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RESEARCH ARTICLE

The impact of admission procalcitonin on prognosis in acute coronary syndromes: a pilot study

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Abstract

Background: Available evidence on the prognostic role of procalcitonin levels in acute coronary syndromes (ACS) is so far controversial.

Aims: To evaluate the association between procalcitonin, major cardiovascular events (MACE) and total mortality in acute coronary syndromes.

Methods: Procalcitonin levels were measured in 247 patients admitted to our Intensive Cardiac Care Unit (ICCU) with ACS. Three subgroups were considered according to procalcitonin levels.

Results: At Cox regression analysis, procalcitonin levels were both an unadjusted and an adjusted predictor (corrected for diagnosis and TnI) of intra-ICCU mortality and of 1-year follow-up MACE and total mortality.

Conclusions: In ACS, admission procalcitonin values identify a "higher risk" group of patients for short and long-term mortality.

Keywords: Procalcitonin, cardiogenic shock, acute coronary syndromes, prognosis, mortality

Introduction

The clinical significance of serum procalcitonin (PCT) levels has been recently investigated in acute coronary syndromes (ACS; Buratti et al. 2001; Geppert et al. 2003; Kafkas et al. 2008; Remskar et al. 2002), but available evidence is so far controversial (Biasucci et al. 2009; Senturk et al. 2011)

Data on the prognostic role of PCT in ACS patients are few and controversial. Senturk et al. failed to find any correlation between PCT early prognosis in patients with STEMI, acute myocardial infarction without ST elevation (NSTEMI) and unstable angina (UA; Senturk et al. 2007). Conversely, Ataoğlu et al. observed, in a small group of ACS patients, that PCT levels were higher in patients who died during hospital stay and in those who died at 6-month follow-up (Ataoğlu et al. 2010).

Up to now, no studies are available on a more heterogeneous ACS population, which includes a wide range of inflammatory burden, from unstable angina to cardiogenic shock complicating STEMI.

Thus, the present investigation was aimed at determine whether admission PCT levels could be an independent predictor of total mortality and MACE both at short-term (during Intensive Cardiac Care Unit or ICCU staying), and at long-term (that is 1-year follow-up, median 6.5 months), in 247 patients with ACS consecutively admitted to our intensive Care Unit.

Methods

Study population

This was a retrospective, non-randomized study which included 247 consecutive patients (mean age 69.3 years, 170 males and 77 females) with acute coronary syndrome admitted to our 12-bed Intensive Cardiac Care Unit (ICCU) in Florence, a tertiary center, from 1st January 2008 to 31st January 2011. To be eligible for the present study, all patients had to be free of infection at the time of blood sampling, as evidenced by both clinical and microbiological examinations, including

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urinary and blood cultures, and microbiological examinations of tracheal aspirates in mechanically ventilated patients.

In our population, different subgroups were considered according to the admission diagnosis (Hamm et al. 2011) as follows (Figure 1): (i) non-ST-elevation myocardial infarction or unstable angina (NSTEMI/UA, $n = 112$); (ii) uncomplicated ST-elevation acute myocardial infarction (STEMI, Killip class I, $n = 95$; Thygesen et al. 2007); (iii) cardiogenic shock complicating STEMI (CS, $n = 40$; Califf et al. 1994; Hochman et al. 2003; Webb et al. 2003; Valente et al. 2008). All patients were submitted to coronary angiography and the majority (91.5%) underwent coronary revascularization, either mechanical (percutaneous coronary angioplasty—PCI—with or without stent implantation, 88.3%) or surgical (coronary artery bypass graft or CABG, 3.2%).

Transthoracic two-dimensional echocardiography was performed on admission to evaluate left ventricular ejection fraction (LVEF).

Fasting blood samples were obtained, on the day after ICCU admission, for the measurements of the following variables: glycemia (g/L; Lazzeri et al. 2011a), insulin (mU/L), glycated hemoglobin (HbA1c, %), uric acid (mg/dL; Lazzeri et al. 2010b), alanine transaminase (ALT, IU/L), aspartate transaminase (AST, IU/L) and gamma-glutamyl transpeptidase (GGT, IU/L) (Lazzeri et al 2010 c, Lazzeri et al 2011b), erythrocyte sedimentation rate (ESR, mm/h), creatinine (mg/dL) to calculate glomerular filtration rate (eGFR, mL/min/1.73 m^2), microalbuminuria (μg/minute, in a 24-h urine collection; Lazzeri et al. 2010a), leucocytes count (10^3/μl), C-reactive protein (CRP, mg/dl), N-terminal proBNP (NT-proBNP, pg/ml; Valente et al. 2009) and serum procalcitonin measurements (sPCT). sPCT was measured with a novel highly sensitive commercial assay (BRAHMS PCT sensitive LIA; B.R.A.H.M.S. AG, Hennigsdorf, Germany), with an interassay coefficient of variability of 10%–20%, lower limit of detection in our laboratory: 0.05 ng/mL (Picariello et al. 2009).

Peak cardiac troponin I (TnI, ng/mL; Lazzeri et al., 2008) and peak glycemia levels (mg/dL) were also recorded.

Homeostatic model assessment (HOMA) index was calculated according to the following formula: {[fasting insulin (microU/mL)]×[fasting glucose (mmol/L)]}/22.5. Subjects whose values exceeded the sex-specific 75thpercentile (i.e. 1.80 for women and 2.12 for men) were considered to have insulin resistance (HOMA-IR; Lazzeri et al. 2009).

According to PCT serum levels, the following subgroups were considered: (i) Group "A" ("normal") had sPCT levels ≤ 0.05 ng/mL, that is undetectable levels ($n = 153$, 61.9% of total population); (ii) Group "B" ("intermediate") had sPCT levels between 0.06 and 0.49 ng/mL ($n = 52$, 21.1%); (iii) Group "C" ("high") had sPCT ≥ 0.5 ng/mL ($n = 42$, 17.0%).

Among discharged patients ($n = 222$), 87.8% ($n = 195$) were followed-up and major cardiovascular adverse

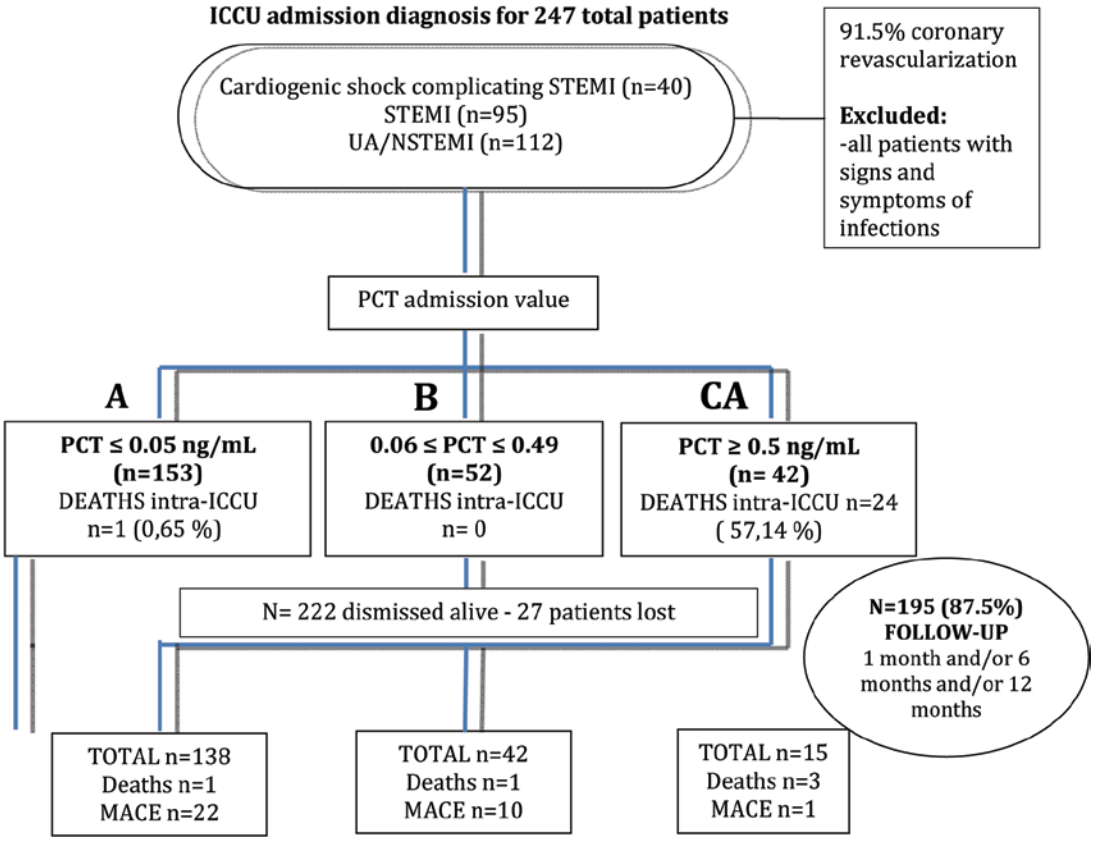

Figure 1. Study design.

events (MACE) were recorded. MACE were defined as one or more between acute myocardial infarction, ischemic stroke, coronary arterial occlusion and all cause death (Kelly et al. 2010).

The study protocol was in accordance with the declaration of Helsinki and approved by the local Ethic Committee.

Statistical analysis

Statistical analysis was conducted with SPSS 17.0 (SPSS inc, Chicago, ILL). Normally distributed variables are expressed as mean ± standard deviation (SD) and analyzed with ANOVA. Non-normally distributed variables are reported as medians (25th–75th percentile) and analyzed by means of Kruskal-Wallis H-test for trend. Discrete variables are reported as frequencies (percentages) and analyzed by means of chi-square test. Post-hoc (that is, each group vs. each other) comparisons were performed considering standardized residuals for χ^2 (Z score >| 1.96 |:$p < 0.05$; discrete variables) and with Bonferroni test for continuous data.

After assessment of risk proportionality, a Cox regression analysis was conducted in order to assess whether sPCT levels were predictors of intra-ICCU death, taking into account days of ICCU staying. Long-term survival with respect to sPCT levels (coded as stated above) was analyzed with Cox regression analysis, both unadjusted and adjusted for diagnosis and TnI, for a composite end point (MACE and total mortality).

In all multivariate analyses, candidate variables were chosen among those being strongly different at univariate analysis in relation to outcome; number of predictors was carefully chosen to avoid overfitting of models. Diagnosis was coded as follows: 1-NSTEMI/UA; 2-STEMI; 3-CS; for procalcitonin levels the coding was: 1-Group A; 2-Group B; 3-Group C, peak TnI values were coded as increments of 10 ng/mL. In all cases, a two-tailed p value <0.05 was considered statistically significant.

Results

Table 1 depicts demographic data and baseline characteristics according to admission diagnosis in our series. In-hospital mortality rate was higher in CS group (55%, $p < 0.001$) than in STEMI (2.1%) and NSTEMI/UA (0.9%).

As shown in Table 2, the highest percentage of patients in Group C was observed in CS, whereas patients with NSTEMI/UA showed the lowest PCT values (Group A). Group C showed the highest values of peak glycemia ($p < 0.001$), HOMA index positivity ($p = 0.013$), CRP positivity ($p < 0.001$), NT-proBNP ($p < 0.001$), ALT, AST ($p < 0.001$ and $p < 0.001$, respectively) and GGT ($p < 0.001$) together with the lowest values of eGFR ($p < 0.001$).

At Cox regression analysis, sPCT level was both unadjusted (HR 12.49, 95% CI 3.46–45.10, $p < 0.001$) and adjusted predictor of intra-ICCU mortality (HR 8.24, 95%CI 2.02–33.66, $p = 0.003$, when corrected for diagnosis and TnI).

Follow-up [Median 6.0 months (interquartile range 1.9–12.0 months)].

Mortality rate at follow-up was 2.6% (5/195) and MACE were 16.9% (33/195).

At 1-year follow-up (Cox regression analysis), PCT was an unadjusted predictor for MACE and mortality (for each

Table 1. Demographic and baseline characteristics of our population according to admission diagnosis.

	Total ($n = 247$)	CS ($n = 40$; 16.2%)	STEMI ($n = 95$; 38.5%)	NSTEMI/UA ($n = 112$; 45.3%)	p
Age, years (mean ± SD)	69.3 ± 12.3	71.4 ± 12.0	67.6 ± 13.6	69.9 ± 12.2	0.229
Males/females (%)	170/77 (68.8/31.2)	29/11 (72.5/27.5)	62/33 (65.3/34.7)	79/33 (70.5/29.5)	0.617
Risk factors					
Body mass index, Kg/m^2 (mean ± SD)	26.5 ± 4.6	25.7 ± 4.8	27.1 ± 5.1	26.2 ± 4.0	0.208
Hypertension (%)	159 (64.4)	25 (62.5)	61 (64.2)	73 (65.2)	0.954
Current smoking (%)	69 (27.9)	5 (12.5) ‡	32 (33.7)	32 (28.6)‡	0.042
Diabetes mellitus II type (%)	63 (25.5)	12 (30.0)	23 (24.2)	28 (25.0)	0.769
Family history (%)	74 (30.0)	6 (15.0)	27 (28.4)	41 (36.6)	0.035
Dyslipidemia (%)	86 (34.8)	13 (32.5)	32 (33.7)	41 (36.6)	0.858
Comorbidities					
Chronic renal failure (%)	18 (7.3)	3 (7.5)	9 (9.5)	6 (5.4)	0.524
Previous POAD (%)	35 (14.2)	8 (20.0)	14 (14.7)	13 (11.6)	0.417
Previous acute myocardial infarction (%)	21 (8.5)	8 (20.0) ‡	7 (7.4)	6 (5.4) ‡	<0.001
COPD (%)	22 (8.9)	4 (12.5)	8 (8.4)	10 (8.9)	0.958
Admission LVEF, % (mean ± SD)	45.2 ± 11.6	29.0 ± 9.8 *,§	44.2 ± 8.9 *,†	50.5 ± 9.5 §,†	<0.001
In-hospital death (%)	25 (10.1)	22 (55.0) ‡	2 (2.1) ‡	1 (0.9) ‡	<0.001
Length of stay, days [median (IQR)]	5 (3–7)	7 (3-12) *,§	4 (4-6) *	4 (3-6) §	0.004

Note: Post-hoc test results (Bonferroni for normally distributed data; Kolmogorov-Smirnov for nonnormally distributed data): * = $p < 0.05$ CS vs. STEMI; § = $p < 0.05$ CS vs. NSTEMI/UA; † = $p < 0.05$ STEMI vs. NSTEMI/UA. ‡ = Z-test > |1.96|: $p < 0.05$

CS: cardiogenic shock; STEMI: ST-elevation myocardial infarction; UA: unstable angina; NSTEMI: non ST-elevation myocardial infarction; POAD: Periferic obstructive arterial disease; COPD: Chronic obstructive pulmonary disease; LVEF: left ventricle ejection fraction; IQR: interquartile range (25th–75th percentile); SD: standard deviation.

Table 2. Laboratory data of three subgroups according to PCT levels.

	Total ($n=247$)	Group A $n=153$ (61.9%)	Group B $n=52$ (21.1%)	Group C $n=42$ (17.0%)	p
CS (%)	40 (16.2%)	2 (1.3%) ‡	10 (19.2%)	28 (66.7%) ‡	
STEMI (%)	95 (38.5%)	61 (39.9%)	23 (44.2%)	11 (26.2%) ‡	<0.001
UA/NSTEMI (%)	112 (45.3%)	90 (58.8%) ‡	19 (36.5%)	3 (7.1%) ‡	
Admission glycemia, mg/dL [median (IQR)]	125 (101–158)	111 (97–138) *,§	146 (110–175) *	160 (126–227) §	<0.001
Peak Glycemia, mg/dL [median (IQR)]	148 (124–198)	136 (112–164) *,§	164 (137–213) *,†	217 (174–260) §,†	<0.001
Fasting insulin, mU/L [median (IQR)]	7.6 (4.2–15.7)	7.0 (4.5–13.6)	8.5 (4.6–25.6)	10.0 (3.5–23.9)	0.325
HOMA index positivity (%)	30 (13.6%)	12 (8.7%)	9 (18.8%)	9 (26.5%) ‡	0.013
Hb1ac > 6.5 % (%)	66 (29.1%)	37 (25.9%)	16 (34.0%)	13 (35.1%)	0.381
C-reactive protein positivity (%)	136 (56.7%)	59 (39.6%) ‡	40 (78.4%) ‡	37 (92.5%) ‡	<0.001
ERS, mm/h [median (IQR)]	20 (11–35)	16 (8–27) *,§	30 (19–47) *	36 (14–61) §	<0.001
Leucocytes, 10^{12}/L (mean ± SD)	10.5 ± 3.9	9.3 ± 3.2 *,§	12.2 ± 4.2 *	13.0 ± 4.0 §	<0.001
Microalbuminuria, μg/min [median (IQR)]	23.6 (9.2–57.2)	15.4 (6.8–40.2) *,§	34.7 (13.3–101.0) *	65.4 (23.5–207.7) §	<0.001
Uric acid, mg/dL (mean ± SD)	6.2 ± 2.1	5.8 ± 1.6 *,	6.6 ± 1.8 *,†	7.5 ± 3.0 †	<0.001
Troponin I peak, ng/mL [median (IQR)]	47.0 (5.6–151.5)	36.6 (2.2–81.0) *,§	94.7 (27.9–243.1) *	185.7 (47.9–414.7) §	<0.001
NT-proBNP, pg/mL [median (IQR)]	1410 (524–4585)	781 (287–1868) *,§	2958 (1248–7768) *,†	12592 (3235–27347) §,†	<0.001
eGFR, mL/min/1.73 m^2 (mean ± SD)	85.1 ± 31.3	95.2 ± 26.5 *,§	76.0 ± 32.9 *,†	59.8 ± 28.4 §,†	<0.001
Alanine transaminase, GPT, IU/L [median (IQR)]	32 (20–54)	27 (19–39) *,§	39 (19–60) *,†	72 (38–154) §,†	<0.001
Aspartate transaminase, GOT, IU/L [median (IQR)]	64 (29–148)	39 (26–82) *,§	110 (33–205) *,†	214 (102–466) §,†	<0.001
Gamma-glutamyl transpeptidase, IU/L [median (IQR)]	25 (14–43)	21 (13–39) §	28 (14–44) †	38 (22–77) §,†	<0.001

Note: Post-hoc test results (Bonferroni for normally distributed data; Kolmogorov-Smirnov for non-normally distributed data): * = $p<0.05$ Group A vs. B; § = $p<0.05$ Group A vs. C; † = $p<0.05$ Group B vs. C. ‡ = Z-test > |1.96|; $p<0.05$.
CS: cardiogenic shock; STEMI: ST-elevation myocardial infarction; UA: unstable angina; NSTEMI: non ST-elevation myocardial infarction; IQR: interquartile range (25th-75th percentile); HOMA: homeostatic model assessment; HbA1c: glycated hemoglobin; ESR: erythrocyte sedimentation rate; NT-proBNP: N-terminal pro brain natriuretic peptide; eGFR: estimated glomerular filtration rate.

category step, from Group A to Group C, HR 1.68; 95% CI 1.05–2.68; $p=0.030$). PCT remained a predictor for adverse events at follow-up when adjusted for admission diagnosis and TnI levels (Group B vs. Group A adjusted HR 2.12; 95% CI 1.00–4.50; $p=0.051$; Group C vs. Group A adj. HR 8.89; 95% CI 3.90–20.29; $p<0.001$; Group C vs. Group B adj. HR 3.86; 95% CI 1.73–8.61; $p<0.001$; Figure 2).

Discussion

The main finding of our investigation, performed in consecutive ACS patients, is that admission PCT levels are an independent predictor for intra-ICCU mortality and, when adjusted for admission diagnosis and TnI peak levels, even for long-term events (MACE and all cause mortality).

Data on PCT levels in ACS are so far controversial. Although some studies (Geppert et al. 2003; Kafkas et al. 2008) reported that PCT levels were increased in ACS patients on admission, other investigations (Buratti et al. 2001, Remskar et al. 2002) documented that plasma PCT concentrations were in the normal range in patients with uncomplicated acute myocardial infarction. In an unselected population of ACS patients, we recently observed (Picariello et al. 2009) that PCT showed higher values only in some patients with ST elevation myocardial infarction (STEMI) and in all patients with cardiogenic shock (CS).

In the last years, few studies focused on the role of PCT for in hospital mortality in ACS. In 77 ACS patients Ataoğlu et al. found that higher PCT levels within 48 h postadmission are associated with increased early (in-hospital) and 6-month mortality (Ataoğlu et al. 2010). In 977 patients with ACS (780 STEMI, 197 NSTEMI), Kelly et al. showed a strict association between PCT levels and MACE on uni- and multivariate analysis at a median follow-up of 671 days, with an adverse outcome if PCT was above the median values (Kelly et al. 2010). In a more recent series, Sinning and colleagues evaluated the prognostic role of PCT in long-term cardiovascular risk prediction for 2,131 patients with documented CAD, both stable angina and acute coronary syndromes (Sinning et al. 2011). At long-term follow-up (3.6 years), baseline PCT levels were related to higher cardiovascular mortality and higher cardiovascular event-rate during follow-up. However, the population described in this study comprised patients at very low risk, such as stable angina (61% of patients), so that long-term cardiovascular events interpretation could result misleading.

Conversely, our investigation includes a high-risk population of acute coronary syndrome patients, from unstable angina to cardiogenic shock following STEMI. We observed for the first time, a progressive increase of in-hospital mortality for higher PCT values, and a gradual

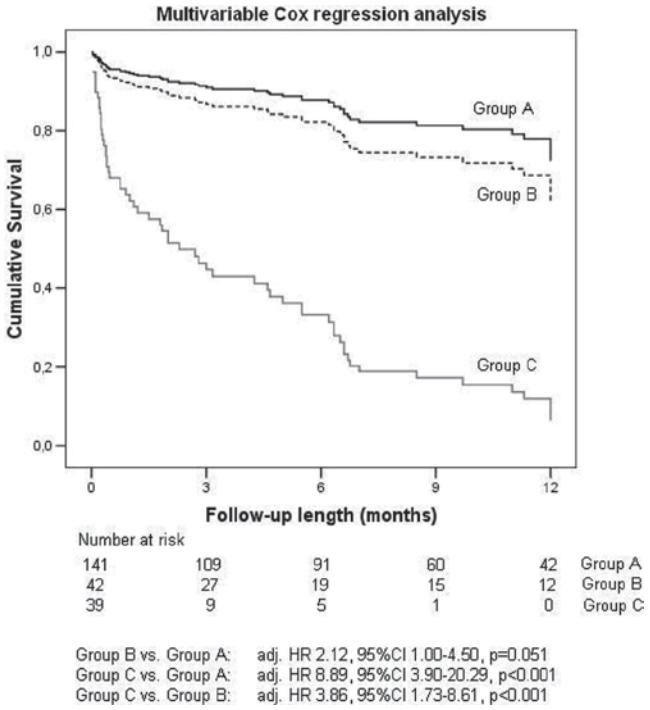

Figure 2. Event-free functions according to procalcitonin levels.

rise in 1-year events risk for high PCT categories (MACE and all cause mortality). In our series, most patients (about 60%) exhibited normal PCT values, but if sPCT values are higher than 0.05 ng/mL (in absence of infection) an increased risk for short- and long-term all cause mortality is detectable.

This phenomenon can be related to the fact that higher values of PCT correlate with the following: a greater severity of illness, showing the highest values in CS; a greater myocardial infarction extension, as inferred by TnI peak levels; a more pronounced acute stress response, as indicated by glucose values and acute insulin resistance (Lazzeri et al. 2011); a greater inflammatory burden, as indicated by CRP levels; and a worst clinical profile (as inferred by eGFR and proBNP values).

Thus, in clinical practice, attending physicians working in ICCU should care about admission PCT levels in acute coronary syndromes without signs of infection, independently from ACS subgroup: patients with high PCT levels should be closely monitored as they can have a worse clinical outcome and a higher risk of intra-ICCU death.

Study limitations

The main limitation of our investigation is the small number of patients, as this is a single center experience. However, our population includes consecutive patients, quite all submitted to mechanical revascularization, thus mirroring the "real world scenario". Furthermore, we only collected baseline PCT measurements at admission instead of sequential blood sampling (Picariello et al. 2010), but the aim of the present investigation was to assess whether admission PCT value could help in the risk stratification of ACS patients.

Conclusions

Our investigation further extends previous data on the prognostic role of PCT in ACS. In ACS patients without signs of infections, high admission PCT values allow the identification of a higher risk subgroup of patients for short- and long-term mortality characterized by the following: a greater severity of illness, showing the highest values in CS; a wider myocardial infarction extension, as inferred by TnI peak levels; a greater acute stress response, as indicated by glucose values and acute insulin resistance; a higher inflammatory burden, as indicated by CRP levels; with a worst clinical setting, as indicated by eGFR and NT-proBNP levels.

Declaration of interest

The authors report no declarations of interest.

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