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Prognostic value of cardiac troponin I in patients with COPD acute exacerbation

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ABSTRACT

Background: Chronic obstructive pulmonary disease (COPD) is frequently associated with right ventricular loading and pulmonary hypertension. We aimed to evaluate a possible association between cardiac troponin I (cTnI) levels and adverse events in hospitalised patients with acute exacerbation of COPD.

Methods: Retrospective cohort study, with analysis of admissions for acute exacerbation of COPD, with cTnI obtained in the first 48 hours of admission. A positive cTnI test was defined as 0.012 ng/ml or higher (99th percentile). Baseline and peak troponin I levels were taken as independent variables, and outcome variables included length of hospital stay, complications during hospitalisation, and in-hospital and extra-hospital mortality (evaluated 18 months post-discharge).

Results: Data concerned 173 patients (105 male, 68 female), with a median age of 77 years (interquartile range of 11 years). The median baseline cTnI was 0.030 ng/ml (n=173), and the median peak cTnI was 0.040 ng/ml (n=173; absolute peak value of 1.260 ng/ml). Nearly 70% of cases had a positive cTnI at admission. Both baseline and peak cTnI correlated significantly with the need for noninvasive ventilatory support. We were not able to find significant differences in in-hospital survival associated with the two troponin groups, but overall 18-month survival was significantly higher among patients with lower values of baseline and peak cTnI.

Conclusions: In patients hospitalised for acute COPD exacerbations, elevated baseline and peak cTnI were associated with a greater need for noninvasive ventilatory support and were significant predictors of 18-month overall survival.

KEYWORDS

COPD, survival, troponin

INTRODUCTION

At present, elevation of plasma cardiac troponin I (cTnI), a biomarker highly specific for myocardial muscle, is a major criterion in the diagnosis of myocardial infarction.[1,2] Nonetheless, the development of cTnI measurement assays has made it possible to study plasma levels amongst the general population, without myocardial ischaemia or necrosis. Increased values of cTnI have been seen in a number of other conditions, including left ventricular hypertrophy, chronic renal insufficiency, diabetes, heart failure and pulmonary embolism.[3,4] Whereas increased troponin I plasma values in the setting of heart failure probably originate in the left heart, in pulmonary embolism the right chambers constitute the probable source for the release of troponin into the circulation.

Chronic obstructive pulmonary disease (COPD) is a lung disease characterised mainly by airflow limitation that is not fully reversible,[5] and corresponds to the major cause of chronic respiratory insufficiency and Cor pulmonale. Cor pulmonale can be defined as right ventricular enlargement (hypertrophy and/or dilatation), caused by pulmonary artery hypertension resulting from diseases affecting the structure and/or function of the lungs, which may, in time, lead to right ventricular failure.[6]

During acute exacerbations, COPD patients, whether or not with a history of Cor pulmonale, have an increased cardiac burden, as stated by Currie *et al.*[7] Therefore, there may be a release of cTnI in these circumstances, and this could have prognostic implications. In fact, Baillard *et al.*, studying a cohort of 71 patients with COPD acute exacerbations, found elevated troponin I values to be a strong predictor of in-hospital death.[8] Higher in-hospital mortality was also seen among heart failure patients with increased plasma troponin levels.[9] Additionally, Harvey *et al.*[10] noted that serum troponins are commonly raised in acute exacerbations of COPD

and appear to reflect the severity of the exacerbation. More recently, Brekke *et al*. studied 897 patients with COPD exacerbation and verified that patients with elevated troponin T levels were at increased risk of death after discharge.[11]

Noninvasive ventilatory support (NIVS) is a crucial strategy in treating the most severe acute COPD exacerbations.[12] In this setting, release of cTnI at presentation, possibly reflecting cardiac strain resulting from respiratory insufficiency, may be an important predictor of patients who will require more aggressive treatment, such as NIVS.

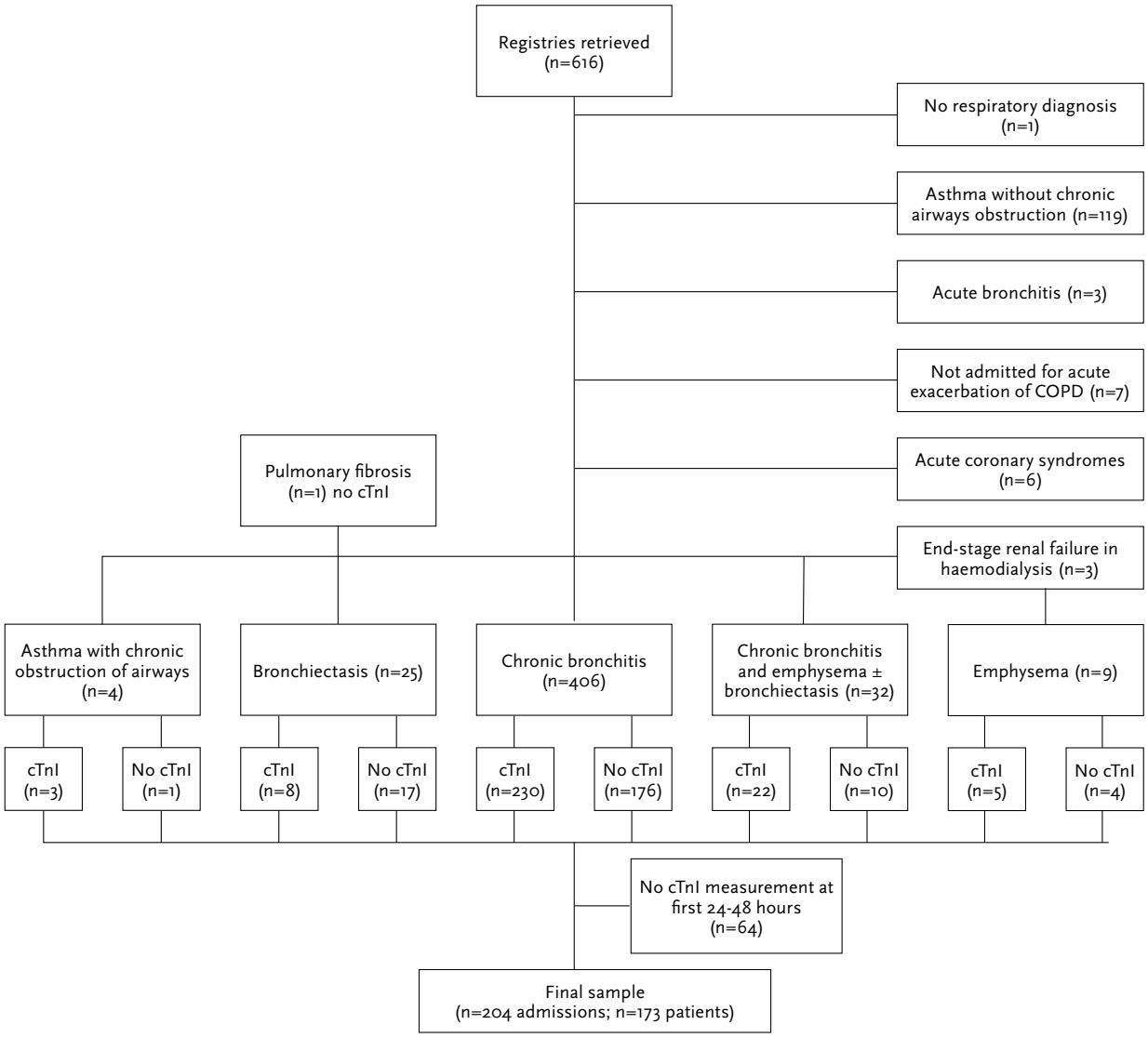
The aims of the present study were to ascertain the range of cTnI values during acute exacerbations of COPD, and evaluate their prognostic implications, namely on mortality and need for NIVS.

METHODS

The present report consists of a retrospective cohort study, which was approved by the institutional ethics board of São João Hospital, Porto, Portugal (*figure 1*). Given the nature of the study, written informed consent was not considered necessary. Nevertheless, verbal consent was obtained from a sample of patients interviewed by means of telephone calls, in which the patients were informed about the study's aim and were asked some questions.

The initial study population consisted of all patients older than 18 years, consecutively admitted for acute exacerbation of COPD to the Department of Internal Medicine of a central university hospital, throughout the year 2007. From this population, we selected the patients who had cardiac

Figure 1. *Flowchart describing a retrospective cohort study involving 173 patients with acute exacerbation of chronic obstructive pulmonary disease*



N = number; cTnI = cardiac troponin I.

troponin I measurement at presentation. Exclusion criteria included marked renal failure (estimated glomerular filtration rate <15 ml/min),[13] persistent haemodynamic instability requiring inotropic or vasoactive support, pulmonary embolism, myocardial infarction and cardiac arrest before admission. All diagnoses were made by the attending physician.

Cases were identified by consulting the electronic records for all admissions to the hospital during the year 2007, with primary discharge coding diagnosis of COPD exacerbation. We retrospectively collected demographic, clinical and analytical data. Missing data were completed by telephone interviews. Demographics included information about gender, ethnic group, age and job. Clinical data incorporated symptoms and signs at admission, comorbidities (particularly obesity, arterial hypertension, ischaemic heart disease, previous myocardial infarction, atrial fibrillation, diabetes, tobacco use), apparent cause of exacerbation, results of standard diagnostic procedures (arterial blood gas (ABG), chest X-ray and electrocardiogram; whenever available, lung function tests and echocardiography), length of hospital stay, and in-hospital complications. Analytical data comprised haematological (globular volume, haemoglobin) and plasma biochemical parameters (cTnI, natriuretic peptide type B (BNP), MB fraction of creatine kinase (CK MB), myoglobin, C-reactive protein, creatinine, sodium and potassium). We considered cTnI measured at baseline (first measurement) and peak troponin level (maximum value recorded during hospitalisation). For patients with more than one admission, we used data concerning only the first admission.

At least 18 months after discharge from hospital, all patients with available contact numbers were contacted by telephone in order to ascertain their vital status and after-discharge intercurrences.

COPD definition fulfilled the ICD-9 classification, and COPD acute exacerbation was ascertained by certifying an increase in cough and dyspnoea, as well as a change in sputum abundance and purulence. Cor pulmonale conformed to the Weitzenblum criteria.[6] Other diagnoses were determined by the attending/discharging physician as documented in the medical records. The severity of COPD exacerbations was evaluated by clinical (altered level of consciousness, signs of respiratory rate exceeding 25 breaths/min, paradoxical abdominal breathing, ineffective cough) and ABG (while breathing room air, arterial oxygen pressure lower than 50 mmHg and blood pH below 7.35) criteria, as stated in medical records. Baseline and peak values of cardiac troponin I were taken as independent variables. For comparison of categorical outcome variables incidence according to both troponin values, we considered them to be categorical variables. For that reason, both baseline and peak cTnI were each categorised in two classes according to the 99th percentile of the cTnI assay available in our hospital: 1) ≤0.012 ng/ml; 2) >0.012 ng/ml. BNP determination, when available, was also included in the calculations. Outcome variables included length of hospital stay, overall complications during hospitalisation, need of NIVS, in-hospital and 18-month post-discharge extra-hospital mortality. The first variable was treated as a continuous variable, and the others as dichotomous categorical variables.

Cardiac troponin I measurement assay

cTnI and BNP measurements were made by means of chemiluminescence's microparticle immunoassay, using the ARCHITECT STAT system, of Abbott Diagnostics (Abbott Park, Illinois, USA). The analytical sensitivity of this cTnI assay was demonstrated to be at ≤0.010 ng/ml, with a 95% confidence level. The 99th percentile of troponin I in a normal population with this assay was established at 0.012 ng/ml. Therefore, all values above this threshold were taken as positive.

Statistical analysis

Continuous variables were presented as median and interquartile range, and categorical variables as absolute and relative frequencies. Univariate comparison of continuous data was performed using the Mann-Whitney U test. Categorical variables were compared using a χ^2 test or Fisher's exact test as appropriate. The correlations between BNP and both baseline and peak cTnI were determined using Pearson's test, and their significance assessed by Kruskal-Wallis test. The correlation of baseline and peak cTnI and length of hospital stay was assessed by simple linear regression, and for that purpose we performed a logarithmic transformation of the independent and dependent variables, as their distributions were asymmetrically positive.

In-hospital and 18-month survival curves were determined using Kaplan-Meier estimates, and were compared between groups (negative *vs* positive) of both baseline and peak cTnI by the log-rank test. To account for baseline covariates, Cox-proportional hazards survival modelling was used. Covariates included gender, age, creatinine and BNP.

For all comparisons a two-sided p value of 0.05 was considered statistically significant. Data analysis was performed using the SPSS 16.0 software programme.

RESULTS

We retrieved 616 registries of admissions with main discharging diagnosis of chronic obstructive pulmonary disease (COPD) and related conditions (codes 490 to 496 of ICD-9), of which 123 were primarily excluded since the patients did not have chronic airways obstruction (119 with

asthma without chronic airways obstruction, three with acute bronchitis, and one with no respiratory diagnosis at all). After analysis of the medical records, we additionally excluded 16 cases (seven were not admitted for acute exacerbation of COPD, six had acute coronary syndromes, and three had end-stage renal disease on haemodialysis). Cardiac troponin I (cTnI) was only ordered in about 56% (n=268) of the remaining 477 admissions confirmed to be COPD with acute exacerbations. For the purpose of our study we considered only those patients who had a first cTnI measured at no more than 48 hours after admission, therefore attaining a final sample of 204 reports, corresponding to 173 patients, whose baseline characteristics are depicted at *table 1*.

Patients had a median age of 77 years (interquartile range of 11 years), with male predominance (105 male, 68 female). Chronic bronchitis was the main diagnosis related to COPD, and previous Cor pulmonale was only seen in 17 cases. Previous medical conditions did not vary significantly in relation to sex, but women were clearly more prone to using β-blockers and diuretics, and men, domiciliary oxygen supplementation (*table 1*).

The median cTnI of the first serum determination was 0.030 ng/ml (n=173), and the median value of peak cTnI was 0.040 ng/ml (n=173; absolute peak value of 1.260 ng/ml). *Tables 2* and *3* summarise the results, according to baseline and peak cTnI categories. Nearly 70% of the patients with COPD who presented to the emergency room with acute exacerbations had positive results of cTnI in the first 48 hours after admission.

The patients with cTnI >99th percentile were significantly older, and in agreement with previous reports, more often had a previous history of congestive heart failure, chronic renal failure and atrial fibrillation or flutter. In addition, they were more likely to have higher values of natriuretic peptide type B (BNP), and also attained higher values of peak cTnI during hospital stay (*table 2*).

BNP was available in only 149 patients. The median of BNP serum determination was 268.4 ng/ml (interquartile

Table 1. *Baseline characteristics of 173 chronic obstructive pulmonary disease (COPD) patients admitted for acute exacerbations*

	Males, n (%) 105 (60.7)	Females, n (%) 68 (39.3)	P value
Age, median (interquartile range)	75.0 (11)	78.5 (9.5)	0.012
COPD, n (%)			
• Chronic bronchitis	90 (85.7)	66 (97.1)	0.140
• Emphysema	25 (23.8)	6 (8.8)	0.011
• Bronchiectasis	16 (15.2)	10 (14.7)	0.822
Known history of Cor pulmonale, n (%)	12 (11.4)	5 (7.4)	0.367
Comorbidities, n (%)			
• Hypertension	58 (55.2)	48 (70.6)	0.074
• Diabetes	29 (27.6)	28 (41.2)	0.201
• AF/AFlt	38 (36.2)	26 (38.2)	0.612
• Chronic heart failure	47 (44.8)	41 (60.3)	0.077
• Ischaemic heart disease	23 (21.9)	19 (27.9)	0.465
• Chronic renal failure	18 (17.1)	11 (16.2)	0.876
• Obesity	20 (19.0)	20 (29.4)	0.292
• Dyslipidaemia	34 (32.4)	31 (45.6)	0.262
• Cerebral vascular disease	18 (17.1)	13 (19.1)	0.741
Chronic medication, n (%)			
• ACEIs	38 (36.2)	30 (44.1)	0.298
• ARBs	9 (8.6)	11 (16.2)	0.132
• Antiplatelets	33 (31.4)	26 (38.2)	0.357
• β-blockers	6 (5.7)	11 (16.2)	0.030
• CCBs	12 (11.4)	8 (11.8)	0.946
• Digitalic	15 (14.3)	16 (23.5)	0.125
• Diuretics	55 (52.4)	47 (69.1)	0.030
• Hypolipidemic drugs	32 (30.5)	21 (30.9)	0.955
• Nitrates	15 (14.3)	11 (16.2)	0.734
• Domiciliary oxygen	54 (51.4)	24 (35.3)	0.038
Hospital stay length (days), median (interquartile range)	10 (6)	9 (4.8)	0.238
Laboratory data, median (interquartile range)			
• Baseline cTnI	0.030 (0.065)	0.026 (0.046)	0.263
• Peak cTnI	0.040 (0.093)	0.031 (0.064)	0.552
• CRP	36.6 (96.3)	19.1 (57.6)	0.027
• Creatinine	1.10 (0.51)	0.94 (0.36)	0.003
• BNP	246.1 (524.6) (n=83)	275.3 (455.4) (n=66)	0.829

N = number; p = probability; AF = atrial fibrillation; Aflt = atrial flutter; ACEI = angiotensin converting enzyme inhibitor; ARB = angiotensin receptor blocker; CCB = calcium channel blocker; cTnI = cardiac troponin I; peak cTnI = maximum value for cTnI; CRP = C-reactive protein; BNP = natriuretic peptide type B.

Table 2. *Data as obtained in 173 chronic obstructive pulmonary disease (COPD) patients admitted for acute exacerbations, according to baseline cTnI categories*

	cTnI ≤0.012 ng/ml (n=52)	cTnI >0.012 ng/ml (n=121)	P value
Age, years, median (interquartile range)	74 (12.3)	78 (9)	0.001
Sex, n (%)			
• Female	22 (42.3)	46 (38.0)	0.596
• Male	30 (57.7)	75 (62.0)	
Peak cTnI, ng/ml, median (interquartile range)	0.010 (0.002)	0.060 (0.100)	0.000
Creatinine, mg/dl, median (interquartile range)	0.90 (0.38)	1.05 (0.52)	0.011
BNP, pg/ml, median (interquartile range)	131.75 (216.1) (n=46)	322.20 (621.7) (n=103)	0.000
Hospital stay length, days, median (interquartile range)	8 (5)	11 (6)	0.014
In-hospital mortality, n (%), median (interquartile range)	2 (3.9)	9 (7.4)	0.383
Noninvasive ventilatory support, n (%)	12 (23.1)	54 (44.6)	0.009
Comorbidities, n (%)			
• Hypertension	31 (59.6)	75 (62.0)	0.618
• Diabetes	14 (26.9)	41 (33.9)	0.314
• AF/AFlt	11 (21.2)	53 (43.8)	0.004
• Chronic heart failure	22 (42.3)	69 (57.0)	0.054
• Ischaemic heart disease	10 (19.2)	33 (27.3)	0.229
• Chronic renal failure	5 (9.6)	23 (19.0)	0.117
• Obesity	11 (21.2)	27 (22.3)	0.798
• Dyslipidaemia	18 (34.6)	48 (39.7)	0.452
• Cerebral vascular disease	13 (25)	17 (14.0)	0.101

N = number; p = probability; cTnI = cardiac troponin I; peak cTnI = maximum value for cTnI; AF = atrial fibrillation; Aflt = atrial flutter; BNP = natriuretic peptide type B.

Table 3. *Data as obtained in 173 chronic obstructive pulmonary disease (COPD) patients admitted for acute exacerbations, according to maximum cTnI categories*

	Peak CTnI ≤0.012 ng/ml (n=42)	Peak CTnI >0.012 ng/ml (n=131)	P value
Age, years, median (interquartile range)	74 (10)	78 (10)	0.001
Sex, n (%)			
• Female	15 (35.7)	53 (40.5)	0.584
• Male	27 (64.3)	78 (59.5)	
Baseline cTnI, ng/ml, median (interquartile range)	0.010 (0)	0.040 (0.062)	0.000
Creatinine, mg/dl, median (interquartile range)	0.91 (0.39)	1.04 (0.48)	0.102
BNP, pg/ml, median (interquartile range)	114.75 (219.01)	308.60 (585.10)	0.000
Hospital stay length, days, median (interquartile range)	8 (5)	10 (6)	0.005
In-hospital mortality, n (%)	1 (2.4)	10 (7.6)	0.252
Noninvasive ventilatory support, n (%)	7 (16.7)	59 (45.0)	0.020
Comorbidities, n (%)			
• Hypertension	25 (59.5)	81 (61.8)	0.789
• Diabetes	11 (26.2)	44 (33.6)	0.372
• AF/AFlt	8 (19.04)	56 (42.7)	0.007
• Chronic heart failure	18 (42.9)	73 (55.7)	0.148
• Ischaemic heart disease	8 (19.1)	35 (26.7)	0.319
• Chronic renal failure	5 (11.9)	23 (17.6)	0.390
• Obesity	9 (21.4)	29 (22.1)	0.923
• Dyslipidaemia	14 (33.3)	52 (39.7)	0.461
• Cerebral vascular disease	8 (19.0)	22 (16.8)	0.737

N = number; p = probability; cTnI = cardiac troponin I; peak cTnI = maximum value for cTnI; AF = atrial fibrillation; Aflt = atrial flutter; BNP = natriuretic peptide type B.

range = 482.1 ng/ml). Baseline and peak cTnI values were significantly correlated with one another (r=0.74, p<0.001). Conversely, neither showed any significant correlation with BNP (r=0.06, p=0.438 and r=0.07, p=0.430 for baseline and peak cTnI, respectively). As depicted in *table 4*, the larger proportion of patients had BNP levels between 100 and 500 pg/ml. Kruskal-Wallis test demonstrated that median peak troponin (but not baseline cTnI) varied significantly (p=0.031) among the BNP categories.

Table 4. *Data as measured in a subset of 149 chronic obstructive pulmonary disease (COPD) patients admitted for acute exacerbations*

	BNP categories			
	≤100 pg/ml	100.01-499.99 pg/ml	≥500 pg/ml	Total
Baseline cTnI ≤0.012 ng/ml	16	18	11	45
Baseline cTnI >0.012 ng/ml (a)	20	51	33	104
Peak cTnI ≤0.012 ng/ml	13	13	10	36
Peak cTnI >0.012 ng/ml (b)	23	56	34	113
Total	*36*	*69*	*44*	*149*

Patients were divided by natriuretic peptide type B (BNP) levels, according to cardiac troponin I (cTnI), either baseline (top) or peak value (bottom). (a) Probability of 0.057 *vs* patients with baseline cTnI ≤0.012 ng/ml, Kruskal-Wallis test; (b) probability of 0.031 *vs* patients with peak cTnI ≤0.012 ng/ml, Kruskal-Wallis test.

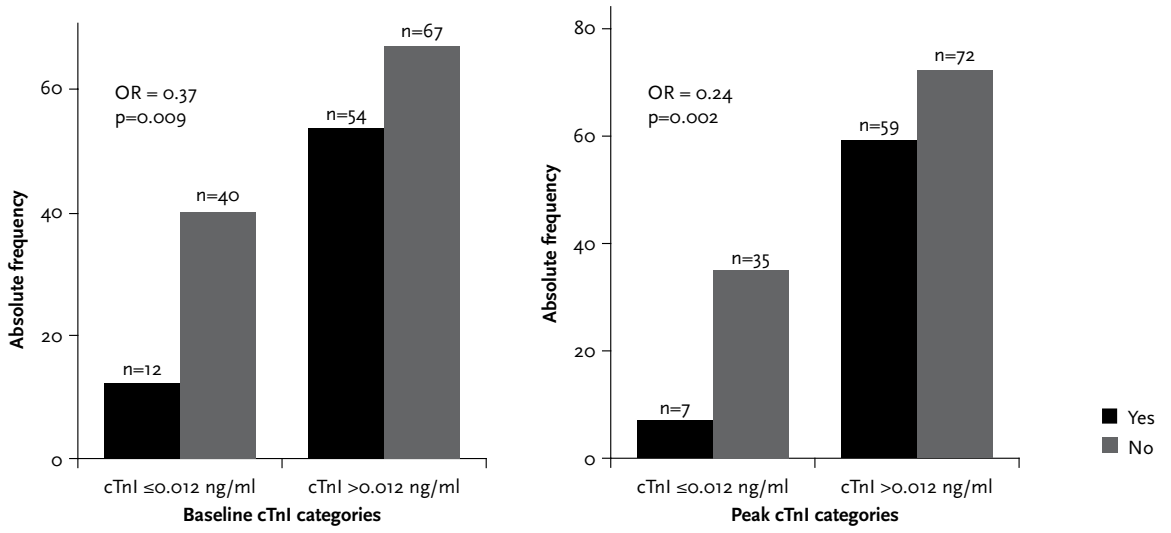
Length of hospital stay differed significantly when comparing both baseline (p=0.014) and peak cTnI (0.005) in the two patient groups (negative/positive). Nonetheless, when applying the linear regression method, only peak cTnI predicted (p=0.046) the hospitalisation period.

Overall complications were significantly correlated with cTnI at admission; they were less likely to occur (OR=0.397) if the cTnI was under the 99th percentile (p=0.007). The same relation was observed with peak cTnI (OR=0.344, p=0.005). Requirement of noninvasive ventilatory support was the main complication, occurring in nearly 38% of patients (n=66). We found that patients had a 63% lower probability (p=0.009) of requiring noninvasive ventilatory support if they had basal cTnI values under 0.012 ng/ml (*figure 2*). Noninvasive ventilatory support requirement also differed significantly according to the peak cTnI categories (OR=0.24, p=0.002). As shown in *figure 2*, patients with lower levels of peak cTnI had a 76% lower probability of needing that type of ventilation.

The in-hospital death rate was 5.9%. Vital status at discharge did not vary significantly according to either baseline or peak cTnI. Kaplan-Meier survival analysis did not show any significant differences in overall in-hospital survival, whether the patients first cTnI measurement was positive or negative. The same holds true for peak cTnI. Controlling for sex, age, BNP and creatinine, Cox regression analysis did not demonstrate any statistically significant differences in terms of in-hospital survival either.

Data concerning 18-month survival could not be obtained for 13 patients, whose records did not include a valid telephone number. The 18-month post-discharge death rate was 21.1%. In contrast to the data concerning in-hospital

Figure 2. *Distribution of patients in terms of need for noninvasive ventilatory support (NIVS), according to baseline and peak cardiac troponin I (cTnI) categories, using the Mantel-Haenszel common odds ratio estimation**



***From a cohort of 173 chronic obstructive pulmonary disease (COPD) patients admitted for acute exacerbations.**
The probability of requiring noninvasive ventilatory support was significantly higher in patients with either baseline or peak cTnI above 0.012ng/ml. N = number; OR = odds ratio; p = probability.

survival, overall 18-month survival analysis revealed a statistically significant difference (p=0.007) when comparing patients who had a baseline cTnI lower than the 99th percentile to those in the other group (*figure 3*). Identical to baseline cTnI, peak cTnI also predicted overall 18-month survival (p=0.012). Moreover, by means of Cox regression analysis, adjusting for the same variables, it was shown that overall survival continued to be significantly higher (p=0.030) among those patients with negative initial cTnI. A similar finding was observed with peak cTnI (p=0.021).

DISCUSSION

In the present study, about 70% of patients with COPD acute exacerbations had a positive cTnI at presentation. It has been shown previously that patients with COPD encompass a great prevalence of cardiovascular diseases,[15] mainly chronic heart failure and pulmonary embolism, but also ischaemic heart disease. In this cohort, according to the retrospective character, the diagnoses were made by the attending/discharging physician, posing a problem in terms of accurate exclusion of all those comorbidities known to raise cardiac troponin.

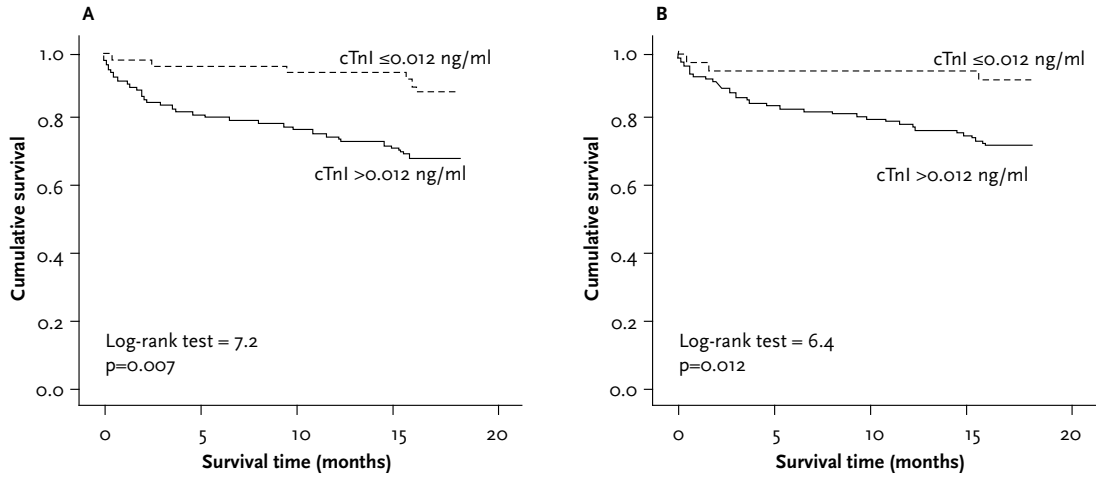
Regarding interpretation of small increases of cardiac troponin, mostly in patients with nonischaemic heart disease, many studies have been carried out during the last decade. Initially, increased plasma levels of cardiac troponin I were presumed to be due to myocardial necrosis, as happens in myocardial infarction. Antman *et al*. studied a group of 1404 patients with acute coronary syndrome and found a higher mortality rate in patients with increased values of plasma cardiac troponin I.[2] Moreover, increased values of plasma cardiac troponins (either I or T) were found in a number of other clinical situations, including heart failure and sepsis.[16,17] Pulmonary embolism is also a relatively frequent cause of increased plasma troponin.[18] In the case of pulmonary embolism, myocardial necrosis is not a prominent phenomenon, and right ventricular strain could be the cause of troponin release.[19] COPD patients could have a similar mechanism for troponin release as pulmonary embolism patients, since right ventricular overload is a prominent phenomenon in this condition.

Hessel *et al*. showed that in the presence of compounds that stimulate stretch-responsive integrins, viable cardiomyocytes release intact cardiac troponin I.[20] Previous work by Feng *et al*. had shown preload, rather than ischaemia, to induce troponin I degradation.[21] COPD patients frequently have hypoxia, and thus the hypothesis that hypoxia also plays a role in troponin release in COPD patients cannot be ruled out.

Peacock *et al*. studied troponin levels in 84,872 patients with heart failure, and found higher in-hospital mortality in patients with elevated troponin levels.[9] In COPD patients, both Baillard *et al*.[8] and Brekke *et al*.[11] found troponin levels to correlate with certain outcomes – in-hospital death and death after discharge, respectively. The present results are in good agreement with these latter findings.

It seems important to account for heart failure (whether overt or overlooked) when dealing with COPD patients presenting for an acute exacerbation. In fact, it was recently demonstrated that left ventricular dysfunction is closely related to COPD acute exacerbations.[22,23] BNP has a

Figure 3. *Kaplan-Meier survival analysis of 160 chronic obstructive pulmonary disease (COPD) patients admitted for acute exacerbations, comparing baseline cardiac troponin I (cTnI) values (A) and peak cTnI levels (B)*



The overall 18-month mortality was significantly higher in cTnI-positive when compared with cTnI-negative patients (probability = 0.007 and 0.012, respectively). p = probability.

negative predictive value for heart failure when under 100 pg/ml and is strongly in favour of left ventricular failure when above 500 pg/ml.[22] However, increased BNP has been seen in mitral stenosis, a disease in which the left ventricle is unaffected.[24]

As stated previously, in the present study, patients who had positive cardiac troponin I were older, had higher BNP values, more often had a history of congestive heart failure and atrial fibrillation/flutter. In addition, cardiac troponin I values, either positive or negative, varied significantly among BNP classes. Myocardial strain could be an important cause of troponin release in these patients. As the majority of patients had BNP levels between 100 and 500 pg/ml, cardiac troponin may result from right ventricular dysfunction, from moderate left ventricular failure, or from both.

The overall in-hospital complications were found to be significantly higher among patients with either baseline cTnI or peak cTnI plasma levels above the 99th percentile. These patients were also more prone to have longer hospital stays, leading to the hypothesis that those complications could be related to hospital stay.

Patients with either baseline cTnI or peak cTnI plasma levels ≥0.012 ng/ml were more likely to require noninvasive ventilatory support. According to this finding, we might add hypoxaemia as a contributing factor for troponin release, as well as subsequent myocardial strain (perhaps also because of tachycardia).

Both Baillard *et al.*[8] and Brekke *et al.*[11] found troponin levels to correlate with in-hospital death and death after discharge, respectively. Consistent with this, our study demonstrated significantly higher odds of dying during the first 18 months after discharge amongst patients with elevated cTnI at presentation or at peak level. Patients with elevated cTnI had a higher prevalence of both heart and renal failure; these diseases are in their own right predictors of survival, and this could contribute to our findings.

Study limitations

As the present report derives from a retrospective study, data were ascertained from past records, possibly leading to information bias; in addition, the outcomes (hospital stay length, in-hospital complications, in-hospital survival and survival after discharge) were evaluated at a later date. However, the outcomes chosen are objective parameters. Bias from nonresponse and losses to follow-up were seen in what concerns the 18-month mortality data (13 patients). Lack of control for possible independent variables may have occurred, namely concerning the fact that cTnI was measured only in 56% of acute COPD exacerbations. Given these limitations, it would be better to have the conclusions of the present report confirmed by other studies.

CONCLUSIONS

The present study showed that a great proportion of patients hospitalised for acute exacerbations of COPD have levels of cTnI above the 99th percentile. Both left ventricular and right ventricular dysfunction could be implicated as sources of cTnI release in this context.

Noninvasive ventilatory support requirement was significantly more likely to occur among patients with elevated cTnI (≥0.012 ng/ml – 99th percentile), when compared with those patients with lower levels. Elevated levels of both baseline and peak cTnI were also found to be significant predictors of 18-month overall survival.

NOTE

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