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FREQUENCY OF RAISED CARDIAC TROPONIN-I DURING ACUTE EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND ITS CO-RELATION WITH CLINICAL OUTCOMES

Original Research

Taha Nazir Warraich[1], Tahreem Raza[2], Talha Laique[1]*
[1]Department of Medicine, Mayo Hospital, Lahore-Pakistan.
2Department of Medicine, Hameed Latif Hospital, Lahore-Pakistan.
Corresponding Author: Talha Laique, Department of Medicine, Mayo Hospital, Lahore-Pakistan. talhalaique51@gmail.com
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ABSTRACT

Background: Cardiovascular disease is a major comorbidity in chronic obstructive pulmonary disease (COPD), contributing to increased morbidity and mortality. Acute exacerbations of COPD (AECOPD) impose additional cardiovascular stress, potentially leading to myocardial injury. Elevated cardiac troponin-I (cTnI) has been associated with poor clinical outcomes, but its prognostic significance in AECOPD remains underexplored, particularly in the Pakistani population. Identifying patients at higher risk of mortality and invasive ventilation can aid in optimizing clinical management and reducing complications.

Objective: To evaluate the prognostic significance of cardiac troponin-I (cTnI) levels in patients with acute exacerbation of COPD.

Methods: A descriptive case series was conducted at the Department of Medicine and TB & Chest Diseases, Mayo Hospital, Lahore. A total of 135 patients diagnosed with AECOPD were enrolled using a non-probability consecutive sampling technique. Detailed history and clinical examination were performed. Blood samples for cTnI were collected at admission and after 24 hours, analyzed via the immunofluorescence method, and considered positive at levels ≥0.017 µg/L. Clinical parameters, including arterial blood gases, electrocardiography, echocardiography, spirometry, and complete blood profiles, were assessed. Independent t-tests were applied to analyze differences in hospital stay duration and oxygen saturation, while the chi-square test evaluated associations between troponin levels, mortality, and need for invasive ventilation.

Results: Among 135 patients, 40.7% (n=55) had elevated cTnI. Invasive ventilation was required in 36.4% of troponin-positive patients compared to 20.0% in troponin-negative patients (p=0.035). Mortality was significantly higher in troponin-positive patients (29.1%) compared to troponin-negative patients (10.0%) (p=0.004). Oxygen saturation (SpO2) was significantly lower in troponin-positive patients (73.7 ± 11.1%) than in troponin-negative patients (81.0 ± 8.1%) (p<0.001).

Conclusion: Elevated cTnI levels are significantly associated with higher mortality and increased need for invasive ventilation in AECOPD patients. This biomarker can serve as an early predictor of adverse outcomes, aiding in clinical decision-making and risk stratification.

Keywords: Acute Disease, Cardiac Troponin-I, Chronic Obstructive Pulmonary Disease, Hospital Mortality, Oxygen Saturation, Prognosis, Ventilator Support.

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INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a progressive airway disorder characterized by persistent respiratory symptoms and airflow limitation due to airway and alveolar abnormalities. It manifests clinically with chronic cough, sputum production, and wheezing. The World Health Organization (WHO) predicts that by 2030, COPD will become the third leading cause of death worldwide (1,2). Despite its significant burden, COPD and asthma often remain underdiagnosed in low- and middle-income countries, contributing to increased morbidity and mortality. In South Asia, COPD accounts for approximately 8% of all disabilities, with an estimated prevalence of 2.1% in Pakistan, while asthma affects around 4.3% of the population (3). The pathogenesis of COPD is multifactorial, encompassing genetic predisposition, immune dysregulation, lung tissue damage, and recurrent infections (4). Environmental risk factors such as exposure to air pollution, occupational dust, biofuel smoke, and cigarette smoking further contribute to disease progression (5). Additionally, COPD frequently coexists with other systemic conditions, including metabolic syndrome, depression, diabetes, lung cancer, osteoporosis, and cardiovascular diseases (6). Among these, cardiovascular diseases represent a major comorbidity, significantly influencing patient outcomes. Both COPD and cardiovascular disease share common risk factors, including aging, male sex, and smoking, making cardiovascular complications particularly relevant in COPD management (7).

Acute exacerbations of COPD (AECOPD) are triggered by various factors, including bacterial and viral infections. However, in a subset of patients, the underlying cause remains unidentified, with acute left ventricular (LV) dysfunction being a possible contributing factor (8,9). The presence of chronic pulmonary hypertension and cor pulmonale further increases cardiac burden, raising the risk of cardiovascular events during exacerbations. Given the substantial overlap between COPD and cardiovascular disease, identifying patients at risk of adverse cardiac events is critical. Biomarkers such as cardiac troponin-I (cTnI), which are easily accessible through simple diagnostic tests, may serve as valuable tools in detecting myocardial injury and guiding clinical decision-making. Previous studies have suggested an elevated frequency of cTnI in AECOPD patients, with one estimate reporting a prevalence of 34% (10). Despite the known association between COPD exacerbations and cardiovascular complications, limited data exist on the role of cTnI in predicting prognosis among COPD patients in Pakistan. No prior studies have been conducted at Mayo Hospital or locally to assess this association. This study aimed to evaluate the frequency of elevated cTnI levels in patients experiencing AECOPD and its correlation with clinical outcomes. By identifying high-risk patients, this research seeks to enhance the understanding of cardiovascular involvement in COPD exacerbations, ultimately guiding clinical protocols to improve management strategies and reduce morbidity.

METHODS

The study was conducted in the Department of Medicine and TB & Chest Diseases at Mayo Hospital, Lahore. A total of 135 patients diagnosed with acute exacerbation of chronic obstructive pulmonary disease (AECOPD) were enrolled using a non-probability consecutive sampling technique. The inclusion criteria encompassed adult patients presenting with AECOPD confirmed through clinical assessment and spirometry. Patients with pre-existing cardiac conditions, recent myocardial infarction, chronic renal disease, or those receiving thrombolytic therapy were excluded to minimize confounding factors. Ethical approval was obtained from the institutional review board, and written informed consent was secured from all participants prior to enrolment (11). Upon admission, patients underwent a comprehensive clinical evaluation, including detailed history-taking and physical examination. Demographic details such as age, occupation, smoking status, residential address, and disease duration were recorded. Blood samples for cardiac troponin-I (cTnI) levels were collected at the time of admission and repeated after 24 hours. The samples were analyzed using the immunofluorescence method, and patients were classified as cTnI positive if the biomarker level was ≥0.017 µg/L. Additional investigations, including arterial blood gases (ABGs), electrocardiography (ECG), echocardiography, spirometry, and a complete blood profile, were conducted to assess the severity of the exacerbation and any associated complications (12).

Clinical outcomes such as duration of hospital stay, need for assisted ventilation, and in-hospital mortality were documented for each patient. Data were processed and analyzed using SPSS version 26.0. Continuous variables, including age, respiratory rate, hospital stay duration, and oxygen saturation (SpO2), were expressed as mean ± standard deviation (SD). Categorical variables, such as gender, invasive ventilation requirement, and mortality, were presented as frequencies and percentages. An independent sample t-test was

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employed to compare mean respiratory rate, duration of hospital stay, and SpO2 levels between cTnI-positive and cTnI-negative patients. The chi-square test was utilized to assess associations between troponin positivity, the need for invasive ventilation, and mortality outcomes. A p-value of ≤0.05 was considered statistically significant (13).

RESULTS

The study included 135 patients with a mean age of 58.5 ± 8.5 years. Among them, 105 (77.8%) were male, and 30 (22.2%) were female. Cardiac troponin-I (cTnI) was elevated in 55 (40.7%) patients, while 80 (59.3%) had normal levels. The clinical characteristics of the study population revealed significant respiratory distress. The mean respiratory rate was 30.8 ± 4.3 breaths per minute, ranging from 20.0 to 41.0 breaths per minute. The average duration of hospital stay was 5.5 ± 2.3 days, with a minimum stay of 2 days and a maximum of 12 days. Oxygen saturation (SpO2) levels were critically low, with a mean of 78.1 ± 10.0%, ranging from 50.0% to 95.0%.

Regarding patient outcomes, 36 (26.7%) required invasive ventilation, while 99 (73.3%) did not. Mortality was recorded in 24 (17.8%) patients, while 111 (82.2%) survived. The comparison between cTnI-positive and cTnI-negative groups demonstrated significant differences in clinical outcomes. Among the patients with elevated cTnI, 20 (36.4%) required invasive ventilation, compared to 16 (20.0%) in the cTnI-negative group (p=0.035). Mortality was notably higher in the cTnI-positive group, with 16 (29.1%) deaths, compared to 8 (10.0%) in the cTnI-negative group (p=0.004), indicating a significant association between elevated cTnI levels and worse clinical outcomes.

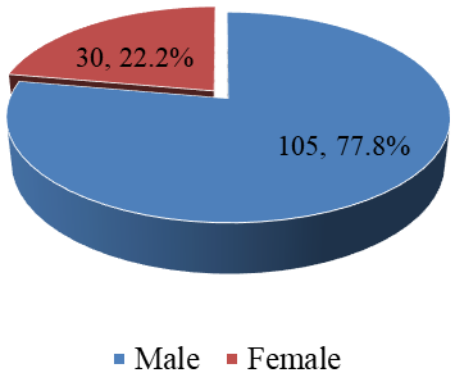

Figure-1: Gender distribution of patients

Table: Summary of Clinical Parameters of Patients

Clinical Parameters	Mean ± SD	Min.		Max.
Respiratory Rate	30.8 ± 4.3	20.0		41.0
Duration of Hospital Stay	5.5 ± 2.3	2.0		12.0
SPO_2	78.1 ± 10.0	50.0		95.0

Table-2: Outcomes of Patients Based on Invasive Ventilation and Mortality

Outcome	Categories	Frequency	Percentage
Invasive Ventilation	Yes	36	26.7
	No	99	73.3
Mortality	Yes	24	17.8
	No	111	82.2

Table: Comparison of Outcomes between Troponin I Positive and Negative Patients

Outcome	Troponin I Positive (n=55)	Troponin I Negative (n=80)	p-value #

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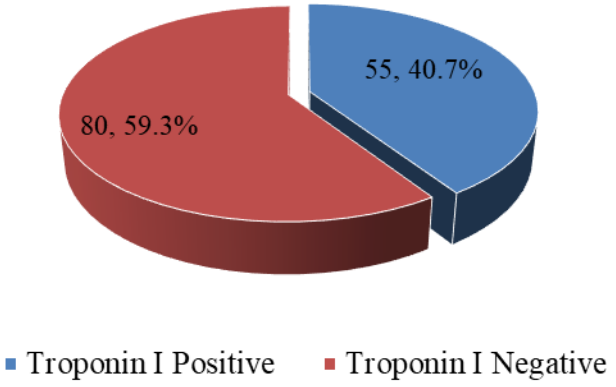
Invasive Ventilation	20 (36.4%)	16 (20.0%)	0.035*
Death	16 (29.1%)	8 (10.0%)	0.004*

*Significant



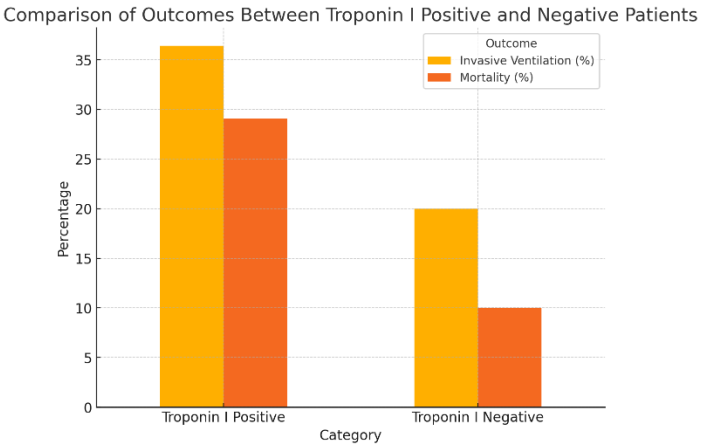


Figure-2: Prevalence of elevation of cTnI in patients

DISCUSSION

Chronic inflammation in chronic obstructive pulmonary disease (COPD) contributes to alveolar wall destruction and small airway obstruction, mediated by inflammatory enzymes such as metalloproteinases and serine proteases (10). Acute exacerbations of COPD (AECOPD) place significant stress on both the right and left ventricles, leading to an increased risk of cardiovascular complications. Cardiovascular disease is a major contributor to morbidity and mortality in COPD, with studies indicating that it accounts for 12–37% of COPD-related deaths (11). In the present study, the overall mortality rate was 17.8%, while 82.2% of patients survived, underscoring the substantial burden of cardiovascular involvement in COPD exacerbations (14). Cardiac troponin-I (cTnI) is a highly specific marker of myocardial injury, typically associated with acute coronary syndromes. However, elevated levels have also been observed in conditions such as sepsis, renal failure, stroke, and trauma (5). In this study, 40.7% of patients with AECOPD tested positive for cTnI, aligning with previous research reporting troponin elevation in 32–34% of COPD patients (11,12). These findings suggest that myocardial injury is not uncommon in AECOPD and may be a consequence of increased cardiac workload, hypoxia, or pulmonary hypertension (15-17).

One of the proposed mechanisms for troponin elevation in COPD is acute pulmonary arterial pressure elevation secondary to hypoxemic vasoconstriction (13). Hypoxia, hypercapnia, increased work of breathing, and left ventricular afterload due to negative intrathoracic pressure contribute to myocardial stress and subsequent troponin release (12,13). The present study demonstrated a mean oxygen saturation (SpO2) of 78.1±10.0%, with values as low as 50.0%, highlighting severe hypoxemia among patients. Previous research has indicated that elevated cTnI is associated with higher PaCO2 levels, decreased pulse oximetry readings, and hypercapnia, which are consistent with the findings of this study (9,14,18). Clinical outcomes were significantly worse in patients with positive cTnI, with a higher need for invasive ventilation and increased mortality. Among cTnI-positive patients, 36.4% required invasive ventilation compared to 20.0% in the cTnI-negative group, with a statistically significant p-value of 0.035. These findings are in line with prior research reporting a higher requirement for invasive or noninvasive ventilation in troponin-positive patients (p=0.016) (14). Similarly, mortality was markedly higher in the cTnI-positive group (29.1%) compared to the cTnI-negative group (10.0%), with a p-value of 0.004. This association has been corroborated by previous studies, where patients with elevated troponin levels experienced significantly higher mortality rates during AECOPD episodes (14,19-21).

The study presents valuable insights into the prognostic significance of cTnI in AECOPD; however, certain limitations should be considered. The single-center design and relatively small sample size limit the generalizability of findings. Financial constraints restricted the inclusion of additional biomarkers and genetic analyses that could have provided a more comprehensive understanding of the underlying mechanisms. Additionally, although patients with pre-existing cardiac disease were excluded, subclinical cardiovascular involvement could not be entirely ruled out. Future studies with larger, multicenter cohorts, detailed echocardiographic assessments, and longitudinal follow-ups are necessary to further delineate the clinical utility of cTnI in COPD management (22,23).

CONCLUSION

The findings of this study highlight the clinical significance of cardiac troponin-I as a biomarker for predicting adverse outcomes in patients experiencing acute exacerbations of chronic obstructive pulmonary disease. Elevated troponin levels were associated with greater disease severity, increased need for invasive ventilation, and higher mortality, indicating underlying cardiac dysfunction in these patients. Recognizing this biomarker as a prognostic tool can aid in early risk stratification, enabling timely interventions to improve patient management and outcomes.

Author Contribution

Author	Contribution
Taha Nazir Warraich	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Tahreem Raza	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Talha Laique*	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published

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