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LETTERS TO THE EDITOR

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Reversibility of acute pulmonary hypertension after resolution of exacerbation in patients with COPD

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To the Editor,

The presence of pulmonary hypertension (PH)in patients with chronic obstructive pulmonary disease (COPD) is associated with an increased risk of acute exacerbation (AE-COPD) [1]. Furthermore, when admitted for AE-COPD, patients with associated PH are at increased risk of lengthy hospital stays and decreased survival [2]. It has been previously reported that pulmonary artery pressure (PAP) can acutely increase during the episodes of AE-COPD presumably due to pulmonary vasoconstriction resulting from arterial hypoxemia [3]. Using right heart catheterization (RHC, the gold standard tool for precise PAP measurement), we retrospectively assessed RHC-derived hemodynamic measurements during- and 6 weeks after the resolution of AE-COPD in 13 patients with severe to very severe COPD. Details of the RHC methodology and derived measurements were previously described [4]. Patients (age: 55 ± 8 years (mean ± SD), 92% males and 11/13 were current smokers) had very severe COPD (forced expiratory volume in 1 s: 25 ± 8%predicted and forced vital capacity: 46 ± 11%predicted)). An arterial blood gas on admission showed PaO_2, 56.5 ± 4.7 mmHg; $PaCO_2$, 76.3 ± 7.2 mmHg; and $HCO3^-$, 26.4 ± 1.6 mEq/L, while breathing room air. Initial RHC measurements showed systolic PAP of 39.1 ± 10.5 mmHg, mean PAP of 27.2 ± 4.4 mmHg, and pulmonary vascular resistance (PVR) of 3.79 ± 0.71 wood units. Cardiac output (CO) and cardiac index (CI) and pulmonary artery wedge pressure (PAWP) were all within the normal range (5.1 ± 0.5 L/min, 3.0 ± 0.4 L/min/m^2, and 7.8 ± 2.2 mmHg, respectively). None of the patients required invasive mechanical ventilation, and AE-COPD was treated according to guidelines at the time of the study [5]. A repeat RHC done 6 weeks following the resolution of AE-COPD showed a drop in systolic PAP, mean PAP, and PVR by 7.1 ± 6.9, 5.7 ± 7.1, and 12.7 ± 11.6%, respectively (all $p < 0.05$), with no significant change in CO, CI, and PAWP (Fig. 1). PaO_2 and $PaCO_2$ also significantly improved on follow up.

It is currently well-acknowledged that the likely mechanism of elevated PAP in patients with COPD is an increase in PVR rather than changes in the driving pressures within the pulmonary circulation (i.e., changes in CO and PAWP) [1, 4, 6]. Increased PVR in patients with COPD is, in turn, closely related to arterial hypoxemia leading to pulmonary vasoconstriction and/or pulmonary vascular remodeling directly induced by tobacco smoking [4, 7–10]. Indeed, PAP is expected to increase during episodes of acute exacerbation in patients with COPD as a result of associated gas exchange abnormalities (hypoxemia with or without hypercapnia) leading to pulmonary vasoconstriction and an increase in PVR [11, 12]. Increased air trapping due to tachypnea and worsening of airflow obstruction could also lead to an increase in PAP during AE-COPD [6, 13]. Thus, after the complete resolution of AE-COPD and the improvement in

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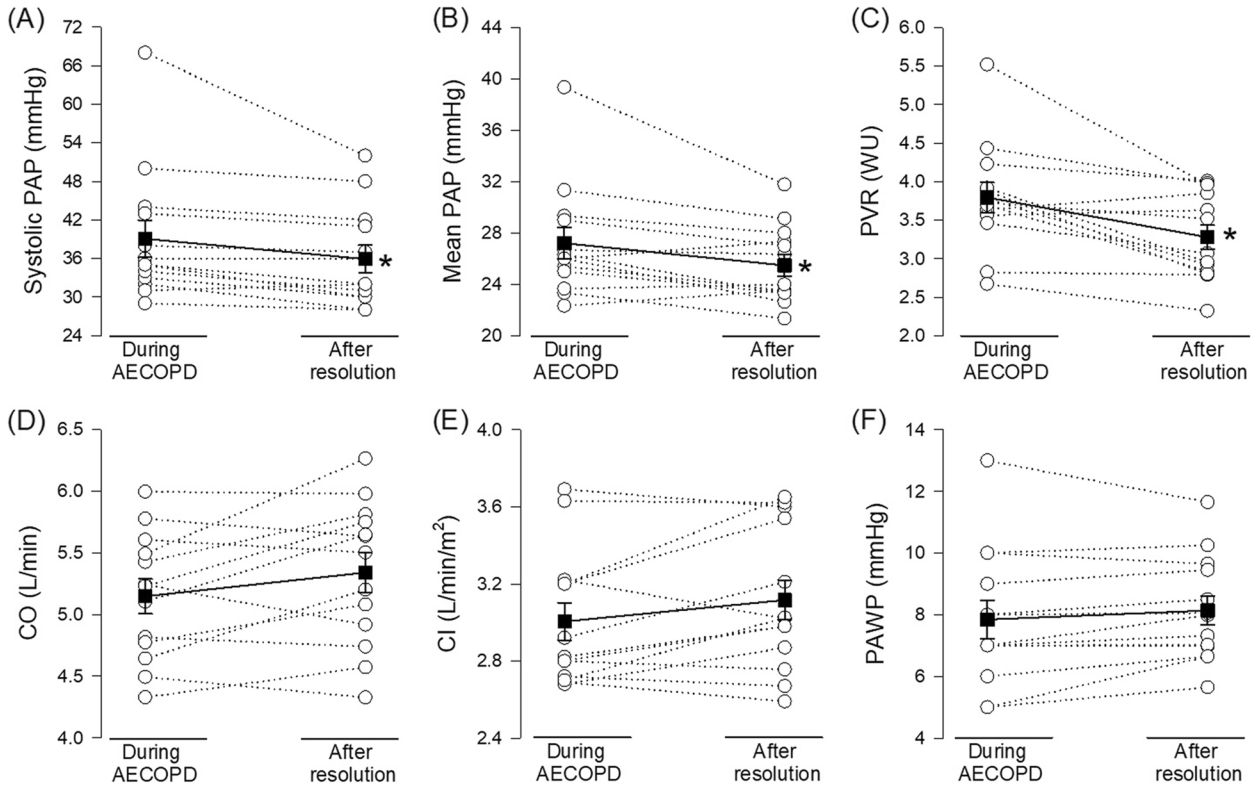

Fig. 1 Right heart catheterization measurements during and 6 weeks after the resolution of acute exacerbation of chronic obstructive pulmonary disease (AE-COPD). The figure shows individual data ($n = 13$) with square symbols representing the means ± SEM. *$p < 0.05$ during AE-COPD versus 6 weeks after resolution. *Abbreviations:* CI, cardiac index; CO, cardiac output; PAP, pulmonary artery pressure; PAWP, pulmonary artery wedge pressure; PVR, pulmonary vascular resistance; WU, wood units

pulmonary gas exchange, PAP could theoretically return to its baseline value before the incident of the acute exacerbation. Supporting this notion and in the current analysis, we included a well-characterized sample of patients with COPD, and despite being a small sample, it was enough to show significant reductions in both PVR and PAP pressures following the complete resolution of AE-COPD (Fig. 1). Future well-designed larger studies are required to further examine the effect of AE-COPD on pulmonary artery pressure and assess its long-term course.

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Authors' contributions
All authors meet the criteria for authorship as recommended by the International Committee of Medical Journal Editors. All authors played a role in the content and writing of the manuscript. In addition, M.E.A., Y.M.K., and A.F.E. provided the original idea for the study; A.F.E. wrote the first draft of the manuscript, and all authors have read and approved the manuscript.

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Availability of data and materials
The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate
This is a retrospective analysis and measurements were performed during different clinical research studies (***unpublished data***) that were ethically approved by the ethical committee of Alexandria University (Egypt). Written informed consent was obtained from all patients before their initial study participation.

Consent for publication
Not applicable.

Competing interests
The authors declare that they have no competing interests.

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