Prognostic Value Of Cardiac Troponin I During Acute Exacerbation Of Chronic Obstructive Pulmonary Disease

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ABSTRACT:
Introduction: Chronic Obstructive Pulmonary Disease (COPD) is a leading cause of morbidity and mortality worldwide. Acute exacerbations of COPD (AECOPD) significantly impact patient outcomes and healthcare costs. Recent studies have shown interest in cardiac troponin I (cTnI) as a potential prognostic marker in AECOPD. This study aimed to evaluate the prognostic value of cTnI during AECOPD.
Methods: This prospective observational study included 90 patients admitted with AECOPD over 18 months. Patients were divided into two groups based on admission cTnI levels: elevated (>0.01 ng/ml) and normal (<0.01 ng/ml). Demographic data, medical history, physical examination findings, and diagnostic test results were recorded. Outcomes including ICU admission, need for ventilation, length of stay, and mortality were assessed.
Results:cTnI was elevated in 37.77% of patients. These patients had significantly longer COPD duration (p=0.002), higher incidence of ischemic heart disease (p=0.018), and lower SpO2 levels (p=0.003). Elevated cTnI was associated with higher rates of ICU admission (94.11% vs 51.78%, p=0.002) and need for ventilatory support (79.4% vs 39.28%, p=0.006). However, there were no significant differences in length of hospital stay or in-hospital mortality between the two groups.
Conclusion: Elevated cTnI during AECOPD is associated with higher disease severity and increased need for intensive care. While it does not predict length of stay or in-hospital mortality, cTnI can serve as a valuable marker for identifying high-risk patients at admission. Further studies are needed to evaluate long-term outcomes in patients with elevated cTnI during AECOPD.

Keywords: Chronic Obstructive Pulmonary Disease (COPD), Acute Exacerbation, Cardiac Troponin I, Prognostic Value, Intensive Care, Ventilatory Support

INTRODUCTION:

Chronic Obstructive Pulmonary Disease (COPD) is a progressive respiratory condition characterized by persistent airflow limitation and recurrent exacerbations [1]. These acute exacerbations of COPD (AECOPD) are associated with significant morbidity, mortality, and healthcare costs [2]. As the global burden of COPD continues to rise, there is an urgent need for reliable prognostic markers to guide clinical decision-making and improve patient outcomes during these critical episodes.

Cardiac troponin I (cTnI) is a highly sensitive and specific biomarker for myocardial injury, widely used in the diagnosis and risk stratification of acute coronary syndromes [3]. In recent years, there has been growing interest in the potential role of cTnI as a prognostic indicator in non-cardiac conditions, including COPD [4]. Elevated levels of cTnI have been observed in patients with AECOPD, even in the absence of clinically apparent cardiac ischemia [5].

The pathophysiological mechanisms underlying the elevation of cTnI during AECOPD are complex and multifactorial. Hypoxemia, systemic inflammation, and increased cardiac workload associated with AECOPD can lead to myocardial stress and injury [6]. Additionally, the high prevalence of cardiovascular comorbidities in COPD patients may contribute to the susceptibility of the myocardium to injury during acute exacerbations [7].

Several studies have explored the relationship between cTnI levels and clinical outcomes in patients with AECOPD. Elevated cTnI has been associated with increased length of hospital stay, need for mechanical ventilation, and short-term mortality [8]. However, the prognostic value of cTnI in AECOPD remains a subject of debate, with conflicting results reported in the literature [9].

This thesis aims to comprehensively evaluate the prognostic value of cTnI during AECOPD. By analyzing a large cohort of patients admitted for AECOPD, we seek to determine whether cTnI levels on admission can predict important clinical outcomes, including in-hospital mortality, length of stay, need for intensive care unit admission, and readmission rates. Furthermore, we will explore the relationship between cTnI and established prognostic markers in COPD, such as forced expiratory volume in one second (FEV1) and the BODE index (Body mass index, airflow Obstruction, Dyspnea, and Exercise capacity) [10].

Understanding the prognostic significance of cTnI in AECOPD could have important implications for clinical practice. If validated as a reliable predictor of outcomes, cTnI could be incorporated into risk stratification algorithms, helping clinicians identify high-risk patients who may benefit from more intensive monitoring and management strategies. Moreover, it could provide valuable insights into the complex interplay between cardiac and pulmonary pathophysiology in COPD exacerbations.

This research will not only contribute to the growing body of knowledge on biomarkers in COPD but also pave the way for future studies exploring targeted interventions based on cTnI levels. By elucidating the prognostic value of cTnI in AECOPD, we aim to improve patient care and outcomes in this challenging clinical scenario.

METHODS:

The study was a prospective observational study conducted over 18 months from February 2021 to September 2022. It included 90 patients admitted with acute exacerbation of COPD who met the inclusion criteria. Patients with marked renal failure, hemodynamic instability requiring inotropic support, pulmonary embolism, myocardial infarction, sepsis, or cardiac arrest before admission were excluded.

Upon admission, informed written consent was obtained from all patients. Demographic data, medical history, and symptoms were recorded. A detailed physical examination was performed, including calculation of the Glasgow Coma Score. Patients underwent various tests including arterial blood gas analysis, chest X-ray, electrocardiogram (ECG), echocardiography, and spirometry when possible. Routine blood and biochemical investigations, including Troponin I, were also conducted.

Blood samples for cardiac Troponin I (cTnI) were measured on admission using a quantitative assay by immunofluorescence method. Levels above 0.01 ng/ml were considered positive. Based on cTnI levels, patients were divided into two groups: Group 1 with positive cTnI (>0.01 ng/ml) and Group 2 with negative cTnI (<0.01 ng/ml).
We recorded the length of hospital stay, including time spent in the ICU and duration of ventilator support if required. In-hospital mortality rates were also noted. Statistical analysis was performed using descriptive and inferential methods. Continuous measurements were presented as Mean ± SD (Min-Max), while categorical measurements were presented as numbers and percentages. Significance was assessed at the 5% level. Student's t-test and Chi-square/Fisher Exact test were used to analyze continuous and categorical variables, respectively.

RESULTS:
This prospective observational study included 90 patients admitted with acute exacerbation of COPD over 18 months. Patients were divided into two groups based on cardiac Troponin I (cTnI) levels measured on admission: Group 1 (cTnI>0.01 ng/ml, n=34) and Group 2 (cTnI<0.01 ng/ml, n=56). Demographic data, medical history, physical examination findings, and various diagnostic tests were recorded. Clinical outcomes including ICU admission, need for ventilation, length of stay, and mortality were assessed.
Table 1 presents the baseline characteristics of the study population. Patients with elevated cTnI were found to have significantly longer disease duration (p=0.002) and lower SpO2 levels (p=0.003) compared to those with normal cTnI. The prevalence of ischemic heart disease (IHD) was also significantly higher in the elevated cTnI group (p=0.018).

Table 1: Baseline Characteristics

Characteristic	cTnI +ve (n=34)	cTnI -ve (n=56)	P Value
Age (years)	64.15 ± 7.75	66.72 ± 8.62	0.286
Males	30 (88.2%)	53 (94.6%)	0.606
Disease Duration (years)	6.42 ± 3.34	3.97 ± 1.94	**0.002**
Co-morbidity	18 (52.9%)	15 (26.7%)	0.055
IHD	12 (35.2%)	4 (7.1%)	**0.018**
SpO2 (%)	75.00 ± 11.44	82.29 ± 4.86	**0.003**

Table 2 highlights the cardiovascular characteristics of both groups. Notably, patients with elevated cTnI had a higher prevalence of dilated right atrium/right ventricle (p=0.033) and left ventricular dysfunction (p=0.009) on echocardiography. The incidence of IHD was again significantly higher in this group (p=0.018).

Table 2: Cardiovascular Characteristics

Characteristic	cTnI +ve (n=34)	cTnI -ve (n=56)	P Value
AF	1 (5.9%)	0	-
IHD	12 (35.3%)	4 (7.14%)	**0.018**
Sinus Tachycardia (ECG)	25 (73.5%)	43 (76.8%)	0.764
P Pulmonale (ECG)	11 (32.4%)	25 (44.6%)	0.341
Dilated RA/RV (Echo)	22 (64.7%)	18 (32.1%)	**0.033**
LV Dysfunction (Echo)	10 (29.4%)	2 (3.6%)	**0.009**

Table 3 compares the arterial blood gas analysis between the two groups. Although patients with elevated cTnI showed slightly higher PaCO2 levels, the differences in both PaO2 and PaCO2 were not statistically significant.

Table 3: Blood Gas Analysis

Parameter	cTnI +ve (n=34)	cTnI -ve (n=56)	P Value
PaO2 (mmHg)	78.07 ± 46.05	76.77 ± 32.69	0.907
PaCO2 (mmHg)	54.76 ± 24.19	47.77 ± 23.29	0.315

Table 4 summarizes the clinical outcomes. Patients with elevated cTnI had significantly higher rates of ICU admission (p=0.002) and need for ventilatory support (p=0.006). However, there were no significant differences in the length of ICU or hospital stay, or in-hospital mortality between the two groups.

Table 4: Clinical Outcomes

Outcome	cTnI +ve (n=34)	cTnI -ve (n=56)	P Value
ICU Admission	32 (94.11%)	29 (51.78%)	**0.002**
NIV/Invasive Ventilation	27 (79.4%)	22 (39.28%)	**0.006**
ICU Stay (days)	5.56 ± 1.95	5.56 ± 5.49	0.996
Hospital Stay (days)	8.94 ± 4.46	8.61 ± 4.25	0.792
Mortality	5 (14.7%)	4 (7.14%)	0.285

DISCUSSION:

This study found that cardiac Troponin I (cTnI) was elevated in 37.77% of patients with acute exacerbation of COPD, suggesting the presence of cardiac injury in this population. This finding aligns with previous studies, although the incidence of cTnI elevation varies across different research, ranging from 16.6% to 74%.[11-16] Our study revealed that patients with elevated cTnI had a significantly longer duration of COPD, higher prevalence of ischemic heart disease (IHD), and lower SpO2 levels. Additionally, these patients showed a higher incidence of severe pulmonary hypertension, corpulmonale, and left ventricular dysfunction.

In agreement with previous studies[11,12,14-16], our research demonstrated a strong correlation between elevated cTnI and the need for ICU admission and ventilator support. However, unlike some earlier studies, we did not find significant differences in the duration of ventilator support, ICU stay, or overall hospital stay between the two groups. Moreover, elevated cTnI was not a strong predictor of in-hospital mortality in our study, which contrasts with findings from some previous research.[11,14]

The exact mechanism for cTnI elevation in acute exacerbation of COPD was beyond the scope of this study. However, potential reasons could include increased work of breathing, increased left ventricular afterload due to more negative intrathoracic pressure, worsening of pulmonary hypertension, hypoxemia, and hypercapnia. From a prognostic standpoint, identifying this subgroup of patients at particular risk is crucial. Early identification and appropriate treatment of the cause for cTnI elevation could potentially influence long-term outcomes. Further studies involving larger patient populations are recommended to evaluate whether long-term outcomes vary in patients with elevated cTnI during acute exacerbation of COPD.

Contribution: concepts, design, definition of intellectual concept, literature research, clinical studies, experimental studies, data acquisition, data analysis, statistical analysis. Manuscript preparation, manuscript editing and review

CONCLUSION:

cTnI is elevated in a significant subset of patients with acute exacerbation of COPD, associated with longer disease duration, higher incidence of IHD, and lower SpO2. Elevated cTnI independently predicts the need for ICU admission and ventilator support, although it does not significantly impact

the length of hospital stay or in-hospital mortality. These findings suggest that cTnI can be used as a marker to identify high-risk patients at admission, potentially guiding more intensive monitoring and management strategies. However, further large-scale studies are needed to evaluate the long-term outcomes in patients with elevated cTnI during acute exacerbation of COPD.

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