SSR Institute of International Journal of Life Sciences
ISSN (O): 2581-8740 | ISSN (P): 2581-8732
Hatila *et al*., 2025
doi: 10.21276/SSR-IIJLS.2025.11.2.3

Original Article

open access

Association of Cardiac Troponin-T Level with Pulmonary Function and Cardiovascular Morbidity and Mortality

Deepika Hatila[1], Kailash Chander Khatri[2], Manish Kumar Bairwa[3]*

[1]Medical Officer, Dep of Anaesthesia, GMC Medical College Barmer and attached District Hospital Barmer, India
[2]Assistant Professor, Dep of Skin and VD, GMC Medical College Barmer, India
[3]Assistant Professor, Dep of Respiratory Medicine, GMC Medical College, Barmer, India

***Address for Correspondence:** Dr. Manish Kumar Bairwa, Assistant Professor, Dep of Respiratory Medicine, GMC Medical College, Barmer, India
E-mail: sehara11manish@gmail.com

Received: 14 Oct 2024/ Revised: 23 Dec 2024/ Accepted: 11 Feb 2025

ABSTRACT

Background: A vital biomarker for identifying myocardial damage, cardiac troponin T (cTnT) has long been used to diagnose acute myocardial infarction. However, because of developments in high-sensitivity assays (hs-cTnT), it is now possible to identify minute elevations in troponin levels in people who do not exhibit any symptoms of cardiovascular diseases. We aim to ascertain the predictive significance of hs-cTnT in evaluating the progression of cardiovascular and respiratory diseases by examining pulmonary function tests (PFTs), inflammatory markers, and echocardiography.

Methods: This prospective study of 200 patients with a history of smoking and symptoms of dyspnea aimed to investigate the link between cardiovascular risk, reduced lung function, and elevated hs-cTnT levels. Participants underwent a series of tests, including routine blood chemistry, EKG, pulmonary function tests, echocardiogram, chest X-ray, 6MWT, and HRCT. Statistical analysis was performed using ANOVA to assess significant differences among study groups.

Results: Increased hs-cTnT levels were substantially linked to systemic inflammation (hs-CRP, r=0.520, p<0.001), age (r=0.545, p<0.001), BNP (r=0.342, p=0.005), and RVSP (r=0.321, p=0.009). Strong associations between hs-cTnT and pulmonary function measurements, such as walking distance in 6MWT (r=-0.492, p<0.001), %DLco/VA (r=-0.438, p<0.001), and FEV_1% predicted (r=0.382, p=0.001), indicate that lung dysfunction leads to elevated myocardial stress. The association between structural lung damage and elevated troponin was further supported by HRCT results, which revealed greater LAA% in both the lower and upper lung fields.

Conclusion: This study concluded that elevated hs-cTnT levels are significantly correlated with factors such as age, smoking habits, pulmonary function, and biomarkers of systemic inflammation like hs-CRP.

Key-words: hs-cTnT, cardiovascular risk, pulmonary function, systemic inflammation, Echocardiography

INTRODUCTION

Cardiovascular disease is the major cause of death in patients who also suffer from pulmonary disease. It suggests that heart disease should be differentiated or evaluated with the co-existence of pulmonary disease [1,2]. Cardiac troponin T (cTnT) is a protein component of the troponin complex, which plays an important role in regulating cardiac muscle contraction. Troponin T is mainly located on the thin filaments of muscle fibers and consists of three subunits: troponin T, troponin I, and troponin C. Troponin T binds to tropomyosin that anchors the troponin complex to the actin filament; thus, it helps in facilitating proper alignment of tropomyosin on actin. This alignment helps regulate muscle contraction in response to calcium ion concentrations. It is released into the bloodstream when any injury occurs in the myocardial region. Therefore, Troponin T is an important biomarker for cardiac muscle damage. Increased cTnT levels have been used for the diagnosis of acute myocardial infarction. But in recent years, advancements in high-sensitivity assays have helped us

How to cite this article
Hatila D, Khatri KC, Bairwa MK. Association of Cardiac Troponin-T Level with Pulmonary Function and Cardiovascular Morbidity and Mortality. SSR Inst Int J Life Sci., 2025; 11(2): 7016-7022.

Access this article online
https://iijls.com/

doi: 10.21276/SSR-IIJLS.2025.11.2.3

with the detection of minimal increases in cTnT, even among individuals who do not have severe symptoms of pulmonary & cardiac damage. Thus, it becomes a key biomarker for the detection [3].

Pulmonary function, which is mainly done by spirometric measurements like Forced Vital Capacity (FVC) and Forced Expiratory Volume in one second (FEV_1), helps as an indicator of respiratory health. People who have normal breathing patterns, specifically who have a normal ratio of how much air they can forcefully exhale in one second (FEV_1) to their total exhaled air (FVC) but overall have a reduced total exhaled air (FVC), possess a higher risk of heart diseases and increased chances of death. Currently, there is certain evidence that suggests a significant association between increased cTnT levels and abnormal pulmonary function. As an example, in a study, individuals who showed a restrictive spirometric pattern were found to have higher occurrences of elevated cTnT levels. This increase serves as a marker for the mortality in the population. Thus, it draws a strong link between subclinical myocardial injury and impaired lung function [4].

Increased levels of cTnT are an important marker for adverse cardiovascular outcomes, which include increased risks of mortality and major cardiovascular events. Majorly in community populations, individuals, who have higher levels of cTnT than baseline concentrations, showed an increased risk for these outcomes. Thus, solidifying cTnT as an important biomarker for cardiovascular morbidity and mortality [5,6].

Patients who have chronic obstructive pulmonary disease (COPD) are observed along with increased level of cardiac troponin T (hs-cTnT) levels. However, patients who don't have any cardiovascular problems tend to show higher levels of it. Thus, this increased level of Troponin T shows increased mortality and makes its position as one of the crucial biomarkers for the detection in patients [7].

The relationship between cTnT levels, pulmonary function, and cardiovascular diseases is complex, but it can be used as a potential biomarker for the diagnosis of respiratory, and myocardial diseases [7]. Increased levels of cTnT are an indication of underlying myocardial stress or injury, which might be worsened by impaired pulmonary function. On the other hand, impaired lung function may be responsible for the increased level of cardiac workload and thus can cause myocardial damage that might lead to elevated cTnT levels. Identifying this relationship between myocardial damage & pulmonary dysfunction is essential for developing effective strategies to identify individuals who have a higher risk for adverse cardiovascular events and thus it requires timely interventions [7,8].

Our study aims to provide the interlink between cardiac troponin T levels, pulmonary function, and cardiovascular morbidity and mortality. By finding several kinds of literature as well as clinical studies, we seek to provide the potential mechanisms linking these factors. And thus discuss the implications for clinical practice and future research.

MATERIALS AND METHODS

Research Design- This prospective study included 200 patients over 1 year, all of whom had a lifetime history of smoking and exhibited symptoms of dyspnea during physical activity, sputum production, and persistent coughing. The primary objective was to investigate the relationship between cardiovascular risk, reduced lung function, and elevated hs-cTnT levels. Each participant underwent a series of measurements, including routine blood chemistry, EKG, blood gas analysis, pulmonary function tests, echocardiogram, chest X-ray, 6-minute walking test (6MWT), and high-resolution computed tomography (HRCT) of the chest. Serum samples were collected for hs-cTnT measurement. The study protocol was approved by the ethics committee, and written informed consent was obtained from all participants before enrollment. Statistical analysis was conducted using ANOVA and other standard methods to assess significant differences among the study groups.

Inclusion Criteria

- ✓ Long time smoking history.
- ✓ Present symptoms of dyspnea during physical activity, sputum production, and persistent coughing.
- ✓ Diagnosis of COPD is based on the criteria outlined by the Global Initiative for Chronic Obstructive Lung Disease (GOLD), with a post-bronchodilator FEV1/FVC ratio of 0.70.

Exclusion Criteria

- ✓ Participants with any cardiovascular conditions, including ischemic heart disease,

doi: 10.21276/SSR-IIJLS.2025.11.2.3

- ✓ Hypertension, arrhythmias, and congestive heart failure.
- ✓ Individuals are diagnosed with respiratory conditions such as interstitial pneumonia, bronchiectasis, and bronchial asthma.
- ✓ Patients with a history of myocardial infarction or other serious cardiac illnesses that could interfere with the study outcomes.

Measurements and Procedure- The procedure of the study involved a comprehensive assessment of cardiovascular and pulmonary health in each participant, including routine blood chemistry, electrocardiogram (EKG), blood gas analysis, pulmonary function tests, echocardiogram, chest roentgenography from both sides, 6-minute walking test (6MWT), and HRCT of the chest. The ECG was performed to detect signs of cardiac illness, such as arrhythmia or myocardial infarction history, and serum samples were collected for high-sensitivity cardiac troponin T (hs-cTnT) measurement, which was stored at -80°C. Pulmonary function tests, following American Thoracic Society guidelines, were carried out by skilled personnel to evaluate parameters like forced expiratory volume in 1 second (FEV1) and forced vital capacity (FVC), categorizing participants with COPD if their post-bronchodilator FEV1/FVC ratio was ≤0.70; others were classified as "at-risk." HRCT scans were performed to assess low attenuation areas (LAA%) in the lung fields, with calculations for the top, middle, and lower zones as well as the overall mean values. Echocardiography was conducted using a diagnostic ultrasound machine to evaluate cardiac function, and serum hs-cTnT levels were measured via electrochemiluminescence immunoassay, with standard values below 0.014 ng/mL and a detection limit of 0.003 ng/mL.

Statistical analysis- Statistical analysis was conducted using SPSS 27. Descriptive statistics were computed for all variables. Pearson's correlation examined relationships between hs-cTnT and clinical variables, identifying smoking, lung function (FEV1% predicted), age, and BNP as strongly correlated ($r=0.289–0.545$, $p<0.05$). Stepwise multiple regression found hs-CRP, age, and RVSP as significant predictors of hs-cTnT, with hs-CRP showing the strongest association ($\beta=0.462$, $p=0.002$). A significance level of $p<0.05$ was applied.

Ethical approval- The study was explained in detail to the patients by the authors. Permission from the patients has been obtained. The study's methodology has been approved by the ethical committee of the involved hospital.

RESULTS

A total of 200 patients with an average age of 66.2 years and a male preponderance (180 men, 20 girls) are shown in Table 1. The cohort's average number of pack years is 71.2, which indicates a substantial smoking history. The results of pulmonary function tests indicate lung diffusion capacity with a %DLco/VA of 70.1% and an FEV1 of 2.6L, FVC of 3.9L, and an FEV1% of 60.9%, with a projected FEV1% of 75.2%. Most patients (147 out of 200) had a diagnosis of COPD. Hemoglobin (14.6 g/dL), creatinine (0.86 mg/dL), P_2O_2 (83.4 Torr), and WBC count (6.7×10^9/L) with neutrophils at 4.1×10^9/L are other laboratory data.

Table 1: Characteristics of Patients at Baseline

	Patients (n=200)
Age (years)	66.2±11.6
Gender (male/female)	180/20
Smoking (pack/year)	71.2±44.1
FEV1 (L)	2.6±1.2
FVC (L)	3.9±1.1
FEV 1 % (%)	60.9±17.7
FEV 1 % predicted (%)	75.2±26.5
BMI	21.8±3.1
% DLco/VA (%)	70.1±22.4
Diagnosis (COPD/at risk)	147/53
P_2O_2 (Torr)	83.4±13.7
Creatinine (mg/dL)	0.86±1.19
Hemoglobin (g/dL)	14.6±1.8
WBC (10^9/L)	6.7±2.6
Neutrophils (10^9/L)	4.1±2.1
hs-CRP (mg/dL)	0.187±0.348
hs-cTnT (ng/mL)	0.009±0.007
BNP (pg/mL)	0.79±0.18

The HRCT, six-minute walk test, and echocardiography findings for 200 patients are shown in Table 2. Lung involvement is shown by HRCT results, which show lower lung field LAA% at 28.7% and upper lung field LAA% at 30.2%. An ejection fraction (EF) of 68.7%, a stroke volume (SV) of 70.1 mL, a LAD of 31.8 mm, and an IVS thickness of 8.9 mm are echocardiographic data that indicate intact cardiac function. With an RVSP of 33.9 mmHg and a cardiac output (CO) of 5.6 L/min, this could be a sign of pulmonary hypertension. With an oxygen saturation drop (ΔSpO_2) of 5.6% and an average walking distance of 520.8 meters, the six-minute walk test results demonstrate the patient cohort's capacity for exercise and oxygenation efficiency.

Table 2: HRCT, 6-minute walk test, Echocardiography results

	Patients (n=200)
Chest HRCT	
LAA% for lower lung field (%)	28.7±15.6
LAA% for upper lung field (%)	30.2±17.6
Echocardiography	
IVS Td (mm)	8.9±1.4
LAD (mm)	31.8±5.7
SV (mL)	70.1±17.2
HR (beats/minute)	75.2±12.3
PW Td (mm)	9.2±1.2
LVEDV (mL)	101.7±24.9
EF (%)	68.7±5.9
IVC Ex (mm)	13.4±2.9
CO (L/minute)	5.6±1.9
RVSP (mmHg)	33.9±6.7
Six-minute walk test	
$\triangle SpO_2$ (%)	5.6±5.4
Distance of walking (m)	520.8±97.1

Relationships between hs-cTnT and clinical variables are shown in Table 3, where factors such as BMI and LVEDV show weak or no significant correlation with hs-cTnT while smoking (r=0.289, p=0.047), lung function (FEV1% predicted, r=0.382, p=0.001), age (r=0.545, p<0.001), and BNP (r=0.342, p=0.005) are among the strongest correlations. Key clinical and functional connections within the patient sample are highlighted by these findings.

Table 3: Relationship between each subject's documented hs-cTnT levels and their cardiovascular and demographic characteristics

	Patients (n=200)	
	r	**p-value**
Age	0.545	0.000
BMI	0.081	0.523
Smoking habit	0.289	0.047
FVC	-0.391	0.000
FEV1 % predicted (logged)	0.382	0.001
% DLco/VA (logged)	-0.438	0.000
P2O2	-0.392	0.003
BNP	0.342	0.005
WBC	0.219	0.067
IVS Td	0.176	0.142
hs-CRP (logged)	0.520	0.000
LAD	0.115	0.337
EF	0.062	0.619
PW Td	0.178	0.137
LVEDV	-0.024	0.869
RVSP	0.321	0.009
CO	0.209	0.085
$\triangle SpO_2$	0.212	0.075
Walking distance (logged)	-0.492	0.000
LAA% for lower lung field	0.287	0.013
LAA% for upper lung field	0.236	0.047

doi: 10.21276/SSR-IIJLS.2025.11.2.3

Using a stepwise estimation technique, the multiple regression analysis of hs-cTnT levels is shown in Table 4, revealing important factors. The baseline relationship is established by the constant term, which is -2.803 ($p<0.001$). The highest positive correlation is seen with Hs-CRP ($\beta=0.462$, $p=0.002$), suggesting that elevated systemic inflammation is a major factor contributing to elevated hs-cTnT levels. Additionally, age plays a significant role ($\beta=0.358$, $p=0.008$), indicating that cardiac troponin levels increase with age. Furthermore, a significant predictor that connects high hs-cTnT to pulmonary pressures and possible cardiovascular strain is RVSP ($\beta=0.284$, $p=0.037$).

Table 4: Analysis of hs-cTnT levels using multiple regression (stepwise estimation model, logged)

Explanatory variables	B	95% CI		β	t-value	p-value
(constant)	-1.803	-2.287	-1.325		-13.019	0.000
hs-CRP (logged)	0.167	0.072	0.259	0.462	3.582	0.002
Age	0.009	0.003	0.017	0.358	2.792	0.008
RVSP	0.009	0.002	0.021	0.284	2.212	0.037

DISCUSSION

The relationship between cardiac troponin T levels and pulmonary function has drawn significant attention recently. Increased cTnT levels are normally detected by high-sensitivity assays which have been associated with various degrees of pulmonary impairment. Most individuals, who have a restrictive spirometric pattern show a higher prevalence of elevated cTnT levels which are not similar to the traditional cardiovascular risk factors. This proves that subclinical myocardial injury may be prevalent among the population with compromised lung function.

Patients who are diagnosed with COPD have increased levels of hs-cTnT, which have also been observed even in the absence of overt cardiovascular disease. This increase is associated with increased mortality, showcasing that hs-cTnT could serve as a valuable prognostic marker in these patients [3,8].

The prognostic value of elevated cTnT levels extends beyond pulmonary function impairment. In community populations, higher levels of cTnT than baseline concentrations have been linked with an increased risk of all mortality and major cardiovascular events. This association strengthens the cTnT's role as a biomarker for identifying individuals who are at elevated risk for adverse cardiovascular effects, although they absence symptomatic heart disease [8].

The mechanisms behind the association between increased cTnT levels and abnormal pulmonary function are not fully understood. One of the hypotheses might be that reduced lung function leads to chronic hypoxemia, which in turn increases cardiac workload and thus causes myocardial stress or injury, which is also reflected by elevated cTnT levels. On the other hand, elevated cTnT may be an indication of underlying cardiac pathology which adversely affects pulmonary function. Further studies are required to clarify these pathways and determine the relationships of these associations [9].

One study from Brekke *et al.* revealed a correlation between cardiac troponin T level & cardiopulmonary function. They discovered that high levels of cTnT in stable COPD patients & high-risk individuals are associated with systemic inflammation, age & RVSP, which suggests a link between lung inflammation & cardiovascular dysfunction. They also observed a correlation between cTnT & pulmonary oxygen levels, indicating that chronic hypoxemia might contribute to cardiomyopathy [10].

Another study also suggests a strong association between High-sensitivity cardiac troponin levels & cardiovascular morbidity in COPD patients. They revealed that increased levels of cTnI were specifically linked to Cardiovascular deaths, not COPD exacerbations, although it was correlated with pulmonary function. Thus, it suggests systemic inflammation, hypoxia & right

doi: 10.21276/SSR-IIJLS.2025.11.2.3

ventricular strain due to pulmonary hypertension might be the causes. In their study, they also observed a significant increase in troponin levels over 3 months, which further elevates cardiovascular risk, indicating atherosclerotic conditions [11-13].

Neukamm *et al.* also provided a similar conclusion; they also found that higher levels of high-sensitivity cardiac troponin T (hs-cTnT) in the blood were linked with a higher risk of death in people with stable COPD. Although these patients don't possess any indication of cardiovascular disease. Troponin is one of the significant indicators for the diagnosis of Heart disease, but it is often disregarded as a potential biomarker for pulmonary disease [14].

The study by Vélez-Martínez *et al.* found that out of 255 patients with pulmonary hypertension, 241 patients were found at higher levels (>1.2pg/ml) of cTnT levels. Thus it also solidifies itself as a potential biomarker for the detection of pulmonary disease [15].

The integration of cTnT measurement into clinical investigation for routine assessment of patients with impaired pulmonary function could enhance risk minimization and can help to form disease management strategies. As an example, identifying elevated cTnT levels in patients with abnormal spirometric patterns or diagnosis of COPD may require closer monitoring and might require intervention to decrease cardiovascular risk. However, implementing such strategies can't formed easily due to the guidelines and consideration of potential factors, which can also increase troponin T levels, such as renal function and systemic inflammation [12,13].

CONCLUSIONS

This study concluded that elevated hs-cTnT levels are significantly correlated with factors such as age, smoking habits, pulmonary function, and biomarkers of systemic inflammation like hs-CRP. The multiple regression analysis further highlighted the contribution of hs-CRP, age, and RVSP as strong predictors of elevated hs-cTnT levels, which may indicate underlying cardiovascular strain. These results emphasize the importance of monitoring hs-cTnT levels in patients with pulmonary conditions, particularly those at risk of cardiovascular complications. The study also shows the need for further research to explore the mechanisms linking pulmonary disease and cardiovascular risk, which could lead to more effective management strategies for this patient population.

CONTRIBUTION OF AUTHORS

Research Concept- Manish Kumar Bairwa, Kailash Chander Khatri, Deepika Hatila
Research Design- Manish Kumar Bairwa, Kailash Chander Khatri,
Supervision- Manish Kumar Bairwa
Materials- Manish Kumar Bairwa
Data Collection- Manish Kumar Bairwa
Data interpretation- Kailash Chander Khatri, Deepika Hatila
Literature- Kailash Chander Khatri, Deepika hatila
Writing Article- Kailash Chander Khatri, Deepika Hatila
Critical value- Manish Kumar Bairwa
Final approval - Manish Kumar Bairwa

REFERENCES

[1] Patel AR, Hurst JR. Extrapulmonary comorbidities in chronic obstructive pulmonary disease: state of the art. Expert Rev Respir Med., 2011; 5(5): 647-62.

[2] Decramer M, Janssens W. Chronic obstructive pulmonary disease and comorbidities. Lancet Respir Med., 2013; 1(1): 73-83.

[3] Dinh W, Füth R, Lankisch M, Bansemir L, Nickl W, et al. Cardiovascular function and predictors of exercise capacity in patients with chronic obstructive pulmonary disease. Respir Med., 2011; 105(1): 55-62.

[4] Xiao W, Cao R, Liu Y, Wang F, Bai Y, Wu H, et al. Association of high-sensitivity cardiac troponin T with mortality and cardiovascular events in a community-based prospective study in Beijing. BMJ Open, 2017; 7(6): e013431. doi: 10.1136/bmjopen-2016-013431.

[5] Høiseth AD, Neukamm A, Karlsson BD, Omland T, Brekke PH, et al. Elevated high-sensitivity cardiac troponin T is associated with increased mortality after acute exacerbation of chronic obstructive pulmonary disease. Thorax, 2011; 66(9): 775-81.

[6] Høiseth AD, Omland T, Hagve TA, Brekke PH, Søyseth V. Determinants of high-sensitivity cardiac troponin T during acute exacerbation of chronic obstructive pulmonary disease: a prospective cohort study. BMC Pulm Med., 2012; 12: 1-7.

doi: 10.21276/SSR-IIJLS.2025.11.2.3

[7] Soyseth V, Bhatnagar R, Holmedahl NH, Neukamm A, Hoiseth AD, et al. Acute exacerbation of COPD is associated with fourfold elevation of cardiac troponin T. Heart, 2013; 99(2): 122-6.

[8] Johansson S, Sandin P, Lindgren L, Mills NL, Hedman L, et al. Cardiac troponin and increased mortality risk among individuals with restrictive spirometric pattern on lung function testing. Eur Clin Respir J., 2025; 12(1): 2436203.

[9] Neukamm A, Einvik G, Hoiseth AD, et al. The prognostic value of measurement of high-sensitive cardiac troponin T for mortality in a cohort of stable chronic obstructive pulmonary disease patients. BMC Pulm Med., 2016; 16: 164. doi: 10.1186/s12890-016-0319-9.

[10] Brekke PH, Omland T, Holmedal SH, Smith P, Soyseth V. Troponin T elevation and long-term mortality after chronic obstructive pulmonary disease exacerbation. Eur Respir J., 2008; 31(3): 563-70.

[11] McEvoy JW, Chen Y, Ndumele CE, Solomon SD, Nambi V, Ballantyne CM, et al. Six-year change in high-sensitivity cardiac troponin T and risk of subsequent coronary heart disease, heart failure, and death. JAMA Cardiol., 2016; 1(5): 519-28.

[12] Hattori K, Ishii T, Motegi T, Kusunoki Y, Gemma A, Kida K. Relationship between serum cardiac troponin T level and cardiopulmonary function in stable chronic obstructive pulmonary disease. Int J Chron Obstruct Pulmon Dis., 2015; 309-20. doi: 10.2147/COPD.S76293.

[13] Adamson PD, Anderson JA, Brook RD, Calverley PM, Celli BR, Cowans NJ, et al. Cardiac troponin I and cardiovascular risk in patients with chronic obstructive pulmonary disease. J Am Coll Cardiol., 2018; 72(10): 1126-37. doi: 10.1016/j.jacc.2018.06.051.

[14] Neukamm A, Einvik G, Høiseth AD, Søyseth V, Holmedahl NH, et al. The prognostic value of measurement of high-sensitive cardiac troponin T for mortality in a cohort of stable chronic obstructive pulmonary disease patients. BMC Pulm Med., 2016; 16: 1-8.

[15] Vélez-Martínez M, Ayers C, Mishkin JD, Bartolome SB, García CK, et al. Association of cardiac troponin I with disease severity and outcomes in patients with pulmonary hypertension. Am J Cardiol., 2013; 111(12): 1812-17.