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Original Research Article

The study on association of elevated cardiac biomarker in patients of acute exacerbation of chronic obstructive pulmonary disease

Deepshikha[1,*], Sajeet Verma[1], Mahim Mittal[1]

[1] *Dept. of Medicine, BRD Medical College, Gorakhpur, Uttar Pradesh, India*

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ABSTRACT

Background: Chronic obstructive pulmonary disease (COPD) is the third leading cause of death worldwide, and also resulting in economic and social burden which is substantial and is significantly increasing with time. Chronic obstructive pulmonary disease is frequently associated with right ventricular loading and pulmonary hypertension. Study aimed to evaluate a possible association between cardiac biomarker levels and adverse events in hospitalized patients with Acute exacerbations of chronic obstructive pulmonary disease comorbidities are important determinants of outcome and quality of life of patients with chronic obstructive pulmonary disease.

Materials and Methods: An observational, cross-sectional study was conducted on 89 patients with AECOPD admitted in the medicine emergency ward of Baba Raghav Das medical college and Nehru Hospital Gorakhpur India. Acute exacerbations of chronic obstructive pulmonary disease were diagnosed according to Global Initiative for chronic obstructive lung disease guidelines. cTnI levels were estimated at the time of admission by method based on chemiluminescence with other investigations. Levels ≥ 0.01ng/ml was taken as positive. Tabulation and statistical analysis were performed using Microsoft Excel and SPSS v.17.0 software. p –value< 0.05 at 95% confidence intervals taken as statistically significant.

Results: 89 patients with AECOPD were enrolled, among them mean age was 59.59±8.72. We found that cardiac biomarker elevation (cTnI≥0.01ng/ml) is significantly associated with increased mortality, duration of hospitalization, need of ventilation (both invasive or non-invasive) & cardiovascular morbidity. cTnI positivity predicted increased need of ICU admission and significantly increased duration of hospital stay (P=0.0001). cTnI positivity can prognosticate need of NIV or ventilator support. It was found in our study that ventilator support necessity & mortality was more in cTnI positive patients which shows statical significance (P=0.006).

Conclusion: cTnI in AECOPD has close relation with exacerbation od disease, right ventricular dysfunction, so it can be used as prognostic marker for mechanical ventilation requirement in future.

1. Introduction

Chronic obstructive pulmonary disease (COPD) is a lung disease characterized mainly by airflow limitation that is not fully reversible and corresponds to the major cause of chronic respiratory failure and Cor-pulmonale.[1,2] Globally, COPD is one of the main and significant cause of morbidity, mortality, and health-care costs. It is a global health issue, with cigarette smoking being an important risk factor universally. According to WHO 328 million people globally suffer from moderate to severe COPD, of which 3.5 to 4 million deaths have occurred annually worldwide because of COPD and its complications and in 15 years it will become leading cause of death worldwide.[3–5] Troponin I values are used to assist in the diagnosis of myocardial

* *Corresponding author*.
E-mail address: dr.deeps@rediffmail.com (Deepshikha).

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infarction. cTnI plays an integral role in the regulation of muscle contraction. Cardiac biomarker may aid in the assessment and the prognosis in patients with AECOPD.

Troponin I is a regulatory subunit of the troponin complex in conjunction with troponin C & troponin T. troponin I plays an important role in regulation of cardiac muscle contraction. Following myocardial infarction cTnl release into the blood stream within hours.

The spectrum of cardiovascular complications associated with COPD is clearly broad. Right ventricular (RV) dysfunction and pulmonary vascular disease are common in COPD and progress with time. The cardiovascular alterations are extremely complex. During an acute exacerbation, the increased work and oxygen cost of breathing, the increase in left ventricular after load related to the more negative intrathoracic pressure, the worsening of pulmonary hypertension, and the presence of hypoxemia and hypercapnia may all contribute to the development of cardiac injury.[6,7]

In AECOPD, there is increased cardiac burden even in the absence of cor-pulmonale. Prompt diagnosis of compromised cardiac function in these patients remains difficult because of nonspecific signs and symptoms and echocardiography may not always be available. Therefore, there is a need for biomarkers, not only to confirm cardiac involvement but also to predict the severity and outcome. In developing countries like India, outcome estimation is particularly important because of high costs of treatment and resource paucity. The most critical decisions are taken in the very first hours after admission. Finding a simpler and more readily available indicator of severity to assist in decision making would be of paramount importance.

The aims of the present study were to ascertain the association, of cTnI during acute exacerbations of COPD, and evaluate their prognostic implications, namely on mortality and need for NIVS, need of ventilator support, duration of hospital stay.

2. Materials and Methods

The study design was an observational cross-sectional study conducted in 89 patients with acute exacerbation of COPD admitted to the Medicine Department at in BRD Medical College & Nehru Hospital Gorakhpur. All patients with AECOPD, who are willing to participate in study included and those patients having previous history of coronary artery disease, Chronic systemic illness like CKD, Decompensated chronic liver disease, Prior H/O lung surgeries, genetic disorders (cystic fibrosis, alpha 1 Antitrypsin deficiency) causing lung function compromise& Cardiac arrest on admission, are excluded from study. The informed written consent was obtained from all the patients. The study was approved by the Institutional Research and Ethics Committee.

All patients are examined through detailed medical history and general physical examination was done for every patient at the time of admission. Blood samples were taken at the time of admission to analyse cardiac biomarker levels along with routine investigations. Cardiac Troponin, was considered negative if value<0.01 ng/ml, and positive if value ≥0.01 ng/ml.

2.1. Cardiac troponin I assay

cTnI assessment were done by using the Architect Stat system (Abbott Diagnostics) two-step immunoassay to determine the presence of cTnI in plasma & serum using CMIA (chemiluminescent microparticle immunoassay) for quantitative determination. Heparinized plasma, EDTA plasma & serum specimens may be used for the Architect Stat (Abbott Diagnostics) cTnI Assay.

Tabulation and statistical analysis were performed using Microsoft Excel and SPSS v.17.0 software. Numerical data were summarized by measures of central tendency, mean and standard deviation. Qualitative data was analysed with descriptive statistics & two way univariate analysis were used for comparing the study variables. If p –value< 0.05 at 95% confidence intervals it's taken as statistical significance. The unpaired t-test was used to compare continuous variables. Binary logistic regression analysis was carried out to find the strength of association of Troponin I with various factors.

3. Results

The study was conducted on 89 patients with AECOPD they were 61 males (68%), 28 females (31%) with a median age of 59.59±8.72 years. In this study of cTnI positive in 22 patients and cTnI negative in 67 patients, all baseline characteristics and clinical data of patients with AECOPD and its relation to cardiac Troponin I level are summarized in Table 1, that shows no statistical significant difference in cTnI level in relation to age, sex, smoking habits and tobacco chewing as well as with comorbidities P > 0.05).

hows relation between troponin level and different parameters studied among AECOPD patientsat the time of admission. The meanPaO2, SpO2, FEV_1/FVC level is low among cTnI positive patient as compare to cTnI negative patients and the difference is statistically significant and mean PaCO2 and PaSP level is low among cTnI negative patients in comparison to cTnI positive patients which is statistically significant. No cardiac disease or complications reported during study.

hows a significant difference in Troponin level in relation to Place of admission, need for type of ventilation, duration of days on ventilator, duration of hospital stay and Outcome (P < 0.05)

Table 1: Baseline characteristics & clinical data of patients with AECOPD & its relation to cTnI level (ng/ml)

Parameter	cTnI<0.01 (n=67)	cTnI≥0.01 (n=22)	P-value
Age (Mean ±SD)	59.26±8.34	60.59±9.95	0.54
Sex			
Males (n-61)	49 (80.3%)	12 (19.6%)	>0.05
Females (n-28)	18 (64.2%)	10(35.27%)	
Smoker			
a) Present (n=51)	40 (78.4%)	11 (21.6%)	0.42
b)Absent(n=38)	27 (71.1%)	11 (28.9%)	
Tobacoo CHEWING			
a) Present (n=35)	29(82.9%)	6(17.1%)	0.18
b) Absent(n=54)	38(70.4%)	16 (29.6%)	
Comorbidity			
a) Hypertension			
(i) Present (n=35)	24(68.6%)	11 (31.4%)	0.23
(ii)Absent(n=54)	43(79.6%)	11 (20.4%)	
Diabetes mellitus			
(i)Present (n=24)	19(79.2%)	5(20.8%)	0.6
(ii)Absent(n=65)	48(73.8%)	17 (26.2%)	

Table 2: Relation between cTnI level and different parameters studied among AECOPD Patients.

No.	Parameters	cTnI<0.01	cTnI≥0.01	P-Value
1	PaO2(mmHg)	78.32±8.99	69.00±10.21	0.0001
2	PaCo2 (mmHg)	45.32±9.31	65.68±9.88	0.0001
3	Spo2	93.17±6.49	87.59±10.07	0.003
4	FEV1/FVC	0.67±0.05	0.60±0.04	0.0001
5	Respiratory Rate	21.86±6.84	22.18±5.75	0.84
6	BNP	173.18±548.2	1313.41±895.9	0.0001
7	Pasp	39.71±8.24	48.27±6.06	0.001

Table 3: Relation between cTnI level & different mode of ventilation, duration of hospital stay & outcome studied among AECOPD patients

No.	Parameters	cTnI<0.01	cTnI≥0.01	P-Value
1	Patients (n=89)			
	i) Invasive Ventilation (12)	3 (25%)	9(75%)	
	ii) Noninvasive Ventilation (19)	6 (31.6%)	13 (68.4%)	0.69
	III) N0 (58)	58(100%)		
2	Duration of days on ventilator	3.77±0.97	5.36±1.46	0.006
3	Duration of hospital stay	5.57±3.12	8.95±3.47	0.0001
4	Outcome			
	I) Death (8)	2(25%)	6(75%)	0.001
	II) Alive (81)	65 (80.2%)	16 (19.8%)	

4. Discussion

COPD is life threatening disease with ever increasing incidence and prevalence. Comorbidities are important determinants of outcome and quality of life of these patients with chronic obstructive pulmonary disease (COPD). The risk of cardiovascular events in COPD patients is three to five folds high. Cardiac dysfunction may trigger acute exacerbation in up to 25% to 30% of these patients, whereas acute respiratory failure itself may lead to right ventricular failure.[8] So the identification of cardiac dysfunction during AECOPD remains difficult because of the non-specific clinical signs, so that there is a need for a biomarker not only to provide confirmation of exacerbation but also to predict the severity of such patients.[9]

The elevated levels of serum cardiac troponins have also been documented in RV dysfunction. In RV failure, cardiac troponins are suspected to be elevated secondary to RV ischemia or microinfarction resulting from increased wall tension, metabolic demand, and reduced coronary perfusion with or without atherosclerosis.[10,11]

Troponin I is a component of the contractile proteins present in all muscles. The amino acid sequence of cardiac troponin I (cTnI) contains a section that is unique to cardiac

muscle. The cTnI assay measures these cardio-specific components to provide a highly specific marker for cardiac muscle cell injury.[12,13] It has no cross-reactivity with the two skeletal muscle isoforms. cTnI is a highly sensitive and long-lasting marker of cardiac injury. Measurements of cTnI concentrations in renal failure, in myopathic states, and after acute skeletal muscle injury have shown normal concentrations in the absence of cardiac injury.

Study group consist of total 89 patients out of which 61 patients are male and 28 patients are female. All patients are admitted in medicine ward and intensive care unit according to their symptoms. The patients studied were aged between 40 to 80 years having overall mean age of 59.59±8.72 years. Among cTnI positive patients mean age is 60.59±9.95 years and cTnI negative patients mean age is 59.26±8.34 years. In the study most of the patients were male (68%) and female are (31%). In the present study, no significant difference was found between mean age of patients with positive cTnI and patients with cTnI levels negative. Baillard et al.[8] found no significant difference between positive and negative cTnI patients as regarding age in their study.

In contrast, Harvey and Hancox[14] reported a significant difference in the mean age of cTnI-positive patients compared with cTnI- negative patients. There is no difference in ratio of male to female in our study. There is equal prevalence in both groups of patients. This is in agreement with the results of Harvey and Hancox and Deveci et al.[15] Aksay et al[16] however, reported contrasting findings. They found a significant difference in sex among AECOPD patients. This finding could be attributed to the large number of female patients included in their study (59%), with most of them being diagnosed with pulmonary embolism (PE).

4.1. Risk factors

Smoking and tobacco usage are important risk factors for progression and development of COPD. In our study overall 39.3% are tobacco chewers and 57.3% are smokers, both risk factors are insignificantly associated with cTnI positivity table. This is in agreement with the studies by Baillard et al.[8] and Deveci et al.[16] who excluded the effect of smoking on cTnI levels among all studied patients. These results suggest that smoking has no effect on cTnI level in AECOPD patients.

4.2. Comorbid conditions

Comorbid conditions like Diabetes and Hypertension are associated with equal prevalence in both groups, in our study overall 39.3% are hypertensive and 27% have diabetes mellitus. In our study cTnI positivity higher in hypertensive patients but this association is statistically insignificant as echocardiographic studies also shows no regional wall motion abnormalities and other ischemic changes.

4.3. Staging of COPD

As regards the severity of disease on the basis of FEV1/FVC, there was a significant statistical difference in troponin levels among all studied AECOPD patients when evaluated with pulmonary function tests, as 75% of troponin I-negative patients were in moderate stage, 36% of troponin I-positive patients were in severe stage, and 23% of troponin I-positive patients were in very severe stage.

4.4. Analysis of various difference among cTnI positive and negative patients of aecopd at the time of admission

In our study arterial blood gases parameters showed a significant statistical difference in troponin positivity in relation to pCO2, pO2, and SpO2. Baillard et al. reported a significant statistical difference in troponin positivity in relation to pCO2, pO2, and SO2, but no significant difference in relation to pH and HCO3. Harvey and Hancox[14] demonstrated a significant role of O2 saturation, pCO2, and on cTnI positivity.

ECHO findings showed the Pulmonary artery pressure (PaSP) has been increased in cTnI positive as well as in negative patient & mean value is more among cTnI positive patients as shown in Table 2.

The aim of our study is to establish cardiac biomarker as a prognostic marker in AECOPD patients. In our study 89 patients presented with acute exacerbation out of which 22 patients have cTnI positive and all these patients significant risk in outcome as evidenced by increased need for oxygen both on a quantitative and qualitative basis, increased need for mechanical ventilation i.e. more patients went through NIV rather than invasive ventilation with an endotracheal intubation, more hospitalized days had a significant association based on statistical analysis with a p value<.005.[17,18] In our study 8 patients died because of cardiopulmonary arrest, of which 6 patients are cTnI positive which is again significant thus the aim and objective of study is met with as shown in Table 3. In addition, MacIntyre and Huang[19] found that elevation of troponins was associated with increased severity of exacerbation. However, troponin T and pro-brain natriuretic peptide are elevated in patients with acute left heart failure and may be used to exclude left ventricular dysfunction as the cause of AECOPD. As regards the need for Invasive Ventilation or Noninvasive ventilation cTnI positivity was more prominent among patients who were ventilated versus those who did not need Invasive Ventilation or Noninvasive ventilation (Table 3). This finding is in agreement with those of Aksay et al. Who reported that there was a significant statistical difference in cTnI positivity between patients who needed mechanical ventilation and those who did not need mechanical ventilation.

As regards the duration of hospitalization (Table 3), a significant difference was seen in cTnI positivity in

relation to duration of hospitalization: cTnI positivity was more prominent in patients with longer duration of hospitalization. This could be attributed to the greater severity of the disease, exacerbation, the need for ICU admission, and the need for Mechanical Ventilation.[20,21] These findings are similar with those of Harvey and Hancox who found that patients with greater number of hospital days were more cTnI-positive compared with those with shorter duration. The same findings were made by King et al.[22] who found a significant effect of length of hospitalization on cTnI elevation, and by Martins et al[23] who found a significant effect of hospital length of stay upon cTnI elevation. An retrospective study performed by Harvey et al in 2004 having sample size 188 predicted that Significant association between raised troponin levels and increased length of hospital stay (p<.001) reported. A study performed by Baillard et al in 2003 having sample size 71 reported that Elevated cardiac troponin I is a predictor of in-hospital death in patients admitted for AECOPD (ORa 6.52; 95% CI 1.23 to 34.47) which is similar to our study.

In our study cTnI is positive about one fourth of patients (24.7%) and BNP is also significantly higher in cTnI positive patients and both cardiac biomarkers significantly contribute to cardiovascular mortality and morbidity and this is comparable with other studies which is done in other parts of world. However, a prospective study done by Stolz et al[24] done on 208 patients in 2008 predicted that Raised BNP levels on admission are not significantly associated with mortality at any time point but BNP levels are significantly higher in patients requiring ITU care and correlate well with need for ITU care and duration of stay.

Knowledge of determinants of troponin elevation in COPD exacerbation may improve our understanding of underlying pathophysiological processes, and ultimately, lead to improved therapeutic strategies.

Mechanism of elevation of cardiac biomarker in AECOPD patients is difficult to understand however, Aksay et al.[16] revealed a significant effect of RV dysfunction on cTnI elevation, and those of Harvey and Hancox who suggested that the severity of acute exacerbation may lead to cardiac damage and troponin release. Potential mechanisms of cardiac injury include the following, acute elevation of pulmonary arterial pressure secondary to hypoxemic vasoconstriction with subsequent RV distension (similar to the proposed mechanism of cTnI release in pulmonary embolism), tachyarrhythmia such as atrial fibrillation, and cardiac damage mediated by sepsis and/or metabolic stress due to hypoxia and acidosis. Seyhan et al[25] found a strong correlation between RV dysfunction and cTnI in a group of AECOPD patients.

Baillard et al.[8] did not report a significant effect of either RV dysfunction upon cTnI or corpulmonale. They stated that the reason for cTnI elevation is difficult to determine, because the cardiovascular alterations are complex. During episodes of exacerbation, the increased work and oxygen cost of breathing, the increase in left ventricular after load related to the more negative intrathoracic pressure, the worsening of pulmonary hypertension, and the presence of hypoxemia and hypercapnia may all contribute to the development of cardiac injury.

At last, there are several studies that could support our study that elevated cardiac biomarker can be used as prognostic marker in AECOPD patients in terms of increased length of stay in hospital, requiring long term oxygen therapy or invasive or non-invasive ventilation, and to predict mortality. Increased level of cardiac biomarker in AECOPD is complex phenomena to understand, during acute exacerbation COPD patients have increased cardiac burden as stated by Currie et al[26] due to hypoxia and pulmonary vasoconstriction can cause pulmonary hypertension and right ventricular dysfunction. Tachycardia, ventilation perfusion mismatch and respiratory muscle fatigue also contribute to cardiac stress, which may be exacerbated by an increased oxygen cost of breathing and increased left ventricular after load from dynamic hyperinflation.

Hessel et al[27] showed that in the presence of compounds that stimulate stretch-responsive integrins, viable cardiomyocytes release intact cardiac troponin I.COPD patients frequently have hypoxia, and thus the hypothesis that hypoxia also plays a role in troponin release in COPD patients cannot be ruled out, so in conclusion elevation in cardiac biomarker in AECOPD patients is due to right ventricular strain or overload, hypoxemia and pulmonary artery hypertension not per se due to ischemia because echocardiography of these patients do not show any regional wall motion abnormality.

5. Conclusion

AECOPD patients having cTnI≥0.01ng/ml may suggest severity of exacerbation, pulmonary hypertension, and right ventricular dysfunction. cTnI≥0.01ng/ml can be considered as prognostic marker for the possibility of need of mechanical ventilation, longer duration of hospitalization and outcome in terms of mortality. So, assessment of cTnI levels at the time of admission, can be done to identify the patients at significant risk. Early diagnosis followed by prompt intervention and treatment can improve outcome.

6. Conflict of Interest

None.

7. Source of Funding

None.

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Author biography

Deepshikha, Associate Professor

Sajeet Verma, Junior Resident

Mahim Mittal, Professor and HOD

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