Determinants and prognosis of high-sensitivity cardiac troponin T peak plasma concentration in patients hospitalized for non-cardiogenic shock

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Abstract

Purpose: The aim of this study was to assess the determinants and prognostic value of high-sensitivity cardiac troponin T peak plasma concentration in intensive care unit patients with non-cardiogenic shock.

Material and methods: A prospective observational cohort study was conducted in a single intensive care unit between November 2014 and December 2015.

Results: During the study period, 206 patients were hospitalized in the intensive care unit for non-cardiogenic shock and the median peak high-sensitivity cardiac troponin T was 55.1 [24.5–136] pg/mL. A multivariate analysis combining all variables showed that higher body mass index ($t = 2.52$, $P = 0.01$), lower left ventricular systolic function ($t = -2.73$, $P = 0.007$), higher white blood cell count ($t = 3.72$, $P = 0.0001$), lower creatinine clearance ($t = -2.84$, $P = 0.0005$), higher lactate level ($t = 2.62$, $P = 0.01$) and ST-segment depression ($t = 3.98$, $P = 0.0001$) best correlated with $\log_{10}$-transformed high-sensitivity cardiac troponin T peak plasma concentration. After multivariate analysis, the high-sensitivity cardiac troponin T peak was not associated with a significant reduction of in-hospital mortality (adjusted odds ratio = 0.99 (95% confidence interval: 0.93–1.02)).

Conclusion: High-sensitivity cardiac troponin T elevation was very common in patients hospitalized for non-cardiogenic shock. The factors significantly associated with high-sensitivity cardiac troponin T peak plasma concentration were higher body mass index, decreased left ventricular systolic ejection fraction, higher leucocyte count, decreased renal function, increased lactate level, and ST-segment depression. The high-sensitivity cardiac troponin T peak was not significantly associated with in-hospital mortality in this setting.

Introduction

Increased plasma concentration of cardiac troponin (cTn) is a very sensitive and specific marker of myocardial damage.[1] Studies have shown that increased plasma concentration of cTn is frequently encountered in intensive care unit (ICU) patients.[1,2] More widespread use of highly sensitive cTn assays has led to a substantial increase in the detection of elevated levels of cTn.[3] In patients hospitalized in ICU, myocardial damage is rarely caused by spontaneous coronary artery occlusion (i.e. type 1 acute myocardial infarction).[4] Many hypotheses have been put forward to explain cTn elevation in ICU patients: imbalance between oxygen supply and oxygen demand during shock or respiratory failure (i.e. type 2 acute myocardial infarction or ischaemic imbalance),[5,6] direct damage caused

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by endotoxins, microvascular coagulation dysfunction; renal insufficiency;[7,8] left ventricular diastolic dysfunction; and right ventricular dilatation.[9] Studies have evaluated determinants of cTn elevation in general population,[10] in acute ischaemic stroke,[11] in respiratory failure,[12] and in cardiology.[13] One study did evaluate determinants of high-sensitivity cardiac troponin T (hs-cTnT) elevation in ICU patients with shock, but the analysis focused strictly on septic patients and on echocardiographic data.[9] The aim of this study was to assess the determinants and prognostic value of hs-cTnT peak plasma concentration in ICU patients with non-cardiogenic shock (N-CS).

Material and methods

This observational study was approved by the local Ethics Committee of Félix Guyon University Hospital (R14008). Written informed consent was obtained from patients or from their legally authorized representative. Reporting of this study conforms to 'the Strengthening the Reporting of Observational studies in Epidemiology' recommendations statement for reporting.[14]

Selection of the study sample

This prospective observational cohort study was conducted between November 2014 and December 2015 in a single 23-bed mixed medical/surgical ICU at a French university hospital. Established criteria for the diagnosis of N-CS were as follows:[15,16] (1) vasopressors required despite adequate fluid loading to achieve a mean blood pressure >65 mmHg; (2) absence of elevated left ventricular filling pressures on transthoracic echocardiography assessed with the presence of 2 the 3 following criteria: E-wave velocity (E)/lateral early diastolic mitral annulus velocity >14, peak tricuspid regurgitation velocity >2.8 m/s, and left atrial maximal volume index >34 mL/m^2; (3) signs of impaired organ perfusion; and (4) absence of positive inotropes agents like dobutamine, dopamine, adrenaline, levosimendan, phosphodiesterase-3 inhibitors, and isoprenaline. Non-inclusion criteria were as follows: inability to obtain informed consent; hospitalization for chest trauma, cardiac arrest, presence of extracorporeal membrane oxygenation; hospitalization after cardiac surgery and surgery with cardiopulmonary bypass.

Upon admission, hs-cTnT was systematically measured every 8 h in all patients during 24 h. Blood samples were analysed using a Cobas Roche hs-cTnT assay (Basel, Switzerland).

Data collection

Patient characteristics and comorbidities were recorded on admission. Electrocardiography was systematically performed within 24 h of admission. The following elements were recorded: heart rate; rhythm; ST-segment depression or elevation >0.5 mm, prominent T-wave flattering or inversion >1 mm, and Q wave ≥0.03 s and ≥0.01 mV deep in at least two contiguous leads; and left bundle branch block.

Transthoracic echocardiography was also systematically performed within 24 h of admission. Transthoracic echocardiography examinations were performed by two experienced operators (J.A. and N.A. who already performed more than 2000 examinations) licensed in echocardiography and blinded to the context and clinical data. The following elements were recorded: left ventricular systolic ejection fraction using the monoplane modified Simpson's rule; left ventricular end-diastolic diameter (mm/m^2), interventricular septal thickness (mm) mitral, Doppler patterns with mitral inflow E-wave velocity (E), A-wave velocity (A), and lateral early diastolic mitral annulus velocity measured by tissue Doppler imaging (Ea); right ventricular dilatation assessed by a right ventricular end-diastolic area/left ventricular end-diastolic area ratio >0.6;[17] right ventricular systolic dysfunction assessed by tricuspid annular plane systolic excursion <[17] mm.[18]

Outcome measures

The primary endpoint was hs-cTnT plasma peak in ICU patients with N-CS. The secondary endpoint was in-hospital mortality.

Statistical analysis

Results are expressed as total numbers (percentages) for categorical variables and median [25th–75th percentiles] for continuous variables, as appropriate. Continuous variables were compared using the unpaired Student's *t* test or the non-parametric Mann–Whitney test, as appropriate. Categorical variables were compared using the Chi-square test or the Fisher's exact test, as appropriate. Values of hs-cTnT with exponential distribution were $\log_{10}$-transformed for linear analyses. Pearson's rank correlation coefficient analysis was performed to assess the correlation between $\log_{10}$ (hs-cTnT) plasma peak concentration and continuous variables. Second, the variables independently correlating with $\log_{10}$ (hs-cTnT) $P < 0.1$ were entered into a multivariate logistic regression analysis. Furthermore, risk factors of in-hospital mortality in bivariate analysis with $P < 0.1$ were entered into a multivariate logistic regression analysis using backward selection with $P < 0.05$. Colinearity between independent risk factors was tested; in cases of colinearity between risk factors, the most clinically relevant factor was selected to construct the multivariate models. The receiver operator characteristic curve was constructed to evaluate the capacity of hs-cTnT peak plasma concentration to predict in-hospital mortality. Analyses were performed using SAS statistical software (8.2, Cary, NC, USA).
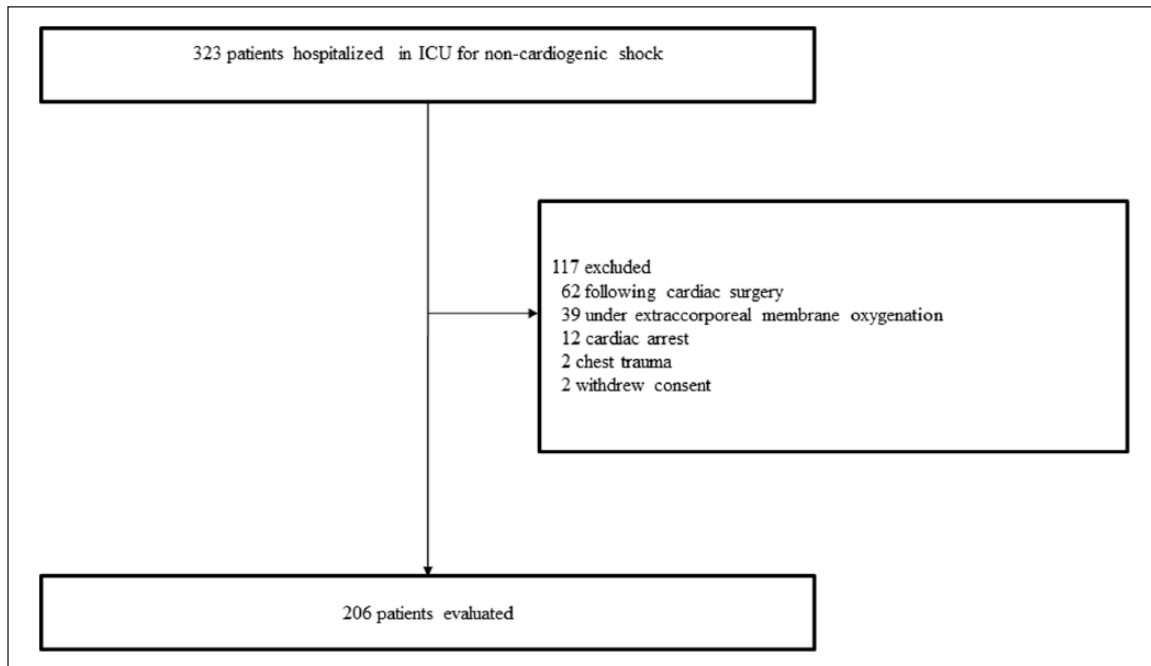
Figure 1. Selection of the study sample.

Results

Patient characteristics

During the study period, 323 patients were admitted to the ICU for N-CS; 117 patients were excluded from the analysis; 12 for cardiac arrest, 39 for receiving extracorporeal membrane oxygenation, 2 for chest trauma, and 62 for hospitalization after cardiac surgery. The remaining 206 patients constituted the cohort (Figure 1).

The main characteristics of the patients are presented in Tables 1 and 2.

The median age of the patients was 59 [51–70] years old, with a mean Simplified Acute Physiology Score 2 of 58 [45–68]. The main aetiology of N-CS was septic shock in 148 cases (71.8%) and haemorrhagic shock in 15 cases (10.1%).

Determinants of hs-cTnT elevation

The median peak plasma concentration of hs-cTnT was 55.1 [24.5–136] pg/mL. After univariate analysis, hs-cTnT plasma peak concentration was associated with a *P* value <0.1 for older age, higher body mass index, hypertension, diabetes mellitus, higher Simplified Acute Physiology Score 2, higher Sequential Organ Failure Assessment score, lower diastolic blood pressure, lower mean blood pressure, higher leucocyte count, lower prothrombin time, lower platelet count, lower haemoglobin level, lower glomerular filtration rate, higher lactate level, renal replacement therapy, higher heart rate, ST-segment depression, lower left ventricular ejection fraction, and lower tricuspid annular plane systolic excursion (Tables 3 and 4).

After univariate analysis, patients with hs-cTnT plasma peak concentration >14 pg/mL were older (*P*=0.0045), had higher Sequential Organ Failure Assessment score (*P*=0.048), lower left ventricular ejection fraction (*P*=0.016), and lower glomerular filtration rate (*P*<0.0001) than patients with hs-cTnT plasma peak concentration <14 pg/mL.

A multiple multivariate analysis showed that hs-cTnT plasma peak concentration was independently associated with higher body mass index, higher leucocyte count, lower glomerular filtration rate, higher lactate level ST-segment depression and lower left ventricular ejection fraction (Tables 3 and 4).

In-hospital mortality

The in-hospital mortality rate was 37.4%. A univariate analysis revealed that the peak plasma concentration of hs-cTnT was associated with a higher in-hospital mortality rate (the peak plasma concentration of hs-cTnT was 50.1 [20.9–121.3] ng/L in survivors and 67.4 [32.4–167] ng/L in non-survivors, *P*=0.03). The other risk factors found to be predictive of in-hospital mortality (based on a bivariate analysis with *P*<0.1) are presented in Tables 1 and 2. The receiver–operator characteristic curve did not show an optimal threshold value of hs-cTnT peak plasma concentration for the prediction of in-hospital mortality (area under the receiver operator characteristic curve was 0.592, 95% confidence interval (CI): 0.514–0.670) (Figure 2).

Table 1. Characteristics of the 206 patients.

Variable	Total (n = 206)	Non-survivors (n = 77)	Survivors (n = 129)	P value
Comorbidities				
Age (years)	59 [51–70]	66 [58–75]	55 [45–63]	0.0004
Male sex	131 (63.6)	44 (57.1)	87 (67.4)	0.13
Body mass index (kg/m^2)	24.2 [20.8–28]	24.7 [20.2–28.7]	24 [20.9–27.6]	0.5
Weight (kg)	70 [55.5–80]	70.5 [55–80]	70 [56.5–80]	0.88
Hypertension	87 (42.2)	41 (53.2)	46 (35.7)	0.01
Hyperlipidaemia	22 (10.7)	9 (11.7)	13 (10.1)	0.72
Diabetes mellitus	66 (32)	37 (48.1)	29 (22.5)	0.0001
Smoking (current or former)	77 (37.4)	28 (36.4)	49 (38)	0.82
History of coronary artery disease	31 (15)	18 (23.4)	13 (10.1)	0.01
Peripheral vascular disease	22 (10.7)	15 (19.5)	7 (5.4)	0.002
History of stroke	18 (8.7)	7 (9.1)	11 (8.5)	0.89
History of atrial fibrillation	14 (6.8)	9 (11.7)	4 (3.1)	0.03
History of congestive heart failure	7 (3.4)	4 (5.2)	3 (2.3)	0.27
Liver cirrhosis	12 (5.8)	8 (10.4)	4 (3.1)	0.03
History of gastro-duodenal ulcer	15 (7.3)	7 (9.1)	8 (6.2)	0.44
COPD with treatment	36 (17.5)	19 (24.7)	17 (13.2)	0.04
Cancer	27 (13.1)	18 (23.4)	9 (7)	0.0007
Immunodepression	49 (23.8)	31 (40.3)	18 (14)	<0.0001
Organ failure				
Simplified Acute Physiology Score 2	58 [45–68]	64 [50–73]	56 [43–64]	0.0004
SOFA score	10 [8–12]	11 [8–13]	10 [7–12]	0.07
Temperature (°C)	38 [37.2–38.8]	37.8 [37.2–38.7]	38.1 [37.3–38.8]	0.1
Systolic blood pressure (mmHg)	90 [82–100]	89 [78–101]	92 [85–100]	0.15
Diastolic blood pressure (mmHg)	50 [43–55]	45 [40–52]	52 [45–55]	0.001
Mean blood pressure (mmHg)	63 [57–67]	60 [55–65]	63 [57–67]	0.004
Lowest O$_2$ saturation (%)	94 [90–96]	92 [90–95]	94 [91–96]	0.09
Leucocyte count (G/L)	15.4 [10.8–22.9]	15.6 [11–22.9]	15.3 [10.8–22.2]	0.84
Prothrombin time (%)	58 [45–71]	52 [38–65]	63 [5171]	0.001
Platelet count (G/L)	144 [72–230]	143 [70–243]	146 [89–227]	0.84
Bilirubin level (µmol/L)	14 [8–30]	16 [10–33]	14 [8–30]	0.29
Haemoglobin level (g/dL)	9.9 [7.8–11.3]	9.1 [7.5–11.4]	10.2 [8.5–11.4]	0.04
Glomerular filtration rate (mL/min)	35 [19.8–54.8]	27.8 [19.5–40.1]	42.7 [20.7–62.3]	0.003
Lactate level (mmol/L)	3.2 [1.9–5.9]	3.9 [2.2–9.2]	2.8 [1.8–4.9]	0.003
Mechanical ventilation	184 (89.3)	74 (96.1)	110 (85.3)	0.015
PaO$_2$/FiO$_2$ ratio	190 [120–271]	166 [106–252]	198 [127–280]	0.15
Norepinephrine (µg/kg/min)	0.54 [0.3–1.01]	0.69 [0.33–1.54]	0.48 [0.29–0.96]	0.04
Renal replacement therapy	79 (38.3)	38 (49.4)	41 (31.8)	0.01
Glasgow Coma Scale score	15 [12–15]	15 [13–15]	15 [11–15]	0.09

COPD: chronic obstructive pulmonary disease; SOFA: Sequential Organ Failure Assessment.
Results are expressed as median [25th–75th percentiles] or n (%), as appropriate.

After multivariate analysis, the peak plasma concentration of hs-cTnT was not associated with a higher in-hospital mortality rate (*P*=0.085). In-hospital mortality was independently associated with older age (per year increment) (////odds ratio (OR): 1.075; 95% CI: 1.041–1.109, *P*<0.0001), higher lactate level (OR: 1.135; 95% CI: 1.027–1.255, *P*=0.001), mechanical ventilation (OR: 5.247; 95% CI: 1.224–22.499, *P*=0.03), and lower left ventricular ejection fraction (OR: 0.966; 95% CI: 0.936–0.936, *P*=0.03) (Table 5). The Hosmer–Lemeshow goodness-of-fit test showed good calibration of the model

(*P*=0.977). The Nagelkerke and Cox/Snell R squares were 0.306 and 0.224, respectively.

Discussion

The main results of this study were as follows: (1) hs-cTnT elevation was very common in patients hospitalized in ICU for N-CS; (2) in this context, the factors significantly associated with hs-cTnT peak plasma concentration after multivariate analysis were higher body mass index, decreased left

Table 2. Cardiologic features at inclusion.

Variable	Total (n = 206)	Non-survivors (n = 77)	Survivors (n = 129)	P value
Electrocardiography				
Heart rate (min)	105 [85–124]	108 [90–129]	102 [81–120]	0.15
Electrocardiographic abnormalities	73 (35.4)	28 (36.4)	45 (34.9)	0.83
Supraventricular arrhythmia	26 (12.6)	12 (15.6)	14 (10.9)	0.33
Prominent T-wave inversion	38 (18.4)	14 (18.2)	24 (18.6)	0.94
Left bundle brunch block	24 (11.7)	13 (16.9)	11 (8.5)	0.07
ST-segment depression	11 (5.3)	3 (3.9)	8 (6.2)	0.48
Electrocardiographic abnormalities	73 (35.4)	28 (36.4)	45 (34.9)	0.83
Echocardiography				
Left ventricular ejection fraction (%)	60 [50–55]	55 [45–65]	60 [55–65]	0.001
Cardiac output (L/min)	6 [4.9–7.3]	6 [4.8–7]	6.1 [5.1–7.7]	0.25
E-wave (cm)	74 [60–90]	68 [60–81]	75 [60–95]	0.26
A-wave (cm)	74 [60–90]	80 [67–95]	70 [58–83]	0.014
E-wave/A-wave	1 [0.8–1.3]	0.8 [0.7–1.1]	1.1 [0.9–1.3]	0.0014
Ea-wave	10 [8.4–12.1]	9.1 [7.7–11.4]	10 [8.5–12.8]	0.03
Ea-wave <10 cm	63 (30.6)	30 (40)	33 (25.6)	0.04
E/Ea ratio	7.2 [5.7–9.7]	7.6 [5.7–9.3]	7 [5.7–9.9]	0.4
Tricuspid annular plane systolic excursion (mm)	18 [16–21]	17 [15–20]	19 [17–22]	0.004
Tricuspid annular plane systolic excursion <17 mm	43 (20.9)	24 (31.2)	19 (14.7)	0.005
Right ventricular dilatation	28 (13.6)	13 (16.9)	15 (11.6)	0.29
High-sensitivity cardiac troponin T peak (ng/L)	55.1 [24.5–136]	67.4 [32.4–167]	50.1 [20.9–121.3]	0.03

Results are expressed as median [25th–75th percentiles] or n (%), as appropriate.

ventricular systolic ejection fraction, higher leucocyte count, decreased renal function, increased lactate level, and ST-segment depression on the electrocardiogram; (3) the peak plasma concentration of hs-cTnT was not associated with an increase in in-hospital mortality after multivariate analysis.

The incidence of hs-cTnT elevation (88.9%) was high in our study, as was that reported by Landesberg et al.[9] in patients hospitalized for septic shock (73%). Detection of myocardial necrosis is more frequent with biological methods that measure hs-cTnT than with older methods.[3] Thus, the reported incidence of myocardial necrosis is below 50% in studies that use more conventional methods of cTn measurement.[1,15,17,19] We noted a significant correlation between hs-cTnT elevation and a decrease in the glomerular filtration rate – which is a well-established association.[9,10,20] We also found a significant correlation between rise in body mass index and hs-cTnT elevation that might be explained by the fact that cardiac mass increases with body mass. This association was already observed in the study by Wallace et al.,[10] who also evaluated the factors linked to cTn elevation, but in a general population. The correlation between leucocyte increase and hs-cTnT elevation may have been related to an inflammatory response secondary to myocardial necrosis with an elevation of circulating plasma cytokines.[21] Similarly, we found a significant association between decreased systolic left ventricular function and elevated hs-cTnT. In cases of septic shock, which concerned a large proportion of

patients in our study, systolic left ventricular dysfunction is often present; it is generally reversible and associated with cTn elevation, whose pathophysiological mechanisms are multiple.[22] Likewise, the association between ST-segment depression on the ECG and hs-cTnT elevation, already observed in the study by Faiz et al.,[11] may be a marker of type 2 myocardial infarction. We found a positive correlation between hs-cTnT elevation and plasma lactate level that may have been related to myocardial necrosis secondary to an imbalance between oxygen supply and oxygen demand during shock, bleeding, or acute respiratory failure (type 2 myocardial infarction). Unlike Landesberg et al.,[9] we did not find a significant relationship between hs-cTnT elevation and right ventricular dilatation or diastolic dysfunction after multivariate analysis. This may be due to a lack of power in our study, to differences in the two study populations, or to the fact that Landesberg et al.[9] primarily examined the correlation between echocardiographic parameters and hs-cTnT elevation, and that they analysed fewer clinical and biological parameters than we did.

The peak plasma concentration of hs-cTnT was associated with in-hospital mortality after univariate analysis, but not after multivariate analysis ($P = 0.085$). It is possible that the study is underpowered while only 206 patients were prospectively evaluated and the lack of difference in mortality could be due to a type II statistical error. Previous studies found that level of peak plasma concentration of cTn was associated with in-ICU mortality.[1,6,23] In the study by Landesberg et al.,[9]

Table 3. Clinical characteristics correlated with $\log_{10}$ high-sensitivity cardiac troponin T.

Variable	Univariate		Multivariate	
	t	p	t	p
Comorbidities				
Age	0.14	0.02		
Male sex	−0.8	0.41		
Body mass index	0.16	0.03	2.52	0.01
Hypertension	−1.9	0.056		
Hyperlipidaemia	−0.5	0.6		
Diabetes mellitus	−2.8	0.006		
Smoking (current or former)	1.2	0.23		
History of coronary artery disease	−1.1	0.29		
Peripheral vascular disease	−1.4	0.16		
History of stroke	0.7	0.46		
History of atrial fibrillation	−1.1	0.26		
History of congestive heart failure	0.2	0.81		
Liver cirrhosis	−0.9	0.33		
History of gastro-duodenal ulcer	0.8	0.45		
COPD with treatment	0.4	0.7		
Cancer	0.9	0.38		
Immunodepression	0.2	0.8		
Organ failure				
Simplified Acute Physiology Score 2	0.18	0.009		
SOFA score	0.14	0.04		
Systolic blood pressure	−0.02	0.74		
Diastolic blood pressure	−0.14	0.04		
Mean blood pressure	−0.14	0.052		
Lowest O_2 saturation	−0.02	0.74		
Leucocyte count	0.25	<0.0001	3.72	0.0001
Prothrombin time	−0.16	0.02		
Platelet count	−0.13	0.06		
Bilirubin level	−0.07	0.29		
Haemoglobin level	−0.16	0.025		
Glomerular filtration rate	−0.29	<0.0001	−2.84	0.0005
Lactate level	0.21	0.002	2.62	0.01
Mechanical ventilation	−1	0.34		
PaO_2/FiO_2 ratio	−0.04	0.63		
Norepinephrine	0.07	0.35		
Renal replacement therapy	−2.2	0.028		
Glasgow Coma Scale score	0.06	0.39		

COPD: chronic obstructive pulmonary disease; SOFA: Sequential Organ Failure Assessment.

who focused on hs-cTnT elevation in septic shock patients, hs-cTnT was associated with in-hospital mortality after univariate analysis, but not after multivariate analysis.

This study has several limitations. It is a single-centre observational study, and no direct causal relationship can be established between isolated variables and hs-cTnT elevation. We collected data from only one transthoracic echocardiography per patient, we did not have access to echocardiographic parameters before ICU admission and we could not collect all data on echocardiography. We did not find significant relationship between hs-cTnT elevation and E/Ea ratio or diastolic dysfunction, and this could be related to the fact that we have excluded patients with elevated left ventricular filling pressures. As a result, the clinical implications of our findings remain uncertain.

Conclusion

hs-cTnT elevation is very common in patients hospitalized for N-CS. The causes for this elevation are multiple (decreased renal elimination of hs-cTnT, inflammation, myocardial necrosis due to inflammation, etc.) and in this context, hs-cTnT elevation does not appear to have any prognostic value.

Table 4. Cardiologic features correlate of $\log_{10}$ high-sensitivity cardiac troponin T.

Variable	Univariate		Multivariate	
	t	*p*	*t*	*p*
Electrocardiography				
Heart rate	0.12	0.08		
Electrocardiographic abnormalities	−3.3	0.001		
Supraventricular arrhythmia	1.1	0.35		
Prominent T-wave inversion	−0.9	0.35		
Left bundle brunch block	1.9	0.51		
ST-segment depression	−4.5	<0.0001	3.98	0.0001
Echocardiography				
Left ventricular ejection fraction	−0.18	0.01	−2.73	0.007
Left ventricular end-diastolic diameter	0.08	0.17		
Interventricular septal thickness	0.1	0.13		
Cardiac output	−0.05	0.52		
E-wave	−0.11	0.21		
A-wave	0.03	0.74		
E-wave/A-wave	−0.12	0.16		
Ea-wave	−0.08	0.37		
Ea-wave <10 cm	−1.7	0.08		
E/Ea ratio	0.04	0.62		
Tricuspid annular plane systolic excursion	−0.17	0.06		
Tricuspid annular plane systolic excursion <17 mm	0.3	0.77		
Right ventricular dilatation	−1.3	0.2		
Abnormalities of left ventricular kinetics	−0.6	0.53		
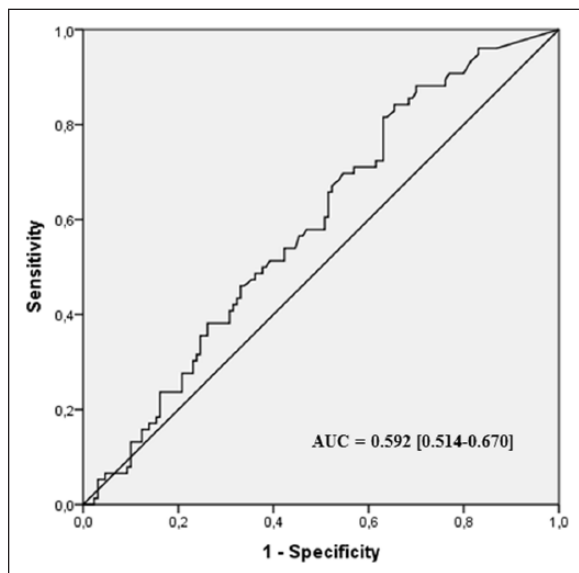
Figure 2. Receiver operator characteristic curve of hs-cTnT for the prediction of in-hospital mortality.

Table 5. Multivariate analysis of risk factors associated with in-hospital mortality.

Variable	Adjusted odds ratio (95% CI)	*P* value
Age (per year increment)	1.075 (1.041–1.109)	<0.0001
Lactate level	1.135 (1.027.1.255)	0.01
Mechanical ventilation	5.247 (1.224–22.499)	0.03
Left ventricular ejection fraction	0.966 (0.936–0.998)	0.03
Haemoglobin level (g/dL)	–	0.056
Prothrombin time	–	0.27
Glomerular filtration rate	–	0.56
Norepinephrine dose	–	0.85
Mean blood pressure	–	0.38
High-sensitivity cardiac troponin T peak	–	0.085

CI: confidence interval.
The Hosmer-Lemeshow goodness-of-fit test *P* value was 0.977. The Nagelkerke and Cox/Snell R squares were 0.306 and 0.224, respectively.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethical approval

Ethical approval for this study was obtained from the local Ethics Committee of Félix Guyon University Hospital (R14008).

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Informed consent

Written informed consent was obtained from patients or from their legally authorized representative.

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