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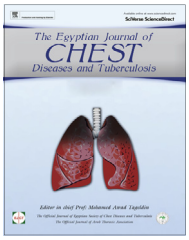

ORIGINAL ARTICLE

Serum Troponin-I as a prognostic marker in acute exacerbated chronic obstructive pulmonary disease patients

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KEYWORDS

Tropinin;
Exacerbation COPD

Abstract *Background:* Chronic Obstructive Pulmonary Disease (COPD) is a major cause of chronic morbidity and mortality worldwide. It is the fifth leading cause of death worldwide. 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. In RV failure, cardiac Troponins (cTn) are suspected to be elevated secondary to RV ischemia or micro infarction.

Objective: To evaluate the prognostic value of cardiac Troponin I level in acute exacerbated chronic obstructive pulmonary diseases (AECOPD) and its impact on the hospital outcome in those patients.

Patients and methods: This study was performed on 60 patients with AECOPD, admitted at Chest Department and respiratory ICU; Zagazig University hospital. All patients were subjected to: thorough history taking, clinical examination, electrocardiography and echocardiography, arterial blood gas (ABG) analysis, measure forced vital capacity (FVC) and forced expiratory volume in first second (FEV1), serum assay of cTnI level which is considered −ve if <0.01 ng/ml and +ve if $\geqslant 0.01$ ng/ml.

Results: cTnI was positive in 42 AECOPD patients. cTnI positivity was more prominent among patients with very severe exacerbation of COPD and in those with past history of LTOT, MV, ICU admission. Also, cTnI positivity was more in patients admitted to ICU rather than those managed

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in the ward and among patients who needed MV rather than who did not need it and in patients who failed weaned rather that who succeeded weaned. P-pulmonate, Rt. ventricular strain, high pulmonary artery pressure, hypoxemia and hypercapnia showed a great effect upon cTnI positivity. The duration of hospitalization was longer among cTnI +ve patients than cTnI −ve ones, Tropinin cutoff value for the prediction of death was >0.055 ng/ml with Sensitivity = 75%, Specificity = 68%.

Conclusion: Positive cTnI in AECOPD patients may suggest exacerbation severity and the occurrence of pulmonary hypertension and right ventricular dysfunction. Positive cTnI is considered as good prognostic marker for the possibility of a need for MV and a longer duration of hospitalization. MV may further elevate cTnI in AECOPD patients and with possible weaning failure. Negative cTnI and cTnI ⩽ 0.055 μg/L can be considered predictors of survival in AECOPD patients.

Introduction

Chronic Obstructive Pulmonary Disease (COPD) is a major cause of chronic morbidity and mortality worldwide. It is the fifth leading cause of death worldwide [1].

Exacerbation of COPD is an event in the natural course of the disease characterized by a change in the patient's baseline dyspnea, cough and/or sputum beyond day-to-day variability sufficient to warrant a change in management. Acute exacerbations of COPD (AECOPD) account for large amounts of the morbidity and mortality attributed to COPD [2].

In patients hospitalized for acute exacerbation of COPD cardiovascular risk factors and cardiac co-morbidity are common. Cardiac dysfunction may trigger acute exacerbation in up to 25% of these patients, whereas acute respiratory failure itself may lead to right or left ventricular failure (LV) [3].

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. Other cardiac diseases found frequently in patients with COPD, including coronary artery disease (CAD) and arrhythmias, present a unique challenge for clinician as the combination of both pulmonary and cardiac disease appears to be additive with regard to morbidity and mortality [4].

The cardiovascular alterations are extremely complex. During an episode of acute exacerbation, the increased work and oxygen cost of breathing, the increase in the left ventricular afterload 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 [3].

So identification of patients with cardiac injury may influence the treatment and outcome. Identification of cardiac dysfunction during acute COPD exacerbation remains difficult because of the non specific nature of clinical signs [5].

Therefore there is a need for a ''biomarker'' not only to provide confirmation of exacerbation but also to predict the severity of such events [6].

Cardiac Troponin I (cTnI) is a specific marker for cardiac injury. It is a component of the contractile proteins present in all muscles. The amino acid sequence of 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. It has no cross reactivity with the two skeletal muscle isoforms. CTnI is a highly sensitive and long-lasting marker of cardiac injury [7,8].

This work was carried out to evaluate the prognostic value of Troponin I level in acute exacerbated chronic obstructive pulmonary diseases (AECOPD) and its impact on the hospital outcome in those patients.

Patients and methods

The study included 60 patients with AECOPD who were admitted at R ICU and Chest Department, Zagazig University Hospitals. They were 39 males and 21 females, with an age range from 35–73 years and its mean 58.22 ± 8.37 years.

They were divided according to their management into:

Thirty-five AECOPD patients requiring mechanical ventilation.

Twenty-five A ECOPD patients non-mechanically ventilated either admitted in ward or RICU.

- Acute exacerbation of chronic obstructive pulmonary disease (AECOPD) was diagnosed on the basis of the patient's medical history, symptoms (increased dyspnea, increased cough and sputum with change of color, wheezing and chest tightness), physical examination and arterial blood gases (ABGs) according to Global Initiative for chronic obstructive Lung Disease (GOLD) guidelines [1].
- Respiratory failure was defined as a PaO_2 < 60 mm Hg and/or SaO_2 < 90% with or without $PaCo_2$ > 50 mm Hg in arterial blood gas measurements made while breathing room air at sea level (GOLD, 1)
- Patients were mechanically ventilated by Servo-i and Puritan Bennett 7200 and were on synchronized intermittent mandatory ventilation (SIMV) plus pressure support ventilation.

Exclusion criteria [9].

Patients were excluded from the study if they had concomitant severe diseases, such as: ischemic heart disease, previous myocardial infarction, heart trauma, aortic valve disease and hypertrophic obstructive cardiomyopathy with significant left ventricular hypertrophy, hypertension (malignant HTN), postoperative noncardiac surgery patients, renal impairment, hypothyroidism and hyperthyrodism.