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Presence of Cardiac Troponin I in Patients with Acute Exacerbation of COPD

U. T. Mane[1*], Rahul S. Patil[1], A. T. Pardesi[1], Anil Bhattad[1] and Vaibhav Agarwal[1]

[1]Department of Medicine, Krishna Institute of Medical Sciences, Karad, India.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Chronic obstructive pulmonary disease (COPD) is frequently associated with right ventricular loading and pulmonary hypertension. We aimed to evaluate a possible association between cardiac troponin I (cTnI) levels and adverse events in hospitalized patients with acute exacerbation of COPD. 120 Patients with acute exacerbation of COPD were studied between 18 months (October 2014 to March 2016). A Male preponderance was found with M: F ratio being 3:2. 35% were current smokers. In hospital mortality in group 1 was 14.3% and in group 2 was 1.9%. Thus cardiac troponin I can be taken as a marker to identify high risk patients during acute exacerbation of COPD.

Keywords: Chronic obstructive pulmonary disease; troponin I; acute exacerbation.

1. INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a common, preventable and treatable disease characterized 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

**Corresponding author: E-mail: kimskarad66@gmail.com;*

gases. World Health Organization (WHO) predict that it will become the third leading cause of mortality by 2030; owing to decrease in cardiac disease and stroke over the period 1970-2002, but that of chronic obstructive pulmonary disease doubled over the same period [1]. It is also associated with significant economic burden. Chronic obstructive pulmonary disease often coexist with other comorbidities which may have a significant impact on its prognosis. Cardiovascular diseases, osteoporosis, lung cancer, diabetes, metabolic syndrome and depression are among a few of them. Cardiovascular disease is major comorbidity in COPD patients and probably the most frequent and most important coexisting illness as these conditions have many risk factors in common like age, male sex, cigarette smoking although its actual prevalence is unknown. COPD is characterized by slowly progressive air flow obstruction, resulting in dyspnea and exercise limitation, and pulmonary arterial hypertension is its major cardiovascular complication [2]. Right ventricular (RV) dysfunction is common in patients with COPD particularly in those with low oxygen saturation. It occurs in up-to 50% of the patients with moderate to severe COPD [3]. When present, it can reduce exercise tolerance, increase dyspnea and contribute to an overall decrease in functional status, and portends a higher mortality rate. Its recognition and treatment may lead to prolonged survival and improved quality of life. Other cardiac diseases found frequently in patients with COPD, including coronary artery disease and arrhythmias, present a unique challenge for clinicians, as the combination of both pulmonary and cardiac disease appears to be additive with regard to morbidity and mortality. There have been several studies to define the course of events in COPD. The major morbidity of COPD is due to the impact on cardiac performances, which is directly due to pulmonary arterial hypertension [4].

2. AIM AND OBJECTIVES

To study presence of positive cardiac troponin I as a prognostic marker in patients with acute exacerbation of chronic obstructive pulmonary disease. To evaluate the incidence of positive cardiac troponin I in patients with acute exacerbation of chronic obstructive pulmonary disease. To study the association of positive cardiac troponin I with the prognosis (outcome) of the patient in relation to mortality.

3. REVIEW OF LITERATURE

Hippocrates (460-369 B.C.) father of medicine described a patient of catarrh with cough and pedal edema. The earliest description of emphysema dates back to the latter half of seventeenth century. Dr. Badham in 1805 described patients with chronic cough, breathlessness and recurrent exacerbations and coined the term bronchitis [5]. In 1800 Humphrey Davy had determined his own lung volume using hydrogen as the test gas. Laennec (1826) described clinical features of emphysema and observed that heart failure can be produced by chronic lung disease. John Hutchinson invented the spirometer in 1846 [6]. A disease state characterized by the presence of airflow obstruction, due to chronic bronchitis or emphysema, airflow obstruction is generally progressive, may be accompanied by airway reactivity and may be partially reversible. A disorder characterized by reduced maximum expiratory flow, and slow forced emptying of the lungs, features which do not change markedly over several months. Chronic bronchitis is defined as a chronic productive cough in three months in each of two successive years in a patient in whom other causes of chronic cough (eg. Bronchiectasis) have been excluded. It may precede or follow development of airflow limitation. Hallmark of chronic bronchitis is cough with sputum production which develop insidiously, occurring initially only in the morning and volume rarely exceeds 60 ml of mucoid sputum. During acute exacerbation there is an increase in cough, purulent sputum, wheezing, dyspnea and occasionally fever which is intermittent and of mild degree. As the disease gets progressive the period between the acute attacks becomes shorter. Emphysema is defined by abnormal and permanent enlargement of the airspace distal to the terminal bronchioles that is accompanied by destruction of the airspace walls, without obvious fibrosis. (i.e. there is no fibrosis visible to the naked eye). As the disease progresses from chronic bronchitis to emphysema, the shortness of breath increases and there is scanty sputum production. In this stage patients develop physical stigmata of COPD like muscle wasting, weight loss, weakness and fatigue. At this stage any acute exacerbation gives rise to erythrocytosis and patients develop polycythaemia. They all start retaining carbon dioxide (hypercapnia) which usually presents with a morning headache and drowsiness due to lack of sleep at night. A patient with COPD tend to develop sleep apnoea

[7]. According to WHO estimates, 65 million people have moderate to severe COPD. More than 3 million people died of COPD in 2005, which corresponds to 5% of all deaths globally. It is known that almost 90% of COPD deaths occur in low and middle income countries. A systematic review and meta-analysis of studies carrying out in 28 countries 1990 and 2004, an additional study from Japan provide evidence that the prevalence of COPD is appreciably higher in smokers and ex-smokers than in non-smokers, in those over 40 years of age than those under 40, and in men than in women [4,8]. It is estimated that there are more than 12 million adults with COPD in India with prevalence rates varying depending upon the population studied and the methodology used. In males the prevalence has varied from 2.12% to 9.4% in north India and from 1.4% to 4.08% in south India [9,10]. The severity of COPD based on degree of airflow limitation if available should be noted. Duration of worsening of symptoms or onset of any new symptoms should be taken. Also important in history is the number of previous episodes of exacerbations and whether they required hospitalization or not. Any comorbidities that the patient is suffering from need to be addressed to. The present treatment regimen that the patient is on, any history of previous use of mechanical ventilation needs to be inquired.

4. MATERIALS AND METHODS

Patients admitted to tertiary care centre both male and female, with signs and symptoms of acute exacerbation of Chronic Obstructive Pulmonary Disease i.e. sudden worsening of Chronic Obstructive Pulmonary Disease symptoms i.e. shortness of breath, quantity and colour of sputum taken for study. COPD and AECOPD were diagnosed according to the global initiative for chronic obstructive lung disease 2011. I have taken all patients admitted and diagnosed with acute exacerbation of Chronic Obstructive Pulmonary Disease during the period of 1 October 2014 to 31 March 2016. Total 120 patients were taken for study.

Blood samples for Cardiac Troponin I detected on admission using qualitative assay by Troponin I test card by Chromatographic Immuno-assay method. Serum of patient separated by centrifugation and tested immediately. If two colored bands visible within 15 minutes, the test result taken as positive and valid. The test result could be read as soon as a distinct colored band appeared in the test area. If test area had no color band and the control area displayed a colored band, the result taken as negative and valid. The test result taken as invalid if a colored band did not form in the control region. That sample was then re-tested, using a new test device.

5. OBSERVATION AND RESULTS

120 patients who fulfilled the inclusion and exclusion criteria were included in the study.

As shown in Table 1, maximum 62.5% patients were between the age group 51 to 60 years, while above 80 age group constituted the second largest group with 15% patients.

As shown in Table 2, maximum 51.7% patients never smoked tobacco, while 35% were still smoking tobacco. 13.3% patients were former smokers (>1 year abstinence).

As shown in Table 3, of all the 120 patients who were included in the study, almost half i.e. 58 (48.3%) patients gave history of smoking tobacco (Current smokers and former smokers both inclusive). Another 41.7% patients gave history of use of biomass as fuel. Others (10%) had risk factors like sugar factory workers, working in boilers, and passive smoking.

5.1 Sensitivity of Test

This test detects cTnI with a concentration of 0.06 ng/ml or greater. Length of hospital stay,

Table 1. Age-wise distribution of patients

Age group in years	No. of patients	Percent (%)
31 – 40	15	12.5%
41 – 50	4	3.3%
51 – 60	75	62.5%
61 – 70	2	1.7%
71 – 80	6	5%
> 80	18	15%
Total	120	100%

including stay in ICU and duration of ventilator support (both non-invasive and invasive) noted and entered in data sheet. Outcome of patient at the time of discharge and in hospital mortality rate noted. With a statistical significant difference as regards the severity of exacerbation, and those with a past history of ICU admission and mechanical ventilation, ($P < 0.05$).

Table 2. Tobacco intake among patients

Tobacco Use	No. of patients	Percent (%)
Current Smoker	42	35.0
Former Smoker	16	13.3
Non Smoker	62	51.7
Total	120	100

6. DISCUSSION

Among the 120 patients included in this study, cardiac troponin I was found to be elevated in 14 patients which corresponds 11.7%. In this study elevated cardiac troponin I was associated with higher prevalence of co-morbidities like HTN, IHD and DM2 but it was not statistically significant. There was not a significant co-relation with the duration of disease; but patients with elevated cardiac troponin I had longer duration, a finding that hasn't been studied in any of previous studies. Positive cardiac troponin I was associated with significantly higher incidence of P pulmonale, dilated RA and RV and Pulmonary arterial hypertension (Cor pulmonale). Patients in Trop negative group had higher incidence of normal 2D Echo. Patients with Trop I positive were having significantly more tachypnea and tachycardia than Trop I negative group. There was significant difference in ABG picture and spirometric readings in both groups. Patients with Trop I positive group were more hypoxemic and hypercapnic. The mean FEV1% predicted and mean FEV1/FVC was significantly lower in Trop I

Table 3. Prevalence of risk factor for COPD among study population based on cardiac troponin-I levels

Sr. no.	Risk factor	No. of patients	Percent (%)
1.	Smokers (Current + Past)	58	48.3%
2.	Chulha Users (Biomass Users)	50	41.7%
3.	Others	12	10%

Table 4. Parameters for COPD among study population

Parameters	cTnI < 0.01 (n = 48)	cTnI ⍰ 0.01 (n = 72)	P-value
Left ventricular dysfunction			
+ve (n: 12)	4 (5%)	18 (21.4%)	<0.05
−ve (n: 108)	44 (95%)	54 (78.6%)	
Right ventricular strain			
+ve (n: 53)	13 (72.2%)	40 (95.2%)	<0.05
−ve (n: 7)	5 (27.8%)	2 (4.8%)	
TR			
+ve (n: 53)	12 (66.7%)	41 (97%)	<0.05
−ve (n: 7)	6 (33.3%)	1 (2.4%)	
AF			
+ve (n: 4)	0 (0%)	4 (9.5%)	>0.05
−ve (n: 56)	18 (100%)	38 (90.5%)	
P-pulmonale			
+ve (n: 57)	15 (83.3%)	42 (100%)	<0.05
−ve (n: 3)	3 (16.7%)	0 (0%)	
Sinus tachycardia			
+ve (n: 34)	4 (22.2%)	30 (71.4%)	<0.05
−ve (n: 26)	14 (77.8%)	12 (28.6%)	
Place admission			
ICU (n: 82)	4 (22.2%)	35 (83.3%)	<0.05
Ward (n: 38)	14 (77.8%)	7 (16.7%)	
Outcome			
Survival (n: 118)	48 (100%)	70 (71.4%)	<0.05
Death (n: 2)	0 (%)	2 (2.6%)	

positive group. Severe and very severe COPD was significantly associated with patients with trop I positive group.

7. CONCLUSION

Cardiac troponin I elevated in significant subset of patients with acute exacerbation of COPD. These patients had significantly higher incidence of hypoxia (low SPO2), tachycardia and tachypnea. P Pulmonale, enlargement of right heart chambers (Dilated RA and RV) and PAH was significantly higher in these patients. Hypoxemia and hypercapnia was significantly higher in these patients. It was a predictor of need for and duration of ventilator support. There was significant difference in duration of ICU and hospital stay in both groups. It predicted significant in hospital mortality. Further studies with quantitative estimation involving larger number of patients are recommended to evaluate whether long term outcome varies with patients with positive cardiac troponin I.

CONSENT AND ETHICAL APPROVAL

As per university standard guideline, participant consent and ethical approval have been collected and preserved by the authors.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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