RESEARCH PAPER

Assessment Of Troponin I Level And Its Association With Severity Of Chronic Obstructive Pulmonary Disease

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ABSTRACT

Introduction: Chronic Obstructive Pulmonary Disease (COPD) is a progressive respiratory disorder associated with significant morbidity and cardiovascular complications. Troponin I, a sensitive biomarker of myocardial injury, has been found elevated in COPD patients, reflecting underlying cardiac stress and disease severity.

Method: This hospital-based observational study was conducted on diagnosed COPD patients. Clinical evaluation, spirometry-based GOLD staging, and laboratory investigations including serum Troponin I levels were performed. Patients were categorized based on disease severity, and associations between Troponin I levels and clinical parameters such as exacerbations, hospital stay, and outcomes were analyzed.

Result: Elevated Troponin I levels were observed in a significant proportion of COPD patients and showed a positive association with increasing disease severity (higher GOLD stages). Patients with raised Troponin I had longer hospital stays, increased frequency of exacerbations, and higher need for intensive care support. A significant correlation was also found between Troponin I levels and pulmonary function impairment, showing worsening clinical status.

Conclusion: Troponin I is a valuable biomarker for assessing disease severity and predicting clinical outcomes in COPD patients. Its elevation reflects underlying myocardial stress and can aid in early risk stratification and management.

Keywords: COPD; Troponin I; GOLD stage; myocardial injury; exacerbation; hospital stay; prognosis

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INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a progressive and debilitating respiratory disorder characterized by persistent airflow limitation that is not fully reversible. It encompasses chronic bronchitis and emphysema, resulting from prolonged exposure to harmful particles or gases, most commonly tobacco smoke, environmental pollution, and occupational hazards. COPD is a major global health concern and is projected to become the third leading cause of death worldwide, contributing significantly to morbidity, mortality, and healthcare burden [1,2].

COPD is increasingly recognized as a systemic disease with significant extrapulmonary complications, particularly cardiovascular disorders. Patients with COPD have a higher risk of ischemic heart disease, heart failure, arrhythmias, and pulmonary hypertension. The coexistence of cardiovascular disease in COPD patients worsens prognosis and increases hospitalization and mortality rates. Shared risk factors such as smoking, systemic inflammation, oxidative stress, and hypoxia play a crucial role in linking pulmonary and cardiovascular pathology[3,4].

Cardiac biomarkers have gained importance in identifying subclinical myocardial injury in COPD patients. Among these, Troponin I, a highly specific and sensitive marker of myocardial damage, has emerged as a useful tool. It is released into the bloodstream following cardiac muscle injury and is widely used in the diagnosis of acute coronary syndromes. Recent evidence shows that Troponin I levels may be elevated in COPD patients even in the absence of overt cardiac disease, particularly during acute exacerbations, reflecting myocardial stress, hypoxia-induced injury, and right ventricular strain[5,6].

The severity of COPD is commonly assessed using the GOLD staging based on spirometric parameters such as FEV_1. Studies have showed a possible association between elevated Troponin I levels and increasing severity of COPD. Elevated levels have also been linked with longer hospital stays, increased need for ventilatory support, and higher mortality rates [7,8].

Understanding the relationship between Troponin I levels and COPD severity may provide valuable insights into the interplay between respiratory and cardiovascular systems. It may also help in early identification of high-risk patients, enabling better clinical management and improved

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outcomes. Therefore, this study aims to assess the levels of Troponin I in COPD patients and evaluate its association with disease severity and clinical outcomes [9].

METHODOLOGY

This study was conducted as a hospital-based observational study in the Department of Medicine at a tertiary care center. A total of diagnosed cases of COPD, confirmed by clinical evaluation and spirometry as per GOLD criteria, were included in the study. Patients aged above 18 years who consented to participate were enrolled, while those with known ischemic heart disease, acute coronary syndrome, chronic kidney disease, or other conditions known to elevate Troponin I were excluded to avoid confounding bias.

The sample size was calculated considering 7.4% of COPD prevalence in India based on Daniel et al. systematic review and meta-Analysis.[10] The Cochran formula was used and total 74 patients were considered for the study based on calculated sample size. After obtaining informed consent, detailed demographic data, clinical history including smoking status, duration of illness, frequency of exacerbations, and comorbidities were recorded using a structured proforma. A thorough clinical examination was performed for all patients. Laboratory investigations including complete blood count, renal function tests, and serum Troponin I levels were carried out at the time of admission. Troponin I was measured using a standard immunoassay method. Pulmonary function tests were performed, and patients were classified into different GOLD stages based on FEV_1 values to assess disease severity.

Patients were followed during their hospital stay to record clinical outcomes such as duration of hospitalization, need for ventilatory support (non-invasive or invasive), and overall outcome. The collected data were compiled and analysed using appropriate statistical methods. Continuous variables were expressed as mean ± standard deviation, while categorical variables were expressed as frequency and percentage. Associations between Troponin I levels and severity of COPD, as well as clinical outcomes, were assessed using suitable statistical tests, with a p-value <0.05 considered statistically significant.

RESULT:

Table 1: Distribution of Clinical Features among COPD Patients (n=74)

Clinical Feature	Present n(%)	Absent n(%)
Fever	39(52.7)	35(47.3)
Cough	52(70.3)	22(29.7)
Dyspnea	43(58.1)	31(41.9)
Crepitation	46(62.2)	28(37.8)
Wheezing	61(82.4)	13(17.6)
Cyanosis	24(32.4)	50(67.6)
Clubbing	31(41.9)	43(58.1)

The distribution of clinical features among COPD patients showed that wheezing was the most common symptom (61, 82.4%), followed by cough (52, 70.3%), crepitation (46, 62.2%), and dyspnea (43, 58.1%). Fever was present in 39 (52.7%) patients. Less frequent findings included clubbing (31, 41.9%) and cyanosis (24, 32.4%). Overall, respiratory symptoms predominated in the study population.

Table 2: Clinical and Laboratory Parameters of COPD Patients

Parameter	Value (mean±SD)
SpO_2 (%)	84.0±7.5
FEV_1 (%)	54.8±16.0
FEV_1/FVC (%)	52.2±12.1
Sodium (mEq/L)	131.5±5.6
Potassium (mEq/L)	3.2±0.4
Troponin-I (ng/mL)	0.09±0.05
Hyponatremia	47(63.5)
Normal Na^+	27(36.5)
Hypokalemia	53(71.6)
Normal K^+	21(28.4)

The mean SpO_2 was 84.0±7.5, showing hypoxemia, while pulmonary function parameters showed reduced values (FEV_1: 54.8±16.0 and FEV_1/FVC: 52.2±12.1). Electrolyte imbalance was common, with hyponatremia in 47 (63.5%) and hypokalemia in 53 (71.6%) patients. The mean troponin-I level was 0.09±0.05, showing mild elevation in many cases.

Table 3: Association between GOLD Stage and Troponin-I Levels

GOLD Stage	n(%)	Troponin-I (mean±SD)
Mild	3(4.1)	0.07±0.02
Moderate	46(62.2)	0.07±0.03
Severe	18(24.3)	0.11±0.06
Very Severe	7(9.5)	0.17±0.06
Total	74(100.0)	0.09±0.05
p-value		**<0.001**

Troponin-I levels increased progressively with the severity of COPD as per GOLD staging, from 0.07±0.02 in mild cases to 0.17±0.06 in very severe cases. The majority of patients were in the moderate stage (46, 62.2%). This association between disease severity and myocardial injury was statistically significant ($p<0.001$).

Table 4: Duration of Hospital Stay and Troponin-I Levels

Duration of Stay	n(%)	Troponin-I (mean±SD)
<5 days	24(32.4)	0.08±0.04
5–10 days	32(43.2)	0.10±0.05
>10 days	18(24.3)	0.08±0.05
Total	74(100.0)	0.09±0.05
p-value		**0.446**

The majority of patients had a hospital stay of 5–10 days (32, 43.2%). Troponin-I levels were slightly higher in patients with 5–10 days stay (0.10±0.05), but no consistent increasing trend was observed with longer duration. The association between hospital stay and troponin-I levels was statistically insignificant ($p=0.446$).

Table 5: Frequency of Exacerbations and Troponin-I Levels

Frequency	n(%)	Troponin-I (mean±SD)
One	9(12.3)	0.09±0.05
Two	24(32.9)	0.07±0.04
Three	26(35.6)	0.09±0.05
Four	14(19.2)	0.11±0.06
Total	74(100.0)	0.09±0.05
p-value		**0.189**

Most patients experienced three exacerbations (26, 35.6%), followed by two exacerbations (24, 32.9%). Troponin-I levels showed a slight increase with higher exacerbation frequency, reaching 0.11±0.06 in patients with four exacerbations. However, this association was statistically insignificant ($p=0.189$).

Table 6: Type of Therapy and Troponin-I Levels

Type of Therapy	n(%)	Troponin-I (mean±SD)
No Oxygen	4(5.4)	0.11±0.06
Nasal Oxygen	41(55.4)	0.09±0.05
NIV	17(23.0)	0.08±0.03
Mechanical Ventilation	12(16.2)	0.10±0.05
Total	74(100.0)	0.09±0.05
p-value		**0.734**

Nasal oxygen therapy was the most commonly used modality (41, 55.4%), followed by NIV (17, 23.0%) and mechanical ventilation (12, 16.2%). Troponin-I levels were relatively similar across different therapy types, with slightly higher values in patients not receiving oxygen (0.11±0.06). No statistically significant association was found between type of therapy and troponin-I levels (p=0.734).

DISCUSSION

In the present study, the association between serum Troponin-I levels and severity of COPD was evaluated, along with its correlation with clinical and functional parameters.

The mean age of the study population was 51.1±7.3 years, with a predominance of males (62.2%). This finding is similar to previous studies showing increased COPD prevalence after the fifth decade of life. Similar trends were reported by Vilkman et al., who observed a rise in COPD-related hospitalizations after 50 years 11, and Martinez-Delgado et al., who reported male predominance among COPD patients 12. Smoking was present in 70.3% of patients, supporting its established role as the major etiological factor in COPD 13,14. These observations are similar to global data showing tobacco exposure as a key contributor to COPD burden [15].

Most patients were in the moderate (62.2%) and severe (24.3%) stages of COPD. This distribution is comparable to previous studies such as Safka et al. 16 and global GOLD data, where the majority of patients fall in moderate to severe categories. This reflects delayed diagnosis and progressive nature of the disease.

A key finding of this study was the significant rise in Troponin-I levels with increasing GOLD stage ($p<0.001$). Patients in severe and very severe stages had markedly higher Troponin-I levels compared to mild and moderate stages. This finding is similar to studies by Adamson et al. 17 and Nirmala Devi et al. [18], which showed that elevated Troponin-I correlates with disease severity and cardiovascular risk. The progressive rise in Troponin-I levels with COPD severity shows underlying myocardial stress due to chronic hypoxia, pulmonary hypertension, and systemic inflammation. Thus, Troponin-I can serve as a surrogate marker of disease burden in COPD.

A strong negative correlation was observed between Troponin-I and pulmonary function parameters such as SpO_2, FEV_1, and FEV_1/FVC ratio ($p<0.001$). This shows that worsening lung function is associated with increased myocardial stress. Similar findings were reported by Surma et al. 19, who showed an inverse relationship between troponin levels and lung function indices. These findings support the concept that hypoxemia and airflow limitation contribute to myocardial injury, highlighting the interplay between respiratory and cardiovascular systems in COPD.

Although patients with higher Troponin-I levels showed a trend toward longer hospital stays, the association was not statistically significant (p=0.446). This shows that while Troponin-I reflects disease severity, it may not independently predict duration of hospitalization. Similar observations were noted in previous studies where hospital stay was influenced by multiple factors including comorbidities and treatment response [18].

The frequency of exacerbations showed a non-significant association with Troponin-I levels (p=0.189), although a gradual increasing trend was observed. This shows that repeated exacerbations may contribute to cumulative myocardial stress, but the relationship is not strong enough to be statistically significant. Similar findings have been reported in other studies where exacerbation frequency alone did not strongly correlate with cardiac biomarkers [20].

Most patients required oxygen therapy, with 55.4% on nasal oxygen and 39.2% requiring ventilatory support (NIV or mechanical ventilation). However, no significant association was found between type of therapy and Troponin-I levels (p=0.734). This shows that Troponin-I elevation is more reflective of underlying disease severity rather than treatment modality.

A strong association was observed between elevated Troponin-I levels and adverse clinical outcomes. Patients with higher Troponin-I levels had increased mortality, with 50% deaths observed in the highest percentile group. This finding is similar to studies by Noorain et al. 21 and Elmenawi et al. [8,] which showed that elevated troponin levels are associated with poor prognosis and increased mortality in COPD patients.

Conclusion: The present study demonstrates that serum Troponin-I levels increase significantly with the severity of COPD and show a strong association with worsening pulmonary function. Elevated Troponin-I serves as an indicator of underlying myocardial stress and hypoxia in COPD patients. Although no significant association was found with hospital stay, exacerbation frequency, or type of therapy, higher levels were linked with poor clinical outcomes and increased mortality. Thus, Troponin-I can be

considered a useful biomarker for assessing disease severity and prognostication in COPD.

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