Cardiac Troponin T for Risk Stratification in Decompensated Chronic Heart Failure

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Summary

Background: The cardiac troponins are highly sensitive and specific markers of myocardial injury. They have been detected in heart failure (HF) and are associated with a bad prognosis.

Objective: To evaluate the association of cardiac troponin T (cTnT) and its ranges with prognosis in decompensated HF.

Methods: A total of 70 patients with chronic HF worsening that needed hospitalization were studied. Cox model was used to evaluate the variables at admission capable of predicting the combined outcome that consisted of death or re-hospitalization due to HF worsening during a 1-year follow-up.

Results: During the follow-up, there were 44 deaths, 36 re-hospitalizations due to HF and 56 combined outcomes. At the multivariate analysis, the predictors of clinical events were the cTnT (cTnT ≥0.100 ng/mL; hazard ratio [HR] 3.95 95% confidence interval [CI]: 1.64-9.49, p = 0.002), left ventricular end diastolic diameter (LVDD ≥70 mm; HR 1.92, 95%CI: 1.06-3.47, p = 0.031) and serum sodium (Na <135 mEq/L; HR 1.79, 95%CI: 1.02-3.15, p = 0.044). To evaluate the association between the cTnT increase and the prognosis in decompensated HF, the patients were stratified in three groups: low-cTnT (cTnT ≤0.020 ng/ml, n = 22), intermediate-cTnT (cTnT >0.020 and <0.100 ng/ml, n = 36), and high-cTnT (cTnT ≥0.100 ng/ml, n = 12).The probabilities of survival and event-free survival were 54.2%, 31.5%, 16.7% (p = 0.020) and 36.4%, 11.5%, 8.3% (p = 0.005), respectively.

Conclusion: The increase in cTnT is associated with a bad prognosis in decompensated HF and the degree of this increase can help the risk stratification. (Arq Bras Cardiol 2009;92(5):372-380)

Key words: Heart failure, congestive; prognosis; troponin T; sodium.

Introduction

Heart failure (HF) represents an important public health problem in modern society, due to its increasing incidence and prevalence, low survival and economic overload caused by prolonged and repeated hospitalizations[1-3]. Thus, the identification of patients with a worse prognosis is essential for the appropriate allocation of health resources, as well as the planning of new researches and therapeutic strategies to improve patient care and the outcome of this life-threatening and debilitating disease.

The cardiac troponins are highly sensitive and specific biomarkers of myocardial injury, which are broadly used for risk stratification in acute coronary syndromes[4]. The detection of troponins in patients with advanced HF by Missov et al[5], has attracted the interest of physicians aiming at prognostic assessment.

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Many prognostic indicators have been described in patients with compensated and decompensated HF[6-10], with several studies focusing on the association between an increase in troponin levels and the HF prognosis, which showed an association of troponin increase with worse clinical outcome[11-22].

Although the current guidelines for the treatment of decompensated HF recommend the routine assessment of troponins in this situation, there are no indications of how these markers should be interpreted regarding patient management[23]. The use of troponins in the risk stratification for decompensated HF can help improve patient care by identifying those with a worse prognosis. Thus, the objective of this study was to evaluate the prognostic value of cTnT levels and the association between its ranges in a cohort of patients that had been hospitalized with chronic decompensated HF and the clinical outcomes after a one-year follow-up.

Methods

Studied population

The studied population consisted of 70 patients with HF that were prospectively admitted at the Emergency Department (ER) of Instituto do Coraçao – The Heart Institute

(InCor) – HCFMUSP, Sao Paulo – SP, Brazil and needed to be hospitalized due to decompensated symptoms of HF and NYHA functional class, between January and September 1999. The study included 62 patients that had participated in a previously published study[17].

The study methods had been previously published[17]; however, in summary, the patients with chronic decompensated HF were eligible for this study if they did not have a recent history of acute myocardial infarction, unstable angina or surgery in the previous 30 days. Patients with chronic renal disease, non-controlled arterial hypertension or active myocarditis were also excluded from the study. The patients included in the study had blood samples collected for laboratory assessment and measurement of serum cardiac troponin T (cTnT) and creatine-phosphokinase fraction –MB mass (CKMB-mass) levels. The levels of cTnT and CKMB-mass were analyzed at the end of the study, so that the patients would receive the conventional treatment for decompensated HF, while the physicians were blinded to the serum levels of these markers.

The present study is according to the principles stated in the Declaration of Helsinki. The study protocol was approved by the Ethics Committee in Research of InCor-HCFMUSP and all patients signed the informed consent form prior to the study participation.

Clinical outcome and patient follow-up

The primary outcome of the present study was a combined outcome, consisting of death or the need for re-hospitalization due to a new episode of HF decompensation during the 1-year follow-up (clinical event). The studied population was followed through contacts every three months to determine the vital status of the patients. The type of death was classified as death by pump failure, myocardial infarction or sudden death.

Analysis of cardiac troponin T and creatine phosphokinase Fraction-MB Levels

The blood samples for the measurement of cTnT and CK-MB levels were obtained at the hospital admission (median: 3 days; interquartiles: 2 to 4 days). cTnT was quantified by a third-generation immunoassay (Elecsys® Troponin T STAT Immunoassay, Roche Diagnostics, Germany). The lower range of detection is 0.010 ng/mL, and cTnT levels that are below this detection threshold were considered as zero. The serum levels of CK-MB mass were analyzed through another immunoassay (Elecsys® CK-MB STAT Immunoassay, Roche Diagnostics, Germany). This assay has a detection threshold of 0.1 ng/mL, with a reference range of 0 a 5.0 ng/mL.

Statistical analysis

The comparisons between the continuous variables were carried out by the Mann-Whitney U-test. The Chi-square test or Fisher's exact test were used to evaluate the categorical variables.

To estimate the probability of clinical events in relation to the ranges of cTnT increase during the 1-year follow-up period (primary outcome), the patients were retrospectively stratified in groups based on the cTnT levels. The low-cTnT group was defined based on the cutoff value below the level determined by the ROC (receiver operating characteristic) curve. The cTnT level ≥0.100 ng/mL was selected to define the high-cTnT group. The intermediate-cTnT group was defined as having cTnT levels above the cutoff and <0.100 ng/mL. The Kruskal-Wallis and the Chi-square tests were used to compare the characteristics among the three groups (low, intermediate and high). The survival and event-free survival probabilities, stratified for the cTnT groups, were estimated by the Kaplan-Meier method.

The analysis of univariate and multivariate regression for the primary outcome predictors were carried out by Cox Proportional Hazard method. The variables with $p<0.200$ at the univariate analysis were selected for the multivariate model. The final model was constructed with the stepwise-forward procedure and included only the variables with $p<0.050$. All the statistical analyses were carried out with the SPSS software and a p value < 0.050 was considered statistically significant.

Results

Basal characteristics and results

The basal characteristics of the studied population are shown in Table 1. The follow-up was complete in all patients with a median of 262 days (range: 3 to 393 days). During the follow-up period there were 44 (62.9%) deaths, 36 (51.4%) re-hospitalizations due to HF, 56 (80.0%) combined outcomes consisting of death or re-hospitalization (clinical event) and 2 (2.9%) heart transplants. Figure 1 shows the natural history of HF, in relation to the events analyzed during the follow-up. The patients that presented clinical events were more hypotensive, presented a more dilated left ventricle, lower levels of serum sodium, more frequently needed the administration of intravenous inotropic drugs (dobutamine) and had a tendency to present more elevated levels of cTnT.

Detection of cardiac troponin t and its prognostic role in decompensated chronic heart failure

Elevated levels of cTnT (>0.010 ng/mL) were detected in 58 patients (82.9%) with decompensated HF and 12 patients (17.1%) presented cTnT ≥0.100 ng/mL. The mean concentration of this marker was 0.062±0.089 ng/mL and Table 2 shows the mean concentration of cTnT and the range (minimum-maximum) according to the HF etiology. The area under the ROC curve was 0.66 and identified a cutoff for cTnT >0.020 ng/mL, for the prediction of clinical events (sensitivity = 0.76, specificity = 0.62). When the patients were stratified based on this cutoff, higher rates of clinical events and deaths were observed among patients with levels of cTnT >0.020 ng/mL (Figure 2).

To evaluate the association between the ranges of cTnT increase and their prognostic value, the studied population was retrospectively stratified in the groups based on the previously described cTnT levels. A progressive and significant decrease in survival and event-free survival (Figures 3 and 4) was observed with the increase in cTnT levels. The cumulative probabilities of survival and event-free survival in the low,

Table 1 - Basal characteristics and clinical outcomes of the studied population

Characteristics	All patients	Clinical events *		p†
	(n = 70)	Yes (n = 55)	No (n = 13)	
Age (yrs)	54.2 ± 15.8	53.0 ± 16.3	60.7 ± 12.2	0.101
Male sex	48 (68.6)	37 (67.3)	9 (69.2)	1.000
NYHA Class IV	59 (84.3)	47 (85.5)	10 (76.9)	0.428
Body mass index (kg/m^2)	24.3 ± 4.2	24.0 ± 4.2	25.8 ± 4.0	0.098
HF duration (year)	4.9 ± 6.1	5.4 ± 6.6	3.0 ± 3.5	0.272
HF etiology				
Ischemic	18 (25.7)	12 (21.8)	5 (38.5)	0.286
Chagasic	19 (27.1)	18 (32.7)	1 (7.7)	0.092
Valvular	13 (18.6)	9 (16.4)	4 (30.8)	0.254
Hypertensive	9 (12.9)	7 (12.7)	2 (15.4)	1.000
Idiopathic Cardiomyopathy	8 (11.4)	6 (10.9)	1 (7.7)	1.000
Others	3 (4.3)	3 (5.5)	0 (0.0)	1.000
Diabetes mellitus	14 (20.0)	12 (21.8)	2 (15.4)	1.000
Hypertension	28 (40.0)	21 (38.2)	7 (53.8)	0.302
Previous MI	15 (21.4)	10 (18.2)	4 (30.8)	0.445
Myocardial Revasc.	8 (11.4)	4 (7.3)	3 (23.1)	0.122
Atrial fibrillation	13 (18.6)	9 (16.4)	4 (30.8)	0.254
Systolic BP (mmHg)	111.7 ± 25.3	108.8 ± 22.8	124.9 ± 31.8	0.069
Diastolic BP (mmHg)	76.7 ± 17.9	74.7 ± 18.2	86.3 ± 14.4	0.025
Mean BP (mmHg)	88.4 ± 18.7	86.1 ± 18.0	99.2 ± 18.7	0.035
Heart rate (bpm)	86.2 ± 19.4	86.3 ± 19.1	87.6 ± 22.4	0.882
LVEF (%)	31.3 ± 8.4	30.5 ± 8.0	35.0 ± 9.2	0.128
LVDD (mm)	71.8 ± 10.0	72.5 ± 9.8	66.6 ± 7.0	0.049
Creatinine (mg/dL)	1.4 ± 0.5	1.4 ± 0.5	1.4 ± 0.6	0.253
GFR (mL/min)	56.8 ± 24.3	55.5 ± 24.6	61.9 ± 23.6	0.362
Sodium (mEq/L)	135.9 ± 5.4	135.0 ± 5.5	138.5 ± 4.1	0.025
cTnT (ng/ml)	0.062 ± 0.089	0.068 ± 0.092	0.041 ± 0.076	0.053
CK-MB mass (ng/mL)	3.3 ± 4.9	3.5 ± 5.5	2.5 ± 1.0	0.815
Medications				
Diuretics	68 (100.0)	55 (100.0)	13 (100.0)	1.000
Digoxin	62 (92.5)	50 (90.9)	12 (92.3)	1.000
ACE inhibitors	58 (85.3)	46 (83.6)	12 (92.3)	0.673
Nitrates	38 (55.9)	28 (50.9)	10 (76.9)	0.124
Hydralazine	16 (23.5)	13 (23.6)	3 (23.1)	1.000
Amiodarone	9 (13.2)	8 (14.5)	1 (7.7)	1.000
Carvedilol	7 (10.4)	4 (7.4)	3 (23.1)	0.127
Dobutamine	34 (50.0)	33 (60.0)	1 (7.7)	0.001

*The data are presented as means ± standard deviation or number (percentage); NYHA - New York Heart Association; HF - heart failure; Revasc. - revascularization; MI - myocardial infarction; BP - blood pressure; LVEF - left ventricle ejection fraction; LVDD - left ventricular-end diastolic diameter; GFR - estimated glomerular filtration rate (Cockcroft-Gault); cTnT - cardiac troponin T; CK-MB - creatine phosphokinase fraction-MB; ACE - angiotensin-converting enzyme; * Two patients submitted to heart transplant were excluded from the analysis; † p value compares the characteristics between patients with and without clinical events.*

Table 2 - Cardiac troponin T levels in relation to the etiology of the decompensated heart failure

Etiology	N	cTnT (ng/mL)	Range (min-max)
Chagasic	19	0.053 ± 0.041	<0.010 – 0.138
Ischemic	18	0.098 ± 0.122	<0.010 – 0.386
Valvular	13	0.037 ± 0.024	<0.010 – 0.093
Hypertensive	9	0.086 ± 0.156	<0.010 – 0.496
Idiopathic	8	0.032 ± 0.031	<0.010 – 0.089
Others	3	0.024 ± 0.022	<0.010 – 0.042
Total	70	0.062 ± 0.089	<0.010 – 0.496

The data are presented as means ± standard deviation and maximum and minimum range; N - number of patients; cTnT - cardiac troponin T.

intermediate and high-cTnT groups were, respectively: 54.2%, 31.5% and 16.7% (p = 0.020) and 36.4%, 11.5% and 8.3% (p = 0.005).

At the multivariate analysis (Table 3), the independent predictors for clinical events, considering only the patients' basal clinical and laboratory variables, were: elevated cTnT levels (cTnT ≥0.100 ng/mL; hazard ratio [HR] = 3.95, 95% confidence interval [CI] = 1.64 – 9.49; p = 0.002), increased left ventricular end-diastolic diameter (LVDD ≥70 mm; HR = 1.92, 95% CI= 1.06 – 3.47; p = 0.031) and low serum sodium levels (Na <135 mEq/L; HR = 1.79, 95%CI = 1.02 – 3.15; p = 0.044). When the need to use dobutamine was introduced in the multivariate analysis, the independent predictors for clinical events were dobutamine (HR = 2.33; 95%CI = 1.27 – 4.26; p = 0.006) and troponin T ≥0.100 ng/mL (HR = 3.07; 95%CI = 1.30 – 7.28; p = 0.001). We did not observe any difference in cTnT levels between patients that received and those that did not receive dobutamine during the hospitalization (0.062 ± 0.094 *vs.* 0.062 ± 0.084 ng/mL, p = 0.094). At the final analysis (Table 3), we considered only the clinical and laboratory variables at the patients' admission for the risk stratification.

The comparison of the clinical characteristics among the three groups of patients in relation to cTnT levels (Table 4) showed that the patients in the high-cTnT group had a tendency to present older mean age (p=0.071), more frequently presented the ischemic etiology (p=0.004), had a previous history of acute myocardial infarction (p=0.001) and presented lower estimated glomerular filtration rates (p=0.010).

Discussion

This study showed that the cTnT was detected in approximately 83% of the patients with decompensated chronic HF and was a long-term independent predictor for clinical events. It was also possible to demonstrate that the degree of cTnT elevation was useful in the one-year prognostic stratification of these patients in three risk groups, based on the cTnT levels: low (cTnT ≤0.020 ng/mL), intermediate (cTnT >0.020 ng/mL and <0.100 ng/mL) and high (cTnT ≥0.100 ng/mL).

The troponins are reliable markers that are broadly used to detect myocardial injury; additionally, several studies[11-22] have associated the presence of these markers with adverse outcomes in HF. In the present study, the minimal myocardial cell injury, detected through the cTnT release, was associated with a bad prognosis in HF. Additionally, it was possible to observe a progressive increase in death and clinical event risk with the increase in cTnT levels. This association had been previously reported for cardiac troponin I[22].

Other authors have described different cutoffs to predict adverse events for troponins in HF, ranging from 0.02 to 0.1

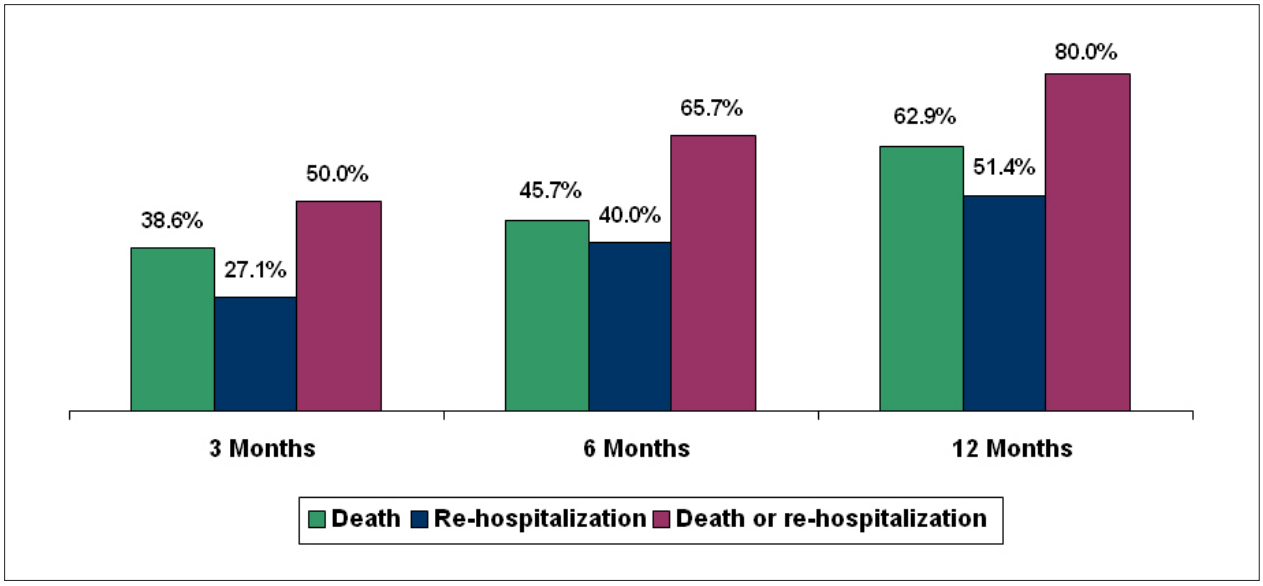

Figure 1 - *Natural history of heart failure (HF) regarding the death rates, re-hospitalization due to a new HF decompensation and combined outcomes of death and re-hospitalization in the studied population during a 1-year follow-up.*

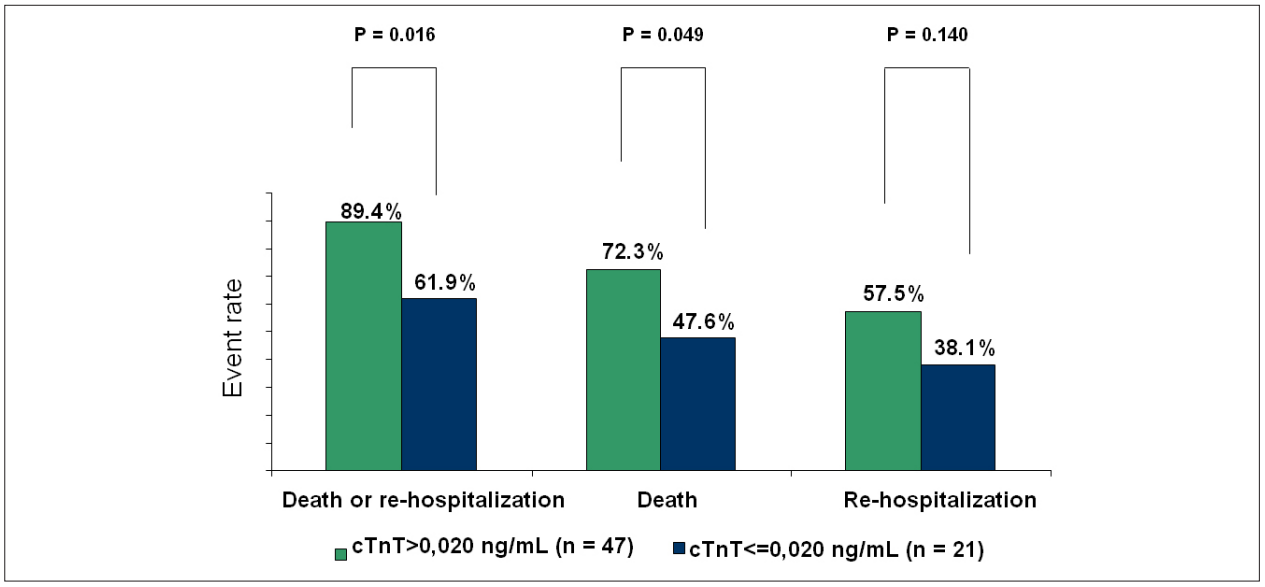

Figure 2 - Comparison between the rates of clinical events among patients with cTnT ≤0.020 ng/mL and cTnT >0.020 ng/mL. Two patients submitted to heart transplant were excluded from this comparison. cTnT - cardiac troponin T.

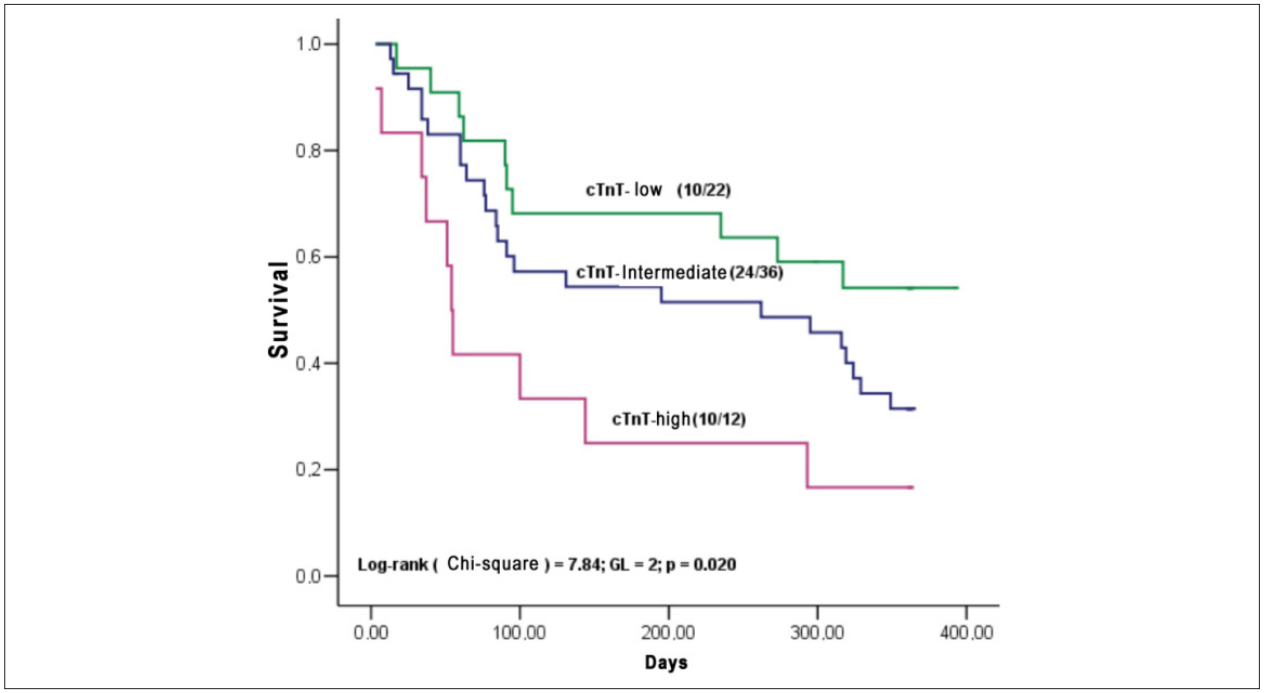

Figure 3 - Survival curves by the Kaplan-Meier method stratified in 3 groups, based on the level of cardiac T troponin (cTnT): low-cTnT (cTnT ≤0.020 ng/mL), intermediate-cTnT (cTnT >0.020 or <0.100 ng/mL), and high-cTnT (cTnT ≥0.100 ng/mL).

ng/mL for troponin T[11,13-15,18-21] and from 0.04 to 0.5 ng/mL for troponin I[12,16,22]. Three studies[13,18,19] described the same cutoff observed in the present study.

The mechanism through which the troponins are released in HF is not completely understood; however, there are several factors that can explain the detection of these biomarkers in this clinical situation. The physiopathological process responsible for the destruction of the contractile apparatus in the HF progression to the terminal stage can be responsible for the release of troponins[24], as follows: ventricular remodeling, apoptosis, endothelial dysfunction, alterations in the coronary microcirculation, recurrent ischemia episodes or infarction.

Additionally, the increase in the pre-load and the decrease in the diastolic perfusion time, which are commonly observed

Table 3 - Univariate and multivariate analysis by Cox Proportional Risk Method and predictors of clinical events after one year of follow-up in decompensated heart failure

Predictors	Hazard Ratio	95%CI	P
Univariate analysis			
Age >55 yrs	0.60	0.34 – 1.06	0.080
Male sex	1.00	0.55 – 1.84	0.976
NYHA Class IV	1.38	0.65 – 2.95	0.404
BMI <25 kg/m2	1.50	0.86 – 2.63	0.157
Ischemic etiology	0.53	0.25 – 1.12	0.097
Chagasic etiology	1.70	0.94 – 3.07	0.077
Diabetes mellitus	1.11	0.55 – 2.23	0.768
Previous MI	0.57	0.26 – 1.28	0.173
Systolic BP <100 mm Hg	1.03	0.52 – 2.02	0.933
Diastolic BP <80 mmHg	1.71	0.97 – 3.01	0.062
Mean BP ≤90 mm Hg	1.77	0.99 – 3.15	0.054
LVEF <0,30	1.08	0.62 – 1.90	0.790
LVDD ≥70 mm	2.19	1.26 – 3.82	0.006
Cardiac troponin T level			
≤0.020 ng/mL	1.00		
>0.020, <0.100 ng/mL	1.86	0.99 – 3.51	0.054
≥0.100 ng/mL	3.66	1.63 – 8.21	0.002
Creatinine >1.3 mg/dL	1.31	0.75 – 2.30	0.345
GFR <60 mL/min	1.59	0.88 – 2.84	0.122
Sodium <135.0 mEq/L	1.88	1.07 – 3.32	0.029
Dobutamine	2.45	1.41 – 4.24	0.001
Multivariate analysis *			
LVDD ≥70 mm	1.92	1.06 – 3.47	0.031
Cardiac troponin T level			
≤0.020 ng/mL	1.00		
>0.020, <0.100 ng/mL	1.83	0.92 – 3.63	0.085
≥0.100 ng/mL	3.95	1.64 – 9.49	0.002
Sodium <135.0 mEq/L	1.79	1.02 – 3.15	0.044

**At the multivariate analysis, the patients' basal clinical and laboratory variables were included, except for the variable dobutamine; CI - confidence interval; NYHA - New York Heart Association; BMI - body mass index; MI - myocardial infarction; BP - blood pressure; LVEF - left ventricle ejection fraction; LVDD - left ventricular-end diastolic diameter; GFT - estimated glomerular filtration rate (Cockcroft-Gault).*

in patients with decompensated HF, facilitate the degradation of troponin I and this finding can also probably be valid for cTnT. The stretching of cardiomyocytes through the pre-load increase and the activation of endogenous proteases leading to the degradation of troponin I seem to be related to the ischemia of the inner layers of the myocardial wall[25,26]. We observed the presence of increased cTnT levels (>0.01 ng/mL) in 16 of the 19 patients with HF due to Chagasic etiology (84,2%), with 3 patients presenting cTnT levels >0.100 ng/mL. This increase can be the consequence of a chronic inflammatory process in Chagasic cardiopathy, leading to myocardial injury and resulting in troponin release.

Thus, the detection of troponins in HF seems to indicate the continuous destruction of cardiomyocytes, with the HF progression to its final stage, which consequently indicates a bad prognosis.

Elevated troponin levels were detected in patients with renal failure in the absence of acute myocardial ischemia. In spite of the uncertainties regarding the mechanisms of serum troponin increase in renal function decrease, the detection of these markers seems to reflect the myocardial necrosis[27]. There is evidence to indicate that the renal function contributes to the elimination of cardiac troponins[28]. However, in acute coronary syndromes, the cTnT levels were strong short-term prognostic predictors, even when the decrease in renal function was present[29]. In the present study, the mechanisms of myocardial injury in decompensated HF associated with the decrease in the cTnT clearance due to renal dysfunction can explain the increased concentrations of this marker in the high-cTnT group.

The worst prognosis associated with the administration of dobutamine must be interpreted with care, as its use probably reflects more advanced disease and consequently, a bad prognosis, instead of a direct effect of this medication on the clinical results.

Study limitations

The sample size of this cohort was relatively small; however, due to the high rates of adverse clinical outcomes in this population with advanced disease, it was possible to demonstrate the association between cTnT levels and prognosis in these patients. Furthermore, this study only included patients with systolic dysfunction and therefore, the prognostic value of the cTnT in HF with preserved systolic function is unknown. Additionally, we did not analyze the serum levels of other prognostic markers in HF, such as the brain natriuretic peptide (BNP or NT-ProBNP), as these markers were not available in our country when the present study was carried out.

However, two studies[14,21] demonstrated that the combination of the levels of cTnT and BNP were capable of stratifying the prognosis of hospitalized patients with decompensated HF. A previous study[30] by our group showed that elevated levels of NT-proBNP were associated with a 3.6-fold higher risk of death within 90 days in patients with decompensated HF.

Another potential limitation of this study was that the coronary angiography was not routinely carried out in these patients. Thus, the undiagnosed coronary artery disease and the presence of micro-infarctions are possible explanations for the increase in cTnT levels. Indeed, the association between the HF of ischemic etiology and previous myocardial infarctions in patients from the high-cTnT group (Table 4) corroborates this hypothesis.

Clinical implications

The present study provides evidence confirming that increases in the cTnT levels are associated with lower survival and event-free survival rates in decompensated HF.

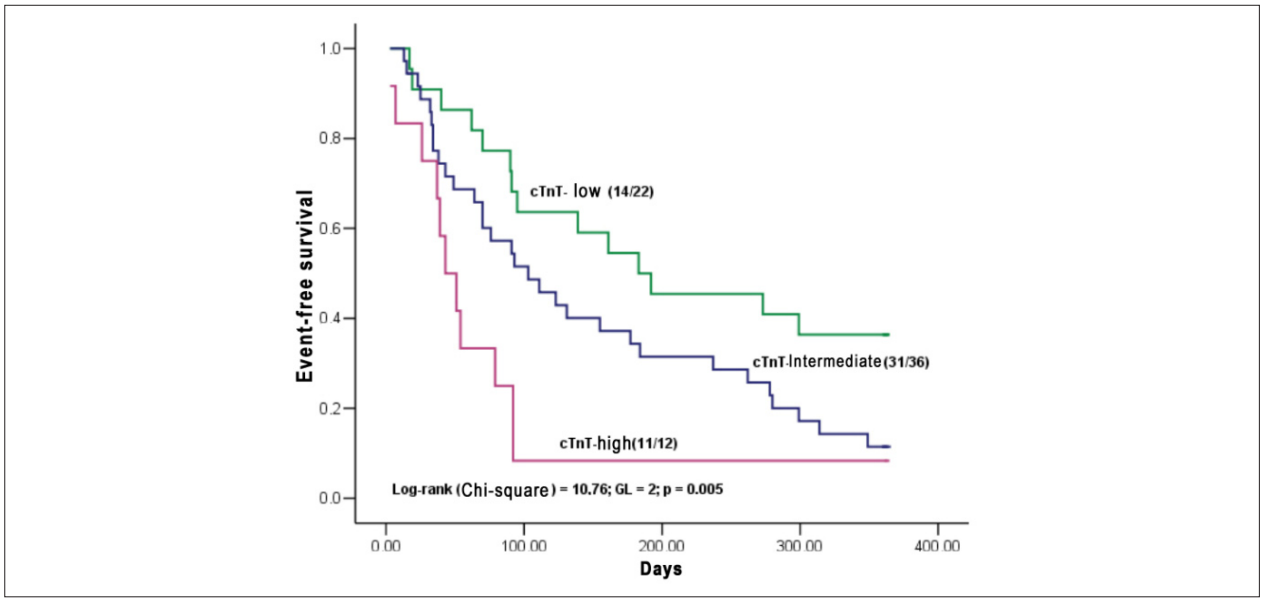

Figure 4 - Event-free survival curves by the Kaplan-Meier method stratified in 3 groups, based on the level of cardiac T troponin (cTnT): low-cTnT (cTnT ≤0.020 ng/mL), intermediate-cTnT (cTnT >0.020 or <0.100 ng/mL), and high-cTnT (cTnT ≥0.100 ng/mL).

Table 4 - Groups of patients in relation to cardiac troponin T and the clinical characteristics

Characteristics	Groups - Cardiac Troponin T			P
	Low	Intermediate	High	
	(n = 22)	(n = 36)	(n = 12)	
Age (yrs)	51.5±15.8	52.4±16.1	64.3±11.3	0.071
Male sex	15 (68.2%)	26 (72.2%)	7 (58.3%)	0.668
NYHA Class IV	18 (81.8%)	31 (86.1%)	10 (83.3%)	0.905
BMI (kg/m2)	24.6±3.7	23.9±4.7	24.9±3.3	0.535
Duration of HF (yrs)	4.2±6.4	4.6±5.9	7.2±6.3	0.120
Ischemic etiology	7 (31.8%)	4 (11.1%)	7 (58.3%)	0.004
Diabetes mellitus	4 (18.2%)	5 (13.9%)	5 (41.7%)	0.110
Previous MI	5 (22.7%)	3 (8.3%)	7 (58.3%)	0.001
Atrial fibrillation	4 (18.2%)	6 (16.7%)	3 (25.0%)	0.812
Systolic BP (mmHg)	115.2±30.0	107.9±17.8	116.7±34.7	0.674
Diastolic BP (mmHg)	77.8±24.7	75.8±12.3	77.5±18.6	0.655
Mean BP (mmHg)	90.3±24.4	86.5±12.6	90.6±22.8	0.681
Heart rate (bpm)	86.7±24.0	85.9±17.8	86.5±16.2	0.979
LVEF (%)	33.0±7.7	29.7±7.9	32.9±10.5	0.289
LVDD (mm)	70.2±8.1	73.3±10.5	70.3±11.5	0.491
Creatinine (mg/dL)	1.3±0.3	1.4±0.5	1.8±0.8	0.063
GFR (mL/min)	66.5±26.6	56.4±21.6	40.4±17.2	0.010
Sodium (mEq/L)	136.1±4.9	135.9±5.7	135.4±5.9	0.995
cTnT (ng/mL)	0.006±0.007	0.046±0.020	0.212±0.128	<0.001
CK-MB mass (ng/mL)	1.9±0.8	2.8±1.4	7.3±11.0	<0.001
Dobutamine	9 (40.9)	22 (61.1)	5 (41.7)	0.249

Cardiac troponin T groups: Low (cTnT ≤0.020 ng/mL); Intermediate (cTnT >0.020 or <0.100 ng/mL); High (cTnT ≥0.100 ng/mL); The data are presented as means ± standard deviation or number (percentage); NYHA - New York Heart Association; BMI - body mass index; HF - heart failure; MI - myocardial infarction; BP - blood pressure; LVEF - left ventricle ejection fraction; LVDD - left ventricular-end diastolic diameter; GFR - estimated glomerular filtration rate (Cockcroft-Gault); cTnT - cardiac troponin T; CK-MB - creatine phosphokinase fraction-MB.

Additionally, this study demonstrated that not only the cTnT increase, but also the degree of this marker increase allowed the stratification of the patients with decompensated HF in risk groups for adverse clinical outcomes.

At an age of frugality, the use of biochemical markers and other prognostic indicators for the identification of patients with HF and high risk of death or adverse events can optimize the allocation of resources and stimulate the development of cost-effective therapeutic strategies to improve the outcomes.

Having observed that troponin-T was detected in patients with Chagasic cardiopathy, we concluded that this marker could be used in the follow-up of these patients together with other biomarkers, such as BNP or NT-ProBNP. Additionally, in the patients with the undetermined form of Chagas' disease, the detection of troponins could indicate those at risk for the development of cardiopathy. However, these hypotheses need further studies to be confirmed.

The use of biomarkers of myocardial injury can help the physicians to perfect the clinical decision-making in individual decisions on therapeutic procedures for patients with decompensated HF. Until more data are available, patients with decompensated HF and increased cTnT levels must be submitted to an intensive evidence-based investigation of the HF.

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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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