However, a potential important confounding factor may explain a part of their results: undiagnosed pulmonary embolism (PE), mimicking (or induced by) COPD exacerbation. Troponin and BNP are factors associated with poor prognosis in PE. COFDP is associated with an increased risk of deep venous thrombosis and PE (especially during exacerbation) and with an increased risk of fatal PE. In particular, COFDP is associated with increased risk of death from undiagnosed PE.

The real incidence of PE during exacerbation of COFDP is not clearly known, ranging from 1.5% to 24.7% corresponding to the incidence of elevated troponin and BNP, as noted by Chang et al in their cohort. Therefore, it would be of great interest if Chang et al could provide us some precise answers:

- In how many of the 250 patients a PE has been evoked and/or eliminated?
- How many patients were under efficient anticoagulant drugs at inclusion?
- How many patients received thromboprophylaxis, as a significant number of patients included presented other PE risk factors such as malignancy or cerebrovascular diseases?

Because of reserved prognosis of COFDP patients with PE, and of the availability of preventive and curative specific drugs, COFDP patients admitted with exacerbation and with abnormal cardiac biomarkers may require a PE screening and effective thromboprophylaxis if PE has been ruled out.

Laurent Bertoletti,1,2,3 Patrick Mismetti,1,2,3 Hervé Decousus1,2,3

1Université Jean-Monnet, Thrombosis Research Group, St-Etienne, France; 2INSERM, CIC-963, St-Etienne, France; 3Centre Hospitalier Universitaire, Service de Médecine Interne et Thérapeutique, St-Etienne, France

Correspondence to Dr Laurent Bertoletti, Centre Hospitalier Universitaire, Service de Médecine Interne et Thérapeutique, CHU de St-Etienne, Saint-Etienne 42023, France; laurent.bertoletti@gmail.com

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Authors’ response

We thank Bertoletti and colleagues for raising the important issue of pulmonary embolism (PE) in the exacerbation of chronic obstructive pulmonary disease (COPD). Although we did not routinely investigate PE in our cohort, we excluded any patients with suspected or confirmed PE from the study. Unfortunately, it is difficult to detect thromboembolic events in this population and it is possible that we included some patients with subclinical pulmonary emboli. It is also plausible that this contributed to the association between elevated cardiac biomarkers and mortality. However, we think that this is unlikely to be the only mechanism.

Thromboprophylaxis was administered to some patients during their admission depending on their immobility and other risk factors, but this would not have influenced the NT-proBNP or troponin T results obtained on presentation. We did not collect information on pre-existing anticoagulation therapy on admission to the study.

Further research into the mechanism linking elevated cardiac biomarkers and mortality in COPD exacerbation is needed. We agree with Bertoletti and colleagues that investigating the contribution of concurrent PE is important, as this is something that can be treated.

C L Chang, R J Hancock1,2

1Department of Respiratory Medicine, Waikato Hospital, Hamilton, New Zealand; 2Department of Preventive and Social Medicine, University of Otago, Dunedin, New Zealand

Correspondence to Dr C L Chang, Department of Respiratory Medicine, Waikato Hospital, Level 01 Menzies Building, Waikato Hospital, Hamilton 3204, New Zealand; contact_cat@hotmail.com

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We thank Bertoletti and colleagues for raising the important issue of pulmonary embolism (PE) in the exacerbation of chronic obstructive pulmonary disease (COPD). Although we did not routinely investigate PE in our cohort, we excluded any patients with suspected or confirmed PE from the study. Unfortunately, it is difficult to detect thromboembolic events in this population and it is possible that we included some patients with subclinical pulmonary emboli. It is also plausible that this contributed to the association between elevated cardiac biomarkers and mortality. However, we think that this is unlikely to be the only mechanism.

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