlipidemia, type of AMI and topography, number of affected leads on the electrocardiogram, sum of ST-segment elevation/depression on the electrocardiogram, LVEF on admission, drugs administered at first aid, performance and effectiveness of thrombolysis, creatine kinase, creatinine and glycemia values on admission, as well as complications during in-patient treatment.

We applied descriptive statistics through percentage analysis and arithmetic mean for each descriptive variable. A multilogistic regression model was used to determine the risk factors of SCA due to malignant ventricular arrhythmia during hospitalization, using SCA as the dependent variable. Odds ratios (OR) and confidence intervals were calculated for 95% reliability. A value of  $p < 0.05$  was considered statistically significant.

## RESULTS

Mean age was similar in both groups, and 73.0% of patients with AMI who presented with SCA due to ventricular arrhythmia at admission were male (**Table 1**). The most frequent associated factor in both groups was high blood pressure, which was found in 77.8% of patients with SCA and in 75.8% of those who did not have cardiac arrest. Smoking turned out to be the second most frequent factor in both groups (44.4% vs. 38.1%). A total of 18.6% of the patients with SCA were obese, exceeding 6.6% of the cases in the other group. All those who did not present SCA had a higher percentage of antiplatelet (87.2% vs. 77.8%) and beta-blockers (24.6% vs. 10.0%) administration at the first medical assistance site. Mean total creatine kinase values were much higher in patients with SCA ( $1736 \pm 22$  vs.  $887 \pm 32$  IU).

**Table 2** shows that 67.2% of patients with SCA due to ventricular arrhythmia presented more than 7 electrocardiographic leads with ischemia on admission. A 54.1%, presented ST-segment elevation/depression on the electrocardiogram  $> 15$  mm. Both figures being much higher than those found in the other control group. It is noteworthy that ST-segment elevation AMI prevailed in both groups (89.7% and 81.5%) where patients with SCA (40.0%) had a predominantly anterior wall topography while those without SCA had a predominantly inferior wall infarction. We must stress the difference between the two groups in relation to biventricular AMIs, which were evident in 24.7% of cases with cardiac arrest and only 8.2% of patients who did not present this complication.

Most patients in both groups presented LVEF during admission above 35%; however, the percentage of cases with lower values was higher in patients who presented SCA (18.9%) when compared to the other group (7.0%). In both groups, there were a number of cases where LVEF could not be measured on admission (**Table 2**).

**Table 2.** Electrocardiographic disorders, left ventricular ejection fraction and type of infarction on admission.

| Aspects  | With SCA<br>(n=90) | Without SCA<br>(n=827) |
|--|--------------------|------------------------|
|  | %                  | %                      |
| <b>Electrocardiographic disorders</b>            |                    |                        |
| More than 7 affected leads                       | 67.2               | 41.9                   |
| Sum of ST-segment elevation/depression $> 15$ mm | 54.1               | 26.3                   |
| <b>Type of infarction</b>                        |                    |                        |
| - ST-segment elevation MI                        | 89.7               | 81.5                   |
| Anterior   | 40.0               | 32.5                   |
| Inferior   | 25.0               | 40.8                   |
| Biventricular                                    | 24.7               | 8.2                    |
| - Non-ST-segment elevation MI                    | 5.6                | 15.9                   |
| - Other  | 4.7                | 2.6                    |
| <b>LVEF</b>                                      |                    |                        |
| LVEF $> 35\%$                                    | 65.6               | 83.5                   |
| LVEF $< 35\%$                                    | 18.9               | 7.0                    |
| Unspecified LVEF                                 | 15.5               | 9.5                    |

LVEF, left ventricular ejection fraction; MI, myocardial infarction.

**Table 3.** Patients with infarction with or without resuscitated sudden cardiac arrest due to ventricular arrhythmia, depending on thrombolysis application and effectiveness.

| Aspects                | With SCA<br>(n=90) | Without SCA<br>(n=827) |
|------------------------|--------------------|------------------------|
|                        | %                  | %                      |
| <b>Thrombolysis</b>    | 53.7               | 69.4                   |
| Effective              | 35.6               | 43.5                   |
| Not effective          | 18.1               | 25.9                   |
| <b>No thrombolysis</b> | 46.3               | 30.6                   |

SCA, sudden cardiac arrest.

More than 50% of all cases received thrombolytic treatment in both groups, but it proved to be greater (69.4% vs. 53.7%) and more effective (43.5% vs. 35.6%) in patients without cardiac arrest (Table 3). In the study group, as expected, there was a higher percentage of patients (46.3% vs. 30.6%) who did not receive thrombolysis.

Table 4 clearly indicates that patients in the study group who presented SCA due to ventricular arrhythmia during admission, later on –at follow-up and due to recurrences of these or other associated complications– presented high mortality compared to the control group (41.1% vs. 10.9%). The first 66.6% presented pump failure III-IV, according to Killip and Kimball's classification, with a higher prevalence of re-infarction (24.4%), high-grade atrioventricular block (23.3%) and mechanical complications (6.6%), than patients in the control group.

When performing the logistic regression study by using SCA due to ventricular arrhythmia as a dependent variable (Table 5), we found that the presence of Killip-Kimball IV (OR=15.3; p<0.0001), LVEF less than 35% (OR=8.51; p<0.0001), creatine kinase values>1200 UI (OR=2.77; p=0.001), obesity (OR=3.16, p=0.011), smoking (OR=2.28, p=0.017), ST-segment elevation/depression on the electrocardiogram>15 mm (OR=2.23, p=0.043) and anterior wall acute myocardial infarction (OR=2.39, p=0.015) were predictors of SCA due to ventricular arrhythmia. Glycemia figures above 15.0 mmol/L and creatinine above 200 µmol/L as well as the presence of more than 7 affected leads on the electrocardiogram, showed OR values above 1, but with non-significant p values, results that may be influenced by the small sample size.

**Table 4.** Other complications during admission, according to the presence or not of resuscitated sudden cardiac arrest due to ventricular arrhythmias.

| Complications                   | With SCA<br>(n=90) | Without SCA<br>(n=827) |
|---------------------------------|--------------------|------------------------|
|                                 | %                  | %                      |
| <b>Death</b>                    | 41.1               | 10.9                   |
| <b>Killip-Kimball III-IV</b>    | 66.6               | 8.9                    |
| <b>Re-infarction</b>            | 24.4               | 2.7                    |
| <b>High degree AVB</b>          | 23.3               | 9.8                    |
| <b>Atrial fibrillation</b>      | 14.4               | 5.8                    |
| <b>Mechanical complications</b> | 6.6                | 1.5                    |

AVB, atrioventricular block; SCA, sudden cardiac arrest.

**Table 5.** Logistic regression model: sudden cardiac arrest due to ventricular arrhythmia as a dependent variable.

| Variables   | OR<br>(95% CI) | p                 |
|---|----------------|-------------------|
| Age >70 años                                      | 0.51           | 0.064             |
| PPH of ischemic heart disease                     | 1.32           | 0.414             |
| Smoking   | 2.28           | 0.017             |
| Obesity   | 3.16           | 0.011             |
| Late arrival at FMC                               | 0.74           | 0.410             |
| Creatinine >200 µmol/L                            | 2.12           | 0.084             |
| Glycemia >15.0 mmol/L                             | 3.77           | 0.088             |
| Anterior AMI                                      | 2.39           | 0.015             |
| Biventricular AMI                                 | 0.83           | 0.713             |
| Total CK >1200 UI                                 | 2.77           | 0.001             |
| More than 7 affected leads                        | 1.78           | 0.212             |
| Sum of ST-segment elevation/<br>depression >15 mm | 2.23           | 0.043             |
| LVEF < 35%  | 8.51           | <0.0001           |
| <b>Killip-Kimball IV</b>                          | <b>15.39</b>   | <b>&lt;0.0001</b> |

AMI, acute myocardial infarction; CK, creatine kinase; FMC, first medical care; LVEF, left ventricular ejection fraction; PPH personal pathological history.

## DISCUSSION

Coronary artery disease is the most common cause of SCD, producing acute complications at this level (plaque rupture, coronary thrombosis) detected at

autopsy in 20-80% of cases. Sudden cardiac arrest in primary ventricular fibrillation is the culprit in more than 60% of patients. Myocardial scarring may be present in those individuals with anterior wall infarction, which produces electrical heterogeneity, thus favoring the occurrence of reentries and ultimately the emergence of ventricular tachycardia. These are the most frequent physiopathological mechanisms of SCA and, therefore, of SCD if recovery is not possible<sup>1,2</sup>.

Understanding the causes of SCA due to ventricular arrhythmia in patients with AMI is not an easy task. Some studies suggest that it increases with age and is greater in those over 75 years old. But there are research studies such as that of Kim *et al*<sup>12</sup> and Garberich *et al*<sup>13</sup> that found no difference between the two study groups in relation to this variable.

Sudden cardiac arrest is more common in males, although after age 65 the ratio drops to 2:1 or less. Gender's effect on post-SCA outcomes was addressed in a retrospective cohort study that enrolled 9651 patients of both sexes. Women were less likely than men to have ventricular fibrillation as a first rhythm (25% vs. 43%); they were also more likely to succeed in resuscitation and therefore had lower incidence of SCD<sup>1,14</sup>.

Smoking has been shown to induce physiological changes that predispose for the occurrence of SCA, namely, increased thrombogenesis, myocardial oxygen demand and decreased coronary blood flow. Smoking is associated with a 2 to 4-fold increase of the risk of SCA. In fact, patients with ICD who persist in smoking have 7 times more events of appropriate ICD shocks<sup>15</sup>.

Obesity is a factor that seems to influence SCD ratio. The Framingham study showed that the percentage of this type of death increased on a linear basis with the increase in body weight from only 39% in normal-weight patients to 70% in the case of those who are obese<sup>1</sup>.

In a study held at the Minneapolis Heart Institute - Abbott Northwestern Hospital between 2005-2014<sup>13</sup>, out of 4001 patients with ST-segment elevation AMI who had received medical assistance, 11.8% of the cases presented SCA prior to percutaneous coronary intervention. These patients were reported to have more cardiogenic shock, with predominance of anterior wall infarctions and lower LVEF, similar results to the ones obtained in our work. It must be noted that LVEF has low sensitivity in predicting sudden death. Less than 50% of patients with anterior wall infarction who present with SCA and eventu-

ally die, will have a LVEF below 35%; however, severe reduction in this parameter of ventricular function is the most widely used predictor of SCD, regardless of the presence of coronary artery disease or not<sup>16</sup>.

Several studies (MADIT<sup>17</sup>, MUSTT<sup>18</sup>, SCD-HeFT<sup>19</sup>) support the importance of this variable as a predictor of PCS and SCD, especially associated with the functional class according to the NYHA (New York Heart Association) classification. Left ventricular ejection fraction is the mainstay at almost every scale in determining ICD implantation, whether as primary or secondary prevention. Despite the fact that in this study there was a small percentage of patients whose LVEF could not be calculated during admission -which is a work limitation that may have influenced the final outcome of this variable's magnitude as a predictor of SCA- it was the second variable most associated with the appearance of SCA due to ventricular arrhythmia in patients who were admitted to hospital for AMI.

Chew *et al*<sup>20</sup> state that the decrease in LVEF early after AMI identifies patients at greater risk of complications; however, the changes that this variable may undergo in the coming weeks or months during follow-up provide more accurate information on prognosis. In this paper the authors evaluated early (2-7 days) and late (2-12 weeks) changes in LVEF following a first AMI, in three different studies (REFINE, CARISMA and ISAR) and split the patients into 3 groups according to changes in LVEF: group 1, no change; group 2, modest increase (1-9%) and group 3, high increase >10%. Patients with no improvement in LVEF had a high risk of death regardless of whether they received percutaneous revascularization or appropriate medication, hence most algorithms for determining ICD implantation take into account the value of LVEF within weeks after the acute coronary event<sup>20</sup>.

A number of electrocardiographic markers have been associated with the appearance of SCD, specifically as predictors of malignant ventricular arrhythmias, including heart rate variability, QRS duration, QT dispersion and prolongation, microvolt T-wave alternans, among others. According to Yodogawa *et al*<sup>21</sup> and Reinier *et al*<sup>22</sup>, the Risk Estimation Following Infarction, Noninvasive Evaluation (REFINE) study showed that the combination of heart rate assessment, microvolt T-wave alternans, and LVEF less than 50%, assessed 8 weeks after AMI, identified patients at risk of SCD, recovered or not, from ventricular arrhythmias. On the other hand, a

sum of ST-segment elevation/depression on the electrocardiogram greater than 15 mm is an indirect variable that may be related to a greater injury surface during AMI and a large area of myocardium at risk which conditions an increase in the ischemic substrate that may be related to greater electrical instability and higher predisposition to sudden ventricular arrhythmias<sup>21,22</sup>.

The higher frequency of cardiogenic shock and high mortality in patients with SCA due to ventricular arrhythmias has been demonstrated in several studies. Garberich *et al*<sup>13</sup> found that 40% of patients with ST-segment elevation AMI and SCA presented cardiogenic shock, in addition to high hospital mortality (29.7%) compared to patients who did not present SCA (2.8%). Our work yielded similar results; however, both, shock frequency and mortality were higher than those found by these authors. Our judgement is based on the fact that there are two aspects to be borne that may explain these results. Firstly, the sample in the study by Garberich *et al*<sup>13</sup> was considerably larger, as data from 4001 patients were analyzed; and secondly –and far more important– percutaneous coronary intervention was performed on the artery responsible for the AMI in 95.6% of cases in that study, with an average door-balloon time of 124 minutes, excellent! A large number of studies agree that early and vigorous revascularization treatments have made it possible to reduce cardiogenic shock, ventricular arrhythmias and, therefore, mortality. The occurrence of this shock represented the main risk factor for the appearance of SCA due to ventricular arrhythmia in our research<sup>13, 23</sup>.

## CONCLUSIONS

Cardiogenic shock, LVEF <35%, creatine kinase >1200 IU, obesity, smoking, having ST-segment elevation/depression on the electrocardiogram >15 mm and anterior wall infarction, were predictors of SCA due to ventricular arrhythmia.

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