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Original Research Article

Cardiac Markers in Acute Exacerbation of Chronic Obstructive Pulmonary Disease with Special Reference to Cardiac Troponin I

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Conflict of interest: Nil

Abstract

Background: Cardiovascular disease is commonly seen in patients with Chronic Obstructive Pulmonary Disease (COPD). Brain Natriuretic Peptide (BNP) is a marker of ventricular dysfunction and troponin I is of myocardial necrosis. Elevations of these markers have been found in many disorders, COPD being one of them. The aim of the study is to assess the association of elevated cardiac biomarkers with the 6-month mortality in patients with Acute Exacerbation of COPD (AECOPD).

Materials and Methods: 109 patients admitted with acute exacerbation of COPD were included in this prospective study. Cardiac Biomarkers- BNP and troponin-T were measured on admission. Patients were followed up for the next 6 months and mortality over this duration was recorded and analysed. Statistical Analysis- Pearson X2 testing was used. Setting and Design- The study was conducted in the Department of Medicine, Gauhati Medical College and Hospital, Gauhatir, Assam. It was a prospective hospital-based observational study.

Results: 109 patients who met the inclusion and exclusion criteria were included in the study. Among them 100 were males and 09 were female.34 were in the age group 50-59 years 47 were between 60-69 years 26 were between 60-69 years 26 were in between 70-79 years and 2 patients were abiove 80 years. TROP I was found to be positive in 41 patients and negative in 68 patients

Conclusion: In COPD patients presenting with acute exacerbation, an isolated elevation of troponin level or a combined elevation of BNP and troponin-T levels are strong predictors of mortality (9 fold and 12 fold increase respectively). However, elevated BNP in isolation is not associated with increased mortality.

Keywords: Pulmonary Disease, Chronic Obstructive, Mortality, Natriuretic Peptide, Brain, Troponin.

Introduction

Chronic Obstructive Pulmonary Disease (COPD) is a commonly encountered non-communicable disease. It constitutes about 30% of all the cases seen in chest clinics and accounts for 1-2.5% of all hospital admissions in India.[1] COPD is also a major cause of morbidity and mortality throughout the world. As per W.H.O, it is the third leading cause of death worldwide behind ischaemic heart disease and stroke.[2] India contributes a significant percentage of COPD mortality, which is estimated to be amongst India contributes a significant percentage of COPD mortality, which is estimated to be amongst the highest in the world with about 5,56,000 (>20%) out of a world total of 2,748,000 annually.[3] Developing countries like India are changing fast. Socioeconomic development, industrialisation, urbanisation, changing age structure and changing lifestyles have the countries at a position where they are facing an ever increasing burden of non-communicable diseases such as COPD.[4] COPD is defined by the Global Initiative for Chronic Obstructive Lung Disease (GOLD) as 'a common preventable and treatable disease characterised by persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases with exacerbations and comorbidities contributing to the overall severity in individual patients.'[5] Established risk factors for development of COPD include tobacco smoking, environmental tobacco smoke, hyper-responsiveness to various exogenous stimuli, occupational coal dust exposure, prolonged exposure to smoke produced by biomass combustion and alpha-1- antitrypsin deficiency.[6] Acute Exacerbations of COPD (AECOPD) are a prominent feature of the natural history of COPD. Exacerbations are defined as- "an acute event characterised by a worsening of the patient's respiratory symptoms that is beyond normal day-to-

day variations and leads to a change in medication."[5] The clinical severity of an AECOPD varies widely. It may be managed in the outpatient setting or maybe severe enough to require hospitalisation. If complicated by respiratory failure, intensive care including noninvasive or invasive ventilator support maybe required.[7] Ball et al reported that AECOPD contributes considerably to the morbidity and the diminished quality of life of people afflicted with COPD. Patients who suffer the most exacerbations have significantly lower health status.[8]

Kanner et al showed that exacerbation frequency predicts an accelerated decline in lung function.[9] Episodes of AECOPD have been associated with diminished physical activity and increase in cardiovascular risk, osteoporosis and neuropsychiatric complications.7 Exacerbations of COPD have considerable impact on healthcare system at both primary and tertiary care levels as they are the major reason for antibiotic use and admissions. Additionally, such episodes lead to indirect costs because of days lost from work.[10]

Most importantly, an increase in in-hospital and future mortality is seen in frequent exacerbators of COPD.[11] A variety of stimuli may result in the final common pathway of airway inflammation and increased symptoms that are characteristic of COPD exacerbations. Factors that precipitate exacerbations of COPD are respiratory tract infection- bacterial or viral, air pollutants, interruption of maintenance therapy or resumption of smoking, pulmonary embolism, congestive cardiac failure, pneumothorax, cardiac arrhythmia, pleural effusion, lower temperature and humidity and higher barometric pressure. In 20-35% cases, no specific precipitant can be identified. [7,12,13]

Brain Natriuretic Peptide (BNP) is an established marker of left ventricular dysfunction. [14,15] It is associated with increased mortality in acute and stable heart disease. It is also increased in right ventricular overload and predicts a poor outcome in pulmonary arterial hypertension. [16,17,18] Cardiac troponins are specific markers of myocardial necrosis.[19]

Elevated troponin levels may also occur in pulmonary thromboembolism, congestive heart failure, tachyarrhythmias, myocarditis, pericarditis, sepsis and stroke.[20] Troponin elevations in these conditions reflect general myocardial injury rather than coronary arterial occlusion. Retrospective observations of troponin elevations in acute exacerbations of COPD have been made and are associated with the severity of exacerbation.

Aims and Objectives: To find out the incidence of cardiac troponin I levels elevation in acute exacerbation of COPD patients

Materials and Methods

Study Setting: The present study was conducted in the Department of Medicine, Gauhati Medical College and Hospital, Gauhati, Assam. Period of Study- The present study was conducted from 1 st June 2021 to 31st may 2022.

Sample Size: A total of 109 patients admitted in the Medicine Ward at GMCH were included in the study after fulfilment of the inclusion and exclusion criteria.

Study Design: The study was a hospital-based prospective observational study.

Methodology: All the patients with clinical features suggestive of Chronic Obstructive Pulmonary Disease (COPD) such as progressive dyspnoea, chronic cough, chronic sputum production with history of exposure to risk factors or a family history of COPD were assessed. Out of these, the patients fulfilling the criteria for Acute Exacerbation of COPD (AECOPD) were enrolled. AECOPD was diagnosed as per the criteria put forth by Anthonisen et al.

Presence of any two of the following symptoms.

- Increased cough
- Increased purulence and/or volume of expectorations and
- Increased severity of dyspnoea

These patients were immediately treated and stabilized. Informed and written consent was obtained from the patients.

A detailed clinical history was teken and complete physical examination was done in all cases. A spirometry was performed on all patients along with bronchodilator revesibilty testing. A post bronchodilator FEV1/FVC of <0.7 confirmed the diagnosis of COPD as per GOLD 2015 guidelines. Severity of COPD was assessed using FEV1 % predicted values. Complete blood count, renal and liver function, cardiac markers (TROP I, BNP), chest radiograpgh, ECG and 2D echocardiography were done in all patients were followed up over the next 6 months and mortality over this duration was recorded.

Inclusion Criteria: All patients above 18 years of age diagnosed with COPD as per GOLD criteria presenting with acute exacerbation were included in the study.

Exclusion Criteria

1. Known cases of malignancy or immuno-suppression.
2. Tuberculosis.
3. Known cases of heart failure.
4. Pneumoni.
5. Asthma.

6. Unwilling patients.

Ethical Clearance: Ethical clearance for the study was taken from the Ethical Committee Gauhati Medical College and Hospital Gauhati, Assam. The study was conducted in accordance with the ethical standards of the responsible committee on human experimentation and with the Revised Helsinki Declaration of 2000.

Statistical Analysis: The data collected were compiled, tabulated and analysed in terms of descriptive statistics using SPSS version 17.0 software.

Continuous variables were presented as mean ± SD and categorical variables were expressed as frequencies and percentages. Categorical data between the groups were compared using Chi-square test. A p value

Results and Observation

Table 1: Gender Distribution

Gender	No. of Patients
Female	9
Male	100
Total	109

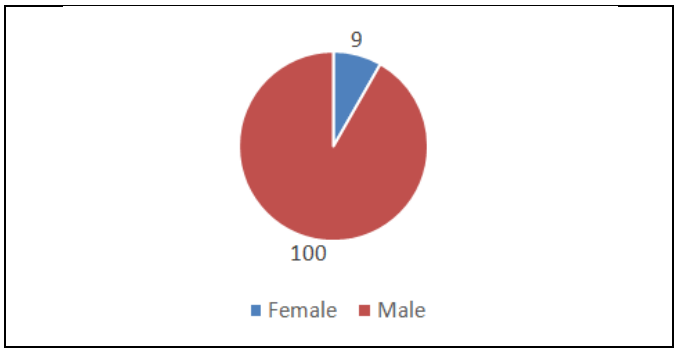

Figure 1: Gender Distribution

109 patients who met the inclusion and exclusion criteria were included in the study. Among them 100 were males and 09 were females.

Table 2: Age Distribution of Patients

Age in Years	No. of patients
50 – 59	34
60 – 69	47
70 – 79	26
>80	2
Total	109

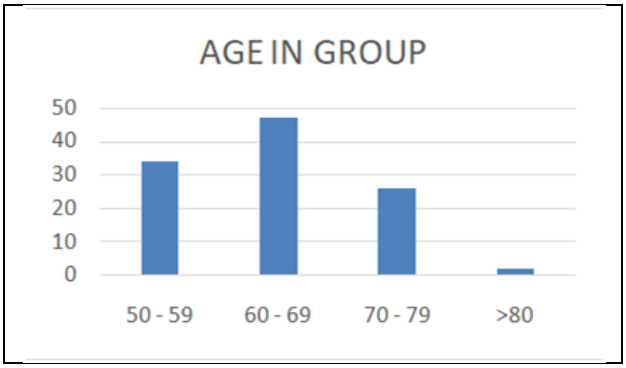

Figure 2: Age in Group

Table 3: Patient Groups According to Trop I Levels

Troponin I	No. Of Patients
POSITIVE (Group 1)	41
NEGATIVE (Group 2)	68
TOTAL	109

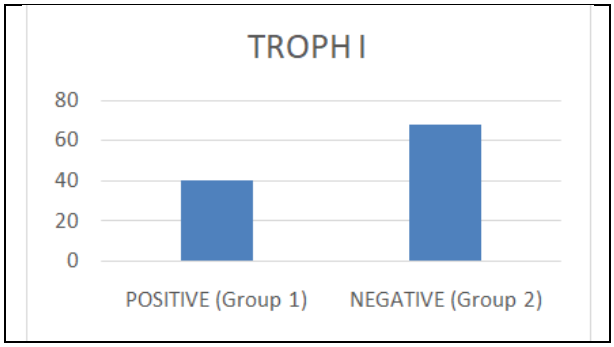

Figure 3: Troph I

Table 4: Age Distribution According to Trop I Levels

Age in years	Troph I +Ve	Troph I – Ve	Percentage
50-59	14	20	34
60-69	18	29	47
70-79	9	17	26
>80	0	2	2
Total	41	68	109

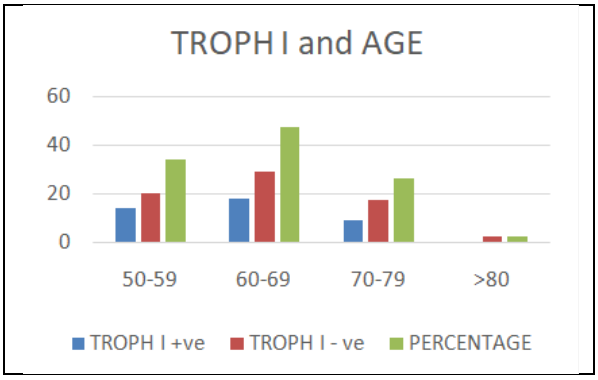

Figure 4: Troph I and Age

Table 5: Comparing Nt-pro-bnp Levels in Both Groups

Variables	Troph I +Ve	Troph I –Ve
Normal Nt Pro Bnp	8	35
High Nt Pro Bnp	33	33

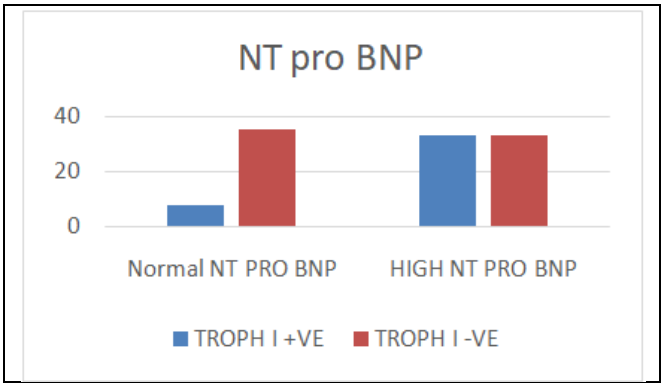

Figure 5: NT pro BNP

Table 6: Out Come in Both Groups

Variables	Discharged	Expired
TROPH I + ve	31	10
TROPH I – ve	64	4

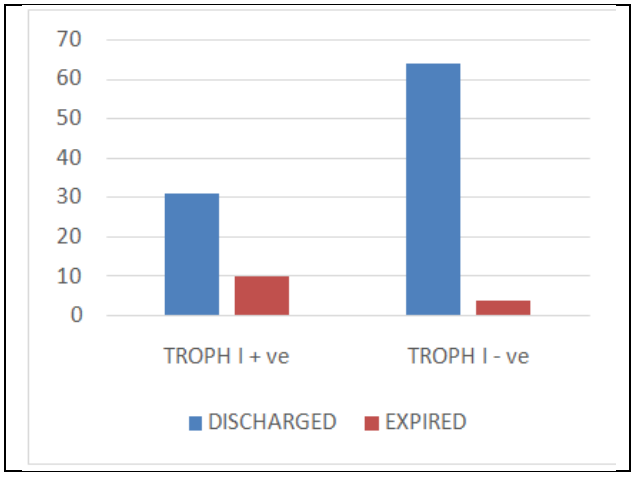

Figure 6: Outcome

Discussion

Isolated Elevation of Troponin I In the present study, elevated levels of troponin I were significantly associated with an elevated mortality by the end of six months (p=0.004; RR=9.0). In the study conducted by Chang et al, data on 244 subjects was collected over a period of one year.[22] 30-day mortality was calculated in these patients. Elevated troponin levels were significantly associated with increased mortality. In the study conducted by Baillard et al, a significant association was noted between an elevated cardiac troponin I level on admission and the in-hospital mortality in AECOPD cases. [23] Brekke et al conducted a study on this subject for a median period of about 1.9 years. Troponin I levels measured on admission were found to be strong independent predictors of mortality in their subjects.[24] These findings are in agreement with the findings of the present study. However, in the study conducted by Noorain et al, elevated troponin I levels were not found to predict inhospital mortality. They were associated with the need for ICU admission and ventilator support.[25] This difference between the present study and the study by Noorain et al maybe because of the fact that the present study included both deaths that occurred after discharge as well as those that occurred in-hospital

Combined Elevation of Troponin I and BNP In the present study, combined elevation of Troponin I and BNP were significantly associated with increased mortality by the end of six months ($p < 0.05$). This finding is in tally with the study conducted by Chang et al.[22] The presence of elevated biomarkers in patients of AECOPD are strong predictors of mortality in patients. All such patients should be advised regular follow up after discharge. They should be counselled regarding the elevated risk of mortality. Advice regarding smoking cessation, inhalational steroid use and domiciliary oxygen use should be offered. In case additional indications are present, surgical interventional measures such as lung volume reduction surgery should be offered.

Conclusion

Chronic Obstructive Pulmonary Disease contributes a substantial proportion to the disease burden of the country. Identifying the clinical profile and the risk factors is of immense importance in diagnosis of the disease as well as for primordial and primary prevention. Our study has led us to the conclusion that patients with a history of smoking, dyspnea and chronic cough should always be evaluated for chronic obstructive pulmonary disease. To determine the airflow restriction and prognosis after clinical evaluation, a rapid spirometric evaluation has to be carried out. Troponin I can be taken as a marker to identify high risk patients during acute exacerbation of COPD. Assessing troponin, I levels in AECOPD patients helps in identifying those who are at higher risks for poor outcomes. The results of the current study demonstrated that Trop I elevation in AECOPD was associated with increased duration of the disease, increased need for admission to ICU, increased ventilatory support requirement and increased mortality. This can help in the proper management of these patients for better outcomes. However, the limitation of this study is relatively smaller sample size, and a short duration. Hence it is difficult to draw definite inference. Therefore, large prospective follow up and analytical studies are required to arrive at a definite conclusion.

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