

Study of Highly Sensitive Cardiac Troponin T and its Clinical Significance in Emergency Patients with Acute Exacerbations of Chronic Obstructive Pulmonary Disease

Ashutosh Kumar¹, Chandan Kumar², Sumit Kumar³, Raj Kumar Deepak⁴, Kumbha Sai Praveen⁵

¹Senior Resident, Department of TB and Chest, Government Medical College and Hospital, Bettiah, West Champaran, Bihar, India

²Senior Resident, Department of General Medicine, Government Medical College and Hospital, Bettiah, West Champaran, Bihar, India

³Associate Professor, Department of General Medicine, Government Medical College and Hospital, Bettiah, West Champaran, Bihar, India

⁴Associate Professor and HOD, Department of General Medicine, Government Medical College and Hospital, Bettiah, West Champaran, Bihar, India

⁵DNB PG Trainee, Government Medical College and Hospital, Bettiah, West Champaran, Bihar, India

Received: 01-08-2026/Revised: 15-08-2026 / Accepted: 21-08-2026

Corresponding author: Dr. Chandan Kumar

Conflict of interest: Nil

Abstract

Background: One of the leading causes of morbidity, mortality, and medical expenses worldwide is chronic obstructive pulmonary disease. Whether or not they have a history of Cor pulmonale, they have a higher cardiac burden during acute exacerbations. Cardiovascular disease is more common in COPD patients, and exacerbations put more strain on the heart. The prognostic and predictive usefulness of very sensitive troponin T observed during COPD exacerbations has been explored. Aim of this study is to assessment the clinical significance of highly sensitive troponin T (Hs cTnT) as a predictive and prognostic factor in patients with acute exacerbations of COPD.

Methods: From March 2026 to July 2026, 79 patients with acute COPD exacerbations enrolled in this observational cross-sectional study. This study excluded patients who had myocardial infarction, cardiac arrest, significant renal impairment, or chronic hemodynamic instability prior to admission. Included Patient detail 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: The study included 79 patients who had an acute COPD exacerbation. When comparing the four reported categories (moderate, severe, very severe and life threatening) of COPD exacerbation severity, the mortality rate was 2.53%, and the Hs cTnT level revealed a statistically significant difference. When compared to moderate and severe forms, only the life-threatening variant had a significantly higher hscTnT level.

Conclusion: According to the study, patients with exacerbations of COPD had considerably higher levels of extremely sensitive cardiac Troponin T. Troponin T could help in the evaluation and prognosis of patients experiencing exacerbations of COPD.

Keywords: Chronic Obstructive Pulmonary Disease; Highly Sensitive Troponin T.

DOI: 10.25258/ijcpr.18.9.192

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Chronic obstructive pulmonary disease (COPD) is a lung illness defined mainly by airflow limitation that is not totally reversible and corresponds to the predominant cause of chronic respiratory failure and Cor pulmonale [1]. One of the leading causes of sickness, mortality, and medical expenses worldwide is COPD. Cigaret smoking is a major risk factor for this worldwide health problem

[2].Chronic obstructive pulmonary disease (COPD) is the third leading cause of death worldwide, causing 3.4 million deaths in 2023, approximately 6% of all global deaths.[3] In India the pooled prevalence of Chronic Obstructive Pulmonary Disease (COPD) among adults is 13%, according to a 2026 Systematic Review published in BMC Pulmonary Medicine. COPD is a long-term lung

disease that makes it hard to breathe. It remains a major and underrecognized health challenge in India.[4] Despite a steady progression of symptoms and decline of respiratory function, the majority of patients experience bouts of symptom aggravation. During exacerbations, patients typically require hospitalization, and fatality rates rise. A relapse might be defined as an exacerbation that occurs within 28 days after a previous exacerbation [5].

COPD exacerbation could put enough load on the heart to trigger myocardial cell necrosis. Edema, mucus hypersecretion, and bronchoconstriction may produce additional breathing impairment, whereas alveolar hypoxia may cause constriction of pulmonary arterioles and increased pulmonary artery pressure, disrupting perfusion.

Exacerbations of COPD typically result in tachycardia, hypoxemia, right ventricular dilatation, and pulmonary arterial hypertension [6]. Currie et al. have demonstrated that patients with or without a history of Cor pulmonale had a higher cardiac burden during acute attack exacerbations [7]. "According to ROC analysis of the results from an earlier test generation of the Elecsys Troponin T assay, the WHO criteria for the definition of acute myocardial infarction for Hs cTnT is 14 ng/L or 0.014 ng/mL" [8]. Patients with COPD are more likely to develop cardiovascular disease, which puts more stress on the heart during flare-ups [9].

The prognostic and predictive usefulness of circulating levels of very sensitive troponin T seen during COPD exacerbations has been explored.

The purpose of this study is to determine the level of highly sensitive cardiac troponin T (hs cTnT) in patients experiencing acute exacerbations of COPD and to assess the short-term prognostic value of highly sensitive cardiac troponin T (hscTnT) with respect to mechanical ventilation, admission to the medical ward, and admission to the intensive care unit (ICU).

Material and Methods

This observational cross-sectional study was conducted in 79 patients with acute exacerbation of COPD admitted to the medicine department at Government Medical College and Hospital, Bettiah, West Champaran, Bihar from March 2026 to July 2026. Both genders, adult patients (more than 40 years old) with acute exacerbation of COPD were included in this study. Patients with severe renal impairment, persistent hemodynamic instability, myocardial infarction and cardiac arrest before admission were excluded in this study. The clinical data were collected 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. Complete blood count, arterial blood gases, renal functions, Serum cardiac enzymes Ck, CK-MB and highly sensitive cardiac troponin T (hs cTnT) were measured. Patients followed up for the effectiveness of the emergency management and outcome.

Results

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

Gender	No. of cases	Percentage
Male	54	68.4%
Female	25	31.6%
Total	79	100%

Table 2: Age among the studied patients (n=79)

Age in years	No. of cases	Percentage
47- <60	32	40.51%
60- <70	24	30.38%
≥70 – 86	23	29.11%
Mean±SD	62.67±9.55	
Range	47– 86	

Mean age of the studied patients is 62.67 years.

Table 3: Risk factor for COPD among the studied patients (n=79)

		No. of cases (79)	Percentage
Risk factor for COPD	Cigarette smoking	63	79.70%
	Shisha (Hukka) 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 3 shows that the most common risk factor for COPD was cigarette smoking whether alone (40.5%) or with shisha (Hukka) smoking (39.2%).

Table 4: Clinical characteristics among the studied patients (n=79)

Cause of exacerbation	No. of cases	Percentage
Infection	72	91.14%
Idiopathic	7	8.86%
Total	79	100.00%

Table 4 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.

Table 5: ECG changes among the studied patients (n=79)

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

Table 5 shows that Sinus tachycardia was the most ECG changes seen among the studied patients (72.15%).

Table 6: ABG on presentation to ER and after initial treatment among studied patients (n=79)

ABG	On presentation to ER	After initial treatment	p-value
	Mean $\pm$ SD	Mean $\pm$ SD	
pH	7.29 $\pm$ 0.11	7.32 $\pm$ 0.08	0.1(NS)
PO ₂	56.1 $\pm$ 14.1	66.72 $\pm$ 12.6	0.001*
PCO ₂	56.4 $\pm$ 15.8	53.6 $\pm$ 13.28	0.2(NS)
HCO ₃	25.8 $\pm$ 6.27	25.41 $\pm$ 5.69	0.6(NS)

*Statistically significant difference (p-value <0.05), NS: no statistically significant difference (p-value >0.05)

Table 6 shows that there was no statistically significant difference regarding ABG findings before and after initial ER treatment except for PO₂.

Table 7: High sensitive cardiac troponin (hs cTnT) among studied patients (n=79)

Cardiac troponin ng/L	
Range	0.007– 0.069
Mean $\pm$ SD	0.0385 $\pm$ 0.0199
Median	0.037

Table 7 shows that mean cardiac troponin level was 0.0385 ng/L with wide range from 0.007 to 0.069 ng/L.

Table 8: 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 $\pm$ SD	0.039 $\pm$ 0.017	0.063 $\pm$ 0.006	0.001*

Table 8 comparison between patients admitted to the general ward and patients admitted to ICU regarding cardiac troponin levels (n=79).

Discussion

The current investigation found that the majority of the patients were males (68.4%), with an average age of 62.67 years (Table 1). This is also similar with another German study by Rabe et al. [9], in which the majority of the patients (71.7%) were men with an average age of 63 years. Cigarette smoking was the most prevalent risk factor in our study for COPD. Only 7.6% of patients were never smokers (occupational exposure), which is consistent with the findings of Roche et al. [10], who found that 6.6% of patients were never

smokers. Cardiomegaly was found to be the most common comorbidity among the investigated patients (27.85%), with chronic liver disease accounting for 7.59%, compared to Chang et al.'s [11] study, which found cardiomegaly to be (30.1%), which matches this study's findings, and chronic liver disease accounting for 0.8%. In 79 included patients comparing the four reported categories (moderate, severe, very severe and life threatening) of COPD exacerbation severity only life threatening from showed significantly higher hs cTnT level compared to moderate and severe forms. More than half of the patients (65%) in our study had arterial O₂ tension less than 60 mmHg and arterial PCO₂ more than 60 mmHg when they arrived at the emergency department. This is

similar to another study conducted by Soyseth et al. [12], in which thirty of the patients in the references had arterial O₂ tension less than 60 mmHg and 50% of the AECOPD patients had arterial PCO₂ greater than 60 mmHg upon admission at the hospital. We observed that AECOPD is related with a considerable rise in troponin T as assessed by a very sensitive assay, which is consistent with the findings of Soyseth et al. [12] Our study found a mean cardiac troponin level of 0.0385 ng/l.

In our investigation, the optimum cutoff point for hscTnT to predict ICU admission was greater than 0.053 ng/l. Our findings are consistent with those of Pal H Brekke et al. [13], who found that hscTnT was raised at values of 0.04 ng/l or higher. At the time, hospital protocol suggested that ahscTnT concentration of 14ng/l or greater be used to diagnose myocardial infarction (MI). Acidosis and hypoxemia were closely linked to cTnT increase. Indeed, in our research of two patients, 2.53% died in the ED, which is similar to Roche et al. [14] study, in which 1.25% died in the ED. We did record the accepted ECG criteria for ischemia, however the ST-T alterations could be generic, leading to false positive ischemia, which was ruled out by measuring CK and CK MB. The current investigation found that AECOPD patients without a history of coronary heart disease have high circulating levels of troponin T, and that troponin T levels are also enhanced in relation to disease severity.

Conclusion

COPD patients with exacerbation had significantly higher levels of the highly sensitive cardiac troponin T. Troponin T will assist physicians in assessing prognosis in COPD exacerbations, but additional research is needed to see whether it will improve therapeutic strategies.

References

1. Global plan for the diagnosis, treatment, and prevention of chronic obstructive pulmonary disease: GOLD executive summary, Rabe KF, Hurd S, Anzueto A, Barnes PJ, Buist SA, et al. (2007). *Am J Respir Crit Care Med* 176: 532-555.
2. Global burden of COPD: risk factors, prevalence, and future projections, Mannino DM, Buist AS (2007). 370: 765-773, *Lancet*.
3. Global Burden of Disease Collaborative Network. Global Burden of Disease 2023: Findings from the GBD 2023 Study. Seattle, United States: Institute for Health Metrics and Evaluation (IHME), 2025.
4. Pritam Halder et al : *BMC Pul. Med.* 2026 Feb 7;26:112.
5. Detectable blood cardiac troponin T as a predictor of poor prognosis in patients with persistent precapillary pulmonary hypertension (Torbicki A, Kurzyna M, Kuca P, 2003). *Circulation* 108: 844-848.
6. The ABC of chronic obstructive pulmonary disease, Currie GP, Wedzicha JA (2006). severe flare-ups. *BMJ* 333: 87-89.
7. Improved troponin T ELISA specific for cardiac troponin T isoform: Assay development and analytical and clinical validation (Müller-Bardorff M, Hallermayer, K, Schröder A, 1997). *Clin Chem* 43: 458-466.
8. Troponin T and CK-MB mass clinical assessment using the Elecsys 2010 analyzer by Kampmann M, Rauscher T, Müller-Bardorff M, Köch M, Klein G, et al. (1997). *Clinical Chemistry Congress of Europe*.
9. 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.
10. A clinical in-hospital prognostic score for acute COPD exacerbations (Roche N, Chavaillon JM, Maurer C, Zureik M, Piquet J, 2014). *Respir Res* 15: 99.
11. Chang CL, Robinson SC, Mills GD, Sullivan GD, Karalus NC, et al. (2011) Biochemical markers of cardiac dysfunction predict mortality in acute exacerbations of COPD. *Thorax* 66: 764.
12. 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.
13. Brekke PH, Omrand T, Holmedahl SH, Smith P, Søyseth V (2009) Determinants of cardiac troponin T elevation in COPD exacerbation- A cross-sectional study. *BMC Pulm Med* 9: 35.
14. 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.