Association between Angiotensin-Converting Enzyme Inhibitors and Troponin in Acute Coronary Syndrome

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Abstract

Background: **Cardiovascular disease is the leading cause of mortality in the western world and its treatment should be optimized to decrease severe adverse events.**

Objective: **To determine the effect of previous use of angiotensin-converting enzyme inhibitors on cardiac troponin I measurement in patients with Non-ST-Elevation Myocardial Infarction (NSTE-ACS) and evaluate clinical outcomes at 180 days.**

Methods: **Prospective, observational study, carried out in a tertiary center, in patients with Non-ST-Elevation Myocardial Infarction (NSTE-ACS). Clinical, electrocardiographic and laboratory variables were analyzed, with emphasis on previous use of angiotensin-converting enzyme inhibitors and cardiac troponin I. The Pearson chi-square tests (Pereira) or Fisher's exact test (Armitage) were used, as well as the non-parametric Mann-Whitney's test. Variables with significance levels of <10% were submitted to multiple logistic regression model.**

Results: **A total of 457 patients with a mean age of 62.1 years, of whom 63.7% were males, were included. Risk factors such as hypertension (85.3%) and dyslipidemia (75.9%) were the most prevalent, with 35% of diabetics. In the evaluation of events at 180 days, there were 28 deaths (6.2%). The statistical analysis showed that the variables that interfered with troponin elevation (> 0.5 ng / mL) were high blood glucose at admission (p = 0.0034) and ST-segment depression ≥ 0.5 mm in one or more leads (p = 0.0016). The use of angiotensin-converting inhibitors prior to hospitalization was associated with troponin ≤ 0.5 ng / mL (p = 0.0482). The C-statistics for this model was 0.77.**

Conclusion: **This study showed a correlation between prior use of angiotensin-converting enzyme inhibitors and reduction in the myocardial necrosis marker troponin I in patients admitted for Non-ST-Elevation Myocardial Infarction (NSTE-ACS). However, there are no data available yet to state that this reduction could lead to fewer severe clinical events such as death and re-infarction at 180 days. (Arq Bras Cardiol. 2014; 103(6):513-520)**

Keywords: **Angiotensin-Converting Enzyme Inhibitors; Troponin; Acute Coronary Syndrome.**

Introduction

Recent records have shown that approximately 1 million individuals are hospitalized in the United States due to Non–ST-segment elevation acute coronary syndrome (NSTE-ACS)[1,2] and an increase in its prevalence has been observed, when compared to ST-segment elevation acute coronary syndrome (STE-ACS)[3], along with the increased use of medications such as beta-blockers, Angiotensin-Converting Enzyme (ACE) inhibitors, angiotensin receptor II-blockers, thienopyridines and statins[3] - all associated with the use of troponin as a marker of myocardial necrosis[4].

The elevation in this biomarker increases the risk of death and re-infarction in the first six months, when compared to troponin-negative patients[5-10]. Thus, the rationale for this study was based on the fact that the reduction in cardiac troponin I in patients with NSTE-ACS could provide a modulation of the renin-angiotensin-aldosterone system (RAAS), preventing the deleterious actions of angiotensin II on myocardial ischemia, such as cardiac hypertrophy and dilation, coronary vasoconstriction, increased oxidation of Low-Density Lipoproteins (LDL) cholesterol, stimulus for PAI-1 release, among others[11], which may be alleviated by the use of ACE inhibitors, of which benefits have been demonstrated[12-14].

Methods

This is a prospective, observational study carried out in a tertiary center from September 8, 2009 to October 10, 2010, in patients with a diagnosis of NSTE-ACS, with a minimum age of 18 years. Patients with ST-segment elevation were excluded, as well as those with confounding ECG changes, such as atrial fibrillation, definitive pacemaker and left bundle branch block, or refusal to participate in the study.

All patients included in the study signed the free and informed consent form. All participants answered a questionnaire that included their personal references, personal pathological antecedents and previous use of medications.

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Laboratory measurements of glucose, hemoglobin, hematocrit, leukocytes, creatinine, potassium and cardiac troponin I were performed at admission. Electrocardiographic changes, such as ST-segment depression when ≥ 0.5 mm in at least two contiguous leads or > 0.5 mm in one lead, in both, except aVR, were analyzed. We also analyzed the inversion of T waves, with amplitude ≥ 1.0 mm in two or more contiguous leads, except aVR. Inpatients were followed until a clinical outcome occurred or until discharge; after that, they were reassessed by telephone contact or by medical record for clinical outcomes at 180 days.

Regarding the statistical methods, descriptive statistics of absolute (n) and relative (%) frequencies were used for qualitative measures, whereas summary statistics of mean, median, standard deviation (SD) and 25th and 75th percentiles (interquartile range) were used for quantitative variables. Associations between qualitative measures and the groups were carried out as follows: positive (> 0.5 ng/mL) and negative troponin (≤ 0.5 ng/mL) and the use and non-use of ACE inhibitors before hospital admission were assessed by Pearson's chi-square[15] or Fisher's exact test[16].

The nonparametric Mann-Whitney test[17] was applied to compare the quantitative measures between the two groups, due to non-normality of data

The variables for the logistic regression model were selected among those that has at least 70% of the observations (n ≥ 319), with absolute frequency of at least five occurrences per category, when qualitative measure, with a significance level < 15% (p < 0.15) in the two-dimensional analysis (univariate), and those which the researcher believed to be of clinical relevance for the assessed outcomes: Systemic Arterial Hypertension (SAH); dyslipidemia; unstable angina (UA); Acute Myocardial Infarction (AMI); prior Coronary Artery Bypass Surgery (CABG); congestive heart failure (CHF); cerebrovascular accident (CVA); typical pain on admission; creatinine and glycemia on admission; medications prior to admission (acetylsalicylic acid - aspirin, beta-blockers, statins, ACE inhibitors); and ST segment depression > 0.5 mm.

The stepwise backward method selected the variables for the final model. The results are shown as odds ratio (OR) and 95% Confidence Interval (95%CI) and descriptive level (p value). For the final model in the logistic regression analysis, only the variables with a significance level < 10% (p < 0.10) were maintained. The fit of the models was evaluated using C statistics (area under the Receiver Operating Characteristic curve - ROC)[18]. The level of significance was set at 5%. The software used was the Statistical Package for Social Sciences (SPSS), release 19.

Results

A total of 457 patients hospitalized with NSTE-ACS were included, of which 288 (63.0%) had UA and 169 (37.0%) non-ST-elevation myocardial infarction (NSTEMI). Among the study population, 291 were males (63.7%) with a mean age of 62.17 years (± 11.04), 390 patients (85.3%) had SAH, 347 (75.9%) had dyslipidemia and 160 (35.0%) were diabetics, in addition to other conditions (Table 1). Regarding medications, 242 patients (53.3%) received ACE inhibitors, 337 (73.9%) AAS, 289 (63.4%) received beta-blockers and other medications (Table 2).

Table 1 – Baseline characteristics of the study population

Characteristic	Patients (n = 457) n (%)
Age, mean (SD)	62.2 (11.0)
Male gender	291 (63.7)
Personal history	
Systemic arterial hypertension	390 (85.3)
Dyslipidemia	347 (75.9)
Congestive heart failure – (NYHA ≥ II)	294 (64.3)
Previous acute coronary syndrome	275 (60.2
Previous revascularization	226 (49.5)
Only PCI	118 (25.8)
Only CABG	67 (14.7)
Diabetes mellitus	160 (35.0)
Family history of coronary artery disease	172 (37.6)
Smoking	110 (24.1)
Cerebrovascular accident	30 (6.6)
Coronary artery disease ≥ 50%	287 (62.8)
Presentation at admission	
Symptoms	
Typical chest pain	391 (85.6)
Atypical chest pain	66 (14.4)
Dyspnea*	14 (3.1)
Syncope*	4 (0.9)
Precordial pain at admission	281 (61.5)
Number of pain episodes in the last 48 hours	
Two or more episodes	253 (55.4)
One episode	204 (44.6)
Hemodynamic instability at admission**	8 (1.8)
Heart rate (bpm)	75.86 (± 16.56)
Systolic blood pressure (mmHg)	139.61 (± 28.24)
Diastolic blood pressure (mmHg)	82.69 (± 17.07)
Congestive heart failure - Killip-Kimball class	
I	451 (98.7)
II	2 (0.4)
III	3 (0.7)
IV	1 (0.2)
Diagnosis	
NSTEMI	169 (37.0)
Unstable Angina III B	287 (62.8)
Unstable Angina III C	1 (0.2)

** Symptoms associated to chest pain; ** systolic blood pressure < 90 mmHg. SD: standard deviation; NYHA: New York Heart Association classification; CABG: coronary artery bypass grafting; PCI: percutaneous coronary intervention; NSTEMI: non-ST-elevation myocardial infarction*

Table 2 – Medications used prior to hospital admission

Medication	Patients (n = 457) n (%)
Acetylsalicylic acid	337 (73.9)
Beta-blockers	289 (63.4)
Statins	278 (61.0)
Angiotensin-converting enzyme inhibitors	242 (53.3)
Nitrates	174 (38.2)
Diuretics	164 (36.1)
Calcium-channel blockers	89 (19.6)
Clopidogrel	32 (7.1)
Digitalis	14 (3.1)
Others	276 (60.4)

Table 3 shows the demographic characteristics taking into account the previous use of ACE inhibitors or not, demonstrating that among the users, there were more hypertensive individuals (91.3% vs. 78.6%, p <0.001), with a previous history of congestive heart failure (CHF), according to the New York Heart Association (NYHA) ≥ II FC (69.0% vs. 59.1%, p <0.027), of ACS (69.0% vs. 50.2%, p <0.001) and revascularization procedures (55.8% vs. 42.3%, p = 0.004), among others.

Due to the relevance of troponin in this study, the patients' demographic characteristics were observed, taking into account the cutoff value of this biomarker. Thus, it was observed that the troponin-positive patients were older (63.9% vs. 61.2%, p = 0.006), had more previous myocardial revascularization procedures (49.7% vs. 49.3% p = 0.034) and had ST-segment depression ≥ 0.5 mm in one or more leads, among others (Table 3).

Of the 457 patients in the study, 319 (69.8%) underwent coronary angiography, of which 103 (32.3%) were submitted to percutaneous coronary intervention (PCI) and 43 (13.5%) to CABG.

According to the TIMI risk score[19], 124 patients were classified as low risk (27.2%), 216 (47.2%) as intermediate risk, and 117 (25.6%) as high risk, showing that this is a population at high risk for cardiovascular events.

During hospitalization, 51 patients (11.2%) had complications, of which ten (19.6%) died, ten (19.6%) developed CHF, six (11.8%) had significant nonfatal cardiac arrhythmias, six (11.8%) had cardiogenic shock (CS), five (9.8) had re-infarction, four (7.8%) AMI, three (5.9%) acute pulmonary edema, three (5.9%) reversed cardiac arrest, two (3.9%) recurrent or refractory angina and two (3.9%), cerebrovascular accident (CVA). The cause of death was CS, AMI and septic shock (SS) in five, three and two patients, respectively. Within 180 days, the primary outcome measures were death from all causes and re-infarction, with a predominance of CS in 11 patients (39.2%) and AMI in eight patients (28.5%).

A statistical analysis model was created according to which troponin levels were compared (> 0.5 ng/dL vs. ≤ 0.5 ng/dL), patients who used ACE inhibitors prior to hospitalization had, a negative beta coefficient (-0.520) and OR = 0.59, 95% CI = 0.35 to 0.99, with p = 0.048.

Discussion

This prospective study carried out in patients with NSTE-ACS demonstrated an association between prior use of ACE inhibitors and reduction in the levels of myocardial necrosis biomarker cardiac troponin I.

Previous studies have demonstrated the role of ACE inhibitors in preventing cardiac events in patients at high cardiovascular risk, with consequent reduction in morbidity and mortality[12-14].

Troponin is considered the most sensitive and specific marker of myocardial necrosis for the diagnosis of AMI[20], although this marker can be elevated in other clinical situations and thus hinder the differential diagnosis of patients with chest pain that seek emergency care[21]. In the 1990s, troponin caused a paradigm shift in the diagnosis[22] and prognostic implications in these patients when evaluated in the short, medium and long term[23-28].

In this sense, this study sought to correlate the use of ACE inhibitors with the release of cardiac troponin I in patients with NSTE-ACS, of which modulation of the RAAS would attenuate the harmful effects associated with excessive production of angiotensin II, both local and systemic. The several benefits would cover a broad spectrum, such as improved coronary and peripheral vasodilator activity, reduction of platelet activity, increased endogenous fibrinolysis and reduced inflammatory activity, among others, providing what might be called a cardioprotective effect[29-31].

The first report on this subject occurred in a 2001 study by Kennon et al[32], who evaluated the correlation between the use of ACE inhibitors and the reduction of troponin in patients with NSTE-ACS, associated with a genetic component (gene polymorphisms: DD, II, DI or ID). The population studied had demographic characteristics that were different from those observed in our study, as although there was a considerable percentage of previous AMI and diabetes, there were low rates of medication use prior to hospitalization, such as aspirin, beta-blockers, ACE inhibitors and statins, which is currently unacceptable for patients with this degree of cardiovascular risk.

Kennon et al[32] concluded that the use of ACE inhibitors reduced troponin levels by approximately 75% and that there might be an association between this reduction and the beneficial effects, both on vascular reactivity and the coagulation system. The genetic assessment showed no association between troponin levels and gene polymorphism (similar in the three groups). In contrast, patients in our study have higher cardiovascular risk, with history of prior ACS (60.2% vs. 27.3%); previous myocardial infarction (39.8% vs. 35.9%); previous revascularization procedures (49.5% vs. 16.5%); and diabetes (35.0% vs. 23.2%).

Medications were also used in higher proportion prior to hospital admission, such as ASA (73.9% vs. 55.1%),

Table 3 – Baseline characteristics according to troponin values

Characteristic		TnI ≤ 0.5 ng/mL Patients (n = 288) n (%)	TnI > 0.5 ng/mL Patients (n = 169) n (%)	Total (n = 457)	p value
Age					
Mean (SD)		61.2 (10.6)	63.9 (11.6)	62.2	0.006
Median (IIQ)		60.0 (53;69)	64.0 (56;72)	-	
Male gender		176 (61.1)	115 (68.0)	291	0.137
Personal history					
Dyslipidemia		227 (78.8)	120 (71.0)	347	0.059
Diabetes mellitus		110 (38.2)	50 (29.6)	160	0.063
SAH		252 (87.5)	138 (81.7)	390	0.088
Smoking		69 (24.0)	41 (24.3)	110	0.942
Family history of CAD		113 (39.2)	59 (34.9)	172	0.357
Previous ACS		180 (62.5)	95 (56.2)	275	0.185
Previous revascularization procedures		142 (49.3)	84 (49.7)	226	0.034
Only PCI		78 (27.1)	40 (23.7)	118	0.487
Only CABG		37 (12.8)	30 (17.8)	67	0.196
Cerebrovascular accident		24 (8.3)	6 (3.6)	30	0.046
CHF (NYHA ≥ II)		199 (69.1)	95 (56.2)	294	0.006
Coronary artery disease ≥ 50%		183 (63.5)	104 (61.5)	287	0.669
Clinical presentation at admission					
Symptoms					
Typical chest pain		254 (88.2)	137 (81.1)	391	0.036
Two or more pain episodes in the last 48 hours		169 (58.7)	84 (49.7)	253	0.062
CHF – Killip-Kimball class ≥ II		0	6 (3.6)	6	0.002F
Treatment performed					
Clinical		210 (72.9)	89 (52.7)	299	< 0.001
PCI		56 (19.4)	51 (30.2)	107	0.012
CABG		22 (7.6)	29 (17.2)	51	0.003
Medications at admission					
Acetylsalicylic acid		222 (77.1)	115 (68.0)	337	0.043
Beta-blockers		193 (67.0)	96 (56.8)	289	0.035
Statins		190 (66.0)	88 (52.1)	278	0.004
ACEI		160 (55.6)	82 (48.5)	242	0.146
Calcium-channel blockers		53 (18.4)	36 (21.3)	89	0.442
ECG alterations					
ST-segment depression ≥ 0.5 mm (one or more leads)		45 (15.6)	54 (32)	99	< 0.001
T –wave inversion ≥ 1.0 mm		97 (33.7)	59 (34.9)	156	0.789
Biochemical analysis					
Glycemia	Mean (SD)	121.4 (54.6)	141.9 (71.7)	-	0.008
	Median (IQR)	105.5 (89;132)	116.0 (95;165)	-	
Creatinine	Mean (SD)	1.13 (0.42)	1.25 (0.59)	-	0.048
	Median (IQR)	1.0 (0.9;1.3)	1.1 (0.9;1.4)	-	

TnI: troponin I; SD: standard deviation; IQR: interquartile range; SAH: systemic arterial hypertension; CAD: coronary artery disease; ACS: acute coronary syndrome; CABG: coronary-artery bypass grafting; PCI: percutaneous coronary intervention; CHF: congestive heart failure; NYHA: New York Heart Association classification; ACEI: angiotensin-converting enzyme inhibitors; ECG: electrocardiogram.

beta-blockers (63.4% vs. 23.1%), ACE inhibitors (53.3% vs. 14.8%) and statins (61.0% vs. 15.6%). Therefore, considering the most recent and national[33] and North-American[2] guidelines, patients with increased cardiovascular risk should be under more intensive medical therapy.

At the end of 2009, dos Santos et al[34] developed the first national risk score for patients with NSTE-ACS. In this study, it was observed that the use of ACE inhibitors prior to hospitalization conferred "protection" to patients, which was confirmed by the presence of this variable in the final risk score model. The population of this research came from a database that was similar to that of the present study, both regarding the degree of cardiovascular risk and the use of medications prior to admission. However, those authors did not intend to correlate the use of ACE inhibitors and the presence of the biomarker cardiac troponin I and its possible clinical events during hospitalization, as well as in the medium and long term.

The benefits of using ACE inhibitors in patients with NSTE-ACS and STE-ACS have been widely confirmed in large clinical trials, especially in those with large infarcts and consequent left ventricular dysfunction[12-14]. Despite the obvious differences in their physiopathology, interesting results were observed in a retrospective study published in 2010 in patients with STE-ACS carried out in a single center in the United States, with 511 patients enrolled between 2004 and 2008[35]. Patients with a history of cardiovascular disease, either in the coronary, cerebral or peripheral beds, as well as those with diabetes were excluded; therefore, the study was on the margin of the exclusion criteria found in the real world. Nevertheless, it was the first study that attempted to correlate the benefit of ACEI prior to an STE-ACS event. Its results confirmed that patients taking ACE inhibitors, when compared to non-users, had lower levels of troponin at admission (79.8 ng/dL vs. 120.0 ng/dL, $p = 0.016$). From a medical viewpoint, this beneficial effect remained, regardless of the concomitant use of aspirin and statins.

In 2010, in a study carried out in Sweden, 87,241 patients that did not take ACE inhibitors prior to hospitalization (40,549 with STE-ACS and 46,692 with NSTE-ACS) were assessed in the period between 1995 and 2005[36]. At hospital discharge, ACE inhibitors were prescribed to 36.5 % of the patients, whose clinical outcomes were evaluated up to a one-year period. The results corroborate previous studies[12-14], with a mortality reduction of 24% in the period ($p < 0.001$). The risk of death at 1 year was lower in patients receiving ACE inhibitors (10.6% vs.12.1%, $p < 0.001$). Limitations of this study were not-randomized registration data, lack of control of comorbidities and treatments used (type and dose of ACE inhibitors), and the fact that clinical outcome evaluation was performed only in patients who survived hospital discharge.

In comparison, our study was prospective and 242 (53.3%) of the patients admitted used ACE inhibitors. Of a total of 28 deaths in 180 days, ten (6.2%) occurred during hospitalization, being higher in ACE inhibitor users (p = NS), in the elderly ($p < 0.001$), those with a history of CHF (NYHA ≥ II) prior to hospitalization ($p = 0.042$) and those with higher creatinine ($p < 0.001$). As limitations, we did not perform left ventricular dysfunction measurement and did not control comorbidities.

In 2011, researchers from the GRACE, GRACE2 and CANRACE studies performed a retrospective analysis from 1999 to 2008 with 13,632 patients[37], correlating prior ACE inhibitors to hospitalization from STE-ACS (3,817 patients) and NSTE-ACS (9,815 patients), aiming to assess the proportion of in-hospital major clinical events.

Users of ACE inhibitors were compared with nonusers by analyzing demographic characteristics such as medical history, current medications, clinical presentation, electrocardiographic changes on admission and laboratory tests, including troponin and creatine kinase MB isoenzyme (CK-MB). When compared to our study, also high-risk, and taking into account the period when data were obtained, the treatment did not follow the most current guidelines for these ischemic syndromes and approximately 20% of patients with ACS and diabetes mellitus did not receive ACE inhibitors. As for the results, users of ACE inhibitors, when compared to non-users, had lower release of biomarkers within the first 24 hours (76.3% vs. 67.3%, $p < 0.001$). Hospital mortality was higher in ACE inhibitor users ($p = 0.012$), but this became non-significant when the model was adjusted for other prognostic factors and use of other medications. The authors concluded that the prior use of ACE inhibitors is not independently associated with reduced in-hospital major clinical events, such as death or re-infarction after NSTE-ACS or STE-ACS. There was underutilization of antiplatelet and anticoagulant agents, as well as of coronary angiographies and percutaneous coronary interventions in patients using ACE inhibitors and in many patients with renal dysfunction, leading to a smaller number of hemodynamic studies and consequently, to the reduction of percutaneous coronary intervention.

Factors such as non-randomization, exclusion of patients who did not survive hospitalization, incomplete data on the use of other antagonists of the renin-angiotensin-aldosterone system, non-reported dose of medications, duration of prior treatment with ACE inhibitors, adherence to medication and peak myocardial necrosis marker, which was not established by the study protocol (and may underestimate its true magnitude and diagnosis of re-infarction) may have affected the results.

In comparison, our study was prospective and observational, did not exclude any patient during the specified period, regardless of age, creatinine level or any other demographic characteristic, and included a population at high cardiovascular risk (Table 1), fully medicated, according to the national[33] and North-American guidelines[2] (Table 2).

Historically, it is known that patients with renal dysfunction and the elderly are often excluded from clinical trials, as they increase the risk of severe clinical events and difficulties in therapeutic management, thus interfering with its optimization, as well as the fact that percutaneous treatment is not contemplated when indicated. In this study, no patient was excluded during the stipulated period and the best approach was used on admission, either percutaneous or surgical, always in agreement with the medical staff, patients and their families. However, patients with renal dysfunction and the elderly showed a significant increase in mortality at 180 days ($p < 0.001$).

The present study was designed in an attempt to demonstrate whether there would be a reduction in myocardial necrosis marker troponin I associated with the use of ACE inhibitors, taking into account other variables that could interfere with this biomarker's release.

In the proposed statistical model, when troponin levels were compared (> 0.5 ng / dL vs. ≤ 0.5 ng / dL) in patients who used ACE inhibitors, a "protective effect" was observed, as the statistical analysis of final model showed a negative beta coefficient (- 0.520) and OR = 0.59, 95%CI = 0.35 to 0.99, p = 0.048) (Table 4). Considering these results, the correlation between the use of ACE inhibitors and lower release of troponin in patients with NSTE-ACS was evident.

However, this correlation has not translated into a reduction in severe clinical events such as death and re (infarction) at 180 days. In our series, in-hospital mortality of 2.2% and mortality at 180 days of 6.2% are considered low; thus, it is observed that the use of ACE inhibitors prior to hospitalization was not associated with the decrease in death rates.

Some authors observed that the beneficial clinical effects from the use of ACE inhibitors became apparent only after about 1 year[38]. In this research, we used a minimum of 30 days prior to admission as time of medication use for inclusion criteria in the study.

Another matter concerns the differences between ACEIs because, in our study, these drugs were considered as belonging to a "class". A Canadian study evaluating different classes of ACE inhibitors used after an AMI, used for a one-year period, showed benefits in survival according to the ACE inhibitor used in patients aged > 65 years[39] However, the three major groups of ACE inhibitors show important pharmacological differences due to their chemical structure, and thus it can lead to different clinical effects[40].

However, despite this correlation of cardiac troponin I with the use of ACE inhibitors, many demographic, clinical and laboratory factors are implicated in this broad-spectrum population with NSTE-ACS to significantly correlate it with the reduction in serious clinical events, such as and death re-infarction within a period of 180 days.

The study of other populations, different from the one evaluated by this study, could lead to other results, but at present there is no record in the medical literature of another study that includes a population with such significant cardiovascular risk or using such comprehensive therapy, widely recommended by the main worldwide renowned cardiology societies and that demonstrates such a statistically significant association between the use of ACE inhibitors and necrosis marker tropinin I in patients with NSTE-ACS.

However, when interpreting this research, in spite of the correlation of cardiac troponin I with the use of ACE inhibitors, many demographic, clinical and laboratory factors would be implicated in this broad-spectrum population with NSTE-ACS, to correlate it significantly with the reduction in severe clinical events such as death and re (infarction) at 180 days.

Conclusion

This study demonstrated that patients hospitalized for NSTE-ACS that had previously used ACE inhibitors showed decreased levels of the myocardial necrosis marker troponin I. However, there are no data available yet to affirm that this reduction could lead to a smaller number of severe clinical events, such as death and re-infarction at 180 days.

Author contributions

Conception and design of the research, Acquisition of data and Critical revision of the manuscript for intellectual content: Minuzzo L, Santos ES, Timerman A; Analysis and interpretation of the data, Statistical analysis, Obtaining financing and Writing of the manuscript: Minuzzo L, Timerman A.

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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Table 4 – Multiple logistic regression analysis for troponin > 0.5 ng / mL

Analyzed variable	Effect	SE	OR	95%CI	p value
Typical pain at admission	-0.878	0.375	0.416	0.199;0.867]	0.0193
Glycemia at admission	0.008	0.003	1.008	1.003 - 1.013	0.0034
Clinical treatment	-	-	-	-	0.0002
PCI	1.089	0.325	2.973	1.572 - 5.622	0.0008
CABG	1.400	0.413	4.055	1.806 - 9.103	0.0007
Previous ACEI use at admission	-0.520	0.263	0.594	0.355 - 0.996	0.0482
ST-segment depression ≥ 0.5 mm	0.971	0.307	2.641	1.447 - 4.818	0.0016

SE: standard error; OR: odds ratio; 95%CI: 95% confidence interval; PCI: percutaneous coronary intervention; CABG: coronary artery bypass grafting; ACEI: angiotensin-converting enzyme inhibitors.

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