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CORRESPONDENCE

Air leak, barotrauma susceptibility, and imaging in acute respiratory distress syndrome: novel application of an old tool

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We read with great interest three recent articles published on Intensive Care Medicine [1–3]. In the first, Knox and colleagues present data on rate of pneumothorax/pneumomediastinum in 2211 patients with coronavirus disease 2019 (COVID-19)-related acute respiratory distress syndrome (ARDS) as compared with 5522 patients with non-COVID-19 ARDS [1]. Data from this large population confirm that these events occur more frequently in patients with COVID-19 ARDS as compared with non-COVID-19 ARDS [4].

We also read with interest the review of Bitker and colleagues on the role of imaging in ARDS [2]. The authors should be congratulated for the detailed description of the use of imaging techniques in management of ARDS.

Interestingly, we have recently demonstrated that a radiological sign, the Macklin effect (also known as interstitial emphysema, visible on chest computed tomography (CT) images as a linear collection of air tracking along bronchovascular bundles, visceral pleura and/or interlobular septa, in a sort of "double-profile" shape [5]), is an accurate predictor of barotrauma in patients with COVID-19 ARDS (sensitivity: 89.2% [95% confidence interval (CI) 74.6–96.9]; specificity: 95.6% [95% CI 90.6–98.4]) and is detectable 8–12 days before clinically overt barotrauma [5]. In short, this radiological sign "captures" air leakage centripetal movement along pulmonary interstitium following alveolar rupture [5]. A possible pathophysiological explanation for the impressive accuracy of Macklin effect in predicting barotrauma could lie in the excessive respiratory effort typical of patients with respiratory failure, which may lead to increased transpulmonary pressure and distal airway rupture. This could happen also due to cough efforts. This hypothesis has been supported also by other authors, as detailed in another recent editorial [3]. Furthermore, the risk of alveolar damage and rupture may be exacerbated by specific conditions, such as COVID-19 ARDS, which is characterized by microthrombi formations in the pulmonary circulation with the potential risk of superimposed ischemic damage to lung cells, and hence increased barotrauma susceptibility.

We hypothesized that early identification of Macklin effect could be used to identify a novel ARDS sub-phenotype characterized by increased barotrauma susceptibility and disease severity; ultimately, these findings could help selecting patients for different treatment algorithms. For example, patients not receiving invasive mechanical ventilation may be selected for advanced monitoring of respiratory mechanics using esophageal pressure to detect patient self-inflicted lung injury (P-SILI) early and be candidates for strategies such as awake pronation, or decision to proceed to early intubation and institution of invasive protective mechanical ventilation. Alternatively, patients may be candidates for use of awake extracorporeal support or ultraprotective invasive ventilation. This might be particularly important during pandemic surges with shortage of intensive care unit (ICU) beds, as delayed intubation has been associated to increased mortality. Notably, avoidance of inspiratory and expiratory intrathoracic pressure swings should be the goal of ARDS management even in absence of Macklin effect. Of

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course, the possible benefit of early detection of Macklin effect should be balanced against the risk and costs of systematic CT scanning of high-risk ARDS patients. Furthermore, therapeutical applications of Macklin sing are attractive but require clinical confirmation, and potential benefits of early invasive ventilation should be balanced against risk of ventilator-induced lung injury (VILI) and prolonged mechanical ventilation.

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Declarations

Conflicts of interest
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