

The Clinical Significance of Highly Sensitive Cardiac Troponin T in Patients with Acute Exacerbation of Chronic Obstructive Pulmonary Disease in the Emergency Department

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Abstract

Introduction: Chronic obstructive pulmonary disease is one of the main causes of morbidity, mortality, and health-care costs entire the world. During acute exacerbations, whether or not with a history of Cor pulmonale have an increased cardiac burden. Patients with COPD are at increased risk of cardiovascular disease, exacerbations increase strain on the heart. The prognostic and predictive value of highly sensitive troponin T seen during COPD exacerbations has been investigated.

Aim: Assessment the clinical significance of highly sensitive troponin T (Hs cTnT) as a predictive and prognostic factor in patients with acute exacerbations of COPD.

Patients and methods: This observational cross sectional study was carried on 79 patients from 2 May 2013 to 1 May 2015 with acute COPD exacerbation.

Inclusion criteria: Patients with acute exacerbation of COPD.

Exclusion criteria: Patients with severe renal impairment, persistent hemodynamic instability, myocardial infarction and cardiac arrest before admission demographics. full medical history, vital signs, ABGs and ECG were recorded. Serum cardiac enzymes CK, CK-MB and highly sensitive cardiac troponin T (hs cTnT) were measured.

Results: 79 patients with acute exacerbation of COPD were enrolled. Mortality rate was (2.53%) Hs cTnT level showed a statistically significant difference comparing four reported categories of COPD exacerbation severity. Only life threatening form showed significantly higher hscTnT level compared to moderate and severe forms.

Conclusion: The study showed that highly sensitive cardiac Troponin T was significantly elevated in COPD patients with exacerbation. Troponin T may help the assessment and the prognosis in patients with COPD exacerbations.

Keywords: Chronic obstructive pulmonary disease; Highly sensitive troponin T

Introduction

Chronic obstructive pulmonary disease (COPD) is a lung disease characterized mainly by airflow limitation that is not fully reversible and corresponds to the major cause of chronic respiratory failure and Cor pulmonale [1]. Globally, COPD is one of the main and significant cause of morbidity, mortality, and health-care costs. It is a global health issue, with cigarette smoking being an important risk factor universally [2]. COPD is presently the fourth leading cause of death in the world, being accountable for 3.8% of total deaths, and it was the sixth leading cause of death in nations of low and middle income, accounting for 4.9% of total deaths. It is predictable to be the third cause of death by 2020 in the world [3]. In spite to a gradual progression of symptoms and deterioration of respiratory function, most patients suffering from episodes of symptom that become worse (exacerbations). During

exacerbations, patients frequently need hospitalization, and mortality is increased. A relapse could be defined when the exacerbation definition is made within 28 days of a previous exacerbation [4]. COPD exacerbation could in itself cause sufficient strain on the heart to induce myocardial cell necrosis. Edema, mucus hypersecretion and bronchoconstriction may cause additional ventilation impairment, and alveolar hypoxia may cause constriction of pulmonary arterioles and enhanced pulmonary artery pressure, disturbing perfusion. Tachycardia, hypoxemia and dilatation of the right ventricle and pulmonary arterial hypertension are generally seen in COPD exacerbations [5]. During the acute attack exacerbations, patients whether or not with a history of Cor pulmonale, have an increased cardiac burden, as proved by Currie et al. [6]. "Based on the WHO criteria for the definition of acute myocardial infarction for Hs cTnT is 14 ng/L or 0.014 ng/mL as determined from ROC analysis of the results from an earlier test generation of the Elecsys Troponin T assay" [7]. Since patients with COPD are at greater risk of cardiovascular disease, exacerbations of which increase strain on the heart [8]. The

prognostic and predictive value of circulating levels of highly sensitive troponin T seen during COPD exacerbations has been investigated.

Aim of the Work

To assess the level of highly sensitive cardiac troponin T (hs cTnT) value in patients with acute exacerbations of COPD and to evaluate the short-term prognostic value of highly sensitive cardiac troponin T (hs cTnT), as regards admission to the medical ward, admission to the Intensive Care Unit (ICU) and mechanical ventilation.

Patients and Methods

This study was made in an observational cross sectional study was conducted in patients with acute exacerbation of COPD admitted to the Emergency Department at Suez Canal University Hospital on the second of May 2013 to the first of May 2015. The calculated sample size was 79 patients.

Inclusion criteria

Both genders, adult patients (more than 40 years old) with acute exacerbation of COPD.

Exclusion criteria

The following patients were excluded from the study: patients with severe renal impairment, persistent hemodynamic instability, myocardial infarction and cardiac arrest before admission. The clinical data were collected by the researcher in a pre-organized data sheet for each patient, the following was studied: Socio-demographic data, clinical evaluation regarding vital signs, general and various body systems examination and grading of the severity of COPD exacerbation.

Laboratory investigations

Complete blood count, arterial blood gases, renal functions, Serum cardiac enzymes Ck, CK-MB and highly sensitive cardiac troponin T (hs cTnT) were measured.

Treatment and follow up on

Patients followed up for the effectiveness of the emergency management and outcome.

Results

This study was approved as an observational cross sectional study. 79 patients with acute exacerbation of COPD were enrolled in the study.

Discussion

The present study showed that most of the studied patients were males (68%) with a mean age of 62.67 years (Figure 1). This also is consistent with another German study by Rabe et al. [9] in which most of the patients were males (71.7%) with a mean age 63 years. The most common risk factor in our study for COPD was cigarette smoking (Table 1). Only 7.6% of patients were never smokers (occupational exposure) which match with another study which was done by Roche et al. [10] in which 6.6% of patients were never smokers. Cardiomegaly was found to be most prevalent comorbidity among the studied

patients (27.85%) and chronic liver disease account (7.59%) compared to Chang et al. [11] study in which Cardiomegaly was found to be (30.1%) which match with this study results while chronic liver disease account (0.8%). More than half of the patients (65%) in our study had arterial O₂ tension less than 60 mmHg and had arterial PCO₂ more than 60 mmHg on presentation to the ED. This match to a near extent to another study was done by Soyseth et al. [12] in which thirty of the patients among the references had arterial O₂ tension less than 60 mmHg and 50% of the AECOPD patients had arterial PCO₂ on more than 60 mmHg arrival to the hospital. We have found that AECOPD is associated with significant elevation of troponin T measured by a highly sensitive assay which matches with Soyseth et al. [12] study. The mean cardiac troponin level in our study was 0.0385 ng/l. In our study, the best cutoff point value for hscTnT to predict ICU admission was more than 0.053 ng/l. Our results match with a study did by Pal H Brekke et al. [13] in which hscTnT was considered elevated at levels equal to or greater than 0.04 ng/l. Hospital policy at that time recommended a hscTnT concentration of 14ng/l or higher as the diagnostic threshold for myocardial infarction (MI). Acidosis and hypoxemia were strongly associated with cTnT elevation. Indeed, in our study of 2 patients died in the ED (2.53%) which was fairly similar to Roche et al. [14] study in which (1.25%) of the patients died in the ED. We did record the established ECG criteria of ischemia, but the ST-T changes may be nonspecific, resulting in false positive ischemia but this was excluded by measuring CK and CK MB. The significant information obtained from the present study is that AECOPD patients without a history of coronary heart disease have high circulating levels of troponin T and troponin T levels were also elevated associated with the severity of disease.

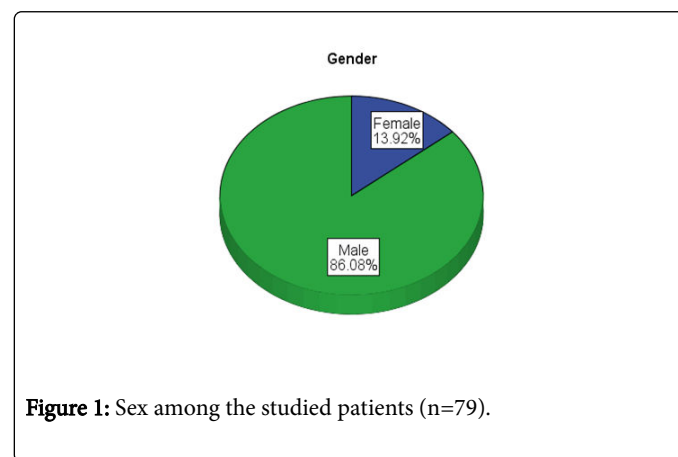


Figure 1: Sex among the studied patients (n=79).

		Number (79)	Percentage
Age	47 - <60	32	40.51%
	60 - <70	24	30.38%
	≥ 70 – 86	23	29.11%
	Mean ± SD	62.67 ± 9.55	
	Range	47 – 86	

Table 1: Age among the studied patients (n=79).

Table 2 shows that the most common risk factor for COPD was cigarette smoking whether alone (40.5%) or with shisha smoking (39.2%).

		Number (79)	Percentage
Risk factor for COPD	Cigarette smoking	63	79.70%
	Shisha smoking	36	45.60%
	Baking	5	6.30%
	Occupational exposure	6	7.60%
Smoking index (pack. Year)	Mean ± SD	43.71 ± 13.48	
	Range	15-122	
Duration of smoking	Mean ± SD	35.92 ± 9.81 years	
	Range	15-55 years	

Table 2: Risk factor for COPD among the studied patients (n=79).

As presented in Figure 2, only life threatening from showed significantly higher hs cTnT level compared to moderate and severe forms.

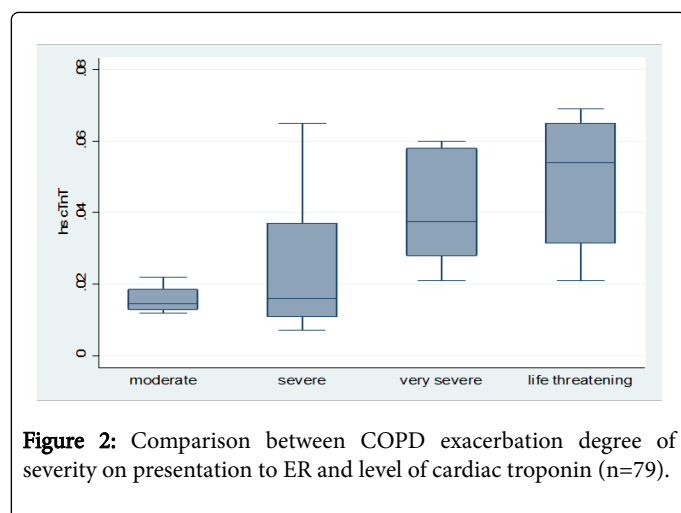


Figure 2: Comparison between COPD exacerbation degree of severity on presentation to ER and level of cardiac troponin (n=79).

Cause of exacerbation	Number	Percentage
Infection	72	91.14%
Idiopathic	7	8.86%

Table 3: Clinical characteristics among the studied patients (n=79).

Table 3 shows that infection was the prevailing apparent cause of exacerbation based on clinical and/or radiological findings (91.14%). Clinical findings of infection include high fever and increased total leucocytic count.

Figure 3 shows that cardiac troponin level was higher among death cases and ICU admitted patients compared to discharged patients and inpatient admitted patients with statistically significant difference.

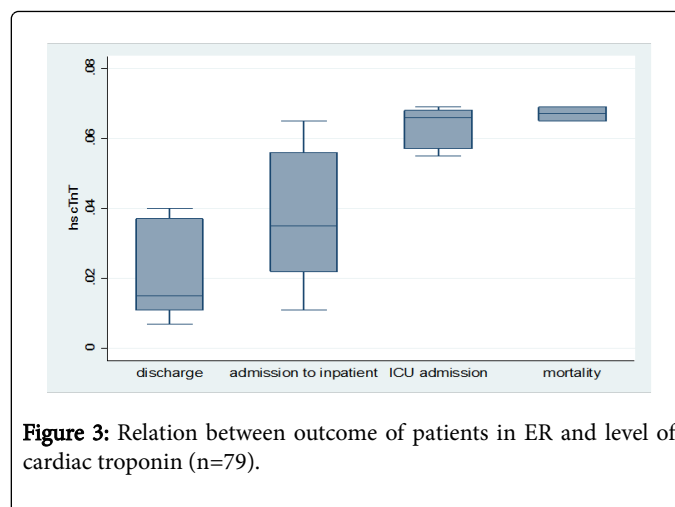


Figure 3: Relation between outcome of patients in ER and level of cardiac troponin (n=79).

Table 4 shows that Sinus tachycardia was the most ECG changes seen among the studied patients (72.15%).

ECG changes	Number	Percentage
Sinus tachycardia	57	72.15%
Nonspecific ischemic changes	18	22.78%
No change	20	25.31%

Table 4: ECG changes among the studied patients (n=79).

Figure 4 shows that discharged patients have significantly lower hs cTnT levels compared to all non-discharged patients.

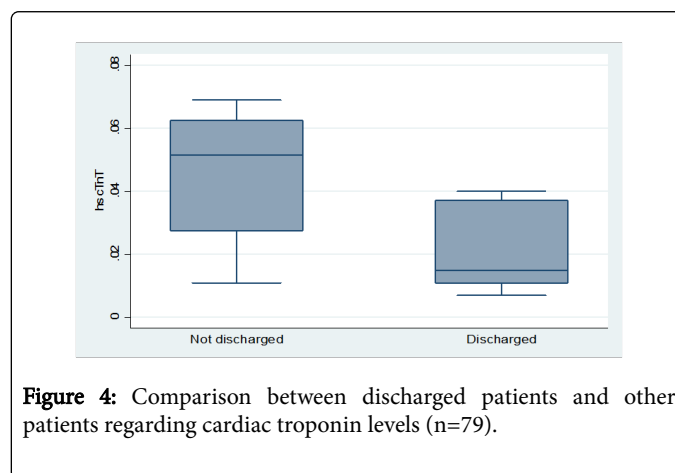


Figure 4: Comparison between discharged patients and other patients regarding cardiac troponin levels (n=79).

Table 5 shows that there was no statistically significant difference regarding ABG findings before and after initial ER treatment except for PO₂.

ABG	On presentation to ER	After initial treatment	p-value
	Mean ± SD	Mean ± SD	
pH	7.29 ± 0.11	7.32 ± 0.08	0.1 (NS)
PO ₂	56.1 ± 14.1	66.72 ± 12.6	0.001*

PCO ₂	56.4 ± 15.8	53.6 ± 13.28	0.2 (NS)
HCO ₃	25.8 ± 6.27	25.41 ± 5.69	0.6 (NS)
*statistically significant difference (p-value <0.05) NS: no statistically significant difference (p-value >0.05)			

Table 5: ABG on presentation to ER and after initial treatment among studied patients (n=79).

Table 6 shows that mean cardiac troponin level was 0.0385 ng/L with wide range from 0.007 to 0.069 ng/L.

Cardiac troponin ng/L	
Range	0.007 – 0.069
Mean ± SD	0.0385 ± 0.0199
Median	0.037

Table 6: High sensitive cardiac troponin (hs cTnT) among studied patients (n=79).

Table 7 comparison between patients admitted to the general ward and patients admitted to ICU regarding cardiac troponin levels (n=79).

Cardiac troponin	Admitted to the general ward (n=42)	Admitted to ICU (n=12)	p-value
Mean ± SD	0.039 ± 0.017	0.063 ± 0.006	0.001*

Table 7: Patients admitted to the general ward and patients admitted to ICU regarding cardiac troponin levels (n=79).

Conclusion

Highly sensitive cardiac Troponin T was significantly elevated in COPD patients with exacerbation. Troponin T will help clinicians to assess prognosis in exacerbations of COPD, but more researches are required to see if they could affect the plan of management.

References

- Rabe KF, Hurd S, Anzueto A, Barnes PJ, Buist SA, et al. (2007) Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. *Am J Respir Crit Care Med* 176: 532-555.
- Mannino DM, Buist AS (2007) Global burden of COPD: risk factors, prevalence, and future trends. *Lancet* 370: 765-773.
- Celli BR, Cote CG, Marin JM (2004) The body mass index, airflow obstruction, dyspnea, and exercise capacity index in obstructive pulmonary disease. *N Engl J Med* 350: 1005-1012.
- Sin DD, Tu JV (2000) Are elderly patients with obstructive airway disease being prematurely discharged? *Am J Respir Crit Care Med* 161: 1513-1517.
- Torbicki A, Kurzyna M, Kuca P (2003) Detectable serum cardiac troponin T as a marker of poor prognosis among patients with chronic precapillary pulmonary hypertension. *Circulation* 108: 844-848.
- Currie GP, Wedzicha JA (2006) ABC of chronic obstructive pulmonary disease. Acute exacerbations. *BMJ* 333: 87-89.
- Müller-Bardorff M, Hallermayer, K Schröder A (1997) Improved troponin T ELISA specific for cardiac troponin T isoform: Assay development and analytical and clinical validation. *Clin Chem* 43: 458-466.
- Kampmann M, Rauscher T, Müller-Bardorff M, Münch M, Klein G, et al. (1997) Clinical evaluation of Troponin T and CK-MB mass on the Elecsys 2010 analyzer. *European Congress of Clinical Chemistry*.
- Rabe KF, Fabbri LM, Vogelmeier C, Kögler H, Schmidt H, et al. (2013) Seasonal distribution of COPD exacerbations in the prevention of exacerbations with tiotropium in COPD trial. *Chest* 143: 711-719.
- Roche N, Chavaillon JM, Maurer C, Zureik M, Piquet J (2014) A clinical in-hospital prognostic score for acute exacerbations of COPD. *Respir Res* 15: 99.
- Chang CL, Robinson SC, Mills GD, Sullivan GD, Karalus NC, et al. (2011) Biochemical markers of cardiac dysfunction predicts mortality in acute exacerbations of COPD. *Thorax* 66: 764.
- Søyseth V, Bhatnagar R, Holmedahl NH, Neukamm A, Høiseth AD, et al. (2013) Acute exacerbation of COPD is associated with four fold elevation of cardiac troponin T. *Heart* 99: 122-126.
- Brekke PH, Omland T, Holmedal SH, Smith P, Søyseth V (2009) Determinants of cardiac troponin T elevation in COPD exacerbation- A cross-sectional study. *BMC Pulm Med* 9: 35.
- Roche N, Zureik M, Soussan D, Neukirch F, Perrotin D (2008) Predictors of outcomes in COPD exacerbation cases presenting to the emergency department. *Eur Respir J* 32: 953-961.