Cardiac troponin trends and severity in chronic obstructive pulmonary disease exacerbation: Are limits properly associated for Intensive Care Unit outcome?

Sir,

We read the report by Noorain in the latest issue of Lung India with great interest. In this single-center prospective study, he tried to highlight the prognostic significance of admission cardiac troponin I (cTnI) in chronic obstructive pulmonary disease (COPD) exacerbation. This is an important and controversial issue because cardiovascular co-morbidities in COPD worsen the outcome of exacerbations. We are agree that cTnI could be a high-density determination factor correlated with Intensive Care Unit (ICU) admission and mechanical ventilator requirement. However, we consider that there are key some practical questions to be answered for a proper clinical extrapolation.

First, regarding cTnI threshold need more consideration. In previous studies by Baillard et al. and Martins et al., cTnI was shown in associations with mortality and worse outcomes. However, there is not clear reason for the threshold of cTnI 0.017 µg/L (0.17 ng/ml) chosen as the cut off point for statistical analysis in Noorain’s study. Is it calculated from receiver-operating characteristic curves or does it represent the mean value (mean value is 0.272 µg/L)? This aspect needs more definitions for clinical implications.

Second, the association of acute COPD exacerbation and other diagnosis such as pulmonary embolism (PE) as relatively frequently cause of increased plasma troponin levels is not clear and this is a key aspect for appropriate diagnosis in this study. PE is particularly difficult to diagnose in critically ill patients because tests that may be suggestive of physiological alterations compatible with PE (e.g., decreased oxygen saturation, increased plasma cTnI) are often nonspecifically abnormal in critically ill patients. Katsios et al. showed that pretest probability scores such as Geneva diagnostic PE score, Wells, modified Wells, and simplified Wells diagnostic scores developed outside the ICU do not correlate with adjudicated PE categories in critically ill patients.

In Noorain’s study probability of PE was evaluated by using Wells score. However, it was stated that 34 (68%) patients (94.7% in Troponin I positive group, 51.6% in Troponin I negative group) were admitted to ICU. He also stated that mean SpO₂ of Troponin I positive group was 75% and 63.2% of these patients had dilated right heart chambers. In our opinion, it seems to be difficult to rule out the PE with Wells score only. It was shown that the negative predictive values the Wells score combined with D-dimer is near to 100%. There were no data
related to patients’ D-dimer levels in this study. It might be better to combine Wells score with D-dimer to rule out PE. In addition, computed tomography pulmonary arteriography was not used for any cases, and it does not mean absolutely all cTnI positive patients do not have PE. There are no any data related to this situation that confirm solid associations of cTnI trends and levels.

Third, Noorain concluded that patients with cTnI elevation were more likely to require ICU care and ventilator support. However, other key additional factors for ICU admission and ventilator support need were not tested in a multivariate analysis. In this line, there are two key factors as SpO₂ (P = 0.003) and disease duration (P = 0.002) that seem to be significant at the end of univariate analysis but these parameters, in our opinion could be evaluated in the multivariate analysis with cTnI.

Fourth, regarding etiologies for acute COPD exacerbation such as decompensated heart failure that may represent the primary clinical problem haven't been evaluated. Although Noorain found that echocardiographically left ventricular dysfunction was 31.4% in cTnI positive group and it was 3.2% in cTnI negative group, final attribution of cTnI elevation to COPD exacerbation may be overlooked.

Fifth, illness duration of cTnI positive group was found longer by Noorain. However, any information about the severity of COPD such as forced expiratory volume in 1 second (FEV1), FEV1/forced vital capacity, GOLD stage, exacerbation rate, systolic pulmonary artery, pressure exercise capacity haven’t been deeply evaluated. This is important for association with disease stage and in-hospital mortality.

Sixth, 94.7% of cTnI positive group and 51.6% of cTnI negative group were admitted to ICU, but mechanical ventilation including noninvasive and invasive ventilation did not seem to be applied to all patients admitted to ICU.

Further prospective clinical trials need to confirm appropriate cTnI trends and value as biochemical markers of severity and practical decisions for ventilator support in COPD exacerbations.

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Conflicts of interest
There are no conflicts of interest.

Fatma Yıldırım, Antonio M Esquinas¹
Department of Pulmonary and Critical Care Medicine, School of Medicine, Gazi University, Ankara, Turkey, ¹Department of Intensive Care Unit, Hospital Morales Meseguer, Murcia, Spain
E-mail: fatma_bodur2000@yahoo.com

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